Aggression

Psychiatric Assessment and Treatment

edited by

Emil F. Coccaro

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The University of Chicago

Chicago, Illinois, U.S.A.

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Series Introduction

Our newspapers are filled with headlines of suicide bombings, of armies massing, and of the deaths of children from abuse. Television news reports similar stories and shows us crime scenes in all too vivid color, the crumpled cars that killed mothers and children, and the bloody dead on the sands of Afghanistan. Our world is a dangerous place, made all the more so by our human behaviors. Despite this, there has been little study and even less understanding of the origins of what we do to hurt each other.

This volume is the outcome of two meetings held over the past several years in an attempt to begin to provide the kind of information essential to furthering the study of this centrally important issue. Although Freud speculated about aggression and tentatively postulated a death instinct opposed to the power of Eros, few have explored these issues in continuing and focused research programs. *Aggression: Psychiatric Assessment and Treatment* provides us with a set of conceptual frameworks, measures of aggression and impulsivity, and a series of approaches to containing the problem. A distinguished group of chapter authors provide us with a summary of what we know and what we need to know, and a set of potential road maps for accumulating the data sets so necessary to future planning of both prevention and treatment.

This is an important and useful book for all of us who care about what the future holds for our children and for the world we have known.

William A. Frosch

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Preface

The genesis of this book dates back to the summer of 1996 when a group of clinical investigators first met in Philadelphia to discuss the state of human aggression research. At the time little was formalized in the way of a system of measures or concepts characterizing primary human aggression as a clinical disorder. A second, similar, meeting was held two years later, at which a plan was formulated to produce a volume containing comprehensive and specific summary articles on primary human aggression that would be of benefit to both clinical researchers and clinicians. The need for such a volume is clear. Despite the apparent relevance of aggression to society, and the emerging empirical scientific literature strongly indicating that aggression, particularly impulsive aggression, is rooted in complex genetic, biological, and psychodevelopmental relationships that can be identified and targeted for treatment intervention, there are few, if any, officially recognized clinical models that allow for the identification of individuals with clinically significant problems of aggression. Without such models there can be little relevant research. Without such research there can be few advances in knowledge that will be useful to those who attempt to study and treat such individuals.

This volume is divided into four sections, based on the idea that a discussion of general models of aggression should be followed by a discussion of more specific clinical models, a discussion of the clinical and research measurement of aggression and impulsivity, and, finally, a discussion of the treatment of human aggression as seen in clinical contexts. Our focus on primary human aggression is noteworthy because the vast majority of the literature is directed toward books on aggression in general, aggression due to other conditions, or aggression in the context of antisocial behavior. This book is concerned with aggression that does not stem from medical, pharmacological (e.g., during intoxication or withdrawal), or psychiatric (e.g., psychosis or mania) factors or from premeditation; accord-

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ingly, it is behavior that is not secondary to some other obvious factor associated with a condition or state about which a good deal is already known.

The first section discusses issues regarding possible models of aggression, including phenomenological models (Chapter 1), genetic models (Chapter 2), developmental models (Chapter 3), and biopsychosocial models (Chapter 4). The fifth chapter considers impulsivity because of its great relevance to aggression, especially impulsive and/or affective or reactive aggression. These chapters set the stage for the chapters in the second section, which discuss clinical models of aggression and anger.

The second section presents four clinical models of aggression that are in various stages of development. Chapter 6 discusses the research category of anger disorders, while Chapter 7 describes a variant of depressive disorder characterized by anger attacks. Chapter 8 discusses the clinical construct of impulsive aggression and Chapter 9 examines Intermittent Explosive Disorder as a categorical expression of impulsive aggression.

The third section focuses on measures of aggression and impulsivity. These chapters provide comprehensive reviews of how aggression and impulsivity can be measured in human and in clinical populations. Chapter 10 presents a comprehensive and critical overview of questionnaire and interview measures of aggression in adults, while Chapters 11 and 12 discuss the two most widely used laboratory measures of aggression. Chapter 13 is similar to Chapter 10 in that it presents a comprehensive review of various psychometric and clinical assessments of impulsivity. Chapter 14 is similar to Chapters 11 and 12 but focuses, instead, on laboratory assessments of impulsivity in human subjects. Chapter 15 concludes this section with a comprehensive review of aggression and impulsivity measures available for use in children and adolescents. The reader interested in obtaining a copy of a specific assessment presented in any of these chapters may refer to the published source cited or to the author of the particular chapter.

The fourth and final section of this volume deals with the clinical treatment of primary aggression. Chapter 16 follows up on Chapter 6, discussing the psychosocial treatment of anger disorders. Chapter 17 does the same for partner aggression. The final three chapters discuss psychopharmacological intervention using neuroleptics and lithium, serotonergic agents, and, finally, anticonvulsants.

Emil F. Coccaro

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1

Phenomenological Models of Aggression and Impulsivity

Implications for Clinical Research and Treatment of Human Aggression

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INTRODUCTION

All of science, including clinical science, begins with observation. The understanding and modulation of aggressive impulsive behavior has been no exception. Even before Hippocrates' attempt to characterize personalities (as phlegmatic, choleric, and sanguine), we have observed and grouped behaviors and then proceeded to study and attempt their manipulation. Webster defines phenomenology as "the branch of a science that classifies and describes its phenomena without any attempt at explanation." It is this classification and description process which has guided the research and clinical modulation of human aggressive behavior utilizing animal models of aggression.

The process of examining the phenomena of aggressive and impulsive behavior has evolved from classical writers including Aristotle, Hippocrates, and Plato continuing through the writers of the Romanticism and Realism schools

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with Locke and Rousseau, into the pre-20th century clinical "scientists" such as Freud. The study changed form to a more structured scientific process after Darwin's *Origin of Species*. Researching animal models of behavior and emotion became a strategy of choice as science attempted to understand man's own behavior and emotions.

This chapter reviews a representative segment of the process of examining phenomenological animal models of aggression and how they guide the study of human aggressive behavior and its modulation.

BASIC CONCEPTS

The underlying premise of the phenomenological study of aggressive behavior is that such aggressive behavior is not uniform but despite its disparity can be grouped according to certain externally observable characteristics. Moreover, the utility of such descriptive grouping provides the structure that leads to a clearer understanding of these phenomena and affords a means to manipulate behaviors. The study ultimately provides an understanding of behavior in the human condition. Said another way, animal models of aggression tell us which questions to ask about human aggression and which biological systems to study in the human animal.

The classical characterization of animal aggression, which has now stood the test of time, is Moyer's classification first published in 1968 (1). He divided animal aggression into seven classes: predatory, intermale, maternal, territorial, fear-induced, irritable, and instrumental. These categories have analogs both in the natural environment and in the laboratory; categories cross phylogenetic lines and can be durably reproduced.

Predatory aggression is self-explanatory on the African veldt or with the barnyard cat. Taken into the laboratory, it has been studied as the "quiet biting attack" of a cat toward a rat (2) or the mouse-killing behavior of rats (3) or even the cricket-killing behavior of mice (4). The attack patterns of the predator are stereotyped, and there is a minimal display of affect or sympathetic arousal. In the laboratory, the latency to attack and the number of attacking animals become the parameters of study.

Inter-male aggression in the field often involves the establishing of social hierarchies, for example, in primate colonies. It may also foster extrusion from a herd or group fostering a species' dispersion. Its classical laboratory analog has been isolation-induced fighting in mice (5,6) where two male mice isolated for several weeks are then placed together. This pair fights until dominance is established. Latency to attack and fight duration are the parameters of study.

Maternal aggression has long been recognized in the field where attacks can readily be elicited by an unwary hiker who finds himself between a grizzly bear sow and her cub or between an Alaskan moose and her calf. Such aggression

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is linked to the presence of young and to the lactating endocrine state (7,8). It can be considered both a special subset of predatory aggression and intruder aggression. Again, latency to attack and the duration of the attack are laboratory parameters for study.

Territorial aggression has been well described by the ethologists in species as disparate as the stickleback fish (9) and the Uganda kob (10). It has been studied as a laboratory model of resident-intruder aggression primarily using rats. In this paradigm, an intruder, usually a male rat, is introduced into an established small colony. Such an introduction will generate an offensive attack by the resident male or males of the colony. Again, the latency and duration of such attacks become the parameters for study.

Fear-induced aggression is illustrated by the behavior of cornered animals. It is marked with intense affective display and characteristic expressions and posturing (11,12).

Irritable aggression is defined by the characteristics of the inducer—being a noxious stimulus. To the extent that the stimulus inducing fear is "noxious," fear-induced aggression might be considered a subset of irritable aggression. However, in the laboratory, the classical model of irritable aggression has been pain-induced aggressive behavior (13). Across species as disparate as snakes and monkeys (14,15), if two conspecifics are given a painful stimulus (in the laboratory, this is a foot shock), they will follow that foot shock with an attack toward the other animal. The laboratory paradigm records the percentage of attacks for a given number of aversive foot shocks. This is the usual experimental variable.

Instrumental aggression was Moyer's final category. In the laboratory it may be the most artificial. Positive (16) and negative (17) reinforcers are utilized to shape and increase the frequency of aggressive behavior. Since aggressive behavior, which affords dominance, can be positively reinforcing, instrumental aggression can be linked with other forms of aggression such as intermale aggression.

Not all categories of aggression fall into Moyer's seven types. Monkeys reinforced on a fixed-ratio operant schedule of reinforcement will become aggressive and attack even proximate inanimate objects (18) when the ratio of behavior to reinforcement is increased too high and too rapidly. This behavior fits the "frustration-aggression" theory of Dollard and Miller (19) but must be stretched to be incorporated into the irritable aggression or instrumental categories of Moyer.

Other phenomenologists have attempt to simplify aggressive behavior into just two categories: predatory aggression and affective aggression (20,21). Reis argued that all categories of aggressive behavior except predatory aggression were accompanied by marked affective arousal and seemed to have many common neurochemical correlates. However, there seem to be physiological and endocrinological differences among the categories of offensive aggression (territo-

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rial and intermale), defensive aggression (fear-induced and irritable), and maternal aggression despite the observation that all of them elicit a significant degree of emotional and sympathetic arousal.

CLINICAL RESEARCH AND TREATMENT RELEVANCE

Beyond the intrinsic "beauty" of describing and classifying, both the researcher and the clinician may require some utility from the study of animal models of aggression. The central thesis of this chapter is that such models do, indeed, have utility both for the researcher of human aggression and the practicing clinician. Several areas can serve to illustrate this premise. Not only can animal model research guide our clinical research, it can guide the way we conceptualize our patients and, using that conceptualization, guide our treatment.

Typology

The animal literature underscores the need to conceptualize aggressive behavior as a collection of behaviors having both shared and distinctive typologies as well as shared and distinctive neurological underpinnings. Brain lesions as well as drugs can produce alternations in one category of aggression distinct from others. Sensory impairment may be critical to one form of aggression, but not to others.

For example, in rats, lesions to the septal nuclei and to the ventromedial nucleus of the hypothalamus (VMH) both increase irritable (pain-induced) fighting. However, the septal rats show an increase in defensive fighting with an increased frequency in upright boxing posture and bites to the opponent's snout whereas VMH rats display an increase in offensive fighting with bites to the opponent's rump (22).

Drugs, too, have differing and even converse effects depending on the type of behavior. Tricyclic antidepressants can enhance affective aggression (23), but they suppress predatory aggression (24).

The role of sensory systems also varies among animal models in enhancing or suppressing particular types of aggressive behavior. Removal of olfactory input (through lesions or the olfactory bulb or even through genetically engineered TRP2 receptor depletion to the vomeronasal organ) in mice suppresses intermale aggression (25,26), but similar olfactory bulb lesions in rats minimally affect irritable aggression (27) and dramatically enhance predatory aggression (27). Removal of the vibrissae of rodents has little to no effect on intermale aggression or predatory aggression, but dramatically suppresses rodent irritable aggression (27).

Social Context

Social context may play a role in either an endogenous chemical change or the effect of an exogenously administered agent. For example, peripheral serotonin

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chemistry appears to change in primates as they ascend to the alpha male role in a colony. Whole-blood serotonin levels are high in an alpha male primate (28). If the primate is removed from that position, his level drops. For the beta male ascending into an alpha position, whole-blood serotonin levels rise (28). In intermale fighting in mice, both mice fight for the same amount of time; however, the activity of the adrenal enzyme PMNT, necessary for the synthesis of adrenal epinephrine, is much higher in the mouse that subsequently becomes the submissive one in the dyad (29).

Exogenous drug effects may also be different. In mice which have been characterized as dominant or submissive within intermale paradigms, alcohol can increase aggression in the submissive mouse, but may produce sedation in the dominant member of the dyad (30).

Temperament

Years ago, Scott and Fuller (31) characterized the temperaments of various breeds of dogs. More recent studies within laboratory rodents and feral mammals have demonstrated that various animal strains differ widely in their levels of aggression. Moreover, they may be strongly aggressive in one category and weakly aggressive in another. NIH Wistar female rats are highly aggressive (fighting at $>50\%$ in the test paradigm) in irritable aggression, but display no predatory aggression toward mice (32). Irritable aggression levels in the laboratory can show a 30-fold variance, ranging from 67% attack rates for male Osborne Mendel rats to 2% for female Brookhaven "sensitive" rats. Such variability can be selectively bred in relatively few generations. This has been readily demonstrated for both laboratory (33) and feral (34) animals.

Interestingly, even when animals are selectively bred for other behaviors, they may also differ markedly in relation to specific categories of aggression. Maudsley rats selectively bred for their performance in the open field into two variants: nonreactive (rats that do not freeze in the open field) and reactive (those that do) show nonoverlapping differences in irritable aggression. The Maudsley nonreactive rats are three times more aggressive than the "fearful" Maudsley reactives. A similar difference is noted between the rats bred for successful performance on an active avoidance learning task. Roman High Avoidance rats were almost six times more aggressive than Roman Low Avoidance counterparts. In both the Maudsley and Roman breeding, rats bred for a "go" versus "no go" diathesis were more aggressive when tested in an irritable aggression paradigm (32).

Strains genetically disparate for aggression can be studied neurochemically and pharmacologically. Rats genetically bred for muscarinic sensitivity (Flanders Sensitive) are more aggressive within an irritable aggression paradigm (35). Across genetic strains, differences in biogenic amines such as serotonin (36) and

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norepinephrine (37) as well as second-messenger systems (38) show correlations between the activity of these neuronal systems and aggressive behavior.

Neuroanatomy, Neurophysiology, and Neurochemistry

Various animal models have provided templates for neuroanatomical research. Initially, deep, phylogenetically primitive brain areas were observed to modulate aggression. Hess (39) demonstrated that diencephalic electrical stimulation could induce feline rage. Shreiner and Kling (40) demonstrated that amgydala lesions could attenuate aggressive behavior in feral cats. Using rodent and feline animal models systems, neuroanatomical sites and pathways could be precisely localized and quantified, demonstrating both facilitatory and inhibitory brain regions. For example, within the limbic system, lesions of the septum and ventromedial hypothalamus enhance irritable aggression, while lesions of the amygdala and cingulated cortex diminish it (22). Such studies began to provide a background of information that has continued to propel research toward answering the question of "how" types of aggressive behavior are initiated and modulated. This neuroanatomical work flowed naturally into using such animal model systems to delineate neural networks, neurotransmitter systems, and more general principles germane to an evolved conceptualization of aggressive behavior.

In the neuropsychological sphere, using the model of electrical stimulation of predatory aggression, Bandler and Flynn (41) demonstrated that brain activity within the hypothalamus (an "excited hypothalamus") enlarges dramatically the sensory trigger points on a cat's paw which, when touched, generate a predatory attack on a rodent target. This electrically enhanced predatory aggression may share a common "kindling" mechanism with the increase in irritability found in rats kindled to develop limbic seizures through chronic lidocaine injections (42).

Neurochemical and pharmacological research concerning aggression has utilized animal models such as the predatory model of mouse killing, the intermale model of isolation-induced fighting in mice, the irritable aggression model of shock-induced (or pain-induced) fighting in rats, and the intruder model in the rat.

The earliest pharmacological work catalogued the effect of various drugs on animals within specific paradigms such as isolation-induced fighting (intermale), or irritable (shock-induced) aggression (43,44). This level of research was replaced by more sophisticated modulation of specific neurotransmitter systems with concomitant assessment of aggression within specific animal model systems.

Research on the serotonin system studied both in relation to predatory aggression, using the mouse-killing model and affective aggression using the model of shock-induced fighting illustrates this neurotransmitter approach. In the case of serotonin (5-HT), in general, manipulations, which diminish 5-HT brain activity,

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enhance both predatory and affective aggression. For example, reduction in brain 5-HT through the inhibition of tryptophan hydroxylase induces mouse killing in nonkiller rats (45) and reduces the latency to kill in killing rats (46). Similarly, inhibition of tryptophan hydroxylase using parachlorophenylalanine increases shock-induced aggression in rats under appropriate stimulus parameters (47).

Serotonin can be reduced in brain in other ways as well. Brain serotonin levels are controlled by the serotonin dietary precursor tryptophan, an essential amino acid. Depleting a rat's diet in tryptophan lowers whole-brain serotonin and induces mouse killing (48) and increases shock-induced fighting in rats (49). Injury to serotonergic nerve terminals using the neurotoxin 5,7-dihydroxytryptamine similarly induces predatory rodent aggression (50) and enhances irritable aggression (51). Finally, neurochemical (50) or electrolytical (52) destruction of the serotonergic raphe nucleus can also induce predatory and irritable aggression.

The converse of enhancing the serotonin system is generally ineffective within rodent models in reducing aggressive behavior. Dietary tryptophan loading, despite increasing brain serotonin levels, does not suppress irritable aggression in untreated laboratory rats (49,53). However, if such rats are made "pathologically aggressive" with septal brain lesions or neurotoxin treatment (5,7-dihydroxytryptamine), then exogenous tryptophan, which induces an elevation in brain serotonin, does block the lesion or toxin-induced enhanced aggression (53).

As the pharmacology of neurotransmitter systems has become more complex, animal models have been useful in clarifying which receptor system for a particular neurotransmitter is most likely responsible for the behavioral effects observed. This can be done by studying in depth the pharmacology of a new or old drug effective in modulating aggressive behavior, studying the behavioral effect of a drug whose receptor pharmacology is known, or utilizing newer technologies such as gene knockouts to identify the critical nature of specific receptor systems in relation to a particular type of aggressive behavior.

This use of animal model systems can be illustrated with the study of serotonin receptors. A battery of serotonin-active agonists can be studied using the resident-intruder model of rodent aggression. This model affords not only an assessment of aggressive behaviors, but also of competing behaviors such as "social interest," "exploration," and "inactivity." Of the drugs tested, those serotonin agonists affecting the serotonin 1B receptor, those that are mixed agonists for the 1A and 1B receptors, and those that are general agonists for 5-HT1 and -2 receptors all show a dose-dependent inhibitory affect on intruder aggression (54). This implication, particularly of the 5-HT1B receptor gained further support which 5-HT1B knockout mice were also demonstrated to be markedly aggressive in an intermale aggression paradigm (55).

Behavioral pharmacology using various rodent models of aggression has also implicated additional neurotransmitter systems. For example, pharmacologi-

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cal or neurochemical treatments that increase central noradrenergic activity through increasing the firing of the noradrenergic locus coeruleus (56), inducing brainstem tyrosine hydroxylase (the rate-limiting enzyme in the synthesis of NE) (57,58), or increasing central NE turnover (59,60), all appear to enhance irritable aggression in rodent models. [This facilitatory effect of NE is less consistent across treatments compared to the modulation of the serotonin system since depletion of brain NE using the neurotoxin 6-hydroxydopa increases irritable aggression (61) while intracerebroventricular infusion of NE depresses irritable aggression (62).]

Receptor mediation of these effects may be occurring in the rat β-adrenergic receptors since rats with an increased number of cortical β-adrenergic receptors show increased irritable aggression in this supersensitive state (63).

Dopamine (D_2) blockade suppresses affective aggression in rodents but always at doses where other behaviors such as locomotion are also affected. Central intraventricular dopamine infusion increases shock-induced fighting (62) and L-dopa (64) or the DA agonist apomorphine (65) both induce defensive aggressive behavior in rats kept in close proximity.

Acetylcholine appears to have both a facilitatory and an inhibitory role in rodent affective aggression models. Muscarinic stimulation appears facilitatory and nicotinic stimulation appears to be inhibitory (66,67).

Initial research modulating the GABA/benzodiazepine system suggested a simple inhibitory effect for relatively high doses of benzodiazepines (68). This was consistent with studies showing enhanced aggression with GABA blockade (68). However, reports were published of benzodiazepines enhancing aggression in rodent animal models (69). Careful review of this literature (70) now appears to indicate that benzodiazepines may increase aggressive behavior in rodents that have low baseline levels of aggression. When baseline levels are high, such drugs have little effect until used at doses that also induce nonspecific sedation.

As new neuromodulators have been discovered and studied, they, too, have been tested in these now-standardized laboratory paradigms. These more recent discoveries have added to the broadening list of CNS modulators. They include enhancing neuropeptides such as vasopressin (71) or cholecystokinin (72), nonamine neurotransmitters such as nitric oxide (73), and, most recently, brain-derived neurotrophic factor (BDNF). BDNF knockout mice behave like 5-HT-depleted mice with enhanced aggressive behaviors (74).

Developmental Factors and Stress

Environmental modulation of an organism can occur in the context of a developing organism through differences in circulating maternal hormones or circulating drugs such as alcohol or cocaine. The environment may impinge on the organ-

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ism as a whole, inducing chronic stress. Within the laboratory, rodent models of affective aggression have provided prototypic examples of both mechanisms of modulation.

For many rat strains, irritable aggression is greater in males than in females, appearing to have some degree of testosterone sensitivity. However, this is not purely a direct effect of testosterone on the brain but, rather, an example of the brain organizing to maintain sensitivity to testosterone in adulthood. Using the laboratory paradigm of irritable aggression (shock-induced aggression), Connor et al. (75) demonstrated that male rats castrated at birth—while brain organization is still occurring—fought less than noncastrated male controls. Similarly, male rats castrated later (at weaning) also fought at a lower level. However, when both castrated low-fighting rat groups were given exogenous testosterone, only the late-castrated rats could respond to this hormone with enhanced aggressive behavior. The early-castrated rats did not have the neural organization to respond to testosterone. The hormonal milieu during fetal brain development controlled the hormonal response of adulthood in this rodent experiment.

Environmental changes, which induce extreme stress in animals, can induce not only acute changes in aggression, but also long-standing durable changes in aggressive behavior and central neurotransmitter systems. Acute sleep deprivation in rats induces a marked increase in shock-induced fighting (57) associated with increased brainstem tyrosine hydroxylase activity and a down-regulation of cortical beta adrenergic receptors (63). More remarkable is the long-term effect of chronic stress on rodent aggression. Young rats immobilized for 2 hours a day over 1 month are, at the end of the month, hypertensive, irritable, and three times as aggressive in the shock-induced fighting paradigm. After 1 month, they again become normotensive and are docile to gentle handling. However, when presented with an aversive foot shock stimulus, they again become aggressive, two to three times more so than their unstressed counterparts (58).

Aggression as a Coping Strategy

The rodent paradigm of shock-induced fighting has generated a provocative observation regarding aggressive behavior as a stress management strategy. In the standard paradigm, two rats receive electric foot shocks delivered to them within the confines of an enclosed environment. In this condition, laboratory rats attack each other approximately 30% of the time they are foot-shocked. Assessment of stress in this setting indicates both a decrease in tail blood pressure (76) and a minimal release of corticotropin (77). In contrast, a rat alone in such a setting shows a marked increase in tail blood pressure and a substantial increase in circulating corticotropin. The aggression paradigm appears to be physiologically less stressful than the experience of receiving the noxious stimulus without the opportunity to aggress.

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IMPLICATIONS FOR CLINICAL RESEARCH AND TREATMENT

Typology

The phenomenological study of animal aggression and the development of animal models of aggression have shown that aggressive behavior is not a unitary behavior. Its instigation, neural mechanisms, and potential for modification are multifactorial. Moreover, neuropharmacological modification of different classes of aggressive behavior by a single agent may have antithetical effects. Therefore, clinical aggression research must be committed to the premise of studying and clinically treating a class of behaviors instigated and modulated in and by a multifactorial system.

Progress regarding instigators, targets of the aggression, characteristics of the behavior, the role of sensory input into initiating the behavior, and the clarification of the neural processes needed for each class of aggressive behavior becomes more manageable and even, over time, feasible by starting with a phenomenological approach to these types of aggressive behaviors.

Problematically, clinical research and treatment continue to lack such a unifying accepted nosology necessary to phenomenologically classify clinically relevant and nonclinically relevant behaviors. Attempts have been made with the American Psychiatric Association's Diagnostic and Statistical Manual (78) structure and, independently, the Carolina Nosology for Destructive Behavior (79). However, more development and unanimity in this area are critically needed.

Social Context

Animal models of aggression provide a warning to clinical researchers to take into account the social context of the behavior under study and treatment. Modification strategies may vary depending on where the aggressor falls in his or her own social hierarchy and how the target's behavior may enhance or diminish the probability for aggression. This caveat can be as mundane, clinically, as to inquire why a patient's assaults always occur on a particular hospital shift or toward a particular staff person.

Temperament

Evaluating people on a continuum of aggression based on their enduring temperament may yield clues to the understanding of behavioral mechanisms. This has happened with genetic studies in rodents, for example. It was this process of evaluating patients of extreme temperament that led to the first clinical association of low cerebrospinal 5-hydroxyindole acetic acid levels and enhanced human aggressive behavior (80).

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Some clues have yet to be pursued. Kinzel (81) noted many years ago that assaultive prisoners had enlarged "body-buffer zones" and experienced greater personal discomfort and aversion than nonassaultive prisoners when approached by others. We still do not know whether this type of reaction was linked to a different sensory perception or the consequent affect (e.g., anxiety) generated by a perception of proximity. However, this effect may, indeed, play an instigating role in assault.

Neuroanatomy, Neurophysiology, and Neurochemistry

Perhaps the most profound conclusion to date from animal research concerning aggressive behavior is that determinants of aggressive behavior are multifactorial in nature. There is no single aggression center, and there appears to be no single aggression neurotransmitter or neuromodulator. Rather, the biological matrix within the organism is much more like an orchestra than a solo trumpet—that is, a synthesis of multiple brain regions and neurotransmitters leading to a final common outcome of attack or alternative behavior. This complexity may affect research, but it offers many additional opportunities for modulation beyond what a "center" mechanism (controlling such behavior) might otherwise afford.

Pharmacologically, for example, there appear to be many developing "handles" for modulating human aggressive behavior. Serotonergic enhancement (primarily at the 5-HT1 level), dopamine blockade (probably at the D_2 level), or central β-adrenergic blockade all might attenuate certain types of human aggressive behavior. Moreover, since there is no demonstration of a clear rate-limiting neurotransmitter system for these behaviors, such polypharmacotherapy affecting differing neurotransmitter systems could well be additive. Ascertaining whether this is the case would be highly valuable for the practicing clinician to know.

Animal neurochemical research also shows the clinical researcher that drugs that may offer therapeutic promise may not necessarily show any significant effect in "normal" populations. Just as antibiotics need to be tested for efficacy in infected patients, so too, will aggression-attenuating pharmaceuticals need to be tested for efficacy in repetitively and excessively aggressive clinical patients.

Finally, in grasping the neurophysiology of aggressive behavior, animal models have illustrated the importance of sensory processes. In the search for an explanation of aggressive behavior in specific populations, more than a "neurotransmitter imbalance" needs to be studied and evaluated.

For example, patients with epilepsy seem to have higher levels of aggressive behavior than the baseline of the population from which they are taken (82,83). Much research effort has been dedicated to the study of whether this effect was a result of actual ictal or peri-ictal occurrences (84). The consensus is that this is not the case. Yet, the observation remains unexplained.

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Clinical researchers could evaluate these patients for irritability or impulsivity, but researchers could also note the animal experiments that show sensory thresholds change with brain stimulation, even to nonsensory regions. It is possible that certain patients either through central kindling or as a consequence of their seizure disorder have alterations in their perception of sensory stimuli that would culminate in a lower threshold for aggression. Could these people hear a baby's cry as more aversive or a barroom comment as more threatening in comparison with a nonictal person? These remain researchable issues.

Developmental Factors and Stress

Animal aggression research provides models to suggest that both the intrauterine environment present during brain development and the organism's external environment (as a generator of stress or trauma) play powerful and durable roles in the genesis of aggressive behavior.

From a clinical research perspective, there remains much to be learned about the consequences of brain organization during abnormal endocrine situations, but also much to be uncovered about the enduring effects of a brain that develops under chronic alcohol, cocaine, or opiate exposure.

From the trauma perspective, the generation of aggressive behavior in attachment-disordered children is observed. Yet, little is known about their cognitive functioning and essentially nothing of the brain chemistry of this population. It is not known whether the abused, now adult, child-abusing parent has a different neurochemistry from the nonabuser, and if it were different, is it related to his own traumatic history or is it genetically driven?

Regardless of the clinical research questions asked, the clinical and public health or public policy mandates seem clearly presented in the animal research. Attention to normalizing the gestational period for the fetus and diminishing the traumas of separation or rejection and the incidence of physical and emotional assault during a child's development may have significant and durable effects in preventing the development of pathological adult aggressive behavior, behavior so difficult to attenuate once established.

Aggression as a Coping Strategy

Finally, the study of the phenomenology of animal aggression continues to affirm its overall selective advantage. While Lorenz (85) has commented wisely that human destructiveness has now obtained the ability to annihilate the species, it still remains critical that both the clinical researcher and the practicing clinician seek to understand human aggressive behavior as a coping strategy, albeit at many times an ineffective and imprudent one. An examination of this behavior may yield a means to replace the observed destructive behavior with an equally efficacious and reinforcing behavior.

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CONCLUSION

This chapter offers a glimpse at animal models of aggression, their topography, and their utility in conceptualizing both research and treatment issues concerning the violent patient. A phenomenological approach suggests multifactorial components of animal aggression, and it offers a promise for a renewed application to the human clinical condition.

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REFERENCES

1. K Moyer. Kinds of aggression and their pathological basis. Comm Behav Biol 2A: 65–87, 1968.
2. M Wasman, JP Flynn. Directed attack elicited from hypothalamus. Arch Neurol 6: 220–227, 1962.
3. P Karli, M Vergnes, F Didiergeorges. Rat-mouse interspecific aggressive behaviour and its manipulation by brain ablation and by brain stimulation. In: S Garattini, EB Sigg, eds. Aggressive Behavior. New York: John Wiley and Sons, 1969, pp 47–55.
4. K Butler. Predatory behavior in laboratory mice: sex and strain comparisons. J Comp Physiol Psychol 85:243–249, 1973.
5. CI Yen, RL Stanger, N Millman. Ataractic suppression of isolation-induced aggressive behavior. Arch Int Pharmacodyn 123:179–185, 1959.
6. L Valzelli. The "isolation syndrome" in mice. Psychopharmacologia 31:305–320, 1973.
7. B Svare. Maternal aggression in mammals. In: DJ Gubernick, PH Klopfer, eds. Parental Care in Mammals. New York: Plenum Press, 1981, pp 179–210.
8. E Endroczi, K Lissak, G Telegdy. Influence of sexual and adrenocortical hormones on the maternal aggressivity. Acta Physiol Acad Sci Hung 14:353–357, 1958.
9. N Tinbergen, JJA van Iersel. "Displacement reactions" in the three-spined stickleback. Behaviour 1:56–63, 1947.
10. HK Buechner. Territorial behavior in the Uganda kob. Science 133:698–699, 1961.
11. K Lorenz. The past twelve years in the comparative study of behavior. In: CH Schiller, ed. Instinctive Behavior. New York: International Universities Press, 1952, pp 288-310.
12. RJ Blanchard, DC Blanchard, T Takahashi, MJ Kelley. Attack and defensive behaviour in the albino rat. Anim Behav 25:622–634, 1977.
13. RE Ulrich, NH Azrin. Reflexive fighting in response to aversive stimulation. J Exp Anal Behav 5:511–520, 1962.
14. NH Azrin, RR Hutchinson. Unpublished study, 1963.

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15. NH Azrin, RR Hutchinson, DF Hake. Pain-induced fighting in the squirrel monkey. J Exp Anal Behav 8:620–621, 1963.
16. R Ulrich,M Johnson, J Richardson, P Wolff. The operant conditioning of fighting behavior in rats. Psychol Rec 13:465–470, 1963.
17. NE Miller. Theory and experiment relating psychoanalytic displacement to stimulus-response generalization. J Abnorm (Soc) Psychol 43:155, 1948.
18. RR Hutchinson, NH Azrin, GM Hunt. Attack produced by intermittent reinforcement of a concurrent operant response. J Exp Anal Behav 11:489–495, 1968.
19. J Dollard, NE Miller, OH Mowrer, GH Sears, RR Sears. Frustration and Aggression. New Haven: Yale University Press, 1939.
20. DJ Reis. Brain monoamines in aggression and sleep. Clin Neurosurg 18:471–502, 1971.
21. DJ Reis. Central neurotransmitters in aggression. Res Publ Assoc Res Nerv Ment Dis 52:119–148, 1974.
22. B Eichelman. Effect of subcortical lesions on shock-induced aggression in the rat. J Comp Physiol Psychol 74:331–339, 1971.
23. B Eichelman, J Barchas. Facilitated shock-induced aggression following antidepressive medication in the rat. Pharmacol Biochem Behav 3:601–604, 1975.
24. RJ Kaatz. Catecholamines in predatory behavior: a review and critique. Aggress Behav 4:153–172, 1978.
25. P Ropartz. The relation between olfactory stimulation and aggressive behavior in mice. Anim Behav 16:97–100, 1968.
26. L Stowers, TE Holy, M Meister, C Dulac, G. Keontges. Loss of sex discrimination and male-male aggression in mice deficient for TRP2. Science 295:1493–1500, 2002.
27. NM Bugbee, B Eichelman. Sensory alterations and aggressive behavior in the rat. Physiol Behav 8:981–985, 1972.
28. MJ Raleigh, MT McGuire, GL Brammer, A Yuwiler. Social and environmental influences on blood serotonin concentrations in monkeys. Arch Gen Psychiatry 41: 405–410, 1984.
29. GD Maengwyn-Davies, DG Johnson, NB Thoa, VK Weise, KJ Kopin. Influence of isolation and of fighting on adrenal tyrosine hydroxylase and phenylethanolamine-n-methyltransferase activities in three strains of mice. Psychopharmacologia 28:339–350, 1973.
30. M Krsiak. Effect of ethanol on aggression and timidity in mice. Psychopharmacology 51:75–80, 1976.
31. JP Scott, J Fuller. Genetics and the Social Behavior of the Dog. Chicago: University of Chicago Press, 1965, p 468.
32. B Eichelman. Variability in rat irritable and predatory aggression. Behav Neural Biol 29:498–505, 1980.
33. KMJ Lagerspetz, KYH Lagerspetz. Changes in the aggressiveness of mice resulting from selective breeding, learning, and social isolation. Scand J Psychol 12:241–248, 1971.
34. NK Popova, NN Voitenko, AV Kulikov, DF Avustinovich. Evidence for the involvement of central serotonin in mechanism of domestication of silver foxes. Pharmacol Biochem Behav 40:751–756.

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35. O Pucilowski, B Eichelman, DH Overstreet, AH Rezvani, D Janowsky. Enhanced affective aggression in genetically bred hyper-cholinergic rats. Neuropsychobiology. 24:37–41, 1990.
36. HW Sudak, JW Maas. Behavioral-neurochemical correlation in reactive and nonreactive strains of rats. Science 146:418-420, 1964.
37. J Slater, DA Blizard, LA Pohorecky. Central and peripheral norepinephrine metabolism in rat strains selectively bred for differences in response to stress. Pharmacol Biochem Behav 6:511–520, 1977.
38. EK Orenberg, J Renson, GR Elliott, JD Barchas, S Kessler. Genetic determination of aggressive behavior and brain cyclic AMP. Psychopharmacol Commun 1:99–107, 1975.
39. WR Hess. The Functional Organization of the Diencephalon. New York: Grune & Stratton, 1957, p 180.
40. L Schreiner, A Kling. Rhinencephalon and behavior. Am J Physiol 184:486–490, 1956.
41. RJ Bandler, JP Flynn. Control of somatosensory fields for striking during hypothalamically elicited attack. Brain Res 38:197–201, 1972.
42. RM Post. Lidocaine-kindled limbic seizures: behavioral implications. In: JA Wada, ed. Kindling 2. New York: Raven Press, 1981, pp 149–160.
43. L Valzelli. Drugs and aggressiveness. Adv Pharmacol 5:79-108, 1967.
44. KA Miczek, H Barry. Pharmacology of sex and aggression. In: SD Glick, J Goldfarb, eds. Behavioral Pharmacology. St. Louis: Mosby, 1976, pp. 176–257.
45. G DiChiara, R Camba, PF Spano. Evidence for inhibition by brain serotonin of mouse killing behavior in rats. Nature 233:272–273, 1971.
46. JL Gibbons, GA Barr, WH Bridger. Effects of para-chlorophenylalanine and 5-hydroxytryptophan on mouse killing behavior in killer rats. Pharmacol Biochem Behav 9:91–98, 1978.
47. MH Sheard, M Davis. Shock elicited fighting in rats: importance of intershock interval upon the effect of p-chlorophenylalanine (PCPA). Brain Res 111:433–437, 1976.
48. JL Gibbons, GA Barr, WH Bridger, SF Leibowitz. Manipulations of dietary tryptophan: effects on mouse killing and brain serotonin in the rat. Brain Res 169:139–153, 1979.
49. KM Kantak, L Hegstrand, J Whitman, B Eichelman. Effects of dietary supplements and a tryptophan-free diet on aggressive behavior in rats. Pharmacol Biochem Behav 12:173–179, 1980.
50. M Vergnes, C Penot, E Kempf, G Mack. Lésion sélective des neurons sérotoninergiques du raphe par la 5,6-dihydroxytryptamine: effets sur le comportement d'agression interspecifique du rat. Brain Res. 133:167–171, 1977.
51. KM Kantak, B Eichelman, L Hegstrand. Facilitation of shockinduced fighting following intraventricular 5,7-dihydroxytryptamine and 6-hydroxydopa. Psychopharmacology 74:157–160, 1981.
52. BL Jacobs, A Cohen. Differential behavioral effects of lesions of the median or dorsal raphe nuclei in rats: open field and pain-elicited aggression. J Comp Physiol Psychol 90:102–108, 1976.
53. KM Kantak, L Hegstrand, B Eichelman. Dietary tryptophan reversal of septal lesion

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and 5,7-DHT lesion elicited shock-induced fighting. Pharmacol Biochem Behav 15: 343–350, 1981.
54. B Olivier, J Mos, M van der Heyden, J Schipper, M Tulp, B Berkelmans, P Bevan. Serotonergic modulation of agonistic behaviour. In: B Olivier, J Mos, BF Brain, eds. Ethopharmacology of Agonistic Behaviour in Animals and Humans. Dordrecht: Martinus Nijhoff, 1987, pp 162–186.
55. D Brunner, R Henn. Insights into the neurobiology of impulsive behavior from serotonin receptor knockout mice. Ann NY Acad Sci (USA) 836:81–105, Dec 29, 1997.
56. M Sheard. The role of drugs affecting catecholamines on shock-elicited fighting in rats. In: E Usdin, IJ Kopin, J Barchas, eds. Catecholamines: Basic and Clinical Frontiers, Vol 2. New York: Pergamon Press, 1979, pp 1690–1692.
57. B Eichelman, L Hegstrand. Stress-induced alterations in aggression and brain biochemistry. Presented at the 13th Collegium Internationale Neuropsychopharmacologicum; June 15, 1982, Jerusalem, Israel.
58. F Lamprecht, B Eichelman, NB Thoa, RB Williams, IJ Kopin. Rat fighting behavior: serum dopamine-B-hydroxylase and hypothalamic tyrosine hydroxylase. Science. 177:1214–1215, 1972.
59. JM Stolk, WJ Nowack, JD Barchas, SR Platman. Brain norepinephrine: enhanced turnover after rubidium treatment. Science 168:501–503, 1970.
60. B Eichelman, NB Thoa, J Perez-Cruet. Alkali metal cations: effects on aggression and adrenal enzymes. Pharmacol Biochem Behav 1:121–123, 1973.
61. NB Thoa, B Eichelman, JS Richardson, D Jacobowitz. 6-Hydroxydopa depletion of brain norepinephrine and the facilitation of aggressive behavior. Science 178: 75–77, 1972.
62. MA Geyer, DS Segal. Shock-induced aggression: opposite effects of intraventricularly infused dopamine and norepinephrine. Behav Biol 10:99–104, 1974.
63. L Hegstrand, B Eichelman. Increased shock-induced fighting with super-sensitive B-adrenergic receptors. Pharmacol Biochem Behav 19:313–320, 1983.
64. AJJC Lammers, JM Van Rossum. Bizarre social behavior in rats induced by a combination of a peripheral decarboxylase inhibitor and dopa. Eur J Pharacol 5:103–106, 1968.
65. B Senault. Effets de lésions de l'hypothalamus et du globus pallidus et d'injections d'apomorphine dans le globus pallidus, le noyau caude, la substantia nigra et le septum sur le comportement d'agressivité induit par l'apomorphine chez le rat. Psychopharmacology 55:135–140, 1977.
66. LH Allikmets. Cholinergic mechanisms in aggressive behavior. Med Biol 52:19–30, 1975.
67. K Bell, DM Warburton, K Brown. Drugs as research tools in psychology: cholinergic drugs and aggression. Neuropsychobiology 14:181–192, 1985.
68. B Eichelman. Neurochemical and psychopharmacologic aspects of aggressive behavior. In: HY Meltzer, ed. Psychopharmacology: The Third Generation of Progress. New York: Raven Press, 1987, pp 697–704.
69. KA Fox, RL Snyder. Effect of sustained low doses of diazepam on aggression and mortality in grouped male mice. J Comp Physiol Psychol 69:663–666, 1969.
70. J Mos, B Olivier. Pro-aggressive actions of benzodiazepines. In: B Olivier, J Mos,

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PF Brain, eds. Ethopharmacology of Agonistic Behaviour in Animals and Humans. Dordrecht: Martinus Nijhoff, 1987, pp 187–206.
71. CF Ferris. Adolescent stress and neural plasticity in hampsters: a vasopressin-serotonin model of inappropriate aggressive behavior. Exp Physiol 85(Spec No):85S–90S, 2000.
72. MV Pletnikov. The participation of the octapeptide cholecystokinin and beta-endorphin in the neurochemical support of the interspecific and intraspecific aggressivity of rats. Zh Vyssh Nerv Deiat Im I P Pavlova 39:770–773, 1989.
73. GE Demas, MJ Eliasson, TM Dawson, VL Dawson, LJ Kriegsfeld, RJ Nelson, SH Snyder. Inhibition of neuronal nitric oxide synthase increases aggressive behavior in mice. Mol Med 3:610–616, 1997.
74. WE Lyons, LA Mamounas, GA Ricaurte, V Coppola, SW Reid, SH Bora, C Wihler, VE Koliatsos, L Tessarollo. Brain-derived neurotrophic factor–deficient mice develop aggressiveness and hyperphagia in conjunction with brain serotonergic abnormalities. Proc Natl Acad Sci USA 96:15239–15244, 1999.
75. RL Conner, S Levine, GZ Wertheim, JF Cummer. Hormonal determinants of aggressive behavior. Ann NY Acad Sci 159:760-776, 1969.
76. RB Williams, B Eichelman. Social setting: influence on the physiological response to electric shock in the rat. Science 174:613–614, 1971.
77. RL Conner, J Vernikos-Danellis, S Levine. Stress, fighting and neuroendocrine function. Nature 234:564–566, 1971.
78. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. Washington; American Psychiatric Association, 1994.
79. B Eichelman, A Hartwig. The Carolina nosology of destructive behavior (CNDB). J Neuropsychiatry Clin Neurosci 2:288–296, 1990.
80. GL Brown, FK Goodwin, JC Ballenger, PF Goyer, LF Major. Aggression in humans correlates with cerebrospinal fluid amine metabolites. Psychiatry Res 1:131–139, 1979.
81. AF Kinzel. Body-buffer zone in violent prisoners. Am J Psychiatry. 127:99–104, 1970.
82. D Williams. Neural factors related to habitual aggression. Brain 92:503–520, 1970.
83. EJ Nuffield. Neurophysiology and behaviour disorders in epileptic children. J Ment Sci 107:438–458.
84. A Delgado-Escueta, RH Mattson, L King, ES Goldensohn, H Speigel, J Madsen, P Crandal, F Dreifuss, RJ Porter. The nature of aggression during epileptic seizures. N Engl J Med 305:711–716, 1981.
85. K Lorenz. On Aggression. New York: Harcourt, Brace, Jovanovich, 1966.

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Genetic Models of Aggression, Impulsivity, and Related Behaviors

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INTRODUCTION

Aggression is a universal phenomenon, spanning every historical period and age group. Although preschoolers are the most aggressive human beings with respect to frequency of aggressive behavior (1), aggressive acts perpetrated by older, more powerful individuals receive greater public attention. Much behavioral genetic research has focused on aggression, impulsivity, and related behaviors. Recent reviews of aggression (2–6) and impulsivity (7,8) are available. Most research indicates that aspects of aggression and impulsivity show at least some genetic influence; however, estimates range from 0% to 94% (2). The picture is complicated by definitional and/or measurement issues, gender differences, the different types of genetic variance (additive vs. nonadditive—e.g., the high estimates that are reported do not take nonadditive genetic variance into account, so the estimates are greater than the identical twin correlations and the heritability has been overestimated), and age differences or developmental factors. The research also results in the inescapable conclusion that aspects of the environment are important for the development of these behaviors—although whether the en-

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vironmental effects are shared (contribute to familial similarity) or nonshared (make family members different from one another) remains equivocal. The purpose of this chapter is to review the recent behavioral genetic research on the etiology of aggression, impulsivity, and related behaviors, to highlight factors that impact estimates of heritability, and to focus on models of the combined effects of genes and environment, specifically genotype-environment (GE) interaction, diathesis-stress models, GE correlation, and G→E effects.

WHAT IS AGGRESSION AND HOW IS IT MEASURED?

Aggression is a constellation of behaviors that range from temper tantrums to participation in violent crimes, and includes anger, hostility, irritability, and impulsivity. According to Anderson and Bushman (9), aggression is any behavior that is directed toward another individual with the intention to cause harm, and two types are frequently discussed—instrumental and impulsive. Instrumental, or proactive aggression, refers to aggressive behavior that is premeditated in nature, whereas impulsive aggression reflects hostile, unplanned behavior that is driven by anger and is a reaction to some perceived provocation; other names for this include affective, hostile, or reactive aggression.

Major limitations to research include both the multiplicity of definitions and the variability in measures; not surprisingly, there remains much disagreement regarding whether aggression is a unitary construct or a multidimensional trait (10). To assess how the components of aggression are interrelated, Choynowski (11) conducted a factor analysis of 826 items that were classified into 13 scales and four factors: Rebelliousness (Nonconformity, Verbal Aggression, Malice, and Negativism); Spontaneous Aggressiveness (Physical Aggression, Boldness, Suspiciousness, and Vicarious Aggression); Intra-aggressiveness (Self-aggression, Resentment, and Suspiciousness) and Irritability (Irritability, Lack of Control, and Revengefulness) that accounted for almost 65% of the variance in the 13 scales. Although four factors emerged, which indicated that aggression has clearly differentiated components, the factors were correlated (r = .26–.58) and the principal component accounted for almost 50% of the variance. Thus, the results provide support for both sides of this definitional debate.

The attributes associated with aggression are then translated into assessment tools; components of most measures include physical and verbal aggression, irritability, anger, hostility, and violent acts (see 12 for a review). Typical measures of aggression are composed of self-report scales like the Buss-Durkee Hostility Inventory (BDHI) or the MPQ aggression scale. Additional assessment techniques include adjective checklists (13), projective tests (14), parent reports (e.g., the externalizing scale of the Child Behavior Checklist (CBCL), which includes bullying, cruelty, getting into fights, temper tantrums) (15–17), maternal observa-

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tions (18), and observations of modeled aggression (19). Related research incorporates estimations of juvenile delinquency or criminal behaviors as well (20).

GENETIC AND ENVIRONMENTAL INFLUENCES ON AGGRESSION

In children, the heritabilities range from 0% (19) for videotaped observations of the number and intensity of hits to a bobo doll to 78% (18) for a parent report on the CBCL Aggression scale; the estimated average heritability for studies focusing on children is .40 (3). In adolescent samples, the estimates are also variable, including 0% on the SAI aggression scale (21), 16% on the ACL aggression scale (22), 27% from videotaped observations of verbal aggression/hostility toward the mother (23), and 30% in a report aggregated from six studies of court records of juvenile delinquency (24). Although the estimates in studies of adolescent populations are more consistent than those found in samples of children, the type of assessment technique does make a difference. In adulthood, estimates also range from 0% (for Direct Assault in an all-female sample (25) to 61% for crimes against property (26).* Typical estimates in adulthood, from self-report questionnaires, are in the 40% range (e.g., 27–29). Two recent meta-analyses have summarized this work. In the first study, analyses of eight aggression scales indicated a heritability of 42% [weighted twin correlations were .49 for identical twins and .28 for fraternal twins (8)] and in the second study, the authors reported that the heritability of aggression may account for up to 50% of the variance (5), but again there is great diversity in the estimates, which may be a function of developmental or methodological effects.

Behavioral genetic research on aggression has also provided an immense literature on the importance of the environment in the development of aggressive behavior. No study concludes that all of the variance is genetic in origin; in fact, the environment is the largest component of variance in most analyses. What is of interest is that the environmental variance tends to be nonshared, rather than shared. In other words, the environment works to make family members different from one another, rather than more similar. Some exceptions to this do exist. For example, some analyses of twins, especially in adolescent populations, show a shared environmental effect, suggesting that common friends, sibling imitation effects, or attributes of the neighborhood may be salient for aggression, primarily delinquency, in youth. These results are discussed in more detail later.

* Cates et al. in 1993 (25) reported heritability estimates of .78 for Indirect Assault, .70 for Verbal Hostility, and 0.98 for Irritability in their all-female sample, but these authors did not take nonadditive genetic factors into account, and the heritability estimates are higher than the MZ correlations. For that reason, these estimates were not included in the reported ranges.

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A few studies have assessed multiple dimensions of aggression, as well as their multivariate relations. Coccaro and colleagues assessed the etiology of aggression using a subsample of the Vietnam Era Twin (VET) Registry (22). Measures of aggression included the subscales of Direct Assault, Indirect Assault, Verbal Assault, and Irritability from the Buss-Durkee Hostility Inventory. These four aspects of aggressive behavior are interrelated, with phenotypic correlations ranging from .34 (Indirect Assault/Direct Assault) to .57 (Verbal Assault/Direct Assault). Heritability estimates for the individual scales were .28 for Verbal Assault, .40 for Indirect Assault, and .37 for Irritability due to nonadditive genetic variance, whereas additive genetic factors were important for Direct Assault, which had a heritability of .47. The remaining variance in all of the scales was due to nonshared environmental factors. Multivariate genetic analyses were used to assess the etiology of the phenotypic associations between these measures; the results indicated that information about the magnitude of a phenotypic relationship tells us little about the underlying etiology of that relationship. For example, the relationships between Verbal Assault with Irritability, Indirect Assault, or Direct Assault were largely mediated by nonshared environmental factors (64–84%), whereas the relationship between Indirect Assault and Irritability was largely mediated by genetic influences (67%).

Multivariate analyses were also used to assess the magnitude of the genetic and environmental correlations. The genetic (environmental) correlation between two variables indicates the extent to which there is overlap in the underlying genetic (environmental) factors that contribute to the observed phenotypic correlation between them. In the case of the BDHI scales, the genetic correlations were relatively high for the Indirect Assault, Verbal Assault, and Irritability scale combinations ranging from .60 to .80. The genetic correlations between Direct Assault with Indirect Assault, Verbal Assault, or Irritability, on the other hand, were quite low, ranging from 0.17 to 0.46. These results suggest that the characteristics measured by the Direct Assault scale of the BDHI may be due to underlying genetic factors that are different from the influences on the other three aggression scales. This is not surprising, given that the univariate model-fitting analyses indicated an additive pattern of genetic variance for Direct Assault and a nonadditive pattern for the other scales of the BDHI. The nonshared environmental correlations indicate a fairly consistent pattern across all combinations of the BDHI scales ranging from .34 to .56. The one exception was the nonshared environmental correlation between Indirect Assault and Irritability, which was .09, indicating that there is little overlap in the nonshared environmental factors influencing these two scales.

Vernon and colleagues (10) asked 247 pairs of adult twins to complete seven self-report questionnaires assessing multiple dimensions of aggression. The factor analysis of the 18 scales of aggressive attributes resulted in a general factor and three rotated (oblimin) factors (Spontaneous Aggression, Aggressive Atti-

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tudes, Verbal Aggression). Univariate analyses of each of these dimensions indicated that the general factor and all three subcomponents were heritable. Specifically, analyses of twin similarity for the general factor indicated that additive genetic influences accounted for 54% of the variance, with the remaining variance (46%) due to nonshared environment. Of the subcomponents, additive genetic influences accounted for 52% and 44% of the variability in Spontaneous and Verbal Aggression, respectively, with the remaining differences due to nonshared environment. Analyses of Aggressive Attitudes also indicated that the majority of the variability was due to heredity (52%), but in this case it was of the nonadditive type. Multivariate analyses to assess the extent to which the same underlying genetic influences contributed to these three subdimensions of aggressive behavior indicated that there was moderate genetic overlap, with an average genetic correlation of 0.44 (range .31–.51).

RECENT STUDIES OF AGGRESSION AND RELATED BEHAVIORS

Behaviors that are frequently related to aggression include hostility, irritability, and anger proneness. Gustavsson and colleagues (30) assessed 70 pairs of twins from the Swedish Adoption Twin Study of Aging (SATSA) on multiple measures of aggression, hostility, and anger-related personality. The twin similarity for Anger Proneness (irritability and trait anger) and Angry Aggression (indirect aggression, verbal aggression, and outward expressions of anger) was attributable to genetic factors, whereas twin similarity for the hostility component (defined as suspiciousness in this study) was ascribed to environmental factors. For all three of the components, nonshared environment accounted for the majority of the variance. Coccaro et al. (31) found a heritability of 41% (nonadditive genetic variance) for a measure of what they called "irritable aggression" in the full SATSA sample. It has been suggested that irritability may be in the gray zone in the interface between aggression and impulsivity (12). Jang and colleagues (32) reported similar findings for measures of labile anger (40%) and irritability (41%) using 236 pairs of MZ and 247 pairs of DZ twins (ages 16–84).

DiLalla et al. (33) included the Hostility scale from the MMPI in an assessment of personality in 111 pairs of twins from the Minnesota Study of Twins Reared Apart. Results indicated a heritability of 37% due to nonadditive genetic factors with the remaining variance (63%) due to nonshared environment. In the SATSA sample, estimates of heritability for the Cook-Medley Hostility Scale were 20%, with shared environment accounting for an additional 20% (34). Weidner et al. (35) assessed subjects from the National Heart, Lung and Blood Institute (NHLBI) Family Heart Study (680 European-American Families including 2525 individuals), also using the Cook-Medley Hostility Scale. Significant familial resemblance was found for all of the subscales, with familiality (assess-

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ment of both shared genes and shared environment) estimates accounting for 42% of the variance in total Hostility—30% in Cynicism, 38% for Aggressive Responding, and 18% for Hostile Affect.

Research has also focused on the familial resemblance for temper outbursts. Mattes and Fink (36) studied patients with temper outbursts (n = 33), a control sample of diverse psychiatric patients without temper outbursts (n = 12), and 179 of their first-degree relatives. They used a modified version of the Family History Research Diagnostic Criteria to rate temper outbursts. Frequency ratings in the patients were relatively severe, with 18.6 verbal outbursts per month for the 6 months prior to the study entry, including 5.8 outbursts per month involving the destruction of property and 1.6 assaults per month. Patients with temper problems had significantly more temper problems in their families than did the control group (18.2 vs. 4.3; $P = .013$).

One measure commonly used in studying aggressive behavior in children is the parent rating on the externalizing scale of the Child Behavior Checklist. In one such study (17), analyses assessed the etiology of externalizing behaviors by both gender and age—younger (ages 8–11; 652 pairs) and older (ages 12–16; 612 pairs). Heritability estimates were .38 and .13 in young boys and girls, respectively, but were not significantly different by gender in the older sample ($h^2 = 0.24$). Shared rearing environment accounted for 46% of the variance in young boys and 62% young girls; there were no significant gender differences in the older group, with shared environment accounting for 57% of the variance. Thus, the results indicated that there were age and gender differences in the etiology of externalizing behaviors, which was reflected in an interaction effect: the gender differences in the relative effects of genes and environment occurred only for the young sample. It should be noted that shared environment played a significant role for boys and girls across both of the ages studied.

Other studies have also used the CBCL to assess the etiology of individual differences in aggressive and nonaggressive antisocial behavior (15). Aggressive antisocial behavior has been defined as a temperamental trait that reflects behaviors such as fighting, bullying, or other physical acts of aggression. Nonaggressive antisocial behavior relates to actions that are only displayed in certain situations (e.g., in the context of delinquent peer affiliations), and include behaviors like truancy or theft. These two types are often referred to as conduct disorder (life course–persistent) and socialized delinquency (adolescence-dependent), respectively. Moffit (37) suggests that these two types of antisocial behavior have different etiologies. Specifically, the life course–dependent type is hypothesized to be due to neuropsychological deficits or aspects of temperament that sustain, and even promote, antisocial behaviors. Adolescence-limited aggression is described in terms of imitation of peer behavior, which is reinforced by respect from members of the peer group. It is considered to be within the normal course of adolescent development, especially as it relates to establishing friend support and

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independence from parents. Thus, it was speculated that aggressive antisocial behavior (life course–dependent), which is more related to heritable aspects of temperament, would show a strong influence of genetic factors, whereas the nonaggressive antisocial behavior (adolescence-dependent) would be more influenced by environmental factors. Results from a study by Eley et al. (15) supported this hypothesis: genetic factors were more important for aggressive antisocial behavior than for nonaggressive antisocial behavior. Because the findings were consistent with those of Moffitt (37) and Caspi and colleagues (38), it was suggested that temperament might mediate the genetic effects on aggressive antisocial behavior.

Other studies have also looked at the relationship between antisocial behavior (especially criminality and juvenile delinquency) and aggression. Finkel and McGue (39) assessed aggression using the MPQ, identifying characteristics such as "physically aggressive," "vindictive," and "likes violent scenes," all components of negative emotionality. Analyzing data from MZ and DZ twins, their siblings, and their parents resulted in a heritability estimate of 39% for males and 35% for females (indicating that there were no significant gender differences in the etiology of these behaviors). The genetic variance was of the nonadditive type, with the remaining variance due to nonshared environment. Jang and colleagues (32) reported heritability estimates ranging from 41% to 58% for characteristics like Interpersonal Violence (.48), Juvenile Antisocial Behavior (.58), Interpersonal Hostility (.41), and Self-harm (.41). The rest of the variance was attributed to environmental factors that make family members different from one another (nonshared environment).

Previous studies of adult antisocial behavior, or criminality, typically concluded that heritability was more important than shared environment, whereas studies of delinquency found that family environment was more salient (e.g., 40). To further test these hypotheses, Lyons and colleagues (20) used 3226 pairs of male twins from the VET Registry. Results of their study indicated that five of the juvenile symptoms were heritable (truant, fights, used weapons, cruel to animals, lies often) with values ranging from 21% to 41%, and five symptoms reflected the importance of shared rearing environment (ran away, cruel to people, damage to property, starts fires, steals, no confrontation), with estimates varying from 18% to 55%. In adults, eight symptoms showed substantial heritability (ranging from 22% to 52%), including "inconsistent work," "aggressive," "fails to conform to social norms," "doesn't honor financial obligations," "reckless," "impulsive," "lacks remorse," and "never monogamous." Only one symptom, "no regard for the truth," showed significant shared environmental effects, but, interestingly, this accounted for 77% of the variance. These researchers estimated that shared environment explained six times more variance in the juvenile characteristics when compared to adult traits, and that the shared environmental effects on juvenile traits overlapped 100% with adult traits. Conversely, additive genetic

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factors accounted for six times more variance in adult versus juvenile traits, and the hereditary influences on the juvenile traits overlapped entirely with the genetic factors contributing to the adult traits. It should be noted, however, that nonshared environmental factors explained the majority of the variance in both types of traits.

Another attribute that is commonly linked with aggression is impulsivity, and a plethora of studies have provided insight into the etiology of impulsive behavior. "Impulsivity" commonly refers to the tendency to engage in behavior without a plan or a clear sense of direction—that is, to "act on the spur of the moment." Results of a study from the SATSA indicated a broad-sense heritability estimate of 45%, with the remaining variance due to nonshared environment (34). Data collected from the twins participating in the Adult Russian Twin Study (ARTS) indicated that 49% of the variability in impulsivity was due to nonadditive genetic influences (41). Eaves and colleagues (42) used four subscales from the Eysenck Impulsivity scale; heritability estimates were 37% for females and 40% for males for Narrow Impulsivity, .36 and .17 for Risk-taking, .36 and .38 for Nonplanning, and .17 and .15 for Liveliness for females and males, respectively. Similarly, Tellegen et al. (29) reported that the age-adjusted heritability for the control scale of the MPQ was 44% in an American sample, and Jang and colleagues (32) reported an estimate of 33% in Canadian twins. Interestingly, similar results emerged from studies of Swedish, Russian, American, and Canadian twins.

In a meta-analysis by McCartney and colleagues (8), the results of 31 studies that contained measures of impulsivity were reported. Although global heritability estimates were not provided, the intraclass correlations, weighted by sample, were .58 for MZ twins and .22 for DZ twins. Because the correlation for DZ twins is less than half of the MZ correlation, nonadditive genetic influences are indicated and heritability estimates cannot be calculated without using model-fitting analyses. It may not be surprising, then, that researchers using family or adoption designs have found substantially lower estimates of heritability for impulsivity than have been reported in twin studies—a difference that may be due to nonadditive genetic influences (see 43 for a review). For example, using data from a sample of 9- to 16-year-olds from the Colorado Adoption Project (CAP), a combined family/adoption study, Plomin and colleagues (44) reported a heritability estimate of 7% with little or no effect of shared environment. In a second study, 16% of the phenotypic variance in impulsivity, observed between the midparent (or average of the two biological parents) and the adopted-away child, was attributable to genetic influences. Analyses based on biologically related siblings and adopted-together siblings indicated a heritability estimate of 30% (45).

In an attempt to determine the extent to which genetic and environmental influences that contribute to impulsivity are shared with those contributing to aggression, Seroczynski et al. (46) performed bivariate behavioral genetic analy-

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ses on a measure of impulsivity and several subtypes of aggressive behavior (Irritability, Direct Assault, Indirect Assault, and Verbal Assault) as measured by the Buss-Durkee Hostility Inventory. Results suggest that impulsivity and physically assaultive behavior (Direct Assault) are less etiologically related, both genetically and environmentally, than are impulsivity and the other subtypes of aggressive behavior. Moreover, impulsivity and irritability showed both the strongest genetic correlation (.59) and the strongest nonshared environmental correlation (.45), suggesting that these two variables have a larger proportion of overlapping genetic and environmental influences than the other three pairs tested.

These results suggest that when aggression is measured in its "purest" form (i.e., physically assaultive behavior), impulsivity and aggression share little etiologically; however, when aggression is measured more loosely, the results become increasingly mixed. One reason for this stronger relationship may be the "impulsive" nature of questions found in the measure of Irritability (e.g., "I lose my temper easily, but get over it quickly"). Because *Irritability* is defined as a readiness to explode at the slightest provocation, it may not be possible to obtain a pure measure of this aggression construct. A second explanation is that different types of aggression have different etiologies and, as such, represent separate aspects of personality. One example of this is the Direct Assault scale from the BDHI, which shows little genetic overlap with the other dimensions of the BDHI (Indirect Assault, Verbal Assault, Irritability) or with impulsivity. Thus, the etiology of premeditated or assaultive aggression, which indicates an additive pattern of genetic variance in the BDHI, may be different from the irritable/impulsive type of aggression, which shows a nonadditive pattern of genetic variance. Further research is needed to empirically test this hypothesis.

It is clear that both genes and environment play an important role in complex behaviors such as aggression and impulsivity. The next section will focus on the crucial role of the interplay between genetic propensities and environmental influences as they contribute to individual differences in aggression-related behaviors.

THE GENE-ENVIRONMENT (GE) INTERFACE

Genetic and environmental influences have been suggested to interrelate in two major ways—via interactions and correlations (47). Genotype-environment interactions reflect the fact that due to genetic propensities, some individuals are more sensitive to specific aspects of the environment. In other words, the effect that the environment has depends on hereditary influences. Related to this is the diathesis-stress model, in which a specific genetic propensity (the diathesis) produces a vulnerability to stressful environmental influences, which results in a negative outcome (typically some type of pathology). Genotype-environment correlations,

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on the other hand, are the extent to which individuals are likely to experience certain environments as a function of their genetic propensities. There are three types that have been described: passive, active, and reactive or evocative, which are defined below. Scarr and McCartney (48) have taken this notion one step further by suggesting that one's genetic propensities play a significant role in development, and that individuals select and even create their own environments based on their genetic characteristics. In other words, it is one's genetic propensities that "drive" their environmental experiences (G→E).

Genotype-Environment Interactions

The concept of GE interaction represents an important perspective for understanding environmental influences on individual development. Rearing environments might differentially affect the expression of genetic propensities; similarly, the effects of the environment on development might depend on an individual's genotype (see 49). Rather than searching for environmental influences that equally affect all individuals on average, GE interaction focuses on environmental influences that may powerfully affect only a small group of individuals with certain genetic propensities. In the case of aggression, genetic predispositions may be expressed differently in hazardous versus protective environments. In other words, the risk might be exacerbated by adverse rearing conditions (e.g., family environment characterized by conflict), whereas a positive family environment could counter a genetic tendency toward aggression (e.g., calm, nurturing, nonreactive parents who provide a buffer against genetically influenced emotional hyperactivity) (50).

The adoption design is an ideal mechanism for estimating GE interactions, because the sources of genetic and environmental variance can be separated. Although adoption studies allow for the assessment of GE interactions, one limitation of this design for the study of aggression is that the variability in possible environmental exposure is constrained by the screening of adoptive families that limits the number of "adverse homes." If there is no variation in genetic or environmental factors in the sample used, the GE interaction is contained in the main effects, and the influence of the interaction will be underestimated. GE interactions may also be underestimated because, in general, main effects are typically larger than interactions—without large sample sizes and sufficient power, GE interactions will go undetected (51). Additionally, genes that affect the sensitivity to environments may be different from those that bring about the main effects (52). Nevertheless, interactions between genetic predispositions and environmental factors are thought to be importantly involved in the development of aggression, impulsivity, and related behaviors.

Although little work has explicitly assessed GE interactions, a series of studies by Cadoret and colleagues (3,53–56) exemplify how GE interactions con-

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tribute to aggressive phenotypes. One study included 367 adoptees, information regarding their biological parents, and aspects of their adoptive home environment. Results indicated that there was a negligible increased risk for adolescent antisocial behavior based on genetic propensity (i.e., antisocial behavior in the biological parent), no effect of adverse environment alone (defined as marital problems, depression, anxiety, substance abuse, legal problems), but substantial effect when both were present (54). Cadoret (53) also suggested that the adverse environmental effect appeared to apply to a greater extent for males, even though both males and females were at biological risk. In a second study (55), he studied 95 male and 102 female adoptees who had biological parents who either had antisocial personality or were alcohol dependent. They also looked at the effects of an adverse adoptive home on adoptee conduct disorder, adult antisocial behavior, and aggressivity. The biological background predicted adolescent aggression (main effect for genetic influences on aggressive behavior); adverse home environment predicted adult antisocial behavior (a main effect of environment on antisocial behavior). There was also a GE interaction such that adverse home environment predicted greater aggression and conduct disorder when the adoptee had a biological predisposition, but not in the absence of genetic influences. Additionally, they reported that the biological influences on antisocial personality disorder in conjunction with an adverse home environment were more specific to antisocial personality disorder as a genetic factor (compared to a genetic susceptibility for alcoholism), and more for aggression as an outcome.

Cadoret also looked at specific aspects of the environment that might contribute to GE interactions using the Family Environment Scale (FES). Aggression was more likely to result in environments that were depicted as high in conflict and control (by parents) and low in independence and expressiveness on the FES, and a GE interaction was especially important in these relations (3). Interestingly, further research indicated that the environment does not have to be adverse for a GE interaction effect to occur. Riggins-Caspers et al. (56) showed that childhood aggression that stemmed from parental psychopathology was greater if the adoptive parents knew about it. Hence, just the expectation of potential problems produced worse outcomes.

In a related area of research, Rowe et al. (57) assessed the moderating effects of school context on genetic and environmental influences on aggression in adolescents from the National Longitudinal Study of Adolescent Health. Results indicated that those schools with greater ethnic/racial heterogeneity had higher mean levels of aggression; schools in which students perceived greater family warmth had lower mean levels of aggression. The authors suggested that a greater genetic effect was required for the expression of aggression in more benign environments, whereas in more adverse environments, processes such as social norms and peer models may promote aggressive behavior even among individuals without a strong genetic predisposition.

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Diathesis-Stress Models

One type of GE interaction that has been discussed in the literature is the diathesis-stress model of development, which has a specific focus on psychopathology (58,59). Diatheses are typically discussed as a genetic liability, which is a predisposition to develop a particular behavior or disorder that is produced when combined with environmental stress. In an interaction model, the diathesis is necessary, but not a sufficient condition for the disorder, whereas in an additive model, stress can produce the disorder in the person with a weak or absent diathesis, but it takes a greater degree of environmental trauma than it will for a vulnerable person with a strong diathesis for pathology. Thus, an aggressive act may be provoked in a vulnerable person by a relatively minor stress or "hassle," whereas only a major event would product the same reaction in a nonvulnerable person.

Genotype-Environment Correlations

Genotype-environment correlation literally refers to a correlation between genetic deviations and environmental differences as they affect a trait (60). Three types have been posited—passive, reactive, and active. Passive GE correlation is most frequently described, and suggests that by virtue of sharing both genes and family environment with their parents, children can passively inherit environments that are correlated with their genetic propensities. For example, parents can pass on attributes of aggressive behavior genetically and also provide an environment high in conflict. Reactive, or evocative, GE correlations relate to experiences of the child that derive from the reactions of other people to their genetic propensities; in other words, people react differently to individuals of different genotypes. Coaches might identify individuals with aggressive tendencies and encourage them to participate in sports like football or boxing. Active GE correlation is sometimes called "niche-picking" and describes the situation in which children actively seek or even create their own environments (that are correlated to their genetic propensities). Individuals with a predisposition toward aggressive behavior might gravitate toward occupations in the military or police force. This last type may play a crucial role in the way the genotype plays out its role in development.

Although we often think about GE correlations in a positive way—the environment supports, or even accentuates, the genetic propensity—it can also work in the other way. That is, environments can be provided or created that counteract or ameliorate one's hereditary predisposition—emotionally labile parents, who are easily angered, can have children with a proclivity to be quick-tempered; yet, the parents are likely to admonish the expression of the anger in their children. GE correlations can also result in positive or negative outcomes. For example, an individual with a genetic propensity for aggressive behavior that is expressed as a high need for excitement or risk could choose a peer group that

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engages in delinquent behavior. On the other hand, he/she may select peers that are stable, reliable, and supportive to minimize interpersonal difficulty. The extent to which influences of this type work differently in individuals—in essence canceling the effect—minimizes the opportunity for identifying a general GE correlation. Nonetheless, there is interesting work in this area that suggests the importance of effects of this type for the study of aggressive behaviors.

In order to consider a GE correlation, the genetically influenced parental characteristics must be associated with major differences in the upbringing they provide for the children. In fact, recent research indicates that there is a strong association between parental psychopathology and the types of family environments that they provide (61,62). One method for assessing these relationships is to compare the parent-child correlations in adoptive and nonadoptive homes. If the phenotypic correlation is larger than would be expected given estimates of heritability and shared environment, GE correlation is implicated. One example of this type of work is a study (63) in which the relation between stress (assessed by self-reported life events in first grade) and the CBCL (based on teacher reports in both first and third grades) was compared in adoptive versus nonadoptive homes in the Colorado Adoption Project. Results indicated that the stress-outcome relation was greater in the nonadoptive families when compared to the adoptive homes. The author concluded that genetic characteristics of parents might mediate children's responses to the stresses of school.

A second way in which genetic tendencies are thought to influence the risk for aggression is via engaging in high-risk environments; in fact, several behavioral genetic studies have focused on the extent to which individuals "inherit" the life events that they experience. Although it may sound counterintuitive to say that one can inherit experiences, researchers have indicated that life events happen (or are perceived to happen) to some people more than others, with heritability estimates ranging from 0% to 51% (64). This finding indicates that "bad luck" is related to genetically influenced attributes of the individual, and that events of the type typically reported (e.g., marital and work difficulties, financial problems, illnesses and injuries, or being robbed or assaulted) do not just happen randomly (64). To test this hypothesis (65), life events reported by sample of Swedish twins (from SATSA) were separated based on the controllability of the life event. Thus, events like divorce or financial difficulties that may be more related to characteristics of the individual, like personality or mood states, were separated from uncontrollable ones (e.g., death of siblings or friends). As predicted, the controllable events showed greater heritability (.43) than the uncontrollable ones (.18). Other studies have reported similar results (66,67).

Interesting research related to aspects of aggressive behavior has focused on peer relationships. A fundamental feature of peers is that they are outside of the family and the selection process of choosing friends opens up new opportunities for genetic contributions to the environment. One study, using sociometric

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ratings of popularity, reported twin correlations of .70 for identical and .52 for fraternal twins, indicating a heritability of 36% and a shared environmental influence of 34% (based on Falconer's formula) (68). Other studies have used the Sibling Inventory of Differential Experiences (SIDE) to assess characteristics of the peer group—College Orientation, Delinquency, Substance Abuse, and Popularity (69). Results indicated that the intrapair differences were significantly less for identical than for fraternal twins, with an average effect size for the Peer Delinquency scale of .52. Heritability estimates based on parental ratings of siblings' peers indicated heritabilities in the 70% range for both Peer Delinquency and Peer Substance Abuse (70). Thus, ratings of characteristics of adolescents' peer groups that have shown convincing relations with aggression in previous studies (i.e., delinquency and substance abuse) demonstrate strong evidence for the involvement of genetic influences. Social selection is important to peer similarities in attitudes, behaviors, and deviant lifestyles. In other words, children and adolescents do not come together accidentally; they choose activities that are well matched to their own dispositions and select similar friends (71).

A variety of research on measures of the environment, using diverse assessment techniques and methods, consistently converge on the conclusion that genetic influences are significant and substantial on widely used measures of the environment (67). Examples of this type of research include social support (72,73), family environment (74–77), perceptions of parenting (78), work environment (79), television viewing (80), exposure to drugs (81), education, and socioeconomic status (82–84). As a result, there is strong evidence that genetic effects consistently emerge in attributes that are typically considered measures of the environment and may provide insight into the process by which genes and environment combine to produce complex behaviors like aggression.

One suggestion is to investigate the antecedents and consequences of the genetic involvement in environmental measures (85). In thinking about antecedents of these relationships, the focus is on genetically influenced characteristics of individuals that result in genetic influences on measures of the environment. For example, sensation seeking is an aspect of personality that shows substantial heritability (86). To the extent that this proclivity contributes to an individual putting him- or herself in a risky situation, the experience of life events, such as accidents, assaults, or arrests due to delinquent or criminal behavior, could result. In terms of a focus on the consequences of genetic-environment relations, the search is for genetic influences that mediate the associations between measures of the environment and outcomes. By using multivariate behavioral genetic analyses it is possible to estimate the extent to which the same underlying genetic influences affect both measures of the environment (e.g., delinquent peer group) and the developmental outcomes of interest (e.g., aggressive behavior). Unfortunately, it is difficult to assess the direction (antecedent versus consequence) of these relations.

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G→E

Scarr and McCartney (48) use the three categories of GE correlation to describe the process involved in the developmental interface between nature and nurture. The primary difference is that these authors define their theory in terms of genotype-environment effects, because they believe that it is the genetic differences between people that prompt differences in which environments are experienced—in other words, genes dictate experiences (G→E). More specifically, these authors suggest that both genes and environment are components of the developmental system, but they have different functions. Genes direct the course of human experience, but experiential opportunities are also necessary. Individual differences can arise from restrictions in environmental opportunities to experience what the genotype would find congruent. If a variety of options are available to the individual, however, genetically determined influences will govern which environments are selected.

CONCLUSIONS

The general finding is that most dimensions of aggression show at least some genetic influence, although there is notable variability in estimates of heritability. One explanation for these disparities relates to the variation in the types of attributes that are often categorized together and called aggression. Hostility is an attitude; anger and irritability are emotions; impulsivity is a drive to act, and aggression is the end product. Thus, aggression may be a process and measures differentially tap into each of these components (12). Additionally, concepts such as criminality or violence are clearly social constructs, and as such are not independent of the social and legal systems. As a result, these attributes may be more related to environmental influences (7). Behavioral phenotypes, such as impulsiveness or chronic aggressiveness, on the other hand, are more culturally independent, and as such may be more influenced by biological factors.

In discussing genetic influences on behavior, it should always be remembered that genes have only a probabilistic effect and that there is a lot of variability in behavioral development—both normal and abnormal. For example, aggressive behaviors can have many developmental roots, each with its own genetic and environmental underpinning; possible examples include high energy level, sensation seeking, low anxiety under conditions that typically reflect inhibition (e.g., walking alone into a dark alley), testosterone levels, and seratonin receptors. Therefore, there exists much genetic heterogeneity (i.e., aggressive behavior can have different genetic roots in different individuals). A related concept, pleiotrophy, suggests that a single genotype can have different behavioral manifestations. In other words, a genetic predisposition toward aggression could be expressed as verbal abuse, indirect assault, or irritability.

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Environmental influences on the expression of aggressive behavior, particularly stress, may be due to both external sources of negative feelings (e.g., adverse events such as frustrations, provocations, loud noises, uncomfortable temperatures, and unpleasant odors) and physiological arousal or to the internal reactions themselves (e.g., changes in blood pressure, or hormones). Different individuals exposed to the same environment experience it, interpret it, and react to it differently. Early experiences can set up defensive attitudes in individuals that lead him/her to project particular interpretations into new social relations and ambiguous situations. Antisocial personalities may also induce responses that confirm and sustain their subjective interpretation of the environment as hostile. Aggressive individuals may also begin to seek out situations that are compatible with their dispositions, especially in the area of friendship formation and mate selection (71).

Behavior genetic researchers have been explicit in the message related to how genes exert influence; clearly, nature and nurture are not independent of one another, and the next era of research must focus on the interplay between them (87). The question is no longer whether genes and environment affect indices of aggressive behavior, but how these influences act together to form the behavioral outcomes of interest. Unfortunately, there is a paucity of research that has explicitly assessed the effects of GE Interaction or GE Correlation on the development of aggression, impulsivity, and related behaviors. What the available research indicates, however, is that this is a promising avenue to pursue.

REFERENCES

1. JD Coie, KA Dodge. Chapter 12: Aggression and antisocial behavior. In: W Damon, NE NE Eisenberg, eds. Handbook of Child Psychology, Vol 3: Social, Emotional, and Personality Development, 65th ed. New York: Wiley, 1998, pp 379–862.
2. CS Bergeman, AD Seroczynski. Genetic and environmental influences on aggression and impulsivity. In: M Maes, EF Coccaro, eds. Neurobiology and Clinical Views on Aggression and Impulsivity. New York: Wiley, 1998, pp 63–80.
3. RJ Cadoret, LD Leve, E Devor, Genetics of aggressive and violent behavior. Psychiatr Clin North Am 20:301–322, 1997.
4. G Carey. Family and genetic epidemiology of aggressive and antisocial behavior. In: DM Stoff, RB Cairns, eds. Aggression and Violence: Genetic, Neurobiological, and Biosocial Perspectives. Mahwah, NJ: Erlbaum, 1996, pp 3–21.
5. DR Miles, G Carey. Genetic and environmental architecture of human aggression. J Person Soc Psych 72:207–217, 1997.
6. R Plomin, K Nitz, DC Rowe. Behavioral genetics and aggressive behavior in childhood. In: M Lewis, SM Miller, eds. Handbook of Developmental Psychopathology. New York: Plenum, 1990, pp 119–133.
7. D Goldman, DA Fishbein. Genetic basis for impulsive and anti-social behaviors: can their course be altered? In: DA Fishbein, ed. The Science, Treatment, and Pre-

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vention of Antisocial Behaviors: Application to the Criminal Justice System. Kingston, NJ: Civic Research Institute, 2000, pp 9-1–9-18.
8. K McCartney, MJ Harris, F Bernieri. Growing up and growing apart: a developmental meta-analysis of twin studies. Psychol Bull 107:226–237, 1990.
9. CA Anderson, BJ Bushman. Human aggression. Annu Rev Psychol 53:27–51, 2002.
10. PA Vernon, JM McCarthy, AM Johnson, KL Jang, JA Harris. Individual differences in multiple dimensions of aggression: a univariate and multivariate genetic analysis. Twin Res 2:16–21, 1999.
11. M Choynowski. Does aggressiveness have a factorial structure? Person Individ Diff 18:167–187, 1995.
12. P Bech, M Mak. Measurements of impulsivity and aggression. In: E Hollander, DJ Stein, eds. Impulsivity and Aggression. New York: Wiley, 1995, pp 25–41.
13. S Scarr. The origins of individual differences in adjective checklist scores. J Couns Psychol 30:354–357, 1966.
14. DR Owen, JO Sines. Heritability of personality in children. Behav Genet 1:235–247, 1970.
15. TC Eley, P Lichtenstein, J Stevenson. Sex differences in the etiology of aggressive and nonagggresive antisocial behavior: results from two twin studies. Child Dev 70:155-168, 1999.
16. H Gjone, J Stevenson, JM Sundet, DE Eilertsen. Changes in heritability across increasing levels of behavior problems in young twins. Behav Genet 26(5):419–426, 1996.
17. JL Silberg, MT Erikson, JM Meyer, LJ Eaves, ML Rutter, JK Hewitt. The application of structural equation modeling to maternal ratings of twins' behavioral and emotional problems. J Consult Clin Psychol 62:510–521, 1994.
18. J Godsian-Carpey, LA Baker. Genetic and environmental influences on aggression in 4- to 7-year-old twins. Aggress Behav 13:173–186, 1987.
19. R Plomin, TT Foch, DC Rowe. Bobo clown aggression in childhood: environment, not genes. J Res Pers 15: 331-342,1981.
20. MJ Lyons, WR True, SA Eisen, J Goldberg, JM Meyer, SV Faraone, LJ Eaves, MT Tsuang. Differential heritability of adult and juvenile antisocial traits. Arch Gen Psychiatry 52:906–915, 1995.
21. SG Vandenberg. Heredity factors in normal personality traits (as measured by inventories). In: J Wortis, ed. Recent Advances in Biological Psychiatry. New York: Plenum Press, 1967, pp 65–104.
22. JS Loehlin, RC Nichols. Heredity, environment, and personality. Austin: University of Texas Press, 1976.
23. TG O'Connor, K Deater-Deckard, D Fulker, M Rutter, R Plomin. Genotype-environment correlations in late childhood and early adolescence: antisocial behavioral problems and coercive parenting. Dev Psychol 34:970–981, 1998.
24. LF DiLalla, II Gottesman. Heterogeneity of causes of delinquency and criminality: lifespan perspectives. Dev Pyschopathol 1:339–349, 1989.
25. DS Cates, BK Houston, CR Vavak, HM Crawford, M Uttley. Heritability of hostility-related emotions, attitudes, and behaviors. J Behav Med 16:237–256, 1993.
26. CR Cloninger II Gottesman. Genetic and environmental factors in antisocial behav-

Marcel Dekker, Inc.
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ior disorders. In: SA Mednik, TE Mofffitt, SA Stark, eds. The Causes of Crime: New Biological Approaches. New York: Cambridge, 1987, pp 92–109.
27. EF Coccaro, CS Bergeman, RJ Kavoussi, AD Seroczynski. Heritability of aggression and irritability: a twin study of the Buss-Durkee aggression scales in adult male subjects. Biol Psychiatry 41(3):273–284, 1997.
28. JP Rushton, DW Fulker, MC Neale, DKB Nias, HJ Eysenck. Altruism and aggression: the heritability of individual differences. J Pers Soc Psychol 50(6):1192–1198, 1986.
29. A Tellegan, DT Lykken, TJ Bouchard Jr, K Wilkox, NL Segal, S Rich. Personality similarity in twins reared apart and together. J Pers Soc Psychol 54:1031–1039, 1988.
30. JP Gustavsson, NL Pedersen, M Åsberg, D Schalling. Exploration into the sources of individual differences in aggression-, hostility- and anger-related (AHA) personality traits. Pers Individ Diff 21:1067–1071, 1996.
31. EF Coccaro, CS Bergeman, GE McClearn. Heritability of irritable impulsiveness: a study of twins reared together and apart. Psychiatry Res 48:229–242, 1993.
32. KL Jang, WJ Livesley, PA Vernon, DN Jackson. Heritability of personality disorder traits: a twin study. Acta Psychiatr Scand 94(6):438–444, 1996.
33. DL DiLalla, G Carey, II Gottesman, TJ Bouchard Jr. Heritability of MMPI personality indicators of psychopathology in twins reared apart. J Abnorm Psychol 105:491-499, 1996.
34. NL Pedersen, P Lichtenstein, R Plomin, U deFaire, GE McClearn, KA Matthews. Genetic and environmental influences on Type A–like measures and related traits: a study of twins reared apart and twins reared together, Psychosom Med 51:428-440, 1989.
35. G Weidner, T Rice, SS Knox, RC Ellison, MA Province, DC Rao, MW Higgins. Familial resemblance for hostility: the National Heart, Lung and Blood Institute Family Heart Study. Psychosom Med 62:197–204, 2000.
36. JA Mattes, M Fink. A family study of patients with temper outbursts. J Psychiatr Res 21:249–255, 1987.
37. TE Moffitt. Adolescence-limited and life-course persistent antisocial behavior: a developmental taxonomy. Psychol Rev 100:674–701, 1993.
38. A Caspi, B Henry, RO McGee, PA Silva. Temperamental origins of child and adolescent behavior problems: from age three to age fifteen. Child Dev 66:19–30, 1995.
39. D Finkel, M McGue. Sex differences and nonadditivity in heritability of the Multidimensional Personality Questionnaire scales. J Pers Soc Psychol 72:929–938, 1997.
40. P McGuffin, II Gottesman. Genetic influences on normal and abnormal development. In: M Rutter L Hersov, eds. Child and Adolescent Psychiatry: Modern Approaches. Boston: Blackwell, 1985, pp 17–33.
41. KJ Saudino, JR Gagne, J Grant, A Ibatoulina, T Marytuina, I Ravich-Scherbo, K Whitfield. Genetic and environmental influences on personality in adult Russian twins. Int J Behav Dev 23:375–389, 1999.
42. LJ Eaves, NG Martin, SBG Eysenck. An application of the analysis of covariance structure to the psychological study of impulsiveness. Br J Math Stat Psychol 30: 185–197, 1977.

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43. JS Loehlin. Genes and Environment in Personality Development. Austin: University of Texas Press, 1992.
44. R Plomin, R Corley, Caspi, A, DW Fulker, J DeFries. Adoption results for self-reported personality: evidence for nonadditive genetic effects? J Pers Soc Psychol 75(1):211-218, 1998.
45. S Scarr, PL Webber, RA Weinberg, MA Wittig. Personality resemblance among adolescents and their parents in biologically related and adoptive families. J Pers Soc Psychol 40(5):885–898, 1981.
46. AD Seroczynski, CS Bergeman, EF Coccaro. Etiology of the impulsivity/aggression relationship: genes or environment? Psychiatry Res 86(1):41–57, 1999.
47. M Rutter, J Silberg. Gene-environment interplay in relation to emotional and behavioral disturbance. Annu Rev Psychol 53:463–90, 2002.
48. S Scarr, K McCartney. How people make their own environments: a theory of genotype greater than environment effects. Child Dev 54(2):424–435, 1983.
49. CS Bergeman, R Plomin. Genotype-environment interaction. In: M Bornstein J Bruner, eds. Interaction in Human Development. Hillsdale, NJ: Lawrence Erlbaum Associates, 1989, pp 157-171.
50. SJ Suomi. A behavioral perspective on developmental psychopathology: excessive aggression and serotonergic dysfunction in monkeys. In: AJ Sameroff, M Lewis, S Miller, eds. Handbook of Developmental Psychopathology, 2nd ed. New York: Plenum Press, 2000, pp 237–256.
51. D Wahlsten. Experimental design and statistical inference. In: WE Crusio, RT Gerlai, eds. Handbook of Molecular Genetic Techniques for Brain and Behavior Research (Techniques in the Behavioral and Neural Sciences). Amsterdam: Elsevier, 13:40-57, 1990.
52. GE McClearn, GP Vogler, SM Hofer. Environment-gene and gene-gene interactions. In: EJ Masoro, SN Austad, eds. Handbook of the Biology of Aging, 5th ed. New York: Academic Press, 2001, pp 423–444.
53. RJ Cadoret, C Cain. Sex differences in predictors of antisocial behavior in adoptees. Arch Gen Psychiatry 37(10):1171–1175, 1980.
54. RJ Cadoret, CA Cain, RR Crowe. Evidence for gene-environment interaction in the development of adolescent antisocial behavior. Behav Genet 13(3):301–310, 1983.
55. RJ Cadoret, WR Yates, E Troughton, G Woodworth, MA Stewart. Genetic-environmental interaction in the genesis of aggressivity and conduct disorders. Arch Gen Psychiatry 52:916–924, 1995.
56. K Riggins-Caspers, RJ Cadoret, W Panak, et al. Gene × environment interaction and the moderating effect of adoption agency disclosure on estimating genetic effects. Pers Individ Dif 27:357–80, 1999.
57. DC Rowe, DM Almeida, K Jacobson. School context and genetic inflences on aggression in adolescence. Psychon Sci 10:277-280, 1999.
58. SM Monroe, AD Simons. Diathesis-stress theories in the context of life-stress research: implications for the depressive disorders. Psychol Bull 110(3):406–425, 1991.
59. M Zuckerman. Vulnerability to Psychopathology: A Biosocial Model. Washington. American Psychological Association, 1999.

MARCEL DEKKER, INC.
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60. R Plomin, JC DeFries, JC Loehlin. Genotype environment interaction and correlation in the analysis of human behavior. Psychol Bull 84:309–322, 1977.
61. L Murray, PJ Cooper, eds. Postpartum Depression and Child Development. New York: Guilford, 1997.
62. M Rutter. Psychiatric disorders in parents as a risk factor for children. In: D Scaffer, I Phillips, NB Enzer, eds. Prevention of Mental Disorders, Alcohol and Other Drug Use in Children and Adolescents. OSAP Prevention Monogr 2. Rockville, MD: Office of Substance Abuse and Prevention, U.S. Department of Health and Human Services, 1989, pp 157–189.
63. RD Rende. The stress of first grade and its relation to behavior problems in school. In: JC DeFries, R Plomin, DW Fulker, eds. Nature and Nurture in Middle Childhood. Cambridge, MA: Blackwell, 1994.
64. R Plomin. Genetics and Experience: The Interplay Between Nature and Nurture. Thousand Oaks, CA: Sage Publications, 1994.
65. R Plomin, P Lichtenstein, NL Pedersen, GE McClearn, JR Nesselroade. Genetic influence on life events during the last half of the lifespan. Psychol Aging 5:25–30, 1990.
66. KS Kendler, M Neale, R Kessler, A Heath, L Eaves. A study of recent life events and difficulties. Arch Gen Psychiatry 50:789-796, 1993.
67. R Plomin, CS Bergeman. The nature of nurture: genetic influence on "environmental" measures. Behav Brain Sci 14:414–424, 1991.
68. M Roff, SB Sells, MM Golden. Social adjustment and personality development in children. Minneapolis: University of Minnesota Press, 1972.
69. LA Baker, D Daniels. Nonshared environmental influences and personality differences in adult twins. J Pers Soc Psychol 51:1173–1182, 1990.
70. B Manke, S McGuire, D Reiss, EM Hetherington, R Plomin. Genetic contributions to adolescents' extrafamilial social interactions: teachers, friends, and peers. Soc Dev 4(3):238-256, 1995.
71. A Caspi. Why maladaptive behaviors persist: sources of continuity and change across the life course. In: DC Funder, RD Parke, C Tomlinson-Keasey, K Widaman, eds. Studying Lives Throughout Time. Washington. American Psychological Association, 1993, pp 343–377.
72. CS Bergeman, R Plomin, NL Pedersen, GE McClearn, JR Nesselroade. Genetic and environmental influences on social support: the Swedish Adoption/Twin Study of Aging (SATSA). J Gerontol 45:101–106, 1990.
73. RC Kessler, KS Kendler, A Heath, MC Neale, LJ Eaves. Social support, depressed mood, and adjustment to stress: a genetic epidemiologic investigation. J Pers Soc Psychol 62:257–272, 1992.
74. R Plomin, NL Pedersen, GE McClearn, JR Nesselroade, CS Bergeman. EAS temperaments during the last half of the life span: twins reared apart and twins reared together. Psychol Aging 3:43–50, 1988.
75. R Plomin, NL Pedersen, GE McClearn, JR Nesselroade, CS Bergeman. Genetic influence on adults' ratings of their current family environment. J Marriage Fam 51(3):791–803, 1989.
76. KW Schaie, SL Willis. Family environments across generations. In: VL Bengston, KW Schaie, L Burton, eds. Adult Intergenerational Relations: Effects of Societal Change. New York: Springer, 1995, pp 174–209.

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77. DC Rowe. A biometrical analysis of perceptions of family environment: a study of twin and singleton sibling kinships. Child Dev 54:416–423, 1983.
78. DC Rowe. Environmental and genetic influences on dimensions of perceived parenting: a twin study. Dev Psychol 17:203–208, 1981.
79. SL Hershberger, P Lichtenstein, SS Knox, GE McClearn. Genetic and environmental influences on perceptions of organizational climate. J Appl Psychol 79:24–33, 1994.
80. R Plomin, R Corley, JC DeFries, DW Fulker. Individaul differences in television viewing in early childhood: nature as well as nurture. Psychol Sci 1:371–377, 1990.
81. MT Tsuang, MJ Lyons, SA Eisen, WT True, J Goldberg, W Henderson. A twin study of drug exposure and initiation of use. Behav Genet 22:756, 1992.
82. P Lichtenstein, SL Hershberger, NL Pedersen. Dimensions of occupations: genetic and environmental influences. J Biosoc Sci 27:193–206, 1995.
83. P Lichtenstein, NL Pedersen, GE McClearn. The origins of individual differences in occupational status and educational level: a study of twins reared apart and together. Acta Sociol 35:1285–1292, 1992.
84. K Tambs, JM Sundet, P Magnus, K Berg. Genetic and environmental contributions to the covariance between occupational status, educational attainment and IQ: a study of twins. Behav Genet 19:209–222, 1989.
85. R Plomin, JM Neiderhiser. Genetics and experience. Curr Directions Psychol Sci 1:160–163, 1992.
86. M Zuckerman, JC Ballinger, RM Post. The neurobiology of some dimensions of personality. Int Rev Neurobiol 25:391–346, 1984.
87. M Rutter. Nature-nurture integration: the example of antisocial behavior. Am Psychol 52(4):390–398, 1997.

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3

Developmental Models of Aggression

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INTRODUCTION

In this chapter, we review developmental theories related to aggressive behavior. The development of aggression and other forms of antisocial behavior has been a central concern in the field of developmental psychopathology (1), and a large body of research has focused on elucidating the development of these behaviors. As will be evident, however, current developmental models do not always distinguish aggression and other antisocial acts. Whenever possible, this review will focus specifically on aggression, and we will describe one developmental model that explicitly illustrates different pathways to aggression versus other antisocial behaviors. In addition to reviewing prominent theories on the development of aggression, we will also consider social information processing approaches to understanding aggressive behavior, discuss risk factors that cut across developmental models, and outline prevention and treatment efforts as they relate to these models. Although we recognize the importance of genetic and physiologic factors in the development of aggression, consideration of these factors is beyond the scope of the current chapter (see 2, 3 for reviews).

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DEVELOPMENTAL THEORIES OF AGGRESSION AND ANTISOCIAL BEHAVIOR

Moffitt's Life Course–Persistent vs. Adolescence-Limited Developmental Taxonomy

During the past decade, several models for the development of aggression and other forms of antisocial behavior have been proposed. One such model is the developmental taxonomy proposed by Moffitt (4). This taxonomy emerged from Moffitt's longitudinal investigation of a large sample of New Zealand children who were assessed every 2 years between the ages of 3 and 15 (5). Approximately 5% of the males in this sample displayed deviant levels of antisocial behavior at every assessment. However, for a much larger segment—about 33% of the total sample—such behavior did not emerge until subjects were at least 11 years old.

These divergent pathways to antisocial behavior are reflected in Moffitt's hypothesis that there are two types of offenders with distinct profiles of risk factors and developmental courses: life course–persistent offenders and adolescence-limited offenders. As implied by the label, the hallmark of life course–persistent offenders is the continuity of antisocial behavior across the life course, with the actual form this behavior takes changing with development. For example, such individuals might engage in: "biting and hitting at age 4, shoplifting and truancy at 10, selling drugs and stealing cars at age 16, robbery and rape at age 22, and fraud and child abuse at age 30" (4:679). In addition to this temporal continuity in their antisocial behavior, these individuals also display such behavior across a range of contexts (e.g., with parents, with peers, and with teachers). Despite representing little more than 5% of the male population, Moffitt suggests that life course–persistent offenders account for a substantial percentage of violent crime.

In contrast to these offenders whose antisocial behavior begins early in life and persists across development, there is a much larger group of individuals whose antisocial behavior begins and ends during adolescence. Adolescence-limited offenders comprise the vast majority of antisocial youth in any cross-sectional sample of the adolescent population, and Moffitt regards them very differently from life course–persistent offenders in terms of why their antisocial behavior begins and why it desists.

According to Moffitt, life course–persistent antisocial behavior is rooted in neuropsychological deficiencies that are present early in childhood. These deficiencies include poor verbal skills and executive functioning deficits such as inattention and impulsivity—characteristics that can make these children more difficult to parent. In environments of risk and adversity, which are disproportionately represented in the lives of children with such characteristics, parents often lack the necessary psychological and physical resources to cope with a challeng-

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ing child. It is this juxtaposition of a "vulnerable and difficult infant with an adverse rearing context that initiates risk for the life course–persistent pattern of antisocial behavior" (4:682). These children enter school ill-equipped to succeed either academically or socially, and, over time, accumulating consequences of the child's academic and social difficulties increasingly limit the options for positive change. The result is a lifelong pattern of antisocial behavior that often escalates to include crimes of interpersonal violence.

For adolescence-limited offenders, the developmental course and associated risk factors are quite different. These individuals are not afflicted with the various neuropsychological deficits that are common among life course–persistent offenders, and their early development is not marked by conflictual parent-child relationships and the early emergence of antisocial behavior. For these offenders, antisocial behavior does not begin until adolescence, when Moffitt argues it is sufficiently common among males to be considered "normative." Moffitt believes this surge in antisocial behavior occurs largely because of the "maturity gap" that exists for adolescents in contemporary Western society; that is, there is a gap between adolescents' desires for independence and access to adult privileges and the reality of remaining largely dependent upon, and controlled by, parents and other adults. For adolescents trapped in this maturity gap, the behavior they observe in their life course–persistent peers who appear relatively free of familial constraints becomes increasingly appealing during this time. Moffitt hypothesizes that many adolescents begin engaging in similar behavior through a process of "social mimicry" as a way of "proving that they can act independently and conquer new challenges" (6:500).

As these individuals move through adolescence and an ever-increasing range of legitimate and tangible adult roles becomes available, shifting environmental contingencies make alternatives to criminal activity more rewarding, and they have skills to take advantage of newly available opportunities. Because access to desired outcomes no longer requires antisocial acts, such behavior ends with the transition to young adulthood. Their brief antisocial careers are thus attributed to social and contextual variables rather than to deviant individual characteristics that set the stage for an ongoing pattern of conflictual relationships, skill deficits, and antisocial behavior. Thus, apart from similarities in antisocial activity during the peak offending adolescent years, the developmental trajectories for life course–persistent and adolescence-limited offenders is dramatically different.

Moffitt's developmental taxonomy represents an elegant interpretation of the available literature and provides a compelling theoretical account for how antisocial behavior that appears similar during adolescence may have dramatically different antecedents and consequences. Recently, however, a prospective study that followed individuals from birth through age 16 calls into question certain key elements of her theory (7). Consistent with Moffitt's theory, an early-onset/persistent group and an adolescence-onset group of antisocial individuals

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were identified by these researchers. Contrary to Moffitt's theory, however, these groups were not distinguished by measures of temperament and neuropsychological functioning during the first 3 years but instead differed on measures of early psychosocial adversity. It was not until late childhood and adolescence that differences in neuropsychological functioning emerged. In addition, adolescence-onset offenders reported higher levels of internalizing symptoms and life stress than nonoffenders, suggesting that adolescence-onset antisocial behavior is not necessarily a benign phenomenon as Moffitt describes.

Patterson's Early vs. Late Starter Model

A related developmental model referred to as the early vs. late starter model has been proposed by Patterson and colleagues (8). Like Moffitt, these investigators believe there is an important distinction between individuals whose antisocial behavior begins at different developmental stages. Similar to the life course–persistent offenders in Moffitt's taxonomy, it is the early starters in Patterson's model who are hypothesized to be at greater risk for chronic antisocial behavior that extends beyond adolescence and into adulthood.

Although similar to Moffitt's developmental taxonomy in these respects, Patterson's early vs. late starter model differs in the factors hypothesized to place individuals on the different antisocial paths. For Moffitt, it is individual child characteristics—primarily verbal and executive functioning deficits—that set the stage for difficult parent-child relationships and a cascading pattern of ensuing difficulties that result in life course–persistent offending. Although such characteristics are not sufficient by themselves for the life course–persistent pattern to develop, they are viewed as critical risk factors for initiating the transactional processes that Moffitt describes. Patterson, in contrast, argues that the early starter path is initiated by poor family management practices, particularly unskilled discipline that is characterized by negative reinforcement of children's coercive and noncompliant behavior.

In the typical exchange, a parent's attempts to discipline a child are ignored or met with protest. Rather than calmly but firmly enforcing the demand, the parent reacts in a neutral or even positive manner and often withdraws. The child's noncompliance is thus negatively reinforced, and when such exchanges are consistently repeated, the child learns to use coercive behaviors to gain control over family members. In the absence of countervailing forces, what begins as trivial aversive behaviors within the family often extends to similar behaviors with other people in other settings, and eventually to more serious antisocial behaviors including aggressing, lying, and stealing.

For many children with this early training in coercive behavior, there are accompanying social skill deficits that contribute to social rejection by peers during elementary school. Peer rejection, in turn, contributes incrementally to early

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starting antisocial behavior problems (9). Peer rejection is such an important risk factor for the development of aggression that it will be considered further in a subsequent section of this review. In the context of Patterson's model, coercive parent-child interactions and peer rejection exacerbate the risk for early starting aggressive children to become chronically antisocial adolescents and adults.

Patterson and colleagues also hypothesize a set of causal factors to explain the onset of antisocial behavior in adolescence that is different from that suggested by Moffitt. Recall that, for Moffitt, adolescence-onset antisocial behavior is viewed as a normative part of the transition to young adulthood, and occurs primarily as a means of establishing independence for youngsters caught in a maturity gap. Antisocial behavior that begins in adolescence is thus the product of macro-level contextual variables rather than being attributed to either deviant individual characteristics or problems in the more immediate family environment. In Patterson's model, in contrast, parent management skills are again accorded an important causal role. He argues that when parents with marginal behavior management skills are impacted by negative life events (e.g., divorce, illness, unemployment) or the normal strains that can accompany parent-child relations during adolescence, parental monitoring and supervision suffer, which contributes to adolescents' increasing involvement with deviant peers. The "training" and support for antisocial behavior by the deviant peer group leads late-starting youth to become involved in delinquent activity. For Patterson, therefore, the antisocial behavior of late starters is not seen as part of a normative transition to young adulthood that is determined predominantly by macro-level social forces to which virtually all adolescents are exposed. Instead, it is an outcome of difficulties in children's immediate family environment that results in increasing involvement with deviant peers. Unlike early starters, however, these adolescents have generally acquired the social and academic skills that enable them to desist from antisocial behavior when shifting environmental contingencies make other options more attractive. Thus, Patterson's explanation for desistance among later starters is similar to Moffitt's account of desistance in her adolescence-limited group.

Unresolved Issues Related to These Developmental Models

The developmental models of Moffitt and Patterson provide a valuable organizational framework for understanding the development of aggression and other forms of antisocial behavior. Although there are important differences between them, both describe distinct pathways leading to antisocial behavior during adolescence. The importance of distinguishing between antisocial behavior according to whether it develops during childhood or adolescence has received strong empirical support (see 10,11 for review). Furthermore, the fourth edition of the Diag-

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nostic and Statistical Manual of Mental Disorders (12) reflects these developmental models in the requirement to designate whether Conduct Disorder is of the childhood-onset type (i.e., characteristics of the disorder present prior to age 10 years) or adolescent-onset type (i.e., criteria for conduct disorder absent prior to age 10 years). There are, however, several important issues pertaining to these models that await resolution.

One such issue is whether early vs. late onset models are sufficient to capture the complexity of how antisocial behavior develops. While recognizing the utility of the conceptualization of early and late onset type, some researchers argue that the dual pathway oversimplifies the trajectories that serious antisocial behavior takes in many individuals (13). For example, some children show high levels of aggression at an early age but desist from such behavior later in childhood (14), a pathway not defined by either Moffitt or Patterson. In addition, although they are a distinct minority, some individuals who commit acts of violence as adults do not have an antecedent pattern of aggressiveness early in life (15). These late-onset antisocial individuals are also not represented in the developmental taxonomies of Moffitt or Patterson.

A second issue awaiting resolution is whether the Moffitt and Patterson models apply to antisocial behavior in girls. Knowledge of the processes that characterize the development of antisocial behavior in girls is relatively scarce. Although Moffitt's New Zealand sample included males and females, the developmental taxonomy she describes is clearly intended as an explanatory framework for understanding the development of antisocial behavior among boys. The sample in the longitudinal study that formed the basis of Patterson's theorizing was exclusively male.

Recently, Silverthorn and Frick (16) reviewed the limited literature on antisocial behavior in girls and concluded that the childhood vs. adolescent onset distinction, which has utility for conceptualizing the development of severe antisocial behavior for boys, may not apply to girls without important modifications. These researchers suggest that although antisocial girls show many of the correlates associated with early-onset antisocial behavior in boys (e.g., neuropsychological deficits, dysfunctional family environment), these factors typically do not lead to severe antisocial behavior until adolescence. They refer to this "delayed onset" pathway as being analogous to the childhood-onset pathway in boys. In addition, they believe that an adolescence-onset path that has relatively benign consequences does not exist for females. The interesting developmental model proposed by these researchers remains to be tested.

Loeber's Model of the Development of Aggression vs. Other Antisocial Behaviors

An additional issue not specifically addressed in Moffit's or Patterson's models is their applicability to the development of aggression specifically as opposed to

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antisocial behavior in general. Although there is an implicit assumption within each model that early onset individuals are more likely to commit crimes of interpersonal violence, neither presents an explicit account for the development and desistance of aggression specifically.

Currently, there is some disagreement among researchers as to whether the development of aggression requires a separate explanation from the development of antisocial behavior more broadly defined. Patterson and colleagues (17) argue that serious aggression is generally preceded by a variety of antisocial acts during childhood and adolescence rather than a distinct developmental pathway that is unique to aggression. In support of this view, other researchers (e.g., 18) have found little evidence that individuals specialize in different types of crimes (i.e., person vs. property).

In contrast to this position, Loeber and Stouthamer-Loeber (13) believe it is important to preserve the distinction between overt (i.e., aggression) and covert (i.e., property crime) forms of antisocial behavior, and note that orderly developmental progressions of each type have been identified (19). They argue that "a single pathway of antisocial behavior tends to obfuscate the development of violence and impede its explanation" (13:249), and that different familial, physiological, and genetic influences have been linked to overt and covert antisocial trajectories. Although overt and covert antisocial behaviors are highly correlated, and the majority of individuals who progress to violence on the overt pathway also commit serious covert antisocial acts, some individuals do seem to "specialize" in primarily one type of antisocial behavior (20). In the overt pathway, bullying and annoying behaviors develop into physical fighting, which in turn may develop into rape and other forms of violent attacks (see 13). In contrast, in the covert pathway, behaviors such as shoplifting and lying may develop into vandalism and other forms of property damage, which might in turn develop into fraud and burglary.

Loeber has also identified a separate authority conflict pathway in which stubborn behavior develops into defiance and disobedience, which may then develop into behaviors such as truancy and running away. Loeber and Stouthamer-Loeber (13) assert that a single causal model to explain the development of antisocial behavior is not adequate, and will hamper efforts to uncover developmental precursors that are specific to different types of offending. Thus, these researchers propose a distinct developmental model for aggression, in contrast to Moffitt's and Patterson's models, which do not distinguish pathways to aggression versus other types of antisocial behavior.

Social Information Processing Theory

The previously described models emphasize factors such as the importance of age of onset, parent-child interactions, peer relationships, and neuropsychological deficiencies in the development of aggression over time. However, these models

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do not explain the proximal factors that give rise to aggression in particular situations. That is, what factors influence, in real time, the likelihood that an individual will behave aggressively? Within the field of developmental psychopathology, social information processing theory has been the major theoretical framework for addressing this important question.

Part of this model is the "database" of prior experiences with parents and peers that children bring to new situations. As a result of prior social interactions, children develop cognitive schemata that influence their processing of social information in new situations. For example, interaction styles that children learn through coercive interactions with parents may generalize to children's interactions with peers (21). Children's beliefs about peers predict later aggression, even after controlling for earlier aggression (22). Thus, social information processing theory describes a set of cognitive mechanisms that have been found to account, in part, for the link between a host of risk factors and the subsequent development of aggression. According to this theory, the way children interpret a particular event influences how they will respond to that situation (23). Dodge and colleagues (e.g., 24,25) have proposed five steps in a model of social information processing: encoding, making attributions, generating responses, evaluating responses, and enacting responses.

Encoding is the process of taking in information from the environment. Encoding problems related to aggression arise if children either are hypervigilent to hostile cues or neglect to take in relevant nonhostile cues. For example, children who have been physically maltreated become more attentive to hostile cues in the environment and less attentive to other relevant social cues; poor encoding is, in turn, related to higher levels of subsequent aggression (24). Thus, encoding mediates the association between physical maltreatment and subsequent aggression, and also may mediate effects of other risk factors.

The second step in the model, making attributions, involves deciding what motivates the behavior of other people. On the basis of information children encode from a particular situation, they could decide that others acted with benign, hostile, or ambiguous intent. Children and adolescents who have hostile attribution biases are more likely than others to behave aggressively (26).

The third step, generating responses, is the process of thinking of behavioral responses to a given situation. Children may generate either aggressive or nonaggressive responses to problems. Children who later behave aggressively are likely to access fewer responses to social situations overall and to have a more easily accessible reservoir of aggressive responses available to them than do nonaggressive children (27).

Fourth, evaluating responses occurs when children assess whether a response is a good one to use in a particular situation and whether that response will be associated with desired outcomes. Aggressive children are more likely to endorse aggressive responses to problems positively and to believe that aggression will lead to desired interpersonal and instrumental outcomes (28). Furthermore, aggressive

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children feel more confident in their ability to enact hypothetical aggressive behaviors than do nonaggressive children, but less confident in their ability to withdraw from a provocative situation or inhibit an aggressive response (25).

Finally, enacting responses is the manner in which a child actually behaves. Children differ in the level of skill they demonstrate in enacting different behaviors. Aggressive children are relatively more skilled in enacting aggressive behaviors and relatively less skilled in enacting nonaggressive behaviors (29).

Recent evidence suggests that deficits in different aspects of social information processing are related to different types of aggression. Encoding errors and hostile attribution biases are associated with reactive aggression, which is characterized by hostile responses to perceived provocation; in contrast, positively evaluating the outcomes of aggressing is associated with proactive aggression, which is used instrumentally to obtain desired outcomes (30). These findings are consistent with hypothesized developmental courses for reactive versus proactive aggression. Reactive aggression is associated with early maltreatment, peer rejection, physiologic overarousal, and attention problems; proactive aggression is associated with having aggressive role models, friendships with other proactively aggressive children, physiological underarousal, and psychopathy (see, e.g., 31). Thus, a consideration of social information processing mechanisms is important in understanding the development of different forms of aggressive behavior.

These mechanisms cut across other developmental models of aggression because they deal with specific proximal links between environments, cognition, and behavioral outcomes. For example, peer rejection and problematic parent-child relationships, which play an important role in Patterson's account of early starting aggression, are associated with a host of social information processing deficits (24,32). Furthermore, social information processing deficits partially mediate the effects of early peer rejection and harsh parent-child relationships on later aggressive behavior (24,33). Thus, social information processing theory provides one explanation for proximal mechanisms leading to aggression in particular situations.

Other Risk Factors for Aggression

The theories described above integrate multiple risk factors in their accounts of the development of aggression. Risk factors that deserve particular attention apart from these theories include peer rejection and association with deviant peers, poverty, exposure to violent media, impulsivity, and male gender. Exposure to these factors singly or in combination with one another increases the risk for the development of aggressive behavior problems.

Peer Rejection and Association with Deviant Peers

Early aggression is one of the best predictors of peer rejection during elementary school (see 34 for review), but empirical studies have supported the role of peer

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rejection not just as a consequence or marker of childhood aggression, but as an independent contributor to subsequently higher levels of future aggression (e.g., 35,36). Dodge et al. (33) followed two large samples of elementary-school children for up to 5 years and found that early peer rejection predicted later aggressive behavior, even after controlling for prior aggression and other factors related to the development of aggression. Furthermore, Rabiner and colleagues (37) found that for adolescents who committed a felony assault, prior rejection by peers significantly predicted the persistence of aggressive criminal behavior into young adulthood.

Associating with deviant peers also contributes to the development of aggression. Analyses of videotaped interactions between 13- and 14-year-old boys and their best friends showed that antisocial dyads were more likely than low antisocial dyads to reinforce delinquent behavior in their discussions, particularly through laughing at antisocial comments (38). Similarly, Bagwell and Coie (39) found that when 10-year-old boys were observed with their best friends, antisocial dyads encouraged each other to cheat and break the rules set for them in a controlled task more than did nonantisocial dyads. Thus, rejection by conventional peers and association with deviant peers both appear to play an important role in the development of aggression.

Poverty

Poverty is related to higher levels of aggression among children, adolescents, and adults (e.g., 40). The association between poverty and children's aggression is, in part, mediated by effects of poverty on parents' psychological distress and their ability to parent effectively (41). Yet, in some contexts, the root of aggression appears to lie not within the child or parents but within an environment that makes engaging in antisocial behavior beneficial. For example, although joining a gang places children at risk for a number of negative outcomes, including aggression, children who live in dangerous neighborhoods may also be placed at risk by not joining a gang. From the child's perspective in such a situation, aggression may be quite adaptive (42,43). Thus, poverty may contribute to the development of aggression through a number of mechanisms including family stress and dangerous neighborhoods.

Exposure to Violent Media

An additional risk factor that has emerged as a consistent and strong predictor of aggressive behavior is exposure to violent media. Meta-analyses have shown that the association between exposure to violent media and aggressive behavior is almost as strong as the association between smoking and lung cancer; the link between exposure to violent media and aggressive behavior is stronger than other well-established links such as those between condom use and sexually transmitted HIV and between exposure to lead and children's IQ scores (44). Scientific confidence in these findings was asserted by the American Academy of Child and

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Adolescent Psychiatry, the American Academy of Family Physicians, the American Academy of Pediatrics, the American Medical Association, the American Psychiatric Association, and the American Psychological Association in a joint statement regarding the overwhelming evidence pointing to a "causal connection between media violence and aggressive behavior in some children" (45:1).

Anderson and Bushman's General Aggression Model (46) illustrates how repeated exposure to violent media contributes to aggressive behavior by altering individuals' cognition, affect, and arousal. According to the model, each exposure to violent media teaches individuals ways to aggress, influences beliefs and attitudes about aggression, primes aggressive perceptions and expectations, desensitizes individuals to aggression, and leads to higher levels of physiological arousal (46). Each of these mediating variables, in turn, leads to higher levels of aggressive behavior. Most evidence suggests that it is not merely that already aggressive children seek out violent media, but that exposure to violent media contributes to the development of aggressive behavior (47).

Impulsivity and Hyperactivity

Impulsivity, inattention, and hyperactivity are yet other risk factors for aggression (37). Individuals who have problems with impulsivity, inattention, and hyperactivity may be at increased risk for aggression because they are impaired in their ability to inhibit aggressive responses once they are cognitively accessed (48). Taylor et al. (49) followed a group of boys who had been identified as early starters (antisocial behavior present by age 11), late starters (antisocial behavior present at age 14 or 17, but not by age 11), or nondelinquent controls (no indicators of antisocial behavior at any time). They found that 40% of their early starters, 8% of their late starters, and 0% of their control group met DSM-III-R criteria for an attention-deficit/hyperactivity disorder (ADHD) diagnosis; thus, ADHD appears to be a particular risk factor for early onset of aggressive behavior problems. Hyperactivity is arguably the personal characteristic most strongly associated with the long-term development of antisocial behavior (10). In a high-risk sample of 1037 boys followed from the age of 6 to 15 years, Nagin and Tremblay (50) found that high levels of hyperactivity and oppositional behavior in kindergarten each increased the odds of boys' displaying a developmental trajectory of high aggression by a factor of 3; in combination, kindergarten hyperactivity and opposition increased the odds of a highly aggressive trajectory by a factor of 9.

Gender

Male gender itself also has been identified as a risk factor for the development of aggression. Crime statistics attest to the gender imbalance in aggression; during 2000, 83% of individuals arrested for violent crimes in the United States were males (51). Among children, compared to girls, boys are four times more likely to receive diagnoses of conduct disorder (52). The preponderance of empirical

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studies of aggression has focused on males, largely because of the higher levels of aggression among them. Recently, however, researchers have documented types of relational, social, or indirect aggression that are more common among girls than among boys. In contrast to overt aggression, which generally takes the form of physical acts or direct insults, relational aggression causes harm through its effects on social relationships, such as by exclusion of a peer or negative gossip. Crick and Grotpeter (53) used third- through sixth-grade peer nominations to construct groups of children who were 1) not aggressive, 2) overtly aggressive, 3) relationally aggressive, and 4) both overtly and relationally aggressive. They found that 16% of the boys compared to 0.4% of the girls were classified as exclusively overtly aggressive, whereas 2% of the boys compared to 17% of the girls were classified as exclusively relationally aggressive. Crick (54) found that girls were more distressed by hypothetical relationship dilemmas than were boys; thus, girls may engage in higher levels of relational aggression because this is a more effective way to inflict harm in girls' than in boys' peer groups. Attempts to understand the development of aggression benefit from taking into account risk factors such as peer rejection and association with deviant peers, poverty, exposure to violent media, impulsivity, and gender in the context of overarching developmental models.

PREVENTION AND TREATMENT

Protective Factors

Although most antisocial adults had behavior problems as children, most children with conduct problems do not grow up to be antisocial adults (11). Thus, it is important to identify protective factors that disrupt the progression from an aggressive childhood to an aggressive adulthood. Maughan and Rutter (11) identified three categories of experiences that could serve as turning points away from continuing antisocial trajectories: the introduction of new opportunities, radical changes in the environment, and experiences that alter individuals' view of themselves and others. For example, supportive marriages, positive work, and geographical moves have the potential to deflect aggressive trajectories (55), and having a single good relationship may serve as a buffer against otherwise negative early environments for a child (56). The developmental course of aggression is related to the presence of risk and protective factors (57), and both influences need to be considered in efforts to understand the development of such a complex and multidimensional construct as aggression.

Prevention and Treatment Approaches Suggested by Different Developmental Models

The developmental models described above suggest different ways of preventing and treating aggressive behavior problems. For example, both Moffitt's and Pat-

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terson's models imply that the onset of aggression in adolescence is related to fewer long-term difficulties than is aggression that begins earlier in life; thus, there would be value in delaying the onset of aggression, even if its occurrence could not be prevented entirely. In addition, Patterson's model suggests that intervening early with parents to prevent their use of coercive disciplinary strategies and instead to encourage contingent responding and consistent practices will result in less aggression for children. Loeber's work intimates that different interventions would be needed for children on the pathway to aggression vs. those on the pathway to covert forms of antisocial behavior.

Peer affiliation models suggest that teaching children social skills that will help them be more accepted by their peers could interrupt a cycle of peer rejection, association with deviant peers, and aggression. Furthermore, because affiliations with antisocial peers tend to propagate aggression, interventions that group antisocial children together would be ill advised. Consistent with this position, McCord (58) reported that the number of times antisocial boys from the Cambridge-Sommerville project were sent to a summer camp for delinquent youth was related to higher rates of negative outcomes. McCord postulates that at summer camp, delinquent youth were able to form strong bonds with each other; thus, the treatment inadvertently increased rather than decreased the boys' risk for negative outcomes. Coie and Miller-Johnson (59) concluded from their review of group interventions that "group discussions among adolescents may inadvertently reinforce antisocial attitudes that emerge as part of the unplanned dialogue among group members, as well as promoting antisocial friendships that continue outside group sessions" (5:23). Interventions are complicated in contexts such as high-risk neighborhoods where aggression is normative rather than deviant. In these contexts interventions may need to be at the level of the neighborhood rather than at the level of the individual child (42).

Social information processing models suggest the need for intervention at the level of children's cognitive processing. Intervention based on social information processing theory would aim to reduce aggressive behavior by promoting the encoding of relevant social cues, modifying children's hostile attribution biases, helping children generate nonaggressive responses to provocation, decreasing children's positive evaluations of aggressing, and training children to enact nonaggressive solutions to problems. Problem-solving skills training has been shown to have the potential to reduce antisocial behavior over time (e.g., 60,61). These interventions typically provide children with a series of individually administered sessions in which therapists use cognitive and behavioral techniques to attempt to improve children's problem-solving skills (e.g., generating nonaggressive solutions) in social situations (62). Children are asked to role-play and practice effective behaviors, and therapists provide feedback and reinforcement. Despite the promise of these interventions, social cognition is only one component in understanding the development of aggression. We turn next to a description of a specific intervention program that incorporates many features

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that follow from an understanding of the different developmental models described above.

The Fast Track Program

Fast Track (Families and Schools Together) is a comprehensive intervention that incorporates parent training, home visits, social skills training, academic tutoring, and classroom intervention for a high-risk group of 445 children, with the goals of preventing conduct problems and promoting social competence, positive peer relationships, and academic performance (63). Children in four geographic regions of the United States were screened during kindergarten for disruptive behavior and problems with peer relationships; schools were randomly assigned to intervention and control groups. Compared to a control group of 445 children who did not receive the intervention, children in the intervention group showed fewer behavior problems at school and improved peer relations after the first year of the intervention (64). In addition, classrooms in which the Fast Track curriculum had been implemented had fewer problems with aggression than did control classrooms (65). Fast Track is reviewed in some detail here because it represents the most comprehensive effort to date to develop a prevention program for aggression based on the developmental theories discussed previously and because it has shown promise as an effective approach for preventing the development of aggressive behavior (see 63 for additional details).

Parent training is an important component of the Fast Track program (63). In early elementary school, weekly sessions held with groups of parents have a structured curriculum that focuses on developing positive family-school relationships and on teaching parenting skills that will promote positive parent-child relationships and decrease child conduct problems. After each parent group, parents and children are brought together for a 30-min session to focus on applying concepts that were covered during the parent group. A second important component of the program that also involves parents is biweekly home visiting. During these home visits, family coordinators work with parents to solve problems they are currently facing, with an emphasis on empowering parents to solve future problems on their own, and promoting parents' efforts to provide a safe and supportive home environment for their child.

Another important component of Fast Track involves providing social skills training to targeted children (63). Each group lesson focuses on a specific skill (e.g., communication, emotional expression, taking turns), with time allotted for practicing the skill and receiving feedback on their enactment of it. In addition, children are each paired with a classroom peer for a weekly 30-min guided play session. The overall goal of this component is to improve children's social information processing skills and promote positive peer relationships.

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The fourth program component involves tutoring in reading skills. Tutors meet with individual children three times a week, and one of these meetings includes the children's parents to encourage them to be involved in their children's schooling. A goal of this component is to reduce children's disruptiveness in the classroom and increase their acceptance by peers through facilitating academic achievement.

The final component of Fast Track is intervention at the level of the classroom (63). Three times a week, teachers present lessons from the Promoting Alternative Thinking Strategies curriculum (66), which is designed to promote self-control, problem-solving skills, positive peer relationships, and emotional awareness. In addition, teachers work with Fast Track staff to implement classroom procedures such as setting clear rules, reinforcing appropriate behaviors, and responding to problematic behaviors to minimize disruptions in the classroom. Unlike the other program components that are directed specifically at children who are part of the Fast Track intervention, this final component is directed at all of the children in the classroom and is thus not just for children receiving the other components of the intervention.

The Fast Track intervention model is in large part based on developmental models of conduct problems, and focuses on factors that place children at risk for early starter aggression. The intervention incorporates many features that are hypothesized to be related to a reduction in early starter aggressive behavior from the perspective of the developmental models previously described. For example, the parent training component is intended to decrease the problematic parent-child interaction sequences that Patterson has found to predict early starting aggression, the peer component may increase children's acceptance by conventional peers and thereby decrease their involvement with deviant peers, and the classroom component may enhance children's social information processing skills. Because the Fast Track program is designed to address multiple risk factors for the development of serious aggressive behavior problems, it has the potential to alter developmental pathways toward aggression.

SUMMARY AND CONCLUSIONS

Our goal has been to review major developmental models of aggression and other forms of antisocial behavior, risk factors that cut across these models, and intervention efforts that emanate from this developmental perspective. Different models attempt to account for early versus late onset of aggression, developmental pathways of aggression compared to other antisocial behaviors, and social information processing mechanisms that increase aggressive behaviors. Risk factors such as peer rejection, poverty, exposure to violent media, and impulsivity promote the development of aggression, whereas protective factors such as positive social relationships may inhibit aggression. Understanding why and how aggres-

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sion develops is crucial to designing effective intervention programs to prevent and reduce aggression over time.

REFERENCES

1. Sroufe, L. A., Rutter, M. (1984). The domain of developmental psychopathology. Child Dev 54, 17–29.
2. Brain, P. F., Susman, E. J. (1997). Hormonal aspects of aggression and violence. In D. M. Stoff, J. Breiling, J. D. Maser (Eds.), Handbook of Antisocial Behavior. New York: Wiley, pp 314–323.
3. Carey, G. (1994). Genetics and violence. In A. J. Reis, K. A. Miczek, J. A. Roth (Eds.), Understanding and Preventing Violence, Vol 2. Biobehavioral Influences. Washington: National Academy Press, pp 21–58.
4. Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. Psychol Rev 100, 674–701.
5. Moffitt, T. E. (1990). Juvenile delinquency and attention-deficit disorder: developmental trajectories from age 3 to 15. Child Dev 61, 893–910.
6. Caspi, A., Moffitt, T. E. (1995). The continuity of maladaptive behavior: from description to understanding in the study of antisocial behavior. In D. Cicchetti, D. J. Cohen (Eds.), Developmental Psychopathology, Vol 2. New York: Wiley, pp 472–511.
7. Aguilar, B., Sroufe, L. A., Egeland, B., Carlson, E. (2000). Distinguishing the early-onset/persistent and adolescence-onset antisocial behavior types: from birth to 16 years. Dev Psychopathol 12, 109–132.
8. Patterson, G. R., Capaldi, D., Bank, L. (1991). An early starter model for predicting delinquency. In D. J. Pepler, K. H. Rubin (Eds.), The Development and Treatment of Childhood Aggression. Hillsdale, NJ: Erlbaum, pp 139–168.
9. Miller-Johnson, S., Coie, J. D., Maumary-Gremaud, A., Bierman, K. (2002). Conduct Problems Prevention Research Group. Peer rejection and aggression and early starter models of conduct disorder. J Abnorm Child Psychol 30, 217–230.
10. Dishion, T. J., French, D. C., Patterson, G. R. (1995). The development and ecology of antisocial behavior. In D. Cicchetti, D. J. Cohen (Eds.), Developmental Psychopathology, Vol 2. New York: Wiley, pp 421–471.
11. Maughan, B., Rutter, M. (1998). Continuities and discontinuities in antisocial behavior from childhood to adult life. In T. H. Ollendick, R. J. Prinz (Eds.), Advances in Clinical Child Psychology, Vol 20. New York: Plenum Press, pp 1–47.
12. American Psychiatric Association. (1994). Diagnostic and Statistical Manual of Mental Disorders, 4th ed. Washington: American Psychiatric Press.
13. Loeber, R., Stouthamer-Loeber, M. (1998). Development of juvenile aggression and violence: some common misconceptions and controversies. Am Psychol 53, 242–259.
14. Kingston, L., Prior, M. (1995). The development of patterns of stable, transient, and school-age onset antisocial behavior in young children. J Am Acad Child Adolesc Psychiatry 34, 348–358.
15. Farrington, D. P. (1994). Childhood, adolescent, and adult features of violent males.

MARCEL DEKKER, INC.
270 Madison Avenue, New York, New York 10016

In L. R. Huesmann (Ed.), Aggressive Behavior: Current Perspectives. New York: Plenum Press, pp 215–240.
16. Silverthorn, P., Frick, P. J. (1999). Developmental pathways to antisocial behavior: the delayed-onset pathway. Dev Psychopathol 11, 101–126.
17. Patterson, G. R., Reid, J. B., Dishion, T. J. (1992). Antisocial Boys. Eugene, OR: Castalia.
18. Farrington, D. P. (1991). Childhood aggression and adult violence: early precursors and later life outcomes. In D. J. Pepler, K. H. Rubin (Eds.), The Development and Treatment of Childhood Aggression. Hillsdale, NJ: Erlbaum, pp 73–93.
19. Loeber, R., Wung, P., Keenan, K., Giroux, B., Stouthamer-Loeber, M., Van Kammen, W. B., Maughan, B. (1993). Developmental pathways in disruptive child behavior. Dev Psychopathol 5, 101–132.
20. Garber, J., Hollon, S. D. (1991). What can specificity designs say about causality in psychopathy research? Psychol Bull 110, 129–136.
21. Patterson, G. R., Dishion, T. J., Bank, L. (1984). Family interaction: a process model of deviancy training. Aggress Behav 10, 253–267.
22. MacKinnon-Lewis, C., Rabiner, D., Starnes, R. (1999). Predicting boys' social acceptance and aggression: the role of mother-child interactions and boys' beliefs about peers. Dev Psychol 35, 632–639.
23. Crick, N. R., Dodge, K. A. (1994). A review and reformulation of social information-processing mechanisms in children's social adjustment. Psychol Bull 115, 74-101.
24. Dodge, K. A., Bates, J. E., Pettit, G. S. (1990). Mechanisms in the cycle of violence. Science 250, 1678–1683.
25. Dodge, K. A., Crick, N. R. (1990). Social information-processing bases of aggressive behavior in children. Person Soc Psychol Bull 16, 8–22.
26. Dodge, K. A., Price, J. M., Bachorowski, J., Newman, J. P. (1990). Hostile attributional biases in severely aggressive adolescents. J Abnorm Psychol 99, 385–392.
27. Asarnow, J. R., Callan, J. W. (1985). Boys with peer adjustment problems: social cognitive processes. J Consult Clin Psychol 53, 80–87.
28. Crick, N. R., Ladd, G. W. (1990). Children's perceptions of the outcomes of aggressive strategies: do the ends justify being mean? Dev Psychol 26, 612–620.
29. Dodge, K. A., McClaskey, C. L., Feldman, E. (1985). A situational approach to the assessment of social competence in children. J Consult Clin Psychol 53, 344–353.
30. Dodge, K. A., Lochman, J. E., Harnish, J. D., Bates, J. E., Pettit, G. S. (1997). Reactive and proactive aggression in school children and psychiatrically impaired chronically assaultive youth. J Abnorm Psychol 106, 37–51.
31. Cornell, D. G., Warren, J., Hawk, G., Stafford, E., Oram, G., Pine, D. (1996). Psychopathy in instrumental and reactive violent offenders. J Consult Clin Psychol 64, 783–790.
32. Dodge, K. A., Feldman, E. (1990). Issues in social cognition and sociometric status. In S. R. Asher, J. D. Coie (Eds.), Peer Rejection in Childhood: Origins, Consequences, and Intervention. New York: Cambridge University Press, pp 119–155.
33. Dodge, K. A., Lansford, J. E., Burks, V. S., Bates, J. E., Pettit, G. S., Fontaine, R., Price, J. M. (2001). Peer rejection and social information processing factors in the development of aggressive behavior problems in children. (Submitted).

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

34. Coie, J. D., Dodge, K. A., Kupersmidt, J. (1990). Peer group behavior and social status. In S. R. Asher, J. D. Coie (Eds.), Peer Rejection in Childhood. New York: Cambridge University Press, pp 17–59.
35. Bierman, K. L., Smoot, D. L., Aumiller, K. (1993). Characteristics of aggressive-rejected, aggressive (nonrejected), and rejected (nonaggressive) boys. Child Dev 64, 139–151.
36. Coie, J. D., Lochman, J. E., Terry, R., Hyman, C. (1992). Predicting early adolescent disorder from childhood aggression and peer rejection. J Consult Clin Psychol 60, 783–792.
37. Rabiner, D. L., Coie, J. D., Miller-Johnson, S., Lochman, J. E. (2001). Predicting the persistence vs. desistance of aggressive and non-aggressive offending. (Submitted).
38. Dishion, T. J., Patterson, G. R., Griesler, P. C. (1994). Peer adaptations in the development of antisocial behavior. In L. R. Huesmann (Ed.), Aggressive Behavior: Current Perspectives. New York: Plenum Press, pp 61–95.
39. Bagwell, C. L., Coie, J. D. (1999, April). The friendship relations of antisocial boys. Presented at the biennial meeting of the Society for Research in Child Development, Albuquerque, NM.
40. Guerra, N. G., Huesmann, L. R., Tolan, P. H., Van Acker, R., Eron, L. D. (1995). Stressful events and individual beliefs as correlates of economic disadvantage and aggression among urban children. J Consult Clin Psychol 63, 518–528.
41. McLoyd, V. C. (1990). The impact of economic hardship on black families and children: Psychological distress, parenting, and socioemotional development. Child Dev 61, 311–346.
42. Coie, J. D., Jacobs, M. R. (1993). The role of social context in the prevention of conduct disorder. Dev Psychopathol 5, 263–275.
43. Richters, J. E., Cicchetti, D. (1993). Mark Twain meets DSM-III-R: conduct disorder, development, and the concept of harmful dysfunction. Dev Psychopathol 5, 5–29.
44. Bushman, B. J., Anderson, C. A. (2001). Media violence and the American public: scientific facts versus media misinformation. Am Psychol 56, 477–489.
45. Joint statement on the impact of entertainment violence on children: Congressional Public Health Summit. (2000, July 26). http://www.senate.gov/brownback/violence1.pdf
46. Anderson, C. A., Bushman, B. J. (2001). Effects of violent video games on aggressive behavior, aggressive cognition, aggressive affect, physiological arousal, and prosocial behavior: a meta-analytic review of the scientific literature. Psychol Sci 12, 353–359.
47. Huesmann, L. R., Eron, L. D., Berkowitz, L., Chafee, S. (1991). The effects of television violence on aggression: a reply to a skeptic. In P. Suedfeld, P. Tetlock (Eds.), Psychology and Social Policy. New York: Hemisphere, pp 192-200.
48. Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive function: constructing a unified theory of ADHD. Psychol Bull 121, 65–94.
49. Taylor, J., Iacono, W. G., McGue, M. (2000). Evidence for a genetic etiology of early-onset delinquency. J Abnorm Psychol 109, 634–643.
50. Nagin, D. S., Tremblay, R. E. (2001). Parental and early childhood predictors of

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persistent physical aggression in boys from kindergarten to high school. Arch Gen Psychiatry 58, 389–394.
51. Federal Bureau of Investigation. (2000). Crime in the United States: 2000 Uniform Crime Reports. Washington: Government Printing Office.
52. Cohen, P., Cohen, J., Kasen, S., Velez, C. N., Hartmark, C., Johnson, J., Rojas, M., Brook, J., Streuning, E. L. (1993). An epidemiological study of disorders in late childhood and adolescence. 1. Age and gender specific prevalence. J Child Psychol Psychiatry 34, 851–867.
53. Crick, N. R., Grotpeter, J. K. (1995). Relational aggression, gender, and social-psychological adjustment. Child Dev 66, 710–722.
54. Crick, N. R. (1995). Relational aggression: the role of intent attributions, feelings of distress, and provocation type. Dev Psychopathol 7, 313–322.
55. Sampson, R., Laub, J. (1993). Crime in the Making: Pathways and Turning Points Through Life. Cambridge, MA: Harvard University Press.
56. Rutter, M., Giller, H. (1983). Juvenile Delinquency. Harmondsworth, Middlesex, England: Penguin.
57. Loeber, R., Farrington, D. P. (2000). Young children who commit crime: epidemiology, developmental origins, risk factors, early interventions, and policy implications. Dev Psychopathol 12, 737–762.
58. McCord, J. (1997, April). Some unanticipated consequences of summer camps. Paper presented at the biennial meetings of the Society for Research in Child Development, Washington.
59. Coie, J. D., Miller-Johnson, S. (in press). Peer factors in early offending behavior. In R. Loeber, D. P. Farrington (Eds.), Child Delinquents. Thousand Oaks, CA: Sage.
60. Kazdin, A. E. (2000). Treatments for aggressive and antisocial children. Child Adolesc Psychiatr Clin North Am 9, 841–858.
61. Spivack, G., Shure, M. B. (1985). ICPS and beyond: Centripetal and centrifugal forces. Am J Commun Psychol 13, 226–243.
62. Spivack, G., Shure, M. B. (1974). Social Adjustment of Young Children: A Cognitive Approach to Solving Real-Life Problems. San Francisco: Jossey-Bass.
63. Conduct Problems Prevention Research Group. (1992). A developmental and clinical model for the prevention of conduct disorder: the FAST Track Program. Dev Psychopathol 4, 509–527.
64. Conduct Problems Prevention Research Group. (1999). Initial impact of the Fast Track prevention trial for conduct problems. I. The high-risk sample. J Consult Clin Psychol 67, 631–647.
65. Conduct Problems Prevention Research Group. (1999). Initial impact of the Fast Track prevention trial for conduct problems: II. Classroom effects. J Consult Clin Psychol 67, 648–657.
66. Greenberg, M. T., Kusche, C. A., Cook, E. T., Quamma, J. P. (1995). Promoting emotional competence in school-aged children: the effects of the PATHS curriculum. Dev Psychopathol 7, 117–136.

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4

Biopsychosocial Approaches to Aggression

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INTRODUCTION

Despite a century of published scientific research on human aggression, definitive answers about the causes and control of human aggressive behavior remain elusive. The ability of mental health professionals to predict which individuals will engage in violent behavior remains poor (1), and the relative stability of violence rates suggests that little progress has been made toward the prevention of aggressive acts. Indeed, data from the U.S. Department of Justice Bureau of Justice Statistics reveal that the homicide rates over the last hundred years have fluctuated from ~5 to 10 murders per 100,000, with peaks and troughs interspersed (2). These data, along with evidence from epidemiological studies showing that physical aggression in everyday relationships is a frequent occurrence (3), provide little reason for optimism that effective interventions for reducing violence in the community will be developed in the near future. On the positive side, over the

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past century a rich literature has accumulated that supports the notion that reliable correlates of aggressive behavior can be identified in the field and laboratory (4,5). For example, biological (e.g., central serotonin activity, endogenous testosterone activity, alcohol consumption), psychological (e.g., perceived threat, aversive mood states, frustration), and social (e.g., economic fluctuations, normative influences, political instability) factors have been associated with aggressive behavior. However, no single variable or theoretical model has been able to satisfactorily explain aggressive behavior in the general case. This has had the indirect benefit of allowing researchers to reach a consensus on what may be the guiding principle for aggression research in the next century; specifically, that the causes of aggressive behavior are complex and multi-factorial.

The idea that human behavior is determined by a complex interplay of biological, psychological, and contextual factors is hardly new or unique. Writers have long argued that psychiatry (6,7) and medicine (8,9) would benefit from a paradigm shift to so-called biopsychosocial frameworks in which the contribution of each of these factors is considered in a systematic and integrated manner. Interest in biopsychosocial models of human aggression is also not new (10–14). The obvious appeal of such models is that if biological, psychological, and social factors account only in part for aggression, examining how these factors operate in concert may provide a more complete understanding of this important and ubiquitous human behavior. In this chapter we discuss aggression from a biopsychosocial perspective. We begin by discussing biopsychosocial approaches in general. We then suggest reasons why the biopsychosocial perspective may be particularly germane to aggression research, and how knowledge about aggression could develop within a biopsychosocial framework. We conclude with some thoughts on biopsychosocial theories of aggression and clinical application.

WHAT IS "BIOPSYCHOSOCIAL"?

The term biopsychosocial has been used to describe a variety of behaviors displayed by clinicians and researchers. These include incorporation of ethical and pragmatic issues in clinical practice (15); examination of the relative contributions of biological, psychological, and social variables on specific behavioral outcomes (16); and a formal assessment approach in which all three domains are considered (17). The term biopsychosocial also represents a belief system in which human behavior is thought to occur in interlocking and interdependent systems ordered in a hierarchy of increasing complexity, from microsystems (e.g., molecular) to macrosystems (e.g., political structures). When these factors are integrated, the effect of the system on human functioning may be greater than the individual contributions of its component parts (7–9,18). Accordingly, the biopsychosocial practitioner considers the patient's psychological response to ill-

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ness and treatment, and the cultural, economic, and political context in which the illness occurs. These psychosocial factors are assumed to reciprocally affect biological functioning at the micro level, potentially altering the course of the illness.

Numerous articles advocating biopsychosocial medical practice have appeared in the past three decades. Unfortunately, biopsychosocial treatments have not challenged the preeminence of traditional psychiatry (15,19); that is, treatment for behavioral abnormalities involves either pharmacology (or other somatic intervention) or psychotherapy, or the simple combination of the two. Familial and cultural factors may be considered in treatment planning, but these considerations are usually secondary and not meaningfully integrated with traditional treatment choices.

Commentators also disagree about the usefulness of the biopsychosocial approach. Some have argued that multifactorial models reflect the maturity of a scientific discipline (9,16), and that ethical and humane practice requires biopsychosocial case conceptualizations (15). In contrast, others have noted that the term biopsychosocial may have lost much meaning through overuse and intellectualization (20), that seemingly complex phenomena may ultimately be accounted for by simple explanations as scientific knowledge advances (21), and that the drive to do biopsychosocial practice or research may atrophy as modestly efficacious somatic treatments are developed (22).

The causes and phenomenology of aggression have been investigated by workers with disparate professional backgrounds, including psychologists, sociologists, neuroscientists, biologists, anthropologists, and political scientists. The different conceptual and empirical paradigms used in these disciplines make it difficult to formulate comprehensive explanatory models or to develop common research methodologies. Although a rich literature on aggression exists within each field, interdisciplinary training opportunities for aggression researchers are limited, which may impede the development of biopsychosocial models of human aggression (16). However, as violence at all levels (from the personal to the international) continues to affect countless lives, aggression researchers must look toward more integrated paradigms to "interweave the complex tapestry of violence and its origins" (23).

WHY BIOPSYCHOSOCIAL CONCEPTUALIZATIONS OF AGGRESSION?

Although it seems intuitively correct to assume that aggression is multiply determined, the reasons for this assumption are seldom elucidated. Three reasons for adopting a biopsychosocial perspective are: 1) Aggressive behavior is by definition a complex psychosocial construct; 2) few human behaviors are caused by a

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single pathogen or pathway; and 3) multiple biological, psychological, and social variables have been associated with aggressive behavior. Following is a brief discussion of these issues.

Aggression comprises several complex psychological and social events, and thus requires a multivariate approach. The following definition includes the general elements that most researchers agree represent aggressive behavior (5,24):

> *Aggression is any form of behavior directed toward the goal of harming or injuring another human being who is motivated to avoid such treatment.*

Note that this definition assumes that aggression is an overt behavior that occurs in a complex psychosocial context. Nothing in this definition requires the behavior to be a physical act that results in bodily injury. Thus, a verbal tirade or property damage that results in psychological harm (e.g., intentionally breaking an antagonist's favorite coffee mug) can be an act of aggression. The harmful behavior must also be goal directed; that is, it must be intentional. Accidental harms, such as an unintended motor vehicle crash, are therefore not aggression. Motivational factors also play a role in aggression. Specifically, the target must be motivated to avoid injury in order to label a social interaction as aggression. Thus, dynamic psychological processes, including formation of intent in the aggressor and a specific motivational state in the target, are central to the notion of aggression. In sum, "aggression" is a rather broad psychosocial construct subsuming a wide range of behaviors, outcomes, and psychological processes operating in an integrated fashion. For these reasons, we believe that aggression can only be fully understood from a multifactorial perspective.

A second reason for viewing aggression as a biopsychosocial event is that few complex behaviors have a single identified cause. Examples of behavioral abnormalities that can be linked to a single pathogen or causeway include a subset of severe mental retardation associated with an inherited enzymatic deficiency (i.e., phenylketonuria), and some cases of psychoticlike behaviors caused by advanced treponema pallidum spirochete infection (parenchymatous neurosyphilis; general paresis). These behaviors, however, are best conceptualized as disease endpoints. Given that virtually all humans exhibit aggressive behavior at some point during the lifespan, and that otherwise healthy toddlers are capable of indulging in clearly aggressive acts, it seems that mild aggressive behaviors can hardly be construed as pathological. It would therefore be difficult to argue that aggression, in the general case, is a syndrome, disorder, or disease.

It is reasonable to ask, however, whether subtypes of extreme violence represent a disease endpoint or mental disorder that can be accounted for by a single pathogen or simple models. The current classification system of psychiatric disorders used in the United States, the DSM-IV-TR (25) includes a categorical

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subtype of aggressive behavior, Intermittent Explosive Disorder (IED). This disorder is characterized by an inability to resist "aggressive impulses," resulting in serious aggression or destruction of property (12:667, criterion A). The aggressive episodes must also be out of proportion to precipitating environmental stressors or provocation (criterion B). To assign this diagnosis, the aggressive episodes must not be better accounted for by another cooccurring mental disorder, intoxicating substance, or medical condition (criterion C).

To date, evidence supporting the validity of IED as a discrete entity is mixed at best, and the diagnostic criteria set has been substantially revised over time owing to these shortcomings (26). Moreover, even if IED does represent a syndrome of severe impulsive and aggressive behaviors, no pathogen or simple theoretical model can account for its development or expression. Indeed, the diagnostic criteria and features of IED described in the DSM-IV-TR imply that IED is a biopsychosocial phenomenon. For example, laboratory and physical findings for IED indicate that serotonergic neurotransmitter abnormalities and neurological "soft signs" may be present. These are not specific to IED, however. In addition, both psychological (e.g., failure to resist aggressive impulses) and social (e.g., presence of provocative stimuli or stressors) factors play a role in IED. Note that IED is not unique in the DSM, as virtually all disorders in this compendium have biological, psychological, and social aspects.

Although IED has a distinct biopsychosocial flavor, precisely how these biological, psychological, and contextual factors interact is not made clear in the DSM, and the constructs central to the IED diagnostic set are poorly defined and difficult to assess. For example, what is meant by "aggressive impulses," and how can these be observed or measured? Additionally, DSM-IV-TR does not provide guidance on how to determine when a response is out of proportion to a stressor or provocation. A final difficulty with the IED diagnosis is that topographically identical aggressive behaviors can occur in many different contexts, such as combat, prizefights, or self-defense. From the perspective of an observer (or the target of the aggression), extremely violent responses in these contexts could also appear impulsive or out of proportion. Thus, the social context in which the behavior occurs may be highly relevant for an IED diagnosis.

A third and final reason for viewing aggression as a multi-factorial phenomenon has to do with the sheer number of variables that have been implicated in its expression. Though many correlates of aggression have been identified, these associations are, on average, of modest magnitude (4). Unexplained variance in bivariate associations could be accounted for, at least in part, by measurement error. It is more likely, however, that small to moderate main effects reflect the presence of one or more moderator variables (27). Indeed, given the number of variables associated with aggression in the research literature, the additive variance would far exceed unity. Of course, common underlying processes could account for the relation between several variables and aggression. A more reason-

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able explanation, however, is that some of these variables serve as mediators or moderators.

There are at least two risks to using complex mediator or moderator models to explain aggression. The first is that highly complex models are likely to be less parsimonious and comprehensible. As an illustration, the reader may remember a time when challenged to interpret a four- or five-way interaction. Comprehending such relationships can be daunting. The second is that complex models that fit data may be sample specific and not generalize to other populations, settings, or times. Thus, complex models may not be any more useful for explaining or predicting aggression than simple models that use a few reliable correlates of aggression (e.g., male gender or alcohol intoxication). Thus, even if aggression as it occurs in the "real world" is a biopsychosocial phenomenon, it is reasonable to argue that a measurable improvement in prediction must be present for a multifactorial model to be useful. Despite these risks, general models of how knowledge develops in a research area support the notion that the biopsychosocial perspective may be the logical next step in understanding aggression. We therefore discuss development of knowledge bases next.

Biopsychosocial Conceptualizations: Development of Knowledge Bases

Theorists have proposed that biopsychosocial knowledge develops systematically and follows a predictable course (9,16). In behavioral medicine, a field that is, by nature, dependent on biopsychosocial models, clinical knowledge is thought to progress in four stages representing increasingly complex ways of thinking about specific disorders (9). In the initial stage, observed clusters of signs or symptoms lead to the formation of patient categories that discriminate category members from nonmembers. The second stage is characterized by examination of simple relationships among variables, collection of data to support cause-and-effect relationships among these variables, and identification of variables that may moderate the relationships. In the third stage, more complex moderating effects are identified that may attenuate or reverse the effects of the pathogens, and competing explanations for a disorder are tested against each other. As explanatory models become more complex, the behavior of investigators becomes important in how knowledge develops. For example, they might develop simpler models that are subsets of the complex model, ignore data that do not fit with specified models, abandon the research line due to its complexity, or synthesize competing data in a more comprehensive model. This last represents the fourth and final stage. In this last stage, multicategory, multicause, and multieffect conceptual models are developed and tested with the goal of identifying the relative importance of different variables in the model system.

A similar stage model for the accrual of scientific knowledge on human

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aggression has been proposed (16), with knowledge developing in a relatively predictable manner in three stages. The first stage of knowledge accrual begins when anecdotal reports link a variable to aggressive behavior. Following identification of potential risk factors, surveys are conducted and archival records examined, often using samples of convenience. Experimental and epidemiological studies are rare at this stage. Data examination tends to be largely descriptive in nature and usually involves only a small set of variables examined in bivariate analyses. Attempts are made to rule out alternative explanations for observed associations in this stage.

Effect sizes reported at stage 1 tend to be large, and thus attractive, to potential investigators. If attempts at replication produce substantially lower effect sizes, theorists may explain this state of affairs by nominating moderating variables. If consistent, albeit imperfect, main effects are found, knowledge accrual transitions to stage 2. At this point journals and professional societies in the area proliferate, and funding agencies devote increasing resources to research efforts. When sufficient replications have occurred, the literature is summarized in qualitative reviews, followed by meta-analyses. Experimental, epidemiological, and longitudinal investigations are conducted to determine whether the variable is causally related to aggression. The systematic exploration of potential moderating and mediating variables marks the end of this stage, along with attempts at theory building to guide future research efforts. By this time, both researchers and laypeople believe that a variable (e.g., alcohol intoxication or childhood abuse) is a risk factor for human aggression.

During the third and final stage, the variable is placed in a larger context. This involves understanding the relative importance of each variable and pathway, and how these change across time, populations, and environments. Paradoxically, as the number of variables called into play to explain a behavior expands, developing accurate, parsimonious, and comprehensible models may become more difficult. Opportunities to test the accuracy of complex models depend on perceived social needs, researcher training, guild biases, and cultural mores. Thus, macro social and psychological variables in the system that seem tangential to the process in stages 1 and 2 may determine whether variables deemed important continue to play a role in conceptualizations of aggression. To date, aggression research generally occurs at stages 1 and 2.

A critic may argue that these stage models of knowledge development are artificial conveniences with little or no empirical support. Although such models may have heuristic value and seem intuitively correct, it is not known whether the proposed trajectories represent the true state of the world (16). Indeed, research on how knowledge accrues in psychology and medicine consists primarily of historical narratives rather than quantitative analyses. Accordingly, research on how scientific knowledge develops may lead to a better understanding of how social factors influence biopsychosocial model development at various stages. Use of

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such meta-models may minimize the likelihood that a potentially fruitful line of inquiry will be abandoned because of individual biases and group pressures associated with the research process.

BIOPSYCHOSOCIAL THEORIES OF AGGRESSION

The research literature on single variable explanations for aggression is extensive, and many standard textbooks provide an overview of the field (5,24). Basic research examining how variables combine in the expression of aggression is less extensive, but not without interest. For example, domestic violence (28), alcohol-related violence (11), the developmental course of violent criminal offending (29) and homicide (30), evolutionary influences on the expression of aggression (12), and neurotransmitter correlates of aggression (31) have all been examined from a biopsychosocial perspective. To date, no comprehensive theory for aggression in the general case has emerged from these research efforts, for at least two reasons. First, the relationships examined to date involve a modest number of variables in relatively simple models. Second, variables and pathways selected for study are strongly influenced by individual research interests, training experiences, and guild associations.

Perhaps the earliest biopsychosocial models related to aggressive behavior were those that focused on antisocial personality disorder (ASPD) or psychopathy. In general, these models were concerned with the combined effects of physiological arousal, personality traits, and learning environment on the expression of ASPD behaviors (32,33). These theories were beneficial in that they generated interest in more integrated conceptualizations. However, they have also been criticized as being overly simplistic, and as not being strongly supported by the empirical literature (13). More recent biopsychosocial theories of ASPD have focused on developmental factors. Buikhuisen (34), for example, posited that antisocial behavior results from improper socialization when appropriate avoidance behavior is not acquired through operant conditioning. This deficit can be due to biological, cognitive, or psychological characteristics of the individual, or to pathological environmental factors that impede the acquisition of avoidance behavior. Moffitt's developmental theory of antisocial behavior also stresses the interplay of biological (particularly neurological), psychological, and environmental factors. Her theory differs, however, in its focus on behavioral and information-processing deficits that impede the acquisition and expression of prosocial behaviors (35).

Although studies testing contemporary biopsychosocial models of antisocial behavior exist (13,29), these theories were not developed to address aggressive behavior specifically. Aggressive acts are common in antisocial individuals, but aggression and antisocial behavior represent separate but related constructs. Most people engage in aggressive acts during their lifetime, but few meet the

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criteria for antisocial personality disorder or psychopathy. In addition, ASPD is characterized by a variety of other behaviors that reflect a general propensity to violate the rights of others, and can include nonviolent criminal acts, deceitfulness, impulsiveness, and lack of remorse. Indeed, it is possible to assign an ASPD diagnosis even if the individual does not have a marked history of aggressive behavior (25). Accordingly, contemporary researchers have begun to develop biopsychosocial models specific to aggressive behavior.

The alcohol literature provides a good example of the growing interest in biopsychosocial approaches to understanding aggression. A relation between alcohol intoxication and violence is one of the most replicated findings in the aggression literature, but theorists and researchers have long realized that multiple biological, psychological, and contextual variables increase or decrease the probability of alcohol-related violence occurring (11,36,37). One representative biopsychosocial model of alcohol-related aggression incorporates developmental (e.g., executive functioning, parental abuse), psychological (e.g., mood, alcohol-aggression expectancies), and contextual (e.g., setting, provocation) influences (11). This model has begun to generate more complex research questions, including the hypothesis that the link between acute alcohol intoxication and aggression may be mediated or moderated by prefrontal cortical executive cognitive functioning (ECF), and empirical studies to test these predictions are under way (38). ECF subsumes many of the cognitive abilities that have been implicated in the alcohol-aggression link in the past (e.g., planning, abstract reasoning, perspective taking, etc.). With respect to mediation, alcohol intoxication may compromise executive-level information processing, which, in turn, affects self-regulation of aggressive behavior. The moderation effect assumes that low-executive cognitive functioning (i.e., impulsive) individuals are more prone to the aggression-eliciting effects of alcohol when compared to their high executive-cognitive functioning counterparts. Although intriguing, the accuracy of the ECF model has not yet been conclusively established.

Biopsychosocial approaches have also been used to guide the development of multifactorial models of domestic violence (28,39). For example, White and Kowalski (39) posit a multi-layered developmental model of male-to-female violence. In this model, historical and sociocultural factors serve as distal influences, with the interaction among the perpetrator, victim, and situation as proximal factors. Biological status, genetic makeup, psychological characteristics, and environmental experiences of both the aggressor and victim can all influence the expression of aggression from this perspective. Unfortunately, White and Kowalski do not make specific predictions about how these factors interact, but suggest that such models can guide future theory development and research. McKenry and colleagues investigated the separate and combined roles of biological (testosterone, alcohol use, serotonin status), psychological (psychiatric symptoms), and social variables (family income, social stress, social support, relationship quality)

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on men's ($N = 105$) violence toward a female partner (28). Violence was assessed using a standard interview procedure with both members of the dyad. Hypotheses were generated concerning potential interaction effects (e.g., men with high levels of testosterone who used alcohol were expected to be at greater risk for aggression). Multivariate analysis revealed main effects for alcohol, relationship quality, and family income, but no significant interactions emerged to support a biopsychosocial model. The authors accounted for the lack of interaction effects by their relatively small sample and restricted range for some variables. Thus, testing hypotheses derived from biopsychosocial models requires particular attention to methodological issues, such as sample and instrument selection, and design parameters related to statistical power.

Accepting the notion that aggression is a multidetermined behavior does not, of course, guarantee that efforts to explain and control aggressive behavior will be advanced. First, as noted above, few biopsychosocial theories of human aggression are sufficiently developed to allow relevant hypotheses to be derived and tested. Second, even a casual observer of the field would note that most contemporary aggression research focuses on variables from a single domain. That is, even if researchers and clinicians believe that aggression is multidetermined, most behave as if aggression has relatively simple causes. This inconsistency is reflected in the aggression treatment literature.

BIOPSYCHOSOCIAL TREATMENT RESEARCH

Research on the treatment of aggression from a biopsychosocial perspective is virtually nonexistent. The aggression treatment outcome literature consists of studies examining specific medications or psychosocial treatment packages (the reader is referred to the chapters in this volume devoted to treatment for an overview). One could argue that the most basic biopsychosocial treatment for aggressive behavior would be to combine the best available psychopharmacological and psychological interventions. To our knowledge, however, no published clinical trial has examined the separate and combined effects of psychosocial and psychopharmacologic treatments for aggression. This does not mean that there is no potential benefit to combining treatments for aggressive behavior. Over the past several decades, clinical researchers have examined the efficacy of separate and combined treatments for a variety of psychiatric disorders, and authors of recent literature reviews have concluded that combined treatments may indeed provide benefits over and above those seen with single treatments (19,40). For example, combining behavior therapy and selective serotonin reuptake inhibitors (SSRIs) may produce greater short-term reduction in panic disorder symptoms as compared to psychotherapy or pharmacotherapy alone (41,42). Research also indicates that combination therapy may be the most effective mode of treatment for patients with chronic major depression (43), and may have the added benefit

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of increasing treatment compliance and acceptability (44). Combined treatments, however, are not always more effective, and may be inferior to individual treatments (19).

Although of practical clinical importance, the trend toward comparing single and combined treatments may have a subtle but significant negative effect on the development of biopsychosocial treatments in general. Specifically, use of combined psychosocial and psychopharmacologic treatments in the context of a traditional provider-patient relationship is, at best, only a rudimentary form of biopsychosocial practice. The principal rationale for combined treatments is that providing both psychopharmacology and psychotherapy will have an additive effect, which is almost never the case. Moreover, if clinicians assume that combined treatments are the sine qua non of biopsychosocial treatment, there will be little motivation for exploring and applying more complex biopsychosocial models posited by researchers and theorists. Given that biopsychosocial research is still in the formative stage, however, it is not surprising that clinical thinking relies on relatively simple treatment approaches.

The methods used to study both separate and combined treatments are derived largely from the traditional biomedical model. That is, the controlled randomized clinical trial (RCT) is the "gold standard" for both individual and combined efficacy studies. The RCT provides good internal validity, and has the potential for addressing such questions as the effects of sequential treatment, matching patient to therapist, portability of treatments, and the influence of culture on treatment delivery, outcome, and acceptability. Unfortunately, the RCT literature for psychiatric disorders is still in its infancy, and is characterized by medication comparisons to the best available treatment or comparison to a placebo control. Thus, psychiatric treatment outcome research, irrespective of disorder, is at a relatively early stage of knowledge development.

Results from RCTs, of course, cannot answer all questions relevant to biopsychosocial practice. Indeed, RCTs involve fairly constrained design parameters for patient selection and treatment delivery (to maximize internal validity). Critics of RCTs have therefore argued that results from clinical trials have little relevance to everyday practice. Effectiveness studies, in which clinical outcomes are examined in more naturalistic settings, may provide additional information about the cultural, psychological, economic, and social factors relevant to behavior change, and thus complement findings from RCTs.

A related problem for biopsychosocial practice is that combined treatments have been politicized to advance professional guild interests. For example, psychiatrists have argued that they are posed to be the ultimate biopsychosocial practitioners because their training curricula provide exposure to both psychopharmacology and psychotherapy training (20), and that having combined treatments administered by one provider may be more effective (19). Similarly, psychologists have argued that they are in the best position to offer biopsychosocial treat-

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ments, based on their extensive training in clinical research and psychosocial interventions. Some psychologists have therefore argued that they should receive expanded psychopharmacological training and associated prescription authority. Both arguments miss the mark. The ability to provide both psychotherapy and psychopharmacology does not ensure biopsychosocial practice. Humans are complex and dynamic biological and psychological organisms operating in a changing social environment (45). Although individual cases may benefit from psychotherapy or psychopharmacology, current psychological and psychiatric approaches are unlikely to produce sustained reductions in violence in the community.

CONCLUSIONS

Aggression is a multidetermined social behavior that is only modestly understood. Although biopsychosocial models of aggression have been proposed and tested, these have limited utility for explaining aggression in the general case. Research on the treatment of aggression lags behind basic research, and has relied largely on the traditional biomedical model for knowledge development and application. Although a full understanding of human aggressive behavior will almost certainly require biopsychosocial conceptualizations, researchers and clinicians behave, at this time, as if aggressive behavior were a one-dimensional phenomenon that can be accounted for by a small number of pathogens or pathways. Awareness and understanding of the social context surrounding knowledge development for aggression may help guide future research efforts and clinical practice.

REFERENCES

1. S Bjorkly. Prediction of aggression in psychiatric patients: a review of prospective prediction studies. Clin Psychol Rev 15:475–502, 1995.
2. JA Fox, MW Zawitz. Homicide trends in the United States. U.S. Department of Justice, Bureau of Justice Statistics http://www.ojp.usdoj.gov/bjs/homicide/homtrnd.htm, 2001.
3. L Magdol, TE Moffitt, A Caspi, DL Newman, J Fagan, P Silva. Gender differences in partner violence in a birth cohort of 21-year-olds: bridging the gap between clinical and epidemiological approaches. J Consult Clin Psychiatry 65:68–78, 1997.
4. CA Anderson, BJ Bushman. External validity of "trivial" experiments: the case of laboratory aggression. Rev Gen Psychol 1:19–41, 1997.
5. RA Baron, DR Richardson. Human Aggression, 2nd ed. New York: Plenum Press, 1994.
6. GM Abroms. Psychiatric serialism. Comp Psychiatry 22:372–378, 1981.
7. MF Reiser. Implications of a biopsychosocial model for research in psychiatry. Psychosom Med 42:141–151, 1980.

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

8. GL Engel. The clinical application of the biopsychosocial model. Am J Psychiatry 137:535–544, 1980.
9. GE Schwartz. Testing the biopsychosocial model: the ultimate challenge facing behavioral medicine? J Consult Clin Psychiatry 50:1040–1053, 1982.
10. M Berman. Biopsychosocial conceptualizations of aggression: the first thirty years. Clin Psychol Rev 17:585–588, 1997.
11. ST Chermack, PR Giancola. The relation between alcohol and aggression: an integrated biopsychosocial conceptualization. Clin Psychol Rev 17:621–649, 1997.
12. DM Buss, TK Shackelford. Human aggression in evolutionary psychological perspective. Clin Psychol Rev 17:605–619, 1997.
13. PA Brennan, A Raine. Biosocial bases of antisocial behavior: psychophysiological, neurological, and cognitive factors. Clin Psychol Rev 17:589–604, 1997.
14. PL Van den Berghe. Bringing beasts back in: Toward a biosocial theory of aggression. Am Sociol Rev 39:777–788, 1994.
15. JZ Sadler, YF Holgus. Clinical problem solving and the biopsychosocial model. Am J Psychiatry 149:1315–1323, 1992.
16. ME Berman. Accruing knowledge on the biological bases of aggression: comment on Giancola. Exp Clin Psychopharmacol 8:601–603, 2000.
17. FJ Huyse, JS Lyons, F Stiefel, J Slaets, P De Jonge, C Latour. Operationalizing the biopsychosocial model: the INTERMED. Psychosomatics 42:5–13, 2001.
18. GL Engel. The need for a new medical model: a challenge for biomedicine. Science 196:129–136, 1977.
19. GO Gabbard, J Kay. The fate of integrated treatment: whatever happened to the biopsychosocial psychiatrist? Am J Psychiatry 158:1956–1963, 2001.
20. PJ Fink. Response to the Presidential Address: is "biopsychosocial" the psychiatric shibboleth? Am J Psychiatry 145:1061–1067, 1988.
21. L Thomas. Biomedical science and human health. Yale J Biol Med 51:133–142, 1978.
22. L Hartmann. Presidential Address: reflection on humane values and biopsychosocial integration. Am J Psychiatry 149:1135-1141, 1992.
23. JS Grisolia, JL Lujan, J Sanmartin, S Grisolia. Preface: the Valencia statement on violence. In: JS Grisolia, J Sanmartin, JL Lujan, S Grisolia, eds. Violence: From Biology to Society. New York: Elsevier, 1997.
24. L Berkowitz. Aggression: Its Causes, Consequences, and Control. New York: McGraw-Hill, 1993.
25. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 4th ed, Text Revision. Washington: American Psychiatric Publishing, 2000.
26. EF Coccaro, RJ Kavoussi, ME Berman, JD Lish. Intermittent explosive disorder revised: development, reliability, and validity of research criteria. Comp Psychiatry 39:368–376, 1998.
27. RM Baron, DA Kenny. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. J Pers Soc Psychol 51:1173–1182, 1986
28. PC McKenry, TW Julian, SM Gavazzi. Toward a biopsychosocial model of domestic violence. J Marriage Fam 57:307–320, 1995.
29. A Raine, P Brennan, B Mednick, SA Mednick. High rates of violence, crime, aca-

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demic problems, and behavioral problems in males with both early neuromotor deficits and unstable family environments. Arch Gen Psychiatry 53:544–549, 1996.
30. DO Lewis, E Moy, LD Jackson, R Aaronson, N Restifo, S Serra, A Simos. Biopsychosocial characteristics of children who later murder: a prospective study. Am J Psychiatry 142:1161-1167.
31. ME Berman, JI Tracy, EF Coccaro. The serotonin hypothesis of aggression revisited. Clin Psychol Rev 17:651–665, 1997.
32. HJ Eysenck. Crime and Personality. London: Methuen, 1964.
33. SA Mednick, L Kirkegaard-Sorensen, B Hutchings, J Knop, R Rosenberg, F Schulsinger. An example of biosocial interaction research: the interplay of socioenvironmental and individual factors in the etiology of criminal behavior. In: SA Mednick, KO Christiansen, eds. Biological Bases of Criminal Behavior. New York: Gardner, 1977, pp 9–24.
34. W Buikhuisen. Chronic juvenile delinquency: a theory. In: W Buikhuisen, SA Mednick, eds. Explaining Criminal Behavior. Leiden, Netherlands: EJ Brill, 1988, pp 27–50.
35. TE Moffitt. Adolescent-limited and lifecourse-persistent antisocial behavior: a developmental taxonomy. Psychol Rev 100:674–701, 1993.
36. TA Ito, N Miller, VE Pollock. Alcohol and aggression: a meta-analysis on the moderating effects of inhibitory cues, triggering events, and self-focused attention. Psychol Bull 120:60–82, 1996.
37. BJ Bushman, HM Cooper. Effects of alcohol on human aggression: an integrative research review. Psychol Bull 107:341–354, 1990.
38. PR Giancola. Executive functioning: a conceptual framework for alcohol-related aggression. Exp Clin Psychol 8:576–597, 2000.
39. JW White, RM Kowalski. Male violence towards women: an integrated perspective. In: RG Geen, E Donnerstein, eds. Human Aggression: Theories, Research, and Implications for Social Policy. New York: Academic Press, 1998, pp 203–228.
40. ME Thase, JH Greenhouse, E Frank, CF Reynolds, PA Pilkonis, K Hurley, V Grochocinski, DJ Kupfer. Treatment of major depression with psychotherapy or psychotherapy-pharmacotherapy combinations. Arch Gen Psychiatry 54:1009-1015, 1997.
41. E De Beurs, AJLM van Balkom, A Lange, P Koele, R van Dyck. Treatment of panic disorder with agoraphobia: comparison of fluvoxamine, placebo, and psychological panic management combined with exposure and of exposure in vivo alone. Am J Psychiatry 152:683–691, 1995.
42. E De Beurs, AJLM van Balkom, R van Dyck, A Lange. Long-term outcome of pharmacological and psychological treatment for panic disorder with agoraphobia: a 2-year naturalistic follow-up. Acta Psychiatr Scand 99:59–67, 1999.
43. MB Keller, JP McCullough, DN Klein, B Arnow, DL Dunner, AJ Gelenberg, JC Markowitz, CB Nemeroff, JM Russell, ME Thase, MH Trivedi, J Zajecka A comparison of nefazodone, the cognitive behavioral-analysis system of psychotherapy, and their combination for the treatment of chronic depression. N Engl J Med 342: 1462–1470, 2000.
44. F De Jongle, S Kool, G van Aalst, J Dekker, J Peen. Combining psychotherapy and antidepressants in the treatment of depression. J Affect Disord 64:217–229, 2001.
45. AM Freedman. The biopsychosocial paradigm and the future of psychiatry. Comp Psychiatry 36:397–406, 1995.

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5

Impulsivity

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INTRODUCTION

Although impulsivity and aggression are frequently co-occurring elements of psychopathology, the relationship between these behavioral dimensions is not fully understood. In fact, the construct of impulsivity alone continues to remain somewhat ambiguous as many researchers continue to devise, and continually revise, often discrepant theoretical and etiological models of the trait. Reflecting this discontinuity, existing impulsivity instruments generally correlate poorly with one another, especially across modalities of assessment. While numerous research studies have offered some insight into the conceptualization of the construct, there are many disparate findings. A review of the theoretical models and research follows, with an emphasis on furthering the understanding of the construct and offering direction for future investigation. Following this review, research relevant to the relationship between the constructs of anger and impulsivity is presented.

THE CONSTRUCT OF IMPULSIVITY

Early on, psychodynamic theorists noted the components of speediness, pleasure attainment, and the absence of deliberation as characteristic of impulsivity. The

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development of impulse control was theorized to be dependent on memory, hierarchical delay mechanisms, and language development and production. According to this model, impulsive behaviors result from a mounting drive toward action behaviorally expressed in inappropriate settings (1,2). While the concept of "drives" has lost favor, the remaining elements originally noted by Frosch and Wortis (1) continue to echo in more recent conceptualizations of impulsivity.

Duffy (3) later modified the drive model and proposed that genetically determined preferences for arousal lead to variations in the degree of impulsivity expressed, with elevated arousal preferences and a tendency to act out impulses resulting in impulsive behaviors (3). Similarly, Eysenck (4) shifted the theory further from drives, focusing on personality. Eysenck conceptualized that differences in personality traits, including impulsivity, stem from variability in the inherited biological systems, such as arousal systems, predisposing the individual to certain temperaments (4). The mediating mechanism for impulsive behaviors, therefore, is the variability in biological systems related to arousal. Thus, Eysenck proposed that personality is genetically defined, with traits present or absent as a result of the inherited biological systems. Supporting evidence of a genetic component to the trait of impulsivity has been obtained (see Chap. 2) with genetic influence underlying impulsivity estimated as high as 45%. While Eysenck also noted that environment plays a key role in impulsivity, he theorized that environmental influences on impulsivity are exerted only when conditions are extreme. In contrast, Pedersen et al. (5), using twin studies, noted that twins reunited at a young age were likely to display greater similarity in impulsiveness than those who met only later in life or were never reunited. This finding suggests that environment plays a role in impulsivity, even when conditions are not particularly severe.

Like the psychoanalysts, Eysenck also addressed the notion of a lack of conformity to social norms and its relationship to impulsivity. In doing so, Eysenck suggested that impulsivity was related to Psychoticism, a construct that reflects a nonconforming, asocial, pathological tendency in one's character (7). Accordingly, the construct of impulsivity contains a dysfunctional element. Eysenck also observed a notable, although weaker, association with Extraversion, suggesting that impulsivity also reflects an outgoing, social orientation and a preference for novelty and change (8). In summary, Eysenck was the first to note both socially acceptable and unacceptable qualities of impulsivity.

Based on prior conceptualizations, impulsivity became viewed as linked to genetically determined brain functioning (arousal), while also influenced by the environment. The roles of various specific brain functions outside of arousal (i.e., memory, inhibitory mechanisms, and language development) were also noted as causal factors. Later, impulsivity became conceptualized as a largely dysfunctional personality trait (although also embodying a socially acceptable quality under some circumstances).

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Expanding upon arousal theories, Gray presented the Limbic System Theory (9), a general behavioral theory of learning and emotions related to personality development. Gray suggested that arousal, in conjunction with sensitivity to reward and punishment, plays a role in determining personality. Gray highlighted the "biology" of impulsivity, but cited new mechanisms as mediators of impulsivity. Specifically, Gray proposed the role of limbic system (in particular, the behavioral inhibition system, the behavioral activation system, and the fight/flight system) function in the modulation of behavior. Additionally, Gray suggested that the "reward system" sensitizes the impulsive individual to reinforcing stimuli while simultaneously desensitizing the individual to punishment. This sensitivity, teamed with a more reactive behavioral activation system, results in impulsive behaviors. While Gray also noted the impact of the environment on behaviors, Gray suggested that one chooses certain environments based on personality variables, rather than the environment acting on the individual in a detrimental or beneficial manner (see the discussion on gene-environment correlations in Chap. 2).

While Gray's theory has utility, it has also been criticized on two grounds. First, it does not account for the tendency of individuals to approach or withdrawal from novel stimuli (as it is based on previous learning experiences only). Second, opposing behavioral responses (such as inhibition/freezing and activation/running) are proposed to be mediated by two different systems, suggesting these opposing processes could theoretically co-occur simultaneously (10). Although the theory has been criticized, some aspects have also received support. Specifically, the desire for immediate reinforcement has received empiric support. For example, individuals with psychiatric diagnoses consistent with impulse control deficiencies tend to discount delayed rewards relative to those without impulsive-type diagnoses (11,12), and will respond immediately rather than wait for a larger reward. Notably, these individuals do not differ in regard to their perception of a low-probability vs. high-probability reward or in their performance on motor inhibition tasks. These data then offer support for Gray's theory of immediacy of responding and reinforcement. Finally, it has been shown that impulsive individuals are "socially fearless" and less concerned than others about the prospect of aversive encounters despite equal awareness of such potential consequences (13). This corroborates the notion of a diminished sensitivity to punishment. Thus, new evidence both clarifies and further complicates the model of impulsivity and the notion of a biologically preset arousal preference remained, with Gray adding to the model new contributing factors, including learning, emotions, and personality.

Cloninger echoed the idea that a diminished concern for punishment and a desire for stimulation are important elements of impulsivity. In Cloninger's model (14), two categories are used to depict impulsivity: (low) harm avoidance and (high) novelty seeking. Those who are impulsive tend to seek out novel or

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"interesting" stimuli and are unlikely to demonstrate a concern for possible negative consequences. As with previous theorists, Cloninger believed these personality dimensions were under substantial genetic influence.

As the scientific study of the brain and personality progressed, the focus further shifted to more distinct neuronal functioning and cognitive processes as the mechanisms of impulsivity. Daruna and Barnes (15) define impulsivity as the "manifestation of dynamic features of brain organization that determine a) neural representational capacity, dimensionality, and symmetry; b) the activation threshold of motor systems; and/or c) the temporal convergence of neural signals regulating motor outputs." Impulsivity, therefore, may be conceptualized to reflect a decreased capacity to consider several factors at one time or in close temporal proximity. Daruna and Barnes suggested that decreased ability in these areas may be due to a limited capacity for mental representations or low dimensionality (i.e., a single representation may require a large portion of the available capacity). Another possible mechanism may be the presence of numerous representations that surface randomly at the same time, which results in difficulties attending to the appropriate cues while processing information. Threshold factors have also been cited as a possible mechanism for impulsivity. Here, the ease with which certain representations can elicit motor acts determines the degree of impulsivity expressed. Finally, Daruna and Barnes cited timing factors, or the temporal overlap of signals from several representations that function to control actions, as possible causal mechanisms of impulsivity. The level of myelination of the axons, the precise point of synaptic contact at the postsynapse, or other characteristics of the transmitters (15) may be the means by which timing and threshold factors are regulated. Thus, according to Daruna and Barnes, impulsivity is mediated by deficient cognitive abilities stemming from impaired brain functioning or organization.

With regard to neurophysiology, decreased serotonergic activity has been implicated as related to impulsivity (16–18), with diminished sensitivity to 5-HT at the postsynaptic sites proposed as a possible causal factor (19). While the specificity of the chemical abnormality and the relationship of functional and dysfunctional impulsivity with chemical processes may have yet to be fully known or understood, there exists a preponderance of findings linking reduced central 5-HT activity metabolism to impulsivity, suggesting that 5-HT is functionally important in the expression of impulsive behaviors.

Although up to this point the impact of affect on impulsivity had been largely neglected, affect may play a role in modulating impulsivity as it interacts with cognitive processes and brain functioning. Evidence to support the notion of the critical influence of affect on the expression of impulsive behaviors is demonstrated by the observation that impulsive responding is related to the cognitive perception that one can positively alter one's mood by engaging in an impulsive activity (20). When led to believe that no improvement can be attained, there

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is a decreased tendency to engage in pleasurable activities, to seek immediate gratification, and to engage in frivolous procrastination. In other words, the individual is less likely to engage in impulsive behaviors when affectively discouraged, and perceives little hope of affect change.

Additionally, it has been suggested that emotional distress may shift priorities from long-term goals to more immediate desires when there is a sense that these more immediate goals will result in the desired mood changes. Unfortunately, because the long-term costs of reducing behavioral controls may outweigh and outlast the short-term affective relief, this pattern results in self-defeating behaviors, perhaps reflecting the common belief that impulsive responding tends to be dysfunctional. Often, deficient information processing is cited as the mechanism by which self-defeating impulsive responses are produced. In other words, it is suggested that there is a trade-off, with the individual responding immediately without consideration of all possible options or ramifications (incomplete processing) in order to obtain satisfaction, and therefore possibly making a poor choice.

With regard to impulsive responding, however, information processing is not *always* deficient. Dickman and Meyer (21) found that highly impulsive individuals are generally no quicker or more inaccurate than nonimpulsive individuals during the response-execution phase of information processing; i.e., impulsive individuals complete this particular phase of information processing as slowly and as accurately as others. Additionally, highly impulsive individuals are actually more accurate than low-impulsive individuals when they are required to process information extremely rapidly. Finally, those who are extremely reflective or extremely impulsive when successfully solving conventional problems tend to be poor solvers of nonconventional problems (22). Thus, a simple speed-accuracy trade-off model of impulsivity does not appear to be accurate.

Barratt (23) proposes a general systems theory of impulsivity that highlights a behavioral control system, which is the product of the combination of genetics, brain functioning, cognitions, behaviors, and the environment. These elements interact, altering one another and ultimately producing impulsive behaviors. Specifically, Barratt (24) notes lessened cortical arousal and diminished brainstem reticular activation among impulsive individuals, with deficiencies in the brain processes that regulate performance of timing and rhythm for cognitive and behavioral tasks, yielding variability of responding and visual tracking, decreased efficiency, and increased errors. Cognitively, impulsive individuals spend less time completing thought tasks (24) and perceive of times as passing unusually quickly (25,26). Temporal information processing is thought to play an instrumental role (27). The rate of cognitive processing preceding a response appears out of sync with the task demands (24), producing behaviors that appear somewhat ineffective and "impulsive." Thus, Barratt's theory combines much of the thinking from the past, adding new findings, to suggest that many of the above

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noted elements are of significance and work in concert to produce impulsive behaviors.

In contrast to the study of impulsivity per se, knowledge regarding the opposing process of inhibition can be of use to further understand impulsivity. In contrast to impulsiveness, the ability to inhibit behaviors necessitates the following interrelated processes: 1) the inhibition of an initial response likely to be reinforced; 2) the cessation of an ongoing response (thereby allowing time for a decision to respond to be made); and 3) the opportunity for self-directed responses to occur (28). Inhibition does not cause the executive, or self-directed, actions to occur. Rather, it *allows* these processes to occur by creating the delay. The development of internalized, self-directed speech, in part, functions to facilitate the process. Gratification is often deferred in aspiration of an eventual greater goal. Conflicts between immediate and later consequences "signal" the activation of executive functions, with the process of inhibition and self-regulation mediated by conditioned cues of punishment and/or prior socialization. Deficiencies in inhibition have been linked to the disruption of several executive functions that facilitate self-regulation and goal-directed persistence. Thus, the study of inhibition has closely paralleled the study of impulsivity, with a focus on the ability to inhibit responses and the capacity for cognitive processing and self-directed thoughts (language). It is clear that the model of impulsivity is complex, involving the interaction of a myriad of factors, from chemicals in the brain, to the ability to "inhibit" a response, to executive functioning of the brain and cognitive processes including self-directed speech and learning, to motor functioning, all of which are influenced by both genetics and the environment.

To date, theorists and researchers have exposed various facets of the construct of impulsivity, and have conceptualized it as a biologically determined preference for stimulation and arousal (4,14) combined with increased attention to pleasurable or reinforcing elements and decreased consideration of stimuli signaling the possibility of punishment (8,9), as well as diminished concern for planning and for future behaviors (8,29). This orientation generally results in maladaptive immediate gratification–serving behaviors. Impulsive responding tends to be variable, less efficient, and more error filled (24,30). This is not due solely to deficiencies in information processing, although cognitive processes do differ among impulsive individuals, most notably those with difficulty in attending to multiple cognitions and timing (15). On the other hand, available data also suggest that, at times, impulsivity may reflect quick and efficient processing of information and a carefree, rapid execution of behavior, which may be productive and free of negative consequence.

THE FACTORS OF IMPULSIVITY

Another clue to understanding impulsivity may be provided through the examination of the subfactors that have been identified and previously studied. While

there has been a large discrepancy in the number and nature of subfactors identified, several dimensions appear to have merit. The nature of impulsivity is complex and includes both adaptive and dysfunctional elements. Two factors termed functional and dysfunctional impulsivity have been identified and indicate that a decreased tendency to reflect prior to acting may be either beneficial or detrimental (31,32). Offering support of this distinction, statistical analyses reveal a relationship between Dysfunctional Impulsivity and the tendency to ignore facts when making decisions (33), the Psychotisicism scale on the Eysenck Personality Questionnaire, and traditional measures of impulsivity (32). Dysfunctional impulsivity is also negatively correlated with self-esteem and attitudes toward the family (32). In contrast, Functional Impulsivity is associated with orderliness and cognitive structure (two constructs that are inversely correlated with Dysfunctional Impulsivity) (31).

In addition to functional valence, numerous factor analyses have identified a dimension often referred to as Behavioral Impulsivity which is described as reflecting a lack of behavioral control, action on the spur of the moment, lack of perseverance, motor impulsiveness, and/or an inability to delay gratification (8,34–38). Behavioral impulsivity is associated with delinquency, aggression, and an undercontrolled and disinhibited approach to the world (38).

A factor dubbed Cognitive Impulsivity has been used to refer to inattention, cognitive instability, inability to switch between cognitive sets, and rapid decision making (36–38). Cognitive Impulsivity is associated with performance on measures of difficult cognitive tasks requiring mental control, the ability to shift between mental sets, and IQ (38).

Finally, many researches have cited the dispositional tendencies of spontaneity, living for the moment, and a lack of forethought and concern for future outcomes, plans, or consequences. These qualities are summed to reflect a factor designated Non-Planning Impulsivity (8,36,39,41,42).

The factors of impulsivity may also lead to the confusion regarding impulsivity since the factors are varied and reflect how "impulsivity" can be expressed in many different ways (e.g., appearing to be a cognitive process, a behavioral pattern, or a personality trait). It may also be functional or dysfunctional, depending on situational contingencies and contexts. At present, it appears that impulsivity may be best defined as a tendency to quickly process information when faced with the opportunity to obtain an instantaneous reward or a positive stimulus of some sort, leading to either a failure to inhibit behavior or a tendency to engage in an action, which is ultimately functional or dysfunctional, depending on task demands and situational contexts.

IMPULSIVITY AND AGGRESSION

As noted, impulsivity is related to aggression, antisocial behaviors, and delinquency. Impulsivity may be of an aggressive or a nonaggressive nature, just as

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aggression can be impulsive or premeditated so as to gain some sort of benefit. The relationship between behavioral impulsivity and delinquency is strong, suggesting that children who are undercontrolled may engage in physical fights on the spur of the moment if the rewards appear to be large and the negative consequences appear small and distant. Further illustrating the significance of the relationship between these two constructs, impulsivity strongly differentiates stable, serious delinquents from other delinquents in early adolescence (43,44). Boys with difficulties with impulsivity, hyperactivity, and attention and concurrent conduct disorder problems most closely resemble psychopathic adults (44). They are disinhibited, choose to delay gratification less frequently, and often display neuropsychological impairments (44). Thus, Behavioral Impulsivity, in particular, is of noteworthy significance to aggression. Non-Planning Impulsivity also demonstrates a strong association with both serious/stable and increasing delinquency (38,40). Cognitive Impulsivity has also shown some relationship, although only to a mild degree (40). Barratt (24,25) found Behavioral/Motor and Cognitive components of impulsivity to distinguish between antisocial and normal subjects. Thus, all elements of impulsivity appear to be related to aggression, although there is greater agreement on the Behavioral and Non-Planning aspects.

Chemical processes within the brain and body influence both aggression and impulsivity, with evidence suggesting that diminished central serotonergic functioning plays a role in both of these behavioral difficulties (45,46). Testosterone is also known to play a role in both aggression and impulsivity. Among women, a positive relationship between testosterone and impulsivity has been identified, as measured by a continuous performance task (with a distracter) (47). In contrast, measures of attention (correct detections) are not related to testosterone levels. Thus, testosterone appears to be more specifically related to impulsive responding and not mere difficulties in attending. Executive cognitive functions, which are more fully engaged during the distracter CPT, guide goal-directed behaviors and are associated with impulsive responding and increased testosterone levels. Testosterone may be related to the ability to restrain motor behavior under conditions where selective attention and working memory are required for rapid, accurate decision making. Thus, testosterone may correlate with both impulsive and aggressive acts due to a specific relationship of higher-order cognitive processes and the inhibition of behaviors. When individuals demonstrate difficulties with both aggression and impulsivity, the cognitive deficits are compounded. Barratt and coworkers (48) found differences on neuropsychological and cognitive psychophysiological measures of information processing between inmates who are impulsive and aggressive from those who are aggressive only. Thus, while cognitive deficits are associated with both types of behavioral difficulties, when both problems are simultaneously present, these deficits are worse. The cognitive deficits are linked to chemical functioning, including, at a minimum, both testosterone and serotonin.

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The specific cognitive errors of impulsive and aggressive individuals are of interest. Among aggressive and nonaggressive female parolees, those who had committed violent crimes demonstrated an inability to select a response strategy that yields the greatest financial gains despite an awareness that the strategy employed was not optimal. Thus, they are able to correctly process the stimuli in order to determine the optimal response strategy, but are unable to employ it. The female parolees generally cited an inability to tolerate the long delay associated with the preferred response strategy as the reason for the poor response choices (49). Thus, an impulsive response style is implemented by those who are aggressive. In contrast, nonviolent offending females were more likely to select a less impulsive response strategy, utilizing reward amount as the more important decision-making factor. Thus, impulsivity and aggression are again linked with regard to processing.

Another study demonstrated that individuals with Borderline Personality Disorder respond to the experience of financial loss in laboratory tasks with greater aggressive responses than controls (11), suggesting a possible link among affect, impulsivity, and aggression. In other words, when provoked by frustration, the impulsive individual is more likely to behave in an aggressive manner. These individuals also displayed higher Buss-Durkee Hostility Inventory scores, Brown-Goodwin Life History of Aggression scores, and Retrospective Overt Aggression Scale scores than controls. Berkowitz (50) suggests that all forms of negative affect contribute to an increase in aggression, with aggressive acts occurring as the result of a loss of self-control while under emotional distress (i.e., affect regulation overrides impulse control in favor of improving one's mood).

Although there is a strong relationship between aggression and impulsivity, there is also evidence that irritability and impulsivity are more closely genetically and phenotypically related than impulsivity and direct assault, with verbal and indirect assault demonstrating a moderate relationship with impulsivity (51). Physical and irritable impulsive aggression may represent distinct categories (51), with impulsivity less etiologically related to physically aggressive behaviors. Seronczynski et al. (51) suggest that this finding may be an artifact of the measure used, but also caution that more examination is needed with regard to those who display both impulsive and aggressive behaviors.

Finally, the environment exerts an influence and sometimes is a mediating factor regarding the relationship between aggression and impulsivity. For example, the effects of impulsivity on the delinquent behaviors of juveniles are greater in poorer neighborhoods (52); conversely, impulsivity has little effect on the delinquent behaviors of boys in more affluent settings. The effects of the context, however, appear to be small relative to other factors.

Despite the lack of complete knowledge regarding the complex interaction of impulsive and aggressive tendencies, treatment is effective. A meta-analysis of 23 studies that focused on cognitive behavior modification utilized with

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school-aged children produced a mean effect size of 0.74, with 89% of the studies showing treatment participants experiencing greater gains than controls subjects at both posttest and maintenance (53). The cognitive behavioral modification treatment reduced the occurrence of hyperactive-impulsive and aggressive behaviors, enabling the students to control their behaviors.

CONCLUSIONS

There remains much to discover about the constructs of aggression and impulsivity, when considered together as well as individually. A complex interaction of higher order executive functions linked to chemical processes in the brain as well as basic behavioral inhibitory processes should be further explored as they appear to play a critical role in both impulsivity and aggression. Additionally, the nature of the subfactors of impulsivity and types of aggressive responding should be further explored. While these two constructs are distinct, they are intimately related. Without a clear understanding of the constructs themselves, an investigation of their relationship is hampered. The role of affect and its relationship to both constructs also beckons further investigation. Identification of the biological or procedural deficits that produce impulsive and/or aggressive responding is necessary to further understand the constructs as well as to facilitate the treatment of these difficulties.

REFERENCES

1. Frosch, J., Wortis, S. B. (1954). A contribution to the nosology of the impulse disorders. Am J Psychiatry 111, 132-137.
2. Monroe, R. R. (1970). Episodic Behavioral Disorders: A Psychodynamic and Neurophysiologic analysis. Cambridge, MA: Harvard University Press.
3. Duffy, E. (1957). The psychological significance of the concept of "arousal" or "activation." Psychol Rev 64, 265–275.
4. Eysenck, H. J. (1983). A biometrical genetical analyses of impulsive and sensation-seeking behavior. In M. Zuckerman (Ed.), Biological Bases of Sensation Seeking, Impulsivity, and Anxiety. Hillsdale, NJ: Lawrence Erlbaum.
5. Pedersen, N. L., Plomin, R., McClearn, G. E., Friberg, L. (1988). Neuroticism, extraversion, and related traits in adult twins reared apart and reared together. J Pers Soc Psychol 55, 950–957.
6. Deleted in proofs.
7. Eysenck, S. B. G., Eysenck, H. J. (1978). Impulsiveness and venturesomeness: their position in a dimensional system of personality description. Psychol Rep 43, 1247–1255.
8. Eysenck, S. B. G., Eysenck, H. J. (1977). The place of impulsiveness in a dimensional system of personality description. Br J Soc Clin Psychol 16, 57–68.

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

9. Gray, J. A. (1987). Perspectives on anxiety and impulsivity: a commentary. J Res Pers 21, 493–509.
10. Zuckerman, M. (1979). Sensation Seeking: Beyond the Optimal Level of Arousal. Hillsdale, NJ: Lawrence Erlbaum.
11. Dougherty, D. M., Bjork, J. M., Huckabee, H. C. G., Moeller, F. G., Swann, A. C. (1999). Laboratory measures of aggression and impulsivity in women with borderline personality disorder. Psychiatry Res 85, 315–326.
12. Crean, J. P., de Wit, H., Richards, J. B. (2000). Reward discounting as a measure of impulsive behavior in a psychiatric outpatient population. Exp Clin Psychopharmacol 8, 155–162.
13. Wolfe, R. N., Kasmer, J. A. (1988). Type versus trait: extroversion, impulsivity, sociability, and preferences for cooperative and competitive activities. J Pers Soc Psychol 54, 864–871.
14. Cloninger, R. (1987). A systematic methods for clinical description and classification of personality variants. Arch Gen Psychiatry 44, 573–588.
15. Daruna, J. H., Barnes, P. A. (1993). A neurodevelopmental view of impulsivity. In W. G. McCown, J. L. Johnson, M. B. Shure (Eds.), The Impulsive Client: Theory, Research, and Treatment. Washington: American Psychological Association.
16. Stein, D. J. (1996). Cognitive science models of compulsivity and impulsivity. In J.M. Oldham, E. Hollander, A. E. Skodol (Eds.), Impulsivity and Compulsivity. Washington: American Psychiatric Press.
17. Hollander, E., Stein, D. J., DeCaria, C. M., Cohen, L., Islam, M., Frenkel, M. (1992). Neurobiology of obsessive compulsive disorders. Clin Neuropharmacol 15(Suppl 1), 259A-260A.
18. Linnoila, M., Virrkunen, M., Scheinin, M., Nuutila, A., Rimon, R., Goodwin, F. K. (1983). Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from nonimpulsive violent behavior. Life Sci 33, 2609–2914.
19. Coccaro, E. F., Siever, G. S., Siever, L. J. (1990). Buspirone challenge: Preliminary evidence for a role for 5-HT-1 a receptors in impulsive aggressive behavior in humans. Psychopharm Bull 26, 393–405.
20. Tice, D. M., Bratslavsky, E., Baumeister, R. F. (2001). Emotional distress regulation takes precedence over impulse control: if you feel bad, do it! J Pers Soc Psychol 80, 53-67.
21. Dickman, S. J., Meyer, D. E. (1988). Impulsivity and speed accuracy tradeoffs in information processing. J Pers Soc Psychol 54, 274–290.
22. Duemler, D., Mayer, R. E. (1988). Hidden costs of reflectiveness: aspects of successful scientific reasoning. J Educ Psychol 80, 419–423.
23. Barratt, E. S. (1991). Measuring and predicting aggression within the context of a personality theory. J Neuropsychiatry 3, S35-S39.
24. Barratt, E. S. (1985). Impulsiveness subtraits: arousal and information processing. In J. T. Spence, C.E. Izard (Eds.), Motivation, Emotion, and Personality. Amsterdam: Elsevier Science, pp 137–146.
25. Bradshaw, C. M., Szabadi, F. (1992). Choice between delayed reinforcers in a discrete-trials schedule: the effect of deprivation level. Q J Exp Psychol 44B, 1–16.

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26. Van den Broek, M. D., Bradshaw, C. M., Szabadi, E. (1992). Performance of impulsive and non-compulsive subjects on two temporal differentiation tasks. Pers Individ Dif 13, 169-174.
27. Stanford, M. S., Barratt, E. S. (1994). Verbal skills, finger tapping, and cognitive tempo define a second order factor of temporal information processing. Brain Cogn 31, 35–45.
28. Barclay, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. Psychol Bull 121, 65–94.
29. Barratt, E. S. (1965). Factor analysis of some psychometric measures of impulsiveness and anxiety. Psychol Rep 16, 547-554.
30. Lawrence, J. B., Stanford, M. S. (1999). Impulsivity and time of day: effects on performance and cognitive tempo. Pers Individ Dif 256, 199–207.
31. Dickman, S. J. (1990). Functional and dysfunctional impulsivity: personality and cognitive correlates. J Pers Soc Psychol 58, 95–102.
32. Heaven, P. C. L. (1991). Personality correlates of functional and dysfunctional impulsiveness. Pers Individ Dif 12, 1213–1217.
33. Dickman, S. J. (1996). Adverse (and beneficial) consequences of impulsive behavior. In R. S. Feldman (Ed.), The Psychology of Adversity. Amherst: University of Massachusetts Press, pp 199–216.
34. Buss, A. H., Plomin, R. (1975). A Temperamental Theory of Personality Development. New York: Wiley-Interscience.
35. Malle, B. F., Neubauer, A. C. (1991). Impulsivity, reflection, and questionnaire response latencies: no evidence for a broad impulsivity trait. Pers Individ Dif 12, 865–871.
36. Patton, J. H., Stanford, M. S., Barratt, E. S. (1995). Factor structure of the Barratt impulsiveness scale. J Clin Psychol 51, 768–774.
37. Schalling, D., Edman, G., Asberg, M. (1983). Impulsive cognitive style and inability to tolerate boredom: psychobiological studies of temperamental vulnerability. In M. Zuckerman (Ed.), Biological Bases of Sensation-Seeking, Impulsivity, and Anxiety. Hillsdale, NJ: Lawrence Erlbaum.
38. White, J. L., Moffit, T. E., Caspi, A., Bartusch, D. J., Needles, D. J., Stouthamer-Loeber, M. (1994). Measuring impulsivity and examining its relationship to delinquency. J Abnorm Psychol 103, 192–205.
39. Barratt, E. S. (1994). Impulsiveness and aggression. In J. Monoham, H. J. Steadman (Eds.), Violence and Mental Disorders: Developments in Risk Assessment. Chicago: University of Chicago Press, pp 61–79.
40. Luengo, M. A., Carrillo-de-la-Pena, M. T., Otero, J. M., Romero, E. (1994). A short-term longitudinal study of impulsivity and antisocial behavior. J Pers Soc Psychol 66, 542–548.
41. Luengo, M. A., Carrillo-de-la-Pena, M.T., Otero, J. M. (1991). The components of impulsiveness: a comparison of the I.7 Impulsiveness questionnaire and the Barratt Impulsiveness Scale. Pers Individ Dif 12, 657–667.
42. Parker, J. D. A., Bagby, R. M., Webster, C. D. (1993). Domains of the impulsivity construct: a factor analytic investigation. Pers Individ Dif 15, 267–274.
43. Mischel, W., Shoda, Y., Rodriguez, M. L. (1989). Delay of gratification in children. Science 244, 933–938.

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44. Lynam, D. R. (1998). Early identification of the fledgling psychopath: locating the psychopathic child in the current nomenclature. J Abnorm Psychol 107, 566–575.
45. Barratt, E. S. (1987). Impulsiveness and anxiety: information processing and electroencephalograph topology. J Res Pers 21, 453–463.
46. Manuck, S. B., Flory, J. D., McCaffery, J. M., Matthews, K. A., Mann, J. J., Muldoon, M. F. (1998). Aggression, impulsivity, and central nervous system serotonergic responsivity in a nonpatient sample. Neuropsychopharmacology 19, 287–299.
47. Bjork, J. M., Moeller, G., Dougherty, D.M., Swann, A. C. (2001). Endogenous plasma testosterone levels and commission errors in women: a preliminary report. Physiol Behav 73, 217–221.
48. Barratt, E. S., Stanford, M. S., Kent, T. A., Felthous, A. (1997). Neuropsychological and cognitive psychophysiological substrates of impulsive aggression. Biol Psychiatry 41, 1045–1061.
49. Cherek, D. R., Lane, S. D. (1999). Laboratory and psychometric measurements of impulsivity among violent and nonviolent female parolees. Biol Psychiatry 46, 273–280.
50. Berkowitz, L. (1989). Frustration-aggression hypothesis examination and reformation. Psychol Bull 106, 59–73.
51. Seroczynski, A. D., Bergeman, C. S., Coccaro, E. F. (1999). Etiology of the impulsivity/aggression relationship: genes or environment? Psychiatry Res 86, 41–57.
52. Lynam, D. R., Caspi, A., Moffit, T. E., Wikstrom, P. H., Loeber, R., Novak, S. (2000). The interaction between impulsivity and neighborhood context on offending: the effects of impulsivity are stronger in poorer neighborhoods. J Abnorm Psychol 109, 563–574.
53. Robinson, T. R., Smith, S. W., Miller, M. D., Brownell, M. T. (1999). Cognitive behavioral modification of hyperactivity-impulsivity and aggression: a meta-analysis of school-based studies. J Educ Psychol 91, 195–203.

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6

Anger Disorders

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INTRODUCTION

Anger is a frequently experienced emotion for most people (1). When mild to moderate in intensity and expressed in constructive, nonhostile ways, anger can lead to positive, adaptive behaviors such as expressing feelings; asserting one's rights, thoughts, and feelings; problem solving; redressing concerns; setting appropriate limits on the behavior of others; and motivating effective behavior (1,2). Anger, however, is a double-edged sword, often leading to many negative outcomes. For example, chronic anger and hostility are associated with health problems such as coronary artery disease, hypertension, compromised immune functioning, dental problems, and overall mortality (3). Intense anger is often associated with marital discord, abusive parenting, intimate partner violence, and other relationship problems (4,5). Anger has also been implicated in school violence, bullying, and disrupted teen relations. Anger may also lead to major or minor property damage (4), and an overall propensity to aggression and violence (6). Anger is also associated with reduced work effectiveness and problems such as being placed on probation, demotion, and termination (7). For some angry individuals, their anger is totally externalized and entirely justified, leaving their sense of self untouched. For others, however, their anger impairs their sense of

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psychological well-being; they feel out of control and overwhelmed by their anger and feel anxious, embarrassed, guilty, ashamed, and depressed by their anger and anger-related reactions. While these are but a few examples, anger and its dysfunctional expression affects the physical, psychological, interpersonal, educational, and vocational lives of many people.

Because of such anger-related consequences, anger is a frequent issue in therapy. Nearly all mental health workers recognize problematic anger, but it is often not clear how they should conceptualize it or intervene. When asked to diagnose an angry patient and a comparable anxious patient, mental health workers reached little consensus in diagnosing the angry patient, needed more information in making the diagnosis, tended toward more severe diagnoses, and felt the diagnosis was not as helpful in treatment planning (8). Our diagnostic system did not appear to be much help to them. This chapter will address how dysfunctional anger is and might be integrated into our diagnostic system. It begins with a review of where anger appears in DSM-IV and moves to a potential framework addressing problematic anger and aggression.

THE STATUS OF ANGER IN DSM-IV

Intermittent Explosive Disorder (IED) is one of the most common diagnoses given by mental health workers when confronted with an angry patient (8). IED refers to the inability to control aggressive impulses that result in discrete episodes of aggression, assault, or property destruction that is grossly out of proportion to external provocation. These episodes are often experienced as uncontrollable experiences preceded by tension and arousal buildup and followed by a sense of tension reduction and relief. DSM-IV notes that the person often reports intense angry feelings, but these angry feelings are not part of IED. IED is an impulsive, aggressive behavioral problem, not an anger-related condition. Moreover, IED cannot capture most problems of dysfunctional anger, because although prevalence data are lacking, IED is described as apparently rare. Something more is needed.

An alternative is to subsume problematic anger under the mood disorders. That is, anger problems are really a manifestation of or secondary to mania or depression. Anger and irritability are certainly involved in mood disorders. For example, presence of anger/irritability is one of the signs of major depression. In fact, for children and adolescents, it can be a defining criterion. Nonetheless, a patient does not have to experience anger/irritability to be diagnosed with a major depression. Anger is at best a secondary symptom (i.e., the presence of which helps rule in major depression, but which is not necessary for the diagnosis). Anger may also be involved in bipolar disorder. The prime definer of a manic episode is a week or more of a persistent mood that is elevated, expansive, or irritable. Anger, as defined by irritability, is thus one of the defining elements

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of a manic episode, but it must be accompanied by a variety of other symptoms such as inflated self-esteem and grandiosity. Thus, anger may be an important element in a manic episode, but only when it is accompanied by affective, cognitive, and/or behavioral characteristics that are not anger-related.

Dysthymia is another possibility. Dysthymia describes chronic, moderate depression of at least 2 years' duration or 1 year's duration of depression or angry/irritable mood in youth. However, two or more conditions not related to anger such as insomnia or poor appetite must also be present. Dysthymia is interesting in that DSM-IV mentions that "subjective feelings of irritability or excessive anger" are one of the most commonly encountered symptoms reported by dysthymics, yet anger is not one of the defining or secondary symptoms of dythymia for adults, only for youth. That is, patients report it as one of frequent, disquieting experiences, but it is not part of the defining symptomatology. In summary, for some of axis I mood disorders, anger is, at most, a secondary symptom or partial definer of the mood disorder.

None of the disorders is defined primarily by the presence of dysfunctional anger. In all cases, a diagnosis can be rendered without the presence of anger, and conditions unrelated to anger must be present for the diagnosis. Certainly diagnoses of mood disorders should be considered when anger exists in the constellation of factors characteristic of mood disorders. However, mood disorders do not accurately capture or describe the individual whose primary issues are dysfunctional anger. Another logical problem exists in considering anger as an outgrowth of depression. The argument assumes that anger is not a primary mood, but there is a large psychological literature (e.g., 9,10) that suggests anger is a core or primary emotion. Perhaps, dysfunctional anger would be better considered a problem of this primary affect, much as depression may be seen as an extension of the primary affect of sadness.

Posttraumatic Stress Disorder (PTSD) is another axis I disorder involving anger. PTSD is a persistent pattern of anxiety, avoidance, and psychic numbing following the direct or indirect exposure to a situation in which the person's or another's physical well-being was threatened or damaged. Irritability and anger outbursts are among the signs of persistent arousal. Anger is thus one of several indices of increased arousal, the presence of which supports a diagnosis of PTSD. Again, anger need not be present. Anger may also be involved defensively as part of numbing. For example, anger and aggressive outbursts may work to distance others and keep psychic numbing and avoidance in place. Nonetheless, it is unclear how PTSD accurately captures the experience of many angry patients. First, many have not experienced, directly or indirectly, the life threatening conditions necessary for PTSD. Second, many do not show the primary psychological characteristics of PTSD (e.g., anxiety, avoidance, numbing). They are more angry than anxious. Third, even though some individuals may learn anger outbursts and defensive aggression as a means of numbing and protecting one's self in

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PTSD or PTSD-like conditions, it may be more beneficial to outline disorders of dysfunctional anger if these predominate the clinical picture, rather than as part of PTSD per se.

Anger is common in some axis II personality disorders and is mentioned directly and indirectly in the diagnostic criteria of some. For example, paranoid personality disorder involves anger. Individuals with paranoid personality disorder are marked by a pervasive mistrust and suspicion of others. They misinterpret the motives of others and anticipate deceit, exploitation, harm, and malevolence. Because of this interpretive bias, they tend to be on guard, hostile, abrasive, and harboring of grudges. Anger is a relatively common emotion stemming from their perceptions of others, and aggression or counterattack is a common defensive reaction. Anger is specifically mentioned in criterion 6, "perceives attacks on his/her character or reputation that are not apparent to others and is quick to react angrily or counterattack." Anger and aggression are common to the paranoid personality disordered individual, but they would not have to be present for a diagnosis. Anger is thus a secondary symptom.

Anger, irritability, impulsive aggression, and destructive behavior are part of some antisocial personality disordered individual's emotional and behavioral responses. This is specifically mentioned in criterion 4, "irritability and aggressiveness as indicated by repeated physical fights and assaults." As with several other disorders, the presence of anger supports a diagnosis of antisocial personality disorder, but not all individuals with antisocial personality disorder necessarily display anger and aggression.

Anger is also seen in individuals with narcissistic personality disorder, who are characterized by grandiosity and an inflated sense of self-importance and by a sense of being special, gifted, and above others. Their inflated self-esteem, however, is often fragile and must be supported by the admiration, attention, and confirmation from others. Their sense of entitlement, combined with their relative lack of empathy and sensitivity to others, sets them up for misunderstanding and strained interpersonal relations. Denial of their entitlement often leads to significant anger and manipulative or attacking behavior. It can escalate into a temper tantrum, a narcissistic rage. Even given this kind of involvement, anger is not mentioned in the diagnostic criteria for narcissistic personality disorder, and the only element that comes close to describing anger, hostility, or aggression is criterion 9, "shows arrogant, haughty behaviors or attitudes." Thus, at best, anger is a secondary symptom of narcissistic personality disorder.

Histrionic personality disorder parallels narcissistic personality disorder in terms of the inclusion of anger and aggression. Histrionic personality disorder is characterized by marked attention seeking, dramatic behavior, and excessive emotionality. They often demonstrate poor delay of gratification, being easily frustrated by and intolerant of situations that do not allow for immediate gratification of their needs. An exaggerated, angry, impulsive reaction is not uncommon

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in such situations, but anger, irritability, and impulsive aggressive behavior are not formally mentioned in the diagnostic criteria.

Obsessive-compulsive personality disorder is another of the personality disorders in which a form of anger is common. Individuals with obsessive-compulsive personality are preoccupied with order, perfectionism, control, rules, and the like. They tend to be excessively concerned with responsibility, conscientiousness, principles, and morality. This sets them up to experience considerable anger and resentment when others do not follow their rules or morals, when others do not do things "right," or in situations in which they cannot maintain control. Sometimes, their anger is expressed as righteous indignation, but often is expressed in passive-aggressive complaining and resistive behavior. Nonetheless, anger is not mentioned in the diagnostic criteria. As close as aggressive behavior is described is in criterion 8, "shows rigidity and stubbornness," which must be interpreted as passive-aggressive behavior.

The final personality disorder, the borderline personality disorder, is one in which anger is mentioned specifically in the diagnostic criteria. Borderlines are characterized by pervasive instability in self-image, interpersonal relations, and emotional control and by marked abandonment issues. They tend to dichotomize relationships as "good" if they can take care of them and prevent abandonment, and "bad" when they do not. The result is intense, stormy, unstable relationships because few, if any, relationships can withstand their constant need for reassurance and prevention of rejection, separation, and abandonment. Borderlines demonstrate poor emotional self-regulation, especially for anger, and often engage in impulsive and aggressive behavior, which is often aimed at the caregiver who has failed them in some way, including therapists. Anger is mentioned directly in criterion 6, "affective instability due to marked reactivity of mood (e.g., . . . irritability lasts a few hours and only rarely more than a few days)," and criteron 8, "inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights)." Thus, anger is mentioned specifically for borderline personality disorder, but is not necessary for a diagnosis, even though most clinicians would agree that, at least emotionally speaking, anger dyscontrol is one of the prime features of borderlines.

In summary, anger and sometimes aggression are involved in a number of axis II disorders. However, even though anger is apparent in the lives of many of these individuals, it is not mentioned at all in some criteria, is mentioned only indirectly and inferentially in others, and specifically in a few. However, there is no personality disorder in which the presence of anger is necessary for a diagnosis. Parallels certainly exist between some processes in individuals with anger problems and some personality disorders. For example, some of the attributional processes in paranoid personality disorder are similar to attributional processes in problematic anger (e.g., hostile attributional bias). Chronic anger and interper-

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sonally aggressive behavior characterize some antisocial individuals and some individuals with dysfunctional anger. Some individuals with anger problems show a narcissistic entitlement and anger and aggression when their entitlement is thwarted. Some individuals with anger control problems show anger dyscontrol not unlike that of borderlines. However, many individuals with anger problems do not share other characteristics with personality disordered individuals, and there are simply too many dissimilarities to consider all problematic anger as a subcase of one or more personality disorder.

Axis III disorders to may reflect anger. If anger arousal can be reasonably shown to trigger, contribute to, or exacerbate a medical condition, then anger can become a diagnosable condition. For example, if anger is shown to trigger headaches or to contribute to coronary artery disease or hypertension, then anger is a diagnosable condition. However, anger is, in this case secondary to or in conjunction with a medical condition. While this is positive feature for anger-related medical conditions, it contains two problems. First, not all people with significant anger problems experience anger-related medical issues. Second, even within anger-related medical conditions, it does not legitimize anger as a problem prior to medical condition. For example, the angry, Type A individual must wait until the first coronary before his/her anger can be addressed, rather than having anger be a diagnosable problem for which they could receive treatment that might prevent or delay the initial heart attack.

This review of DSM-IV shows that there are several axis I and axis II disorders in which anger is a secondary (i.e., the presence helps rule in the disorder) or correlated (i.e., frequently found in individuals with the disorder but not mentioned in the criteria) symptom. Anger may be involved in some axis III disorders as well. However, there are no anger-based disorders (i.e., where anger must be present for a positive diagnosis). Quite simply, anger disorders are missing from DSM-IV.

Put another way, there were numerous diagnostic categories for involving disturbances two other major emotions—anxiety and depression. For example, if a person is chronically, moderately depressed or chronically, moderately anxious and unrealistically worried, then diagnoses of dysthymia and generalized anxiety disorder may be warranted. But, what of the individual who is chronically, moderately angered? There is no parallel generalized anger disorder. For example, if an individual is intensely fearful and avoidant of driving or situations in which he/she might be evaluated, criticized, and rejected, then a diagnosis of simple or social phobia may be appropriate. But what of the person who is intensely angered and perhaps aggressive in exactly the same situations? There is not an analogous situational anger reaction diagnosis. As a final example, if an individual has suffered psychosocial stress and experiences exaggerated or dysfunctional anxiety, depression, or a combination of anxiety and depression, then relevant adjustment disorders with anxious, depressed, or mixed anxious and de-

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pressed mood are relevant. But what of the person who has experienced the same stressor, but is reacting with anger, but not aggression or other disturbances of conduct? Again, there is no parallel adjustment disorder with angry mood. Absence of diagnostic criteria, however, does not mean such problems do not exist. They do exist. People hurt and suffer because of them, and in some cases bring considerable suffering to others. They are worthy of our conceptual, clinical, and scientific attention.

A POTENTIAL CLASSIFICATION OF ANGER-RELATED DISORDERS

Classification in psychopathology can serve several important functions (11). For example, it can provide a common language that facilitates communication among scientists and practitioners. Diagnostic systems assist in identifying important similarities and differences among patients which may help differentiate underlying psychological and physiological processes, facilitate treatment planning, and aid in prognosis. Without common language and conceptual systems, researchers and clinicians do not have the language to compare and contrast patients with anger control problems and begin identifying important variables and subtypes. All of this, plus the theoretical models that are adapted to account for phenomena, advances scientific and clinical knowledge. A nosology may assist clinical science in another way. Funding of research is a critical process. However, funding is currently limited primarily to established diagnostic categories. However, as established previously, there are no anger-based diagnostic categories. Without such diagnostic categories, funding for research and clinical trials for anger problems will lag, further hampering the understanding and treatment of individuals with problematic anger and aggression.

What is proposed next is a way of thinking about and conceptualizing anger disorders. It is presented not as a final or empirically validated system. To the contrary, it is presented as a heuristic way of thinking and organizing information about individuals with problematic anger that could facilitate research and clinical understanding. In describing potential disorders, each will be framed in terms of the following issues: 1) the nature/degree of life interference that leads the anger to be considered dysfunctional or pathological; 2) the nature of anger; 3) the presence or relative absence of aggression; 4) the context or the apparent triggers of dysfunctional anger; and 5) other disorders that must be ruled out.

Dysfunctional/Pathological Anger: Is It Possible?

If anger did not lead to sufficient dysfunction, it could not be considered disordered, and proposing anger disorders would be moot. Psychopathology implies significant distress, pain, or suffering to the individual and/or others, and/or dis-

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ruption in typical roles, relationships, and goal attainment. DSM-IV's definition is as follows:

> In the DSM-IV, each mental disorder is conceptualized as a clinically significant behavioral or psychological syndrome or pattern that occurs in the individual and that is associated with present distress, (e.g., a painful syndrome) or disability (i.e., impairment in one ore more areas of functioning) or with a significant increased risk of suffering death, pain, disability, or an important loss of freedom. In addition, this syndrome or pattern must not be merely an expected and culturally sanctioned response to a particular event, for example, the death of a loved one. Whatever its original cause, it must currently be considered a manifestation of a behavioral, psychological, or biological dysfunction in the individual (12:xxi–xxii).

As noted in the opening paragraph of this chapter, anger can serve many adaptive functions. However, anger also can be associated with significant personal distress (e.g., feelings of guilt, anxiety, self-recrimination), and adverse physical (e.g., associated with disease processes, injury to self or others), relational (e.g., marital discord, damaged relationships), role (e.g., youngster leaves home or avoids a parent because of how anger is expressed), legal (e.g., arrest for assault or child abuse), educational (e.g., dismissed from school for ways anger is expressed), financial (e.g., medical, legal, or property costs associated with anger expression), and vocational (e.g., being terminated for ways anger is expressed) costs. These types of costs and suffering appear to more than meet the threshold for DSM-IV's definition of maladaptiveness or dysfunction, and there are therefore no criteria that would automatically exclude anger-based categories.

The Nature of Anger

Anger is a "syndrome," an internal, cognitive-emotional-physiological experience, consisting of emotional/experiential feelings (e.g., feeling mad or furious), physiological arousal (e.g., elevated heart rate, muscle tension), and cognitive processes (e.g., attributions of intentional harm, blaming, inflammatory labeling). Anger appears in response to a range of external and internal prompts and exists in a cultural context which specifies appropriate and inappropriate forms of anger experience and expression. Anger leads to personal, social, legal, educational, and vocational consequences, which, on occasion, may be negative enough to be considered a disorder. The experience of anger, however, is conceptually separable from the way the person behaves when angry (see next section).

Angry emotions and feelings are central to all proposed diagnoses and must be present for a positive diagnosis. That is, the patient must report or show sig-

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nificant elevations of angry feelings, feelings that interfere with or lead to conditions that negatively impact the person. The patient may report strong feelings of anger such as being furious, mad, livid, enraged, pissed off, red hot, and the like. Other patients may report strong feelings of anger more in "cold" terms such as cold fury, frozen in rage, or icy anger. Anger, however, is not solely described by emotions or feeling states. There are physiological and cognitive elements as well. Physiologically, the patient may report symptoms arousal such as: 1) increased heart rate; 2) increased muscle tension generally or in specific areas such as clenched hands or jaws; 3) trembling or shaky feelings; 4) sweating or clammy skin; 5) rapid, often shallow breathing; 6) flushes or hot sensations; 7) restlessness or agitation such as in feeling as if one is going to jump out of one's skin, cannot sit still, or pacing about; 8) feeling keyed up or on edge; 9) jumpiness or exaggerated startle response; and 10) gastrointestinal upset, pain, nausea, or a sickening feeling in the stomach. Cognitively, the patient may report or demonstrate some of the following cognitive processes:

1. An exaggerated belief or appraisal that he/she has been treated unfairly, trespassed upon, or violated in some way
2. An unsubstantiated or exaggerated belief that others are out to hurt, harm, damage, or control the person (hostile attributional bias)
3. Categorizing another person as a member of a hated group or as the enemy
4. Thoughts or images of hurting, harming, damaging, getting back at, exacting revenge upon, or retaliating upon the perceived source of anger
5. An exaggerated sense of the importance of goal-directed behavior that has been blocked or impeded
6. Demands that others or events should be as he/she desires
7. Demands that he/she should not be exposed to, deal with, or endure angering events
8. Mentally labeling people, things or events in highly negative, overgeneralized or obscene ways such as cursing, name calling, derogatory labeling, etc.
9. A strong belief that his/her level of anger and/or aggression is justified by the nature of outside events
10. Blaming others or outside events for the extent of angry feelings and reactions
11. Brooding or angry rumination about the source of anger such as turning a terminated relationship or job over and over in one's mind
12. Racing thoughts and difficulty in concentration
13. Being very guilty and self-recriminating for having angry thoughts, feelings, and urges.

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Although no individual will show all cognitive or physiological indices nor will the profile of cognitive and physiological characteristics be the same from person to person, individuals with problematic anger are expected to show several of the cognitive and physiological elements of anger.

Anger Expression: Aggression and Other Dysfunctional Behavior

The behavioral or expressive element of anger is important, but not all individuals behave the same when angry. Some engage in modulated expression, negotiation, problem solving, taking time out, and the like. Individuals who consistently engage in such behaviors are not likely to have problems with anger, much less a diagnosable anger disorder. Others, however, behave much more maladaptively. Some behave aggressively, but the form of aggression may take many different forms: 1) loud verbal outbursts, yelling, or screaming; 2) verbal intimidation and threats (e.g., threatening to hit the person, "kick their ass"); 3) cursing, name calling, insults, belittling, etc.; 4) repeated sarcasm, cutting verbal remarks, or hostile humor; 5) physical intimidation and threats (e.g., making obscene gestures, shaking his/her fist at others); 6) physically assaultive behavior toward others (e.g., hitting, slapping, kicking, punching, grabbing, or throwing things at another); 7) physically assaultive behavior towards property or the environment (e.g., throwing, slamming, banging or pounding on, or breaking things); 8) brandishing or threatening with a weapon (e.g., pointing a knife or gun at someone); 9) use of a weapon (e.g., stabbing or slashing with a knife, using one's vehicle to threaten or intimidate); 10) actively seeking out or provoking an aggressive confrontation; 11) behavior the purpose of which is to hurt, harm, damage or seek revenge on another (e.g., starting a rumor about another person or purposefully destroying part of a document to get back at another person); 12) intense, hostile nonverbal behavior (e.g., icy stares and glares, menacing looks); and 13) belligerent or stubborn refusal to cooperate with reasonable requests in dealing with angering events. Others respond to anger maladaptively, but not necessarily aggressively.

Such maladaptive behavior could include things such as: 1) becoming intoxicated or drug involved; 2) inappropriate withdrawal; 3) disengagement from important life roles and responsibilities; 4) anger suppression with continued arousal leading to psychophysiological involvement; 5) prolonged angry brooding and rumination; 6) engaging in risky, dangerous behavior such as speeding and reckless driving; and 7) acting out in other nonaggressive ways. Thus, not all negative consequences from poorly handled anger necessarily stem from aggression, and this is an important distinction, both conceptually and clinically.

Proposed anger disorders are differentiated by whether aggression is present or not. An anger disorder with aggression will be one in which a significant

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level of aggressive behavior is present in addition to anger, and an anger disorder without aggression describes significant interfering anger, but a high level of aggression is not present. This differentiation is made because of its implications for conceptualization and treatment. Aggression is often a serious problem leading to serious consequences and should be treated in its own right. Specifying anger with aggression provides this focus. On the other hand, a relative absence of aggression does not mean that angry but not highly aggressive individuals do not suffer significantly. Their suffering should not be overlooked owing to a diagnostic emphasis on aggression. That is, anger can be problematic without being associated with aggression and should receive attention in its own right.

Triggers/Prompts of Anger

Dysfunctional anger can also be described in terms of the contexts in which it appears or conditions that elicit it. There appear to be four relatively distinct prompts of anger. For some individuals, anger does not appear prompted by external events or appears grossly out of proportion to provocation present. The patient experiences a kind of "anger attack" with intense anger welling up over a 10- to 20-min period. Other anger reactions, however, are very situation or context bound. This anger is triggered by specific events (e.g., a driver cuts the person off or a child's irresponsibility and lying) or by a series of events that are tied together by a specific theme (e.g., being criticized or disrespected). In these cases, anger is elicited by and limited to a small number of events or situations. Such anger, like phobias, is situational. For others, anger is elicited by a wide range of provocations, frustrations, insults, and encroachments on the personal domain, internal emotional and physical conditions, or it may appear as a frequent angry, irritable mood state. For these individuals, the sources of anger are not delimited, but are many, diffuse, and generalized (general anger). Typically, anger for such individuals is experienced more frequently than situational anger reactions. Finally, an increase in anger or an angry, irritable mood may follow psychosocial stress; that is, while some individuals may show elevations of anxiety or depression following psychosocial stress, others show an elevation in anger and irritability in the weeks and months following the stressor. To summarize, sources or triggers for anger may be 1) unclear with anger quickly welling up within the person, 2) relatively situational or context-bound, 3) many and generalized, and 4) psychosocial stress.

The four sources or triggers of anger can be combined with dysfunctional anger and the presence or absence of aggression to describe eight potential anger disorders—anger attacks with and without aggression, situational anger disorder with and without aggression, generalized anger disorder with and without aggression, and adjustment disorder with anger and with anger and aggression. In every disorder, angry feelings or emotion must be present along with some cognitive

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and physiological involvement. For the anger reaction to reach the level of a disorder, anger and its expression must lead to significant distress or dysfunction. Finally, the clinician must consider the anger in the context of other presenting concerns and information in order to rule out other diagnoses.

ANGER ATTACKS WITHOUT AGGRESSION

Patients with this type of anger reaction do not have clear external triggers of their anger. They experience intense anger reactions or "anger attacks" over a 10- to 20-min period followed by a sense of relief. These attacks are not accompanied by a great amount of aggression, but are often quite distressing to the individual and those around him/her, and the patient may become quite worried and anxious about the reoccurrence of such intense anger experience and about whether he/she is "going crazy" and out of control. Such anger attacks have been documented by Fava and colleagues (e.g., 13,14) in both depressed patients and mental health patients generally. In many ways, the experience of many of these patients parallels that of IED patients, except that the anger attack is not accompanied by the marked aggressive or destructive behavior during an IED episode.

Anger attack without aggression is exemplified in the following case. A 38-year-old woman experienced anger attacks every few months. Episodes did not appear related to any environmental events and were unpredictable, although the patient could feel them coming on a few minutes before they became most intense. Over a 10- to 15-min period, intense feelings of anger were experienced accompanied by cardiovascular arousal, short rapid breathing, tight constricted feelings across her chest, and a sense of tension and agitation. She felt out of control, like she was going crazy, and like she wanted to yell and scream and hit someone or something. She did not act on these impulses. She typically withdrew as much as possible, pacing about, grimacing, and wringing her hands. If she was alone, she reported that she did sometimes yell and scream, but not if others were present. She just "toughed it out," with the emotionality tending to subside in 15–20 min, leaving her emotionally exhausted and fatigued, sometimes with headaches and lasting irritability. She was quite distressed about these reactions and was afraid she was going to do something destructive or hurtful to others. She reported that these episodes scared her husband and family and that the anger and the aftermath reduced her personal effectiveness and role functioning as a mother and wife.

ANGER ATTACKS WITH AGGRESSION

These individuals not only experience anger attacks but also engage in physically assaultive or destructive behavior that is out of proportion to provocation present.

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For example, with minimal provocation the person flies into a rage and verbally and physically assaults others or destroys property. This clinical picture describes the experience of patients with IED. If criteria for IED were revised to include the intense anger arousal experienced by patients with IED, then anger attacks with aggression and IED would describe essentially the same patients. Revisions might also include issues outlined by Coccaro et al. (15). Linking anger attacks without aggression and IED in this manner would facilitate an evaluation of their similarities and differences and an exploration of potential underlying mechanisms.

Anger attack with aggression is captured in a case of a 25-year-old male who had experienced four such episodes over the past 4 years. When in college, he experienced the first episode. He reported being tense and irritable while studying and that his anger increased rapidly for no reason. When his roommate turned on the television, he flew into a rage, screaming obscenities at his roommate, and then repeatedly shoving him, throwing a chair, destroying a stereo, breaking a window, and damaging the walls. The second and third incidents involved the same kind of rapid escalation of anger with him destroying a table he was refinishing in one case and causing several hundred dollars' damage to his car as he smashed the windows, hood, headlights, and sides with a large wrench, as well as putting a 12-stitch gash in his hand during the melee. The fourth incident was similar in terms of the rapid onset of anger, but involved him yelling at his passenger and driving aggressively and reckless through approximately 15 miles of heavy interstate traffic (e.g., driving on the back bumper and flashing lights, screaming obscenities at and flipping other drivers off, and driving in excess of 100 mph). He was distressed by these incidents, feared that he was losing his mind, experienced significant interpersonal and financial consequences, and was worried that he was going to seriously injury himself or others or be arrested.

It would be important for the clinician to assess whether intense anger episodes with and without aggression were not better conceptualized as conditions such as: 1) a diagnosable psychotic episode; 2) a kind of dissociative flashback in a patient with PTSD; 3) an aspect of a major depressive, bipolar, or substance-induced mood disorder; 4) an episode involving a cognitive disorder or medical condition; 5) part of antisocial, paranoid, or borderline personality disorder; or 6) apparent only when drugs or alcohol were ingested.

SITUATIONAL ANGER DISORDER WITHOUT AGGRESSION

Individuals with this type of anger problem experience intense angry feelings in response to specific situations or to a cluster of situations sharing a common theme. They become very angry, but do not engage in frequent or intense aggressive behavior. Aggressive behavior may be present on some occasions, but tends

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to be infrequent and not severe (e.g., cursing to oneself, mild derogatory comments, or slamming a door). They may show other dysfunctional or self-defeating behavior such as inappropriate withdrawal, becoming intoxicated, being very self-disparaging, or physical problems such as irritable bowel, headaches, and bruxism, but they are not highly aggressive.

Situational anger without aggression and its consequences are exemplified in a case of a 38-year-old woman who commuted 90 minutes to and from work and became very angry at other drivers on nearly every commute. Physiologically, she experienced marked tension in the neck, back, and shoulders, general physical tension all over, pain in her hands from clenching the wheel, and an upset stomach. Cognitively, she engaged in a nearly constant internal dialogue about other drivers and their behavior, marked by silent name calling and cursing, demanding for different behavior from them, many critical, derogatory comments about their behavior, difficulties concentrating, and "being so pissed I can't think straight." Although frequently and intensely angered, she engaged in minimal aggression, only occasionally cursing aloud to herself regarding other drivers. She reported that her commutes were miserable and that her irritable mood often carried over to work or home. At work, she experienced difficulties in concentration for the first hour or two and was often impatient with others. At home, she consumed more alcohol than she wanted, often just "vegged out" wasting time in ways she did not want, and was "not worth anything at home most evenings" because of the anger she experienced driving home from work. She also indicated that she tended to be more irritable with her husband and children in the evenings than she was on weekends when she did not commute. Her physician suggested that her headaches might be due in part to this chronic anger as well. Although she recognized that her anger was excessive, she felt incapable of doing anything about it.

Situational prompts need not always be present as exemplified in the next case. A mild-mannered man's wife left him for another man. Even though the relationship terminated three years later, he continued to be very angry and bitter toward her. Three or four times a week, he would think about how "she screwed me over" and would experience intense anger, gastrointestinal upset, hot sensations across his chest, and many negative thoughts and images about his ex-wife. These periods of angry rumination generally lasted from 1 to 3 hours and led him to feel out of control, like he was "continuing to waste time on her," to lost time with friends and other activities, and to bouts of colitis which he had experienced since the breakup. Although his ex-wife lived in the same town, he did not act out his anger toward her, save occasional negative comments about her to his friends.

In examples such as these, a small number of situations elicit strong anger reactions, but minimal aggression. Nonetheless, such individuals suffer negative consequences. Their health, self-esteem, work and family roles, relationships in

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and out of the family, time, energy, and in some cases finances are impacted adversely. At the same time, anger appears to be the primary problem and does not appear to be better accounted for by another disorder (e.g., PTSD, dysthymia, psychotic disorder, major depression, or generalized anxiety disorder).

SITUATIONAL ANGER DISORDER WITH AGGRESSION

Individuals with situational anger disorder with aggression are very similar, except that they also behave aggressively. When angry, they may become loud and argumentative, belittle and put others down, curse and name call, or threaten physical behavior (e.g., "I'm going to knock your head off" or "I'm going to kick the crap out of you"). They may physically assault others or the environment around them (e.g., push or shove someone or throw things). They may not react aggressively every time the encounter the situation, but across events they show an elevated probability of aggression.

Clinically, this pattern is exemplified in the owner of a small firm. He was technically capable, but also very perfectionistic. When he encountered inferior work, he became very angry and loudly berated, belittled, cursed at, and put down his employees. Although he did not mean to become physically intimidating, he often appeared so because he was a large man and often got very close to others when being verbally abusive. Such emotional outbursts led him to feel stupid and out of control and to problems of employee morale, reduced work productivity, and staff turnover. In addition, employees tended to avoid him and not seek consultation from him (which was precisely one of the situations that angered him most), because they did not wish to broach one of his tirades. He stated that he felt his anger had cost his company tens of thousands of dollars in lost productivity and staff turnover.

Sometimes things rather than people are the targets of the aggression. For example, one woman became very angry at things that did not work easily. When encountering such events, she would fly into a rage, cursing and verbally abusing the item, and hitting, throwing, breaking, or pounding the offending object, resulting in, among other things, a destroyed stereo and laptop computer, breaking things such as vases, mirrors, and windows, breaking her finger in one incident, and lacerating her hand requiring several stitches on another occasion.

In summary, for individuals with Situational Anger Disorder with Aggression certain types of situations elicit strong anger reactions and, at least on a significant number of occasions, aggressive behavior. These individuals suffer a variety of negative consequences directly and indirectly from both their high levels of anger and their aggressive behavior.

Validity of a proposed conceptual scheme would be strengthened if there is research supporting it. Although there is no empirical research on situational

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anger disorders, data on high-anger automobile drivers seeking counseling for driving anger reduction provides an example of the kinds of characteristics suggested in this type of disorder (16). Although not separated into nonaggressive and aggressive subtypes, these high-anger clients were very different from low-anger drivers. Although both groups drove as often and as many miles, suggesting that differences were not due to different exposure to provocation, 3.4 times more situations elicited much or very much anger for high-anger drivers. They were angered 2.7 times more often and reported significantly more intense anger behind the wheel. High-anger drivers, however, were not universally angry, as they did not report more anger when driving unimpeded on a country road. High-anger drivers expressed their anger in more aggressive, less constructive ways, reporting they are more likely to express anger verbally (e.g., yell or swear at another driver), physically (e.g., make a hostile gesture to another driver), or vehicularly (e.g., purposely speed up or slow down to frustrate an offending driver). They also reported being less likely to express their anger in an adaptive manner (e.g., focus on safe driving or engage in calming behaviors). They engaged in 3.5–4.0 times more aggressive (e.g., tailgating in anger) and twice as much risky (e.g., darting in and out of traffic) behavior on the road. They were more than twice as likely to hit or damage their vehicles in anger and more than 10 times as likely to sustain injury as a result of such aggressive behavior (e.g., cutting one's hand from hitting the car). High-anger drivers also experienced more moving violations, close calls, crashes, and near crashes, although not more major-injury accidents. They also tended to be more generally angry, anxious, and impulsive, which may only exacerbate their anger behind the wheel. In summary, high-anger drivers were an at-risk group that parallels characteristics of situational anger disorders as they were more easily, frequently and intensely angered on the road, expressed their anger in more hostile/aggressive and less adaptive/constructive ways, engaged in significantly more aggressive and risky behavior, and were at elevated risk for many crash-related outcomes.

In the situational anger disorders, anger is elicited by a small number of situations or by a number of situations which share a common denominator. Although anger is intense, it tends to be relatively limited to these situations and, therefore, typically limited in frequency and pervasiveness. The next set of disorders, however, describe individuals who experience anger frequently and pervasively, either from a wide number of sources and/or as frequent periods of angry/irritable mood.

GENERAL ANGER DISORDER WITHOUT AGGRESSION

This disorder describes a group of people who are frequently angry, often several times per day or for lengthy periods of time, but who do not typically respond

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aggressively. For some, anger appears triggered by a wide range of external situations. Affronts, injustices, violations, frustrations abound everywhere in their worlds, and they respond with anger. Portions of their anger may be triggered by internal cues such as feelings of disappointment, rejection, or hurt, as recalling memories of previous hurts, losses, and frustrations, as ruminating about past or present mistreatment, difficulties, or grudges, or as imaging revenge, retaliation, and retribution. For yet others, anger appears to be a function of deeply ingrained attitudinal and attributional sets (e.g., beliefs that life should go the way that the individual desires and that he/she should not have to put up with negative events). Anger is frequent and pervasive as a great number of events are frustrating by these criteria. A variant on this theme is the invocation of various narcissistic rules (e.g., life "should" be "fair," "just," "kind and courteous," "easy"). Again, anger is frequent and pervasive as these rules are repeatedly broken. Another cognitive pattern is one of intentionality, preventability, and blame, in which the individual tends to interpret the behavior of others as intentional frustration and mistreatment for which they are to blame. Anger, resentment, and distrust eventuate and are justified because they perceive themselves the victim of others' purposeful misdeeds. Yet others tend to be very opinionated and dichotomize their worlds ("right/wrong," "good/bad," "respectful/disrespectful," etc.) and then respond with anger whenever the positive polarity is disconfirmed, which happens often. Although these are but a subset of the ways anger may be triggered, they describe individuals for whom anger is frequently and diversely triggered.

Anger threshold for these individuals is often lowered by the presence of negative emotional, cognitive, and physiological states. Since these individuals are frequently angered, their angry state carries forward and increases the chances of anger in other situations. Other negative states (e.g., fatigue, feeling ill, stressed) facilitate anger as they seem to increase the presence of aversive images, feelings, and memories and lower the threshold for anger. Often these types of conditions operate in circular fashion wherein the aversive condition increases anger, which in turn exacerbates the very condition contributing to anger in the first place, eventuating in cycles of anger and reduced coping.

Whatever the sources of anger, these individuals experience chronic, pervasive anger. To describe this quality and to separate it from a limited period of life in which the person was angry, it is suggested that pervasiveness and chronicity be captured in two ways. First, the pattern of anger is to be present more days than not for at least a year. Second, the person is to have been free from frequent anger reactions or periods of angry/irritable mood for no more than a month during the past year. Although the temporal criteria of a year's and a month's length are somewhat arbitrary, they establish anger as a persistent pattern and separate it from reactions to a specific stressor or period of difficulties. Although these diagnostic dimensions focus on the pervasiveness of anger, they do not imply that such individuals are irate every waking moment and never experience

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happiness or other mood states. To the contrary, they may experience happy, positive reactions, but they also experience frequent, pervasive, dysfunctional anger reactions as well.

While frequently angry, individuals fitting the proposed general anger disorder without aggression do not engage in aggressive or destructive behavior. On occasion, they may show verbal aggression (e.g., make a few negative comments or curse) or physical aggression toward objects (e.g., slam doors), but these behaviors tend to be infrequent compared to the number of times that they are angry and relatively mild in intensity and severity. Because of their anger, they suffer negative consequences to their health, sense of self, quality of life, personal or work relationships, and the like.

A low-level manager in a large industrial firm who felt he had been passed over in promotions demonstrates this disorder. He experienced anger frequently at work. He felt that supervisees and supervisors did not respect him as they should, that others were lazy and unproductive, that others purposefully made his job hard, and that supervisors made fun of him and gave him all the thankless tasks. Anger did not stop at work, however. He felt "shafted" by the rest of life. For example, he felt that his ex-wife, who had residential custody of their two children, was a controlling, money-grubbing person who made life miserable and prevented him from having quality access to his daughters. For him, life was chronic frustration and hassle, but he engaged in little aggression. He reported thoughts and images of retaliation and of times when he really would like to give others a "real piece of his mind," but tended to suppress his anger, often brooding about it in the evenings, sometimes experiencing gastrointestinal upset and drinking too much. He indicated his only respites were camping, traveling, television, and some visits from his children.

GENERAL ANGER DISORDER WITH AGGRESSION

Individuals meeting the proposed general anger disorder with aggression not only experience frequent anger, but also frequently engage in aggression as well. They may engage in forms of verbal or physical aggression, for example. Moreover, although the individual need not aggress every time he/she is angry, the pattern of aggression is nonetheless fairly habitual with the individual either showing a variety of aggressive behaviors across episodes or being fairly consistent in the form of aggression.

One female patient showed this pattern. To name just a few things, she was angered by traffic, lines, people who did not do their jobs correctly, her own or others' mistakes, unsolicited phone calls, any comments perceived as critical of her, things that did not go well or quickly, and almost anything her husband did or did not do her way. She stated she "felt tight and ready to explode" and "like a bomb just ticking away." Her existence was constantly hurried, a desper-

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ate, angry, impatient chaos punctuated by periodic yelling and screaming laced with vitriolic criticism, name calling, cursing, and verbal abuse. Sometimes, she threatened and postured physically and occasionally followed through on such threats (e.g., slapped or threw things at her husband). Anger and aggression appeared exacerbated by negative physical states as she indicated that when she was tired or premenstrual, she was a "total screaming mess." Her health, sense of self, and work and family relationships suffered because of the anger and abuse.

In summary, these individuals are not only frequently and pervasively angry, but also frequently aggressive as well. The form of aggression can vary across people or within the same person over time, but it is frequently present. They too suffer many negative consequences, not only from their anger, but also from aggression.

As with dysfunctional situational anger reactions, validity of the proposed diagnostic category would be enhanced by supportive research. Studies on high-anger individuals seeking counseling for general anger reduction (17) provides an analog of such patients. Although these clients were not separated into aggressive and nonaggressive groups, these generally angry clients showed the following characteristics compared to low-anger individuals. In diary studies, they reported more frequent and intense anger in day-to-day living and reported levels of anger that they considered more interfering or disruptive. In survey studies, they reported greater anger across a wide range of situations, and more situations that elicited much or very much anger, suggesting more events angered them. They reported more intense anger-related physiological arousal and spontaneously reported additional signs of arousal even when not asked to do so.

They also reported engaging in more verbal and physical aggression and antagonism, and on measures of anger expression reported both more anger suppression and outward, negative, aggressive expression and less controlled or modulated expression, supporting tendencies toward greater aggressiveness. High-anger clients also reported expressing their anger more via verbal assault, noisy arguing, physical assault on people, physical assault on objects and the environment, dirty looks, and negative body language, and less through reciprocal, assertive communication, taking of time outs, cognitive reflection, and thinking before responding, and anger control strategies. They also suffered more anger-related consequences. High-anger clients experienced more frequent adverse consequences of nearly every type (e.g., anger led to lowered self-esteem, feelings of tension and anxiety, arguments with others, physical altercations, property damage, damaged relationships, alcohol consumption, and the like). Moreover, they reported from two to 14 times as many negative consequences, suggesting their anger led to greater impact on their lives. High-anger clients also reported more frequent and, in some cases, more severe consequences experienced in their worst anger-related event of the past year (e.g., physical harm to self or others, property

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damage, damaged or destroyed relationships, etc.). In summary, high-anger clients represent a clinically meaningful group that experiences anger more easily, frequently, and intensely; expresses anger in hostile/aggressive ways; and experiences more negative anger-related consequences.

ADJUSTMENT DISORDER WITH ANGER

Adjustment disorders consist of maladaptive responses occurring within 3 months of one or more psychosocial stressors. Maladaptiveness is judged in terms of an excessive reaction in light of developmental or cultural norms, and/or of impairment in social or vocational performance. Currently, there are adjustment disorders defined primarily in terms of their emotional features, namely with anxiety, depression, or a mixture of the two (12); that is, an adjustment disorder diagnosis is available if an individual reacts to one or more psychosocial stressors primarily with anxiety and/or depression. However, if the individual reacts primarily with anger, but does not act out aggressively or with other significant problems of conduct or deportment, then there is no available diagnosis. To address this oversight, an adjustment disorder with anger is being proposed in a manner parallel to DSM-IV adjustment disorders with anxiety or depressed mood. Specifically, this diagnosis would be for the individual experiences major psychosocial stress and responds with anger to stressor-related cues and/or with increased angry/irritable mood sufficient to violate cultural and developmental expectations and/or disrupt or impair the person's life. In order to be parallel to other adjustment disorders, specific levels of cognitive and physiological involvement are not outlined, although they are commonly present. The individual may show mild aggressiveness or acting out (e.g., mild argumentativeness or alcohol misuse), but not sufficient to be considered conduct problems.

For example, a woman lost her job owing to downsizing. For the next 4 months, she experienced frequent periods of anger and irritability and strong anger at nearly any mention or reminder of her prior employment. Cognitively, she expressed a strong sense of being treated unfairly and unjustly and believed that the company could have and should have prevented the layoffs. Although anger was predominant, there was minimal aggression or other dysfunctional behavior. She felt preoccupied and out of control; her anger negatively influenced interviewing for other jobs; and her angry preoccupation with the company distanced her husband and two teenage children.

Another example involves a couple who were unable to have children as they had wanted and planned for years. Shortly after a definitive medical workup, the husband lapsed into a fairly chronic angry/irritable mood punctuated by less frequent intense anger. At these times, he would become agitated with severe tension in the jaw and face, stomach pain and nausea, and a series of thoughts about how unfair the infertility was, how somehow it was someone's fault, even

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though he knew better, how he should not have to deal with this, how medical science should have a cure, and streams of subvocal curse words which were atypical for him. Except for occasional irritable words with his spouse and one occasion in which he had thrown his glass into the sink when he was in one of his angry ruminative periods, he did not show significant problems of aggression or other dysfunctional behavior.

In summary, such individuals have suffered one or more sources of psychosocial stress and reacted to these stressors with increased anger and/or angry/irritable mood, but not with other dominant mood, conduct, or work/school problems. The problems are primarily anger-based, hence the proposed adjustment disorder with anger (i.e., an excessive anger reaction to a psychosocial stressor).

ADJUSTMENT DISORDER WITH ANGER AND AGGRESSION

Adjustment disorder with anger and aggression describes individuals who experience psychosocial stress and are reacting with maladaptive levels of anger and aggression. For example, in case examples in the prior section if the woman had made repeated irate or threatening phone calls to her former work site or if the man had become verbally or physically abusive, such a diagnosis might be warranted.

It is arguable that this diagnostic category is not necessary. Within a DSM-IV framework, aggression can be accommodated within the current adjustment disorder diagnoses. If aggression predominates over anger, then adjustment disorder with disturbance of conduct is appropriate. Where both anger and aggression are salient, an adjustment disorder with mixed disturbances of emotion and conduct is appropriate, assuming anger is considered a dysfunctional emotion if mixed with conduct problems. Thus, aggression can be accommodated within the current system in terms of a disturbance of conduct. The proposed adjustment disorder with anger and aggression is proposed for consistency in conceptualization and because it more descriptively focuses on the two issues of clinical relevance, namely anger and aggression, rather than the vaguer reference to mood and conduct. At the very least, what seems missing diagnostically is an adjustment disorder in which anger is primary and aggression is minimal, for which an adjustment disorder with anger should be considered.

CONCLUSIONS AND DIRECTIONS

This chapter reviewed DSM-IV regarding the place of dysfunctional anger and aggression and found it lacking. Ways anger could be maladaptive or dysfunctional were outlined, and eight anger-based disorders were described in terms of

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the intersection of apparent triggers of dysfunctional anger and the presence or absence of aggression. The latter distinction was made to acknowledge that anger can be dysfunctional in the absence of aggression and to highlight aggression as a target of intervention and treatment planning. Any proposals such as those above are clearly preliminary, a place from which to initiate much-needed scientific work. They attempt to legitimize anger as an area worthy of greater clinical inquiry, and to provide, albeit perhaps insufficiently, a framework from which to explore anger-based difficulties. However, more questions arise than have been answered. For example, have critical dimensions been overlooked? Should some criteria or characteristics be weighted more heavily than others? Are there subtypes within groups that should be differentiated? While these are but a few questions that could be raised, it is hoped that the proposed scheme will facilitate research to answer them. Epidemiological studies are needed to establish prevalence rates, and comorbidity studies are necessary to establish how anger disorders are related to other disorders. Outcome studies are needed in which psychological and pharmacological interventions are investigated regarding their potential for anger reduction with specific types of anger disorders. While such research remains to be done, it is hoped that this chapter has provided clinicians with some additional ways of thinking about anger, researchers with clearer definitions of anger disorders from which to initiate needed research, and all readers with a pause for thought about how anger may fit into their clinical thinking.

REFERENCES

1. Averill, J. R. (1982). Anger and Aggression: An Essay on Emotion. New York: Springer-Verlag.
2. Novaco, R. W. (1975). Anger Control. Lexington, MA: Heath.
3. Siegman, A.W., Smith, T. A. (1994). Anger, hostility and the heart. Hillsdale, NJ: Lawrence Erlbaum.
4. Deffenbacher, J. L., Oetting, E. R., Lynch, R. S., Morris, C. D. (1996). The expression of anger and its consequences. Behav Res Ther 34, 575–590.
5. Jacobson, N., Gottman, J.M. (1998). When Men Batter Women. New York: Simon & Schuster.
6. Baumeister, R., Smart, L., Boden, J. (1996). Relation of threatened egotism to violence and aggression: the dark side of high self-esteem. Psychol Rev 103, 5–33
7. Folger, R., Baron, R. (1996). Violence and hostility at work: a model of reactions to perceived injustice. In G.R. Vanden Bos, E. Bulatao (Eds.), Violence on the Job: Identifying Risks and Developing Solutions. Washington: American Psychological Association, pp 51–85.
8. Lochmond, E., DiGiuseppe, R. (1995). How clinicians diagnosis angry clients and what categories they fit. Presented at the 103rd Annual Convention of the American Psychological Association, New York.

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

9. Ekman, P. Davidson, R. (1994). The Nature of Emotions: Fundamental Questions. New York: Oxford University Press.
10. Izard, C. (1977). Human Emotions. New York: Plenum.
11. Blashfield, R. K., Sprock, J., Fuller, A. K. (1990). Suggested guidelines for including or excluding categories in the DSM-IV. Compr Psychiatry 31, 15–19.
12. American Psychiatric Association (1994). Diagnostic and Statistical Manual of Mental Disorders, 4th ed. Washington: American Psychiatric Publishing.
13. Fava, M., Rosenbaum, J. F., McCarthy, M., Pava, J., Steingard, R., Bless, E. (1991). Anger attacks in depressed outpatients and their response to fluoxetine. Psychopharmacol Bull 27, 275–279.
14. Fava, M., Alpert, J., Nierenberg, A. A., Ghaemi, N, O'Sullivan, R., Tedlow, J., Worthington, J., Rosenbaum, J. F. (1996). Fluoxetine treatment of anger attacks: a replication study. Ann Clin Psychiatry 8, 7–10.
15. Coccaro, E. F., Kavoussi, R., Berman, M. E., Lish, J. (1998). Intermittent Explosive Disorder—revised: development, reliability, and validity of research criteria. Compr Psychiatry 39, 368–376.
16. Deffenbacher, J. L. (2000). Characteristics of generally angry individuals. Paper presented at the 34th Annual Convention of the Association for the Advancement of Behavior Therapy, New Orleans.
17. Deffenbacher, J. L. (2000). Characteristics of individuals high in driving anger: Prevention and intervention. presented at the 108th Convention of the American Psychological Association, Washington.

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7

Anger Attacks

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INTRODUCTION

Anger attacks are sudden spells of intense anger that resemble panic attacks but lack the predominant affects of fear and anxiety associated with panic attacks. Anger attacks appear to be a form of dysregulated anger, which is a state in which there is an exaggerated angry and aggressive response to provoking or frustrating events. Thus, an anger attack may be viewed as a combination of predisposition—or some enduring state of vulnerability—and provocation. It is possible that this form of dysregulated anger is a response to the fight/flight reaction expressed cognitively and behaviorally as anger and fight instead of fear and flight (1). On the other hand, the predisposition may be related to underlying mood disorder, with anger and irritability being part of a depressive symptomatology (2), or of a hypomanic/manic/mixed state (3). Irritable mood is a core symptom of major depressive disorder in children and adolescents but is emphasized less as a symptom of depression in adults. Nevertheless, Snaith and Taylor (4) report that 37% of depressed inpatients had moderate to severe outwardly directed irritability, and findings from the Epidemiologic Catchment Area surveys indicate that depression is related to violent behavior

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in samples taken from the community (5). This chapter discusses the development of the concept of anger attacks, the presence of anger attacks in depression and other psychiatric disorders, and the current treatment of anger attacks.

ANGER ATTACKS

In 1990, we (6) reported on a series of patients who experienced sudden outbursts of anger resembling panic attacks. These anger attacks were described by the patients as uncharacteristic behavior that was inappropriate for the situation at hand and was followed by remorse. The outbursts of angry behavior were accompanied by physical features of autonomic activation that included sweating, trembling, tachycardia, and hot flashes. Since the anger attacks improved with antidepressant treatment, they were postulated to be variants of major depression. A self-rating Anger Attacks Questionnaire (7) was subsequently designed as an ad hoc instrument for assessing the presence of anger and was administered to patients already participating in clinical trials. Anger attacks—as defined by the questionnaire—were subsequently found to be significantly more common among depressed outpatients than in healthy volunteers with no known psychiatric history (7).

CRITERIA AND ASSESSMENT OF ANGER ATTACKS

The criteria to define anger attacks (7) adopted by our group, and incorporated in the Anger Attacks Questionnaire, include:

1. Irritability during the previous 6 months.
2. Overreaction to minor annoyances with anger.
3. Occurrence of one or more anger attacks during the previous month.
4. Inappropriate anger and rage directed at others during an anger attack.
5. An additional criterion—modeled in part from the DSM-IV criteria for panic disorder—includes the occurrence of at least four of the following autonomic and/or behavioral features in at least one of the attacks: heart palpitations, flushing, chest tightness or pressure, paresthesias, light-headedness or dizziness, excessive sweating, shortness of breath, shaking or trembling, intense fear or anxiety, feeling out of control, feeling like attacking others, physically and/or verbally attacking others, and throwing or destroying objects.

The Anger Attacks Questionnaire is the self-rating instrument that has been used in almost all the studies in the literature (7). Although it has not been formally validated, the instrument has been translated into several languages and used in a variety of clinical settings (3,8,9).

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ANGER ATTACKS IN UNIPOLAR DEPRESSION

The prevalence rates of anger attacks in U.S. outpatients with major depressive disorder were found to be 44% (56/127) in one sample (10), 39% (64/164) in a subsequent sample (11), and 38% (36/94) in patients with atypical major depression, 28% (21/74) in patients with dysthymia, and 0% (0/38) in screened normal controls (12). On the basis of these findings, the prevalence of anger attacks in depressed populations appears to be ~30–45%. In a European study assessing the prevalence of anger attacks among 103 depressed outpatients by administering the French version of the Anger Attacks Questionnaire (8), the prevalence of anger attacks during the previous month was 47%. A recent study assessed the prevalence of anger attacks in a non-Western depressed population, by administering the Turkish version of the Anger Attacks Questionnaire to 88 medication-free consecutive outpatients diagnosed as having major depressive disorder according to DSM-IV criteria (9). Forty-three (49%) of these patients had reported having anger attacks. Therefore, the prevalence rate of anger attacks in depressed patients seems to hover between 50% and 30%, depending on the population.

PSYCHOLOGICAL AND CLINICAL CORRELATES OF ANGER ATTACKS IN DEPRESSION

In a sample of 127 depressed outpatients, 56 (44%) reported a mean ± SD of 7.4 ± 13.0 anger attacks per month when assessed with the Anger Attacks Questionnaire (10). The most frequently reported autonomic arousal symptoms and behavioral features that occurred during the anger attacks were tachycardia, hot flashes, the feeling of being out of control, and the feeling of wanting to attack others. While 63% of the patients with anger attacks reported physical or verbal attacks directed at others, only 30% actually threw objects or destroyed property. After the attacks, guilt or regret was almost universal (93%) (Fig. 1) (10). On the Symptom Questionnaire, measuring global psychological distress, depressed patients with anger attacks scored higher on the anxiety, somatization, and hostility scales than depressed patients without anger attacks. No significant difference was noted on the depression scale of the Symptom Questionnaire between patients with and without anger attacks, a finding that corresponded with Hamilton Depression Rating Scale (HAM-D) scores (Fig. 2) (10). High scores on the anxiety scale may be a reflection of the autonomic symptoms experienced or a possible link between dysregulated anger and anxiety (see below).

In a European study among 103 depressed outpatients (8), the most frequently reported symptoms among patients with anger attacks were feelings of panic (85%), tachycardia (84%), and feeling out of control (81%).

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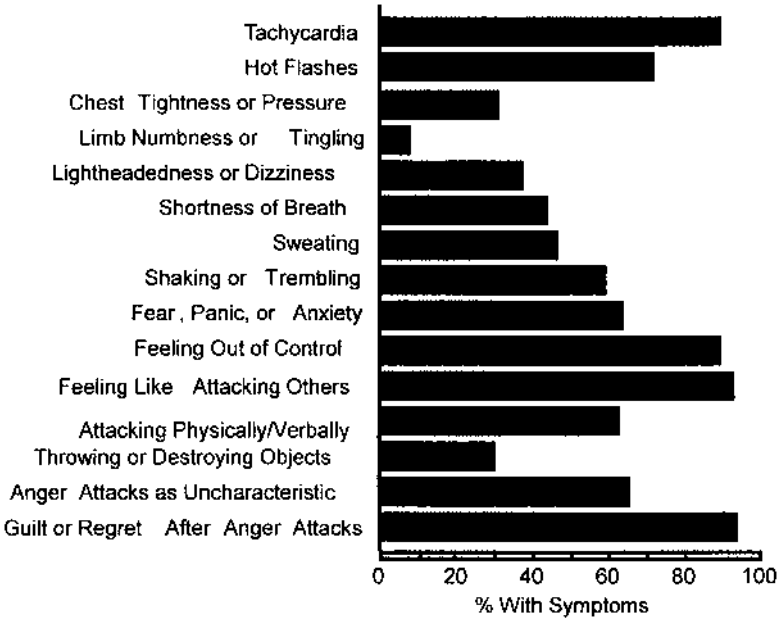

FIGURE 1 Frequency of autonomic arousal symptoms and behavioral outbursts during anger attacks (N = 56). (From Ref. 10.)

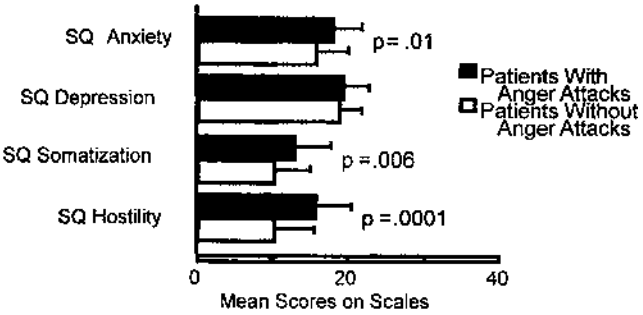

FIGURE 2 Scores of depressed patients with and without anger attacks on symptom questionnaire. (From Ref. 10.)

In a study of Turkish depressed patients, patients with anger attacks were significantly more depressed and anxious than patients without anger attacks, with anger-out and trait anger measures being also significantly higher in depressed patients with anger attacks than patients without anger attacks (9).

ANGER ATTACKS IN DEPRESSION AND PERSONALITY DISORDERS

Irritability and anger in depressed patients are sometimes viewed as evidence for a personality disorder, and the self-rated Personality Diagnostic Questionnaire–Revised (PDQ-R) was used in one of our studies to assess comorbid personality disorders in depressed patients with and without anger attacks. Depressed patients with anger attacks were more likely to meet criteria for histrionic, narcissistic, borderline, and antisocial personality disorders than depressed patients without anger attacks (10). In another sample of 306 depressed outpatients with major depressive disorder, the clinician-rated SCID-II (Structured Clinical Interview for Personality Disorders) and the Anger Attacks Questionnaire were used for evaluation, and depressed patients with anger attacks were significantly more likely to meet criteria for dependent, avoidant, narcissistic, borderline, and antisocial personality disorder than depressed patients without anger attacks (13).

ANGER ATTACKS AND BIPOLAR DISORDER

Anger attacks and irritability have been suggested as indicators of a bipolar depressive episode in adults (3). Perlis et al. (14) explored anger attacks in a sample of depressed (unipolar and bipolar) patients and found higher rates of anger attacks among bipolars. Tedlow et al. (13) reported that switches into mania or hypomania are no more frequent in unipolar patients with anger attacks than in unipolar patients without anger attacks. This study suggests that the presence of anger attacks in depression is not necessarily indicative of a bipolar diathesis.

ANGER ATTACKS IN PANIC DISORDER AND OTHER ANXIETY DISORDERS

In a two-site study by Gould et al. (15), the prevalence of anger attacks in patients with panic disorder was ~33% at both sites. Anger attacks were not unique to panic disorder, and similar rates emerged for patients with other anxiety disorders. Depressed patients were twice as likely to report anger attacks as patients with anxiety disorder. Additionally, anxiety disorder patients with anger attacks were significantly more depressed than anxiety disorder patients without anger attacks. In a study assessing the prevalence of anger attacks in a European de-

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pressed population (8), the most frequently reported symptoms among patients with anger attacks were feelings of panic (85%). The occurrence of anger attacks was also significantly associated with intensity of loss of control and history of panic attacks, while there was no significant association with age, gender, severity of depression, and anxiety; or history of suicidal attempts. The relationship among panic disorder, depression, and anger attacks has also been supported by a study from our group, which showed that, while there were no significant differences in rates of lifetime comorbid anxiety, eating, and substance use between depressed patients with and without anger attacks, 8% of depressed patients with anger attacks met criteria for current comorbid panic disorder compared with 3% of depressed patients without anger attacks, with a trend toward a statistically significant difference (13).

A recent study (16) employed the panicogenic agent sodium lactate to examine the response of perpetrators of domestic violence to this challenge. Using a double-blind, placebo-controlled design, comparing a select group of subjects who perpetrate acts of domestic violence and two nonviolent subject groups, perpetrators evidenced more lactate-induced rage and panic and showed greater changes in speech, breathing, and motor activity than did nonviolent control subjects. One may interpret these results as suggesting that the lactate-induced physiologic arousal might have induced/triggered a fight/flight reaction expressed cognitively and behaviorally as anger and fight instead of fear and flight (1).

ANGER ATTACKS IN OTHER PSYCHIATRIC DISORDERS

Anger attacks have also been reported in women with eating disorders (17). Of 132 female patients with anorexia nervosa and/or bulimia nervosa and 39 normal female volunteers, 41 (31%) of eating-disorder women met the criteria for anger attacks compared with four (10%) of the control subjects. Severely bulimic patients reported the highest prevalence of anger attacks, and women with eating disorders with anger attacks had more depressive symptoms than women with eating disorders without anger attacks (17). These studies suggest that the severity of depressive symptoms may be a significant predictor of the presence of anger attacks among patients with eating and anxiety disorders.

A study by Mammen et al. (18) examined the prevalence and correlates of anger attacks in a psychiatric clinic for women who were either pregnant or up to 18 months postpartum. Thirty (60%) of 50 patients reported anger attacks. Of those with anger attacks, 76.7% worried about them, and 73.3% had tried to prevent them. Compared with women without anger attacks, those with anger attacks were significantly more likely to report higher state and trait anger ($P < .001$), have a diagnosis of unipolar depression ($P < .01$), report more aggression directed at immediate family, and avoid their children.

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SEROTONERGIC FUNCTION AND ANGER ATTACKS

Alterations in serotonergic function have been reported in both depression and aggressive behavior. Coccaro et al. (19) evaluated central serotonergic function in 45 male patients with clearly defined major affective disorder (N = 25) and/or personality disorder (N = 20), and in 18 control patients. A single dose of 60 mg p.o. fenfluramine hydrochloride, a serotonin-releasing/uptake-inhibiting agent, resulted in a reduced prolactin response in both groups of patients compared with the response in controls. Additionally, reduced prolactin response was also correlated with high ratings of impulsive aggression in patients with personality disorder (19).

We (20) conducted a study to determine whether a subset of depressed patients with anger attacks had a different prolactin response to thyrotropin-releasing hormone (TRH) stimulation from that of depressed patients without anger attacks. TRH infusion was administered to 25 patients with major depression, 12 of whom reported having anger attacks; the depressed patients with anger attacks had a blunted prolactin response to TRH stimulation compared with depressed patients without anger attacks. Twenty-two subjects participated in the TRH test again after 8 weeks of fluoxetine (20 mg/day) treatment, and the drug significantly increased prolactin response to TRH in patients with anger attacks (20). These results suggest that patients with major depression and anger attacks may have a relatively greater serotonergic dysregulation than depressed patients without such attacks.

In a subsequent study (21), we compared the prolactin response to fenfluramine challenge, as an indirect measure of central serotonergic function, in depressed patients with and without anger attacks. We recruited 37 outpatients (22 men and 15 women; mean age: 39.5 $\pm$ 10.5) with DSM-III-R major depressive disorder, diagnosed with the SCID-P. All patients received a single-blind placebo challenge followed by a fenfluramine challenge (60 mg p.o.) the next day. There were no significant differences in age, gender, fenfluramine, or norfenfluramine blood levels between depressed patients with (n = 17) and without (n = 20) anger attacks. Depressed patients with anger attacks showed a significantly blunted prolactin response to fenfluramine challenge compared to patients without anger attacks, supporting the hypothesis that depressed patients with anger attacks may have a relatively greater serotonergic dysregulation than depressed patients without these attacks.

DRUG TREATMENT OF ANGER ATTACKS

A number of studies from our group have evaluated the treatment of anger attacks with antidepressants, and anger attacks subsided in 53–71% of depressed outpatients treated with the selective serotonin reuptake inhibitors (SSRIs) fluoxetine

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or sertraline or the tricyclic antidepressant imipramine. In an open-label study (10), 44% of depressed outpatients reported anger attacks at baseline and demonstrated significantly higher scores on measures of anxiety, somatization, and state and trait hostility than did the subjects without anger attacks. After 8 weeks of treatment with a fixed dose (20 mg/day) of fluoxetine, significant reductions in these measures occurred and the anger attacks disappeared in 71% (24/34) of patients who previously reported them. In a subsequent open-label study (11), 64% (41/64) of the depressed patients who reported anger attacks at baseline reported no attacks after fluoxetine treatment. The change in the severity of depression after fluoxetine treatment in patients both with and without anger attacks was comparable whether measured by the 17-item HAM-D, the eight-item HAM-D, or the Clinical Global Impression-Severity of Illness (CGI-S) scale. In a double-blind, placebo-controlled study (12), the efficacy of sertraline (up to 200 mg/day) versus imipramine (up to 300 mg/day) treatment of anger attacks was compared in 168 outpatients with diagnoses of atypical depression or primary dysthymia. Anger attacks ceased in 53% (9/17) of patients taking sertraline, 57% (12/21) of patients taking imipramine, and 37% (7/19) of the placebo group. These treatment studies suggest that antidepressants are effective treatment for anger attacks.

Patients who fail to meet the full criteria for anger attacks before antidepressant treatment occasionally do so after antidepressant treatment. The emergence of anger attacks in depressed patients after fluoxetine treatment has been reported to be 6–7% (10,11), and after sertraline treatment as 8%, after imipramine treatment as 10%, and after placebo as 20% (12). Large placebo-controlled studies comparing SSRIs with relatively noradrenergic tricyclic antidepressants may help to determine whether depressed patients with anger attacks show a distinct response to specific drug treatment.

Anecdotally, when depressed patients with anger attacks do not respond to an SSRI, clinicians find useful augmentation of the SSRI with drugs such buspirone, atypical antipsychotics, and anticonvulsants (valproic acid in particular). However, no studies in resistant depressed patients with anger attacks have been conducted.

Finally, the presence of anger attacks does not predict poorer response to antidepressant treatment, as suggested by a study from our group with the SSRI fluoxetine (22).

CONCLUSION

Anger attacks appears to be relatively common among psychiatric populations, in particular among patients suffering from unipolar depression. Severity of depression seems to predict the presence of anger attacks across a variety of psychiatric populations. Panic attacks and panic disorder may also be somewhat related

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to the occurrence of this clinical phenomenon. The presence of anger attacks in depressed patients appears to be associated with a relatively greater serotonergic dysfunction. The presence of anger attacks in patients with depression predicts a good response to antidepressant treatment, particularly treatment with SSRIs. Finally, the assumption that antidepressant treatment may mobilize anger attacks in depressed patients is challenged and calls for additional studies.

REFERENCES

1. Fava M, Anderson K, Rosenbaum JF. Are thymoleptic-responsive "anger attacks" a discrete clinical syndrome? Psychosomatics 1993; 34:350–355.
2. Riley WT, Treiber FA, Woods MG. Anger and hostility in depression. J Nerv Ment Dis 1989; 177:668–674.
3. Fava M, Rosenbaum JF. Anger attacks in depression. Depression Anxiety 1997; 6: 1–5.
4. Snaith RP, Taylor CM. Irritability: definition, assessment and associated factors. Br J Psychiatry 1985; 147:127–136.
5. Swanson JW, Holzer CE III, Ganju VK, et al. Violence and psychiatric disorder in the community: evidence from the Epidemiologic Catchment Area surveys. Hosp Community Psychiatry 1990; 41:761–770.
6. Fava M, Anderson K, Rosenbaum JF. "Anger attacks": possible variants of panic and major depressive disorders. Am J Psychiatry 1990; 147:867–870.
7. Fava M, Rosenbaum JF, McCarthy M, et al. Anger attacks in depressed outpatients and their response to fluoxetine. Psychopharmacol Bull 1991; 27:275–279.
8. Morand P, Thomas G, Bungener C, Ferreri M, Jouvent R. Fava's Anger Attacks Questionnaire: evaluation of the French version in depressed patients. Eur Psychiatry 1998; 13:41–45.
9. Sayar K, Guzelhan Y, Solmaz M, Ozer OA, Ozturk M, Acar B, Arikan M. Anger attacks in depressed Turkish outpatients. Ann Clin Psychiatry 2000; 12(4):213–218.
10. Fava M, Rosenbaum JF, Pava JA, et al. Anger attacks in unipolar depression, part I. Clinical correlates and response to fluoxetine treatment. Am J Psychiatry 1993; 150:1158–1163.
11. Fava M, Alpert J, Nierenberg AA, et al. Fluoxetine treatment of anger attacks: a replication study. Ann Clin Psychiatry 1996; 8:7–10.
12. Fava M, Nierenberg AA, Quitkin FM, et al. A preliminary study on the efficacy of sertraline and imipramine on anger attacks in atypical depression and dysthymia. Psychopharmacol Bull 1997; 33:101–103.
13. Tedlow J, Leslie V, Keefe BR, Alpert J, Nierenberg AA, Rosenbaum JF, Fava M. Axis I and axis II disorder comorbidity in unipolar depression with anger attacks. J Affect Disord 1999; 52(1–3):217–223.
14. Perlis RH, Smoller JW, Fava M, Rosenbaum JF, Nierenberg AA, Sachs GS. The prevalence and clinical correlates of anger attacks during depressive episodes in bipolar disorder. Journal of Affect Disord, in press.
15. Gould RA, Ball S, Kaspi SP, et al. Prevalence and correlates of anger attacks: a two site study. J Affect Disord 1996; 39:31–38.

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16. George DT, Hibbeln JR, Ragan PW, Umhau JC, Phillips MJ, Doty L, Hommer D, Rawlings RR. Lactate-induced rage and panic in a select group of subjects who perpetrate acts of domestic violence. Biol Psychiatry 2000; 47(9):804–812.
17. Fava M, Rappe SM, West J, et al. Anger attacks in eating disorders. Psychiatry Res 1995; 56:205–212.
18. Mammen OK, Shear MK, Pilkonis PA, Kolko DJ, Thase ME, Greeno CG. Anger attacks: correlates and significance of an underrecognized symptom. J Clin Psychiatry 1999; 60(9):633–642.
19. Coccaro EF, Siever LJ, Klar HM, et al. Serotonergic studies in patients with affective and personality disorders: correlates with suicidal and impulsive aggressive behavior. Arch Gen Psychiatry 1989; 46:587–599.
20. Rosenbaum JF, Fava M, Pava JA, et al. Anger attacks in unipolar depression, part II. Neuroendocrine correlates and changes following fluoxetine treatment. Am J Psychiatry 1993; 150:1164–1168.
21. Fava M, Vuolo RD, Wright EC, Nierenberg AA, Alpert JE, Rosenbaum JF. Fenfluramine challenge in unipolar depression with and without anger attacks. Psychiatry Res 2000; 94(1):9–18.
22. Fava M, Uebelacker LA, Alpert JE, Nierenberg AA, Pava JA, Rosenbaum JF. Major depressive subtypes and treatment response. Biol Psychiatry 1997; 42(7):568–576.

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8

Impulsive Aggression

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INTRODUCTION

Clinicians and behavioral scientists are increasingly recognizing impulsive aggression as one of several different types of aggressive behaviors with correspondingly different approaches for effective treatment and management. The realization and appreciation that impulsive aggression is essentially distinct, even if overlapping, in occurrence with other types of aggression, is one of the most important recent developments toward a reasoned approach to research on aggressive behavior on the one hand and clinical management on the other. Development and conceptualization of impulsive aggression can and should be framed as both a categorical condition and as a psychological dimension.

Current nosologies of mental disorders, developed to serve research and clinical purposes, evolved from the categorical conceptualizations which at least

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suggest a phenomenon of impulsive aggression that, in individual cases, is sufficiently pronounced and dysfunctional to attain clinical importance. The closest approximation to impulsive aggression in the first Diagnostic and Statistical Manual of the American Psychiatric Association in 1952 (1) was *emotionally unstable personality*, formally designated as "psychopathic personality with emotional instability." The essential emotional instability of this disorder anticipated the present borderline personality disorder and its possible association with impulsive aggression. A core defect in this disorder was the *poor ability to control* "hostility, guilt, and anxiety." The next edition, DSM-II in 1968 (2), dropped this concept of general emotional instability and poor self-control and introduced "explosive personality," also then known as "epileptoid personality disorder" and the forerunner of DSM-III's "intermittent explosive disorder" (3), wherein the aggressive outbursts are strikingly out of character for the individual. DSM-II listed impulsivity as a feature of antisocial personality (2). Likewise, the third edition, DSM-III (1980) (3), did not identify a disorder of impulsive aggression, though several disorders were relevant for differential diagnostic considerations: intermittent explosive disorder (IED) (but lacking "generalized impulsivity or aggressiveness between episodes" [pp 295–297]), antisocial personality disorder (APD) one manifestation of which was "irritability and aggressiveness as indicated by repeated physical fights or assault" (p 321), borderline personality disorder (BPD) (pp 321–323) with "inappropriate, intense anger or lack of control of anger" (p 322), and "organic personality syndrome" (pp 118–120), manifestations of which can include "explosive temper outbursts" and impaired impulse control (pp 119–120). The third edition Revised (DSM-III-R) (4) remained unchanged on these specific points.

In DSM-IV (1994) (5) and DSM-IV-TR (2000) (6), the manifestations of impulsivity and aggressive behavior for APD and BPD were carried over, but some significant nosological changes were made in the organic condition and IED. The organic condition is now termed "personality change due to a general medical condition" (6:187–190), and relevant subtypes include disinhibited (with poor impulse control), aggressive, and combined (with disinhibition and aggression).

Today, generalized impulsivity is no longer an exclusionary factor for IED. In contrast, the current description of associated features includes, "signs of generalized impulsivity or aggressiveness . . . between explosive episodes." Moreover, some of the potential biological correlates of impulsivity (e.g., altered serotonicity and CNS electrophysiological measures) are thought to be associated with impulsive aggression. IED-Revised, proposed by Coccaro and colleagues (7; also see Chap. 9 by Coccaro, this volume), with demonstrated reliability and validity, is even more inclusive and allows for comorbid generalized impulsivity. Conceivably, many patients with impulsive aggression would today be diagnosed with IED, since the former does not exist as a separate diagnostic category in the DSM.

Although the ICD-10 (8) includes neither IED nor impulsive aggression,

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the emotionally unstable personality disorder, impulsive type, corresponds to impulsive aggression and is distinct from the borderline type which in turn is the counterpart of BPD in DSM-III, -III-R, -IV, and IV-TR. "The essential character traits [of the ICD-10 diagnosis of impulsive type of emotionally unstable personality disorder] are emotional instability and deficient impulsive control. Outbursts of violent and threatening behavior are frequent, especially in response to criticisms from others" (p 215).

To place the construct of impulsive aggression in relevant current clinical and research contexts, this chapter is organized into six sections: 1) Construct validity of impulsive aggression based on research findings; 2) Defining and measuring impulsive aggression as a disorder; 3) Delineation of impulsive aggression from other disorders; 4) Assessment; 5) Treatment considerations; and 6) Social and legal issues. This overview is selective and not exhaustive. The first two sections, addressing the conceptual validity and definition of impulsive aggression, can be understood within the framework of a general systems model. Barratt (9) and Barratt and Slaughter (10) modified a model proposed by Ashby (11) and incorporated psychiatric concepts emphasized by Lazare (12). The resulting discipline neutral—but not atheoretical—model is a closed feedback system extending over time from the genetic contribution at conception through birth and over an individual's life-span. The four interacting categories for organizing concepts and measurable dimensions are *biological*, including molecular, cellular, and systemic (e.g., nervous system) contributions; *behavioral*, such as simple reflexes to complex socially defined acts; *environmental*, ranging from physical energy (e.g., nutrition), familial, and cultural influences; and *cognitive* (e.g., perception and cognition). Within each of these four categories, measurable dimensions are identified in developing and refining the concept of impulsive aggression. A current conceptual counterpart of this model is the new subdiscipline of social cognitive neuroscience (13,14). Within this general systems model we will compare our concept of impulsive aggression and its supportive empirical findings with the approach recommended by Robins and Guze (15) to establish the validity of a mental disorder, an approach with which they distinguished chronic schizophrenia from acute schizophrenia before the DSM (viz., DSM-III) introduced a criterion of chronicity for the diagnosis of schizophrenia.

CONSTRUCT VALIDITY OF IMPULSIVE AGGRESSION BASED ON RESEARCH FINDINGS: ETIOLOGICAL FACTORS

Impulsivity

For several decades investigators have differentiated and contrasted "impulsive" or reactive from nonimpulsive, planned, or proactive aggression (16–19), a dis-

tinction supported by both construct and predictive validity studies (20–23). This distinction has resulted in a better understanding of the role of aggression in selected applied clinical problems. For example, recidivism has been shown to be related to impulsive aggression.

Coccaro et al. (19) defined impulsive aggression using the Buss-Durkee Hostility Inventory (24) and the Barratt Impulsivity Scale (25) to arrive at a distinction between "irritable-impulsivity" and "aggression-assault" components of aggressive acts. Irritability-impulsivity requires a lower threshold for a response to be perceived as noxious, corresponding to our concept of impulsive aggression. Other investigators (26,27), using different techniques, arrived at similar definitions of impulsive aggression. Thus, the construct of impulsive aggression is well supported in the literature.

Cortical and Electrophysiological Studies

An experiment by Barratt and colleagues (28) demonstrated that subjects showing impulsive aggression, in contrast to those manifesting premeditated aggression, differed on selected self-report personality traits, neurophysiological measures, and cognitive psychophysiological measures, representing respectfully the behavioral, biological, and cognitive categories of his general systems model. The study included two groups of prison inmates and a control group of noninmates. All inmates satisfied DSM-III-R criteria for antisocial personality disorder (APD). Using a structured interview, all inmates were classified into impulsive aggressive and nonimpulsive aggressive groups. After screening with the Psychiatric Diagnostic Interview-Revised (PDI-R) (29), anyone with an axis I disorder was excluded.

The results indicated that impulsive aggressive inmates, in contrast to nonimpulsive aggressive inmates, were significantly deficient in verbal abilities (e.g., reading skills and verbal memory), had lower scores on a personality scale of emotional "warmth," higher scores on gregariousness, and temporal and parietal lobe differences in event-related cortical potentials (ERPs) recorded while subjects performed visual information-processing tasks.

The differences in cognitive and biological measures between inmates as a whole and noninmate controls on these measures were consistent with what has been reported in the literature (30). It is important to note that inmates with APD could be separated into impulsive and premeditated aggressive groups using a convergence of neuropsychological and cognitive psychophysiological measures. Others (26,27) have reported lower verbal abilities among impulsively aggressive subjects and differences in ERPs related to aggression and psychopathy (30) and impulsivity (31). Consistent with Barratt's study of prisoners, college students (32) and psychiatric inpatients (33) who are impulsively aggressive also have prolonged latencies and diminished P300 amplitudes in ERPs recorded dur-

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ing the performance of selected sensory discrimination tests (e.g., oddball tasks). In comparing studies, it is important to recognize that these findings are not due to criminality per se (34,35).

Imaging studies, using pneumoencephalogram (PEG), CT scan, and/or fMRI, summarized elsewhere (36), have shown significant anatomical and functional abnormalities in frontal and temporal lobes of neurologic patients who manifest impulsive aggression. Consistent with these findings, neural circuitry has been proposed which includes the prefrontal cortex, the anterior cingulate cortex, insular cortex, the amygdala, the hippocampus, the hypothalamus, and the ventral striatum in the activation, control, and modulation of impulsive aggression (37). Frontostriatal functional impairment with consequent impulsivity has also been discussed as an adverse outcome of drug abuse (38).

Genetic Studies

Many disorders (e.g., diabetes and hypertension) can result from an interaction of genetic and environmental factors. Some well-recognized illnesses and impairments are caused by physical or emotional trauma without a genetic predisposition. Nonetheless, clear genetic etiologic agency itself is a strong indication of a discrete disorder. Impulsive aggression, as specifically defined here, has not been studied in terms of intergenerational familial inheritance patterns or specific gene defects. However, genetic influence may variably affect specific components of impulsive aggression including impulsivity, the personality trait of anger, cognitive ability to process information, and these may be related to impulsive aggression at a secondary level. Numerous studies suggest a genetic component to impulsivity and aggression in humans, and animal models have proven useful in identifying specific molecular genetic processes that may have relevance for the pathogenesis of impulsive aggression in humans.

Twin Studies

Genetic predispositions have been demonstrated for verbal skills (39), cognitive psychophysiological measures (40), and self-report psychometric measures (41), constructs that are closely associated with impulsive aggression. Selected studies suggest a genetic component to the etiology of Antisocial Personality Disorder (APD) [see, e.g., Raine 1993 (42)], which is often attended by impulsive aggression (28), though several studies tracked patterns of criminality without actual diagnosis of APD. Other studies suggest a genetic predisposition to anger and hostility (43) and impulsivity (42,44).

Interesting results were reported from two twin studies of 182 male monozygotic twin pairs and 118 dizygotic taken from the Vietnam Era Twin Registry (45,46). Results of the first study demonstrated significant hereditability for indirect assault, irritability, and verbal assault respectively, with environmental in-

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fluences contributing to 53–72 % of the variance. In the second study (46), irritability and impulsivity showed substantial genetic and environmental mediation, whereas assault and impulsivity did not. Taking into account other studies that pointed to a genetic component for impulsivity (42,44), this finding raises the possibility that impulsivity is substantially genetically mediated, but other nongenetic factors are required for it to result in impulsive aggression (also see Bergeman and Montpetit, Chap. 2, this volume).

Molecular Genetics

Lesch and Merschdorf (47) recently reviewed leading research into the molecular genetic cause for impulsive and aggressive behaviors in animal models. As will be discussed, several central neurotransmitters have been implicated in the etiology of impulsive aggression, but serotonin (5-HT) has received the most investigative attention. Of the various lines of evidence that suggest a pivotal role for the serotonergic gene in the expression of impulsive aggression, not the least derive from studies of inbred and knockout strains of mice, and studies of rhesus monkeys. In rhesus monkeys a gene-linked polymorphic region (5-HTTLPR) modulates the transcription of the 5-HT transporter (5-HTT), which occurs in short (s) and long (l) alleles. The s allele in Rhesus monkeys results in lower CSF 5-HIAA (reflecting lower CNS serotonicity) and increased impulsive aggression. Despite their in-depth clarification of evidence for molecular genetic mechanisms in impulsive aggression, these authors stress the multifactorial etiology of the phenomenon and the importance of psychosocial factors in particular.

Psychosocial Studies

Another approach to sorting out the environmental from genetic influences is to investigate whether specific environmental influences are associated with impulsive aggression. One of the most consistent findings in this regard is that early subjection to severe parental punishment or physical abuse is associated with aggressive behavior among boys (47–49) and later aggression (50). A rather wide range of studies support social intergenerational transmission of aggressive behaviors (36,51–55), including "impulsive aggression."

Straus and Mouradian (56), in a study of 933 mothers of children 2–14 years of age, found that corporal punishment, especially if impulsively inflicted, was strongly associated with antisocial and impulsive behaviors of the child. Others (57), examining the influence of different types of violent exposures, emphasize that behavioral outcomes are heterogeneous and a function of the child's level of development and psychological and social assets. In controlled laboratory studies using behavioral methods, investigators have demonstrated that social cues and manifestations of the victim's suffering can reinforce aggressive behaviors and enhance their impulsive qualities (16). Taken together, research suggests

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both genetic and psychosocial contributions to the etiology of impulsive aggression are present, though congenital, traumatic, and toxic factors, including neurotoxic effects of commonly abused drugs (38), have to be considered as having an interactive influence.

Ultimately the most promising approach to studying etiology will integrate the approaches of familial genetics, molecular genetics, and psychosocial factors, including accurate clinical assessments. For now, genetic predisposition appears to be an important etiologic factor, particularly for the dysregulation of impulsive control, but psychosocial and other internal/external factors also contribute.

Neurotransmitters

Of the more than 50 molecules identified as neurotransmitters, the monoamines, viz, serotonin (5-HT), norepinephrine (NE), and dopamine (DA), have been most frequently implicated in aggressive behavior (58). Of these three, serotonin has been the most studied and discussed with regard to poor impulse control and impulsive aggression. Low CSF-5-HIAA, thought to reflect low CNS serotonicity, has been associated with aggressive behavior in military men (59); violent criminal acts, homicide, or arson, characterized as impulsive (60,61). Diminished 5-HIAA in the brain and CSF of suicidal and impulsively aggressive individuals is "one of the most replicated findings in psychobiology" (47). Consistent with the hypothesis that deficient serotonicity results in poor control over aggressive impulses, Kent and colleagues (62), using platelet serotonin uptake as a peripheral indication of CNS serotonicity, found diminished platelet serotonin uptake in aggressive subjects who showed general impulsivity in comparison with aggressive subjects who did not demonstrate high impulsivity on the Barratt Impulsivity Scale. Major serotonergic tracts project onto the prefrontal cortex (37), an area of the brain of central importance in regulating emotions and behavior as noted.

CNS dopamine activity is, like serotonicity, thought to be inversely associated with aggressive behavior in humans (58). For example, violent criminal recidivism was found to be associated with low CSF HVA (a metabolite of dopamine) (63). Research on animal models suggests that chronic consumption of commonly abused drugs leads to cortical dopaminergic hypofunction (38). Reduced dopamine transmission in the prefrontal cortex is thought to impair modulation of subcortical dopamine function, resulting in behaviorally poor inhibition of drug-seeking behavior, proneness to stress-precipitated relapse to drug dependence, and generally increased impulsivity. Neurobiological research on drug abuse and addiction suggests two important possibilities: First, dopamine, in addition to serotonin, may play an important role in the neurophysiology of impulsivity. Second, the possibility of chronic drug abuse–promoting impulsivity must be added to genetic and psychosocial factors in the pathogenesis of impulsivity and, therefore, impulsive aggression. Norepinephrine, a neurotransmitter related

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to arousal, may be directly associated with aggression (59,64), but this finding is not consistently reported. The serotonin system is the most widely distributed neurotransmitter system in the brain. This system projects to a variety of brain structures including cortex, amygdala, and hippocampus, and regulates the activity of several other transmitter systems (47) and may be related to dopamine turnover (65). Thus, serotonin's apparently significant role in impulsive aggression could be related to its mediating effects on other neurotransmitter systems. As Gabbard (66) nicely summarized, a convergence of evidence, using both animal and human models, increasingly demonstrates the dynamic interactions of neurobiological and psychosocial factors in the pathogenesis of mental disorders and abnormal behavioral patterns. No doubt future advances in understanding these interactions will integrate relevant categories of information in a manner consistent with the general systems model.

DEFINING AND MEASURING IMPULSIVE AGGRESSION AS A DISORDER

Impulsive aggression is one of three categories of human aggression proposed by Barratt (9), the other two being medically related (secondary) and premeditated aggression. *Medically related or secondary aggression* is symptomatic of a primary medical disorder including neurological disorders such as head injury or psychiatric disorders such as schizophrenia. Typically, efficacious treatment of the primary disorder results in control of the associated aggressive behavior. *Premeditated aggression*, intended to benefit the actor, is best understood within a social context using principles from learning theory (67). Even though these three types of aggression can co-occur (28), the distinction is clinically important for accurate assessment and effective treatment.

Impulsive aggression is characterized by a "hair-trigger" response to a stimulus which results in a sudden agitated state that lasts from a few minutes to several hours (68); the agitation builds to a crescendo and culminates in an aggressive act. During this state, interpersonal communication appears inefficient, and recall of the related events may be poor. Impulsive and premeditated aggression can co-occur with each other and with other psychiatric disorders.

Phenomenologically impulsive aggression, in contrast to premeditated aggression, is spontaneous, unplanned, and lacking in self-control. Outbursts are explosive and driven more by impulse or erupting affect than by acquisitive or self-promoting goals. Impulsive and premeditated aggression have also been labeled as *reactive* versus *proactive* aggression (69), *affective* versus *predatory* (70), *hostile* versus *instrumental* (23), and *expressive* versus *instrumental* aggression of behavioristic (71) and criminological categorizations (72). The distinction between impulsive and nonimpulsive aggression is supported by both construct and validity studies (16–23). Research by Barratt and colleagues demonstrates that impulsive and premeditated aggression differ not only phenomenologically

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but also in neurophysiological, cognitive, psychophysiological, and selected personality trait measures (28). Moreover, among aggressive offenders, the higher the level of impulsiveness, especially motor impulsiveness, the greater the *variety* of criminal acts committed by the prisoners (73,74). This is consistent with studies that found elevated impulsiveness to be associated with disorders marked by poor control of self-stimulating impulses such as alcohol abuse, binge eating, and heavy cigarette smoking (75) and with treatment prognosis for cocaine consumption (76).

A few observations about the nature of impulsivity contribute to the understanding of impulsive aggression. The Barratt Impulsivity Scale, now in its 11th revision (25), has been translated into many languages and has been found to be internally consistent across subcultures (77,78). An adolescent version of the BIS-II, developed by Fossati (79) in Italy, was shown to have good internal consistency when administered to male and female Italian high school students. Barratt (80,81) identified three subfactors of impulsivity: 1) motor impulsiveness, i.e., acting without thinking; 2) cognitive impulsiveness, i.e., making quick decisions; and 3) nonplanning impulsiveness (showing little regard for future planning). Although different populations show different subtest patterns, prisoners who manifest impulsive aggression demonstrate elevation in all three of these subfactors (82).

Barratt (9) proposed that impulsive aggression is primarily related to the two personality *traits* of impulsiveness and anger/hostility, respectively. In a study of impulsivity involving inmates and matched controls, Barratt et al. (28) found that prisoners had higher levels of anger and impulsiveness than nonprisoners, but these traits did not distinguish those prisoners with impulsive aggression from those with premeditated aggression. All of the inmates met the criteria for APD which implicates impulsivity. Having concluded that these personality traits are necessary but not sufficient to cause impulsive aggression (10), Barratt et al. (83) recently demonstrated that both impulsivity and anger/hostility are correlated with impulsive aggression but not premeditated aggression, further suggesting that the 1997 results were related to all inmates having APD.

Herpertz and Sass (84) hypothesize that impulsiveness is a quality of drive that is associated with both temperament and control mechanisms. Consistent with this suggestion, aggression itself, Barratt explains, involves two components: 1) the aggressive drive, and 2) a corresponding control system (85). Thus, aggressive behaviors, and especially impulsive aggression, could be understood as the vectorial product of these opposing forces.

RECOGNITION OF A CLINICAL DISORDER

In North America the most commonly diagnosed mental disorders, as a rule, are those found in the current edition of the Diagnostic and Statistical Manual of Mental Disorders [DSM-IV-TR, 2000 (6)]. The manual is considered authorita-

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tive because experts in a respective field of psychopathology developed the criteria for disorders in their particular areas of expertise. Close adherence to criteria in this manual is presumed to improve diagnostic reliability. Administrative requirements (government, managed care, insurance companies) insist on conformity of usage. Even while realizing that diagnostic criteria and specific disorders are subject to revision over time, most clinicians' operational assumption typically is: "If a disorder is in the DSM, it is valid; if not, it is suspect."

The general importance of and widespread confidence in the DSM, however, should not becloud the process by which disorders rise to official recognition. The panel of experts for a given class of potential disorders, bringing their knowledge, research, and the known literature to the table, arrives at a consensus. This is basically no different from classifying disorders by other medical disciplines. It takes a consensus of experts, for example, to establish threshold blood pressures for official recognition of hypertension. Clearly the experts have examined the research and available clinical reports and must apply some guiding principles for recognizing a disorder and its diagnostic criteria. Clinicians and researchers in the field who then rely these on diagnostic manuals are typically not privy to specific guidelines used to arrive at a specific mental disorder, which can vary from one disorder or group of disorders to another.

In considering whether the construct of impulsive aggression should be recognized as a psychiatric disorder, we next apply the five criteria outlined by Robins and Guze (15) for establishing the validity of a psychiatric disorder: 1) *Clinical description* including symptoms and "other items" that serve to "define" the clinical picture more accurately; 2) *Laboratory studies* that consist of biological measures as well as reliable psychological test results; 3) *Delimitation from other disorders* whereby exclusionary criteria of other disorders can be applied to ensure the study group is optimally homologous in terms of the proposed disorder; 4) *Follow-up study* to determine if the patients develop another disorder that could explain the original findings; and 5) *Family study* with genetic investigation for familial inheritance. These criteria have played a significant role in constructing current nosological schemes in psychiatry.

In applying these five criteria, impulsive aggression clearly has a clinical description and specific confirmatory laboratory studies. Despite strong comorbidities and dimensional features, impulsive aggression can be delimited from other disorders as will be demonstrated in the next section of this chapter, though arguably less so from the DSM-IV-R definition of IED which is now expanded to include both episodic and impulsive aggression. Genetic studies, though not definitive, support a genetic contribution. In classifying impulsive aggression as a disorder using Robins and Guze's five criteria, the need for follow-up studies is admittedly a weak link. Follow-up studies of impulsively aggressive patients to determine outcome have provided external validation for impulsive aggression (17,20,22,23). However, these studies did not follow patients over an extended

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time course or continuously to determine whether other disorders could have resulted in the outcomes. The prison study by Barratt and colleagues involved a 13-week observation period during which findings remained consistent (28,86).

Incidentally, though not included among the Robins and Guze criteria, further confirmation of the validity of a disorder should be provided by a consistently favorable response to a specific treatment intervention, with normalization of both the clinical manifestations of the disorder and accompanying physiological abnormalities. As will be described under pharmacotherapy of impulsive aggression, this, too, has been demonstrated (86).

With at least three of the five Robins and Guze criteria required, impulsive aggression ought to be recognized as a disorder, as numerous officially recognized disorders do not satisfy these minimal criteria. Even more importantly, however, recognition of the disorder carries specific and demonstrably effective treatment implications.

Beyond providing support for recognition of impulsive aggression as a disorder, empirical data accumulated to date lend themselves to synthesis within the previously mentioned general systems model. The *behavioral* aspect is represented by the behavioral manifestation of impulsive aggression itself as well as the associated features of increased gregariousness and diminished emotional "warmth." Although many *environmental* factors have been associated with violent and criminal behaviors, one of the most replicated findings for the predisposition of impulsive aggression is parental brutality. The triggering event for an aggressive outburst is typically a social or interpersonal provocation, however mild in the eyes of others. *Cognitive* findings include deficient verbal abilities such as reading and verbal memory, perhaps secondary to deficient behavioral inhibition (87). *Biological* characteristics from direct research includes temporal and parietal lobe differences in event-related cortical potentials (ERPs) while the subject is performing information-processing tasks. Other research findings supportive of altered molecular genetics in impulsively aggressive animal models, diminished serotonicity in impulsively aggressive humans, and familial studies consistent with genetic inheritance of components of impulsive aggression and associated factors are the most salient contributions to impulsive aggression within the biological realm.

DELINEATION OF IMPULSIVE AGGRESSION FROM OTHER DISORDERS

Are we not all, mentally disordered or not, capable of acts that are both impulsive and aggressive? Of course! What distinguishes pathological from nonpathological behavioral phenomena is essentially a matter of degree. As we have begun to see, however, in using the criteria of Robins and Guze as a standard, and in applying a general systems model of personality variations, recurrent and serious

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impulsive aggression is set apart from "normalcy" and APD without impulsive aggression by measures of quite different dimensions. At this point, then, we examine whether impulsive aggression can be distinguished from IED, wherein uncontrollable aggressive outbursts are the sine qua non of the disorder, and from other disorders of adults, not reaching the level of serious mental illness, wherein aggressive outbursts are symptomatic but not essential features.

IED

The phenomenon of anger outbursts in IED is, to be sure, quite similar to that described for impulsive aggression. McElroy et al. (88) found, in a study of 27 patient referrals who met DSM-IV criteria for IED, substantial association with lifetime diagnoses of mood disorders in particular (93%), but also with substance use disorders (48%), anxiety disorders (48%), eating disorders (44%), other impulsive control disorders (44%), and anxiety disorders (22%). It is therefore reasonable to ask whether the anger attacks in depression described by Fava and Rosenbaum (89,90), IED, and impulsive aggression represent only phenomenological variations of essentially the same pathophysiology. From a slightly different perspective, impulsivity alone is better regarded as a dimension (91) than a disorder, representing a common if not essential feature, for example, of Attention-Deficit/Hyperactivity Disorder (ADHD), Borderline Personality Disorder (BPD), Antisocial Personality Disorder (APD), and different subtypes of mania. Although this could be said of impulsive aggression, as we shall see, the phenomenon also exists outside the context of major mental illness and clinical mood disorders, and, unlike pre-DSM IV definitions of IED, is attended by pronounced impulsivity, not confined to the violent episodes per se.

In the Galveston Anger Management Project (GAMP), we (92) conducted a descriptive study of aggressive behaviors in adult men from the community to examine IED in particular. Of 443 violent men, 79 were identified whose violent outbursts were sufficiently severe and frequent and were not due to another psychiatric disorder. Of these 79, 38 were excluded from further study because of presence of other mental disorders, proportionate provocation, or insufficient data. Of those excluded on the basis of other disorders, most manifested an affective or substance abuse disorder, suggesting, as already supported in the literature, that these other psychopathologies can also play a role in violent behavior. After 26 subjects were excluded owing to excessive impulsivity, then a DSM-III-R exclusionary criterion for IED, 15 subjects remained with diagnosable IED. Thus, 18.9% of sufficiently violent men or 1.49% of all 443 aggressive subjects were diagnosed with IED. Although this study, using volunteer subjects from the community, was not an epidemiologic investigation, the evaluations were thorough. Results supported the impression that pure IED is an uncommon but

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not a rare cause of violence. We concluded that the prospect for both helpful treatment and promising research justifies retention of the diagnosis of IED.

As early as 1963, Karl Menninger (93) described two forms of a phenomenon termed "ego rupture" involving aggressive behavior which anticipated impulsive aggression and IED. The first syndrome, manifested by chronic, repetitive aggression, is similar to impulsive aggression. IED may have been represented by the second syndrome wherein aggressive acts were infrequent but nonetheless sudden and explosive. Megargee's (94) contrasting types of undercontrolled and overcontrolled personality types compare with the Menninger duality. No measures of impulsivity were related to these early descriptive reports.

Two observations from the GAMP study are relevant here: The first relates to the role of impulsivity in IED. As noted, 26 subjects were not considered to have IED because their elevated BIS scores indicated a high level of impulsivity. If generalized impulsivity had not been an exclusionary criterion, the incidence of IED in the GAMP study would have been higher (36). Since DSM-IV discarded this exclusionary criterion, one can expect the diagnosis of IED to be used more often than before. However, this does not refute an essentially different pathophysiology where the aggression is associated with diffuse impulsivity. Further suggesting an essential difference, only aggressive subjects in the GAMP study who were also impulsive showed reduced platelet absorption of serotonin (62).

The second, unpublished observation in the GAMP study was that a crossover double-blind study testing the effect of placebo, lithium, and phenytoin demonstrated a large placebo effect which washed out any possible effects of lithium and phenytoin on aggression. In this study aggressive subjects included those both with and without generalized aggression. This negative result was a prime reason for follow-up research to concentrate on impulsive aggression rather than explosive aggression in the absence of other signs of impulsivity.

Given the positive psychological and laboratory findings associated with impulsive aggression, adequately defined, and the robust behavioral and electrophysiological normalization with anticonvulsant medication, impulsive aggression should probably be considered as a disorder apart from IED without generalized impulsivity, even though this exclusionary criterion has been removed from the DSM-III-R (4).

The DSM-IV change eliminating general impulsivity as an exclusionary criterion for IED allowed a place in the diagnostic scheme for pure impulsive aggression. On the other hand, this change also blurred a potentially useful distinction between IED in the traditional sense, i.e., involving an otherwise sufficiently if not overly controlled personality style, and impulsive aggression with generalized impulsivity. Both are disorders of control over anger leading to agitation, but IED involves relatively increased control with a significantly greater

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quantum of affect or impulsive drive required to breach the control mechanisms resulting in the characteristic "out-of-character" violent outbursts.

Other Disorders

Other disorders to be differentiated from impulsive aggression are those in which aggressive outbursts are symptomatic but not essential: Personality Change Due to a General Medical Condition, disinhibited, aggressive, and combined subtypes; ADHD in adults, especially the predominantly hyperactive-impulsive subtype; BPD; and APD.

Of critical diagnostic importance is the need to establish presence of supporting symptoms to establish the diagnosis of one of these other conditions. An essential feature of ADHD, for example, is attentional dyscontrol. Although impulsive aggression also involves some measure of compromised attention, pervasive disruption of attention is most striking for ADHD. Individuals with BPD show pronounced dysregulation of affect with emotional response to specific stressors, especially the threat of abandonment (95).

APD is characterized by emotional detachment, manifested by lack of fear and compassion (95), allowing expression of destructive impulses even when control mechanisms appear to be functional. Physiological markers, including diminished electrodermal responsiveness to aversive stimuli, absence of the startle reflex, and decreased autonomic arousal in general, are consistent with decreased emotional responsiveness (95). This lack of emotional responsiveness has been implicated to explain the psychopath's craving for excitement and stimulus-seeking behavior. Diminished emotional responsiveness may also lead to the individual's defective ability to profit from experience and to learn from corrective punishment. Thus, aggressive antisocials often, but not always, exhibit impulsive aggression, but they invariably demonstrate premeditated aggression.

Distinguishing impulsive aggression from other disorders of commonality, in terms of both phenomenological similarity and comorbidity, is useful for making specific treatment decisions. At least of heuristic value, however, should be the hypothetical consideration that a similar underlying dyscontrol mechanism is essential or commonly associated with these disorders. In BPD, for example, emotional dysregulation and impulse dyscontrol are closely associated (95). The expression of impulsivity as impulsive aggression requires both an angry, hostile emotional component and weakened impulse control mechanisms. Thus, although individuals with APD show less self-restraint owing to diminished anxiety, their level of impulsive aggression will be a function of impaired control itself. Those with BPD inevitably manifest emotional dysregulation and impaired impulse control; whether impulsive aggression occurs, depends on the amount of externally directed anger.

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ASSESSMENT

Assessment for impulsive aggression begins with the basics: Mental status examination and personal and family history are essential, and even routine physical assessment can be contributory. For a complete, more definitive evaluation, specific rating instruments and neurophysiological probes are recommended. Beyond specific techniques for evaluating impulsive aggression, a more comprehensive assessment should serve to rule out other disorders that can result in aggressive acts, and to establish comorbidities that must be taken into account for proper treatment.

The mental status exam is probably both underutilized and undervalued as an approach for assessing impulsive aggression. If sufficiently comprehensive and individualized, the mental status exam can elicit pertinent data regarding subjective mood states, perceptions and thoughts, and more objective information on cognitive processes. It is not sufficient to simply inquire about suicidal and homicidal thoughts. Inquiring into assaultive and physically aggressive ideation should include questions about individual sensitizing factors ("What ticks you off?"), precipitating perceptions of others ("What do people do to make you angry?"), specific aggressive thoughts, perceived payoffs for acting aggressively, level of intent and seriousness, sense of control or loss of control, expected predisposing contingencies, and desire to resist the impulses.

Just as attempts to predict violence can overlook the treatable and preventable causes, assessment of the nature of aggressive behavior can underemphasize actual risk assessment. Yet clinical assessment and safe management need to go hand in hand. However it is accomplished, the following issues must be addressed (96): Is the patient dangerous to others? Is the danger due to serious mental illness? Is the danger imminent? Are potential victims of the danger reasonably identifiable? Answers to these questions will help the clinician make protective decisions regarding hospitalization or warnings. Though threats of future harm do not necessarily correspond with the phenomenon of explosive, unplanned aggression, the clinician must be prepared to assess threats directed against identifiable individuals. For this Bohrum and Reddy (97) offer a well-reasoned, logical approach.

The evaluator should take care not to completely compartmentalize suicidality and homicidality. If either state is considered exclusionary of the other, important data will be missed. Particularly with acts of combined homicide-suicide, aggression and despair can occur together, providing the energy for a tragedy greater than either killing alone (98).

A comprehensive cognitive screen provides useful data as well. Does the patient approach problems quickly and impulsively or slowly and methodically? What is the individual's estimated level of intellectual functioning? Is laterality

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demonstrated (performance substantially better than verbal memory)? Are there specific verbal deficits or deficiencies in reading ability? Finally, proverb interpretation and judgmental responses can suggest indications of impulsivity (e.g., burning theater: "Yell fire!") or hostility and aggression (e.g., "When the cat's away . . .": "The cat won't kill the mice.").

General observations are also of significance. Is the affect one of anger with tension? Is underlying hostility and resentment recognizable? Does the patient appear impetuous, impatient and easily frustrated in interacting with the interviewer?

The personal history should address the nature, severity, and frequency of aggressive outbursts, with special consideration given to suddenness of onset, disproportionality to any provocation, length of episode, and pre and post mood states. Other conditions to be addressed include drug or alcohol consumption and behaviors associated with intoxication or withdrawal, depression or mania, epileptic phenomena, earlier history of physical abuse, head injury, parental abandonment, neurological illness, and toxic or metabolic conditions that can affect brain functioning. While it is important to rule out medical conditions that could themselves account for impulsive and aggressive behavior, subclinical factors may also help to explain the disorder.

Of the various rating scales used to assess impulsive aggression, four in particular hold promise of special utility. The Barratt Impulsivity Scale (BIS-11) is perhaps the most widely used and validated instrument for measuring the trait of impulsivity (25). The Spielberger Anger Trait State (99) scale is particularly useful in assessing both anger and hostility. To follow and monitor aggressive outbursts, we recommend either the Yudofsky Overt Aggression Scale (100) or the Coccaro-modified version of this instrument (101). Another technique for monitoring course of the disorder and recurrence of episodes, is to enlist the patient's collaboration in keeping a daily log. Of course collateral source information can be most useful in this regard: guards in prisons, nurses and technicians in hospitals, and spouses in domestic settings, for example. Finally, Barratt et al. (83) are in the process of developing an instrument especially designed to measure the degree of impulsive aggression in a given episode. This should be useful in identifying, characterizing, and measuring impulsive aggressive episodes, thereby improving the accuracy of assessments and effectiveness of interventions.

TREATMENT CONSIDERATIONS

Pharmacotherapy

No medication is FDA approved for pharmacoptherapy of impulsive aggression, or for that matter, for the more widely recognized IED. Nonetheless, various

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classes of drugs have been used in practice for these conditions: mood stabilizers, anticonvulsants, β-blockers, and SSRIs. As mentioned above, our early attempt to test the effects of lithium and phenytoin on IED with and without generalized impulsivity yielded disappointing results primarily because of a large placebo effect. Once we further defined the nature of impulsive aggression, however, we found that phenytoin (and in a separate, unpublished study, carbamazepine) had a significant effect in reducing impulsively aggressive outbursts and normalizing electrophysiological evoked potentials in the same objects (86). Whenever phenytoin curbed impulsive aggression, the medicine was 100% effective and attended by substantially increased amplitudes of P300 waveforms of event-related potentials (ERPs). Abnormally low amplitudes reflect less efficient cortical information processing in impulsively aggressive individuals. Such changes in ERPs were not observed when impulsively aggressive subjects received a placebo or when nonimpulsive but aggressive subjects received either placebo or phenytoin. Specifically, phenytoin resulted in significantly decreased frequency and intensity of aggressive acts and in lowered tension and anxiety among the impulsively aggressive individuals but had no effect on the aggressive acts of nonimpulsive subjects.

While the risk of medication side effects is always of appropriate concern, the risks should be lower than that which therapeutic doses to manage epilepsy, because the effective serum level is substantially lower (4–5 μg/mL versus 10–20 μg/mL) (86). Thus, where the condition is clearly and purely impulsive aggression, we would recommend an initial trial with one of these two anticonvulsants in particular. In a further investigation of phenytoin's effects on impulsive aggression, Stanford et al. (102) found in a community, noncriminal sample of aggressive men that phenytoin resulted in a significant reduction in frequency and intensity of aggressive acts. Moreover, tension/anxiety and anger/hostility were also reduced and electrophysiological abnormalities were normalized (viz., PI amplitude increased, EP latencies lengthened, NI amplitudes reduced). The authors interpret these favorable psychophysiological, mood, and behavioral changes to mean that phenytoin-mediated improvement in information processing results in reduced impulsiveness, irritability, and aggressive conduct.

One of the most exciting implications of this research is the possibility that impulsive aggression may be brought under control, even in the context of APD, a condition often considered as unresponsive to therapeutic interventions (103). One might expect SSRIs to be more useful with underlying depression (89,90), or mood stabilizers where hypomanic features are detectable, and β-blockers where more pronounced organicity is present, approaches to be covered elsewhere in this text. The important point with regard to medication is that impulsive aggression contrasts with premeditated aggression, which is singularly unresponsive to medication. An exception is among persons for whom the objective of

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aggressive behavior is not the enhancement of status or acquisition of material gain, but a powerful and deviant sex drive (104), suggesting the possibility of a comorbid disorder such as paraphilia.

Although our work supports an impressively favorable response of impulsive aggression, adequately defined, to the administration of phenytoin (86), empirical studies also support the use of carbamazepine (105) and valproate (106–108) in the treatment of anger outbursts (109). Because these anticonvulsants have demonstrated mood stabilization qualities, they are referred to as mood stabilizers when used to manage poorly controlled aggression. As in the control of epilepsy, however, phenytoin has been observed to have a significant effect on brain function within an hour of administration (110), presumably by stabilizing neuron membranes within the control system of the CNS. Thus, more than mood stabilizers, except where irresistible affective drive is the force behind the aggressive outbursts as in hypomania, anticonvulsants may function as stabilizers of control.

Psychotherapy

A variety of psychotherapeutic approaches have been employed, but unfortunately most without follow-up studies to ascertain efficacy. An empirical advantage to behavioral therapy is that measured monitoring is incorporated into the technique itself. Token economies have been shown to be of some success in controlling episodic aggression in inpatients. The impression is that the effectiveness of these programs is a function of how methodically they are designed and implemented. A hybrid program, the "therapeutic economy," attempts to incorporate elements of the therapeutic community to enhance social responsibility and effectiveness without diluting the rigor of a classical token economy (111).

Of interest among the individual and group therapeutic techniques is dialectic behavioral therapy (DBT), which can be provided on inpatient settings or in outpatient clinics. DBT is designed to address impulsive aggression and impulsive behaviors in general, and a developing body of empirical research supports its effectiveness in treating impulsive behaviors, especially in patients with BPD (112). Also of interest is the use of CRCST, a manualized anger management treatment (see Deffenbacher, this volume). By acting to reduce anger, CRCST may also work to reduce impulsive aggressive acts in impulsively aggressive individuals.

Environmental Adjustments

As with other mental disorders, the amount of environmental control and structure should be titrated to the patient's dangerousness and ability to self-control. Often the patient can be managed in the community with pharmacotherapy, psychother-

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apy, and monitoring. Until the patient is better able to control him/herself, he/she should be advised to avoid situations that are likely to spark explosive dyscontrol. Where lethal weapons pose a risk, measures should be taken to neutralize them, though the safest approach where such danger is acute is physical containment of the patient (e.g., hospitalization).

Because outbursts of impulsive aggression are sudden and relatively unprovoked, discrete acts are difficult to predict. However, if such outbursts are becoming more serious and frequent, despite appropriate outpatient measures, hospitalization may have to be considered. Even voluntary hospitalization is not without controversy owing to the risks posed to other, nonaggressive patients, but an inpatient setting can often provide an appropriate level of control and safe management in comparison with outpatient care. If the patient does not consent, emergency detention and court-ordered hospitalization may be warranted, though civil commitment codes are not invariably permissive of involuntary hospitalization where major mental illness is lacking. Of course, jail and prison can be natural outcomes of impulsive aggression where criminal injury is inflicted on another. The hopeful point of this chapter is the possibility that treatment and management will serve to prevent assaultive injuries and, in some cases, the expense of criminal procedures and penal containment.

SOCIAL AND LEGAL ISSUES

One naturally associates impulsive aggression with the harm the afflicted individual inflicts upon *others* and the enormous cost to society in housing impulsive, aggressive offenders within the criminal justice system. These are worthy concerns. Acts with serious results are appropriately considered as criminal, though not always with the same degree of moral turpitude and criminal culpability, as when an offender premeditates his violent act and executes it with exquisite self-control. The criminological dilemma is that the responsibility of a criminal act should weigh less where the individual lacked self-control, yet some impulsive individuals are most predictably dangerous and society deserves to be protected from them. The hope is that with more effective treatments and infrastructures for their delivery, assaultive injuries can be prevented and lengthy incarcerations obviated.

Of the various situations where recognition of impulsive aggression could have special value, one is certainly at disciplinary hearings in correctional settings. States vary greatly in whether mental health professionals are allowed to participate in such hearings and for what purpose. Presumably a most valued and least controversial clinical role would be to assess for presence of impulsive aggression that can be treated, reducing the probability of future infractions due to this disorder. Without interfering in the disciplinary procedure itself, the provision of voluntary, effective treatment for impulsively aggressive inmates could

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make a substantial improvement in maintaining an orderly and peaceful incarcerated community of offenders.

Less well appreciated than the harm inflicted on others is the harm that impulsive aggression causes the subject him/herself. Even without the extreme consequences of self-injury and even death or lengthy imprisonment, impulsive aggression can be severely damaging to familial and social relationships and can jeopardize one's opportunities for employment and promotion.

Most individuals who display impulsive aggression do so involuntarily. They also experience feelings of remorse following the act. Although society has the responsibility of protecting its citizens, it should also consider what is to be gained by changing the impulsive aggressive behavior patterns of incarcerated individuals. Many, though not nearly enough, sexual paraphiliacs have obtained effective treatment; substance abusers, too, with rehabilitation, have maintained a satisfying quality of life. As treatment becomes more available and visible for individuals with impulsive aggression, substantial benefits will undoubtedly accrue to the individuals themselves and to the general public. The first step down this propitious pathway will be the general recognition of impulsive aggression, its nature, and most promising treatment approaches.

REFERENCES

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. Washington: American Psychiatric Publishing, 1952, p 52.
2. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 2nd ed. Washington: American Psychiatric Publishing, 1968.
3. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 3rd ed. Washington: American Psychiatric Publishing, 1980.
4. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 3rd ed. revised. Washington: American Psychiatric Publishing, 1987.
5. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. Washington: American Psychiatric Publishing, 1994.
6. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. text revision. Washington: American Psychiatric Publishing, 2000.
7. EF Coccaro, RJ Kavoussi, ME Berman, JD Lish. Intermittent explosive disorder-revised: development, reliability, and validity of research criteria. Comp Psychiatry 39(6):368–376, 1998.
8. World Health Organization. Tenth Revision of the International Classification of Diseases, Chapter V(F): Mental and Behavioral Disorders (Including disorders of psychological development). Clinical Descriptions and Diagnostic Guidelines. Geneva: World Health Organization, 1991.
9. ES Barratt. Measuring and predicting aggression within the context of a personality theory. J Neuropsychol 3(2):535–539, 1991.

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10. ES Barratt, L Slaughter. Defining, measuring, and predicting impulsive aggression: a heuristic model. Behav Sci Law 16(3):285–302, 1998.
11. WR Ashby. Design for Brain. New York. New York: Wiley, 1960.
12. A Lazare. Hidden conceptual models in clinical psychiatry. N Engl J Med 288: 345–350, 1973.
13. B Azar. At the frontier of science: social cognitive neuroscience merges three distinct disciplines in hopes of deciphering the process behind social behavior. Monit Psychol 42–41, 2002.
14. KN Ochsner, MD Lieberman. The emergency of social cognitive neuroscience. Am Psychol 56(9):717–734, 2001.
15. E Robins, SB Guze. Establishment of diagnostic validity in psychiatric illness. Am J Psychiatry 126:107–111, 1970.
16. L Berkowitz. Some determinants of impulsive aggression: role of mediated associations with reinforcements for aggression. Psychol Rev 81(2):165–176, 1974.
17. AB Heilbrun, IJ Knopf, P Brunna. Criminal impulsivity and violence subsequent parole outcome. Br J Criminol 16:367–377, 1976.
18. M Linnoila, M Virkkunen. Low cerebrospinal fluid 5-hydroxy-indoleacetic acid concentration differentiates impulsive from non-impulsive violent behavior. Life Sci 33:2609–2614, 1983.
19. EF Coccaro, LJ Siever, HM Klar, G Maurer, K Cochrane, TB Cooper, RC Mohs, K Davis. Serotonergic studies in patients with affective and personality disorders. Arch Gen Psychiatry 46:587–599, 1989.
20. AB Heilbrun, LC Heilbrun, KL Heilbrun. Impulsive and premeditated homicide: an analysis of subsequent parole risk of the murderer. J Crim Law Criminol 69: 108–114, 1978.
21. M Virkkunen, MS Rawlings. CSF biochemistries, glucose metabolism, and diurnal activity rhythms in alcoholics, violent offenders, fire setters, and healthy volunteers. Arch Gen Psychiatry 51:20–27, 1994.
22. J DeJong, M Virkkunen, M Linnoila. Factors associated with recidivism in a criminal population. J Nerv Ment Dis 180:543–550, 1992.
23. MS Atkins, DM Stoff, ML Osboune, R Brown. Distinguishing instrumental from hostile aggressions: does it make a difference? J Abnorm Child Psychol 21:355–365, 1993.
24. AH Buss, A Durkee. An inventory for assessing different types of hostility. J Consult Clin Psychol 21:343–349, 1957.
25. JH Patton, MS Stanford, ES Barratt. Factor structure of the Barratt Impulsiveness Scale. J Clin Psychol 51(6):768–774, 1995.
26. B Vitiello, D Behar, et al. Subtyping aggression in children and adolescents. J Neuropsychiatry 2:189–192, 1990.
27. D Mungas. An empirical analysis of a specific syndrome of violent behavior. J Nerv Ment Dis 171:357–361, 1963.
28. ES Barratt, MS Stanford, TA Kent, AR Felthous. Neurological and cognitive psychophysiological substrates of impulsive aggression. Biol Psychiatry 41:1045–1061, 1997.
29. E Othmer, EC Penick, BJ Powell, MR Read, SC Othmer. Psychiatric Diagnostic

Marcel Dekker, Inc.
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Interview–Revised (PDI-R): Manual. Los Angeles: Western Psychological Services, 1992.
30. A Raine, A Scerbo. In: Biological Theories of Violence in Neuropsychology of Aggression, eds JS Miliners, MA Boston. Dordrecht: Kluwer Academic Publishers, 1991, pp 1–26.
31. AJ Fallgatter, MJ Herrmann. Electrophysiologische Korrelate von Impulsivität. Annual Meeting of the German Psychiatric Society. Nervenheilkunde: Zeitschrift für Interdisziplinäre Fortbildung 20(suppl 3):137, p487A, 2001. Abstract.
32. CW Matthias, MS Stanford. P300 under standard and surprise conditions in self-reported impulsive aggression. Neuro-Psychopharmacol Biol Psychiatry 23:1037–1051, 1999.
33. E Harmon-Jones, ES Barratt, C Wigg. Impulsiveness, aggression, reading, and the P300 of the event-related potential. Pers Individ Dif 22(4):439–445, 1997.
34. ES Barratt, TA Kent, SG Bryant, AR Felthous. A controlled trial of phenytoin in impulsive aggression. J Clin Psychopharmacol 11:388–389, 1991.
35. ES Barratt. The use of anticonvulsants in aggression and violence. Psychopharmacol Bull 29:75–81, 1993.
36. AR Felthous, ES Barratt. Impulsive und episodische aggressivität: biologische und psychosoziale Forschung in den USA. In: HL Kröber, KP Dahle, eds. Sexualstraftaten und Gewaltdelinquenz: Verlauf-Behandlung-Opferschutz. Heidelberg; Kriminalistic-Verlag, 1998, pp 95–117.
37. RJ Davidson, KM Putnam, CL Larson. Dysfunction in the neural circuitry of emotion regulation—a possible prelude to violence. Science 289(5479):591–594, 2000.
38. JD Jentsch, JR Taylor. Impulsivity resulting from frontostriatal dysfunction in drug abuse: implications for the control of behavior by reward-related stimuli. Psychopharmacology 146:373–390, 1999.
39. WH Gaddes. Learning Disabilities and Brain Function. New York; Springer-Verlag, 1980.
40. MS Buchsbaum. Average evoked response and stimulus intensity in identical and fraternal twins. Physiol Psychol 2:365–370, 1974.
41. A Tellegen, DT Lykken, TJ Bouchard, K Wilcox, N Segal, S Rich. Personality similarity in twins reared apart and together. J Pers Soc Psychol 54:1031–1039, 1988.
42. A Raine. The Psychopathology of Crime. New York; Academic Press, 1993.
43. DS Cates, BK Houston, CR Vavak, et al. Heritability of hostility-related emotions, attitudes, and behaviors. J Behav Med 16:237–256, 1993.
44. L Eaves, HJ Eysenck, D Martin. Genes, Culture and Personality: An Empirical Approach. New York: Academic Press, 1989.
45. EF Coccaro, CS Bergeman, RJ Kavoussi, AD Seroczynski. Heritability of aggression and irritability: a twin study of the Buss-Durkee Aggression Scales in adult male subjects. Biol Psychiatry 41:273–274, 1997.
46. AD Seroczynski, CS Bergeman, EF Coccaro. Etiology of the impulsivity/aggression relationship: genes or environment? Psychiatry Res 86:41–57, 1999.
47. KP Lesch, U Merschdorf. Impulsivity, aggression, and serotonin: a molecular psychobiological perspective. Behav Sci Law 18:589–604, 2000.

MARCEL DEKKER, INC.
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48. EH Hollenberg, MS Sperry. Some antecedents of aggression and effects of frustration in doll play. Personality 1:32–43, 1951.
49. LD Eron, LO Walder, R Toigo, NM Lefkowitz. Social class, parental punishment for aggression and child aggression. Child Dev 34:857–899, 1963.
50. KA Dodge, JE Bates, GS Pettit. Mechanisms in the cycle of violence. Science 250:1678–1683, 1990.
51. S Glueck, E Glueck. Unraveling Juvenile Delinquency. Cambridge: Harvard University Press, 1950.
52. W McCord, J McCord, IK Zola. Origins of Crime: A New Evaluation of the Cambridge-Somerville Youth Study. New York: Columbia University Press, 1959.
53. LB Silver, CC Dublin, RS Lourie. Does violence breed violence? Contributions from a study of the child abuse syndrome. Am J Psychiatry 126:404–407, 1969.
54. A Bandura. Social learning through imitation. In: MR Jones, ed. Nebraska Symposium on Motivation. Lincoln: University of Nebraska Press, 1962.
55. A Bandura, RH Walters. Adolescent Aggression. New York: Ronald Press, 1959.
56. MA Straus, VE Mouradian. Impulsive corporal punishments by mothers and antisocial behavior and impulsiveness of children. Behav Sci Law 16(3):353–374, 1998.
57. G Margolin, EB Gordis. The effects of family and community violence and children. Annu Rev Psychol 51:445–479, 2000.
58. ME Berman, EF Coccaro. Neurobiological correlates of violence: relevance to criminal responsibility. Behav Sci Law 16:303–318, 1998.
59. GL Brown, FK Goodwin, JC Ballenger, PF Goyer, LF Major. Aggression in humans correlates with cerebrospinal fluid amine metabolites. Psychiatr Res 1:131–139, 1979.
60. M Virkunnen, A Nuutila, FK Goodwin, M Linnoila. Cerebrospinal fluid monoamines: metabolite levels in male arsonists. Arch Gen Psychiatry 44:241–247, 1981.
61. M Linnoila, M Virkunnen, M Scheinen, A Nuutila, R Rimon, FK Goodwin. Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from non-impulsive violent behavior. Life Sci 33:2609–2614, 1993.
62. TA Kent, CS Brown, ES Barratt, AR Felthous, RM Rose. Blood platelet uptake of serotonin in episodic aggression: correlation with red blood cell proton T. and impulsivity. Psychopharmacol Bull 24:454–457, 1988.
63. M Virkkunen, J DeJong, J Bartko, FK Goodwin, M Linnoila. Relationship of psychological variables to recidivism in violent offenders and impulsive fire setters. Arch Gen Psychiatry 46:600–603, 1989.
64. M Virkkunen, A Nuutila, FK Goodwin, M Linnoila. Cerebrospinal fluid metabolite levels in male arsonists. Arch Gen Psychiatry 44:241–247, 1987.
65. H Ågren, IN Mefford, MV Rudorfer, M. Linnoila, WZ Potter. Interacting neurotransmitter symptoms. A non-experimental approach to the 5-HIAA-HVA correlation in human CST. J Psychiatry Res 20:175–193, 1986.
66. GO Gabbard. A neurobiologically informed perspective on psychotherapy. Br J Psychiatry 177:117–122, 2000.
67. A Bandura. Aggression: A Social Learning Analysis. Englewood Cliffs, NJ: Prentice-Hall, 1973.

MARCEL DEKKER, INC.
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68. FA Elliot. Neurology of aggression and episodic dyscontrol. Sem Neurol 10(3): 303–312, 1990.
69. KA Dodge. The structure and function of reactive and proactive aggression. In: Pepler DJ, Rubin KH, eds. The Development and Treatment of Childhood Aggression. Hillsdale, NJ: Lawrence Erlbaum, 1991.
70. B Vitiello, D Behar, J Hunt, D Stoff, A Ricciuti. Subtyping aggression in children and adolescents. J Neuropsychiatry 2(2):189–192, 1990.
71. S Feshbach. The function of aggression and the regulation of aggressive drive. Psychol Rev 71:257–272, 1964.
72. D Glaser. The classification of offenses and offenders. In: D Glaser, ed. Handbook of Criminology. Chicago: Rand McNally College Publishing Company, 1974, pp 45–85.
73. MS Stanford, ES Barratt. Impulsivity and the multi-impulsive personality disorder. Pers Individ Dif 13(7):831–834, 1992.
74. HG Kennedy, DH Grubin. Hot-headed or impulsive? Br J Addict 85:639–643, 1990.
75. JH Lacey, CDH Evans. The impulsivist: a multi-impulsive personality disorder. Br J Addict 81:641–649, 1986.
76. FG Moeller, DM Dougherty, ES Barratt, JM Schmitz, AC Swann, J Grabowski. The impact of impulsivity on cocaine use and retention in treatment. J Substance Abuse Treatment 21:193–198, 2001.
77. A Fossati, AD Ceglie, A Acquarini, ES Barratt. Psychometric properties of an Italian version of the Barratt Impulsiveness Scale–II (BIS-II) in nonclinical subjects. J Clin Psychol 57(6):815–828, 2001.
78. T Someya, K Sakado, T Seki, M Kojima, C Reist, SW Tang, S Takahashi. The Japanese version of the Barratt Impulsivity Scale, 11th version (BIS-II): its reliability and validity. Psychiatry Clin Neurosci 55:111–114, 2001.
79. A Fossati, ES Barratt, E Acquarini, A Di Ceglie. Psychometric properties of an adolescent version of the Barratt Impulsiveness Scale–II (BIS–II-A) in a sample of Italian high school students. Percept Motor Skills. In press.
80. ES Barratt. The biological basis of impulsiveness: the significance of timing and rhythm disorders. Pers Indiv Dif 4:387–391, 1983.
81. ES Barratt. Impulsiveness subtraits: arousal and information processing. In: JT Spence, CE Izard, eds. Motivations, Emotions, and Personality. Amsterdam: Elsevier Science, 1985, pp 137–146.
82. ES Barratt. Impulsiveness and aggression. In: J Monahan, HJ Steadman, eds. Violence and Mental Disorders: Developments in Risk Assessment. Chicago: University of Chicago Press, 1994, pp 61–79.
83. ES Barratt, MS Stanford, L Dowdy, MJ Liebman, TA Kent. Impulsive and premeditated aggression: a factor analysis of self-reported acts. Psychiatr Res 86(2): 163–173, 1999.
84. S Herpertz, H Sass. [Impulsiveness and impulse control. On the psychological and psychopathological conceptualization.] (In German.) Nervenarzt 68(3):171–183, 1997.
85. ES Barratt. Aggression/impulsivity: neurobiological correlates. In: G Adelman,

MARCEL DEKKER, INC.
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BH Smith, eds. Elsevier's Encyclopedia of Neuroscience, 2nd ed. New York: Elsevier Science, 1999, pp 35–37.
86. ES Barratt, MS Stanford, AR Felthous, TA Kent. The effects of phenytoin on impulsive and premeditated aggression: a controlled study. J Clin Psychopharmacol 17(5):341–349, 1997.
87. RA Barkley. Attention-deficit/hyperactivity disorder, self-regulation, and time. Toward a more comprehensive theory. Dev Behav Pediatr 18(4):271–279, 1997.
88. SL McElroy, CA Soutullo, DA Beckman, P Taylor, PE Keck. DSM-IV intermittent explosive disorder: a report of 27 cases. J Clin Psychiatry 59(4):203–210, 1998.
89. M Fava, K Anderson, JF Rosenbaum. "Anger attacks": possible variants of panic and major depressive disorders. Am J Psychiatry 147(7):867–870, 1990.
90. JF Rosenbaum, M Fava, JA Pava, MK McCarthy, RJ Steingard, E Bouffides. Anger attacks in unipolar depression. Part 2. Neuroendocrine correlates and changes following fluoxetine treatment. Am J Psychiatry 150(8):1164–1168, 1993.
91. A Kaplan. Researching impulsivity and aggression in mania and IED. Psychiatric Times' Bipolar Disorder Impulsive Spectrum, Supplement to Psychiatric Times, pp 6–8, May 2001.
92. AR Felthous, SG Bryant, CB Wingerten, ES Barratt. The diagnosis of intermittent explosive disorders in violent men. Bull Am Acad Psychiatry Law 19(1):71–79, 1991.
93. KA Menninger. The Vital Balance. New York: Viking Press, 1963.
94. EI Megargee. Undercontrolled and overcontrolled personality types in extreme antisocial aggression. Psychol Monogr Gen Appl 80(3):1–29, 1966.
95. SC Herpertz, HJ Kunert, A Schurkens, EM Steinmeyer, H Sass, R Freese, M Flesch, R Muller-Isberner, M Osterheider. [Impulse control and affect regulation in personality disorders.] (In German.) Psychother Psychosom Med Psychol 50(11):435–442, 2000.
96. AR Felthous. The clinician's duty to protect third parties. In: PJ Resnick, ed. The Psychiatric Clinics of North America. Forens Psychiatry 22(1):49–60, 1999.
97. R Bohrum, M Reddy. Assessing violence risk in *Tarasoff* situations: a fact-based model of inquiry. Behav Sci Law 19(3):375–386, 2001.
98. AR Felthous, A Hempel. Combined homicide-suicides: a review. J Forens Sci 40(5):846–857, 1995.
99. CD Spielberger. State-Trait Anger Expression Inventory (STAXI Professional Manual). New York: Psychological Assessment Resources, 1988.
100. SJ Yudofsky, J Silver, W Jackson, et al. The Overt Aggression Scale for the objective rating of verbal and physical aggression. Am J Psychiatry 143:35–39, 1989.
101. Coccaro EF, Harvey PD, Kupsaw-Lawrence E, Herbert JL, Bernstein DP. Development of neuropharmacologically based behavioral assessments of impulsive aggressive behavior. J Neuropsychiatry Clin Neurosci 3:S44–S51, 1999.
102. MS Stanford, RJ Houston, CW Mathias, KW Greve, NR Villemarette-Pittman, D Adams. A double-blind placebo-controlled crossover study of phenytoin in individuals with impulsive aggression. Psychiatry Res 103:193–203, 2001.
103. RD Hare, MA Clark, M Grann, D Thornton. Psychopathy and the predictive valid-

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ity of the PCL-R: an international perspective. Behav Sci Law 18(5):623–646, 2000.
104. A Rösler, E Witztum. Pharmacotherapy of paraphilias in the next millennium. Behav Sci Law 18(1):43–56, 2000.
105. JA Mattes. Comparative effectiveness of carbamazepine and propranolol for rage outbursts. J Neuropsychiatry Clin Neurosci 2:159–164, 1990.
106. WJ Giakas, JP Seibyl, CM Mazure. Valproate in the treatment of temper outbursts [letter]. J Clin Psychiatry 51:525, 1990.
107. MY Hasan, RDE Sewell, PJ Nichols. Does the anticonvulsant agent sodium valproate display behaviorally selective anti-offensive activity? J Pharmacol 42(suppl):185, 1998. Abstract.
108. PE Keck Jr, SL McElroy, LM Friedman. Valproate and carbamazepine in the treatment of panic and post-traumatic stress disorder, withdrawal states, and behavioral dyscontrol syndromes. J Clin Psychopharmacol 12(suppl):365–415, 1992.
109. E Hollander. Managing aggressive behavior in patients with obsessive-compulsive disorder and borderline personality disorder. J Clin Psychiatry 60(suppl 15):38–44, 1999.
110. ES Barratt, DM Faulk, ME Brandt, SG Bryant. Effects of phenytoin on N100 augmenting/reducing and the late positive complex of the event related potential: a topographic analysis. Pharmacoelectroencephalography 15:201–207, 1986.
111. A Nahor, AR Felthous. Therapeutic economy: an effective model of residential psychiatric treatment. J Behav Ther Exp Psychiatry 7:77–78, 1976.
112. MM Linehan. Cognitive Behavioral Treatment of Borderline Personality Disorder. New York: Guilford Press, 1993.

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9

Intermittent Explosive Disorder

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INTRODUCTION

Intermittent Explosive Disorder (IED) is a diagnostic category in the Diagnostic and Statistical Manual of Mental Disorders (DSM) (1–3) that purportedly identifies individuals with recurrent impulsive aggressive behavior not due to other known mental or physical disorder. However, because DSM criteria for IED are poorly operationalized, IED has been the subject of little systematic research to date (4).

The paucity of attention paid to IED is striking for several reasons. First, impulsive aggressive behavior is relatively common. Data from community epidemiological surveys suggest that up to 12–25% of men and women in the United States report a history of physical fighting as an adult (5). While data on "IED-type" diagnoses from large community surveys have not been published, the lifetime prevalence of DSM-IV IED may be as high as 6% in psychiatric outpatients (6) and 4% in the general community (7). If the latter rate is correct, about 11.2 million people among the U.S. population could have DSM-IV IED. Second, impulsive aggressive behavior is associated with substantial psychosocial distress/dysfunction (8,9). In addition, it is now widely appreciated that impulsive

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aggressive behavior directed at children by parents is associated with the later development of various forms of psychopathology including impulsive aggressive behavior directed at others (10). Third, there is evidence that impulsive aggressive behavior is influenced by genetic (see Chap. 2 for review) and neurochemical (11) factors and, accordingly, is responsive to treatment with specific psychopharmacological agents (12–14). This chapter briefly reviews the history of IED and highlights some of the epidemiologic, phenomenologic, familial, biologic, and treatment correlates of this disorder, particularly as currently conceptualized in research circles.

NOSOLOGY OF IED BY DSM AND RESEARCH CRITERIA SYSTEMS

Despite the wealth of data regarding aggression and/or impulsive aggression, there are few systematic data regarding the clinically corresponding entity which may best be categorized as Intermittent Explosive Disorder. This is largely because of problems in diagnostic criteria that were often due to the individual perspectives of the "framers" of the various DSMs that, in turn, were not grounded in empiric data. Review of the history of "IED-type" disorders in the DSM reveals a critical reversal on how to grant a specific diagnosis to individuals with "recurrent, problematic, impulsive aggressive behavior." At first, DSM highlighted "recurrent, problematic, impulsive aggressive behavior" as a diagnostic entity. Later revisions of DSM, however, progressively moved away from this position and tended to restrict "IED-type" diagnostic criteria set and, as a consequence, these DSM criteria could not identify most individuals characterized by the type of impulsive aggression repeatedly shown to correlate with specific developmental and psychosocial variables, measures of central neurobiologic function and responses to specific psychopharmacologic agents.

DSM Through DSM-III-R

Initially, DSM identified "recurrent, problematic, impulsive aggressive behavior" as a diagnostic entity. Individuals with a "persistent reaction to frustration with irritability, temper tantrums, and destructive behavior" were diagnosed with "Passive-Aggressive Personality (aggressive type)," which was considered characterological in nature. After the first DSM, the "criteria" were progressively restricted in nature. In DSM-II, Passive-Aggressive Personality (aggressive type) evolved into "Explosive Personality" and was described as "intermittently violent behavior occurring in an aggressive person." These individuals were described as generally "excitable, aggressive, and overresponsive to environmental pressures" with "gross outbursts of rage or of verbal or physical aggressiveness" that was "strikingly different from the patient's usual behavior." There were no opera-

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tional diagnostic criteria since specific operational criteria were not included in the DSM for any disorder until version DSM-III. In DSM-III, Explosive Personality was named Intermittent Explosive Disorder for the first time and assigned Clinical Disorder status under the axis I classification.

Four specific criteria were specified for IED. These criteria proved to be quite problematic in a number of ways. For example, "assaultive" and "destructive" acts (criterion A) were not operationalized and clinicians were left without specific guidelines to determine the kinds of behaviors of sufficient severity to satisfy the criteria. The frequency and minimal time frame for putative assaultive/destructive acts were also unclear. In addition, subjects who were generally aggressive or impulsive in between the ill-defined "aggressive episodes" were excluded from receiving the diagnosis (criterion C). This was unfortunately overrestrictive because individuals with *recurrent*, problematic, impulsive aggressive behaviors are also impulsive and aggressive in general (i.e., between more severe outbursts). The Antisocial Personality Disorder exclusion (criterion D) also restricted the diagnosis. These issues were not resolved in the revised DSM-III and DSM-III-R diagnostic criteria for IED proved to be even more restrictive. The exclusionary criterion (criterion D) was expanded to include *both* Borderline and Antisocial Personality Disorder. Because of some symptom overlap (i.e., intense inappropriate anger), a sizable minority of subjects with problematic impulsive aggressive behavior could also meet criteria for Borderline Personality Disorder. Ultimately, it was shown that only 20% of clinically valid IED cases could actually receive an IED diagnosis by DSM-III as a consequence of these changes (15).

Development of Research Criteria for IED

The limitation of DSM-III-R criteria to identify individuals with "recurrent, problematic, impulsive aggression" made it necessary to develop Research Diagnostic Criteria for IED. This process began by modifying the DSM-III-R criteria for IED and then applying it to a series of subjects in whom relevant clinical and psychometric data about aggression and impulsivity had already been collected.

Intermittent Explosive Disorder-Revised (IED-R): Rationale for Criteria Selection

The research criteria for IED-R involved five aspects that critically operationalize the criteria for IED:

1. They broadened the scope of aggressive behavior encompassed by the IED diagnosis by including verbal and indirect physical aggression (provided that these behaviors are impulsive, frequent, and associated with distress and/or impairment; see points 2–4 below). Less severe impulsive aggressive behavior was included because these forms of aggression had been shown to be sig-

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nificantly reduced in impulsive aggressive subjects in double-blind, placebo-controlled clinical trials with fluoxetine (14,16).

2. They required the specified aggressive behavior to be impulsive in nature. If the presence of "impulsive aggression" is not stipulated, individuals with "premeditated" (i.e., criminal) aggression could be diagnosed with IED. Inclusion of "premeditated" aggression in IED would make the diagnosis nonspecific since existing data strongly suggests a psychosocial (17), biologic (18,19), and treatment response (12,13) distinction between "impulsive" and "premeditated" aggression.

3. They required a minimal frequency over time that is critical in order to make the diagnosis of IED reliable across clinicians and to ensure that subjects with only occasional impulsive aggressive outbursts (especially those of low severity; see the first point, above) are not assigned this disorder.

4. They required the presence of subjective distress (e.g., in the individual) and/or social or occupational dysfunction. This formally focused the concept of IED as a behavioral "disorder" and further enables the criteria to rule out subjects for whom their impulsive aggressive behavior is not functionally severe enough, and again, not to "overdiagnose" this disorder.

5. They modified the "diagnostic exclusionary" criteria to: a) allow subjects with antisocial or borderline personality disorder (AsPD/BPD) to be given IED, and b) rule out subjects with current histories of major depression.

The former modification was made for two reasons. 1) Impulsive aggressive subjects with AsPD and/or BPD would not be identified by IED criteria. It is understood that the presence of AsPD and/or BPD appears to be a justifiable exclusion criteria for IED. Since BPD subjects are impulsive and prone to angry outbursts, and AsPD subjects are often irritable and aggressive, why use two diagnoses to describe the same person? However, many BPD/AsPD subjects are not particularly impulsive aggressive. 2) There are few data to support the idea that impulsive aggressive behavior should be hierarchically placed under the constructs of BPD/AsPD. Familial (20), twin (21), biological (22,23), and treatment response (12–14,24,25), data suggest that impulsive aggressive behavior, while present in many BPD/AsPD subjects, has specific clinical relevance apart from the remaining diagnostic features of these personality disorders. The latter modification regarding the exclusion of major depressive disorder was made to formally recognize the fact that impulsive aggressive outbursts are characteristic of a number of individuals suffering from a current major depressive disorder (see Chap. 7, this volume). While it is possible that these outbursts identify a clinically meaningful subgroup of depressives, it is widely appreciated that the presence of major depression is associated with irritability, a behavioral symptom manifested by quickness to react to aversive stimuli with negative affect. In fact, the presence of irritability was part of the "A" criterion in the Research Diagnostic Criteria (RDC; the precursor to the DSM-III) set for major depressive disorder.

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Moreover, a reduction in irritability scores is usually noted with successful psychopharmacologic treatment of major depression.

Application of IED-R Criteria

DSM-III-R and IED-R criteria were applied to 188 personality disordered subjects, nearly half of whom were seeking evaluation and/or treatment for impulsive aggressive behavior. Despite the large proportion of treatment seeking individuals, only 2% met DSM-III-R (note that 10% would eventually have met DSM-IV) criteria while a much larger 40.4% met IED-R criteria for IED (26). Interrater reliability for IED-R was very high ($\kappa = 0.92$), and IED-R-positive subjects were found to have significantly greater aggression (e.g. by Life History of Aggression) and impulsivity (e.g., by Barratt Impulsivity Scale) scores than IED-negative subjects. This finding remained even after correcting for the influence of variables that also correlated with these measures of aggression and impulsiveness (e.g., age, race, gender, etc.). Further analysis noted that the variance accounted for by "impulsiveness" completely overlapped with that accounted for by "aggression." This suggested that impulsiveness was secondary to aggression as might be expected if IED was describing individuals with prominent "impulsive aggressive" behaviors. IED-positive subjects were also found to have lower scores of psychosocial function (GAF score) than IED-negative subjects. This finding remained even after correcting for the influence of various characteristics (e.g., age, race, gender, etc.) that also correlated with the GAF score.

Regarding comorbidity with axis I disorders, IED-positive subjects were more likely to have a current and life history of mood disorder than IED-negative subjects. They were also more likely to have a life history of alcohol and substance dependence. Further analysis found that scores of aggression and impulsiveness remained elevated in IED-positive subjects even after accounting for the presence of these comorbid disorders into the statistical model. Regarding axis II disorders, IED-positive subjects were more likely to have diagnosis of borderline (but not antisocial) personality disorders than IED-negative subjects. Notably, however, most (62%) IED-positive subjects did not have either borderline or antisocial personality disorder. Further analysis found that scores of aggression and impulsiveness remained elevated in IED-positive subjects even after accounting for the comorbid presence of Borderline Personality Disorder into the statistical model. These data suggest that IED-R could reliably describe individuals with substantial impulsive aggressive behavior associated with a reduction in psychosocial functioning.

DSM-IV Criteria for IED: An Imperfect Revision

DSM-IV criteria, published in the mid-1990s, contained revisions that addressed some of the issues that led to the development of the IED-R criteria set. Most importantly, DSM-IV eliminated the "C" criterion that required IED subjects not

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be "generally impulsive and aggressive" between explosive episodes. Another potential change in DSM-IV criteria was that the exclusionary disorders could "not better account for" the intermittent explosive episodes. This allowed for the possibility that subjects meeting criteria for BPD or AsPD could be given a DSM-IV IED diagnosis if the diagnostician felt that the explosive episodes were above and beyond that seen in those personality disorders. While most clinicians may not make this distinction, it led the way for subsequent research criteria to allow for the "comorbid" presence of these personality disorders.

Another change noted in the DSM-IV criteria was the new wording that the assaultive/destructive behavior of the subject resulted from a "failure to resist aggressive impulses." This wording was included to bring the "A" criterion for IED into line with the other Impulse Control Disorders in DSM-IV. This wording change suggests the possibility that the explosive episodes of IED subjects are "premeditated" and not "impulsive" in nature. This is problematic in that it may not allow subjects with "impulsive" explosive episodes to be diagnosed with DSM-IV IED depending on how this criterion is interpreted by the clinician or researcher. More importantly, the distinction between "impulsive" and "nonimpulsive" aggression is extremely relevant as it is impulsive, not nonimpulsive aggressive behaviors that correlate with biological characteristics (18,19) and that are responsive to treatment interventions (12,13). If so, some of the revisions of DSM-IV may not truly "correct" the deficiencies of the IED criteria sets of DSM-III-R.

Further Development of Research Criteria for IED

Close examination of the DSM-IV (3) criteria for IED revealed that several modifications to the "A" criterion were necessary before DSM-IV-like diagnoses of IED could be applied in a systematically assessed series of subjects. First, the "A" criterion did not define the number of "aggressive acts" required for diagnosis. Second, there was no guideline on the time frame during which these acts occurred. Third, there was no definition of "serious assaultive acts" or of "destruction of property." The present author's query of the DSM-IV subcommittee charged with revising the criteria sets for the Impulse Control Disorders revealed that this subcommittee had not discussed these issues in their deliberations and that there were, in fact, no official guidelines to follow in this regard (Michael Wise, personal communication, March 1997). This led us to propose the following:

1. That at least three "aggressive acts," during any proposed time frame, would be required for a DSM-IV IED research diagnosis (IED-IV-R).
2. That proposed time frames for study include adult lifetime, 1 year, 6 months, and 3 months.
3. "Aggressive acts" were defined as: a) any act of physical aggression on another person which did not lead to physical evidence of injury (e.g., push/

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shove, slap, etc.) or destruction of property with a relatively trivial value (e.g., breaking one or two dishes); or b) any act of physical aggression on another person which did lead to physical evidence of injury (e.g., push/shove, slap, etc.) or destruction of property of nontrivial value (e.g., destroying several dishes, windows, appliances, etc.).

Application of these criteria, during a retrospective research-chart review, revealed that these research criteria for IED-IV-R over the lifetime could be diagnosed with good reliability (κ for lifetime IED-IV-R = 0.87 in a sample of 76 subjects). Analyses using research criteria for IED-IV-R on the same sample of 188 subjects reported above revealed similar findings to those reported above. That is, IED-IV-R subjects were (as expected) more aggressive, more impulsive, and less functional, than non-IED-IV-R subjects.

Relationship Between IED-R and IED-IV-R Criteria Sets

In review of the same research records, there was substantial overlap (69%) between subjects meeting research criteria for IED-R and IED-IV-R. Despite this, there were potentially important conceptual differences between IED-R and IED-IV-R. Most importantly, IED-R described subjects with frequent (i.e., twice weekly or greater) explosive episodes that may or may not (usually not) rise to the level of physical assault on persons or destruction of property. In contrast, IED-IV-R described subjects whose explosive episodes rise to the level of physical assault on other persons, and/or to the destruction of property, at a relatively low frequency over time (i.e., certainly less than twice weekly). In addition, IED-R required the presence of impulsive aggression (rather than aggression of any kind) and the presence of distress and/or impairment due to the explosive outbursts.

It is not known which description of IED best describes the population of impulsive aggressive individuals in the community or in mental health populations. However, it is likely that there are several clinically relevant subtypes of intermittent explosive disorders. For example: a) subjects with frequent small-scale, nonassaultive/destructive, explosive episodes associated with distress and/or impairment ("IED-R Only"); b) subjects with relatively infrequent assaultive/destructive explosive episodes in addition to more frequent small-scale, nonassaultive/destructive, explosive episodes associated with distress and/or impairment ("IED-R and IED-IV-R"); c) subjects with relatively infrequent assaultive/destructive explosive episodes associated with distress and/or impairment ("IED-IV-R Only"). In our initial sample, 69% met research criteria for both IED-R and IED-IV-R, 20% met criteria for IED-IV-R Only, and 11% met criteria for IED-R Only. Importantly, analysis of our data found no differences among these three subgroups with respect to measures of aggression and impulsivity (MANCOVA result: Wilks $\lambda = .46$, $P = .348$); in contrast, these three groups did differ from Non-IED-Type subjects in the same type of analysis

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TABLE 1 Current Research Criteria for Intermittent Explosive Disorder

A.	Recurrent incidents of aggression manifest as either[a]:
	A1. Verbal or physical aggression towards other people, animals, or property occurring twice weekly on average for 1 month. OR
	A2. Three episodes involving physical assault against other people or destruction of property over a 1-year period.
B.	The degree of aggressiveness expressed is grossly out of proportion to the provocation or any precipitating psychosocial stressors.
C.	The aggressive behavior is generally not premeditated (e.g., is impulsive) and is not committed in order to achieve some tangible objective (e.g., money, power, intimidation, etc.).
D.	The aggressive behavior causes either marked distress in the individual or impairment in occupational or interpersonal functioning.
E.	The aggressive behavior is not better accounted for by another mental disorder (e.g., Major Depressive/ Manic/Psychotic Disorder; ADHD); General Medical Condition (e.g., head trauma, Alzheimer's disease); or to the direct physiological effects of a substance.

[a]Original A criteria from Coccaro et al. (26) recurrent incidents of aggression manifest as verbal or physical aggression toward other people, animals, or property occurring twice weekly on average for one month.

(MANCOVA result: Wilks $\lambda = .36$, $P < .001$) with all available measures of aggression and impulsivity demonstrating significant differences in this regard (all P's $< .01$). This result suggested that these two research criteria sets may not meaningfully differentiate impulsive aggressive subjects along the primary dimension of behavior and that the application of one criteria set, but not the other, will not fully identify the population of interest. Accordingly, one research diagnostic criteria set may be used that allows for subjects from each of these potential three subgroups (and others) to be identified if empirically justified (Table 1).

EMPIRIC STUDIES OF IED BY DSM OR RESEARCH CRITERIA

Epidemiology of IED

Few community-based data are available regarding the epidemiology of IED. What data are available are from clinical studies involving IED and clinical surveys of samples of psychiatric patients. While the DSM-IV stated that "IED is apparently rare," recent data suggests that IED is far from rare. However, as there

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has not been diagnostic clarity as to who does, and does not, meet criteria for IED, comparative estimates across studies are problematic.

Clinical Surveys of Psychiatric Patients

Using a chart review of 830 hospitalized patients in the early 1980s, Monopolis and Lyons (27) found that while 2.4% of patients were given the diagnosis of IED, only 1.1% of the patients actually met the DSM-III criteria for IED. Few data of this type appeared in the literature until the recent report of Zimmerman et al. (6), who found that 3.8% ($CI_{95\%}$ = 1.9%), and 6.2% ($CI_{95\%}$ = 2.3%), of 411 outpatient psychiatric subjects at the Rhode Island Hospital had, respectively, a current or a lifetime diagnosis of IED by DSM-IV using the Structured Clinical Interview for DSM-IV Diagnoses (SCID). Reanalysis of a much larger data set (n = 1300) from the same study site (Coccaro and Zimmerman, unpublished data; hereafter referred to as the Rhode Island Hospital Study) reveals that the results from the smaller data set approximate that of the much larger data set with, respectively, 3.1% ($CI_{95\%}$ = 0.9%), and 6.3% ($CI_{95\%}$ = 1.3%), of the psychiatric outpatients meeting DSM-IV criteria for IED at time of interview or at some time in the past.

Surveys of Patients Recruited for Studies of IED

As part of a pharmacologic treatment study of IED by DSM-III in the 1980s, Felthous et al. (15) evaluated 433 men with histories of aggression. Of these, only 1.8% ($CI_{95\%}$ = 1.3%) met full DSM-III criteria for IED. From the author's own research program, we have found that 12.3% ($CI_{95\%}$ = 2.6%) of subjects scheduling examination for an aggression problem in response to a public service announcement for a pharmacologic treatment study of IED meet DSM-IV criteria for lifetime IED and that 20.7% ($CI_{95\%}$ = 3.2%) of the same group of subjects meet Current Research Criteria for lifetime IED. These higher rates [compared with the Felthous et al. (15) study] reflect the changes in the IED criteria sets from DSM-III to DSM-IV. Among subjects simply seeking to be paid as study volunteers (i.e., non-treatment seeking), we have found that 5.3% ($CI_{95\%}$ = 2.4%) meet DSM-IV criteria for lifetime IED and that 8.8% ($CI_{95\%}$ = 3.0%) meet Current Research Criteria for lifetime IED.

Pilot Survey of Community Subjects

In collaboration with Nestadt and colleagues, the author conducted a pilot survey of community subjects in Baltimore (hereafter referred to as the Baltimore Study) (7). The subjects were part of a follow-up study of ECA subjects in Baltimore (28). In this pilot study, trained interviewers conducted brief structured interviews (in up to 253 subjects) in order to collect data regarding the presence or absence of DSM-IV and Current Research Criteria for IED. Preliminary analyses of the raw data suggest that the community rate of lifetime IED by DSM-IV criteria is

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4.0% ($CI_{95\%}$ = 2.4%) and the community rate by Current Research Criteria is 5.1% ($CI_{95\%}$ = 2.7%). Given the 95% confidence limits bounding this estimate, the community rate for lifetime IED could range from about 1.6% to 6.4% for DSM-IV IED and from about 2.4% to 7.9% by present Research Criteria. Mindful of the confidence intervals in this small pilot study, the lifetime rate of IED in the community could range from 4.5 million to 18.0 million using DSM-IV criteria or from 6.7 million to 22.2 million using Research Criteria. Using either sets of estimates, the lifetime rate of IED appears to be far from rare and equal to, if not greater than, that seen with other major psychiatric disorders such as schizophrenia or bipolar illness. More definitive data about the rate of IED in the community, however, are expected when data from the current National Co-Morbidity Study, directed by Ronald Kessler, are available for analysis in the next few years.

Phenomenology of IED

While diagnostic criteria have been inconsistent across time, there is general consensus in clinical descriptions of IED. IED appears to begin as early as childhood (8) and/or adolescence with a mean age of onset at 15 years of age and an average duration of about 20 years (9). This early age of onset has been confirmed in the most recent analysis from the Rhode Island Hospital Study. The mean age of onset in this sample was 16 years, with 31% of DSM-IV IED subjects reporting onset of IED during their first decade (preteen years), 44% during their second decade (teenage years), and finally 19% and 6% during their third and fourth decades (adulthood), respectively. Aggressive outbursts have a rapid onset (9), often without a recognizable prodromal period (8,15). Episodes are short-lived ($<$ 30 min) (9), and involve verbal assault, destructive and nondestructive property assault, or physical assault (8,9). Episodes are associated with substantial distress, impairment in social functioning, occupational difficulty, and legal or financial problems (8,9). The outbursts most commonly occur in response to a minor provocation by a close intimate or associate (9,15). In some cases episodes may appear to occur without identifiable provocation (9). Finally, IED subjects may have less severe episodes of verbal and nondestructive property assault in between more severe assaultive/destructive episodes (9,26).

Gender Ratio in IED

In clinical studies of IED, the gender ratio in IED is typically reported at 3 male to 1 female (e.g., 8,9,26). However, data from both the Rhode Island Hospital Study and the Baltimore Study (7) suggest that the male-to-female ratio is closer to unity with the male to female ratio for DSM-IV IED being 0.86 in both samples.

Comorbidity with Axis I Disorder

IED is reported to be highly comorbid with a variety of DSM-IV disorders including mood, anxiety, and substance use disorders. McElroy et al. (9) reported that

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current/lifetime diagnoses of other axis I disorders are often present in IED subjects. Current axis I disorders present in the IED subjects to the greatest degree were: mood disorder (89%; 33% major depression, 56% bipolar spectrum); anxiety disorder (37%); impulse control disorders (33%); eating disorder (19%); and substance abuse or somatoform disorder (7% each). It is important to note that the investigators took care not to make the IED diagnosis in subjects in whom aggressive episodes were circumscribed to these comorbid disorders. Rates for these disorders over the lifetime were higher for anxiety disorder (48%), substance use disorders (44% for alcohol, 33% for nonalcohol), and for impulse control disorder (44%).

Except for bipolar disorder, which was excluded from the overall sample, Coccaro et al. (26) reported similar findings. A current depressive mood disorder (i.e., major depression, dysthymia, depressive disorder NOS) was noted in 39.5% of IED-R subjects (lifetime rate: 72.4%). A current anxiety disorder was also noted in 19.7% of IED-R subjects (lifetime rate: 27.6%). A lifetime diagnosis of alcoholism and/or other substance use disorders (53%) was present at nearly the same rate as that reported by McElroy et al. (9). In a recent preliminary study of individuals presenting for behavioral treatment of "aggressive driving" (n = 30; hereafter referred to as the Aggressive Drivers Study), the rate of lifetime mood, anxiety, and substance use disorder among those diagnosed with IED (n = 10) was reported at 30%, 50%, and 70%, respectively (29). Analysis of data from the Rhode Island Hospital Study also reveals the rate of lifetime mood disorder (70%), anxiety disorder (75%), and substance use (60%) disorders to be as high or even higher. However, examination of the age of onset for these disorders reveals that in DSM-IV IED subjects, the onset of IED occurs at a significantly earlier age than that for mood disorder (16.0 years vs. 22.5 years; $P < .05$ by paired t-test) or substance use disorder (16.0 years vs. 19.7 years; $P < .05$ by paired t-test). In the case of the anxiety disorders, the age of onset of phobic disorders (i.e., social/specific phobia) occurs at a significantly earlier age than that of IED (16.0 years vs. 10.7 years; $P < .01$ by paired t-test), while the onset of the nonphobic anxiety disorders (i.e., panic, obsessive-compulsive, posttraumatic stress, and generalized anxiety disorders) occur somewhat later than IED (16.0 years vs. 18.3 years; P = ns). The relatively high rate of comorbid phobic disorder (49%) in the DSM-IV IED subjects, coupled with the earlier age of onset of these disorders, suggests the possibility that the early development of a phobic anxiety disorder might be associated with an increased risk of developing IED later on in adolescence or young adulthood.

Comorbidity with Bipolar Disorder and Other Impulse Control Disorders

A relationship between IED and bipolar disorder has been suggested by McElroy (30) for two reasons. First, a substantial comorbidity appears to exist between IED and bipolar illness (total = 56%: Bipolar I: 33% Bipolar II: 11%; Bipolar

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NOS or Cyclothymia: 11%) (9). Second (following from this observation), the aggressive episodes of these IED subjects appeared, on close examination, to resemble "microdysphoric manic episodes" (30). Affective symptoms noted prior to and during the aggressive episodes included irritability (79–92%), increased energy (83–96%), racing thoughts (62–67%), anxiety (21–42%), and depressed mood (17–33%). While Coccaro et al. (26) did not report any history of bipolar disorder among their IED subjects, that study specifically excluded subjects with bipolar disorder. In the Aggressive Drivers Study (29), the rate of bipolar disorder in those diagnosed with IED was only 10% (only one of 10 subjects met criteria for a bipolar II disorder). This is similar to the rate in the Rhode Island Hospital Study where only 11% of the DSM-IV IED subjects met lifetime criteria for any form of bipolar disorder (Bipolar I: 5%; Bipolar II: 5%; and Bipolar NOS: 1%). Notably, the age of onset for IED in this sample tended to precede that for Bipolar Disorder (16.0 years vs. 21.0 years). These data suggest that while bipolar disorder may not be highly comorbid with IED in general, its presence may be relevant in some specialty clinic samples (9,30). Sensitivity to the possibility of bipolar disorder in such cases of IED should prompt a full psychiatric evaluation to rule in or rule out bipolar disorder. If bipolar disorder of any kind is present, mood stabilizers (rather than SSRIs) should probably be used in such individuals as the first-line agent (30).

Comorbidity with Other Impulse Control Disorders

A relationship between IED with other impulse control disorders is implied in the DSM-IV because IED is placed in the Impulse Control Disorders section of the DSM. McElroy et al. (9) reported that 12 of 27 (44%) DSM-IV IED cases had any type of Impulse Control Disorder. Notably, this figure included impulsive control disorders that are not formally in DSM-IV (e.g., compulsive buying at 37%). For the DSM-IV impulse control disorders, this rate ranged from 0% (tricotillomania) to 19% (kleptomania). In contrast, no IED subjects were given an impulsive control diagnosis in the Aggressive Drivers Study (29). In the Brown Study, the rate of any impulse control disorder among the IED subjects was only 5% with all comorbid cases meeting DSM-IV criteria for Pathological Gambling. The age of onset for IED preceded that for Pathological Gambling (16.0 years vs. 26.2 years). It is likely that these differences in comorbidity rates represent differences in ascertainment. If so, the comorbidity of IED and other impulse control disorders among the general psychiatric patients may be lower that currently assumed.

Comorbidity with Borderline Personality Disorder (BPD) and Antisocial Personality Disorder (AsPD)

DSM-IV criteria may allow the diagnosis of IED in the presence of BPD or AsPD. However, most clinicians (and several researchers) question whether IED

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exists apart from BPD/AsPD in such patients. In our first study, using the original research diagnostic criteria set for IED, we found that nearly 80% of BPD/AsPD subjects met research criteria for IED (26). In a later analysis of a larger group of personality-disordered subjects, we found this percentage to be ~ 82% with a marked difference based on whether subjects were seeking treatment for impulsive aggression or not (Coccaro, unpublished data). Among those seeking treatment for impulsive aggression, the rate of IED among BPD/AsPD subjects was extremely high (93%). However, among those not seeking treatment for impulsive aggression, the rate was substantially lower (45%) and similar to the rate among subjects in the Rhode Island Hospital Study (51%; n.b.: these were BPD/AsPD subjects who would have met DSM-IV IED criteria except for the fact that they were BPS/AsPD). Among those in the Aggressive Drivers Study (29) the rate of BPD/AsPD in those diagnosed with IED was 60%. Among the subjects studied in the Baltimore Study, only 23% of BPD/AsPD subjects would have met DSM-IV criteria for IED. Accordingly, it is likely that degree of comorbidity of BPD/AsPD with IED may be a function of treatment seeking (e.g., a sampling artifact) rather than a finding that is generalized to the community. That is, as samples are enriched with individuals seeking treatment (particularly for impulsive aggression), the frequency of comorbidity with BPD/AsPD increases (i.e., 23% in a community sample to 51% in a sample seeking general mental health treatment to 93% in a sample seeking treatment for impulsive aggression). Even so, there is emerging evidence that meeting criteria for IED has added relevance in BPD/AsPD patients. In a recent analysis of our study subjects we have found that patients with BPD/AsPD and IED have significantly higher scores on measures of aggression and significantly lower scores on general psychosocial function than patients with BPD/AsPD but not IED.

Genetic/Familial Correlates of IED

There are no twin or adoption studies of IED. However, existing family history data suggest that IED (or IED-type) behavior is familial. The first-degree relatives of patients with histories of violent behavior have a high incidence of violent behavior (31,32). An increased frequency of first-degree relatives with history of temper outbursts and a strong trend for familial aggregation of IED (defined as the first two DSM-III criteria for IED) was reported in psychiatric patients with history of temper outbursts compared to other patients (33). While not a controlled study (i.e., no comparison group), McElroy et al. (9) noted that 32% of first-degree relatives of IED probands had IED. Recently, the author conducted a blinded, controlled, family history study using Current Research Criteria for IED (34). In this Family History Study of IED we compared the morbid risks of IED (by research criteria) of first-degree relatives of IED Probands (n = 30 with 160 relatives) with those observed in first-degree relatives of control pro-

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bands (n = 20 with 81 relatives) without IED or any other history of "recurrent, problematic, impulsive aggression." Three independent raters were used in this controlled, blinded, family history study. All data were presented to a Best-Estimate Board for final diagnostic assessment. Interrater reliability for IED in probands ($\kappa = 0.87$) and in relatives ($\kappa = 0.90$) was excellent. Sensitivity for the IED diagnosis comparing direct interview with the family history method employed in this study was as expected for a family history study (35) with moderate sensitivity (57%), but greater specificity (81%) for the IED diagnosis.

Analysis of the family history data revealed significantly elevated morbid risk for IED in relatives of IED probands compared to control probands (26% vs. 8%, OR = 3.25, $P < .01$). Examination of comorbid conditions among the IED probands revealed no differences in MR of IED in relatives when IED probands were broken down into having (and not having) a life history of suicide attempts, major depression, alcoholism, drug use disorders, or any anxiety disorder. This indicates that familial aggregation of IED was not due to the presence of these comorbid conditions in the probands in this sample. Though there were increases in MR of major depression, alcoholism, drug use disorders, and any anxiety disorder, in the relatives of IED probands, there was no relation between a diagnosis of IED and any (or all) of these comorbid disorders in these relatives (ϕ coefficients from .01 to .07, P's = ns). That is, for example, the chance of a relative with IED having a life history of major depression was no greater than that of a relative without IED. These data suggest that while these comorbid conditions aggregate in relatives of IED probands, the aggregation of IED is not due to an epiphenomenon of the liability to have these comorbid conditions. These findings, which are being currently reinvestigated in a direct family study of IED, strongly suggest that IED, as defined by research criteria, is familial and independent of other conditions.

Biologic and Treatment Correlates of IED

Nearly all studies in the area of the biology and treatment of aggression focus on aggression as a dimensional, as opposed to a categorical, variable, and are reviewed in detail elsewhere (11). However, since the development of Current Research Criteria for IED, we have had the opportunity to explore biological and treatment response correlates in our ongoing studies. In our pilot data, we have found that the maximal prolactin response to d-fenfluramine challenge, and the number of platelet 5-HT Transporter Binding sites, are reduced in subjects meeting Current Research Criteria for IED and are, overall, inversely correlated with dimensional measures of impulsive aggression. Preceding these studies, Linnoila et al. (18) and Virkunnen et al. (19) reported reduced CSF 5-HIAA concentrations in subjects diagnosed as DSM-III IED compared with subjects who were not IED and/or those who demonstrated nonimpulsive aggression.

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Regarding treatment response, it is noteworthy that all subjects in our published fluoxetine trial of impulsive aggression met Current Research Criteria for IED (14). This study demonstrated that the core feature of IED, impulsive aggressive behavior, is responsive to a treatment targeting the central serotonergic system. Reanalysis of the data, however, suggests that SSRIs may work best in IED subjects who are only "moderately aggressive" (see Chap. 19, this volume), perhaps because the central serotonergic system of such individuals is less impaired than that of those who are "highly aggressive" (36). Impulsively aggressive subjects who do not respond to treatment with SSRIs may respond to a mood stablizer (37). Antiaggressive responsiveness to a mood stabilzer in IED-like subjects has also been reported by Sheard et al. (38), Cowdry and Gardiner (39) and Barratt et al. (13) (diphenylhydantoin in impulsively aggressive prison inmates). Recently, Hollander et al. (40) reported greater reduction over placebo in overt aggression scores in Cluster B IED subjects (BPD: 55%, B Spectrum NOS: 21%, Narcissistic PD: 13%, AsPD: 10%, and Histrionic PD: 1%) treated with divalproex (40). This study followed the basic design of the Coccaro and Kavoussi study, used the same outcome measures (14), and entered subjects that met both DSM-IV and Current Research Criteria for IED. Curiously, a divalproex effect was not observed in IED subjects without Cluster B Personality Disorder as these subjects responded equally to divalproex and placebo. Reasons for a difference in divalproex response, as a function of Cluster B Personality Disorder, are unknown and highlight the need for further research in this area to determine predictors of antiaggressive response in IED subjects. Clinically, McElroy (30) has suggested that IED subjects with unipolar affective symptoms be treated first with SSRIs (or other antidepressants) while IED subjects with bipolar affective symptoms be treated first with mood stabilizers; IED subjects without affective symptoms should be treated first with SSRIs.

CONCLUSION

While IED has been poorly characterized to date, the behavioral phenomena underlying this disorder (i.e., impulsive aggression) have been well studied for a number of years. This research has led to important insights into the biology and treatment of impulsive aggressive behavior (11). Recently, new data have emerged to indicate that IED is more common than once thought, that it runs in families apart from other disorders, that its age of onset appears earlier than that of most of its comorbid disorders, and that it may be associated with variable deficits in central serotonergic functioning. With several psychopharmacologic options available to treat recurrent, problematic, impulsive aggression now available (e.g., SSRIs, mood stabilizers, etc.; see chapters in this volume by Donovan, Fava, Lee, Malone), IED should now be systematically investigated so that patients with this disorder can be offered potentially efficacious treatments (psycho-

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pharmacologic and/or cognitive-behavioral treatments) and can be identified for further research in this area.

REFERENCES

1. Diagnostic and Statistical Manual of Mental Disorders, 3rd ed. Washington: American Psychiatric Publishing, 1980.
2. Diagnostic and Statistical Manual of Mental Disorders, 3rd ed revised. Washington: American Psychiatric Publishing, 1987.
3. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. Washington: American Psychiatric Publishing, 1994.
4. J Bradford, J Geller, HR Lesieur, R Rosenthal, M Wise. Impulse control disorders. In: TA Widiger, AJ Francis, HA Pincus, MB First, R Ross, W Davis, eds. DSM-IV Source Book, Vol II. Washington: American Psychiatric Publishing, 1994.
5. LN Robins, DA Regier. Psychiatric Disorders in America. New York: Free Press, 1991.
6. M Zimmerman, J Mattia, S Younken, M Torres. The prevalence of DSM-IV Impulse Control Disorders in Psychiatric Outpatients. APA New Research Abstracts #265. Washington: American Psychiatric Publishing, 1998.
7. EF Coccaro, CA Schimdt, JF Samuels, WW Eaton, G Nestadt. Lifetime rates of intermittent explosive disorder in a community sample. APA New Research Abstract, 155th Annual Meeting of the American Psychiatric Association, Philadelphia, 2002.
8. JA Mattes. Comparative effectiveness of carbamazepine and propranolol for rage outbursts. J Neuropsychiatry Clin Neurosci 2:159–164, 1990.
9. SL McElroy, CA Soutullo, DA Beckman, P Taylor, PE Keck. DSM-IV intermittent explosive disorder: A report of 27 cases. J Clin Psychiatry 59:203–210, 1998.
10. LR Huesmann, E Leonard, M Lefkowitz, L Walder. Stability of aggression over time and generations. Dev Psychopathol 20:1120–1134, 1984.
11. EF Coccaro, LJ Siever. Pathophysiology and treatment of aggression. In: KL Davis, D Charney, JT Coyle, C, Nemeroff eds. Psychopharmacology: The Fifth Generation of Progress, 1709–1724. Phildelphia: Lippincott Williams & Wilkins, 2002.
12. M Sheard, J Manini, C Bridges, A Wapner. The effect of lithium on impulsive aggressive behavior in man. Am J Psychiatry 133:1409–1413, 1976.
13. ES Barratt, MS Stanford, AR Felthous, TA Kent. The effects of phenytoin on impulsive and premeditated aggression: a controlled study. J Clin Psychopharmacol 17: 341–349, 1997.
14. EF Coccaro, RJ Kavoussi. Fluoxetine and impulsive aggressive behavior in personality disordered subjects. Arch Gen Psychiatry 54:1081–1088, 1997.
15. AR Felthous, G Bryant, CB Wingerter, ES Barratt. The diagnosis of intermittent explosive disorder in violent men. Bull Am Acad Psychiatry Law 19:71–79, 1991.
16. C Salzman, AN Wolfson, A Schatzberg, et al. Effect of fluoxetine on anger in symptomatic volunteers with borderline personality disorder. J Clin Psychopharmacol 15:23–29, 1995.

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17. KA Dodge, G Pettit, J Bates. Socialization mediators of the relation between socioeconomic status and child conduct problems. Child Dev 65:649–665, 1994.
18. M Linnoila, M Virkkunen, M Scheinin, A Nuutila, R Rimon, FK Goodwin. Low cerebrospinal fluid 5-hydroxylndolacetic acid concentration differentiates impulsive from nonimpulsive violent behavior. Life Sci 33:2609–2614, 1983.
19. M Virkkunen, R Rawlings, R Tokola, et al. CSF biochemistries, glucose metabolism, and diurnal activity rhythms in alcoholic, violent offenders, fire setters, and healthy volunteers. Arch Gen Psychiatry 51:20–27, 1994.
20. JM Silverman, L Pinkham, TB Horvath, EF Coccaro, HM Klar, S Schear, S Apter, M Davidson, RC Mohs, LJ Siever. Affective and impulsive personality disorder traits in the relatives of borderline personality disorder. Am J Psychiatry 148:1378–1385, 1991.
21. S Torgerson. Genetic and nosological aspects of schizotypal and borderline personality disorder. Arch Gen Psychiatry 41:546-554, 1984.
22. EF Coccaro, LJ Siever, HM Klar, G Maurer, K Cochrane, RC Mohs, KL Davis. Serotonergic studies in affective and personality disorder: correlates with suicidal and impulsive aggressive behavior. Arch Gen Psychiatry 46:587–599, 1989.
23. HB Moss, JK Yao, GL Panzak. Serotonergic responsivity and behavioral dimensions in antisocial personality disorder with substance abuse. Biol Psychiatry 28: 325–338, 1990.
24. RW Cowdry, DL Gardner. Pharmacotherapy of borderline personality disorder: alprazolam, carbamazepine, trifluroperazine, and trancypromine. Arch Gen Psychiatry 45:111–119, 1988.
25. MM Linehan, DA Tutek, HL Heard, HE Armstrong. Interpersonal outcome of cognitive behavioral treatment for chronically suicidal borderline patients. Am J Psychiatry 151:1771–1776, 1994.
26. EF Coccaro, RJ Kavoussi, ME Berman, JD Lish. Intermittent explosive disorder-revised: development, reliability and validity of research criteria. Compr Psychiatry 39:368–376, 1998.
27. S Monopolis, JR Lion. Problems in the diagnosis of intermittent explosive disorder. Am J Psychiatry 140:1200–1202, 1983.
28. J Samuels. WW Eaton, OJ Bienvenu, CH Brown, PT Costa, G Nestadt. Prevalence and correlates of personality disorders in a community sample. Br J Psychiatry 180: 536–542, 2002.
29. T Galovski, EB Blanchard, C Veazey. Intermittent Explosive Disorder and other psychiatric co-morbidity among court-referred and self-referred aggressive drivers. Behav Res Ther 40:641–651, 2002.
30. SL McElroy. Recognition and treatment of DSM-IV Intermittent Explosive Disorder. J Clin Psychiatry 60(suppl 15):12–16, 1999.
31. G Bach-Y-Rita, JR Lion, CF Climent, F Ervin. Episodic dyscontrol: a study of 130 violent patients. Am J Psychiatry 127:1473–1478, 1971.
32. BM Maletsky. The episodic dyscontrol syndrome. Dis Nerv Syst 36:178–185, 1973.
33. JA Mattes, M Fink. A family study of patients with temper outbursts. J Psychiatr Res 21:249–255, 1987.
34. EF Coccaro. Family history study of intermittent explosive disorder. APA New

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Research Abstract. 152nd Annual Meeting of the American Psychiatric Association, Washington, 1999.
35. SV Faraone, MT Tsuang. Methods in psychiatric genetics. In: MT Tsuang, M Tohen, GEP Zahner, eds. Textbook in Psychiatric Epidemiology. Wiley-Liss, New York: NY 1995, pp 81–134.
36. EF Coccaro, RJ Kavoussi, RL Hauger. Serotonin function and antiaggressive responses to fluoxetine: a pilot study. Biol Psychiatry 42:546–552, 1997.
37. RK Kavoussi, EF Coccaro. Divalproex sodium for impulsive aggressive behavior in patients with personality disorder. J Clin Psychiatry 59:676–680, 1998.
38. MH Sheard, JL Marini, CI Bridges, E Wagner. The effect of lithium on impulsive aggressive behavior in man. Am J Psychiatry 133:1409–1413, 1976.
39. RW Cowdry, DL Gardner. Pharmacotherapy of borderline personality disorder: alprazolam, carbamazepine, trofluroperazine, and tranycypromine. Arch Gen Psychiatry 45:111-119, 1988.
40. E Hollander, AC Swann, EF Coccaro, KA Tracy, SL McElroy, D Burt, Peris E, Sommerville K, Nemeroff CB. Divalproex sodium is superior to placebo for impulsive aggression in Cluster B personality disorders. Neuropsychopharmacology (in press).

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10

Questionnaire and Interview Measures of Aggression in Adults

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INTRODUCTION

The goal of this chapter is to provide a review of the psychometric properties of measures used to assess the frequency of and propensity for aggression in adults. This review includes self-rating scales and clinician-rated interviews. Observational measures of aggression are not included. For a review of observational aggression measures the reader is referred to the chapter written by Bech and Mak (1). Furthermore, this review is restricted to measures that examine actual aggressive behavior. Measures that solely assess common emotive (e.g., anger), cognitive (e.g., hostility), or behavioral (e.g., antisocial behaviors) corollaries of aggression were not included. Scales that assess a general propensity toward aggression, sometimes referred to as "trait" aggression, were the focus of the chapter. However, some measures examining aggressive behavior in specific contexts (e.g., partner violence and sexual aggression) are also discussed. A list of the measures discussed in this review, along with some of their basic properties, is presented in Tables 1 and 2.

Measures for the following review were identified via searching Medline

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TABLE 1 Description of General Aggression Measures in Adults

Measure	Items	Subscales			Mode of administration		Reliability[a]	
		Physical	Verbal	Sexual	Self-report	Interview	Internal	Test-retest
Buss-Perry Aggression Questionnaire (12)	29	X	X		X		.72–.89	.72–.80
STAXI-2 (Anger Expression-Out) (46)	57 (8)		X		X		.73–.94 (.73–.80)	.21–.81[b]
Past Feelings and Acts of Violence (62)	12				X		.77	
Aggression Inventory (66)	28	X	X		X		.64–.86	.60–.79[c]
Brief Anger-Aggression Questionnaire (68)	6				X		.82	.84
Overt Aggression Scale-Modified (87)	25	X	X			X		.46–.54
Life History of Aggression (90)	10					X	.74–.88	.80–.97
Intermittent Explosive Disorder Interview	20[d]	X	X			X		

[a] Based on original validation study referenced next to scale name.
[b] These reliabilities are for the original STAXI. No reliability estimates for the STAXI-2 were found. The STAXI state scale test–retest reliability was scale. .21–.27; all other scales were .61–.81.
[c] No test–retest reliabilities were given in for the scale initially. Test–retest reliabilities were taken from Harris (33).
[d] The items have many subitems. The measure takes 20–30 min complete for someone with a significant history of aggression.

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TABLE 2 Description of Specific Aggression Measures in Adults

Measure	Items	Subscales			Mode of administration		Reliability[a]	
		Physical	Verbal	Sexual	Self-report	Interview	Internal	Test-retest
Partner violence								
Conflict Tactics Scales-2 (69)	39	X	X	X	X	X[b]	.79–.95	
Index of Spouse Abuse (73)	30	X	X		X		.90–.97	
Abusive Behavior Inventory (75)	30	X	X		X		.70–.92	
Psychological Maltreatment of Women (76)	58		X		X		.90–.93	
Severity of Violence Against Women (77)	46	X	X		X		.89–.96	
Sexual aggression								
Sexual Experiences Survey (79)	10				X	X[c]	.74–.89	.93
Coercive Sexuality Scale (85)	18				X		.96	

[a]Based on original validation study referenced next to scale name.
[b]The original CTS was developed as an interview. The CTS-2 has been used as both a questionnaire and interview, but has been developed as a questionnaire. Psychometric data are given for the questionnaire version of the CTS-2.
[c]The SES was given as an interview to validate the SES questionnaire. The two versions showed strong agreement. Reliabilities given in the table are for the questionnaire version of the SES.

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and PsychINFO computerized databases using the keyword "aggression" combined with the keywords "interview," "measure," "inventory," and "questionnaire." Aggression scales that met the aforementioned criteria and that had published psychometric properties (reliability and/or validity) were included in the review to the extent allowed given space restraints. Although it is possible that this search failed to capture all available measures of aggression, it is hoped that this chapter will serve as useful review of aggression measures in clinical and/or research settings. Prior to discussing specific aggression measures, some issues relevant to all aggression assessment should be highlighted.

DEFINING AGGRESSION

Aggression, Hostility, and Anger

Researchers have yet to agree on a uniform definition of aggression (2–4). However, what almost all (1,3–5) current definitions of aggression have in common is the classification of aggression as a behavior. Despite this, studies often use the term aggression interchangeably with "hostility" and "anger" (6). Adding to the confusion, measures of aggression often include subscales that assess these related constructs (and vice versa), and subscales purported to measure "aggression" sometimes include items that actually assess anger or hostility. Thus, it is important to understand the differences among the concepts.

Anger is most often conceptualized as an emotion or experiential state that is accompanied by hostile cognitions and physiological tension (7). Dysfunctional anger has been conceptualized as a syndrome that causes distress or impairment in the individual, and which may be related to aggressive responding (8). Though anger can and often does facilitate aggression, angry feelings are neither a necessary nor a sufficient component of aggression. In fact, the overwhelming majority of anger episodes do not involve any aggression (9).

Hostility is an enduring cognitive style that is characterized by a pattern of suspicion and resentment (10). Not surprisingly, individuals high in hostility are frequently angry and tend to act aggressively (11). However, the association between hostility and aggression is far from perfect, with weak to moderate correlations between hostility and aggression common in the literature (12). Thus, although aggression, anger, and hostility overlap, they are separate constructs.

Aggression and Self-Aggression

Some theorists define aggression solely within the context of behaviors directed toward another individual or object (5), whereas others (13) include destructive behavior directed against the self (e.g., self-mutilation and suicide). Though self-directed aggression is an area of obvious importance, almost all measures of

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"aggression" assess outwardly directed aggression. Accordingly, our review focuses exclusively on outwardly directed aggression. Reviews of self-aggression measures have been presented elsewhere (14,15).

Aggressive Acts

Definitions of aggression typically require delivery of a noxious stimulus with the intent to do harm (5,16). Theorists generally categorize these acts along two dichotomous dimensions—verbal versus physical, and direct versus indirect (17). The goal of physical aggression is injury to person or property, whereas the goal of verbal aggression is humiliation, rejection, and threat. Direct aggression occurs when the aggressor is easily identifiable by the victim during the aggressive act (e.g., punching someone in the face, calling someone an "asshole"). Indirect aggression occurs when the victim is not present during the aggressive act (e.g., gossip), and/or when a victim's objects are taken or damaged (17). Measures of aggression vary in the extent to which they assess these four types of aggressive acts, so the same individual may receive very discrepant scores on two measures of "aggression." Furthermore, no aggression measure was found that contained a scale dedicated to the assessment of indirect aggression, and many measures have no any items that sample indirect verbal aggression. This limitation may spuriously inflate gender differences in aggression, as it has been suggested that women are more likely than men to engage in these forms of aggression (18). Few aggression measures provide separate indices of verbal and physical aggression, and very few measures discriminate among each of the four types of aggression; thus, individuals with very different aggression profiles may look quite similar.

Impulsive Versus Instrumental Aggression

The aggression literature also classifies aggression based on the motivation for the aggressive act. Impulsive aggression occurs in response to a perceived attack or provocation. It is motivated by anger and its primary goal is harm to another. In comparison, instrumental aggression is more thoughtful (premeditated), is less influenced by anger, and uses aggression as a means to obtain a goal (e.g., power) rather than as an end in itself (19). Despite this distinction in the literature, measures of aggression rarely discriminate between the two types. This is likely, in part, due to the difficulty in distinguishing between purely instrumental and purely impulsive acts. It has been suggested that most aggressive acts may have both impulsive and instrumental components, and that this dichotomy should be abolished (5). However, other studies have suggested that this distinction is valid and that separate neurological substrates may be involved in the two types of aggression (20).

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Though self-report frequency measures of general aggression do not distinguish between impulsive and instrumental aggression, two questionnaires have been developed to examine aggression within this nosology. The Expagg (21,22) is a 20-item questionnaire that assesses an individual's tendency to attribute his/her aggression as expressive (impulsive) or instrumental. The Aggressive Acts Questionnaire (23) asks 22 questions (e.g., "I felt guilty" and "the act was planned") to evaluate the impulsiveness and instrumentality of the four most extreme acts of aggression the participant engaged in over the past 6 months. Both measures have demonstrated reliability. However, the Aggressive Acts Questionnaire does not have any published validity estimates and the construct validity of the Expagg has been questioned (24). Thus, there are currently no well-validated measures of aggression nosology in the literature.

Defining Aggression: Summary

The lack of consensus in defining the typology and nature of aggression is reflected in the measures used to assess aggression. Instruments that purport to assess aggression can vary greatly in the types of behavior they sample. Distinctions between physical and verbal aggression, direct or indirect aggression, and instrumental versus impulsive aggression are often absent or blurred. This is understandable considering that the underpinnings of these distinctions are still under debate. However, knowledge of these issues will result in a more informed choice of aggression instrument for the clinician or researcher.

SELF-REPORT AGGRESSION QUESTIONNAIRES

Advantages and Limitations of Self-Report in Assessing Aggression

The utility of self-report measures in assessing behavior has been debated at length elsewhere (25) and will only be briefly addressed here. Retrospective self-report is subject to multiple forms of potential bias. Apart from the inherent difficulty in accurately remembering past behavior, which limits the validity of all self-report measures (26), self-report aggression scales are additionally hindered by a social desirability bias. Participants typically attempt to present themselves in a favorable way. Behaving aggressively is considered socially unacceptable in most situations. Therefore, participants will often underreport their aggressive behavior (27). This is especially true for more severe acts of aggression (28,29). Self-report measures have also been criticized as being so abstract compared to the actual behavior of interest (pencil marks vs. actual aggression) as to be of doubtful utility. Despite these criticisms, others have argued that the private and/or indirect nature of most aggressive behavior makes self-report ques-

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tionnaires the optimal method of studying such socially undesirable behavior (30). Furthermore, research comparing self-report and behavioral measures of aggression suggests that self-report measures provide a useful, albeit flawed measure of aggression (17).

General Aggression Questionnaires

The following measures assess an individual's general tendency to act verbally or physically aggressive. This is done either by measuring the frequency with which the individual engages in a set of aggressive behaviors, or by measuring the one propensity for behaving aggressively. The two most cited self-report questionnaires of general aggression are the Buss-Perry Aggression Questionnaire (previously Buss-Durkee Hostility Inventory) and the State Trait Anger Expression Inventory, which has recently been revised. Both of these measures, as well as three additional self-report questionnaires of trait aggression, are presented.

Buss-Perry Aggression Questionnaire (BPAQ)

The BPAQ (12) is the most frequently used self-report measure of aggression (31). Developed in 1992 as a revision to the Buss-Durkee Hostility Inventory (10), the BPAQ is a 29-item self-report instrument designed to measure physical and verbal aggressive tendencies as well as anger and hostility. The BPAQ requires participants to rate items on a scale from 1 ("extremely uncharacteristic of me") to 5 ("extremely characteristic of me"). Items 4 and 14 are reverse scored. Factor analysis revealed four factors: physical aggression—nine items; verbal aggression—five items; anger—seven items; and hostility—eight items. Aggression items almost exclusively assess direct aggression.

Using a sample of >1200 undergraduate students, Buss and Perry (12) demonstrated adequate internal consistency for the four scales (Physical Aggression [PA] = .85, Verbal Aggression [VA] = .72, Anger = .83, Hostility = .77, Total = .89). Later studies using undergraduate students (32,33) supported these findings. With the exception of verbal aggression (α = .50), similar results were also found using an offender population (34). Buss and Perry (12) had a subsample of 372 students complete the measure a second time 9 weeks after the initial administration of the BPAQ. Results showed acceptable test–retest reliability (PA = .80, VA = .76, Anger = .72, Hostility = .72, Total =. 80). Others studies have reported similar test–retest reliabilities (e.g., 33).

The four-factor structure of the BPAQ has also been supported via multiple confirmatory factor analyses (12,32,35,36). Some confirmatory factor analyses suggest that the four-factor solution would be further improved by deleting two of the hostility items (32,36), though others believe that the items contribute to

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the scale (37). One confirmatory factor analysis using an offender population suggested that a two-factor model combining Physical Aggression and Anger as one factor and Verbal Aggression and Hostility as the other would better fit their data (34).

Construct validity of the BPAQ is provided by significant correlations between BPAQ scales and peer rating of aggressiveness (PA = .45, VA = .20, Anger = .29, Hostility = .24, Total = .31) as well as with impulsiveness, competitiveness, and assertiveness (12). Significant correlations between the BPAQ and other self-report questionnaires of recent and lifetime aggression were also found among Hispanic and Anglo populations (38). BPAQ items also correlated with salivary testosterone and negatively correlated with a prosocial personality factor of empathy, nurturance, and altruism (39), and BPAQ scales were negatively correlated (−.19 to −.44) with the amount of time since the participant's last fight (40). Scales of the BPAQ have also correlated with aggression-related scales on the Personality Assessment Inventory and Aggression Inventory (33). The physical aggression scale of the BPAQ was found to predict enjoyment of hockey fights and self-reported propensity for escalating acts of crowd aggression among men watching a hockey game (41). The physical aggression subscale also predicted the number of aggressive penalties among high school hockey players (31).

Detailed norms using a stratified sample are not provided. However, the original article (12) does provide the mean and standard deviation of each scale for men and women separately. Means were based on a college sample of 1253 students (612 men, 641 women). The means and standard deviations for men were: Physical M = 24.4, SD = 7.7; Verbal = M = 15.2, SD = 3.9; Anger M = 17.0, SD 5.6; Hostility M = 21.3, SD 5.5; Total M = 77.8, SD 16.5. The means and standard deviations for women were: Physical M = 17.9, SD = 6.6; Verbal M = 13.5, SD = 3.9; Anger M = 16.7, SD 5.8; Hostility M = 20.2, SD 6.3; Total M = 68.2, SD = 17.0.

The BPAQ has been translated into multiple languages, including Swedish (42), Japanese (43), and Dutch (36). These versions appear to have reliabilities similar to the original BPAQ. In addition, a peer report version of the BPAQ has been developed with initial results suggesting adequate internal consistency and significant correlations with the original BPAQ among a group of 77 men (44).

The BPAQ is a frequently used measure of aggression that provides separate scales to assess the propensity for physical and verbal aggression. Strong evidence of the BPAQ's reliability and validity has been demonstrated via numerous studies using undergraduate and community samples. Results using offender populations (e.g., 34,45) are more equivocal.

State Trait Anger Expression Inventory 2 (STAXI-2)

The STAXI-2 (46) is a multidimensional self-report measure of anger and aggression. A recent revision, the STAXI-2 retains 42 of the original STAXI's

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(47) 44 items, as well as adding 15 additional items for a total of 57 items. The STAXI-2 and its predecessor were designed for and are commonly used in behavioral medicine research (46) and in anger treatment outcome research (48,49). All items are statements that participants endorse using a four-point scale, ranging from 1 (not at all/almost never) to 4 (very much so/almost always). The STAXI-2 has six scales: State Anger, Trait Anger, Anger Expression-Out, Anger Expression-In, Anger Control-Out, and Anger Control-In, as well as an Anger Expression Index. The development of three state anger subscales and the creation of an Anger Control-In scale constitute the major revisions of the STAXI-2. Factor analysis was used to determine item and scale/subscale selection for the STAXI-2.

The State Anger Scale (S-Ang), consisting of the first 15 items, asks the participants to describe how they feel "right now." The scale is comprised of three five-item subscales that assess general angry feelings (S-Ang/F), a desire to express anger verbally (S-Ang/V), and desire to express anger physically (S-Ang/P). Items 16–25, which comprise the Trait Anxiety Scale (T-Ang), ask participants to describe how they "generally feel." The four-item Trait Anger Temperament (T-Ang/T) subscale measures baseline levels of anger (e.g., I have a fiery temper), whereas the six-item Angry Reaction (T-Ang/R) subscale assesses anger reactivity to minor provocation (e.g. "get angry when slowed down by others' mistakes").

The final 32 items focus on the expression and inhibition of aggression when "angry or furious." The two eight-item anger expression scales measure the tendency to hold anger in or express it passively (e.g., pout or sulk) without actively aggressing (Anger Expression-In; AX-I) as well as the tendency to engage in aggression against others or objects (Anger Expression-Out; AX-O). This scale is weighted toward verbal aggression, with only one item likely to be assessing direct physical aggression ("strike out at whatever is infuriating"). The two eight-item anger control scales assess the frequency with which individuals use strategies to reduce their anger (Anger Control-In; AC-I) and aggression (Anger Control-Out: AC-O). The Anger Expression Index provides a measure of total anger expression. The index is derived by subtracting the summed anger control scales from the summed anger expression scales, and then adding 48 to the total to eliminate the possibility of negative numbers.

Research supports the reliability of the STAXI-2. Internal consistencies for the six scales and five subscales were adequate to excellent across gender among community (largely undergraduate) and psychiatric samples and (α = .73–.94). Test–retest reliabilities for the STAXI-2 were not provided in the test's otherwise comprehensive manual. Earlier versions of the STAXI demonstrated adequate test–retest reliability (.62–.81) with the exception of the S-Ang scale (.21–.27), which would not be expected to have high test–retest reliability (3).

Separate factor analyses of male (N = 667) and female (N = 977) nonpsychiatric (normal control) participants from the normative sample were used to

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derive STAXI-2 scales and subscales. There is significant overlap between the factor structures of the STAXI and STAXI-2. Factor analytic studies of the STAXI supported its factor structure (50,51). Similar confirmatory factor analytic studies will be needed to validate the proposed factor structure of the STAXI-2.

Validity studies of the STAXI-2 are still in development. However, studies have demonstrated the validity of the STAXI. Trait anger scale scores were found to correlate with anger measures such as Buss-Durkee Hostility Inventory and the Hostility and Overt Hostility scales of the Minnesota Multiphasic Personality Inventory (46). Trait Anger scores were each correlated with increased blood pressure (e.g., 52) and coronary heart disease (53). State and Trait Anger scale scores were also correlated with Neuroticism and Psychoticism scales of the Eysenck Personality Questionnaire, suggesting that individuals with greater levels of psychopathology have higher levels of anger (46). State and Trait anger scale scores also decreased after psychological anger intervention, demonstrating sensitivity to change (54).

The Anger Expression and Anger Control scales are most relevant to an aggression review, as the AX-O scale measures one's tendency to aggress when very angry and the AC-O scale measures attempts to restrict aggression. Studies have shown that AX-O scores correlate with physiological reactivity when provoked or harassed (55,56). AX-O scores have also been linked to myocardial infarction (57) and carotid artery arteriosclerosis (58). AX-I and AC were also found to discriminate between healthy individuals and those with coronary heart disease (59). PTSD veterans were found to have higher scores than non-PTSD Vietnam veterans on all anger expression and control scales (60), and within a community sample of maritally dissatisfied men with a history of violence scored higher on the AX-O scale and lower on the AC scale than a group of maritally dissatisfied but nonviolent men (61).

The STAXI-2 has a professional manual that includes norms for nonpsychiatric males and females (both separately and combined) aged 16 and older, as well as male and female psychiatric patients (separately) aged 18 and older.

The STAXI-2 is a revision and expansion of the oft-used STAXI. The STAXI has well-demonstrated validity and reliability. The STAXI-2 is well normed and internally consistent within scales; however, additional studies are needed to demonstrate the test–retest reliability and validity of this new revision. The STAXI-2 assesses both anger and aggression. State and Trait Anger scales assess angry feelings and the desire to behave aggressively. Anger Expression scales measure propensity for internalized (AX-I) and aggressive (AX-O) expressions of anger. The form of aggression measured by the AX-O scale is primarily direct and verbal. Thus, the STAXI-2 is not as comprehensive a measure of aggression as the BPAQ. However, the STAXI-2 also offers two Anger Control Scales to assess the frequency with which strategies are used to reduce aggressive responses (AC-O) and angry feelings (AC-I), which the BPAQ does not offer.

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Past Feelings and Acts of Violence (PFAV)

The PFAV was developed by Plutchik and colleagues (62) to assess risk of violence (physical aggression). The PFAV has been used primarily by its creators, and as a result has been validated on inpatient psychiatric populations (62,63). Based on the 36-item Feelings and Acts of Violence (64), the 12-item PFAV uses multiple-response formats. The first nine items have the four response options of 0 (never), 1 (sometimes), 2 (often), and 3 (very often). The first three of these items assess frequency of anger; the next six items assess frequency of violent behaviors and accessibility of weapons. Items 10 and 11 ask about history of aggressive and nonaggressive criminal behavior and have the response options never, once, twice, and more than two times. It is believed that these items are also coded 0 through 3, though this is not specifically stated (63). The final question asks if the person keeps weapons in the home that he/she knows how to use, to which the subject responds yes or no. It is not clear how this item is scored. The PFAV was shown to be internally consistent among a population of 100 psychiatric patients ($\alpha = .77$).

Evidence for the predictive validity of the PFAV comes from a strong agreement between documented history of violence and a determination of the patient as violent based on the PFAV in a sample of psychiatric inpatients. The determinations of violence on the PFAV are operationally defined as either 1), an endorsement (score of 1 or higher) on items 6 and 7, which collectively assess if the patient has ever "hit or attacked" another individual, or 2), endorsement (score of 1 or higher) on either item 8 (ever used a weapon to harm someone) or item 11 (arrested for a violent crime). The authors admit the definition is arbitrary, but "reasonable." Correlations between PFAV total score and documented history of violence were not given. Using the same definition of violence, the PFAV distinguished between a group of inpatients and a college control group. This time an additional comparison of total scale scores was performed, showing that the college control group had lower PFAV scores ($M = 3.88$, $SD = 2.56$) than the psychiatric inpatient ($M = 6.5$, $SD = 5.08$) group ($P < .001$). Using hit rate analysis, the authors found that a score of 5 on the PFAV provided 71% specificity and sensitivity in identifying psychiatric patients as violent versus nonviolent, whereas a score of 4 best discriminated violent from nonviolent college students (sensitivity and specificity each 75%).

Preliminary evidence supports the PFAV's utility in discriminating violent from nonviolent individuals, and it may provide a useful measure of physical aggression. However, the PFAV would benefit from an assessment of test–retest reliability, a more detailed explanation of how items are scored and additional validity studies (e.g., comparing the PFAV to other measures of aggression such as the BPAQ). Though the PFAV was designed as a tool for aggression research (63), it appears well suited for use as a brief violence screen in clinical settings.

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Aggression Inventory (AI)

Developed from the Olweus Multifaceted Inventory (65) to assess gender differences in aggression, the AI (66) is a 28-item self-report questionnaire in which each item is scored on a five-point Likert scale ranging from 1 (the statement does not apply to me at all) to 5 (the statement applies exactly to me). Based on exploratory factor analyses, Gladue has used 20-item, four-scale (physical aggression—four items; verbal aggression—seven items; impulsivity—seven items; and aggression avoidance—2 items) and 22-item, five-scale (physical aggression—seven items; verbal aggression—six items; impulsivity—four items; impatience—three items; and aggression avoidance—two items) versions of the AI. The two-item aggression avoidance scale has poor to adequate internal consistency (α = .28–.70) (35,66). Internal consistencies of the other scales ranged from .69 to .86. Test–retest reliabilities over a period of at least 14 weeks for the verbal aggression (r = .79), impulsivity (r = .67), aggression avoidance (r = .60), and impatience (r = .76) were marginal to adequate (33). Test–retest data were not found for the aggression scale or for any of the scales using the four-factor solution. Confirmatory factory analysis of the four-factor solution suggested that it may not be the optimal fit for the data (35).

Convergent validity for the AI is supported by significant correlations among the physical, verbal, and impulsive scales of the AI with all four scales of the BPAQ (r's = .18 [AI physical vs. BPAQ hostility] to .64 [AI physical vs. BPAQ physical, N = 320]. Aggression avoidance on the AI was also negatively correlated with BPAQ physical (r = −.29) and BPAQ verbal (r = −.20) aggression. Sex differences were also found on the AI, with men scoring higher than women on physical and verbal aggression scales in multiple studies (35,66). However, other researchers failed to replicate gender differences found by Gladue for aggression avoidance (66–68). Physical, verbal, and impulsive scales using the five-factor AI were positively correlated with testosterone level for men (r = .27–.31). Conversely, physical (r = −.51) and verbal (r = −.30) aggression scales were negatively correlated with testosterone level for women.

Though significant correlations with BPAQ scales are promising, the AI requires further psychometric development. A single version of the measure with a consistent scale structure and item-scale composition needs to be agreed upon and developed. The poor psychometric properties of the aggression avoidance scale will also need to be addressed.

Brief Anger-Aggression Questionnaire (BAAQ)

The BAAQ (68) is a six-item measure designed to provide rapid screening and identification of anger levels in violence prone men in environments such as emergency rooms and community clinic settings. The six items were designed to represent six of the seven scales from the Buss-Durkee Hostility Inventory

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(10)—assault, indirect hostility (interpreted as aggression against objects), irritability, negativism, resentment, and verbal hostility. Thus, three (assault, indirect hostility, and verbal hostility) of the six items ask about the likelihood of behaving aggressively. All items are scored on a five-point scale (0–4) with response options that range from extremely unlikely/not at all to very likely/very frequently. The assault question assesses likelihood of physical aggression. All other questions assess frequency of behaviors or feelings.

Internal consistency ($\alpha = .82$, N = 137) and test–retest reliability ($r = .84$, N = 44) were good. Construct validity for the scale was supported by a significant correlation between the BAAQ and BDHI scores ($r = .78$) among a sample of 137 men, the majority of whom had a history of violence. Criterion validity was supported by significantly (all P's $< .05$) higher scores among men with a history of domestic abuse (N = 30, M = 11.13, SD = 5.00), general assault (N = 26, M = 10.38, SD = 4.48), or both (N = 37, M = 11.51, SD = 4.92) as compared to a nonviolent control group (N = 26, M = 11.51, SD = 2.38). The BAAQ also was found to be sensitive to change as a result of group psychotherapy. A cut score of 9 was found to correctly identify 42 of 59 individuals with a history of violence (29% false-negative) and 15 of 15 nonviolent controls (0% false-positive). The BAAQ would benefit from additional studies showing its reliability and validity. However, preliminary results suggest the measure is an effective aggression screen, and potentially a useful outcome measure.

Domain-Specific Aggression Self-Report Questionnaires

The above questionnaires are intended to assess the frequency and/or likelihood of aggressive behavior in any setting. However, other questionnaires have been developed to assess aggression in specific circumstances. Two applications of particular interest to aggression researchers are partner violence and sexual aggression. The following is a review of some of the more often used self-report questionnaires to assess aggression in these contexts.

Partner Violence

Partner aggression and violence has been a primary area of interest to aggression researchers for some time. Studies using verbal reports of partner violence have been dominated by the use of the Conflict Tactics Scales (3). However, a number of other measures have been developed for this purpose.

Conflict Tactics Scales-2 (CTS-2). Introduced in 1996, the CTS-2 (69) was developed to address limitations of the Conflict Tactics Scales (70). Originally developed as an interview measure, the CTS and CTS-2 have been used in both interview and questionnaire formats. It is reported in this section because the CTS-2 was validated using the questionnaire version.

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The CTS-2 consists of four scales that measure the extent to which partners in a dating, cohabitating, or marital relationship engage in negotiation (six items), psychological aggression (eight items), physical assault (12 items), and sexual coercion (seven items). A fifth scale (six items) assesses the extent to which individuals were physically injured by their partner. The negotiation scale was conceptually divided into emotional and cognitive subscales. Other scales are similarly partialed into minor and severe subscales. For each of the 39 items, participants are asked to report how often they have engaged in the behavior, followed by how often their partner has engaged in the behavior. Response options range from 0 (never) to 6 (more than 20 times) for each of behavior "over the past year." Of the eight items on the physical aggression scale, six assess direct verbal aggression (e.g., "shouted at partner"), one assesses indirect physical aggression (destroying property), and one assesses covert aggression (refusing to discuss an issue). The sexual coercion scale comprised items assessing direct verbal (three items) and physical (two items) aggression, with one item for which this distinction was unclear (i.e., "made my partner have sex without a condom"). All physical assault items assess direct physical aggression.

Over half of the items on the CTS have been replaced or modified on the CTS-2. The sexual coercion and injury scales are new to the CTS-2, the result being that the CTS-2 is twice as long as its predecessor. Other changes in the CTS-2 include altering the wording of item stems from "his/her" to "my partner," and interspersing items randomly rather than ordering them hierarchically (69). Totals are derived by simple summing of coded responses. However, alternate forms of scoring have also been presented (69).

The CTS-2 was developed using a college sample of 317 students. Internal consistencies (69) for the CTS-2 scales are good (psychological aggression α = .79; negotiation α = 86; physical assault α = 86; sexual coercion α = .87) to excellent (injury α = .95). Marginal to excellent internal consistencies (α = .62–.91) were found for the CTS-2 scales in studies of incarcerated females (71) and high-risk postpartum women (72). No studies were found that provided test–retest reliability for the CTS-2.

Modest preliminary evidence of validity for the CTS-2 was demonstrated by significant correlations between subscales of the CTS-2 (e.g., physical assault–psychological aggression r = .71 [men] and .67 [women]), significantly stronger correlations between sexual coercion and other aggression related scales (i.e., injury, psychological aggression, physical assault) for men as compared to women, and poor correlations between negotiation and either sexual coercion or injury (69). Significant correlations were also found between general assault items and history of being abused in relationships among a sample of incarcerated women (71).

The dimensionality of the CTS-2 was examined in two studies. Confirmatory factor analyses of items from the negotiation, psychological aggression, and

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physical assault scales supported the factor validity of the negotiation scale and less definitively supported the factor structure of psychological aggression and physical assault scales (72). A second factor analytic study of incarcerated females suggested a four-factor solution that combined items from physical assault and psychological aggression (71).

The CTS-2 is the revised version of a measure that has helped shape much of what is believed about partner violence. The CTS-2 includes individual scales that, essentially, assess direct verbal aggression and physical aggression. Another scale assesses combined verbal and physical sexual aggression. The measure is internally consistent, and there is some evidence for the validity of the scales; however, the evidence of the measure's validity is not definitive. More validity studies are needed and will likely occur as the CTS-2 replaces its predecessor in research programs. The CTS-2 addressed some of criticisms of the CTS. Other criticism of the CTS not addressed by the CTS-2 are true of most aggression measures; namely, inability to measure patterns of violence, assessment of only a subset of aggressive behavior, insensitive scaling, and susceptibility to response bias (3).

Index of Spouse Abuse (ISA). The ISA (73) is a 30-item self-report scale developed to measure the severity of physical and nonphysical aggression inflicted on a woman by her spouse or partner. The ISA was designed for use in clinical settings to monitor and evaluate spouse abuse treatment progress. Items on the ISA are rated on a scale from 1 (never) to 5 (very frequently). Factor analytic studies suggest that the items break down into two scales—a nonphysical abuse scale (19 items; e.g., my partner orders me around), and a physical abuse scale (11 items; e.g., my partner punches me with his fists). Individual items are weighted to produce a score between 0 and 100 for both physical and nonphysical abuse. The scale takes $\sim$ 5 min to complete and is easy to score. Internal consistency for each of the scales is strong for both nonvictim (nonphysical $\alpha = .91$, physical $\alpha = .90$) and victim (nonphysical $\alpha = .94$, physical $\alpha = .97$) samples. Test–retest reliabilities were not found.

Validity was assessed via discriminant and convergent validity. Significant correlations were found between the ISA and other problems related to spouse abuse (e.g., marital problems, depression) and poor, nonsignificant correlations were found between the ISA and theoretically unrelated variables (age, selected social and personal problems). Concurrent validity was also demonstrated by showing ISA cutoff scores (10 for physical abuse, 25 for nonphysical abuse) discriminated spouse abuser from nonabusers with $> 90\%$ accuracy. Similar results have been found in other studies using the ISA (e.g., 74). Correlations between ISA scores and clinical abuse status were .73 and .80 for physical and nonphysical scales, respectively (73).

The ISA appears to be a valid, reliable measure of partner violence. Though

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possibly long for a screening tool, the psychometric properties of the ISA have made it a popular measure in medical settings. Some have reported that the ISA has been used as an interview. However, psychometric data on the interview version of the ISA were not found.

Abusive Behavior Inventory (ABI). The ABI (75) was developed to measure the physical and psychological abuse committed by men toward their female partners. The ABI is a 30-item self-report instrument that uses a five-point ("never" to "very frequently") Likert scale to measure the frequency of abusive behavior during a 6-month period. There are identical forms for men and women, except that the different pronouns focus on the abusive behavior by men only. Therefore, it reflects a feminist perspective, which guides most partner abuse programs. Unlike the CTS-2, the ABI does not set the violence in the context of family disagreement or the result of attempts to deal with conflict. The author's rationale is that violence is used to maintain dominance, not resolve conflict.

The ABI contains a physical abuse scale and a psychological abuse scale. The physical abuse scale (ABI-Physical) consists of 10 items assessing assaultive behaviors, including three items that assess sexual assault. The psychological abuse scale (ABI-Psychological) consists of 20 items that assess emotional abuse, isolation, intimidation, and threats. The two scales were shown to be internally consistent with samples of male abusers (ABI-Psychological $\alpha = .82$, ABI-Physical $\alpha = .88$) and nonabusers (ABI-Psychological $\alpha = .79$, ABI-Physical $\alpha = .82$) and females with (ABI-Psychological $\alpha = .88$, ABI-Physical $\alpha = .70$) and without (ABI-Psychological $\alpha = .92$, ABI-Physical $\alpha = .88$) a history of being abused. Validity for the measure was supported by significant differences between individuals with and without abuse histories on both physical and psychological scales. Furthermore, significant correlations were found between ABI scores and other abuse measures, and correlations of ABI scores with age or household size were not significant, indicating that the measure has convergent and discriminant validity (75). The ABI has been used in several research studies, and has been found to be a good measure of potential abusive behavior.

Other Partner Violence Questionnaires. The aforementioned measures of partner violence do not represent a comprehensive list. Two other partner violence questionnaires found repeatedly in the literature were the Psychological Maltreatment of Women Inventory (76) and Severity of Violence Against Women Scales (77). Space limitations prevent a detailed discussion of these instruments. However, basic information about the instruments is presented in Table 2.

Sexual Aggression

Some of the partner violence questionnaires have items or scales that assess sexual aggression. However, few measures are devoted exclusively to the assessment

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of sexually aggressive behavior. Below are two measures of sexual aggression. Of these, the questionnaire that dominates the sexual aggression research appears to be the Sexual Experiences Survey.

Sexual Experiences Survey (SES). Originally developed to evaluate the prevalence of sexual aggression among college students, the SES (78,79) is the most commonly used self-report measures of sexual aggression (80). The SES comprises 10 behavioral items that assess the engagement in sex play, attempted intercourse, and achieved intercourse since age 14 using the following three types of aggressive behavior: coercion, abuse of authority, and physical force (e.g., "Have you engaged in sex play with a woman when she didn't want you to"). The 10th item assesses physically forced sodomy. Items are arranged hierarchically. Some studies have used frequency scales to score the SES (81). However, the original reliability and validity studies for the SES were conducted using a yes/no format. Degree of sexual perpetration/victimization can be assessed either by adding all affirmative responses or by recording the highest level of abuse endorsed. The SES has two parallel forms—one to assess male perpetration, and one to assess female victimization. However, modifications of the SES have been developed that assess female sexual aggression and/or male victimization (80,82).

Internal consistency (women $\alpha = .74$; men $\alpha = .89$) and test-retest reliability ($r = .93$) for the SES appear strong (78). However, these psychometric indices may be biased by a low base rate of yes responses. A study using a German adaptation of the SES (83) found that temporal stabilities for yes responses were poor (46% for female respondents, $N = 84$; 42% male for male respondents, $N = 27$) despite high overall test–retest reliability ($r = .95$, $N = 1028$).

Concurrent and construct validity have been demonstrated via significant correlations between the SES and other self-report or interview measures of sexual aggression. The SES has been shown to correlate ($r = .54$), with the Coercive Sexuality Scale (84), as well as measures of nonsexual aggression and relationship distress (66,67). Koss (78) showed significant correlation between the SES and the subsequently administered SES interview for women ($r = .73$; $N = 242$) and men ($r = .61$; $N = 144$). Though both men and women were much more likely to report less sexual aggression/victimization when interviewed than when filling out the SES self-report, the difference was more striking for men. A second study using only men found a similar pattern of results (84), suggesting that for behaviors strongly societally eschewed such as sexual aggression, there is a tendency to report less sexual aggression during a clinical interview than when completing a self-report.

The SES is a quick, easily administered self-report measure of sexual aggression (male) or victimization (female). The SES, when given as a self-report, appears to be a reliable measure, though reliability may be less convincing among

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samples of individuals with sexual aggression histories. Evidence of validity does exist for the SES. However, considering the extent to which this scale is used, it is somewhat surprising that more validation studies have not been performed. That being said, the SES is the best validated of the unidimensional sexual aggression scales examined. Also, unlike most other sexual aggression scales, the SES also has versions to assess male victimization and female perpetration, though those scales are not as well validated as the original SES.

Coercive Sexuality Scale (CSS). Of the other sexual aggression measures, the Coercive Sexuality Scale (85) is one of the more common. The CSS is an 18 item self-report measure of coercive sexual behaviors. The CSS measures coercive sexual behavior and coercive sexual methods using a four-point scale (never, once or twice, several times, often). The CSS has been shown to have high reliability ($\alpha = .96$).

Concurrent validity for the CSS was shown by significant correlations between self-report measures of aggressive sexual beliefs and acceptance of violence (85) as well as the SES (84). Additional validity for the measure was supported by a finding that CSS scores discriminated between groups of men who would and would not show violent sexual films to a female subject while participating in a laboratory analogue of sexual aggression (86). The CSS would benefit from estimates of test–retest reliability. However, it was the only sexual aggression measure found that was validated against an observed behavioral measure.

AGGRESSION INTERVIEWS

In comparison to questionnaires, fewer interviews exist to assess the retrospective self-report of one's aggression. The CTS (presented above) is a measure of partner aggression that has often been used as both a questionnaire and an interview. Because of a dearth of general aggression interviews, the author (E.F.C.) has developed two measures—one to assess trait aggressiveness (Life History of Aggression), and one to assess frequency of aggression over a 1-week span (Overt Aggression Scale-Modified for Outpatient Use). The author is also currently developing a clinical interview to assess the phenomenology of aggression as it relates to Intermittent Explosive Disorder.

Interview Versus Questionnaire Measures of Aggression

Interview measures, like questionnaires, are dependent on the retrospective self-report of the participant and are thus subject to the same threats to validity. However, the interpersonal aspect of the interview creates unique advantages and disadvantages. As stated earlier, social desirability bias may lead to under-

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reporting of aggressive behavior. This bias may be accentuated for interviews, where the participant has to directly relay this information to another individual. Indeed, when the SES was administered first as a questionnaire, then subsequently as an interview, there was a strong tendency to report fewer acts of sexual aggression during the interview (78). The use of interviews also requires some level of training for the interviewer, the level of training being dependent on the complexity of the interview and the concepts assessed, and produces another potential source of error variance. However, well-trained interviewers can assess nonverbal behavior, clarify questions, and request additional information to arrive at a more accurate response, distinct advantages over questionnaire measures.

Overt Aggression Scale-Modified for Outpatient Use (OAS-M)

The OAS-M (87) is one of three measures that were based on the Overt Aggression Scale (OAS) (88). This modification of the OAS was developed to measure treatment outcome in an outpatient population. The largest difference between the OAS-M and OAS or its other modifications is that the OAS-M is not an observational scale, but rather an interview that is based on the subject's retrospective self-report of the past week, whereas the other two measures are based on behavioral observation, something that is less feasible when treating an outpatient population.

The OAS-M is a semistructured interview that assesses four clusters of aggressive behavior: verbal assault, assault against objects, assault against others, assault against self. Within each behavior cluster, the OAS-M distinguishes five levels of aggression severity in each group that are weighted 1 (e.g., "snapped or yelled") through 5 (e.g., "threatened to hit a stranger"). The OAS-M obtains the frequency of aggressive episodes that involve each of these types of behavior. Using the OAS convention, an episode is operationally defined as a period of aggression followed by a 30-min or greater period of no aggression. The frequency of each behavior within an aggression category is multiplied by its weight and then summed. This number is multiplied by the categories weight (1 for verbal assault, 2 for assault against objects, and 3 for both assault against others and assault against self) to derive a weighted category score. The four weighted category scores are summed to arrive at an aggression score. The choice of weights for both aggression clusters and aggressive behaviors within a cluster was determined rationally.

The OAS-M also includes a two-item irritability scale and a three-item suicidality scale. The irritability scale assesses subject evaluation and behavioral presentation of irritability. Irritability items are scored 0 (not at all) to 5 (extreme). Suicidal items assess ideation and (if an attempt has occurred) intent and lethality of suicide attempt. Suicidal ideation is scored on a scale of 0 (no ideation) to 6

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(very extreme). Other suicidal items are assessed on a 0-to-5 scale. Both scales were based on questions from the Schedule for Affective Disorders and Schizophrenia (89). Examples of behaviors that would meet scoring criteria are presented next to each item option on the OAS-M.

The OAS-M was administered to 22 psychiatric patients to evaluate the psychometric properties of the scale (87). Interrater reliability for total aggression and irritability scales were high (both ICC $>$.90). The suicidality scale was unable to be assessed owing to a lack of variance in scores. One-week test-retest reliability scores for total aggression (ICC = .46) and irritability (ICC = .54) were low, which was not unexpected for a state measure. Internal consistency was not assessed. Some evidence of validity for the measure is demonstrated by significant correlations between aggression and irritability scales (r = .53, P < .011), and between OASM irritability and specific verbal assault items with a questionnaire measure of irritability and assault. Somewhat stronger evidence of construct validity was evidenced by significant correlations between the scores on the OAS-M aggression scale and the Life History of Aggression total (r = .45) and aggression (r = .52) scales among a group of personality disordered subjects (90). OAS-M aggression and irritability scales have also been shown to be sensitive to changes in aggression associated with pharmacotherapy (91).

The scale takes 10–20 min to administer and requires some clinical savvy in addition to training on the OAS-M scoring system. It is suggested that persons administering the instrument have previous clinical assessment experience. A training manual is available from the author.

The OAS-M was an attempt to fill a void in aggression assessment, and shows promise as an aggression treatment outcome measure. However, most of the OAS-M's psychometric properties are either preliminary or undetermined. The measure would greatly benefit from improved scoring guidelines and significant additional validation research.

Life History of Aggression (LHA)

The LHA (90) is a semistructured interview that assesses frequency of aggressive and antisocial behaviors since adolescence. The LHA was adapted from the Brown-Goodwin Aggression scale for military personnel (92). The LHA consists of three subscales: a five-item aggression (temper tantrums, verbal aggression, fighting, any physical assault, and destruction of property) subscale; a four-item antisocial behavior (school behavioral problems, problems with supervisors, antisocial behavior not involving the police and antisocial behavior that did involve the police) subscale; and a two-item self-aggression (suicidal behavior and self-injurious behavior) subscale. All items are rated on a six-point scale based on the total number of occurrences of the behavior since the age of 13. Scores are coded as follows: 0 (no occurrences), 1 (one event), 2 (two or three events) 3

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(four to nine events), 4 (10 or more events recalled) or 5 (more events than can be counted). Each subscale score is derived by simply adding the scores from each item comprising the subscale. The total score is derived by adding the scale scores. However, the total score is not often used in studies that employ the LHA. The measure takes ~5–10 min to administer.

Psychometric properties of the LHA were obtained using a mixed sample of personality disordered (N = 165) and "normal" (i.e., no Axis I or II psychopathology; N = 63) volunteers. Total, Aggression and Antisocial Behavior scales exhibit good to adequate internal consistency, with α coefficients of .88, .87, and .74, respectively. Internal consistency for the self-aggression scale is poor (α = .48). The LHA demonstrated high interrater agreement for total score and subscales with ICCs of .95 (total), .94 (aggression), .88 (antisocial behavior), and .84 (self-aggression; N = 48). Test–retest reliability was also strong with coefficients of stability (mean test–retest interval = 146.5 days; SD = 91.7 days; N = 20) of .91 (total), .80 (aggression), .89 (antisocial behavior), and .97 (self-aggression). Evidence for the validity of the LHA, particularly the aggression scale, is based on significant correlations of LHA total and LHA aggression scales with other self-report and interview measures of aggression. LHA aggression scores also discriminated between personality-disordered and non-personality-disordered subjects ($P < .001$). In addition, other studies have shown that LHA aggression scores are correlated ($r = .56$, $P < .05$) with laboratory measure of aggressive behavior (93), and that individuals who score high on the LHA aggression scale are more likely to respond to tryptophan depletion with increased aggression (94). Variants of the LHA to assess the frequency of witnessed and experienced aggression are currently in development.

The LHA would benefit from a revised self-aggression scale and additional validation studies (including factor analytic studies to better explore the underlying nature of the measure). However, early research suggests that the aggression scale of the LHA is a valid, reliable measure of direct aggression that is inclusive of both verbal and physical aggression.

Intermittent Explosive Disorder (IED) Interview

IED is a behavioral disorder that is defined by acts of impulsive aggression. The authors have developed a structured diagnostic interview of aggression that assesses the lifetime and current frequency of verbal aggression, aggression against property, and physical aggression. Contextual descriptive information (e.g. "what was the provocation," and "what were the consequences of this outburst") about the three most serious episodes of each type of aggression during the 1-year period in which it occurred most frequently is elicited. Additional phenomenological information about aggressive acts are also obtained, including but not limited to: age of onset and offset of each type of aggression; the effects of the aggressive

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behaviors on relationships with family and friends; subjective level of distress; emotions and physical symptoms prior to and after an outburst; and frequency of substance use during aggressive outbursts. The interview was designed to allow for the diagnosis of IED using either the DSM criteria or integrated research criteria for IED (95). The interview takes ~20–30 min to complete. Psychometric information for this measure is still in the process of being collected.

CONCLUSIONS

Self-report measures provide a quick, easily accessible method of learning about aggressive behavior. A number of measures, most notably the BPAQ, do an adequate job of assessing general aggression, and instruments such as the CTS have been shown to be valid, reliable measures of aggression in specific contexts. However, there are numerous pitfalls that continue to exist with questionnaires and interview measures of aggression.

One set of obstacles, the limitations of retrospective self-report, is universal to self-report research and can only be partially minimized by the continuing efforts to improve a measure's test–retest reliability. Unfortunately, test–retest reliability was noticeably lacking for many of the measures discussed, particularly the partner violence measures. A second limitation of self-report measures more specific to behaviors such as aggression is the likelihood that individuals intentionally misrepresent their history of aggression. To this end, additional validation of measures against observed aggression, either in naturalistic or in laboratory settings, is needed.

A final area of concern is the paucity of measures that assess the full span of aggressive behaviors. Aggressive behavior can be direct or indirect, passive or active, physical or verbal, instrumental and/or reactive. However, current aggression self-report methods typically assess only direct aggression (often predominantly verbal or physical), and little to no consideration is usually given to other dimensions such as instrumental versus impulsive. The field would be advanced by a multidimensional aggression inventory that includes measures of all of these dimensions in a single instrument. In sum, while currently available measures of aggression appear adequate, these measures can be improved upon with further development and research.

REFERENCES

1. P Bech, M Mak. Measures of impulsivity and aggression. In: E Hollander, DJ Stein, eds. Impulsivity and Aggression. New York: John Wiley & Sons, 2002, pp. 25–41.
2. D Zillman. Hostility and Aggression. Hillsdale, NJ: Erlbaum, 1979.
3. JW White, P Hall-Smith, MK Koss, AJ Figueredo. Intimate partner aggression: what have we learned? Psychol Bull 126:690–696, 2000.

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

4. J Archer. Sex differences in aggression between heterosexual partners: a meta-analytic review. Psychol Bull 14:651–680, 2000.
5. BJ Bushman, CA Anderson. Is it time to pull the plug on the hostile versus instrumental aggression dichotomy. Psychol Rev 108:273–279, 2001.
6. MK Biaggio, RD Maiuro. Recent advances in anger assessment. In: Spielberger C.D., Butcher J.N., eds. Advances in Personality Assessment, Vol. 5. Hillsdale, NJ: Erlbaum, 1985, pp. 71–111.
7. H Kassinove, DG Sukhodolsky. Anger disorders: basic science and practice issues. In: H Kassinove, ed. Anger Disorders: Definition, Diagnosis, and Treatment. Washington: Taylor & Francis, 1995, pp. 1–26.
8. CI Eckhardt, J Deffenbacher. Diagnosis of anger disorders. In: H Kassinove, ed. Anger Disorders: Definition, Diagnosis, and Treatment. Washington: Taylor & Francis, 1995, pp. 27–49.
9. JR Averill. Anger and Aggression: An Essay on Emotion. New York: Springer-Verlag, 1982.
10. A Buss, A Durkee. An inventory for assessing different kinds of hostility. J Consult Clin Psychol 21:343–349, 1957.
11. AH Buss, Fischer H., AE Simmons. Aggression and hostility in psychiatric patients. J Consult Psychol 26:84–89, 1962.
12. A Buss, M Perry. The aggression questionnaire. J Pers Soc Psychol 63:452–459, 1992.
13. AJ Bond. Pharmacological manipulation of aggressiveness and impulsiveness in healthy volunteers. Prog Neuro-Psychopharmacol Biol Psychiatry 16:1–7, 1992.
14. LM Range, EC Knott. Twenty suicide assessment instruments: evaluation and recommendations. Death Studies 21:25–34, 1997.
15. GK Brown. A Review of Suicide Assessment Measures for Intervention Research with Adults and Older Adults. National Institute of Mental Health, 2002.
16. RG Geen. Human Aggression. Pacific Grove, CA: Brooks/Cole, 1990.
17. BJ Bushman, CA Anderson. Methodology in the study of aggression: integrating experimental and nonexperimental findings. In: RG Geen, E Donnerstein, eds. Human Aggression: Theories, Research, and Implications for Social Policy. New York: Academic Press, 1998, pp. 23–48.
18. N Crick, JK Grotpeter. Relational aggression, gender, and social-psychological adjustment. Child Dev 66:710–722, 1995.
19. EF Cocarro. Aggression. In: G Fink, ed. Encyclopedia of Stress. San Diego: Academic Press, 2000.
20. ES Barrat, MS Stanford, AR Felthous, TA Kent. The effects of phenytoin on impulsive and premeditated aggression: a controlled study. J Clin Psychopharmacol 17: 341–349, 1997.
21. A Campbell, S Muncer, E Coyle. Social representation of aggression as an explanation of gender differences. Aggress Behav 18:95–108, 1992.
22. A Campbell, S Muncer, IC McManus, D Woodhouse. Instrumental and expressive representation of aggression: one scale or two? Aggress Behav 25:435–444, 1999.
23. ES Barrat, MS Stanford, L Dowdy, MJ Liebman, TA Kent. Impulsive and premeditated aggression: a factor analysis of self-reported acts. Psychiatry Res 86:163–173, 1999.

MARCEL DEKKER, INC.
270 Madison Avenue, New York, New York 10016

24. S Forrest, M Shevlin, V Eatough, M Gregson, MNO Davies. Factor structure of the expagg and revised expagg: failure to replicate using confirmatory factor analysis. Aggress Behav 28:11–20, 2002.
25. RL Piedmont, RR McCrae, R Reimann, A Angleitner. On the invalidity of validity scales: evidence from self-report and observer ratings in volunteer samples. J Pers Soc Psychol 78:593, 2000.
26. A Baddeley. The limitations of human memory: implications for the design of retrospective surveys. In: L Moss, H Goldstein, eds. The Recall Method in Social Surveys. London: University of London Institute of Education, 1979, pp. 13–27.
27. P Bech. Rating Scales for Psychopathology, Health Status, and Quality of Life. A Compendium on Documentation in Accordance with the DSM-III-R and WHO Systems. Berlin: Springer-Verlag, 1993.
28. RT Ammerman, M Hersen. Current issues in the assessment of family violence: an update. In: Assessment of Family Violence: A Clinical and Legal Sourcebook. New York: Wiley, 1999, pp. 3–23.
29. C Wekerle, DA Wolfe. Dating violence in mid-adolescence. Clin Psychol Rev 19: 435–456, 1999.
30. RA Barron, D Richardson. Human Aggression, Vol. 2. New York: Plenum Press, 1994, pp. 39–85.
31. BJ Bushman, GL Wells. Trait aggressive and hockey penalties predicting hot tempers on the ice. J Appl Psychol 83:969–974, 1998.
32. JA Harris. Confirmatory factor analysis of the aggression questionnaire. Behav Res Theory 33:991–993, 1995.
33. JA Harris. A further evaluation of the aggression questionnaire: issues of validity and reliability. Behav Res Ther 35:1047–1053, 1997.
34. TY Williams, JC Boyd, MA Cascardi, N Poythress. Factor structure and covergent validity of the aggression questionnaire in an offender population. Psychol Assess 8:398–403, 1996.
35. J Archer, G Kilpatrick, R Bramwell. Comparison of two aggression inventories. Aggress Behav 21:371–380, 1995.
36. C Meesters, P Muris, H Bosma, E Schouten, S Beuving. Psychometric evaluation of the Dutch version of the aggression questionnaire. Behav Res Ther 34:839–843, 1996.
37. IH Bernstein, RP Gesn. On the dimensionality of the Buss/Perry Aggression Questionnaire. Behav Res Theory 35:563–568, 1997.
38. MB Harris. Aggressive experiences and aggressiveness: relationship to ethnicity, gender, and age. J Appl Soc Psychol 26:843–870, 1996.
39. JA Harris, JP Rushton, E Hampson, DN Jackson. Salivary testosterone and self-report aggressive and pro-social personality characteristics in men and women. Aggress Behav 22:321–331, 1996.
40. J Archer, R Holloway, K McLoughlin. Self-reported physical aggression among young men. Aggress Behav 21:325–342, 1995.
41. MB Harris, K Knight-Bonhoff. Gender and aggression II: personal aggressiveness. Sex Roles 35:27–42, 1996.
42. H Prochazka, H Agren. Aggression in the general Swedish population, measured with a new self-rating inventory: The Aggression Questionnaire-Revised Swedish version (AQ-RSV). Nord J Psychiatry 55:17–23, 2001.

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43. K Nakano. Psychometric evaluation on the Japanese adaptation of the Aggression Questionnaire. Behav Res Ther 39:853–858, 2001.
44. DB O'Conner, J Archer, FWC Wu. Measuring aggression: self-reports, partner reports, and responses to provoking scenarios. Aggress Behav 27:79–100, 2001.
45. J Archer, AM Haigh. Do beliefs about aggressive feelings and actions predict reported levels of aggression? Br J Soc Psychol 36:83–105, 1997.
46. CD Spielberger. STAXI-2 State Trait Anger Expression Inventory-2, Professional Manual, Odessa, FL: Psychological Assessment Resources, 1999.
47. CD Spielberger. Manual for the State-Trait Anger Expression Inventory, Odessa, FL: Psychological Assessment Resources, 1988.
48. J Deffenbacher, DA Story, AD Brandon, JA Hogg. Cognitive and cognitive-relaxation treatments of anger. Cogn Ther Res 12:167–184, 1988.
49. J Deffenbacher, K McNamara, RS Stark, PM Sabadell. A comparison of cognitive-behavioral and process-oriented group counseling for general anger reduction. J Couns Dev 69:167–172, 1990.
50. DR Fuqua, E Leonard, MA Masters, RL Smith, JL Campbell, PC Fischer. A structural analysis of the state trait anger expression inventory. Educ Psychol Meas 51: 439–446, 1991.
51. HM Van der Ploeg. The factor structure of the state trait anger scale. Psychol Rep 63:978, 1988.
52. JH Markovitz, KA Matthew, RR Wing, LH Kuller, EN Meilahn. Psychological, biological and health behaviour predictors of blood pressure changes in middle-aged women. J Hypertens 9:399–406, 1991.
53. GD Bishop, SH Quah. Reliability and validity of measures of anger/hostility in Singapore: Cook & Medley Ho Scale, STAXI and Buss-Durkee Hostility Inventory. Pers Indiv Diff 24:867–878, 1998.
54. ML Lanza, H Satz, J Stone, HL Kayne. Assessing the impact of group treatment for aggressive inpatients. Group 19:195–219, 1995.
55. S Boyle and AW Siegman. Dimension of anger expression and CVR in angered men. University of Maryland, College Park, unpublished doctoral dissertation, 1992.
56. TO Engebretson, KA Matthews, MF Scheier. Relations between anger expression and cardiovascular reactivity: reconciling inconsistent findings through a matching hypothesis. J Pers Soc Psychol 57:513–521, 1989.
57. CF Mendes de Leon. Anger and impatience/irritability in patients of low socioeconomic status with acute coronary heart disease. Behav Med 15:273–284, 1992.
58. J Julkunen, R Salonen, GA Kaplan, MA Chesney, JT Salonen. Hostility and the progression of carotid atherosclerosis. Psychosom Med 56:519–525, 1994.
59. M Atchison, J Condon. Hostility and anger measures in coronary heart disease. Aust NZ J Psychiatry 27:436–442, 1993.
60. NB Lasko, TV Gurvits, AA Kuhne, SP Orr, RK Pitman. Aggression and its correlates in Vietnam veterans with and without chronic posttraumatic stress disorder. Compr Psychiatry 35:373–381, 1994.
61. KA Barbour, C Eckhardt. The experience and expression of anger in maritally violent and maritally discordant-nonviolent men. Behav Ther 29:173–191, 1998.
62. R Plutchik, H Van Praag, HR Conte. Suicide and violence risk in patients. In: C Shagass, ed. Biological Psychiatry. New York: Elsevier, 1986, pp. 761–763.

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63. R Plutchik, HM van Praag. A self-report measure of violence risk. II. Compr Psychiatry 31:450–456, 1990.
64. R Plutchik, C Climent, R Ervin. Research strategies for the study of human violence. In: WL Smith, A Kling, eds. Issues in Brain/Behavior Control. New York: Spectrum, 1976.
65. D Olweus. Aggression and hormones: behavior relationships with testosterone and aggression. In: D Olweus, J Block, M Radke-Yarow, eds. Development of Antisocial and Prosocial Behavior. Orlando: Academic Press, 1986.
66. BA Gladue. Qualitative and quantitative sex differences in self-reported aggressive behavioral charecteristics. Psychol Rep 68:675–683, 1991.
67. BA Gladue. Aggressive behavioral characteristics, hormones, and sexual orientation in men and women. Aggress Behav 17:313–326, 1991.
68. RD Maiuro, PP Vitaliano, TS Cahn. A brief measure for the assessment of anger and aggression. J Interpers Violence 2:166–178, 1987.
69. MA Strauss, SL Hamby. The revised Conflict Tactics Scales (CTS2): development and preliminary psychometric data. J Fam Issues 17:283–316, 1996.
70. MA Strauss. Measuring intrafamily conflict and violence in the family. J Marriage Fam 41:75–88, 1979.
71. NT Jones, P Ji, M Beck, N Beck. The reliability and validity of the Revised Conflict Tactics Scale (CTS2) in a female incarcerated population. J Fam Issues 23:441–457, 2002.
72. RR Newton, CD Connelly, JA Landsverk. An examination of measurement characteristics and factorial validity of the revised conflict tactics scale. Educ Psychol Meas 61:317–3350, 2001.
73. WW Hudson, SR McIntosh. The assessment of spouse abuse: two quantifiable dimensions. J Marriage Fam 43:873–888, 1981.
74. AA Ernst, SN Weiss, TG Nick, J Casalletto, A Garza. Domestic violence in a university emergency department. South Med J 93:176–177, 2000.
75. MF Shepard, JA Campbell. The abusive behavior inventory: a measure of psychological and physical abuse. J Interpers Violence 7:291–305, 1992.
76. RM Tolman. The development of a measure of psychological maltreatment of women by their male partners. Violence Victims 4:159–177, 1989.
77. LL Marshall. Development of the severity of violence against women scales. J Fam Violence 7:103–121, 1992.
78. MP Koss, CA Gidycz. Sexual Experiences Survey: reliability and validity. J Consult Clin Psychol 53:422–423, 1985.
79. MP Koss, C Oros. Sexual Experiences Survey: a research instrument investigating sexual aggression and victimization. J Consult Clin Psychol 50:455–457, 1982.
80. KM Ryan. The relationship between courtship violence and sexual aggression in college students. J Fam Violence 13:377–394, 1998.
81. NM Malamuth, RJ Sockloskie, MK Koss, JS Tanaka. Characteristics of aggressors against women: testing a model using a national sample of college students. J Consult Clin Psychol 59:670–681, 1991.
82. PB Anderson. Correlates of college women's self-reports of heterosexual aggression. Sexual Abuse: A Journal of Research and Treatment 8:121–131, 1996.
83. B Krahe, T Reimer, R Scheinberger-Olwig, I Fritsche. Measuring sexual aggression:

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the reliability of the sexual experiences survey in a German sample. J Interpers Violence 14:91–100, 1999.
84. PC Ouimette, J Shaw, JF Drozd, J Leader. Consistency of reports of rape behaviors among nonincarcerated men. Psychol Men Masc 1:133–139, 2000.
85. K Rapaport, BR Burkhart. Personality and attitudinal characteristics of sexually coercive college males. J Abnorm Psychol 93:216–221, 1984.
86. GC Nagayama Hall, R Hirschman. The relationship between men's sexual aggression inside and outside of the laboratory. J Consult Clin Paychol 62:375–380, 1994.
87. EF Coccaro, PD Harvey, E Kupsaw-Lawrence, JL Herbert, DP Bernstein. Development of neuropharmacologically based behavioral assessments of impulsive aggressive behavior. Assess Impuls Aggress Behav 3:S44–S51, 1991.
88. SC Yudofsky, JM Silver, W Jackson, J Endicott, D Williams. The overt aggression scale for the objective rating of verbal and physical aggression. Am J Psychiatry 143:35–39, 1986.
89. RL Spitzer, J Endicott. Schedule for Affective Disorders and Schizophrenia. New York: New York State Psychiatric Institute, 1978.
90. EF Coccaro, ME Berman, RJ Kavoussi. Assessment of life history of aggression: development and psychometric characteristics. Psychiatry Res 73:147–157, 1997.
91. RJ Kavoussi, EF Coccaro. Divalproex sodium for impulsive aggressive behavior in patients with personality disorder. J Clin Psychiatry 59:676–680, 1998.
92. GL Brown, FK Goodwin, JC Ballenger, PF Goyer, LF Major. Aggression in human correlates with cerebrospinal fluid amine metabolites. Psychiatry Res 1:131–139, 1979.
93. EF Coccaro, RJ Kavoussi, TB Cooper, RL Hauger. Relationship of prolactin response to D-fenfluramine to behavioral and questionnaire assessments of aggression in personality disordered males. Biol Psychol 40:157–164, 1996.
94. DM Dougherty, JM Bjork, DM Marsh, FG Moeller. Influence of trait hostility on tryptophan depletion-induced laboratory aggression. Psychiatry Res 88:227–232, 1999.
95. EF Coccaro. Intermittent explosive disorder. Cur Psychiatry Rep 2:67–71, 2000.

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11

Laboratory Measures

The Taylor Aggression Paradigm

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INTRODUCTION

The individual and social costs associated with violence, and the consequent desire to understand and reduce aggressive behavior, have made aggression a popular area of scientific research. Experimental designs are an excellent means to control extraneous and confounding variables, and thus have the potential to allow research with a high degree of internal validity. In addition, experimental methodologies allow for the testing of causal hypotheses. Accordingly, researchers have developed behavioral analogs to experimentally examine various correlates of aggressive behavior under controlled, laboratory conditions. Of these measures, the most well known are the Buss Aggression Machine (BAM) (1), the Point Subtraction Aggression Paradigm (PSAP) (2), and the Taylor Aggression Paradigm (TAP) (3). Over the past 40 years, these measures have generated a

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great deal of knowledge about aggression and have been both praised (4) and criticized (5) for their effect on the development of the aggression research field. The present chapter will discuss the utility of laboratory measures of aggression, focusing on the Taylor Aggression Paradigm (TAP).

LABORATORY MEASURES OF AGGRESSION: ADVANTAGES AND DISADVANTAGES

In the typical laboratory aggression paradigm, a participant interacts with another "subject," either a live confederate or a simulation of another subject made credible by using video or audio recordings of a confederate. A cover story provides a rationale for the task and masks the true purpose of the study. For example, the participant may be led to believe that his or her task is to function as a teacher in a learning experiment, or that they are playing another "subject" in a competitive reaction-time game. During the interaction, the faux subject may be preprogrammed to behave in a provocative manner to elicit aggressive responses from the participant. For example, the faux subject may take points with monetary value from the participant, or administer increasingly stronger noxious stimuli, such as loud noise or electric shock, to the participant. The participant is provided the opportunity to respond in turn. Aggression is operationally defined as the intensity of the response, whether application of electric shock or taking money away from an opponent.

Some argue that the artificiality of the laboratory setting so severely limits the external validity of aggression tasks as to make them virtually useless in predicting correlates of aggression in extralaboratory ("real-world") settings (6). However, relations between dispositional and situational variables and aggression found in naturalistic settings typically parallel those observed in the laboratory, supporting the notion that laboratory aggression tasks do offer some degree of external validity (7). Furthermore, experimental aggression paradigms offer a number of advantages over nonexperimental methodologies, the foremost of which are increased experimental control and the ability to determine causal relations between variables. Experimental tasks also allow researchers to examine associations between variables that occur infrequently in extralaboratory settings. Of course, naturalistic and field approaches to studying aggression offer a high degree of external validity, usually at the cost of decreased internal validity. Ideally, aggression research should progress by incorporating findings from both experimental and nonexperimental studies, triangulating in on the underlying causes of aggression by integrating these complementary sources of information.

Another criticism of laboratory measures is that the results obtained may be more a measure of the cover story's construct than aggression (5). There is some evidence that this may be true of the Buss Aggression Machine Paradigm in which the participant, acting in the role of "teacher," may administer shock

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to the "student" in an effort to help him/her learn. Thus, shock-setting behavior in the Buss context may not always measure aggression (8). This does not appear to be true of the TAP, during which shock selections occur in the context of a competitive reaction-time game. Specifically, shock setting during the TAP is not related to measures of competitiveness (9).

Critics have also pointed out that masking the true purpose of a particular study by using a cover task and deceiving participants into believing they are interacting with "another person" raises ethical concerns. Indeed, whether or not deception should be used at all in behavioral research has generated substantial commentary among ethicists, theorists, and front-line researchers. Research on socially undesirable behaviors, however, may require the use of deception in order to prevent the participant from inhibiting the behavior of interest, and thus providing invalid data. This is why the American Psychological Association's Ethical Principles of Psychologists and Code of Conduct (10) acknowledges that deception may be appropriate based on the prospective scientific value of the study and if no equally effective design alternative exists, conditions that appear to apply to aggression research (11). As added protection, participants in laboratory aggression studies are debriefed about the deception as soon as possible after participation, and they are not deceived about aspects of the study that might affect their decision to participate. For example, the use of electric shock in the TAP is discussed as part of the informed-consent process before participation.

Another criticism is that laboratory measures of aggression actually measure compliance to experimenter demands (12). However, recent research by Berman and colleagues (13) has shown that aggressive responding on the TAP is unrelated to whether the participant is concerned with how the experimenter views him/her. A final criticism of laboratory measures of aggression is that a nonaggressive option is not usually provided. In a potentially violent "real-world" encounter, many nonaggressive response options are available to adversaries, including conciliatory or submissive behavior. Critics have therefore argued that because behavioral responses are constrained to differing intensities of aggressive responding, laboratory measures may not provide useful information about aggression in naturalistic settings. To address this criticism, the PSAP and more recent versions of the TAP provide an unequivocally nonaggressive response option.

DEVELOPMENT OF THE TAP

Stuart Taylor developed the TAP at the University of Massachusetts as an alternative to the Buss Aggression Machine (BAM) Teacher-Learner Task developed earlier by Arnold Buss. For the BAM, two "subjects," the participant and a confederate of the experimenter, are asked to participate in an experiment on the effects of punishment on a learning task (e.g., recognizing patterns in a series

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of stimuli), with the participant assigned to the teacher role by lottery. After demonstrating a shock apparatus by applying a mild shock to both subjects, electrodes are attached to the confederate and the participant is led to an adjoining room. The participant presents a series of stimuli to the confederate and rewards the learner for a correct response by turning on a light (the sequence of correct and incorrect responses by the confederate is predetermined, of course), and punishes an incorrect answer by application of shock. Shocks of varying intensities may be selected (though no shock is actually administered to the confederate). Aggression is defined by the intensity and duration of selected shock ostensibly administered to the confederate.

Despite the many advantages of the BAM, including a clearly defined and easily measured aggressive response, there are also several methodological limitations. First, the confederate cannot retaliate or act in an aggressive manner toward the participant during the task itself, a social context that may not represent aggressive interactions in naturalistic settings. Second, extra task manipulations must be employed to instigate aggressive behavior, and provocation by the confederate during the task itself is not possible. Finally, there has been debate about whether the motivation for selecting a higher shock on the BAM is to aggress or to help the participant learn faster, with some research suggesting that it is actually the latter (8). For these reasons, Taylor modified the BAM by changing the cover story from a cooperative task to a competitive reaction time task in which both the participant both sends and receives electrical shock. A detailed description of the TAP is presented below.

DESCRIPTION OF THE TAP: FEATURES AND PROCEDURE

Although the procedure has undergone many minor modifications, some of which will be discussed later, we will first describe the prototypical TAP procedure (14–17). The participant is seated in front of the "reaction-time task apparatus" and an electrode is attached to the participant's nondominant wrist or hand. The participant is informed that he or she is Subject A, and that they will be competing in a task with another participant, Subject B, who will be hooked up in an adjoining room. The participant is also informed that task instructions will be presented simultaneously to both subjects via an intercom. The experimenter then immediately excuses himself, ostensibly to prepare the opponent for the experiment.

The TAP apparatus has an array of button switches labeled in ascending order from left to right. These are used by the participant to select a shock level that is ostensibly administered to Subject B. Early versions of the TAP provided five or eight shock levels; more recent versions use 10–12. The version presented in Figure 1 uses 12 buttons representing 12 response options: 0, 1–10, and 20. The 0 represents a nonaggressive response, and the 20 an extreme aggressive

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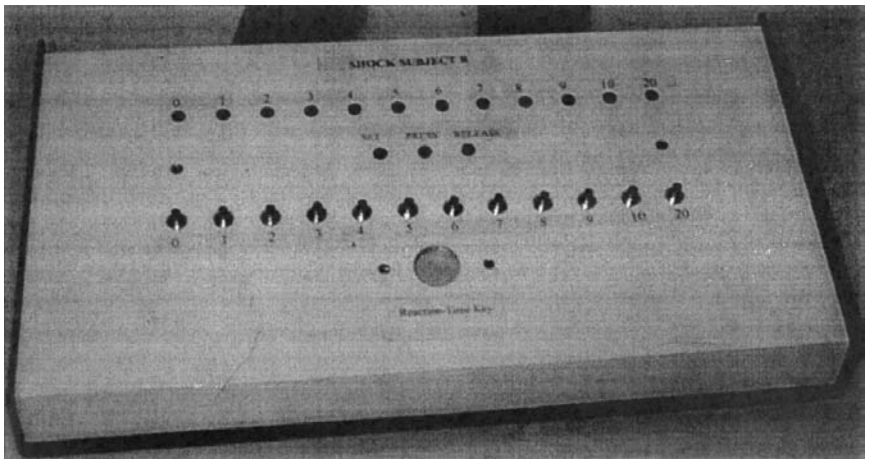

FIGURE 1 Taylor Aggression Paradigm (TAP) apparatus.

response, which are discussed in detail below. The other buttons are associated with increasingly aggressive responses. A series of lights are located above the switches, labeled in ascending order from left to right. The participant is told that these are feedback lights representing the shock level set for him/her by Subject B before each trial. Another array of three lights labeled "set," "press," and "release" are positioned between the switches and feedback lights. These are used to inform the participant when to press down on, and then release, a reaction-time button at the bottom of the panel (Fig. 1).

Following a short delay, lower and upper shock thresholds are determined for the participant and the fictitious opponent using scripted (often tape-recorded) instructions. The lower threshold is merely the lowest current at which the shock stimulus is detectable. The participant then determines his/her own upper shock threshold by informing the experimenter, during a sequence of increasingly intense shocks, when the shock becomes "definitely unpleasant." The participant then hears the threshold procedures repeated for the fictitious opponent. The "subjects" are then told that the upper shock threshold is set to correspond to the highest shock level available on the apparatus. Thus, if the TAP apparatus used has shock options 1–10, the upper shock threshold for each "subject" would be equivalent to a 10 shock.

The second part of the instructions consists of elaborating on the cover task. For example, if the effects of alcohol on aggression are being studied, both "subjects" might be told the purpose of the task is to determine the effect of alcohol on the speed with which a finger can be pulled off a reaction-time key,

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and that for each trial the person who does not get his/her finger off in the shortest time will receive a shock of the intensity decided by the opponent. The participant and Subject B are reminded that the highest shock corresponds to the level of the opponent's upper threshold, and that both are free to choose any of the available shocks options on any trial. The "subjects" are told that they will know how much shock their opponent set for them on each trial in one or two ways: first, by the intensity of the shock they actually feel on trials that they lose, and second, by which of the numbered lights at the top of the panel is illuminated after each trial. Thus, the participant knows the shock set for him or her by the opponent on all trials, including those in which the participant "wins."

The participant then competes with the fictitious opponent over a series of reaction-time trials. Each trial consists of six specific events: 1) a signal to set the intensity of shock the participant wishes to administer to the opponent on winning reaction-time trials; 2) a signal to depress the reaction-time key; 3) a signal to release the reaction-time key as fast as possible; 4) the presence or absence of a shock; 5) visual feedback informing the participant of the intensity of shock the opponent had set for the participant; and 6) a brief intertrial interval. The frequency of wins and losses, as well as the sequence of feedback lights across trials, is predetermined by the experimenter. Studies using the TAP usually have the participant win 50% of the trials.

The exact number of trials employed varies from experiment to experiment, but most often 21–28 trials are used. Using 21 trials as an example, a typical TAP study would consist of an initial trial, and three six-trial blocks of increasingly intense average shock by the opponent to provoke the participant into setting higher shocks for the opponent. Between blocks is a transition trial that is used to bridge the gap between levels of provocation and is not included in the data analysis. For example, the fictitious opponent may set 2- and 3-level shocks in the first block of trials, followed by a single transition trial in which the opponent sets a 4 shock. For the next block of trials, the opponent may set 5 and 6 shocks, and so forth. Immediately after the reaction-time task, participants are often given a posttask questionnaire to examine the participant's perception of the experimental situations, as well as other potential questions of interest.

The two primary dependent variables in a TAP study are initial shock administered on the first trial and the average shock level administered in each block of trials. Because the initial shock is selected on the first trial, before receiving any feedback about the opponent's shock setting strategy, it provides a measure of aggression in the absence of provocation. Average shock is the mean shock setting for each block of trials, and is a measure of aggression in response to varying levels of provocation. Other secondary indices of aggressive behavior are derived from the TAP. For example, the proportion of trials in which the participant selects the most intense shock available for the opponent is thought to represent a more extreme form of aggression. Similarly, the frequency with which the par-

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ticipant selects the least intense shock response (0 or 1, depending on the study) provides a measure of nonaggressive behavior. Duration of shock has also been used as indirect measures of aggression on variants of the TAP that allow participants to control the length of time the shock is administered (18,19).

VARIANTS OF THE TAP

Over the years, the TAP has been modified in a variety of ways to help experimenters accomplish their objectives or in response to criticisms of earlier versions of the TAP. Examples of minor variations include altering the number of trials (15,20), the number of shock options (16,21), or the percentage of winning trials (22). In addition, some investigators have the participant select the shock level to be administered to the opponent at the end of winning trials, rather than before each reaction-time trial (23). This modification was designed in response to the criticism that shock selection chosen before one knows if the shock will be administered to an opponent may not be a sufficiently unambiguous act of aggression. Four additional modifications are discussed below.

Inclusion of an Extreme Aggression Option

In the classic Taylor paradigm, the maximum level of shock that can be administered by the participant is equal to 100% of what the opponent reports as a "definitely unpleasant" shock. It could be argued that administration of a stimulus that is merely unpleasant does not represent aggression. Taylor was aware of this limitation, and modified his TAP to include an additional shock selection option twice both "subjects" upper threshold, which could be administered by pressing a button twice the number (e.g., 20) as the next highest button (e.g., 10). The participant is told that this level of shock is "severe" or "extremely painful." As it would be unethical to deliver such a painful shock to participants, the faux opponent either never uses the extreme shock option (22,24), or the faux opponent sets the extreme option on trials the participant "wins" (15,25). The use of an extreme shock option allows for a clearly aggressive response by the participant and a high level of provocation by the opponent.

Inclusion of a Nonaggressive Option

A major criticism of laboratory measures of aggression is that, in contrast to aggression as it occurs in the real world, a nonaggressive option is often not available (5). Thus, the participant's responses are constrained in a way that may not represent complex social interactions in naturalistic settings. In response to this criticism, Zeichner and associates modified the TAP to include a "0" shock option that provides no shock (26,27). Studies using a zero shock option show

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that the percentage of zero shocks chosen has a strong inverse correlation with mean shock intensity and percentage of extreme shock selections (27,28). The inclusion of a nonaggressive option represents an improvement over the classic TAP paradigm, and is becoming more common in aggression research (e.g., 28–30).

Non-Shock Stimuli

The TAP has been used safely in hundreds of studies on thousands of participants, without any reported significant adverse reaction to shocks delivered. However, not all researchers have access to the equipment required to construct a shock apparatus, and some local oversight committees to protect human subjects do not approve the use of electrical stimulation in research. For these reasons, some investigators have employed alternative aversive stimuli in TAP research, including loud noise. For example, Bushman and colleagues have used sound intensities ranging from 60 dB to 105 dB in lieu of shock to assess aggression (30), with both the intensity and the duration of noxious noise controlled by the participant. The two measures are standardized and summed into a single measure for greater reliability (30). Loud noise has been used in other TAP-like studies (e.g., 31,32); the results of these studies indicate that the use of noise appears to provide a useful alternative when the use of electrical stimulation is not an option. Presentation of loud noise directly into the ear is not without human-subject risk, however. The current administered by the standard shock apparatus used in the TAP is analogous to the mild electrical stimulation used by physical therapists to treat muscle injuries, is highly replicable and accurately measured, never exceeds the self-determined discomfort threshold of the participant, and is supported by the largest literature base. Accordingly, alternative noxious stimuli should be used in the TAP cautiously.

The Self-Aggression Paradigm (SAP)

One of the more novel modifications of the TAP was developed by Berman and colleagues (33,34). Berman noted that despite the wealth of experimental research on aggression, there is a dearth of experimental research on self-aggression. Accordingly, the TAP was modified to assess self-aggressive behavior by having the participant's shock selections administered to him/her, rather than the opponent, on losing reaction-time trials. In one of the studies participants were also provided the option to self-administer an "extremely unpleasant" severe self-shock that they believed was twice the intensity of their previously self-selected upper shock threshold. In reality, this option delivered a shock equal to their upper threshold, and thus the deception may not be credible after just one administration of this extreme shock level. Accordingly, the level of analysis for SAP extreme shock is any use the extreme shock, rather than the total number of

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extreme shocks as in the TAP. Studies using this new Self-Aggression Paradigm (SAP) have shown that viewing a self-aggressive model (33,34) and acute alcohol intoxication (34) increase average level of self-administered shock. With respect to alcohol, intoxicated participants were also three times more likely (45%) than nonintoxicated participants (15%) to self-select an extreme shock. These results parallel nonexperimental findings on self-aggression and support the external validity of the SAP. SAP behavior was also correlated with depression, suicidal ideation, and history of self-injurious behavior, providing evidence for the validity of the inferences that can be drawn from SAP behavior. Correlations were particularly large when the decision to self-select an extreme shock was chosen, suggesting that extreme shock selection may be a particularly sensitive measure of self-aggression (34).

COMPARISON OF THE TAP TO OTHER LABORATORY AGGRESSION MEASURES

Buss Aggression Machine

The TAP was developed as an improvement of the Buss Aggression Machine, and, not surprisingly, shares a number of similarities to the Buss paradigm. The most obvious similarity is that both measures use the severity of electrical shocks chosen by the participant as a dependent measure of aggression. In addition, both paradigms involve an interaction between a participant and a confederate or fictitious opponent, and thus capture the social nature of aggression. Furthermore, as aggression is typically considered an undesirable behavior, and one which participants may be reluctant to exhibit, cover tasks are used to provide an opportunity for the experimenter to examine aggression while minimizing the influence of social desirability response bias. However, the two tasks differ on the type of cover task used (the BAM task in which a teacher administers shock to a learner confederate versus the TAP competitive reaction-time task in which both "subjects" can administer and receive a noxious stimulus). The implications associated with these differences were discussed in detail earlier.

Point Subtraction Aggression Paradigm (PSAP)

The TAP and the PSAP are the two laboratory measures most often used in current aggression research. The TAP and PSAP share many features. Both involve an interaction between "subjects," the participant and another subject, who is actually fictitious. Both use provocation as a means of eliciting aggressive behavior, and the degree and pattern of provocation can be modified to suit the experimenter's needs in both paradigms. Thus, the two measures each focus on reactive aggression, though they also provide measures of unprovoked aggres-

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sion. What most researchers consider to be the major difference between the two measures is the topography of the aggressive response.

In the PSAP, provocation by the fictitious subject and aggression by the participant are defined by the amount of money one takes from the other. In this task, the participant selects from among three response options. One response accumulates points with monetary value (e.g., $0.15 per 100 button presses). A second response takes money away from an opponent. For example, $0.15 may be deducted from the opponent after 10 button presses. Upon completing the response requirements, the opponent is barred from deducting points from the participant for a predetermined time period (called provocation free intervals, or PFIs). The third response option, not used in all studies, is topographically identical to the second response option and provides the same PFI, but no points are removed from the opponent. This third option thus provides a topographically identical avoidance response option. Level of provocation is defined by the number of times during a session that the fictitious opponent takes money away from the participant. The use of money, rather than electrical shock, makes the PSAP more palatable to some human-subjects review boards. Though both paradigms are valid measures of aggression, a potential advantage of the TAP is that shock is a physically noxious stimulus, and may therefore better represent aggressive behavior as it occurs in extralaboratory violent interactions. This last point is speculative, however, as no published study has directly compared the performance characteristics of two measures.

Although the choice of aversive stimulus may be the most salient difference between the TAP and PSAP, other differences exist as well. For example, the PSAP uses the number of point subtractions from the opponent to measure aggression. The TAP uses the intensity of shock selected and percentage of maximum shock selections as measures of aggression. Thus, participants can vary the frequency but not the intensity of aggressive responding on the PSAP, whereas the converse is true of the TAP.

Both the TAP and PSAP take advantage of repeated observations of subject behavior. However, the repeated measures for the TAP usually represent different levels of provocation, with all provocation blocks of trials performed in single session that typically lasts < 30 min. Rarely are multiple sessions performed. In contrast, the PSAP uses multiple sessions, of ~ 30 min each. If provocation is a variable of interest, PSAP PFIs are lengthened or shortened across sessions (35–37). Thus, the TAP may be a better choice when there are participant or experimenter time constraints to completing a study, and sufficiently large samples are available to study group differences. The PSAP, in contrast, was developed for clinical pharmacology trials that require repeated measurement of a small number of subjects over multiple days, and it has proven to be a reliable measure of aggression for this purpose (Chapt. 12).

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For the TAP, only two studies were found that used a multiple-session design. Epstein and Taylor (38) had participants complete the TAP on three separate days. Each day the participant received a different pattern of shock provocation by the opponent (i.e., minimal, matching the participant's last response, or constant high provocation). Session order was counterbalanced but not analyzed. Thus, it is unclear if presentation order had any effect on aggressive responding. The study was able to show that when participants received high provocation they became increasingly aggressive, whereas they were consistently nonaggressive when they were subjected to low or matching patterns of provocation.

The other multiple-session study using the TAP found that, when given on 2 successive days, the TAP discriminated between intoxicated and sober conditions (39). However, significant order effects were also found, with sober participants for the second session setting higher shocks than sober participants for first session. Thus, until additional research can demonstrate the validity of the TAP in multiple-session studies, the PSAP may be a better choice when one is primarily interested in temporally extended repeated-measures observations of aggression in single individuals.

The PSAP has an explicitly nonaggressive option, whereas earlier versions of the TAP did not. However, with the introduction of the "0" shock option for the TAP, this is no longer the case. The function of the nonaggressive response still does differ between the two measures. The two nonaggressive options on the PSAP serve to avoid point losses or to acquire points that are exchanged for money. The nonaggressive option on the TAP serves no secondary function. That is, selection of the "0" option is a simple nonaggressive response, in that no shock is administered to the opponent on winning trials. The PSAP nonaggressive options have the advantage of allowing for the direct comparison of aggressive, protective, and appetitive responses. The TAP has the advantage of providing a nonaggressive option that is uncomplicated by secondary motives.

Unique Feature of the TAP: Extreme Aggression

Unlike other behavioral aggression measures, some modifications of the TAP provide the participant the opportunity to select a shock that is twice their fictitious opponent's shock tolerance level. This is a level that the participant is explicitly told will be extremely painful or unpleasant. This option allows researchers to examine conditions under which participants are willing to cause unequivocal harm to another human being. This modification also allows the experimenter to have the opponent set an extreme shock option for the participant without the participant having to experience the event, allowing for an examination of conditions of extreme provocation without any actual physical risk to the participant.

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PSYCHOMETRIC PROPERTIES OF THE TAP

Multiple articles have discussed the validity of laboratory measures of aggression. These issues were briefly discussed earlier. This section focuses on research studies specifically aimed at demonstrating the psychometric properties of the TAP.

Reliability

Determining reliability for a laboratory measure such as the TAP is difficult. Though the task has separate trials, it does not have different items per se, so split-half or internal consistency estimates are difficult to interpret. Furthermore, because the TAP is almost always only given once, intersession test-retest reliability is unknown. However, one study did give the TAP on separate days, with consistent levels of provocation within each day (38). Results from this study indicated that participants set consistently low (low and moderate provocation days) or rapidly ascending then consistently high shocks (high provocation day) as a function of provocation condition. This supports the notion that the TAP provides a stable measure of aggression when experimental conditions are held consistent across sessions. Similar results were found from single-day studies that used a consistent level of provocation across multiple blocks of trials (17,40).

Validity

Construct validity for the TAP can be explored by examining relevant convergent and discriminant validity evidence. For example, studies have repeatedly shown that responding on the TAP is correlated with self-report and interview measures of trait aggressiveness, including the Buss-Durkee Hostility Inventory (41,42), Buss-Perry Aggression Questionnaire (43), Spielberger Overt Anger Subscale (41), Caprara Irritability Scale (23,44), and the Life History of Aggression scale (28). Furthermore, when participants are divided into groups based on aggression history, high-aggression groups are shown to respond more aggressively on the TAP when unprovoked, and even more so when provoked (3,28,30).

As the TAP uses a competition cover task, it is important that TAP behavior not be confounded by competitive motives on the part of the subject. Bernstein and colleagues (9) tested this possibility by having subjects complete both the TAP and the Prisoners Dilemma Game. The Prisoners Dilemma Game is a measure of competitiveness in which a participant can choose a response that maximizes personal gain, or one that will benefit both the participant and fictitious opponent, but to a lesser extent. Results showed that shock selection on the TAP was unrelated to competitive responding on the Prisoner Dilemma Game. In addition, reaction-time performance is not related to shock selections (34) or self-reported motivation to win at the task (33). Discriminant validity for the TAP is also supported by findings that show no association between shock selections

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and helping behavior (9) or self-reported guilt, suspicion, and internalized anger (41).

External Validity

Laboratory paradigms of aggression are useful only if the findings generated generalize to extralaboratory settings (7). One way to examine this is to see if groups of people who differ in aggressiveness in everyday settings show the same differences on the TAP. As reported earlier, individuals high on measures of trait aggression respond more aggressively on the TAP (e.g., 41,42). Furthermore, studies using the TAP have repeatedly demonstrated the facilitation of aggression by consumption of substances previously associated with case reports of violence and homicide, such as alcohol (45) and cocaine (46), and the reduction of aggression by marijuana consumption (47). The TAP also discriminates groups of subjects theoretically expected to evidence elevated levels of aggression, such as psychopathic individuals (48) and individuals with a racial or sexual orientation prejudice (27,49,50).

Another way to examine external validity is to compare the role of contextual factors on aggression outside the laboratory to those observed under controlled conditions. For example, archival studies have demonstrated that provocation is a major facilitator of aggression (7). Aggressive responding on the TAP is also highly sensitive to provocative contextual cues (e.g., 41,51). Conversely, events that are believed to attenuate aggression in real-world settings, such as social pressure, self-awareness, and conciliatory behavior by an adversary, all have resulted in reduced aggressive behavior using the TAP (52).

RESEARCH FINDINGS FROM TAP STUDIES

The TAP has been employed over the past 35 years to examine the role of dispositional, interpersonal, and situational variables on aggression. Following is a brief synopsis of these findings. TAP studies have shown that racial prejudice (50) and homophobia (27) facilitate aggression. Studies have also shown that the perceived aggressive intent of others, rather than actual behavior, is more important with respect to the expression of aggression (53). Men with inferior cognitive function, particularly on measures of frontal lobe function, also respond more aggressively on the TAP, independent of provocation (18,54).

TAP studies have also identified variables that do not appear to increase aggression. For example, research using the TAP has shown that trait anxiety (55), discordant belief systems (56), and frustration (51) are not always related to aggression. In fact, when rewarded for doing so, individuals who are more successful (and therefore less frustrated) are more likely to become aggressive (57). In addition to these research areas, much TAP research has been devoted

to examining the effects of both provocation and alcohol on aggression. These findings will be reviewed below.

Provocation

Taylor's seminal TAP paper showed that male participants set higher shocks in response to increasing provocation, and that participants with "undercontrolled hostility" responded more to provocation than participants with "overcontrolled hostility" (3). The finding that aggression increases in response to provocation has been replicated numerous times using the TAP for both men (e.g., 17,22,58) and women (43,59), and has become one of the most consistent findings in aggression research (7). Additional research has attempted to determine the conditions under which this relationship is enhanced or mitigated. Taylor (60) showed that participants with a low need for approval are less influenced by provocation, primarily because they are more aggressive under conditions of low aggression. Other studies have shown that Type A personality men with high levels of testosterone (61) are more likely to respond to provocation with severe retaliation (a shock equal to the intensity the sham opponent's maximum threshold). Gender of the opponent can also mitigate the effects of provocation, at least for men. Men are less aggressive toward a woman opponent who is increasingly aggressive compared to an increasingly aggressive male opponent (62,63). However, when men viewed pornography in the presence of a "permissive" female immediately before participating in the TAP, they did become more aggressive in response to provocation by a fictitious female opponent (64). Furthermore, men set higher shocks for a woman opponent when the woman verbally insulted them after an ostensibly poor performance on a rigged strength test. In sum, provocation appears to be an important determinant of TAP aggression that is moderated by various dispositional and contextual factors. More research is needed to better understand the conditions under which provocation elicits aggressive acts.

Alcohol

A rich literature employing the TAP has examined the alcohol-aggression relationship. This research has reliably demonstrated that acute alcohol intoxication facilitates aggression in men (52,65–68). This effect appears to be due to the pharmacological effects of alcohol on information-processing capability, rather than expectations about alcohol's effects on human behavior (45,69). The effect of alcohol on TAP aggression appears to be dose dependent, with higher doses of alcohol associated with significantly more robust effects than low doses of alcohol (67,68,70,71). TAP studies also support the notion that alcohol's effect on aggression may be strongest under conditions of provocation (72,73), and are not attenuated by pain signals from the opponent (73,74).

TAP studies have also shown that alcohol intoxication does not invariably

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lead to aggression. Direct and indirect pressure to set low shock reduces aggression in intoxicated participants (20,69). Similar results were found for increased self-awareness (75) and reduction of threat (14,76). Participants with a nonaggressive disposition are less influenced by alcohol under conditions of moderate or high provocation (72). The evidence for an alcohol-aggression link in women is less compelling, particularly under conditions of high provocation (77–79). Recent research has attempted to determine the inter- and intra-personal variables that make alcohol-related aggression more likely. To this extent, Giancola and colleagues, based largely on research using a modified TAP, have recently developed a theory of alcohol-related aggression that integrates past findings and formulates a biopsychosocial conceptual framework for future research (80).

SUMMARY

The Taylor aggression paradigm is but one of several laboratory behavioral measures of aggression available to researchers. Although no measure represents the "gold standard," sufficient research exists to support the validity of the inferences that can be drawn from TAP behavior to make it an important tool in the aggression research field. Future research is needed to better understand the operating characteristics of all aggression measures, including self-report and clinician rating scales. To this end, we recommend that researchers consider implementing behavioral measures in which aggression is prospectively observed under controlled laboratory conditions in clinical, as well as basic, research endeavors. More research is also need to better understand the relatedness of the various laboratory measures of aggression (the PSAP, TAP, and BAM), and how idiosyncratic modifications of each paradigm affect subject task performance. Gathering evidence for the external validity of these tasks is of particular concern. As the literature base grows, investigators will develop a clearer understanding of the advantages and limitations of these measures, and how they may be used to better understand human violence.

REFERENCES

1. AH Buss. The Psychology of Aggression. New York: Wiley, 1961.
2. DM Cherek. Point Subtraction Aggression Paradigm (PSAP). Houston: University of Texas.
3. SP Taylor. Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression. J Pers 35:297–310, 1967.
4. PR Giancola, ST Chermack. Construct validity of laboratory aggression paradigms: a response to Tedeschi and Quigley (1996). Aggress Viol Behav 3:237–253, 1998.
5. JT Tedeschi, BM Quigley. Limitations of laboratory paradigms for studying aggression. Aggress Viol Behav 1:163–177, 1996.

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

6. MR Gottfredson, T Hirschi. A control theory interpretation of psychological research on aggression. In: RB Felson, JT Tedeschi, eds. Aggression and Violence: Social Interactionist Perspectives. Washington: American Psychological Association, 1993, pp. 47–68.
7. CA Anderson, BJ Bushman. External validity of "trivial" experiments: the case of laboratory aggression. Rev Gen Psychol 1:19–41, 1997.
8. RA Baron, RJ Eggleston. Performance on the "aggression machine": motivation to help or harm? Psychon Sci 26:321–322, 1972.
9. S Bernstein, D Richardson, G Hammock. Convergent and discriminant validity of the Taylor and Buss measures of physical aggression. Aggress Behav 13:15–24, 1987.
10. American Psychological Association. Ethical Principles of Psychologists and Code of Conduct. Washington: American Psychological Association, 1992.
11. HS Bertilson. Can aggression be justified in order to study aggression? Am Behav Sci 33:594–607, 1990.
12. JT Tedeschi, BM Quigley. A future comment on the construct validity of laboratory aggression paradigms: a response to Giancola and Chermack. Aggress Viol Behav 5:127–136, 2000.
13. ME Berman, MS McCloskey. The effects of acute alcohol intoxication and paroxetine on aggression. In preparation, 2002.
14. AB Gantner, SP Taylor. Human physical aggression as a function of alcohol and threat of harm. Aggress Behav 18:29–36, 1992.
15. ME Berman, SP Taylor. The effects of triazolam on aggression in men. Exp Clin Psychopharmacol 3:411–416, 1995.
16. ME Berman, SP Taylor, B Marged. Morphine and human aggression. Addict Behav 18:263–268, 1993.
17. ST Chermack, ME Berman, SP Taylor. Effects of provocation on emotions and aggression in males. Aggress Behav 23:1–10, 1997.
18. PR Giancola, A Zeichner. Neuropsychological performance on tests of frontal-lobe functioning and aggressive behavior in men. J Abnorm Psychol 103:832–835, 1994.
19. R Gustafson. Alcohol-related aggression: a further study of the importance of frustration. Psychol Rep 57:683–697, 1985.
20. SP Taylor, CB Gammon. Aggressive behavior of intoxicated subjects: the effect of third party intervention. J Stud Alcohol 37:917–929, 1976.
21. P Hoaken, J Assaad, R Pihl. Cognitive functioning and the inhibition of alcohol-induced aggression. J Stud Alcohol 59:599–607, 1998.
22. J Shortell, S Epstein, SP Taylor. Instigation to aggression as a function of degree of defeat and the capacity for massive retaliation. J Pers 38:313–328, 1970.
23. PR Giancola. Irritability, acute alcohol consumption, and aggressive behavior in men and women. Drug Alcohol Depend 68:263–274, 2002.
24. SP Taylor, JD Sears. The effects of alcohol and persuasive social pressure on human physical aggression. Aggress Behav 14:237–243, 1988.
25. SP Taylor, GT Schmutte, KE Leonard, JW Cranston. The effects of alcohol and extreme provocation on the use of a highly noxious shock. Motiv Emotion 3:73–81, 1979.

MARCEL DEKKER, INC.
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26. A Zeichner, FC Frey, D Parrott, M Butryn. Measurement of laboratory aggression: a new response-choice paradigm. Psychol Rep 85:1229–1335, 1999.
27. JA Bernat, KS Calhoun, A Zeichner. Homophobia and physical aggression toward homosexual and heterosexual individuals. J Abnorm Psychol 110:179–187, 2001.
28. MS McCloskey, ME Berman, P Posey, V Crawford, EF Coccaro. Experimental investigation of the serotonin hypothesis of aggression. Presented at the 1998 convention for the International Society for Research on Aggression, Mawah, NJ.
29. MW Weisbuch, D Beal, EC O'Neal. How masculine ought I be? Men's masculinity and aggression. Sex Roles 40:583–592, 1999.
30. BJ Bushman, RF Baumeister, CM Phillips. Do people aggress to improve their mood? Catharsis beliefs, affect regulation opportunity, and aggressive responding. J Pers Soc Psychol 81:17–32, 2001.
31. BJ Bushman, AD Stack. Catharsis, aggression, and persuasive influence: self-fulfilling or self-defeating prophecies? J Pers Soc Psychol 76:367–376, 1999.
32. J Cheong, CT Nagoshi. Effects of sensation seeking, instruction set, and alcohol/placebo administration on aggressive behavior. Alcohol 17:81–86, 1999.
33. ME Berman, CJ Walley. Imitation of self-aggressive behavior: an experimental test of the contagion hypothesis. J Appl Soc Psychol. In press.
34. MS McCloskey, ME Berman. The effects of alcohol intoxication on self-aggressive behavior in men. J Abnorm Psychol. In press.
35. DM Dougherty, JM Bjork, HCG Huckabee, FG Moeller, AC Swann. Laboratory measures of aggression and impulsivity in women with borderline personality disorder. Psychiatr Res 85:315–326, 1999.
36. DM Cherek, SD Lane. Acute effects of d-fenfluramine on simultaneous measures of aggressive escape and impulsive responses of adult males with and without a history of conduct disorder. Psychopharmacology 157:221–227, 2001.
37. DM Cherek, SD Lane, CJ Pietras, JL Steinberg. Effects of chronic paroxetine administration on measures of aggressive and impulsive responses of adult males with a history of conduct disorder. Psychopharmacology 159:266–274, 2002.
38. S Epstein, SP Taylor. Instigation to aggression as a function of defeat and perceived aggressive intent of the opponent. J Pers 35:265–289, 1967.
39. MA Lau, RO Phil. Alcohol and the Taylor Aggression Paradigm: a repeated measures study. J Stud Alcohol 55:701–706, 1994.
40. M O'Leary, H Dengerink. Aggression as a function of the intensity and pattern of attack. J Res Pers 7:61–70, 1973.
41. PR Giancola, A Zeichner. Construct validity of a competitive reaction time aggression paradigm. Aggress Behav 21:199–204, 1995.
42. G Hammock, D Richardson. Predictors of aggressive behavior. Aggress Behav 18: 219–229, 1992.
43. ME Berman, MS McCloskey, EF Coccaro, TF Greer. Gender, testosterone, and human aggression. Presented at the 2002 Convention of the American Psychological Association, Chicago, IL.
44. DJ Parrott, A Zeichner. Effects of nicotine deprivation and irritability on physical aggression in male smokers. Psychol Addict Behav 15:133–139, 2001.

MARCEL DEKKER, INC.
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45. ST Chermack, SP Taylor. Alcohol and human aggression: pharmacological versus expectancy effects. J Stud Alcohol 56:449–456, 1995.
46. SP Taylor, MR Hulsizer. Psychoactive drugs and human aggression. In: RG Geen, E Donnerstein, eds. Human Aggression: Theories, Research and Implications for Social Policy. New York: Academic Press, 1998, pp 139–165.
47. R Myerscough, SP Taylor. The effects of marijuana on human physical aggression. J Pers Soc Psychol 49:1541–1546, 1985.
48. H Dengerink. Anxiety, aggression and physiological arousal. J Exp Res Pers 5:232, 1971.
49. RW Genthner, SP Taylor. Physical aggression as a function of racial prejudice and the race of target. J Pers Soc Psychol 27:207–210, 1973.
50. KE Leonard, SP Taylor. Effects of racial prejudice and race of target on aggression. Aggress Behav 7:205–214, 1981.
51. SP Taylor, R Pisano. Physical aggression as a function of frustration and physical attack. J Soc Psychol 84:261–267, 1971.
52. SP Taylor, KE Leonard. Alcohol and human physical aggression. In: Aggression: Theoretical and Empirical Reviews. New York: Academic Press, 1983:, pp 77–101.
53. SP Taylor, RJ Shuntich, A Greenberg. The effects of repeated aggressive encounters on subsequent aggressive behavior. J Soc Psychol 107:199–208, 1979.
54. MA Lau, RO Phil. Cognitive performance. Monetary incentive, and aggression. Aggress Behav 22, 417–430. 1996.
55. FS Dorsky, SP Taylor. Physical aggression as a function of manifest anxiety. Psychon Sci 27:103–104, 1972.
56. C Hendrick, SP Taylor. Effects of belief similarity and aggression on attraction and counteraggression. J Pers Soc Psychol 17:342–349, 1971.
57. RJ Borden, SP Taylor. Pennies for pain: a note on instrumental aggression towards a pacifist by vanquished, victorious, and evenly matched opponents. Victimology 1:154–157, 1976.
58. HS Bertilson, SA Wonderlich, MW Blum. Withdrawal and matching strategies in reducing attack-instigated aggression. Psychol Rep 55:823–828, 1984.
59. D Richardson, S Bernstein, SP Taylor. The effects of situational contingencies on female retaliative behavior. J Pers Soc Psychol 37:2044–2048, 1979.
60. SP Taylor. Aggressive behavior as a function of approval motivation and physical attack. Psychon Sci 18:195–204, 1970.
61. ME Berman, B Gladue, SP Taylor. The effects of hormones, Type A behavior pattern, and provocation on aggression in men. Motiv Emotion 17:125–138, 1993.
62. SP Taylor, S Epstein. Aggression as a function of the sex of the aggressor and the sex of the victim. J Pers 35:474–486, 1967.
63. SP Taylor, I Smith. Aggression as a function of sex of victim and male subject attitude towards women. Psychol Rep 35:1095–1098, 1974.
64. KE Leonard, SP Taylor. Exposure to pornography, permissive and nonpermissive cues, and male aggression towards females. Motiv Emotion 7:291–299, 1983.
65. RJ Shuntich, SP Taylor. The effects of alcohol on human physical aggression. J Exp Res Pers 6:34–38, 1972.
66. R Gustafson. Alcohol and aggression: a validation study of the Taylor Aggression Paradigm. Psychol Rep 57:667–676, 1985.

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67. R Gustafson. Alcohol and aggression: a replication study controlling for potential confounding variables. Aggress Behav 18:21–28, 1992.
68. A Zeichner, RO Pihl. Effects of alcohol and behavior contingencies on human aggression. J Abnorm Psychol 88:153–160, 1979.
69. CM Jeavons, SP Taylor. The control of alcohol-related aggression: redirecting the inebriate's attention to socially appropriate conduct. Aggress Behav 11:93–101, 1985.
70. SP Taylor. Experimental investigation of alcohol-induced aggression in humans. Alcohol Health Res World 17:108–112, 1993.
71. SP Taylor, CB Gammon. Effects of type and dose of alcohol on human physical aggression. J Pers Soc Psychol 32:169–175, 1975.
72. DS Bailey, SP Taylor. Effects of alcohol and aggressive disposition on physical aggression. J Res Pers 25:334–342, 1991.
73. GT Schmutte, SP Taylor. Physical aggression as a function of alcohol and pain feedback. J Soc Psychol 110:235–244, 1980.
74. A Zeichner, R Pihl, R Niaura, C Zacchia. Attentional processes in alcohol mediated aggression. J Stud Alcohol 43:714–724, 1982.
75. DS Bailey, KE Leonard, JW Cranston, SP Taylor. Effects of alcohol and self-awareness on human physical aggression. Pers Soc Psychol Bull 9:289–295, 1983.
76. PR Giancola, A Zeichner. An investigation of gender differences in alcohol-related aggression. J Stud Alcohol 56:573–579, 1995.
77. PR Giancola, EL Helton, AB Osborne, MK Terry, AM Fuss, JA Westerfield. The effects of alcohol and provocation on aggressive behavior in men and women. J Stud Alcohol 63:64–73, 2002.
78. A Bond, M Lader. The relationship between induced behavioral aggression and mood after the consumption of two doses of alcohol. Br J Addict 81:65–75, 1986.
79. A Zeichner, JD Allen, PR Giancola, JM Lating. Alcohol and aggression: effects of personal threat on human aggression and affective arousal. Alcohol Clin Exp Res 18:657–653, 1994.
80. PR Giancola. Executive functioning: a conceptual framework for alcohol-related aggression. Exp Clin Psychopharmacol 8:576–597, 2000.

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12

Laboratory Measures

Point Subtraction Aggression Paradigm

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INTRODUCTION

Definition and Measurement of Aggression

Human aggression can be defined as a) a social behavior that involves the interaction of at least two people; b) is intended to harm another person, who c) finds this harm aversive and would act to avoid it if possible (1). It is well documented that a social context (verbal and nonverbal interaction) is a necessary condition for human aggressive behavior. Furthermore, provocation and retaliation are powerful variables in aggression (2–4), and both require a social context. These variables are central features of the laboratory procedures to be described.

Laboratory Measurement of Aggression

Laboratory measurement of aggression provides several advantages. First, physical violence and other forms of aggression are low-probability behaviors, are sometimes unpredictable, may involve physical injury, and can be difficult to

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identify and measure outside of laboratory settings. Unless an individual is living in a controlled environment (e.g., prison or inpatient setting), data on human aggressive behavior in natural settings must be obtained via verbal report or arrest records, and such reports may not accurately represent the actual aggressive acts. Furthermore, data of this sort are limited to regression analyses, and thus interpretations must be correlational in nature. Second, experimenters are able to avoid the possibility of physical injury to participants by controlling the form of aggressive behavior emitted. This constraint has obvious practical and ethical advantages, as well as providing an operational definition of aggressive behavior in the context of the experiment (5). Third, laboratory methods provide improved precision and manipulation of independent variables—including control over the frequency of presentation of provocative stimuli and subsequent counter attacks. Precision and control over independent variables, in union with an operationally defined dependent measure, allow for exacting experimental designs. These designs in turn provide data that permit interpretations of causality-an important step in scientific inquiry (6). Fourth, drug and alcohol use are known to be important factors in criminal behavior. Approximately 40% of violent crimes are known to involve alcohol (7). Assessment of drug and alcohol effects under controlled conditions is made possible through laboratory measurement of aggression.

This chapter provides an overview of the Point Subtraction Aggression Paradigm (PSAP), a methodology developed to study human aggressive behavior. In addition to the factors noted above, the PSAP offers some distinct experimental advantages. These will be described in greater detail below, but in summary they include a) use in a variety of experimental settings (many institutions will not allow use of real or simulated electric shock); b) use with a variety of subject populations (including children and adolescents); and c) repeated testing, which affords measurement of behavior across a range of conditions within the same subject. This latter factor also provides the opportunity for studying psychopharmacological effects via within-subject drug administration across a range of doses.

FEATURES OF THE POINT SUBTRACTION AGGRESSION PARADIGM

Similarities with Other Aggression Procedures

The PSAP (8) was developed to experimentally investigate aggressive behavior in humans under controlled laboratory conditions. This procedure shares several features with two earlier laboratory procedures designed to study human aggression, the Teacher-Learner paradigm, or Aggressive Machine procedure (9), and the Taylor Competitive Reaction Time Task (10). In all three procedures, subjects are instructed that they will be paired with others in a laboratory task and that they

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may deliver an aversive stimulus to the other individual by making an arbitrary response, such as a button press. Because the response ostensibly delivers an aversive stimulus to the partner, it is operationally defined as aggressive.

Aggressive behavior tends to occur with a low probability. Therefore, laboratory procedures designed to study aggression (including the PSAP) must include features that increase the probability of aggressive responding. In the Teacher-Learner paradigm, subjects are instructed to respond aggressively. Specifically, subjects are told that they will act as a teacher in a learning experiment and that they should deliver an electric shock to the learner whenever he/she makes an incorrect response. Alternatively, in the PSAP and in Taylor's procedure, the probability that subjects will respond aggressively is increased by periodically provoking subjects during experimental sessions. In the PSAP, money is subtracted from the subjects' cumulative earnings, whereas in Taylor's procedure, subjects are given electric shocks and shown the shock intensity set for them by the other ficititous subject. These provocations are attributed to the partner, although they are programmed by the experimenter so that their frequency and intensity can be precisely controlled. Controlling the frequency and intensity of provocations is critical because both of these variables can have a large influence on aggressive responding. For example, Cherek et al. (11) showed that the rate of aggressive responding on the PSAP varied as a direct function of the provocation frequency.

Description of the PSAP

Procedural Details

The design of the PSAP is as follows. Subjects are first instructed that they will be paired with others during experimental sessions, although in actuality they are not. Because the partner is fictitious, there is a risk that subjects may become suspicious of the social deception. The effectiveness of the deception may be evaluated, however, by querying subjects at regular intervals about their interactions with the partner(s) and their perceptions of the task. During experimental sessions, subjects are seated in front of a computer monitor and a response panel containing three buttons labeled A, B, and C. A counter centered on the computer screen shows the subject's cumulative session earnings. Subjects are told that their partner is also seated in front of a computer monitor and response panel. During sessions, subjects are presented with repeated choices between three response options. Opportunities to choose among the three options are signaled by the appearance of three letters on the computer screen—A, B, and C. A single response on an option disables the alternatives. Subjects are instructed that 100 consecutive responses on the A option will add money (e.g., $0.15) to their counter. During a session, money is periodically subtracted from a subject's counter and subjects are told that the subtractions are due to the partner pressing

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the B button on their own response panel. Subjects are also told that the partner keeps the money that is subtracted from them, which provides a motive for the partner's actions. Subjects are instructed that they may subtract money from their partner's counter by making 10 consecutive responses on the B option, but the money they subtract from the partner is not added to their own counter. In addition to ostensibly subtracting money, responses on the B option also prevent further subtractions for brief time periods called provocation free–intervals (PFIs). Subjects are not informed of this latter outcome. Finally, subjects are told that 10 consecutive responses on the C option can protect their earnings from subtractions by the partner for a variable amount of time. Responses on the C option therefore produce the same consequences as responses on the B option. Although responses on the B and C options produce PFIs, not all provocations can be avoided. That is, at the end of a PFI, at least one subtraction must occur before responses on the B or C option can produce another PFI. Therefore, subjects are provoked by money subtractions throughout the session.

Consequences of Aggressive Responding

The consequence of aggressive responding in the PSAP, the production of the PFI, is critical to maintaining aggressive responding over long periods of time. Without this consequence, aggressive responding rapidly extinguishes. For example, Cherek et al. (12) showed that when aggressive responses did not produce PFIs, aggressive responding extinguished in 1-13 sessions. Furthermore, subjects expressed doubts about the social deception.

Proactive (Instrumental) vs. Reactive Aggression

Because aggressive responding in the PSAP occurs primarily following provocations (see 12) and is maintained by escape from provocations rather than by positive consequences, such as money, the aggressive responding may be described as reactive as opposed to proactive (e.g., 13). Reactive aggression is a type of aggressive behavior that occurs following a provocation or threat and is maintained either by the removal of the aversive event, or by the avoidance of further provocations (i.e., negative reinforcement). Proactive or instrumental aggression is a type of aggressive behavior that occurs in the absence of provocation and is maintained by positive consequences (i.e., positive reinforcement), such as social approval or material goods. Previous research has indicated that reactive aggression and proactive aggression are distinct classes of aggressive behavior (e.g., 13,14).

Unique Features of the PSAP

The Aversive Stimulus

The PSAP has several characteristics that distinguish it from earlier aggression procedures. First, both in the Teacher-Learner paradigm and in Taylor's proce-

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dure, electric shock is used as the aversive stimulus whereas in the PSAP, the subtraction of money is used as an aversive stimulus. An advantage of using money subtractions rather than electric-shock presentations as aversive stimuli is that subject participation may be maintained for long time periods. Behavior can therefore be measured repeatedly, and the effects of manipulating independent variables can be evaluated in the same individual using within-subject experimental designsThe PSAP procedure may also be used to study aggressive responding in a variety of subject populations. For example, using a within-subject design Casat et al. (15) investigated the effects of methylphenidate on aggressive responding in children with histories of attention-deficit/hyperactivity disorder (ADHD) on the PSAP and showed that acute methylphenidate administrations decreased rates of aggressive responding. Research in progress in our laboratory has shown that aggressive responding in normal children can be maintained across repeated sessions on the PSAP as well. The PSAP was also used in a recent study in our laboratory to study aggressive behavior in adolescents. Twenty-one subjects, ages 14–18, were assigned to one of two groups: control or high risk for antisocial behavior and substance abuse (N = 11 high risk, 10 control). All subjects in the high-risk group met criteria for at least three of the following five high risk factors: conduct disorder, early-onset drug use, past substance abuse, school dropout, and criminal history. Control subjects met none of these criteria. Subjects took part in six sessions of the PSAP. There were notable differences in aggressive responding between the two groups, with the high-risk group making, on average, three times more aggressive responses than controls [$t(21) = 2.02$; $P = .05$]. Figure 1 shows the mean ($\pm$ 1 SEM)number of aggressive responses per minute for each group.

Provocations

The PSAP also differs from Taylor's procedure in how provocations are programmed. In Taylor's procedure, the intensity of provocations (shock deliveries) is gradually increased across the experimental session, thereby increasing the intensity of aggressive responses within a session. In the PSAP, the provocation intensity remains constant across a session. Furthermore, although responses on the aggressive and escape option decrease the frequency of provocations, all provocations cannot be avoided. Thus, the provocation frequency cannot decrease below the mean programmed value. Aggressive responding on the PSAP can therefore be maintained at stable levels across an experimental session.

Rate of Aggressive Responding

Another unique feature of the PSAP is that the rate of aggressive responding is not controlled. In the Teacher-Learner paradigm and Taylor's procedure, the opportunity to deliver an aversive stimulus to the partner is restricted to a select number of trials. The latency to respond, the magnitude of the aversive stimulus delivered to the partner, or the total number of aggressive responses serve as the

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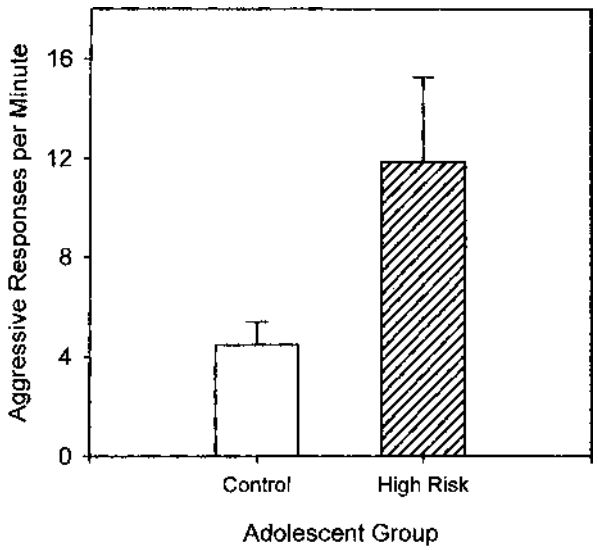

FIGURE 1 Results of a study of aggressive behavior in adolescents (N = 11 high-risk, 10 matched control). The high-risk adolescents had behavioral histories composed of conduct disorder, substance abuse/dependence, and dropping out of school. The bars show the mean number of aggressive responses per minute made across six PSAP sessions; error bars show 1 SEM. The high-risk group made, on average, three times more aggressive responses than controls, indicating greater reactivity to aversive provocation.

main dependent measure. In the PSAP, however, there are multiple opportunities to respond aggressively, and the rate of aggressive responding during a session can vary across a wide range (between zero and hundreds of responses per minute). The rate of aggressive responding is therefore used as the primary dependent measure. An advantage of using response rate as a dependent measure is that it can provide a sensitive baseline with which to evaluate the effects of a variety of independent variables, including drugs.

Alternatives to the Aggressive Option

Unlike previous aggression tasks, the PSAP provides multiple response options: the monetary-reinforced option and the escape option. There are two important benefits of including these alternatives. First, by providing an alternative to the aggressive option that also produces timeout from provocations, the PSAP does not require that subjects respond aggressively. This allows a better assessment of individual differences in aggressive behavior. Second, arranging multiple re-

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sponse options is useful in psychopharmacological studies for evaluating the specificity of drug action. For example, studies using the PSAP have shown that drugs such as d-fenfluramine (16), D-amphetamine (17), paroxetine (18), and diazepam (19) decreased aggressive responding while monetary-reinforced responding increased slightly or was unaffected. These results suggest that the drug action was specific to aggressive responding and was not due to a nonspecific sedative effect.

VALIDITY, RELIABILITY, AND SENSITIVITY OF THE PSAP

External validity of the PSAP

One enduring problem that has confronted investigators involved in laboratory studies of human aggression has been the question of external validity (1,20). Does the aggressive responding exhibited by research subjects under such controlled conditions bear any relationship to aggressive behavior emitted by these subjects outside the laboratory?

Some studies comparing different groups, such as Wolfe and Baron (21), have been criticized because the groups of subjects studied were too different in terms of education and socioeconomic levels to allow meaningful comparisons. To avoid this potential criticism, we decided to compare aggressive responding among male parolees who would have very similar backgrounds. Our initial study would allow us to determine the external validity of our laboratory aggression paradigm, PSAP. Subjects were assigned to the violent and nonviolent groups based upon their criminal history and the Brown History of Violence Questionnaire (BHVQ) score. These subjects would allow us to compare subjects with very similar backgrounds, i.e., time in prison, low socioeconomic levels, and minimal education averaging only 11.0 years. The violent subjects emitted significantly more aggressive responses per session than the nonviolent subjects. The results of our study add to the support of the relationship between aggressive responding observed in the laboratory and aggression which has previously occurred outside the laboratory.

A second study determined aggressive responding on the PSAP laboratory procedure in a larger group of male parolees. These parolees were assigned to violent or nonviolent groups based upon their criminal history. Comparisons of these two similar groups of male parolees indicated that they differed on two psychometric measures of aggression. The number of aggressive responses as measured by the PSAP differed significantly between the two groups. The violent subjects emitted significantly more aggressive responses per session than the nonviolent subjects. A third study, with female parolees, also demonstrated that violent female parolees emitted more aggressive responses than nonviolent female

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parolees (22). The results of these three studies support the relationship between aggressive responding observed in the laboratory and aggression which has previously occurred outside the laboratory.

Construct, Convergent, and Discriminant Validity of PSAP

Dougherty and his colleagues have shown a relationship between psychometric measures of aggression and aggressive responses on the PSAP. Aggressive responses correlated positively with Buss-Durkee Hostility Inventory scores (23) and Buss-Perry Aggression Questionnaire scores (24). In our own studies, we have found a positive correlation between psychometric measures of aggression and frequency of aggressive responses as measured by the PSAP. Only aggressive responses correlated with questionnaire measures of aggression and hostility (22). Males and females with higher scores on psychometric measures of aggression, emitted more aggressive responses on the PSAP (22,25).

Reliability of the PSAP

We have published a number of studies over the past 20 years which have examined the effects of different drugs on aggressive responding using the PSAP procedure (e.g., 26). As discussed in the last section, we have used the PSAP in our own laboratory to examine the role of serotonin in human aggression.

Several other investigators have reported results using the PSAP to measure human aggression. Coccaro and colleagues found a relationship between prolactin response to d-fenfluramine and aggressive responses as measured by the PSAP (27). Dougherty has found that women with borderline personality disorder emitted three times the frequency of aggressive responses as controls (23). Gerra and colleagues have published a number of studies with the PSAP. Neuroendocrine studies by this group have found that frequency of aggressive responses as measured by PSAP is related to plasma norepinephrine and testosterone (28). Other investigators using the PSAP have reported increased aggressive responding following acute administration of testosterone (29), and during marijuana withdrawal among chronic marijuana users (30). Two very recent studies have shown that Ecstasy users made more aggressive responses than control subjects, but two groups did not differ in monetary reinforced responding or escape responding (31), and aggressive responses were greater in methadone-treated heroin-dependent patients than controls (32).

Sensitivity of the PSAP

One of the advantages of the PSAP discussed earlier, is that the frequency of the aggressive response is free to vary. The Taylor Competitive Reaction Time

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procedure controls the number of trials and thus the response can only vary in terms of intensity of shock selected. The PSAP offers several advantages over other laboratory paradigms: 1) subjects are not required to respond aggressively to maintain participation in the experiment; 2) the frequency of aggressive responding is under the control of the subject rather than the experimenter; and 3) greater variability in the aggressive response can occur across subjects, allowing more sensitive determinations of the effects of other variables upon aggressive responding.

Intensity is related to frequency of the aggressive response in the PSAP. We can compare the sensitivity of the PSAP and Taylor procedure by reviewing studies that used the same pharmacological manipulation. Tryptophan depletion allows a temporary reduction in tryptophan and thus serotonin by having subjects drink an amino acid mixture that not contain tryptophan. Such procedures have been used to assess effects of tryptophan depletion on laboratory measures of aggressive responding. Both the Taylor procedure and the PSAP have reported increases in aggressive responses following tryptophan depletion. While three studies employing the Taylor procedure reported nonsignificant increases in aggression (33–35), all four studies employing the PSAP procedure reported significant increases in aggressive responses (36–39). Thus, the PSAP is more sensitive to this pharmacological manipulation, and we suggest that this sensitivity is due to measuring the frequency of the response.

NEUROBIOLOGICAL STUDIES OF AGGRESSION

Many studies have sought to locate structures and functions in the central nervous system that might be involved in aggression. One of the most reliably documented biological variables implicated in aggressive behavior is the neurotransmitter serotonin (5-hydroxytryptamine, or 5-HT) (40–42). The PSAP has been employed in a series of studies investigating the relationship between 5-HT function and aggression. In one study, male and female parolees were divided by their criminal history into violent (e.g., assault, aggravated robbery) and nonviolent (e.g., drug possession, forgery) groups. The violent group made significantly more aggressive responses on the PSAP. Serotonin activity in these same subjects was measured through a neuroendocrine challenge procedure. Subjects received a 0.4 mg/kg dose of buspirone at 9:30 am. Buspirone is an antianxiety drug that affects both serotonin and dopamine neurotransmitters. Its activity in the CNS can be estimated through measurement of prolactin release into the bloodstream. Blood draws taken throughout the day recorded serum prolactin levels. Prolactin release was severely reduced in subjects with a history of violent offenses, suggesting diminished CNS serotonin activity in those violent offenders who exhibited more aggressive behavior on the PSAP task (43). In another study, nonparolee subjects completed testing on the PSAP, and then on a separate day were

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administered a neuroendocrine challenge procedure with the drug ipsapirone. Ipsapirone also stimulates 5-HT activity in the brain. Similar to the buspirone study, subjects who had the highest levels of aggressive behavior as measured by the PSAP showed a blunted neurochemical response to the ipsapirone challenge (44). Coccaro et al. (27) administered a neuroendocrine challenge with the 5-HT-releasing drug d-fenfluramine to 14 personality-disordered males with a history of aggressive behavior. Prolactin response to the d-fenfluramine challenge was significantly negatively correlated with these subjects' levels of responding on the PSAP task.

PSYCHOPHARMACOLOGICAL STUDIES OF AGGRESSION

Neuroendocrine challenge studies provide correlational evidence about the association between aggressive behavior and neurochemical function. More direct evidence can be obtained by directly manipulating the relevant neurotransmitter (e.g., serotonin) and then simultaneously measuring aggressive responding. Studies of this type have been completed using the PSAP procedure. In one study, male parolees with history of conduct disorder (CD) as adolescents and antisocial personality disorder (ASPD) as adults received placebo and a range of three doses of d,l-fenfluramine. This drug directly stimulates the release of both serotonin and dopamine in the CNS (45). Each dose was administered approximately 30 min before the first PSAP session of the day, and data were obtained on aggressive responding throughout the remainder of the experimental day (four 25-min sessions). The data revealed that aggressive responding was decreased at high doses (46). A second study was completed using a range of doses of the drug d-fenfluramine, which is similar to d,l-fenfluramine but more specific, stimulating activity only in the serotonin system. In this study, participants were also male parolees but were divided into two groups based on the presence or absence of a diagnosis of CD/ASPD. At the highest dose, aggressive responding on the PSAP was significantly decreased in the CD/ASPD group, but unchanged in controls (16). A third study with CD/ASPD parolees administered the specific serotonin reuptake inhibitor paroxetine (20 mg/day) or placebo over 21 days. Aggressive responding was decreased in those receiving paroxetine more than those receiving placebo, but the difference did not reach statistical significance (18). One collective interpretation of these three studies is that a) individuals with violent behavioral histories can be identified as aggressive under laboratory conditions using the PSAP, b) this aggressive behavior is related to diminished serotonin activity, and c) stimulation of serotonin produces decreases in aggressive responding.

Another line of investigation using the PSAP to study the relationship between neurochemical function and aggressive behavior has examined participants

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with a history of chronic drug use. Chronic drug use produces substantial changes in brain structure and function (47). For example, Gerra and colleagues (31) studied MDMA (or Ecstasy) users and controls while they were behaving on the PSAP task. Biochemical data were collected both at baseline and in the time surrounding responding on the PSAP. While no neurochemical differences were found between the MDMA users and controls at baseline, levels of the catecholamine norepinephrine were significantly elevated in the MDMA users during responding on the PSAP (31). Work from this same laboratory replicated the effect in heroin-dependent patients versus healthy controls, showing elevated norepinephrine levels during periods surrounding PSAP responding (32). The PSAP was used to discriminate differences in aggression between chronic marijuana users going through withdrawal and control subjects. Over a 28-day detoxification period, chronic marijuana smokers showed a significant elevation in aggressive responding during the first week, compared to controls (30). This increase in aggression was attenuated by the end of the 28-day detoxification.

It is important to note that the demonstration of a relationship between the behavior of an intact organism and the function of a single biological system represents a gross oversimplification of the relevant variables. However, such an approach is germane to science and provides information upon which future work can build. This underscores the importance of focusing research efforts in aggression on the interaction of environmental and biological factors. Understanding the interaction of these factors is critically important to the prediction and control of aggression, as is the use of methods for studying aggression both in the laboratory and the natural environment. The PSAP can be an effective research tool in this process.

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REFERENCES

1. RA Baron, DR Richardson. Human Aggression. New York: Plenum Press, 1994.
2. DR Cherek. Effects of smoking different doses of nicotine on human aggressive behavior. Psychopharmacology 75:339–345, 1981.
3. DR Richardson, RJ Vandenberg, SA Humphries. Effect of power to harm on retaliatory aggression among males and females. J Res Pers 20:402–419, 1986.
4. SP Taylor. Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression. J Pers 35:297–310, 1967.
5. JM Johnston, HS Pennypacker. Strategies and Tactics of Behavioral Research. Hillsdale, NJ: Lawrence Erlbaum, 1993.

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

6. SS Stevens, ed. Handbook of Experimental Psychology. New York: Wiley, 1951.
7. SE Martin. The links between alcohol, crime and the criminal justice system: explanations, evidence and interventions. Am J Addict 10:136–158, 2001.
8. DR Cherek. Point Subtraction Aggression Paradigm (PSAP). Houston: University of Texas.
9. AH Buss. The Psychology of Aggression. New York: Wiley, 1961.
10. SP Taylor. Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression. J Pers 35:297–310, 1967.
11. DR Cherek, R Spiga, RH Bennett, J Grabowski. Human aggression and escape responding: effects of provocation frequency. Psychol Rec 41:3–17, 1991.
12. DR Cherek, R Spiga, JL Steinberg, TH Kelly. Human aggressive responses maintained by avoidance or escape from point loss. J Exp Anal Behav 53:293–303,1990.
13. KA Dodge, JD Coie. Social-information-processing factors in reactive and proactive aggression in children's peer groups. J Pers Soc Psychol 53:1146–1158, 1987.
14. F Vitaro, PL Gendreau, RE Tremblay, P Oligny. Reactive and proactive aggression differentially predict later conduct problems. J Child Psychol Psychiatr 39:377–385, 1998.
15. CD Casat, DA Pearson, MJ Van Davelaar, DR Cherek. Methylphenidate effects on laboratory aggression measure in children with ADHD. Psychopharmacol Bull 31: 353–356, 1995.
16. DR Cherek, SD Lane. Acute effects of d-fenfluramine on simultaneous measures of aggressive, escape and impulsive responses of adult males with and without a history of conduct disorder. Psychopharmacology 157:221–227, 2001.
17. DR Cherek, JL Steinberg, TH Kelley, DE Robinson. Effects of d-amphetamine on aggressive responding of normal male subjects. Psychiatr Res 21:257–265, 1987.
18. DR Cherek, SD Lane, CJ Pietras, JL Steinberg. Effets of chronic paroxetine administration on measures of aggressive and impulsive responses of adult males with a history of conduct disorder. Psychopharmacology 159:266–274, 2002.
19. DR Cherek, JL Steinberg, TH Kelley, DE Robinson, R Spiga. Effects of acute administration of diazepam and d-amphetamine on aggressive and escape responding of normal male subjects. Psychopharmacology 100:173–181, 1990.
20. L Berkowitz, E Donnerstein. External validity is more than skin deep. Some answers to criticisms of laboratory experiments. Am Psychol 37:245–257, 1982.
21. BM Wolfe, RA Baron. Laboratory aggression related to aggression in naturalistic social situations: effects of an aggressive model on the behavior of college students and prisoner observers. Psychon Sci 24:193–194, 1971.
22. DR Cherek, SD Lane, DM Dougherty, FG Moeller, S White. Laboratory and questionnaire measures of aggression among female parolees with violent and nonviolent female parolees. Aggress Behav 26:291–307, 2000.
23. DM Dougherty, JM Bjork, HC Huckabee, FG Moeller, AC Swann. Laboratory measures of aggression and impulsivity in women with borderline personality disorder. Psychiatry Res 85:315–326, 1999.
24. DM Dougherty, JM Bjork, DM Marsh, FG Moeller. Influence of trait hostility on tryptophan depletion–induced laboratory aggression. Psychiatry Res 88:227–232, 1999.

Marcel Dekker, Inc.
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25. DR Cherek, FG Moeller, W Schnapp, DM Dougherty. Studies of violent and nonviolent male parolees. I. Laboratory and psychometric measurements of aggression. Biol Psychiatry 41:514–522, 1997.
26. DR Cherek, JL Steinberg, BR Manno. Effects of alcohol on human aggressive behavior. J Stud Alcohol 46:321–328, 1985.
27. Coccaro EF, Berman ME, Kavoussi RJ, Hauger RL. Relationship of prolactin response to d-fenfluramine to behavioral and questionnaire assessments of aggression in personality-disordered men. Biol Psychiatry 40:157–164, 1996.
28. G Gerra, A Zaimovic, P Avanzini, B Chittolini, G Giucastro, R Caccavari, M Palladino, D Maestri, C Monica, R Delsignore, F Brambilla. Neurotransmitter-neuroendocrine responses to experimentally induced aggression in humans: influence of personality variable. Psychiatry Res 66:33–43, 1997.
29. HG Pope Jr, EM Kouri, JI Hudson. Effects of supraphysiologic doses of testosterone on mood and aggression in normal men: a randomized controlled trial. Arch Gen Psychiatry 57:133–140, 2000.
30. EM Kouri, HG Pope Jr, SE Lukas. Changes in aggressive behavior during withdrawal from long-term marijuana use. Psychopharmacology 143:302–308, 1999.
31. G Gerra, A Zaimovic, R Ampollini, F Giusti, R Delsignore, MA Raggi, G Laviola, T Macchia, F Brambilla. Experimentally induced aggressive behavior in subjects with 3,4-methylenedioxy-methamphetamine ("Ecstasy") use history: psychobiological correlates. J Subst Abuse 13:471–491, 2001.
32. G Gerra, A Zaimovic, MA Raggi, F Giusti, R Delsignore, S Bertacca, F Brambilla. Aggressive responding of male heroin addicts under methadone treatment: psychometric and neuroendocrine correlates. Drug Alcohol Depend 65:85–95, 2001.
33. SN Young, RO Pihl, FR Ervin. The effect of altered tryptophan levels on mood and behavior in normal human males. Clin Neuropharmacol 11(suppl 1):S207–S215, 1988.
34. DG LeMarquand, RO Pihl, SN Young, RE Tremblay, JR Seguin, RM Palmour, C Benekelfat. Tryptophan depletion, executive functions, and disinhibition in aggressive, adolescent males. Neuropsychopharmacology 19:333–341, 1998.
35. DG LeMarquand, C Benkelfat, RO Pihl, RM Palmour, SN Young. Behavioral disinhibition induced by tryptophan depletion in nonalcoholic young men with multigenerational family histories of paternal alcoholism. Am J Psychiatry 156:1771–1779, 1999.
36. JM Bjork, DM Dougherty, FG Moeller, DR Cherek, AC Swann. The effects of tryptophan depletion and loading on laboratory aggression in men: time course and a food restricted control. Psychopharmacology 142:24–30, 1999.
37. JM Bjork, DM Dougherty, FG Moeller, AC Swann. Differential behavioral effects of plasma tryptophan depletion and loading in aggressive and nonaggressive men. Neuropsychopharmacology 22:357–369, 2000.
38. DM Dougherty, FG Moeller, JM Bjork, DM Marsh. Plasma L-tryptophan depletion and aggression. Adv Exp Med Biol 467:57–65, 1999.
39. FG Moeller, DM Dougherty, AC Swann, D Collins, CM Davis, DR Cherek. Tryptophan depletion and aggressive responding in healthy males. Psychopharmacology 126:97–103, 1996.

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40. BS Eichelman. Neurochemical and psychopharmacologic aspects of aggressive behavior. Annu Rev Med 41:149–158, 1990.
41. KA Miczek, E Weerts, M Haney, J Tidey. Neurobiological mechanisms controlling aggression: preclinical developments for pharmacotherapeutic interventions. Neurosci Biobehav Rev 18:97–110, 1994.
42. Zubieta JK, Alessi NE. Is there a role of serotonin in the disruptive behavior disorders? A literature review. J Child Adolesc Psychopharmacol 3:11–35, 1993.
43. DR Cherek, FG Moeller, F Kahn-Dawood, A Swann, SD Lane. Prolactin response to buspirone was reduced in violent compared to nonviolent parolees. Psychopharmacology 142:144–148, 1999.
44. FG Moeller, DR Cherek, DM Dougherty, SD Lane, AC Swann. Ipsapirone neuroendocrine challenge: relationship to aggression as measured in the human laboratory. Psychiatry Res 81:31–38, 1998.
45. NE Rowland, J Carlton. Neurobiology of an anorectic drug: fenfluramine. Prog Neurobiol 27:13–62, 1986.
46. DR Cherek, SD Lane. Effects of d,l-fenfluramine on aggressive and impulsive responding in adult males with a history of conduct disorder. Psychopharmacology 146:473–481, 1999.
47. ND Volkow, L Chang, GJ Wang, JS Fowler, YS Ding, M Sedler, J Logan, D Franceschi, J Gatley, R Hitzemann, A Gifford, C Wong, N Pappas. Low level of brain dopamine D2 receptors in methamphetamine abusers: association with metabolism in the orbitofrontal cortex. Am J Psychiatry 158:2015–2021, 2001.

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13

Psychometric Measurement of Impulsivity

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INTRODUCTION

To assess a construct accurately, measurement tools are created with the definition of the construct as the foundation upon which the scale is developed. With regard to impulsivity, however, the definitions employed are broad, inconsistent, and continually evolving. The lack of an existing coherent and comprehensive framework from which to understand impulsiveness is reflected in the heterogeneity of available measurement techniques (1–4).

Selecting one of the various impulsivity assessments can be difficult. To begin, measurements of impulsivity are available in a variety of forms including written self-reports, personality assessments, interview assessments, observational checklists and rating scales, and, finally, behavioral or cognitive processing tasks (see Chap. 14 for the latter). Each assessment differs with regard to the time required to complete the assessment, the materials required for administration, the population for whom the measure is intended for use, and the conceptualization of the construct assessed by the measure. The length of time required for administration ranges from a few minutes for some self-report and simple behavioral

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measures, to considerably longer for omnibus personality inventories, personality interviews, or computerized continuous performance tasks.

The availability of materials to administer the measure is also a factor that influences the selection of assessment tools. Certain assessments are more expensive to administer because trained professionals or computers are required. Further, each assessment is unique with regard to the population with whom the scale is most appropriately used. Some measures are age dependent and are specifically intended for use with an adult, adolescent, or child population. At times such measures are applied across age groups, however, which can be problematic, raising the issue of ceiling or floor effects.

In addition to age, other population parameters are often important in selecting a measure. For example, Prentky and Knight (5) developed a measure of impulsivity specific to sexual offenders. Use of such a measure with other populations may be inappropriate and invalid. That differences exist among various impulsivity measures is a strength since diversity among assessments may increase the validity of the scale when used with the appropriate population. However, in selecting a measurement tool, clinicians and researchers must be aware of each instrument's inherent differences and/or limitations.

An important variation among impulsivity measures lies in the fact that the construct assessed by each measure is somewhat different (2,4,6–8) so that no single assessment adequately quantifies the construct (2,7). This distinction between measures is of critical importance in both selecting a measure and interpreting interpreting the results from its use. Many instruments provide no operational definition of the construct the scale is intended to assess. Other scales report an idiosyncratic definition of the construct. It is possible that each measure may assess impulsivity, but it is not possible that all assess the entire construct in its completeness and complexity without the inclusion of other, related constructs. The low intercorrelations obtained, especially between modalities of assessment, is evidence supporting the notion that no one scale measures impulsivity as a whole (2,6,8-10). Some scales reflect a combination of impulsiveness and other related constructs such as antisociality, attentional abilities, sensation seeking, maturity (2,11), and/or some incomplete combination of the subfactors of impulsivity.

While several self-report measures include specified subfactor scales along with the assessment of impulsivity, the number and nature of the subfactors represented by the various scales differ depending on the conceptualization of the term used as the basis for the development of the scale (12). One must consider the definition provided (if any), and/or the specific items or tasks included within the measure in order to determine what construct, or which aspect(s) of impulsiveness, is assessed by the measure. Thus, the clinician or researcher interested in measuring impulsivity has a variety of tools from which to choose but much

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to consider in selecting an instrument, particularly with regard to the aspect of impulsivity he/she intends to assess.

Over the past several decades, numerous impulsivity measures have been developed across various modalities of assessment, each unique with regard to its conceptualization of the construct and its factors, the population with whom it is most appropriately used, and the psychometric properties of the measure. The lack of association among many measures suggests that they are not consistent with one another (2,6,8-10). Therefore, in selecting a measure, it is critical that individuals be knowledgeable of the construct and/or subfactors assessed by, and the general utility of, each instrument. Although several specific and more commonly utilized measures are reviewed, it is beyond the scope of this chapter to discuss all measures; however, a comprehensive review is presented in Table 1. Laboratory measures of impulsivity are reviewed in detail in Chapter 14.

SELF-REPORT MEASURES OF IMPULSIVITY

Self-report measures are commonly utilized because of their ease of administration. They are typically completed relatively quickly (unless a part of a larger omnibus personality inventory). Additionally, self-report assessments do not require trained personnel to administer, adding to their utility and cost efficiency. However, self-report measures may be criticized on several grounds. First, there is often some question regarding the construct validity of the measures since many items included in self-report assessments reflect concepts such as preferences, temperaments, cravings, etc., as opposed to impulsive behavior. For example, of the eight items proposed to assess impulsivity within the NEO Personality Inventory (13), five reflect cravings or temptations, rather than impulsive behavior. This reflects the lack of consistency in defining the construct; note that the NEO-PI suggests that impulsivity refers to cravings or a tendency to be tempted, whereas other measures define impulsivity as behaviors. Even when impulsivity is defined behaviorally, most measures include only a few items that focus on what are believed to be actual impulsive behavior. Self-report assessments typically present hypothetical situations and ask the subject to report how he/she would behave in a given situation. However, this type of questioning assesses one's subjective sense of a tendency and/or subjective comparison of self relative to perceived behaviors of others, rather than one's actual behaviors.

Another difficulty among existing self-report assessments of impulsivity is that nonimpulsive individuals may endorse items regardless of their actual behaviors, or perhaps in spite of their discomfort with situations calling for more spontaneous behaviors. For example, an item such as "Do you feel at your best after taking a couple of drinks?" (14) may be endorsed by someone who is socially

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TABLE 1 Review of Existing Impulsivity Measures

Measures	Items/format/ scales	Reliability	Validity
Self-report measures specific to Impulsivity			
Impulsiveness Inventory (I.7) (22) For use with adults	54 items (19 for the Impulsivity Scale). Scales: Impulsiveness, Venturesomeness, Empathy	Test-retest 0.76 Int. consistency 0.73 (17,21). Reliability 0.67–0.87 (14,25)	Suspect: correlations with observational measures are low (9,25,27,28)
Junior Impulsiveness Inventory (23) For use with children	69 items (23 per scale). Yes/no format. Scales: Impulsiveness, Venturesomeness, and Empathy	Adequate (26)	Not comprehensive: this scale measures only the motor aspect of impulsivity.
Barratt Impulsiveness Scale (BIS) (16) For use with adults (patient and criminal populations)	34 items Scales: Motor, Nonplanning, Attentional Impulsiveness	Good internal consistency (16)	Validity has been criticized based on difficulties with the cognitive scale. Otherwise, there is evidence of the scale's validity.
Lifetime History of Impulsive Behaviors Self-Report (30) For use with adults	53 items Scales: Behavioral, Nonplanning, Cognitive	Preliminary data are promising	Preliminary data are promising, although no association with behavioral measures of impulsivity was found.
Self-Report Test of Impulse Control (STIC) (51)	72 items True/ false format	Good test-retest reliability (51)	Adequate concurrent and construct validity (51)
Act Frequency Approach (52) For use with adolescents and adults	100 items Scale: Impulsivity		There is evidence of this scale's validity (52)

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TABLE 1 Continued

Measures	Items/format/ scales	Reliability	Validity
Self-report measures containing Impulsivity scales			
EASI Temperament Survey (53)	Scales: Emotionality, Activity, Sociability, Impulsivity		Later the Impulsivity subscale was removed from the measure and it was renamed the EAS.
Guilford-Zimmerman Temperament Survey (54)	300 items. Impulsivity scale is 30 items. Scales: 10 personality dimensions		
Multidimensional Personality Questionnaire (MPQ) (55)	300 items. True/false format. Scales: several personality dimensions	1 month reliability of 0.82 to 0.86 (55)	
Personality Research Form (PRF-Form E) (31)	352 True/false items. 16-item Impulsivity scale. Scales: 22 personality traits	1 week test-retest reliability and internal reliability 0.85 (31)	
Interpersonal Style Inventory (ISI) (56)			Evidence of concurrent validity (56–58)
NEO-PI (13)	181 items, 5-point scale. Scales: Neuroticism, Extraversion, Openness to Experience, Agreeableness, Conscientiousness (added later)	Test-retest reliability and int. consistency are moderate to good (59)	Evidence of construct validity (60)

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TABLE 1 Continued

Measures	Items/format/ scales	Reliability	Validity
Self-report measures containing Impulsivity scales			
Tridimensional Personality Questionnaire (TPQ) (61)	Scales: Harm Avoidance, Novelty Seeking, Dependence		Suspect: The TPQ correlates with impulsivity but does not not correlate with difficulties commonly associated with impulsiveness (62). Strong construct validity (63).
Karolinska Scales of Personality (63)	3 scales, one of which is a 10-item assessment of impulsivity; 4-point rating scale.	Test-retest 0.71 (63)	Strong construct validity (63)
Interview measures of Impulsivity			
Lifetime History of Impulsive Behaviors Interview (30)	40 items. Scales: Behavioral, Nonplanning, Cognitive	Preliminary data support the reliability of the measure	Preliminary data support the validity, although there is no association with behavioral measures of impulsivity.
Interview measures of personality containing an assessment of Impulsivity			
Diagnostic Interview for Borderlines (DIB) (36)	Structured interview		Evidence of validity (38)
Structured Interview for DSM-III-R Personality Disorders (SID-P)	Semistructured interview		(Used for diagnosis more than as a measure of impulsivity)

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TABLE 1 Continued

Measures	Items/format/ scales	Reliability	Validity
Projective measures			
Various projective measures have been found to be able to identify impulsive individuals with good (Bender Gestalt and Draw-A-Person tests) (64) to fair (Rorschach Inkblot test) (65) accuracy. However, often these measures are time-consuming and subjective (with regard to scoring). Thus, they are not the preferred measures of impulsivity.			

insecure and timid and, with intent and forethought, drinks to feel a bit more relaxed. In this case, the shy individual might endorse the item in the belief that drinking might make him/her or less inhibited and not because he/she is impulsive. Because many instruments do not assess the actual behaviors and motivations of the individual, someone who is not impulsive may endorse items intended to be indicative of impulsiveness.

With regard to most assessments of impulsivity, there is a lack of assessment regarding the behaviors, motivations, situational cues or other relevant stimuli, and consequences of one's impulsiveness. This is unfortunate because impulsivity is a part of many behavioral disorders, and information regarding the actual behaviors and surrounding circumstances could be used for further, more refined study. Although self-report assessments are advantageous with regard to their facility, they are also limited in their assessment of the construct of impulsivity as well as with regard to meaningfully related factors such as situational cues, motivations, and consequences/learning.

The Barratt Impulsiveness Scale

One of the most commonly used self-assessments of impulsivity, the Barratt Impulsiveness Scale [original (15); BIS-11 (16)] measures a predisposition toward intraindividual variability. Now in its 11th version, the BIS is a widely used 34-item self-report measure of impulsivity (15,16). Traditionally, early versions of the BIS measured Barratt's three subfactors of impulsiveness (Motor, Cognitive, and Nonplanning). However, the BIS-10 was criticized owing the absence of an identifiable Cognitive factor (17). After revisions were made and the BIS-11 was examined, the cognitive factor was renamed Attentional Impulsiveness (16). Attentional impulsiveness comprises two first-order factors: attention (focusing on the task at hand) and cognitive instability (thought insertion and racing thoughts). The remaining two factors, Motor and Nonplanning, remained identifiable within

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the revised scale. Analysis of the first-order factors produces an additional four second-order factors: 1) motor impulsiveness/acting on the spur of the moment (Motor); 2) self-control/planning and thinking carefully (Motor); 3) cognitive complexity/enjoyment of difficult mental tasks (Nonplanning); and 4) perseverance/consistent lifestyle (Nonplanning).

Within the BIS, each item is rated on a four-point scale ranging from "Rarely/Never" to "Almost Always/Always." To avoid response bias, several items are worded in the reverse direction so that the lower number, as opposed to the higher, represents high impulsiveness in these cases. The latest revision, the BIS-11, is an internally consistent measure of impulsiveness (0.79-0.83), which can be used with both patient and criminal populations (16). First-and second-order factors correlate significantly, suggesting that the instrument is measuring an overall trait of impulsiveness (16). Additionally, the Motor and Nonplanning subscales demonstrate a strong correlation with Eysenck's impulsiveness narrow and nonplanning factors (16,17), further suggesting the validity of the measure. Finally, the BIS correlates with measures of aggressiveness and hostility, two constructs related to impulsiveness (18). However, the lack of support related to the Cognitive or Attentional factor remains a weakness of the measure. Barratt has suggested that the items reflecting this factor also load on the other two factors, implying that cognitive processes underlie impulsiveness in general (6,16). Another possible explanation for the lack of support regarding the cognitive component is that it may be difficult for individuals to identify underlying thought processes related to impulsivity (16,19,20).

The Impulsiveness Inventory

A second commonly used instrument, the Impulsiveness Inventory (I.7), is a 54-item scale that includes 19 items to measure impulsiveness (21,22). The remaining items yield the Venturesomeness and Empathy scales. Impulsiveness is distinguished from venturesomeness in that impulsiveness refers to action without awareness of risk, whereas venturesomeness describes action taken in light of awareness of risk (23). Within the I.7, two factors of impulsivity are assessed: lack of thoughtfulness/acting and making decisions quickly and lack of planning/living in the present. Impulsiveness, Venturesomeness, and Empathy are robust factors, as measured by the I.7 questionnaire (21). Factor analysis supports the use of subscales (14), and this instrument has been reported to yield moderate to high reliability coefficients (0.68–0.87) (24–26). Test-retest reliability (0.76), internal consistency (Cronbach's α, 0.73), and factor structure of the I.7 have also been supported (17,21). As commonly noted across assessment modalities, however, correlations between the I.7 and observational measures are low to insignificant (9,25,27,28). This suggests that the I.7's validity as an inclusive assessment of behavioral impulsivity is limited.

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There is also a childhood version of the I.7, the Junior Impulsiveness Inventory (23). This version, however, appears to assess only the motor aspect of impulsiveness (26).

Functional and Dysfunctional Impulsivity Scale

With regard to the conceptualization of impulsivity, Dickman (29) identified two types—functional and dysfunctional. Impairment, or dysfunction, has relevance to clinical syndromes. However, to date, the existence, nature, and/or importance of differences with regard to functional and dysfunctional impulsivity, as well as the biological mechanisms that may mediate these processes, are not fully known. The first scale to specifically assess these two factors of impulsivity was Dickman's Functional and Dysfunctional Impulsivity Scale (29). To some extent, other measures "control for" this omission through the predominant inclusion of items that are clearly dysfunctional in nature, creating assessments of (mainly) dysfunctional impulsivity. However, if there is a meaningful, qualitative difference between functional and dysfunctional impulsivity, it is insufficient to refer to and assess impulsivity as only a negative or pathological trait. Although Dickman's Functional and Dysfunctional Impulsivity Scale (29) assesses both "types" of impulsivity, it is limited with regard to its assessment of functionality: within the measure, items are scored as absolutely functional or dysfunctional. In reality, it is unlikely that most behaviors are always functional or always dysfunctional. For example, the item, "Most of the time, I can put my thoughts into words very rapidly," while considered a functional item, could be endorsed by someone who frequently "puts her foot in her mouth" and/or insults others without intention. Thus, the assessment of the functionality of impulsivity is either omitted or crudely assessed within existing measures of impulsiveness.

The Lifetime History of Impulsive Behaviors Self-Report

More recently, the Lifetime History of Impulsive Behaviors Self-Report measure (LHIB-SR) (30) has been developed to specifically assess both functional and dysfunctional impulsivity. Questions specifically address the outcome of impulsive behaviors in order to determine the functionality of one's impulsiveness. Three factors of impulsivity are assessed by the LHIB-SR: Behavioral, Attentional, and Nonplanning Impulsivity. Like most other self-report measures, the LHIB fails to demonstrate an association with behavioral measures. However, the LHIB is associated with other self-report measures of impulsivity and is better able than the BIS and I.7 to correctly classify subjects with personality disorders from those without such a diagnosis. Although the analysis and development of the measure remain in the early stages, preliminary data suggest the LHIB is both reliable and valid.

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The Personality Research Form

The Personality Research Form (PRF-Form E) (31) is a 352-item, true-false, self-report measure with 22 scales aimed at measuring a variety of personality traits, one of which assesses impulsivity (a 16-item subscale). The PRF is based on Murray's hierarchy of needs (32). Jackson (31) found 1-week test-retest reliability and internal reliability to be sufficient ($r = .85$) using a sample of undergraduates. Evidence for its reliability across cultures has also been found (33). While there is evidence suggesting construct validity of this measure (34) and its computerized version (35), only moderate to low coefficients have been obtained between the PRF and adjective self-ratings, trait descriptions, and peer ratings (33). Although this is again consistent with the typically low intercorrelations between self and other rating scales, it has been suggested that these findings imply a lack of validity (33).

Overall, there is evidence of the usefulness of several self-report measures with clinical and nonclinical populations. However, the subfactors assessed across measures differ, and in some cases impulsivity, although multifactorial, is assessed as if it is a unitary construct. Additionally, different self-report measures demonstrate varying degrees of reliability and validity. There is a tendency for self-report measures to correlate moderately to highly with one another, but insignificantly with other forms of assessment (e.g., laboratory assessments), suggesting that self-report measures represent only a partial assessment of the construct of impulsivity.

INTERVIEW ASSESSMENTS OF IMPULSIVITY

Two interview assessments—the Diagnostic Interview for Borderlines (36) and the Structured Interview for DSM-III-R Personality Disorders (37)—incorporate a measure of impulsivity. While these assessments are generally reliable (38), and interview assessments allow for the documentation of more specific information and behaviors, these measures are not typically chosen as direct assessments of impulsivity. Generally, only a few items within the assessment represent the construct of impulsivity, and these items are usually directly related to the specific disorder they are intended to assess. Therefore, a wide range of impulsive behaviors across a variety of domains is not assessed. Additionally, other constructs, such as antisociality, are generally included within the assessment of impulsivity. Further, when using these interview assessments, the subfactors of impulsivity are not assessed. Finally, interview assessments are time-consuming and costly because the interview is generally written to assess a broader construct (i.e., diagnosis) than merely impulsiveness, and a trained rater is required.

Interviews, however, can be valuable in that they offer a thorough exploration of an individual's behavior. Currently, there is only one interview assessment

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that focuses solely on the measurement of impulsive behaviors, the Lifetime History of Impulsive Behaviors Interview (LHIB-I). Like the self-report version, the LHIB-I assesses three subfactors of impulsivity (Behavioral, Nonplanning, and Attentional), as well as the dysfunctionality/functionality of the individual's behaviors over time. The psychometrics for the interview version are very similar to those of the self-report, suggesting reliability and validity (39).

BEHAVIORAL MEASURES OF IMPULSIVITY

Lab, or behavioral, measures, range from simple and brief tasks to lengthy, computerized tests, with most taking the form of either continuous performance tests (CPT), go/no-go tests, motor inhibition tests, or reaction time tests. Laboratory measures of impulsiveness are based on certain assumptions regarding the construct of impulsiveness, namely rapidity of responding and increased likelihood of errors (2). Behavioral measures are advantageous in that actual behaviors are assessed: the effect of the behaviors can be noted as well as the effect's impact on later responding.

However, while a lab task may prompt an individual to react impulsively, if a situation containing similar elements does not present itself outside the lab, the individual may not behave impulsively. Therefore, the assessment measures how the subject will behave given a certain set of circumstances (state impulsiveness). The researcher does not have any information regarding trait impulsivity, or the degree to which the subject actually behaves in an impulsive manner over time. Behavioral measures are limited in that they occur within an artificial setting. Subjects' motivational levels may be reduced, and the emotional salience of the situation is likely to be very different from many real-life settings wherein impulsive behaviors may occur. Further, unlike "real life," subjects are not likely to suffer any ongoing consequences from lab tasks. This may impact on responding as one may be more likely to respond impulsively on a lab task which carries no long-term consequence than in situations wherein the consequences may be more severe and/or long-lasting.

Finally, many behavioral tasks of impulsivity are extraordinarily monotonous, which may cause other factors, such as attention, to influence the score obtained. This is a potential confound in the construct validity of these measures. Unique to behavioral measures, however, is the assessment of a variety of types of errors and response times (e.g., errors of omission and commission, reaction time/discriminability, and anticipatory responding), which can be used, in part, to address the issue of the construct validity of the measure. However, as mentioned previously, lab measures do not correlate highly with other self-report or observational measures of impulsivity (40), again suggesting limitations with regard to the construct validity of the assessments. For a complete review of behavioral or laboratory tests, see Chapter 14.

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CHILDHOOD MEASURES OF IMPULSIVITY

With regard to the assessment of childhood impulsivity, the Matching Familiar Figures Test (MFFT), the most widely used childhood measure of impulsivity (2), assesses cognitive tempo, or impulsivity-reflection. Subjects must identify a figure among a group of similar figures, which exactly matches a presented target image. Response latencies and errors are scored. Those with errors above the median and latencies below the median are considered to be impulsive. Evidence supporting the validity of the MFFT includes its association with the cognitive scale of the BIS (41), behavioral checklist ratings, and the number of impulsive behaviors noted in hospital records of adolescent subjects (42). The MFFT reflects expected developmental changes (43).

Evidence that questions the validity of the measure, however, has also been presented. When the separate contributions of latency and accuracy are evaluated, latency appears to be inconsequential. In addition, when the effects of the two variables are combined, those who are both fast and inaccurate tend to be anxious, hypersensitive, vulnerable, and structure seeking as opposed to impulsive (44). Notably, the MFFT does not correlate with the Eysenck Impulsiveness Questionnaire and teacher ratings of behavior (41). Further, with use of median split analysis, fast-accurate and slow-inaccurate subjects are excluded from study (2,44). As previously discussed, the functional type of impulsivity is disregarded using this method. A further criticism of the MFFT is the lack of reliability of error scores (2,45,46). Thus, although often used, the MFFT has not gone without criticism.

OBSERVATIONAL MEASURES OF IMPULSIVITY

Observational checklists are typically developed on a per-study basis, using behaviors the researcher believes reflect impulsiveness to create the scale. Therefore, information regarding the validity and reliability of most observational "scales" is unknown. However, one scale, the Baker-Mednick teacher-rating scale (BAMED), has been reviewed. This scale consists of 106 items rated on a Likert-type scale. It has been found to be reliable (47). Observational scales are of value in that subject characteristics, such as the desire to present positively or a lack of insight, do not affect the scores obtained, as may be the case with self-report assessments (48). However, it is often difficult to access a reliable observer who has contact with the subject in naturalistic settings.

One recently developed rating scale of adult impulsivity, the Impulsivity Rating Scale (IRS), contains seven items reflecting the mostly commonly cited characteristics of impulsivity (49). While Lecrubier et al. (49) acknowledge that the sample size used in the preliminary study was inadequate, the introductory psychometric properties of this scale appear promising.

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CONCLUSIONS

In sum, while myriad assessment techniques exist, all seem to demonstrate deficits (2,7). To some degree, the deficits spring from the lack of an accepted definition of impulsivity. Ways of conceiving the construct range from behavioral or activity oriented, to cognitive or the ability to reflect, to tendencies or preferences. The proposed Cognitive or Attentional factor of impulsivity remains somewhat ambiguous. Some measures generate scores of attention and impulsiveness, as if these reflect two constructs; however, others conceptualize the ability to attend as part of impulsivity. Without a clear understanding of what the construct is, it is very difficult to measure.

In assessing impulsivity, researchers commonly employ one of two strategies: 1) operationalize impulsivity in an idiosyncratic manner, such as assessing impulsivity as the frequency of an activity believed to be impulsive (i.e., shoplifting); or 2) define impulsivity by use of an already established instrument. Using the former method is problematic in terms of establishing external validity. As Kashden and coworkers (50) have suggested, shoplifting, for example, may be a function of impulsivity, peer pressure, attention-seeking, or other factors. Thus, determining which behaviors are actually indicative of impulsiveness, is extremely difficult.

However, this same issue is also a consideration when using an established measure. Many instruments consist of items that may or may not include elements of attentional abilities, motivational factors, affect-regulatory abilities, and/or other personality traits such as sensation seeking, aggressivity, and antisociality, which may confound the assessment of impulsivity. Using an instrument with good psychometric properties is advantageous because there is some knowledge regarding the construct assessed, as well as the reliability and validity of the measure. What remains unknown, however, is the influence, or the assessment, of other constructs, and/or which subfactors of the construct are being assessed. Because most measures of impulsivity correlate only to a small degree, definitions and research subject groupings based upon existing measures are likely to yield results specific only to the subcomponent(s) measured by the particular instrument. Olson (7) concluded, "Although we have progressed in our understanding of existing measures and their interrelationships, current measures do not do justice to the complex and developmentally important construct of impulsivity." Although this conclusion was drawn in 1989, and many other measures have been created since that time, this statement remains true.

Using existing measures, clinicians and researchers must note the parameters and/or limitations of the various impulsivity scales. Inappropriate use of measures weakens the ability to interpret results accurately and to generalize findings across studies. For example, it is acceptable to use a measure of impulsivity, such as the MFFT, with children, but the assessor should be careful to recall

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that the MFFT assesses a cognitive aspect of impulsivity, or reflectivity. When forming hypotheses or conclusions, this distinction must be noted. If used with adults, or compared to another measure of impulsivity that assesses a motor aspect of the concept, the conclusions drawn may be inaccurate. Use of measures across populations or across subfactors of impulsivity results in ambiguities and misconceptions regarding impulsivity. Finally, most measures do not take into account impulsiveness resulting in correct responses or beneficial outcomes for the individual. The study and assessment of impulsivity, to date, are advancing but incomplete.

Future studies should attempt to clarify the concept of impulsivity, examining if differences in functionality are of significance in the assessment of impulsivity, if the affective salience of the situational cues are important in the production of impulsive behaviors, and if learning influences or plays a role in impulsiveness. It is possible that functional impulsivity represents a completely different process and construct, or a subcomponent of impulsivity. Issues related to the clarification of the construct are central to the accurate assessment of impulsiveness. To achieve a reasonable conceptualization of impulsivity, the subfactors of the construct need to be further assessed. It may be that one cannot meaningfully assess impulsivity without the use of specified subfactors. It may be too broad a construct. Just as one would not study personality without the use of reasonable factors of personality, it is likely the case that impulsivity cannot be discussed without the identification of consistent and reliable subfactors. Once the construct of impulsivity is better understood, the assessment of the construct can be undertaken more efficiently: comprehensive assessments including all identified subfactors can be devised. Additionally, it is reasonable to utilize existing measures but to accurately identify them as measures of a specific subfactor of impulsivity, rather than as measures of impulsivity. The continued use of the term to describe multiple constructs will only result in additional clouding of the construct of impulsivity.

REFERENCES

1. Gerbing, D. W., Ahadi, S. A., Patton, J. H. (1987). Toward a conceptualization of impulsivity: components across the behavioral and self-report domains. Multivariate Behav Res, 22, 357–379.
2. Milich, R., Kramer, J. (1984). Reflections on impulsivity: an empirical investigation of impulsivity as a construct. Adv Learn Behav Disabil, 3, 57–94.
3. Parker, J. D. A., Bagby, R. M., Webster, C. D. (1993). Domains of the impulsivity construct: a factor analytic investigation. Pers Individ Dif, 15, 267–274.
4. Schachar, R., Logan, G. D. (1990). Impulsivity and inhibitory control in normal development and childhood psychopathology. Dev Psychol, 26, 710–720.
5. Prentky, R. A., Knight, R. A. (1986). Impulsivity in the lifestyle and criminal behavior of sexual offenders. Crim Justice Behav, 13, 141–164.

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

6. Barratt E. S., Patton, J.H. (1983). Impulsivity: cognitive, behavioral, and psychophysiological correlates. In Zuckerman, M. (Ed.), Biological Bases of Sensation-Seeking, Impulsivity, and Anxiety. Hillsdale, NJ: Lawrence Erlbaum.
7. Olson, S. L. (1989). Assessment of impulsivity in preschoolers: cross-measure convergence, longitudinal stability, and relevance to social competence. J Clin Child Psychol, 18, 176–183.
8. Paulsen, K., Johnson, M. (1980). Impulsivity: a multidimensional concept with developmental aspects. J Abnorm Child Psychol, 8, 269–277.
9. Bentler, P. M., McClain, J. (1976). A multi-trait-multimethod analysis of reflection-impulsivity. Child Dev, 47, 218–226.
10. White, J. L., Moffitt, T. E., Caspi, A., Bartsch, D. J., Needles, D. J., Stouthamer-Loeber, M. (1994). J Abnorm Psychol, 103, 192–205.
11. Paulsen, K. (1978). Reflection-impulsivity and level of maturity. J Psychol, 99, 109–112.
12. Visser, M., Das-Smaal, E., Kwakman, H. (1996). Impulsivity and negative priming: evidence for diminished cognitive inhibition in impulsive children. Br J Psychol, 87, 131–140.
13. Costa, P. Jr., McCrae, R. (1978). The NEO Personality Inventory, Form S. Odessa, FL: Psychological Assessment Resources.
14. Eysenck, S. B. G., Eysenck, H. J. (1978). Impulsiveness and venturesomeness: their position in a dimensional system of personality description. Psychol Rep, 43, 1247–1255.
15. Barratt, E. S. (1959). Anxiety and impulsiveness related to psychomotor efficiency. Percept Mot Skills, 9, 191–198.
16. Patton, J. H., Stanford, M. S., Barratt, E. S. (1995). Factor structure of the Barratt impulsiveness scale. J Clin Psychol, 51, 768–774.
17. Luengo, M. A., Carrillo-de-la-Pena, Otero, J. M. (1991). The components of impulsiveness: a comparison of the I.7 Impulsiveness questionnaire and the Barratt Impulsiveness Scale. Pers Individ Dif, 12, 657–667.
18. Coccaro, E. F., Harvey, P. D., Kupsaw-Lawrence, E., Herbert, J. L., Bernstein, D. P. (1991). Development of neuropsychologically based behavioral assessments of impulsive-aggressive behavior. J Neuropsychiatry Clin Neurosci, 3(suppl), S44–S51.
19. Barratt, E. S. (1991). Measuring and predicting aggression within the context of a personality theory. J Neuropsychiatry, 3, S35–S39.
20. Barratt, E. S., Stanford, M. S. (1995). Impulsiveness. In, Costello, C.G. (Ed.), Personality Characteristics of the Personality Disordered. New York:Wiley.
21. Eysenck, S. B. G., Pearson, R. R., Easting, G., Allsopp, J. F. (1985). Age norms for impulsiveness, venturesomeness, and empathy in adults. Pers Individ Dif, 6, 613–619.
22. Eysenck, S. B. G. (1993). The I_7: development of a measure of impulsivity and its relationship to the superfactors of personality. In, W. G. McCown, J. L. Johnson, M. B. Shure (Eds.), The Impulsive Client: Theory, Research, and Treatment. Washington. American Psychological Association.
23. Eysenck, S. B. G., Easting, G., Pearson, P. R. (1984). Age norms for impulsiveness, venturesomeness and empathy in children. Pers Individ Dif, 5, 315–321.

MARCEL DEKKER, INC.
270 Madison Avenue, New York, New York 10016

24. Eysenck, S. B. G., Eysenck, H. J. (1977). The place of impulsiveness in a dimensional system of personality description. Br J Soc Clin Psychol, 16, 57–68.
25. Oas, P. (1985). The psychological assessment of impulsivity: a review. J Psychoeduc Assess, 3, 141–156.
26. Saklofske, D. H., Eysenck, S. B. G. (1983). Impulsiveness and venturesomeness in Canadian children. Psychol Rep, 52, 147–152.
27. Edman, G., Schalling, D., Levander, S. E. (1983). Impulsivity and speed and errors in a reaction time task: a contribution to the construct validity of the concept of impulsivity. Acta Psychol, 53, 1–8.
28. Gudjonsson, G. H. (1980). The relationship between the EPI extraversion score and impulsiveness on a perceptual-motor task. Pers Individ Dif, 1, 177–180.
29. Dickman, S. J. (1990). Functional and dysfunctional impulsivity: personality and cognitive correlates. J Pers Soc Psychol, 58, 95–102.
30. Schmidt, C. A., Fallon, A. E., Coccaro, E. F. The development and validation of the self-report version of the Lifetime History of Impulsive Behaviors. Chicago: University of Chicago. In preparation.
31. Jackson, D. N. (1984). Personality Research Form manual (3rd ed.). Port Huron, MI: Sigma Assessment Systems.
32. John, O. P. (1990). The "big five" factor taxonomy: dimensions of personality in the natural language and in questionnaires. In, L. A. Pervin, (Ed.), Handbook of Personality: Theory and Research. New York: Guilford.
33. Wilson, D. J., Doohlabh, A., Cooney, J., Khalpey, M. (1990). A cross-cultural validation of the Personality Research Form in Zimbabwe. Int J Psychol, 25, 1–12.
34. Holden, R. R., Woermeke, C., Fekken, G. C. (1993). Enhancing the construct validity of differential response latencies for personality test items. Can J Behav Sci, 25, 1–11.
35. Fekken, G. C., Holden, R. R. (1989). Psychometric evaluation of the microcomputerized Personality Research Form. Educ Psychol Meas, 49, 875–882.
36. Gunderson, J. G., Kolb, J. E., Austin, V. (1981). The Diagnostic Interview for Borderlines. Am J Psychiatry, 138, 896–903.
37. Pfohl, B., Blum, N., Zimmerman, M. (1995). Structured Clinical Interview for DSM-IV Personality. Iowa City: University of Iowa College of Medicine.
38. Soloff, P. H. (1981). Concurrent validation of a diagnostic interview for borderline patients. Am J Psychiatry, 138, 691–693.
39. Schmidt, C. A., Fallon, A. E., Coccaro. E. F. The Assessment of Behavioral and Cognitive Impulsivity: Development and Validation of the Lifetime History of Impulsive Behaviors interview. Chicago: University of Chicago. In review.
40. DuPaul, G. J., Anastopoulos, A. D., Shelton, T. L., Guevremont, D. C. (1992). Multimethod assessment of attention-deficit hyperactivity disorder: the diagnostic utility of clinic-based tests. J Clin Child Psychol, 21, 394–402.
41. Carrillo-de-la-Pena, M. T., Otero, J. M., Romero, E. (1993). Comparison among various methods of assessment of impulsiveness. Percept Mot Skills, 77, 567–575.
42. Oas, P. (1983). Impulsive behavior and assessment of impulsivity with hospitalized adolescents. Psychol Rep, 53, 764–766.

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43. Messer, S. B. (1976). Reflection-impulsivity: a review. Psychol Bull, 83, 1026–1052.
44. Block, J., Block, J. H., Harrington, D. M. (1974). Some misgivings about the Matching Familiar Figures Test as a measure of reflection-impulsivity. Dev Psychol, 10, 611–632.
45. Ault, R L., Mitchell, C., Hertmann, D. P. (1976). Some methodological problems in reflection-impulsivity research. Child Dev, 47, 227–231.
46. Becker, L. D., Bender, N. N., Morrison, G. (1978). Measuring impulsivity-reflection: a critical review. J Learn Disabil, 11, 626–632.
47. Baker, R. L., Mednick, B. R., Hocevar, D. (1991). Utility of scales derived from teacher judgments of adolescent academic performance and psychosocial behavior. Educ Psychol Meas, 51, 271–286.
48. Bech, P., Mak, M. (1996). Measurements of impulsivity and aggression. In, Hollander, E., Stein, D. J. (Eds.), Impulsivity and Aggression. New York: Wiley.
49. Lecrubier, Y., Braconnier, A., Said, S., Payan, C. (1995). The impulsivity rating scale (IRS): preliminary results. Eur Psychiatry, 10, 331–338.
50. Kashden, J., Fremouw, W. J., Callahan, T. S., Franzen, M. D. (1993). Impulsivity in suicidal and nonsuicidal adolescents. J Abnorm Child Psychol, 21, 339–353.
51. Lazzaro, T. A., Beggs, D. L., McNeil, K. A. (1969). The development and validation of the self-report test of impulse control. J Clin Psychol, 25, 434–438.
52. Romero, E., Luengo, M. A., Carrillo-de-la-Pena, M. T., Otero-Lopez, J. M. (1994). The act frequency approach to the study of impulsivity. Eur J Pers, 8, 119–133.
53. Buss, A. H., Plomin, R. (1975). A Temperamental Theory of Personality Development. New York: Wiley-Interscience.
54. Guilford, J. S., Zimmerman, W. S., Guilford, J. P. (1976). The Guilford-Zimmerman Temperament Survey Handbook. San Diego, CA: Educational & Industrial Testing Service.
55. Tellegan, A. (1982). Brief Manual for the Multidimensional Personality Questionnaire. Unpublished manuscript, University of Minnesota, Minneapolis.
56. Youniss, R. P., Lorr, M. (1972). Varieties of personality style. J Clin Psychol, 28, 140–145.
57. Lorr, M., Wunderlich, R. A. (1985). A measure of impulsiveness and its relations to extroversion. Educ Psychol Meas, 45, 251–257.
58. Forbes, A. R., Braunstein, S. (1981). Relationship between the Eysenck Personality and Interpersonal Style Inventories. Pers Individ Dif, 2, 167–168.
59. McCrae, R. R., Costa, P. T. Jr. (1989). Reinterpreting the Myers-Briggs type indicator form the perspective of the five-factor model of personality. J Pers, 57 17–40.
60. McCrae, R. R., Costa, P. T. Jr. (1989). Rotation to maximize the construct validity of factors in the Neo Personality Inventory. Multivariate Behav Res, 24, 107–124.
61. Cloninger, R. (1987). A systematic methods for clinical description and classification of personality variants. Arch Gen Psychiatry, 44, 573–588.
62. Nagoshi, C. T., Walter, D., Muntaner, C., Haertzen, C. A. (1992). Validation of the tridimensional personality questionnaire in a sample of make drug users. Pers Individ Dif, 13, 401–409.
63. Schalling, D., Edman, G., Asberg, M. (1983). Impulsive cognitive style and inability

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to tolerate boredom: psychobiological studies of temperamental vulnerability. In, M. Zuckerman (Ed.), Biological Bases of Sensation-Seeking, Impulsivity, and Anxiety. Hillsdale, NJ: Lawrence Erlbaum.
64. Oas, P. (1984). Validity of the draw-a-person and Bender gestalt tests as measures of impulsivity with adolescents. J Consult Clin Psychol, 52, 1011-1019.
65. Pantle, M. L., Ebner, D. L, Hynan, L. S. (1994). The Rorschach and the assessment of impulsivity. J Clin Psychol, 50, 633–638.

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14

Laboratory Measures of Impulsivity

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DEFINING IMPULSIVITY

Impulsivity can be defined as "a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to themselves or others" (1). This definition encompasses the key elements of previous definitions of impulsivity (2–6). One of the reasons for multiple definitions of impulsivity has been that impulsive behavior is the expression of complex interactions between biological and environmental factors. Because of this complexity, the measurement of impulsivity has varied widely (7), and the association among various measures of impulsivity is modest. While there are a variety of techniques to measure impulsivity, one summary of this literature suggests that these various measurement techniques fall into one of three broad categories self-report, psychophysiological/biological, and laboratory behavioral measures (1). Each of these approaches to the measurement of impulsivity has its place in clinical and research domains, but each should be selected for use dependent on the setting and information sought (see Summary). In this chapter we focus our discussion on laboratory behavioral measures of impulsivity.

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LABORATORY BEHAVIORAL MEASURES OF IMPULSIVITY

One of the primary motivations for the development and use of laboratory-based assessments of impulsivity is that these measures might offer advantages over other approaches, and therefore may be more appropriate for use in particular clinical or research settings. While there are a number of impulsivity questionnaires that have been developed and refined, their usefulness is generally limited by two factors: 1) these measures are not sensitive to transient (state-dependent) fluctuations in impulsivity; and 2) they typically rely on self-report or subjective observation. The development of objective laboratory behavioral measures sensitive to changes that occur over a brief period is important to both basic and applied research, as well as to clinicians.

For our purpose here, to qualify as a laboratory behavioral measure, two criteria must be met.

1. The behaviors measured must reasonably fit within the definition of impulsivity. Additionally, the question that must be answered is whether the measure is assessing impulsivity directly or whether it is measuring some phenomenon that is associated with impulsivity. An impulsivity measure directly assesses some phenomenon that meets the definition of impulsivity, while an impulsivity correlate would measure some secondary phenomenon that reliably varies as a function of impulsivity (8).
2. The behavior must be measured objectively.

In this chapter we have also defined behavioral measures of impulsivity as measures that are not encompassed in any way by the other two categories of impulsivity measures (self-report and psychophysiological/biological). Most often these behavioral paradigms of impulsivity fit into one of three categories: 1) rapid-decision paradigms, where impulsivity is defined either as responding prematurely, or as failing to inhibit an already initiated response; 2) reward-directed paradigms, where impulsivity is defined as a preference for a smaller-sooner reward over a larger-later reward; or 3) punishment and/or extinction paradigms, where impulsivity is defined as the perseveration of a response despite the consequence of punishment or nonreinforcement. Below we review examples of each of the three categories.

Rapid-Decision Paradigms

These are probably among the most promising and frequently used paradigms for impulsive-behavior measurement in humans. Rapid-decision tasks are sensitive to state-dependent changes in levels of impulsivity and can distinguish the performance of a number of different disinhibited patient or community samples from control group performance. Despite their popularity, however, validation studies are few and have relied almost exclusively on young children with im-

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pulse control disorders (e.g., conduct disorder, attention deficit disorder). Impulsive responding on the rapid-decision tasks involves either an inability to inhibit an already initiated response or as a premature response made before information is completely processed. The three prominent subtypes of tasks in this category are: continuous performance tests (CPT), stop tasks, and matching familiar figures tests (MFFT).

Continuous Performance Tests

The CPT is a common clinical and investigatory tool that was originally devised as a test for brain-injured patients (9). Initially, the CPT was most often used as a measure of attention (9–16). The CPT requires participants to respond selectively to a series of stimuli (e.g., abstract shapes, letters, or numbers) that are presented briefly and rapidly (usually presentations and delays of < 500 msec). Common versions include either a single target stimulus (e.g., "0") or a specific target stimulus pair (e.g., "A" followed by "X") presented periodically during random presentations of numbers or letters. Excessive failures to identify target stimuli, or omission errors, are generally interpreted as attentional deficits, which are found in a variety of populations including those with learning disabilities (11,16), Attention-Deficit/Hyperactivity Disorder (ADHD) (13,15), and schizophrenics or those at risk for schizophrenia (12,14).

Incorrect responses to nonmatching stimuli, or commission errors, have been interpreted as impulsive behavior (17,18). In most forms of the CPT commission errors are defined as any error; in other words, any response to a stimulus other than the one designated as a target is considered a commission error (17–19). Most of the subsequent research investigators have restricted their definitions of impulsive-type commission errors to include only certain kinds of errors, responses to stimuli that are similar to the target (13,20–26). These types of errors are thought to result from anticipatory, or incomplete, processing of the stimulus, which leads to a rapid, but incorrect, response to a stimulus that is similar to the target. These more narrowly defined commission errors are proposed to be more accurate indicators of impulsive behavior (27–29). Regardless of the breadth of definition, most authors interpret commission error responses as impulsive behavior. CPT procedures also typically generate measures of response latencies (measured from stimulus onset to response), and shorter latencies have been related to impulsive responding (30). The use of commission errors and latencies as measures of impulsivity is consistent with the "fast-guess" model of impulsivity, whereby judgment or accuracy is compromised for the sake of speed (31–33). According to this model, making decisions takes a certain amount of processing time and decisions (or responses) made before processing has been completed are impulsive (33–35).

More recently, efforts have been made at validating these procedures as measures of impulsivity. CPT studies have reported increased commission error

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rates and shorter latencies in a number of impulsive populations. For example, commission errors were made more frequently by children with ADHD (11,13,17,22,25,29), disruptive behavior disorders (36-38), adults with histories of Conduct Disorder (39), Bipolar Disorder (40), aggressive female parolees (41), and among healthy controls following consumption of alcohol (42,43). Others also have found reaction times (latencies) were shorter in duration compared to controls (13,22,39). Commission error rates are also elevated in nonalcoholic participants having a parental history of alcohol-related problems (44) or history of DWI (45). Additionally, CPT task performance has been related to the more substantially validated self-report measures of impulsive personality traits (38,42,46,47) such as the Barratt Impulsiveness Scale (5) and the Eysenck Impulsivity, Venturesomeness, and Empathy Questionnaire (48). Figures 1 and 2 illustrate how one CPT, the IMT/DMT, differentiates performance of those with histories of suicide attempts and aggression from others without these index

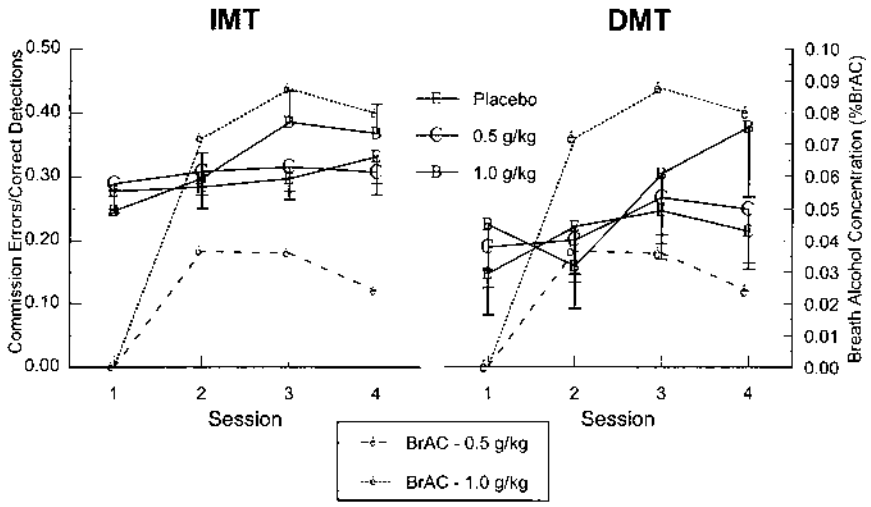

FIGURE 1 The proportion of commission errors to correct detections for both the Immediate Memory Task (left) and the Delayed Memory Task (right) under placebo, 0.5 g/kg, and 1.0 g/kg 95% alcohol treatments. The dotted (1.0 g/kg) and dashed (0.5 g/kg) lines indicate BrAC. Error bars represent the SEM. Participants were twelve men and eight women. Average age was 33.4 and education was 13.4; ethnicity was varied (8 African-American, 10 Caucasian, and 2 Hispanic). During peak BrAC (i.e., session 3) the 1.0 g/kg treatment showed proportionately more IMT commission errors than placebo ($P = .02$) and marginally more than during 0.5 g/kg treatment ($P = .08$). The DMT showed similar decrements with increasing alcohol dose although these were not statistically significant.

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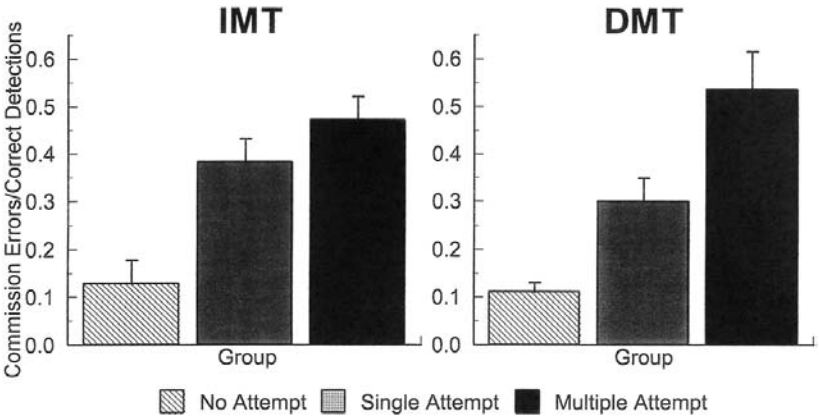

FIGURE 2 The proportion of commission errors to correct detections for both the Immediate Memory Task (left) and the Delayed Memory Task (right) among three groups of adults; those with histories of multiple suicide attempts, those with histories of a single suicide attempt, and controls. Error bars represent the SEM. Subjects were 26 women and 18 men. Average age was 28.8, and education 12.4; ethnicity was varied (23 African-American, 1 Asian, 15 Caucasian, and 5 Hispanic). Controlling for academic achievement (and Bonferroni corrected), there was a stepwise increase in impulsive responding such that the Multiple Attempt group emitted proportionately more commission errors those in the Single Attempt group (IMT $P = .05$; DMT $P = 0.001$), while the Single Attempt group scored higher than controls (IMT $P < 0.001$; DMT $P < .001$).

behavior patterns (49). Collectively, these studies support the idea that commission error rates and response latencies found during CPT performance are related to impulsive behaviors.

Stop Tasks

Stop tasks are more traditionally conceptualized as measuring response inhibition. Within this context, impulsivity is demonstrated by a failure to inhibit an already initiated response. Here again, participants are required to attend to a series of briefly presented stimuli (usually visual) and respond to target "go" stimuli. To provide response conflict situation, a portion of the "go" signals are unpredictably followed by a "stop" signal (frequently auditory), which signals participants to withhold their response (50). Here impulsive behavior is defined as the inability to withhold a response when a stop signal is presented, and these responses are also referred to as commission errors.

Stop tasks have been frequently applied among both humans (37,47,51) and nonhumans (52–54). Even in adult control populations, poor inhibitory control in

a stop task is correlated with high self-reported impulsiveness (55), and alcohol has also been shown to impair stop task response inhibition (50). Stop tasks have been frequently used in conjunction with electrophysiological measures. Results indicate a reliable anteriorization of cortical activation during performance of the procedure (56), which suggests that this measure is assessing some aspect of the executive system. This anteriorization is correlated with scores on self-report impulsivity measures (57). In another example, men with histories of Conduct Disorder prior to age 15 have been shown to have blunted frontal cortex electroencephalographic (P300) activity in stop tasks (58). These P300 amplitude decrements were not evident when participants were deciding whether to respond to target stimuli; instead, the P300 deficit was evident only when participants were presented with stop-type stimulus trials (associated with commission errors). Additionally, P300 amplitude has been shown to be inversely proportional to Barratt Impulsiveness Scale scores (59,60).

Matching Familiar Figures Test

The MFFT (61) is a behavioral measure that is typically described as assessing cognitive tempo. Cognitive tempo may be thought of as decision time under conditions of uncertainty (62). This cognitive tempo ranges from reflective (delayed responding motivated by anxiety to produce an accurate response) to impulsive (rapid responding outweighs the strength of the fear of inaccuracy) (63). In this test the participant must select a stimulus figure that matches a target stimulus from an array of distracter stimuli. Each of the other distracter figures differs from the target by at least one dimension. Scoring methods include latency to first response, total errors, and a composite impulsivity score based on latency and error rates (64).

Although initially developed and applied in child samples, the MFFT, or more commonly one of its variants (65) (e.g., MFF-20), has also been used with adults. For instance, the MFFT has been a useful tool for testing adult samples ranging from ecstasy users (64), adult descendants of alcoholics (66), psychiatric inpatients with impulse control disorders (67), male inmates (68), and women who physically abuse their children (69). However, the use of the MFFT has been largely restricted to children, and, possibly owing to several criticisms regarding methodology and validity (discussed below), its use has diminished in recent years.

While the tendency to make rapid responses resulting in inaccuracy would meet our definition of impulsivity, the MFFT has been criticized on multiple fronts. One criticism is that performance is confounded by intelligence making this test an unreliable measure of impulsive behavior (70,71). Additionally, earlier versions have been criticized on methodological grounds (72), and construct validity has been questioned because the paradigm measures a very narrow aspect of impulsivity that shows little relation to other outcome measures (73,74).

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Reward-Directed Paradigms

In the reward-directed paradigms, or "delay of reward procedures" (75;339), impulsivity is demonstrated by a preference for a smaller-sooner reward over a larger-later reward, because the participant does not tolerate the delay necessary for the larger reward. This inability to tolerate delay is one way that rapid reactions resulting in negative consequences (i.e., our definition of impulsivity) may be expressed. Therefore the reward-directed measures would be considered one form of a laboratory measure of impulsivity rather than an impulsivity correlate.

Reward-directed paradigms were developed for the study of operant behavior in animals (76,77). The advantages of the reward-directed approach are its rich history from which comparisons can be made and its clearly defined responses that are amenable to statistical and theoretical analyses (76,78). As a result, the operant perspective remains a popular model for investigating impulsive choice in both humans and nonhumans (30,79-81). Typically, these procedures require a discrete number of choices between reinforcers of different magnitude and delay, either a smaller reinforcer after a brief delay or a larger reinforcer after a longer delay.

Recent studies with humans have begun to demonstrate the utility of reward-directed paradigms for the measurement of impulsive behavior. For example, Dougherty and colleagues (37), using the Single Key Impulsivity Paradigm (SKIP) where the magnitude of reward is contingent upon the amount of time that passes between responses, demonstrated an impulsive preference for smaller-sooner rewards among adolescent patients with disruptive behavior disorders (DBD), especially in those DBD patients with a history of physical aggression. Comparable findings have been reported in adult women; female parolees with childhood history of initiating fights show a greater preference for smaller-sooner rewards than those without fighting histories (41) (see Fig. 3) A similar, two-option forced-choice procedure where participants choose between a smaller, more immediate reinforcer or a larger, progressively more delayed reinforcer has shown that parolees with histories of violence (82,83), drug abusers (84), and women with borderline personality disorder (30) demonstrated a preference for the more immediate reward and tolerate less of a delay for the larger reinforcer. Figure 4 illustrates such a two-choice procedure among adolescents with disruptive behavior disorders who had a history of initiating physical aggression (38).

While these tasks have demonstrated utility in a number of studies, there have been mixed reports regarding their sensitivity. For instance, a two-choice procedure was ineffective in distinguishing a group of adult women who were self-rated as high in impulsivity and who performed more impulsively on rapid-decision paradigms of impulsivity (described above), from their less impulsive counterparts (47). Further, direct comparisons between the paradigms revealed reward-directed tasks were less sensitive than rapid-decision tasks among high

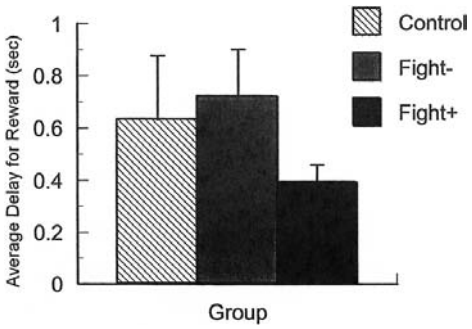

FIGURE 3 The average delay for reward (in seconds) on the Single Key Impulsivity Paradigm for three groups of adult women; those on probation/parole with a history of initiating physical aggression prior to age 15 (Fight-positive), those on probation/parole without any history of initiating physical aggression prior to age 15 or later (Fight-negative), and controls. Error bars represent the SEM. Subjects were 60 women with an average age of 27.9 and education of 12.1; ethnicity was varied (35 African-American, 10 Caucasian, 12 Hispanic, and 3 others). The Fight-positive group had a significantly shorter delay for reward than the women in the Fight-negative group ($P = .04$).

impulsivity groups (37). One explanation for the reduced group discrimination of the reward-directed measures in adults is that the processes measured may be subject to modulation by higher-order cognitive systems. This modulation, then, may serve to obscure underlying differences in impulsivity and reduce measurement sensitivity. Although disruption of executive function has been implicated in impulsivity, prior learning and experience are mechanisms that may act as compensatory processes to inhibit impulsive-type performance (37) on a less demanding task.

A promising finding regarding these measures is their relative independence from other impulsivity measures. Factor analytic studies demonstrate that reward-directed paradigms are unique from rapid-decision paradigms (37), suggesting the potential for incremental sensitivity when used in combination with other impulsivity tasks.

Punishment/Extinction Paradigms

Punishment/extinction paradigms assess impulsivity by examining responses made to stimuli that are either punished or not rewarded. This fits within the definition of impulsivity in that this paradigm measures repeated (or perseverative) responses to stimuli without regard to the consequences of those responses (i.e., punishment or lack of reward). For example, Siegel (85) developed a card-

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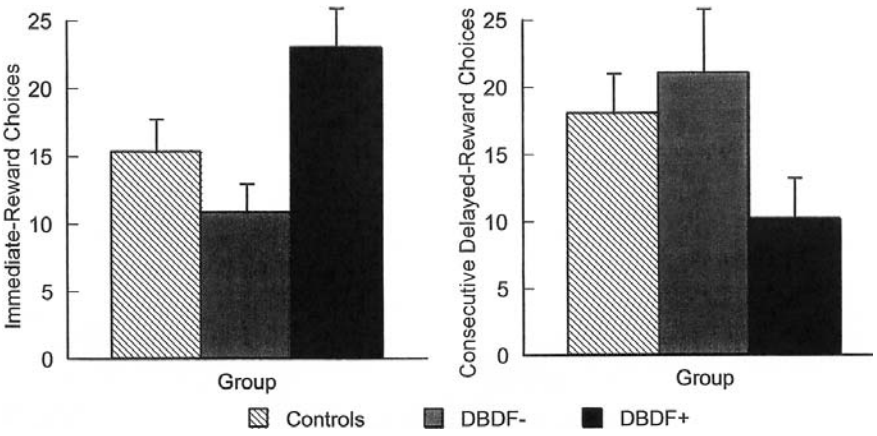

FIGURE 4 The number of smaller-sooner reward choices (left) and consecutive longer-later reward choices (right) on the Two-Choice Delayed Reward Task is presented for three groups of adolescents: inpatients with a disruptive behavior disorder diagnosis who had a history of initiating physical aggression (DBDF-positive), inpatients with a disruptive behavior disorder diagnosis who had no history of initiating physical aggression (DBDF-negative), and controls. Error bars represent the SEM. Subjects were 24 boys and 20 girls. Average age was 14.7 and average IQ was 98.1; ethnicity was varied (14 African-American, 22 Caucasian, and 8 Hispanic). Controlling for IQ (and Bonferroni corrected), the DBDF-positive emitted more immediate-reward responses ($P = .03$) and fewer and exhibited fewer consecutive delayed-reward responses ($P = .04$) than the DBDF-negative group.

playing task in which participants turn over cards from 10 separate decks. Participants turn over as many cards as they want from each of the decks and receive points for "number" cards and lose points for "face" cards. The decks are sequentially "stacked," in that there are successively more face cards in the later decks of cards (10% more from one stack to the next). Impulsive populations, in this case "psychopathic" sex offenders, persisted in playing more cards than "nonpsychopathic" sex offenders or normal controls. Even under conditions where performance feedback is given, these behaviors continue among impulsive populations (86). Similar tasks have been developed for use with children and have found that impulsive boys (with Conduct Disorder and/or ADHD) perform more impulsively than normal controls (87). This type of task is different from delay-discounting tasks, which involve varying probability of reward/punishment and are thought to be more related to risk taking than strictly impulsive behavior.

Other card-playing tasks, like the Wisconsin Card Sorting Test (WCST)

(88) require participants to match cards to a criterion that, unknown to them, alternates across dimensions (including color, number, and form). A response set is established through reward, and then the sorting criterion changes. Persisting to respond to the previously established, but no longer rewarded dimension demonstrates perseveration, which in some cases has been useful in distinguishing the behavior of high impulsive samples from other controls. The WCST is a popular clinical measure with established norms (88) and is frequently administered among brain-injured samples (89). However, the test has also been applied and interpreted from a basic research perspective examining performance of impulsive samples. Some reports indicate more perseverative responding among impulsive clinical groups (90), while others have failed to replicate this effect (91,92). Finally, other reports indicate that among college student impulsive-aggressive (93) and adolescent psychopathic (94) samples performance is relatively poorer than controls on all WCST variables except perseverative responding.

Another manner of assessing perseveration involves testing the ability to alter responding away from some dominant or overlearned response tendency. For instance, The Stroop Color-Word Test (95,96) requires naming the color of ink that words are printed with. The words, however, are color words (i.e., red, green, blue) that conflict with the name of the color of ink that they are printed in. Because, at least among literate populations, reading is such an automatic process naming the color of ink requires active suppression of reading the word and failure to do so may be thought of as one type of perseveration. Slower performance or more errors on the Stroop, indicating diminished inhibition capacity, has been reported among a number of impulsive samples such as mothers with a history of maltreatment of their children (69), violent criminal offenders (97), adult "hyperactives" (98), and among samples with compromised frontal lobe function (99). Among the general public, Stroop errors are related to a self-report measure of dysfunctional impulsivity, while faster completion time was associated with greater functional impulsivity (100). One advantage of the Stroop is that it has been applied clinically and has established norms. This test is objective, repeatable, and sensitive to state changes, although it may be necessary to correct for practice effects (101).

A final method to be considered in the punishment/extinction category includes differential reinforcement for low-rate responding (or DRL schedules) (102-105). In these operant tasks, a response is only rewarded when a specified amount of time as elapsed since the previous response. While this usually results in low response rates, when responding is continuous despite increased punishment and/or nonreward, the behavior is interpreted as impulsive.

Punishment/extinction paradigms of impulsivity have been used extensively with humans, but they have limitations. These paradigms typically incorporate elements of risk taking that make their interpretation difficult. Additionally, while DRL schedules are useful for animal research, with humans they can often

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require extensive training to acquire stability, and they may not be repeatable once the participant determines the reinforcement schedule.

SUMMARY

There are many ways in which to measure impulsive behavior through laboratory technique, and there can be a number of advantages in using this approach to address clinically relevant issues. Some of the advantages of the laboratory measures are that they: a) gather objective and easily quantifiable information; b) typically require little training to administer; c) are cost-effective; d) can be used as repeated measures; and e) can be sensitive to state-dependent changes within an individual. This last advantage is probably one of the most exciting prospects which has the potential for clinically relevant applications that cannot be met by other approaches to the measurement of impulsivity. Valid measurements sensitive to state-dependent changes in impulsivity may prove useful in answering fundamental questions regarding the role of impulsivity in subtypes of psychiatric disorders, in assessing treatment efficacy, and in predicting response to therapeutic interventions targeted for underlying impulsive deficits relating to maladaptive behaviors.

The use of laboratory behavioral tasks in clinical settings is not a new idea. Various laboratory tasks have been developed for clinicians to assess the severity of behavioral impairments in children (106:540). And many clinicians and researchers have acknowledged the potential value of laboratory-based technological developments. For example, after reviewing some of these procedures (mostly developed for use in preadolescent populations), Frick and Loney (106) recently concluded:

> Many of these techniques have proven useful for monitoring the effects of interventions, which seems to be their most immediate clinical use. With further development, several of these techniques have the potential for assessing clinically important processes that may be involved in the development and maintenance of conduct problems in youth, especially processes that may differ across subgroups of children with conduct disorders. The assessment of such processes could contribute to the development of individualized treatment plans for children and adolescents with CDs [Conduct Disorders].

The potential usefulness of objective laboratory techniques would likely extend into adult populations as well.

The disadvantages of measuring behavioral impulsivity through laboratory technique, particularly in adults, are that most of the measures used have not been properly validated and standardized. As a result, their clinical usefulness

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has not been firmly established. Basic research has been promising, indicating that these measures can discriminate among populations and can be sensitive to state-dependent changes in impulsivity produced by drug administration, which has clearly indicated their potential usefulness to the clinician.

When electing to use these tasks for clinical research it is important to select an appropriate task. As can be seen by the limited number of tasks reviewed in this chapter, these tasks differ widely and measure different aspects of impulsivity. It is important to note that each test only measures a component of impulsivity, and no approach should be used in isolation for assessment of the construct of impulsivity. Because impulsivity may result from different etiological sources and not all impulsivity tasks are equally sensitive to group differences (37), determining how to measure impulsivity by behavioral laboratory technique, or through other methodologies, is a decision that should be carefully considered. For example, when trying to assess impulsivity it is important that the task be appropriate to the population of interest. One of the most common mistakes is using a task that is not challenging enough to produce impulsive errors. There are many research reports that find no differences in behavioral impulsivity between impulsive and nonimpulsive samples, and this may be attributed to using a task that was originally developed for young children (or a significantly impaired population) and applying this same task in some high-functioning population. Selection of test measurement should also be driven by both practical and theoretical constraints. The theoretical constraints on test selection are crucial for interpretability of the results, although they are also more difficult to gauge prior to data collection. Theoretical implications to consider are at what level is impulsivity measured by the test and will the outcome of this measure satisfactorily address the testing hypothesis. Specifically, the question that must be answered is whether the measure is assessing impulsivity directly or whether it is measuring some phenomenon that is associated with impulsivity. An impulsivity measure directly assesses some phenomenon that meets the definition of impulsivity.

Other, ancillary phenomena that reliably vary as a function of impulsivity are "correlates of impulsivity" (8:96). This fine distinction must be considered because within the literature there has been a tendency to equate impulsivity correlates as true measures of impulsivity. This issue is further confounded by some authors bias toward allowing their definition of impulsivity to reflect the outcome of a single measurement tool. Hypothesis-driven research provides the direction for proper test selection and allows one to make a statement about the role of impulsivity in the research question.

When selecting a particular laboratory measure, it may be necessary to consider the relative contributions of other sources of data. Consider that self-report measures are generally inexpensive, well-established in terms of psychometric properties, and face-valid. Limitations of self-report techniques are that they are subjective (susceptible to dissimulation) require a certain degree of in-

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sight (impulsive participants often are characterized as having a disability to appreciate phenomena beyond the here and now), and are insensitive to fine-grained fluctuations in state impulsivity (i.e., these are trait measures). On the other hand, psychophysiological/biological measurement is appealing because it offers fine temporal resolution, and depends less on the participant's capacity for cooperation than on self-reports. Limitations of psychophysiological techniques include cost (equipment and personnel), sensitivity to influence by confounding factors (environmental, vegetative, and behavioral), and interpretation of results (are these impulsivity measures or are they impulsivity correlates?).

In summary, laboratory behavioral measures of impulsivity as a whole are developing into promising methodological techniques that offer unique assessments in areas not met by other methodological approaches. Despite their apparent utility, they should not be used in a vacuum—without the benefit of other sources of information. Self-report allows for the participant's perspective on unseen factors like motivations, laboratory behavior provides some objectivity, and psychophysiological measurement allows for assessing a unique aspect of the construct because biological processes have been the proposed causal link between personality and subsequent behavior (107). Therefore, laboratory measurement is one piece of a puzzle aimed at accurately and more completely assessing the construct of impulsivity.

REFERENCES

1. FG Moeller, ES Barratt, DM Dougherty, JM Schmitz, AC Swann. Psychiatric aspects of impulsivity. Am J Psychiatry 158:1783–1793, 2001.
2. SJ Dickman. Impulsivity and information processing. In: WG McCown, JL Johnson, MB Shure, eds. The Impulsive Client: Theory, Research, and Treatment. Washington; American Psychological Association, 1993, pp 151–184.
3. SBG Eysenck, HJ Eysenck. The place of impulsiveness in a dimensional system of personality description. Br J Soc Clin Psychol 16:57–68, 1977.
4. L Hinslie, J Shatzky. Psychiatric Dictionary. New York: Oxford University Press, 1940.
5. JM Patton, MS Stanford, ES Barratt. Factor structure of the Barratt Impulsiveness Scale. J Clin Psychol 51:768–774, 1995.
6. L Smith, L. A Dictionary of Psychiatry for the Layman. London: Maxwell, 1952.
7. P Oas. The psychological assessment of impulsivity: a review. J Psychoeduc Assess 3:141–156, 1985.
8. ES Barratt, JH Patton. Impulsivity: cognitive, behavioral, and psychophysiological correlates. In: M Zuckerman, ed. Biological Basis of Sensation Seeking, Impulsivity, and Anxiety. Hillsdale, NJ: Lawrence Erlbaum, A 1983, pp 77–122.
9. HE Rosvold, A Mirsky, I Sarason, ED Bransome Jr, LH Beck. A continuous performance test of brain damage. J Consult Psychol 20:343–350, 1956.
10. BA Cornblatt, NJ Risch, G Faris, D Friedman, L Erlenmeyer-Kimling. The Contin-

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

uous Performance Test: Identical Pairs Version (CPT-IP), I. New findings about sustained attention in normal families. Psychiatry Res 26:223–238, 1988.
11. RA Dykman, PT Ackerman, DM Oglesby. Selective and sustained attention in hyperactive, learning-disabled and normal boys. J Nerv Ment Dis 167:288–297, 1979.
12. L Erlenmeyer-Kimling, B Cornblatt. High-risk research in schizophrenia: a summary of what has been learned. J Psychiatr Res 21:401–411, 1987.
13. JM Halperin, L Wolf, ER Greenblatt, G Young. Subtype analysis of commission errors on the continuous performance test. Dev Neuropsychol 7:207–217, 1991.
14. KH Nuechterlein, ME Dawson. Information processing and attentional functioning in the developmental course of schizophrenic disorders. Schizophr Bull 10:160–203, 1984.
15. DA Pearson, LS Yaffee, KA Loveland, KR Lewis. A comparison of sustained and selective attention in children who have mental retardation with and without attention deficit hyperactivity disorder. Am J Ment Retard 100:592–607, 1996.
16. L Swanson. Vigilance deficit in learning disabled children: a signal detection analysis. J Child Psychol Psychiatry 22:393–399, 1981.
17. M O'Dougherty, KH Neuchterlein, B Drew. Hyperactive and hypoxic children: signal detection, sustained attention, and behavior. J Abnorm Psychol 93:178–191, 1984.
18. DH Sykes, VI Douglas, G Weiss, KK Minde. Attention in hyperactive children and the effect of methylphenidate (Ritalin). J Child Psychol Psychiatry 12:129–139, 1971.
19. J Kashden, WJ Fremouw, TS Callahan, MD Franzen. Impulsivity in suicidal and nonsuicidal adolescents. J Abnorm Child Psychol 21:339–353, 1993.
20. DM Dougherty. (1999). IMT/DMT Immediate Memory Task & Delayed Memory Task: A Research Tool for Studying Attention and Memory Processes (Version 1.2). [Computer Software Manual.] Houston: Neurobehavioral Research Laboratory and Clinic, University of Texas Health Science Center at Houston.
21. DM Dougherty, DM Marsh, CW Mathias. Immediate and Delayed Memory Tasks: A computerized measure of memory, attention, and impulsivity. Behav Res Meth Instrument Comput 34:391–398, 2002.
22. JM Halperin, LE Wolfe, D Pascualvaca, JH Newcorn, JM Healey, JD O'Brien, A Morganstein, JG Young. Differential assessment of attention and impulsivity in children. J Am Acad Child Adolesc Psychiatry 27:326–329, 1988.
23. A Smith, A Kendrick, A Maben. Use and effects of food and drinks in relation to daily rhythms of mood and cognitive performance: effects of caffeine, lunch and alcohol on human performance, mood and cardiovascular function. Proc Nutr Soc 51:325–333, 1992.
24. AJ Sostek, MS Buchsbaum, JL Rapoport. Effects of amphetamine on vigilance performance in normal and hyperactive children. J Abnorm Child Psychol 8:491–500, 1980.
25. DH Sykes, VI Douglas, G Morganstern. Sustained attention in hyperactive children. J Child Psychol Psychiatry 14:213–220, 1973.
26. GW Wohlberg, C Kornetsky. Sustained attention in remitted schizophrenics. Arch Gen Psychiatry 28:533–537, 1973.

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

27. RA Barkley. The ecological validity of laboratory and analogue assessment methods of ADHD symptoms. J Abnorm Child Psychol 19:149–178, 1991.
28. IL Beale, PJ, Matthew, S Oliver, MC Cornballis. Performance of disabled and normal readers on the continuous performance test. J Abnorm Child Psychol 15: 229–238, 1987.
29. K O'Toole, A Abramowitz, R Morris, MK Dulcan. Effects of methylphenidate and nonverbal learning in children with attention-deficit hyperactivity disorder. J Am Acad Child Adolesc Psychiatry 36:531–538, 1997.
30. DM Dougherty, JM Bjork, HC Huckabee, FG Moeller, AC Swann. Laboratory measures of aggression and impulsivity in women with borderline personality disorder. Psychiatry Res 85:315–326, 1999.
31. SJ Dickman, DE Meyer. Impulsivity and speed-accuracy tradeoffs in information processing. J Pers Soc Psychol 54:274–290, 1988.
32. R Ollman. Fast guess in choice reaction time. Psychonom Sci 6:155–156, 1966.
33. JI Yellot. Corrections for fast guessing and speed-accuracy-tradeoff in choice reaction time. J Math Psychol 8:159–199, 1971.
34. JA Sergeant, CA Sholton. On resources strategy limitations in hyperactivity: cognitive impulsivity reconsidered. J Child Psychol Psychiatry 25:809–819, 1985.
35. G Sperling. A unified theory of attention and signal detection. In: R Parsuraman, DR Davis, eds. Varieties of Attention. New York: Academic Press, 1984, pp 103–181.
36. JM Bjork, DM Dougherty, FG Moeller, RA Harper, K Scott-Gurnell, AC Swann. Laboratory measures of impulsivity in hospitalized adolescents with disruptive behavior disorders. Biol Psychiatry 47:149, 2000.
37. DM Dougherty, JM Bjork, RA Harper, DM Marsh, FG Moeller, CW Mathias. Behavioral impulsivity paradigms: a comparison in hospitalized adolescents with disruptive behavior disorders. (In press.)
38. DM Dougherty, JM Bjork, RA Harper, CW Mathias, FG Moeller, DM Marsh. Concurrent validation of the Immediate and Delayed Memory Tasks in hospitalized adolescents with disruptive behavior disorders. (In press.)
39. DM Dougherty, JM Bjork, DM Marsh, FG Moeller. A comparison between adults with conduct disorder and normal controls on a Continuous Performance Test: differences in impulsive response characteristics. Psychol Rec 50:203–219, 2000.
40. AC Swann, J Anderson, DM Dougherty, FG Moeller. Measurement of interepisode impulsivity in bipolar disorder: a preliminary report. Psychiatry Res 101:195–197, 2001.
41. CW Mathias, DM Dougherty, DM Marsh, FG Moeller, LR Hicks, K Dasher, L Bar-Eli. Laboratory measures of impulsivity: a comparison of women with and without childhood aggression. Psychol Rec 52:289–303, 2002.
42. DM Dougherty, DM Marsh, FG Moeller, RV Chokshi, VC Rosen. Effects of moderate and high doses of alcohol on attention, impulsivity, discriminability, and response bias in Immediate and Delayed Memory Task performance. Alcohol Clin Exp Res 24:1702–1711, 2000.
43. DM Dougherty, FG Moeller, JL Steinberg, DM Marsh, SE Hines, JM Bjork. Alcohol increases commission error rates for a continuous performance test. Alcohol Clin Exp Res 23:1342–1351, 1999.

MARCEL DEKKER, INC.
270 Madison Avenue, New York, New York 10016

44. RC Miller. The effect of low doses of alcohol on human behavior. Unpublished PhD thesis, University of Texas at Austin, 1984.
45. M Koch, M Morguet. Investigation in the use of a vigilance test in psycho-medical assessment. Blutalkohol 22:391–396, 1985.
46. DM Dougherty, JM Bjork, FG Moeller, RA Harper, DM Marsh, CW Mathias, AC Swann. Familial transmission of Continuous Performance Test behavior: attentional and impulsive response characteristics. J Gen Psychol. (In press.)
47. DM Marsh, DM Dougherty, CW Mathias, FG Moeller, LR Hicks. Comparison of women with high and low trait impulsivity using laboratory impulsivity models of response-disinhibition and reward-choice. Pers Individ Dif 33:1291–1310, 2002.
48. SBG Eysenck, PR Pearson, G Easting, JF Allsopp. Age norms for impulsiveness, venturesomeness and empathy in adults. Pers Individ Dif 6:613–619, 1985.
49. CW Mathias, DM Dougherty, VA Oderinde, FG Moeller, RA Harper, DM Marsh, JM Bjork. Suicidality and a laboratory measure of impulsivity. Poster session presented at the 31st annual meeting of the Society for Neuroscience, San Diego, CA, 2001.
50. LE Mulvihill, TA Skilling, M Vogel-Sprott. Alcohol and the ability to inhibit behavior in men and women. J Stud Alcohol 58:600–605, 1997.
51. DG LeMarquand, C Benkelfat, RO Pihl, RM Palmour, SN Young. Behavioral disinhibition induced by tryptophan depletion in nonalcoholic young men with multigenerational family histories of paternal alcoholism. Am J Psychiatry 156: 1771–1779, 1999.
52. V Giardini, L Amorico, L De Acetis, G Bignami. Scopolamine and acquisition of go–no go avoidance: a further analysis of the perseverative antimuscarinic deficit. Psychopharmacology (Berl) 80:131–137, 1983.
53. AA Harrison, BJ Everitt, TW Robbins. Central serotonin depletion impairs both the acquisition and performance of a symmetrically reinforced go/no-go conditional visual discrimination. Behav Brain Res 100:99–112, 1999.
54. K Itoh, A Izumi, S Kojima. Object discrimination learning in aged Japanese monkeys. Behav Neurosci 115:259–270, 2001.
55. GD Logan, RJ Schachar, R Tannock. Impulsivity and inhibitory control. Psychol Sci 8:60–64, 1997.
56. D Aranda, AJ Bartsch, MJ Herrmann, S Eisenack, JC Morinigo, AJ Fallgatter. Reliability of electrophysiological measurements of motor control. Rev Neurol 32: 10–14, 2001.
57. AJ Fallgatter, MJ Herrmann. Electrophysiological assessment of impulsive behavior in healthy subjects. Neuropsychologia 39:328–333, 2001.
58. LO Bauer. Frontal P300 decrements, childhood conduct disorder, family history, and the prediction of relapse among abstinent cocaine abusers. Drug Alcohol Depend 44:1–10, 1997.
59. ES Barratt. Impulsiveness subtraits: Arousal and information processing. In: JT Spence, CE Izard ed. Motivation, Emotion, and Personality. New York: Elsevier Science, 1985, pp 137–146.
60. MH Branchey, L Buydens-Branchey, TB Horvath. Event related potentials in substance abusing individuals after long term abstinence. Am J Addict 2:141–148, 1993.

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

61. J Kagan, BC Rosman, D Day, J Albert, W Phillips. Information processing in the child. Psychol Monogr 78:578, 1964.
62. J Block, JH Block, DM Harrington. Some misgivings about the Matching Familiar Figures Test as a measure of reflection-impulsivity. Dev Psychol 10:611–632, 1974.
63. DW Gerbing, SA Ahadi, JH Patton. Toward a conceptualization of impulsivity: components across behavioral and self-reported domains. Multivar Behav Res 22: 357–379, 1987.
64. MJ Morgan. Recreational use of "Ecstasy" (MDMA) is associated with elevated impulsivity. Neuropsychopharmacology 19:252–264, 1998.
65. E Cairns, T Cammock. Development of a more reliable version of the Matching Familiar Figures Test. Dev Psychol 11:244–248, 1978.
66. JS Baer, NJ Novick, AO Hummel-Schluger. Task persistence after alcohol consumption among children of alcoholics. Alcohol Clin Exp Res 19:955–960, 1995.
67. PC Kendall, JA Moses, AJ Finch. Impulsivity and persistence in adult inpatient "impulse" offenders. J Clin Psychol 36:363–365, 1980.
68. RV Heckel, SS Allen, L Andrews, G Roeder, P Ryba, W Zook. Normative data on the Kagan Matching Familiar Figures test for adult male incarcerates. J Clin Psychol 45:155–160, 1989.
69. CA Rohrbeck, CT Twentyman. Multimodal assessment of impulsiveness in abusing, neglecting, and nonmaltreating mothers and their preschool children. J Consult Clin Psychol 54:231–236, 1986.
70. R Milich, J Kramer. Reflections on impulsivity: an empirical investigation of impulsivity as a construct. In: K Gadow, ed. Advances in Learning and Behavioral Disabilities (vol 3). Greenwich, CT: JAI Press, 1984, pp 57–93.
71. HG Weijers, GA Wiesbeck, J Boning. Reflection-impulsivity, personality and performance: a psychometric and validity study of the Matching Familiar Figures Test in detoxified alcoholics. Pers Individ Dif 31:731–754, 2001.
72. RA Barkley. The assessment of attention deficit-hyperactivity disorder. Behav Assess 9:207–233, 1987.
73. J Block, JH Block, DM Harrington. Some misgivings about the Matching Familiar Figures Test as a measure of reflection-impulsivity. Dev Psychol 10:611–632, 1974.
74. DW Gerbing, SA Ahadi, JH Patton. Toward a conceptualization of impulsivity: Components across behavioral and self-report domains. Multivar Behav Res 22: 357–379, 1987.
75. J Monterosso, G Ainslie. Beyond discounting: Possible experimental models of impulse control. Psychopharmacology 146:339–347, 1999.
76. G Ainslie. Specious reward: A behavioral theory of impulsiveness and impulse control. Psychol Bull 82:463–496, 1975.
77. H Rachlin. The Science of Self Control. Boston: Harvard University Press, 2000.
78. MY Ho, S Mobini, TJ Chiang, CM Bradshaw, E Szabadi. Theory and method in the quantitative analysis of "impulsive choice" behaviour: Implications for psychopharmacology. Psychopharmacology 146:362–372, 1999.
79. L Green, H Rachlin. Commitment using punishment. J Exp Anal Behav 65:593–601, 1996.

MARCEL DEKKER, INC.
270 Madison Avenue, New York, New York 10016

80. CXC Poulos, ADA Le, JLJ Parker. Impulsivity predicts individual susceptibility to high levels of alcohol self-administration. Behav Pharmacol 6:810–814, 1995.
81. H Rachlin, A Raineri, D Cross. Subjective probability and delay. J Exp Anal Behav 55:233–244, 1991.
82. DR Cherek, SD Lane. Laboratory and psychometric measurements of impulsivity among violent and nonviolent female parolees. Biol Psychiatry 46:273–280, 1999.
83. DR Cherek, FG Moeller, DM Dougherty, H Rhoades. Studies of violent and nonviolent male parolees: II. Laboratory and psychometric measurements of impulsivity. Biol Psychiatry 41:523–529, 1997.
84. TJ Allen, FG Moeller, HM Rhoades, DR Cherek. Impulsivity and history of drug dependence. Drug Alcohol Depend 50:137–145, 1998.
85. RA Siegel. Probability of punishment and suppression of behavior in psychopathic and nonpsychopathic offenders. J Abnorm Psychol 87:514–522, 1978.
86. JP Newman, JF Wallace. Diverse pathways to deficient self-regulation: implications for disinhibitory psychopathology in children. Clin Psychol Rev 13:699–720, 1993.
87. W Matthys, SHM Van Goozen, H De Vries, PT Cohen-Kettenis, H Van Engeland. The dominance of behavioral activation over behavioral inhibition in conduct disordered boys with or without attention deficit hyperactivity disorder. J Child Psychol Psychiatry 39:643–651, 1998.
88. RK Heaton, GJ Chelune, JL Talley, GG Kay, G Curtis. Wisconsin Card Sorting Test Manual: Revised and Expanded. Odessa, FL: Psychological Assessment, 1993.
89. KW Greve, L Sherwin, MS Stanford, CW Mathias, JM Love, P Ramzinski. Personality and neurocognitive correlates of impulsive aggression in long-term survivors of severe traumatic brain injury. Brain Inj 15:255–262, 2001.
90. EE Gorenstein. Frontal lobe functions in psychopaths. J Abnorm Psychol 93:133–140, 1982.
91. D Lapierre, CMJ Braun, S Hodgins. Ventral frontal deficits in psychopathy: neuropsychological test findings. Neuropsychologia 33:139–151, 1995.
92. MD van den Broek, CM Bradshaw. An investigation of the relationship between perseveration and impulsiveness. Pers Individ Dif 14:531–534, 1993.
93. MS Stanford, KW Greve, JE Gerstle. Neuropsychological correlates of self-reported impulsive aggression in a college sample. Pers Individ Dif 23:961–966, 1997.
94. S Roussy, J Toupin. Behavioral inhibition deficits in juvenile psychopaths. Aggress Behav 26:413–424, 2000.
95. JR Stroop. Studies of interference in serial verbal reactions. J Exp Psychol 18: 643–662, 1935.
96. CJ Golden. Stroop Color and Word Test. Chicago; Stoelting, 1978.
97. N Deu. Executive function and criminal fantasy in the premeditation of criminal behavior. Criminal Behav Ment Health 8(suppl):41–50, 1998.
98. J Hopkins, T Perlman, L Hechtman, G Weiss. Cognitive style in adults originally diagnosed as hyperactives. J Child Psychol Psychiatry 20: 209–216, 1979.
99. H Chevalier, MN Metz-Lutz, SJ Segalowitz. Impulsivity and control of inhibition in benign focal childhood epilepsy (BFCE). Brain Cogn 43:86–90, 2000.

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100. J Brunas-Wagstaff, A Bergquist, GF Wagstaff. Cognitive correlates of functional and dysfunctional impulsivity. Pers Individ Dif 17:289–292, 1994.
101. J McGarth, S Scheldt, J Welham, A Clair. Performance on tests sensitive to impaired executive ability in schizophrenia, mania and well controls: acute and subacute phases. Schizophr Res 26:127–137, 1997.
102. M Gordon. Manual for the Gordon Diagnostic System. Dewitt, NY: Gordon Diagnostic Systems, 1979.
103. SD Lane, DR Cherek, DM Dougherty, FG Moeller. Laboratory measurement of adaptive behavior change in humans with a history of substance dependence. Drug Alcohol Depend 51:239–252, 1998.
104. FD McClure, M Gordon. Performance of disturbed hyperactive and nonhyperactive children on an objective measure of hyperactivity. J Abnorm Child Psychol 12:561–571, 1984.
105. SK Shapiro, HC Quay, AE Hogan, KP Schwartz. Response perseveration and delayed responding in undersocialized aggressive conduct disorder. J Abnorm Psychiatry 97:371–373, 1988.
106. PJ Frick, BR Loney. The use of laboratory and performance-based measures in the assessment of children and adolescents with conduct disorders. J Clin Child Psychol 29:540–544, 2000.
107. TP Zahn, MJP Kruesi, HL Leonard, JL Rapoport. Autonomic activity and reaction time in relation to extraversion and behavioral impulsivity in children and adolescents. Pers Individ Dif 16:751–758, 1994.

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15

Measurement of Aggression in Children and Adolescents

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INTRODUCTION

A national report by the Office of Juvenile Justice and Delinquency Prevention (1) found that 9% of U.S. high school students had carried a weapon to school within the previous 30 days. Furthermore, 44% of crime guns traced back to their owners belonged to individuals younger than 25, and 11% of the crime guns belonged to juveniles younger than 17. In 1997, 1400 homicides involved a juvenile offender, and this figure is probably an underestimate. These data clearly

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indicate that aggression and violence among children and adolescents is a leading public health concern.

As such, the accurate measurement of aggressive behavior is of paramount importance for both research and clinical applications. Nevertheless, a recent review of the literature indicates that the "gold standard" measure of aggression in children and adolescents has yet to be identified (2). Measures of aggression are needed to identify acts of aggression in children, to assess the persistence and desistance of aggression, to investigate factors associated with aggression, and to evaluate the efficacy of treatments. Aggressive behavior is among the most stable of all early detectable personality characteristics and, when present in childhood, is highly predictive of later delinquent and antisocial behavior (3–7). Early identification of behavior problems in children enables early intervention, which may be more efficacious than treatment started later in life (8).

One factor that complicates the assessment of aggression in youth is the developmental nature of aggressive behaviors, which range from variants of normal to pathological, and may be manifested by different behaviors at different ages. In infants, temperament and emotional regulation have been linked to later manifestations of aggression (9,10). Aggressive behavior in toddlers, in both boys and girls, is typified by physical aggression (e.g., hitting, kicking, throwing) and, to a greater extent, object-related aggression (e.g., grabbing toys) (11–13). Gender differences in the expression of aggression begin to emerge between the ages of 3 and 6 years (14–16), where boys begin to exhibit more physical aggression (17–19). Most children begin to display a decrease in physical aggression as they progress through middle childhood (20–22), although there are some data to suggest that the decrease in aggression begins earlier (21,23).

Considerable data indicate that aggression continues to diminish during adolescence (20) and is replaced by improved social skills; however, in a subgroup of adolescents, aggression steadily increases and the acts of aggression become more serious and violent (14). The adolescent's increased strength and access to weapons combine to make acts of aggression potentially more violent. Some data indicate that overt delinquent behaviors actually decrease during this time, while the incidence of covert delinquent behaviors increases (8). In contrast, the literature on antisocial behavior seems to suggest that adolescence is a period characterized by a generalized increase in antisocial behavior, irrespective of the nature of the aggressive acts (24). Most investigators seem to agree that there is little evidence for "late-onset" physical aggression; the majority of adolescents who are physically aggressive were physically aggressive as young children (25). These data coincide with results indicating that aggression is one of the most stable personality traits, equivalent to the stability evinced by measures of intelligence (6,8,26).

There are numerous risk factors which may act to promote patterns of aggressive behavior in children and adolescents. Children who display aggressive

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behavior often demonstrate compromised verbal abilities and impairments in executive functioning (24,27). Moreover, behavioral (e.g., hyperactivity, impulsivity, inattention), cognitive (e.g., low IQ, poor school performance), social/familial (e.g., gang membership, harsh parenting techniques, child abuse, familial conflict, low SES), and situational risk factors (e.g., access to alcohol, drugs, guns) all collude to perpetuate the cycle of violence (14,28). Recent studies indicate that there are gender differences in risk and protective factors. For example, high Verbal IQ may be a protective factor against the emergence of conduct disorder for boys, but not in girls (29).

The developmental trajectory of aggression has implications for the evaluation of aggressive behavior in children and adolescents. Preschoolers demonstrate aggression (hitting, kicking, grabbing toys) differently from adolescents (assault, acts of delinquency, covert acts of aggression). However, most scales use the same item pool to evaluate aggressive behavior across a large age range. The use of age norms may partially help to circumvent this issue, but in some cases, age-appropriate aggressive behaviors are omitted.

This chapter outlines various issues related to the measurement of aggression in children and adolescents. In addition, some of the more commonly used measures of aggression in children and adolescents will be reviewed. Behaviors such as delinquency and oppositionality/defiance, which are theoretically related to, and often confused with, aggression, will not be addressed other than to illuminate issues relevant to the measurement of aggression. While it is true that many, perhaps even most, aggressive children are oppositional, only a limited subset of oppositional children are physically aggressive (30,31). Furthermore, despite the high association between oppositional/defiant behaviors and aggression, the former are less predictive of course and/or long-term outcome (31,32).

ISSUES CONCERNING MEASUREMENT OF AGGRESSION

Subtypes of Aggression

Aggression, as manifested in children and adolescents, is characterized by a wide array of diverse behaviors. Despite the fact that in animals, different types of aggressive behavior can be readily categorized into subtypes with distinct neuroanatomical pathways and stereotyped patterns of behavior (33), this is not the case in children and adolescents. Investigators have proposed numerous subtypes of aggression in children and adolescents, but they do not correspond neatly to specific neuroanatomical or neurochemical indicators or mechanisms. Nevertheless, if subtypes of aggressive behavior exist in children and adolescents, then distinguishing them may have important implications for the understanding of etiology and treatment (34).

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On the basis of their studies of hostile attributional biases among aggressive children, Dodge and Coie (35) proposed a distinction between proactive (goal-directed, instrumental) and reactive (responding to a threat, retaliating) aggressive behavior. They found that children who were characterized as reactive-aggressive were more likely to 1) misperceive an ambiguous situation as an aggressive threat, and 2) respond with aggressive retaliations more often than proactive-aggressive boys, socially isolated boys, or normal controls. Price and Dodge (36) found that reactive aggression was associated with social rejection, whereas instrumental-proactive aggression (as distinct from bullying-proactive aggression) was associated with leadership skills and positive peer perceptions.

Other researchers have hypothesized that aggressive behavior in children can be subdivided into predatory (deliberate and controlled) and affective (impetuous and poorly controlled) aggressive behavior, and that these subtypes are differentially associated with levels of cognitive functioning (37). Vitiello and colleagues (37) found that children classified as predominantly predatory in nature were more likely to display higher verbal intelligence relative to children characterized as affectively aggressive.

Atkins and Stoff (38) differentiated between instrumental and hostile aggression in children. Instrumental aggression was associated with goal-directed behavior, in which there is some benefit or gain to the aggressor. In contrast, hostile aggression was perceived as an attempt to cause pain to the victim, with no independent gain. Aggressive children with Attention-Deficit/Hyperactivity Disorder (ADHD) were more likely to demonstrate hostile aggressive behavior on an analog task than aggressive children without AD/HD, suggesting that impulsivity plays a role in hostile aggression as well as in ADHD (38).

Finally, an example from the conduct disorder literature may prove useful. Loeber and Schmaling (39) suggested that antisocial behavior varied along a single dimension, whereby one end of the spectrum consisted of overt behavior (e.g., fighting, arguing) and the other end of the spectrum consisted of covert behavior (e.g., stealing, truancy). The decision to categorize these behaviors along a single dimension is an interesting one; however, this model does not account for the frequently observed overlap among overt and covert behaviors.

It has been suggested that the various proposed subtype schemes all are describing two clusters in the expression of childhood aggression: proactive/predatory/instrumental aggression, and reactive/affective/hostile aggression (33). Regardless of how one conceptualizes these dichotomies, the clinical value of these distinctions may be limited. There is some evidence that the distinctions between proactive and reactive aggression can differentially predict later conduct problems (40) or differentiate between those at risk for delinquency and those at risk for dating violence (41). However, discriminant validity is limited by the fact that most aggressive children exhibit both types of aggressive behavior (35,37,42).

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Measurement Issues

A second issue affecting the assessment of aggression relates to the manner in which the target behaviors are measured. Aggressive behavior can be measured by a frequency count (e.g., number of times per week/month/year, or not at all, somewhat, very much, etc.), by tallying the total number of behaviors (fights, argues, bullies, etc.), or through the ascertainment of severity (e.g., attacks peers vs. attacks adults, or bullying vs. homicide). Several scales' aggression scores (43–46) are composed of a combination of a tally of the total number of aggressive acts, and a frequency count. Other scales (47) rely more heavily upon frequency counts, a practice that can result in higher aggression scores for children who engage in daily skirmishes at school as opposed to those who commit a few serious assaults. For example, many children with ADHD who are not temperamentally aggressive get into frequent fights because some of their impulsive and overactive behaviors are annoying to other children who subsequently "retaliate." The manner in which aggressive behavior is measured may partially account for the poor discriminability between ADHD and aggression.

Aggression vs. Oppositionality/Defiance

Many scales confound the measurement of aggression with oppositionality, defiance, or other disruptive behaviors. The IOWA Conners (46) Aggression Index, for example, contains five items, none of which describes physical aggression. The items address emotions or behaviors such as lability, oppositionality, or disruptive behaviors. While these oppositional/defiant items are related to aggression (and represent a related construct which may constitute a precursor to aggression in young children), they do not necessarily tap into the construct of aggression, and may confound the measurement of aggressive behavior.

Situationality

Data suggest that children who are aggressive in multiple settings (e.g., home and school) are at a higher risk for future maladjustment than children who are just aggressive at home, or just aggressive at school (8,24). Aggressive behavior at home, as evidenced by early intrasibling aggression, has been shown to be predictive of later aggressive behavior (9). Other data suggest that a high rate of aggression directed toward a sibling is fairly "normal," is not different between boys and girls, and is not indicative of future maladjustment (14). In either case, information about where the aggression occurs may have important ramifications for the institution of a treatment plan. Current measures of aggression rarely address the issue of where the behavior occurs and whether the aggressive behavior occurs in multiple situations. Gathering data from multiple informants may partially help to circumvent this issue; however, while teachers usually report

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school behavior, parental reports of aggressive behavior probably consist of information gleaned from a variety of sources and settings.

Gender Differences

Considerable data indicate gender differences in the manifestation of aggression and antisocial behavior in children and adolescents (18,20,42,48–52). Gender differences seem to emerge during the preschool years (14,16). Boys tend to resort to more overt forms of aggression, such as fighting, whereas girls tend to utilize more subtle forms of aggression (e.g., ostracism, social isolation, slander, etc.), often referred to as "relational aggression" (18,19). Furthermore, engaging in nonnormative types of aggression (e.g., boys engaging in relational aggression and girls engaging in overt/physical aggression) may be indicative of more severe pathology than engaging in gender-normative types of aggression (53). Current measures of aggression rarely address the subtle types of aggression that are typically observed in females. For this reason, females may be viewed as less aggressive when in reality, such gender differences may be an artifact of how aggressive behavior is measured. In addition, gender differences may account for different patterns of persistence and desistence of aggressive behavior (14) and may impact a clinician's choice of treatment (54).

MEASURES OF AGGRESSION

There exists a wide array of instruments designed to measure aggression. These instruments can be divided into "broad-band" and "narrow-band" measures. Broad-band scales measure childhood psychopathology in general, and contain a subscale which evaluates aggression. Narrow-band scales are specifically designed to measure aggression. In this chapter, we consider both kinds of instruments, as well as other research and laboratory-based tasks.

General Childhood Psychopathology Scales, with Aggression Subscales

Achenbach Scales

The Achenbach scales (43–45) are empirically based tools for the assessment of general psychopathology in children and adolescents. There are three structurally similar scales, which are completed by the parent (Child Behavior Checklist; CBCL), teacher (Teacher's Report Form; TRF), or youth (Youth Self-Report; YSR). All three scales are composed of eight core syndromes, or subscales, which were derived from a principal components/varimax analysis of the CBCL items. The scales are named: Withdrawn, Somatic Complaints, Anxious/Depressed, Social Problems, Thought Problems, Attention Problems, Delinquent Behavior,

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and Aggressive Behavior. Using a secondary factor analysis, these eight subscales were further grouped into Internalizing (Withdrawn, Somatic Complaints, Anxious/Depressed) and Externalizing subscales (Delinquent Behavior, Aggressive Behavior), based on the premise that symptoms and behaviors can be divided into those directed against oneself and those directed at others. Three of the subscales (Social Problems, Thought Problems, and Attention Problems) could not be categorized along this dimension.

The *Child Behavior Checklist (CBCL)* (43) is a 118-item checklist of different behaviors on which parents rate their child's behavior during the past 6 months on a 3-point scale, consisting of "very true/often true," "somewhat/sometimes true," or "not true." The CBCL is normed for children between the ages of 4 and 18; a separate profile is available for 2- to 3-year olds.

The CBCL possesses highly respectable interrater and test-retest reliability. On the Aggressive Behavior subscale, interparent agreement ranged from $r = .66$ to $r = .86$ for boys and girls between the ages of 4 and 18. Test-retest reliability of the Aggressive Behavior subscale was $r = .91$ for both boys and girls. Content validity is supported by the fact that almost all CBCL items (with the exception of "Allergy" and "Asthma") are able to discriminate between referred and nonreferred children. Further, support for validity of the scale is derived from significant correlations with other scales measuring similar constructs, and the fact that all subscale scores are able to discriminate between referred and non-referred children, when demographic information is covaried.

The *Teacher's Report Form (TRF)* (44) is a modified version of the CBCL, in which certain items were added which relate to classroom behavior (e.g., Disturbs other students, Sleeps during class, Late to school or class, etc.) to replace the items that were inappropriate for teachers to rate. Teachers are required to rate the child's behavior on an identical 3-point scale, on the basis of their experience with the child during the past 2 months. The overall structure of the scale remains the same, there are 118 items, and the items load on the same eight subscales. This scale was normed on children ages 5–18 who were attending school.

Test-retest reliability correlations obtained for the Aggressive Behavior subscale over 15-day and 2- and 4-month intervals were significant, with correlation coefficients of .91, .77, and .68, respectively, indicating acceptable reliability. Interteacher reliability correlations were highest for the Aggressive Behavior subscale, indicating excellent agreement among teachers when rating children on aggressive behavior. Content validity was supported by the fact that almost all items (with the exception of "Allergy" and "Asthma") were able to differentiate between referred and nonreferred children. Construct validity was determined by comparing the TRF to other measures of childhood behavior [e.g., Conners Rating Scales-Revised (55)]. Similar to the CBCL, criterion-related validity was supported by the fact that the scale scores were able to discriminate between groups

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of referred and nonreferred children when demographic variables were controlled.

The *Youth Self Report (YSR)* (45) is also a modified version of the CBCL, designed to be completed by the child/adolescent between the ages of 11 and 18. Many of the problem items are the same as those in the CBCL and TRF, but the YSR includes 16 social desirability items which replace those items that were more appropriate for children than adolescents (making a total of 119 items). Adolescents are instructed to respond to the items based on their experiences during the past 6 months. Test-retest reliability of the Aggressive Behavior subscale during a 7-day period was $r = .78$ for ages 11–14 and $r = .87$ for ages 15–18. Seven-month test-retest reliability of the Aggressive Behavior subscale was $r = .48$ for ages 11–12 and $r = .46$ for ages 13–14. Content validity and criterion-related validity were evaluated in a manner similar to that used in the assessment of the CBCL and the TRF.

Comments. The CBCL accounts for developmental changes in aggressive behavior by supplying a different scale for toddlers, and by providing norms for different age ranges. The CBCL does not address the issue of subtypes of aggression. Aggressive behavior is measured using a combination of frequency and a tally of the total number of aggressive acts; however, the CBCL does not take severity of aggressive behavior into account. In addition, the items which load on the Aggression subscale do not necessarily relate to or measure aggression. Most of the items (e.g., Argues, Brags, Easily jealous, Shows off, Stubborn, Mood changes, Talks too much) may better be described as indicators of temperament or emotional lability. The issue of situationality is partially circumvented by the use of multiple informants (i.e., separate parent, teacher, and youth scales). In fact, the similar factor structure of the three Achenbach scales facilitates examination of cross-situationality and concordance. While separate norms are provided for each gender within each age range, the actual items measuring aggressive behavior for both genders are the same.

In general, the Achenbach scales are especially well suited for the global assessment of childhood psychopathology, and for evaluating the overall syndrome of aggression. They are probably less effective, however, for monitoring physical aggression and treatment response, or for the effective development of a behavioral treatment plan for the physically aggressive child.

Conners Rating Scales-Revised

The Conners Rating Scales-Revised (CRS-R) (55) are broad-based measures of childhood psychopathology, with a special emphasis on the assessment of AD/HD. Originally developed as a teacher-rating instrument for use in drug studies with hyperactive children (56), the Conners Rating Scales have undergone many

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revisions during the past 30 years. Loney and Milich (46) reanalyzed the Conners scale and selected five items which discriminated inattentive/overactive children and another five items that discriminated aggressive children. These 10 items were then renamed the IOWA (Inattention Overactivity With Aggression) Conners scale. Pelham and colleagues (57) subsequently renamed the aggression factor to reflect the fact that most of the items are consistent with oppositional defiance, and not aggression. Both factors have been validated in normative and clinic-referred samples (46,58,59).

Recently, Conners (55) has created a new set of parent, teacher, and self-report scales based on the original teacher-rating scale. These scales can be utilized for children between the ages of 3 and 17, with the exception of the self-report scale whose recommended age range is 12–17. There are short and long forms of each scale. In the interest of brevity, the subsequent analysis will only deal with the long form of each scale (parent, teacher, and adolescent self-report).

The *Conners Parent Rating Scale-Revised (CPRS-R)* (55) is an 80-item checklist in which the parent rates the child's behavior in the last month on a 4-point scale ["not true at all (never/seldom)," "just a little true (occasionally)," "pretty much true (often/quite a bit)," or "very much true (very often, very frequently)"]. The CPRS-R contains 14 subscales: Oppositional, Cognitive Problems/Inattention, Hyperactivity, Anxious-Shy, Perfectionism, Social Problems, Psychosomatic, Conners Global Index Total, Conners Global Index Restless-Impulsive, Conners Global Index Emotional Lability, ADHD Index, DSM-IV Symptoms Subscale Total, DSM-IV Symptoms Subscale Inattentive, and DSM-IV Symptoms Subscale Hyperactive-Impulsive.

The *Conners Teacher Rating Scale-Revised (CTRS-R)* (55) is a 59-item questionnaire similar to the parent version (CPRS-R), which is completed by the teacher. The scale utilizes the same response paradigms, the same items, and most of the same subscales. (There is no Psychosomatic subscale on the teacher form.)

The *Conners-Wells Adolescent Self-Report Scale (CASS)* (55) is an 87-item questionnaire for children and adolescents between the ages of 12 and 17, in which the adolescent responds to various items based on the way he/she has felt during the past month. The CASS contains 10 subscales: Family Problems, Emotional Problems, Conduct Problems, Cognitive Problems/Inattention, Anger Control Problems, Hyperactivity, ADHD Index, DSM-IV Symptoms subscale Total, DSM-IV Symptoms subscale Inattentive, and DSM-IV Symptoms subscale Hyperactive-Impulsive.

Reliability of the three scales is acceptable. Internal consistency coefficients range from .75 to .90, while test-retest reliability coefficients range from .60 to .90. The different subscales measure at least partially distinct constructs, as the correlations among the subscales were low to moderate.

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Comments. Thirty years ago, the Conners Rating Scale measured aggression by employing a "Defiance-Aggressive" subscale (56). The aggression factor was so prominent that factor analytic studies of the original scales raised questions as to whether the hyperactivity factor could be distinguished from the aggression factor. In its current incarnation, the Connors scales no longer contain an aggression subscale; however, the aggression items remain the same—they merely load on different subscales. For example, the item "Temper Outbursts," used to load on the Defiance-Aggressive subscale, but now loads on the Emotional Lability and the Global Index subscales. As such, it is difficult to obtain a unitary construct of aggression and/or evaluate the Conners scales as measures of aggression. The Conners scales are normed from ages 3 to 17, but the items relating to aggressive behavior remain the same across the ages. The Conners scales do not address the issue of subtypes of aggression. The scales have a flexible scoring system, whereby one can obtain a raw score based on symptom count, or obtain a norm-based/reference score by comparing the child to other children of roughly the same age. Severity of aggressive acts is not taken into account in either case. The issue of situationality is partially circumvented by the use of multiple informants (separate parent, teacher, and adolescent scales). Although gender differences are addressed through the use of separate norms for boys and girls, the items themselves may be more characteristic of "male" forms of aggression. In summary, the Conners scales inform on the presence of aggressive behavior, but cannot be used specifically to measure aggression.

Child Behavior Scale

The Child Behavior Scale (CBS) is a teacher questionnaire designed to measure children's aggressive, withdrawn, and prosocial behaviors (60–62). The CBS is a 59-item questionnaire, with a subset of items adapted from existing scales. Teachers rate their students on a 3-point scale as to whether the behavior is characteristic of the target child: "certainly applies," "applies sometimes," or "doesn't apply." Six subscales are identified: Aggressive with Peers, Prosocial with Peers, Excluded by Peers, Asocial with Peers, Hyperactive-Distractible, and Anxious-Fearful. The investigators caution that some of the subscales need further investigation (e.g., Prosocial with Peers, Excluded by Peers, and Hyperactive-Distractible).

Internal consistency for the various CBS subscales, including the Aggressive with Peers subscale, was shown to be highly respectable. Test-retest reliability was assessed by administering the scale twice across a 4-month interval, and the subscale scores were generally stable over time. Construct validity was assessed by comparing the CBS subscales to other concurrent measures of children's behaviors. Children who scored high on the CBS aggression scale were more likely to be rated by observers as engaging in aggressive interactions, more

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likely to dominate their peers, and less likely to engage in prosocial behaviors. The CBS Aggressive Behavior subscale correlated more highly with the TRF Aggressive Behavior Scale (44) than with any other subscales on the TRF. Concurrent and predictive validity analyses revealed that, on average, boys were rated as more aggressive than girls, and that higher Aggressive Behavior scores were correlated with lower levels of classroom peer acceptance.

Comments. This scale is useful for evaluating a child's behavior in the school setting and identifying those children who exhibit behaviors which place them at risk for future adjustment problems. The CBS also evaluates positive social factors/behaviors which may act to protect the child from future maladjustment (e.g., Prosocial with Peers). Owing to its restricted age range (normed on children age 5–6 years); however, the CBS does not adequately address the issue of developmental changes in aggression. This scale also does not address the issue of subtypes of aggression. The CBS measures aggression by combining the total number of behaviors and the frequency of the behaviors. Most of the items on the Aggressive with Peers subscale seem to be related exclusively to aggression, with perhaps one or two exceptions (Argues, Taunts/teases). This scale does not address the issue of situationality, since all items pertain to school-based aggression. Finally, gender differences are not addressed by this scale—there are not separate norms for boys and girls, and the full range of behaviors thought to be associated with aggression in girls is not present.

Preschool Behavior Questionnaire

The Preschool Behavior Questionnaire (PBQ) is a 30-item teacher rated questionnaire for preschool-aged children (63). It is based on the Rutter's Children's Behavior Questionnaire (64,65), and was developed as a short screening instrument for problem behaviors in children ages 3–6. Behaviors are rated on a 3-point scale ("Does not apply," "Applies sometimes," or "Frequently applies"). The scale was later modified (66) to include a prosocial factor, and the resulting Preschool Social Behavior Questionnaire (PSBQ) has also been used in the assessment of childhood aggression (67,68).

A factor analysis of the PBQ revealed three main factors: Hostile-Aggressive, Anxious-Fearful, and Hyperactive-Distractible. Mean interrater reliability of the scale was $r = .79$, and the Hostile-Aggressive factor had an interrater reliability coefficient of .76. Test-retest reliability after a period of 3–4 months was $r = .87$, and the Hostile-Aggressive Score specifically had a correlation coefficient of $r = .93$. An analysis of the individual items indicated that most of them, as well as the scale as a whole, were able to differentiate between normal preschoolers and preschoolers with emotional difficulties, supporting the scale's criterion-related validity.

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Comments. The PBQ is specifically designed for preschool children, and as such, contains developmentally appropriate items relating to aggression in preschool children (e.g., Doesn't share toys, Kicks/bites/hits). The score is based on a tally of frequency counts. Some of the items on the aggression scale (Inconsiderate, Blames others) do not describe aggressive behavior per se, but are more typical of oppositional behavior. The scale was modified for use only by teachers, and as such cannot evaluate aggression in the home environment. This scale does not address the issue of gender differences.

Specific Measures of Aggression

Children's Aggression Scales

The Children's Aggression Scale–Parent (CAS-P) (69) and the Children's Aggression Scale–Teacher (CAS-T) (70) were designed to measure the frequency and severity of aggressive acts in noninstitutionalized children, in different settings (e.g., home vs. school), without the confound of oppositional/defiant behavior. The 33-item CAS-P and the 23-item CAS-T were modeled after several existing measures, including the Overt Aggression Scale (71). The CAS-P and CAS-T are designed to capture five different domains: Verbal Aggression, Aggression Against Objects and Animals, Provoked Physical Aggression, Unprovoked Physical Aggression, and Use of Weapons.

Reliability analyses identified excellent overall internal consistency for both the CAS-P ($\alpha = 0.93$) and the CAS-T ($\alpha = 0.92$), and acceptable reliability estimates for the individual subscales. The CAS-P effectively distinguished levels of aggression in children diagnosed with ADHD, ODD, and CD. The CAS-T subscales, however, did not significantly distinguish among children with ADHD only, those with ODD only, and those without a disruptive history disorder. Nonetheless, all three groups were rated significantly lower than children with CD. Correlational data provide support for both the convergent and divergent validity of the CAS-P and the CAS-T. Parent ratings of Verbal ($r = .41$) and Initiated Physical Aggression ($r = .33$) on the CAS-P correlated significantly with teacher ratings of aggression on the IOWA, but none of the subscales correlated significantly with teacher ratings of inattention/overactivity. The CAS-T ratings were found to be significantly and robustly correlated with parent CBCL ratings of aggression and delinquency, but not with ratings of attentional problems. Validity for the Weapons subscale could not be adequately assessed in either the CAS-P or the CAS-T owing to the low frequency with which children in the validation sample carried weapons.

Comments. This is a new scale that attempts to address a variety of issues relating to the measurement of aggression that have not previously been captured in rating scales. Two of the subscales (Provoked and Initiated Physical Aggres-

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sion) were created to accommodate the distinction between instrumental and responsive aggression, although it is unclear whether the distinction of who initiated the aggressive act vs. who was provoked can be determined with much accuracy or precision. Other subscales address the issue of verbal vs. physical aggression, another important descriptive point in the literature on aggression subtypes, which is possibly sensitive to gender differences. Aggressive behavior is measured in this scale using a combination of frequency and severity. Severity of aggressive behavior is determined by rating each subscale of behaviors on a continuum of less severe to more severe aggressive acts (e.g., Snapped or yelled at children living in the home, Cursed or sworn at children who live in the home, Verbally threatened to hit a child who lives in the home). The scale includes only those items that describe aggression, and excludes oppositional or defiant behaviors. To address the issue of aggression in different settings, the parent scale distinguishes between aggressive acts directed toward individuals who live at home and those directed toward individuals who do not live with the child. The scale is not specifically geared to measure gender differences in aggressive behavior, although the inclusion of verbal aggression items may prove somewhat sensitive to gender differences. There are not separate norms for males and females; the standardization sample was composed almost entirely of males between the ages of 7 and 11.

New York Teacher Rating Scale

The New York Teacher Rating scale (NYTRS) for disruptive and antisocial behavior is a measure designed to capture aberrant behavior specific to Conduct Disorder (CD) and Oppositional Defiant Disorder (ODD) (47). It is composed of items from several existing measures, including the Revised Behavior Problem Checklist, Conner Teacher Rating Scale-Revised, IOWA Conners Teacher Rating Scale, DSM-IV (72) items for ODD and CD, as well as new items. There are 36 items, forming four factors (Defiance, Physical aggression, Delinquent aggression, and Peer relations); four additional items describing DSM-IV (72) CD; and two more items relating to global impairment. Items from the Physical and Delinquent Aggression factors, and those pertaining to conduct problems form the Antisocial Behavior Scale. Items from the Defiance factor, in conjunction with the Antisocial Behavior Scale, form the Disruptive Behavior Scale. Teachers are required to rate each child's behavior on a four-point scale: "not at all," "just a little," "pretty much," and "very much."

Internal consistency was high for most factors and composite scales (ranging from $\alpha = 0.78–0.96$), except for the Delinquent Aggression factor ($\alpha = 0.49$ in a CD sample). Test-retest reliability over a 5-week period was established for the factors and scales (range from 0.62 to 0.87) and interrater reliability was moderate (ICC ranged from 0.27 to 0.60). The validity of the NYTRS was established by the comparing the scale to other measures assessing similar constructs,

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by assessing the ability of the scale to identify children with CD from among the general population, and by comparing factor scores with the general composite scores.

Comments. An important strength of the NYTRS is that it effectively separates oppositional/defiant behavior from aggressive behavior. Behaviors are measured using a frequency count; however, there are two items that address the issue of the severity of the child's behavior. This teacher rating scale does not address the issue of developmental changes in the expression of aggression (although the investigators do mention that no grade effects were found for any of the scales); however, it may be useful for distinguishing among different types of aggression (physical vs. delinquent). This scale was specifically composed of items that relate to school and classroom behavior, and therefore is not appropriate for the assessment of behaviors that may occur in the home or outside the school setting. Finally, the issue of gender differences is not addressed in this scale.

Overt Aggression Scale

The Overt Aggression Scale (OAS) was created by Yudofsky and colleagues (71) to measure acts of aggression and to monitor the efficacy of subsequent interventions delivered to children and adults within the context of an inpatient psychiatric setting (71,73–76). The OAS is completed by a trained member of a hospital staff, and is well suited to circumstances in which patients may be too cognitively impaired to complete a self-report inventory. The scale was specifically designed to discriminate between hostility and assaultive behavior, and among different types of aggressive behavior. This 25-item questionnaire measures aggressive behavior in four different domains, each with four levels of severity: Verbal Aggression, Physical Aggression Against Objects, Physical Aggression Against Self, and Physical Aggression Against Others. For each act of aggression, the observer must also document what type of intervention (e.g., holding patient, medication, isolation, seclusion, restraint, etc.) was utilized. The OAS has since been modified into a rated scale (OAS-M) (77), but the OAS-M has not been used extensively in children (78).

The OAS demonstrates good reliability, with intraclass coefficients of $>$ 0.75 for most items. However, while the scale has respectable reliability, the authors caution that the verbal aggression factor and intervention factors have the lowest reliability. OAS scores have been found to be correlated with a more general measure, the Global Clinical Consensus Rating, suggesting that the OAS may be an effective tool in treatment studies (74,75).

Comments. The OAS effectively discriminates between different types of aggression, such as verbal and physical aggression. It uses a combination of frequency and severity to measure acts of aggression. Within each category, ag-

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gressive behavior is listed on a continuum from less violent to more violent, and as such, the scale effectively captures more extreme instances of aggressive behavior. The OAS almost exclusively uses items which relate to aggression, so there are no confounds of items relating to oppositional or defiant behavior. Since this scale was created for use in institutionalized settings, it does not address situational or contextual aspects of aggression. Indeed, its primary use for inpatient settings may render it less valid or appropriate for documenting aggression within outpatient or school contexts. Given that many of the behaviors addressed by the OAS occur with a low degree of frequency (e.g., Threatens to kill someone, Sets fires, Mutilates self, Attacks others causing severe physical injury, etc.), its discriminant validity may be called into question when used for outpatient applications (i.e., the OAS will not discriminate among less severe manifestations of aggression). Finally, this scale does not address the issue of gender differences in the expression of aggression, or diverse ways in which aggression is manifested at different ages.

Additional Scales

There are additional aggression scales which have been primarily designed to address a single hypothetical question relating to aggression, are not formally named, and are used almost exclusively for research purposes (18,35,37). A teacher rating scale by Dodge and Coie (35) was developed to capture the difference between proactive and reactive aggressive behavior in children. Teachers rate each child on a 5-point scale (ranging from "Never," to "Almost always"), indicating the frequency with which each child has engaged in the various behaviors. The scale is composed of three items reflecting proactive aggression and three items reflecting reactive aggression. There is limited support for the independence of the two constructs, suggesting that most teachers perceive aggression as a unitary construct, and do not rate children differently on two different types of aggression (35). Although this scale has been used in research applications (34,36), future research is needed to establish its validity and reliability.

Vitiello and colleagues (37) created a scale designed to capture the difference between predatory and affective aggressive behavior in children. This 16-item, yes-no questionnaire is composed of eight items reflecting predatory behavior, and eight items which describe more affective aggression; it is designed to be completed by hospital staff. Cluster analysis revealed that 10 of the original 16 items fell into two groups, divided along the lines of the predatory vs. affective distinction. Individual scores were best represented by a bimodal distribution, with some children/adolescents scoring high on the predatory items, and others scoring high on the affective items; a third group displayed both predatory and affective aggressive features. Internal consistency of the scale was satisfactory (Chronbach's $\alpha = 0.73$). While intended to address the distinction between types

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of aggression, it is unclear as to whether this scale will have clinical utility in an outpatient setting. Individual items place considerable emphasis on whether the aggression was planned and/or can be controlled, a determination which is often difficult for an outside evaluator. The scale makes no other provisions with respect to other aggression subtypes, does not account for the severity or frequency of each aggressive act, and is designed only for inpatient settings.

Comments. Both of the above scales possess great heuristic value. They address the issue of subtypes of aggression and provide an excellent starting point in our attempt to understand the different manifestations of aggression in children and adolescents. However, it remains to be determined whether they can be adapted for more widespread clinical use. Further, additional research is needed to continue documenting the presence of different subtypes, and these scales provide an important beginning.

Peer Nominations

Peer nominations, in which children identify which of their classmates are the most aggressive (e.g., "Who pushes and shoves other children"), represent an intriguing alternative for assessing aggression in children and are often used in research settings (22,48,51,61,79–81). Although peer nominations can be time-consuming and costly, they have the advantage of utilizing numerous raters for each child. Moreover, since children typically have extensive interactions with their classmates, they may be particularly insightful informants (82). Huesmann and colleagues (82) have developed an innovative system which uses a combination of peer nominations and teacher ratings. They proposed that teachers estimate how their students would complete a peer nomination rating, and then request that the teachers complete the peer nomination from their students' perspective. They found that the teachers' predictions of peer nominations of aggression were highly accurate and valid, but that teachers' predictions of peer nominations for popularity and victimization were particularly poor.

Comments. The use of peer nominations offers a unique perspective on aggression in children by effectively utilizing multiple knowledgeable informants to gauge the level of aggression during peer interactions. However, use of peer nominations is limited by the fact that it involves considerable time and expense. There are also ethical issues to be considered, specifically whether the use of peer nominations is likely to be associated with long-standing changes in how children view their peers. Because of this concern, use of peer nominations in research has recently been subject to more stringent ethical standards, and some institutional review boards have mandated that investigators obtain informed consent from the parents of every child participating in a peer nomination procedure.

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Summary

As described above, behavioral ratings constitute an ecologically sensitive approach to the study of aggressive behavior, allowing investigators and clinicians to better gain insight into aggressive behavior as manifested in everyday settings (e.g., home and school environments). Furthermore, such strategies permit investigators to explore the duration and chronicity of childhood aggression as well as the pervasiveness of aggressive acts.

Despite the utility of behavioral ratings, such strategies are clearly not without their limitations. As previously noted, many behavioral ratings lack the capacity to distinguish between the various subtypes of aggression previously described (e.g., provoked vs. initiated aggression) and often fail to adequately reconcile issues of severity vs. frequency. The assessment of aggression as a unitary measure may pose difficulties for the development of an individual treatment plan. Furthermore, the failure to take into account the heterogeneity of aggression and developmental changes in the manifestation of aggressive behavior may lead clinicians to falsely conclude that aggressive behavior has declined over time when in fact only the nature of such acts has changed.

Secondly, traditional behavioral rating scales may be less capable of distinguishing aggressive behavior from other disruptive behavior problems such as oppositional-defiant behavior and hyperactivity/impulsivity. In part, because a significant proportion of aggressive children meet diagnostic criteria for one or more comorbid psychiatric disorders, it is not surprising that investigators (83) have suggested that the severity of conduct problems may lead informants to endorse difficulties in other behavioral domains (halo effects). While the assessment of a wide array of behavioral difficulties is an essential component of a good clinical evaluation, it is important that aggressive and non-aggressive disruptive behaviors be clearly differentiated.

Finally, behavioral ratings may be susceptible to informant biases, which can distort (i.e., inflate or minimize) the reported severity of childhood aggression. Given that caretakers often pursue clinical assistance at a time when the child's behavior has become especially problematic, clinical ratings, which are supposed to take into account the child's behavior over the past 6 or 12 months, may be inflated by the magnitude of the child's current behavioral difficulties.

Laboratory Measures

Another strategy that may be beneficial to the study of childhood aggression involves the application of laboratory-based measures. Although concerns have been raised regarding their ecological validity and their ability to tap into situational/contextual factors associated with the expression of aggressive behavior, laboratory measures are typically more objective and therefore less apt to be affected by rater biases. In addition, laboratory-based paradigms may be more

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readily able to isolate specific subtypes of aggressive behavior (e.g., covert aggression) and/or related behaviors (e.g., impulsivity) which may underlie aggressive acts. Laboratory measures are gaining popularity in aggression research, yet have not made the crossover into clinical practice (84). Such techniques are often utilized to test specific hypotheses, often related to a type or mechanism of aggression. Nonetheless, laboratory measures may be useful for validating behavioral ratings, and may serve as a valuable adjunct to other clinical assessment procedures. Several of these paradigms/measures are described below.

Provocation paradigms are tasks designed to elicit an aggressive response from the child. The provocations are uniform and standardized, enabling investigators to compare the children's responses across situations; children are usually provided with various aggressive and nonaggressive response options (85). These tasks tap into how individuals react to different stimuli and assess the degree to which they are easily angered.

The *Point Subtraction Aggression Paradigm (PSAP)* (86) is an example of a measure developed to test the ease with which individuals become aggressive, and the nature of their interaction with others. This measure has been developed for use in adults, and it has only recently been studied in children/adolescents (87,88). In this paradigm, participants try to earn money for completing a task, while a fictitious peer periodically provokes them by subtracting money from their "account." Players can respond by either attempting to earn more money, protecting their earnings, or subtracting money from their opponent. This laboratory task has demonstrated considerable external validity in adults (89,90). In addition, scores on this instrument in a group of high-school athletes were able to predict the type of contact sport (high/low) played by the athletes as a function of the number of hostile responses exhibited during the PSAP game (87).

Another computerized provocation task simulates a pinball game, in which the child is allegedly playing against a peer who repeatedly provokes the child (38,91). The child can respond to the provocations by delivering a blast of white noise (hostile response), or temporarily blocking the peer's game (instrumental response). The two different types of responses are intended to reflect the conceptual dichotomy between hostile and instrumental aggression. Analyses investigating the relationships between the two different types of aggressive responses and psychiatric diagnoses found that both aggressive children with ADHD and aggressive children without ADHD utilized instrumental aggressive responses more frequently than the normal controls. However, children with comorbid ADHD and aggression were more likely to utilize hostile aggressive responses than the aggressive children without ADHD, indicating a connection between impulsivity and hostile aggression (38).

The *Intention-Cue Detection Tasks* are a series of laboratory tasks designed to measure the cognitive processing of social cues in two types of aggressive children (35,92–95). The investigators hypothesized that children with reactive

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aggression, as opposed to those with proactive aggression, would display a deficit in the cognitive processing of social and emotional stimuli. Participants observed taped vignettes of children interacting with one another, and provoking one another. The series of vignettes includes episodes of intentional and accidental provocation, as well as vignettes that are intentionally ambiguous with regard to provocation. Participants are asked to rate the provocateur's intent, and to explain how he or she would respond to the provocation. Certain types of aggressive children, namely those with reactive aggression, display a processing deficit called the "hostile attributional bias," whereby they incorrectly attribute hostile intent to children and situations where no such intent is present.

The *Pulkkinen Aggression Machine*, a computerized touch-screen laboratory task, employs a series of pictures designed to elicit different types of aggressive responding (96). Each picture portrays provocative interactions of varying intensity (e.g., children being pushed, slapped, punched, etc.), and the child is told to imagine that they are the target of the aggressive provocation. The child has many different options of responding (e.g., pushing back, slapping back, punching, hitting child with stick, etc.). This laboratory task has been used to study the effects of situational/contextual cues, and to investigate the difference between impulsive and controlled aggression in different types of children (96). This is a very new technique and limited data are available at this point.

Summary. Laboratory measures of aggression are generally designed to measure specific hypothetical constructs. These hypothetical constructs and resulting lab paradigms sometimes evince considerable external validity. However, various practical and ethical concerns, include the use of deception for some laboratory measures, may preclude their implementation in clinical settings (85).

CONCLUSIONS

Accurate assessment of aggressive behavior in youth is the first step toward understanding the determinants of this maladaptive behavior pattern, as well as the development of effective interventions both on individual and societal levels. The most common approach to the assessment of aggression in children is through the use of parent and teacher rating scales, and these are often supplemented by self-reports from adolescents. While these rating scales have been the mainstay of the field for several decades, and have played a key role in research protocols as well as assessment and treatment of individuals, important limitations have placed constraints on their utility. In particular, many of the currently available instruments lack sensitivity to developmental changes in the ways in which aggression is manifested, and to the more subtle behaviors that tend to characterize aggression in girls. Further, many scales do not clearly differentiate aggression from oppositional-defiant behavior, lack sensitivity to differences in severity be-

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tween individual acts, and do not adequately capture the diversity of motivations, behaviors and settings associated with aggression in youth. Some of these limitations are partially rectified by the use of laboratory measures of aggression. However, concerns regarding ecological validity have limited the use of these approaches.

The above constraints upon the accurate assessment of aggressive behavior in children and adolescents clearly limit the validity of any individual assessment instrument. Nevertheless, different instruments tend to have different limitations. As such, the use of a multimethod, multi-informant approach allows clinicians and investigators to most accurately capture the nature, diversity, and severity of aggressive behavior in children and adolescents.

REFERENCES

1. HN Snyder, M Sickmund. Juvenile Offenders and Victims: 1999 National Report. Washington: Office of Juvenile Justice and Delinquency Prevention, 1999.
2. D Gothelf, A Apter, HM van Praag. Measurement of aggression in psychiatric patients. Psychiatry Res 71:83–95, 1997.
3. DP Farrington. Childhood aggression and adult violence: early precursors and later life outcomes. In: DJ Pepler, KH Rubin, eds. The Development and Treatment of Childhood Aggression. Hillsdale, NJ: Lawrence Erlbaum, 1991, pp 5–29.
4. CE Lewis, LN Robins, J Rice. Associations of alcoholism with antisocial personality in urban men. J Nerv Ment Dis 173:166–174, 1985.
5. F Loeber, T Dishion. Early predictors of male delinquency: a review. Psychol Bull 94(1):68–99, 1983.
6. D Olweus. Stability of aggressive reaction patterns in males: a review. Psychol Bull 86(4):852–875, 1979.
7. RE Tremblay, B Mâsse, D Perron, M LeBlanc, AE Schwartzman, JE Ledingham. Early disruptive behavior, poor school achievement, delinquent behavior, and delinquent personality: longitudinal analyses. J Consult Clin Psychol 60:64–72, 1992.
8. R Loeber. The stability of antisocial child behavior: A review. Child Dev 53:1431–1446, 1982.
9. L Kingston, M Prior. The development of patterns of stable, transient, and school-age onset aggressive behavior in young children. J Am Acad Child Adolesc Psychiatry 34(3):348–358, 1995.
10. JE Bates, K Bayles, DS Bennett, B Ridge, MM Brown. Origins of externalizing behavior problems at eight years of age. In: DJ Pepler, KH Rubin, eds. The Development and Treatment of Childhood Aggression. Hillsdale, NJ: Lawrence Erlbaum, 1991, pp 93–120.
11. DF Hay, J Castle, L Davies. Toddlers' use of force against familiar peers: a precursor of serious aggression? Child Dev 71(2):457–467, 2000.
12. K Keenan, DS Shaw. The development of aggression in toddlers: a study of low-income families. J Abnorm Child Psychol 22(1):53–77, 1994.

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

13. M Caplan, J Vespo, J Pedersen, DF Hay. Conflict and its resolution in small groups of one- and two-year-olds. Child Dev 62:1513–1524, 1991.
14. R Loeber, D Hay. Key issues in the development of aggression and violence from childhood to early adulthood. Ann Rev Psychol 48:371–410, 1997.
15. K Keenan, D Shaw. Developmental and social influences on young girls' early problem behavior. Psychol Bull 121(1):95–113, 1997.
16. NR Crick, JF Casas, M Mosher. Relational and overt aggression in preschool. Dev Psychol 33(4):579–588, 1997.
17. R Loeber, M Stouthamer-Loeber. Development of juvenile aggression and violence: some common misperceptions and controversies. Am Psychol 53(2):242–259, 1998.
18. NR Crick, JK Grotpeter. Relational aggression, gender, and social-psychological adjustment. Child Dev 66:710–722, 1995.
19. D Olweus. Bullying at school: basic facts and effects of school based intervention program. J Child Psychol Psychiatry 33(7):1171–1190, 1994.
20. BB Lahey, M Schwab-Stone, SH Goodman, ID Waldman, G Canino, PJ Rathouz, TL Miller, KD Dennis, H Bird, PS Jensen. Age and gender differences in oppositional behavior and conduct problems: a cross-sectional household study of middle childhood and adolescence. J Abnorm Psychol 109(3):488–503, 2000.
21. R Loeber, BB Lahey, C Thomas. Diagnostic conundrum of oppositional defiant disorder and conduct disorder. J Abnorm Psychol 100(3):379–390, 1991.
22. DS Moskowitz, AE Schwartzman, JE Ledingham. Stability and change in aggression and withdrawal in middle childhood and early adolescence. J Abnorm Psychol 94(1):30–41, 1985.
23. EM Cummings, RJ Iannotti, C Zahn-Waxler. Aggression between peers in early childhood: individual continuity and developmental change. Child Dev 60:887–895, 1989.
24. TE Moffitt. Adolescence-limited and life-course-persistent anti-social behavior: A developmental taxonomy. Psychol Rev 100:674–701, 1993.
25. B Brame, DS Nagin, RE Tremblay. Developmental trajectories of physical aggression from school entry to late adolescence. J Child Psychol Psychiatry 42(4):503–512, 2001.
26. D Olweus. The consistency issue in personality psychology revisited—with special reference to aggression. Br J Clin Psychol 19(4):377–390, 1980.
27. JR Séguin, RO Pihl, PW Harden, RE Tremblay, B Boulerice. Cognitive and neuropsychological characteristics of physically aggressive boys. J Abnorm Psychol 104(4):614–624, 1995.
28. DP Farrington, R Loeber. Epidemiology of juvenile delinquents. Child Adolesc Psychiatr Clin North Am 9(4):733–748, 2000.
29. V Koda. Gender differences in the neuropsychology of childhood aggression. Dissertation Abstracts International: Section B: Sciences & Engineering, 60(1-B). US: Univ. Microfilms International, 1999.
30. JM Halperin, JH Newcorn, K Matier, G Bedi, S Hall, V Sharma. Impulsivity and the initiation of fights in children with disruptive behavior disorders. J Child Psychol Psychiatry Allied Disc 36:1199–1211, 1995.

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31. R Loeber, P Wung, K Keenan, B Giroux, M Stouthamer-Loeber, WB van Kammen, B Muughan. Developmental pathways in disruptive child behavior. Dev Psychopathol 5:103–133, 1993.
32. J Haapasalo, RE Tremblay. Physically aggressive boys from ages 6–12: Family background, parenting behavior, and prediction of delinquency. J Consult Clin Psychol 62:1044–1052, 1994.
33. B Vitiello, DM Stoff. Subtypes of aggression and their relevance to child psychiatry. J Am Acad Child Adolesc Psychiatry 36(3):307–315, 1997.
34. NR Crick, KA Dodge. Social information-processing mechanisms in reactive and proactive aggression. Child Dev 67:993–1002, 1996.
35. KA Dodge, JD Coie. Social information-processing factors in reactive and proactive aggression in children's peer groups. J Pers Social Psychol 53:1146–1158, 1987.
36. JM Price, KA Dodge. Reactive and proactive aggression in childhood: Relations to peer status and social context dimensions. J Abnorm Child Psychol 17:455–471, 1989.
37. B Vitiello, D Behar, J Hunt, D Stoff, A Ricciuti. Subtyping aggression in children and adolescents. J Neuropsychiatry Clin Neurosci 2:189–192, 1990.
38. MS Atkins, DM Stoff. Instrumental and hostile aggression in childhood disruptive behavior disorders. J Abnorm Child Psychol 21:165–178, 1993.
39. R Loeber, KB Schmaling. Empirical evidence for overt and covert patterns of antisocial conduct problems: a meta-analysis. J Abnorm Child Psychol 13:337–352, 1985.
40. F Vitaro, PT Gendreau, RE Tremblay, P Oligny. Reactive and proactive aggression differentially predict later problems. J Child Psychol Psychiatry 39(3):377–385, 1998.
41. M Brendgen, R Vitaro, RE Tremblay, F Lavoie. Reactive and proactive aggression: Predictions of physical violence in different contexts moderating effects of parental monitoring and caregiving behavior. J Abnorm Child Psychol 24(4):293–304, 2001.
42. MK Underwood, BR Galen, JA Paquette. Top ten challenges for understanding gender and aggression in children: why can't we all just get along? Soc Dev 10(2): 248–266, 2001.
43. TM Achenbach. Manual for the child behavior checklist 4/18 and 1991 profile. Burlington: University of Vermont Department of Psychiatry, 1991.
44. TM Achenbach. Manual for the Teacher's Report Form and 1991 Profile. Burlington: University of Vermont Department of Psychiatry, 1991.
45. TM Achenbach, T.M. Manual for the Youth Self-Report and 1991 Profile. Burlington: University of Vermont Department of Psychiatry, 1991.
46. J Loney, R Milich. Hyperactivity, inattention, and aggression in clinical practice. Adv Dev Behav Pediatr 3:113–147, 1982.
47. LS Miller, RG Klein, J Piacentini, H Abikoff, MR Shah, A Samoilov, M Guardino. The New York Teacher Rating Scale for disruptive and antisocial behavior. J Am Acad Child Adolesc Psychiatry 34(3):359–370, 1995.
48. K Björkqvist, KMJ Lagerspetz, A Kaukiainen. Do girls manipulate and boys fight?

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

Developmental trends in regard to direct and indirect aggression. Aggress Behav 18:117–127, 1992.
49. EE Maccoby, CN Jacklin. Sex differences in aggression. Child Dev 51:964–980, 1980.
50. M Zoccolillo, R Tremblay, F Vitaro. DSM-III-R and DSM-III criteria for conduct disorder in pre-adolescent girls: specific but insensitive. J Am Acad Child Adolesc Psychiatry 35:461–470, 1996.
51. NR Crick, MA Bigbee, C Howes. Gender differences in children's normative beliefs about aggression: How do I hurt thee? Let me count the ways. Child Dev 67:1003–1014, 1996.
52. RB Cairns, BD Cairns, HJ Neckerman, LL Ferguson, JL Gariepy. Growth and aggression, I. Childhood to early adolescence. Dev Psychol 25:320–330, 1989.
53. NR Crick. Engagement in gender normative versus nonnormative forms of aggression links to social-psychological adjustment. Dev Psychol 33(4):610–617, 1997.
54. K Keenan, R Loeber, S Green. Conduct disorder in girls: a review of the literature. Clin Child Fam Psychol Rev 2(1):3–19, 1999.
55. CK Connors. Conners' Rating Scales-Revised: Technical Manual. New York: Multi-Health Systems, 1997.
56. CK Conners. A teacher rating scale for use in drug studies with children. Am J Psychiatry 126:884–888, 1969.
57. WE Pelham, R Milich, DA Murphy, HA Murphy. Normative data on the IOWA Conners Teacher Rating Scale. J Clin Child Psychol 18:259–262, 1989.
58. R Milich, G Fitzgerald. Validation of inattention/overactivity and aggression ratings with classroom observations. J Consult Clin Psychol 53(1):139–140, 1985.
59. MS Atkins, WE Pelham, MH Licht. The differential validity of teacher ratings of inattention/overactivity and aggression. J Abnorm Child Psychol 17(4):423–35, 1989.
60. GW Ladd, SM Profilet. The Child Behavior Scale: a teacher-report measure of young children's aggressive, withdrawn, and prosocial behaviors. Dev Psychol 32: 1008–1024, 1996.
61. GW Ladd, KB Burgess. Do relational risks and protective factors moderate the linkages between childhood aggression and early psychological and school adjustment? Child Dev 72(5):1579–601, 2001.
62. DAG Drabick, Z Strassberg, MR Kees. Measuring qualitative aspects of preschool boys' noncompliance: The Response Style Questionnaire (RSQ). J Abnorm Child Psychol 29(2):129–139, 2001.
63. L Behar, S Stringfield. A behavior rating scale for the preschool child. Dev Psychol 10(5):601–610, 1974.
64. M Rutter. A children's behavior questionnaire for completion by teachers: preliminary findings. J Child Psychol Psychiatry 8:1–11, 1967.
65. R McGee, S Williams, J Bradshaw, JL Chapel, A Robins, PA Silva. The Rutter scale for completion by teachers: factor structure and relationships with cognitive abilities and family adversity for a sample of New Zealand children. J Child Psychol Psychiatry, 26(5):727–39, 1985.
66. RE Tremblay, F Vitaro, C Gagnon, C Piché, N Royer. A prosocial scale for the

Preschool Behavior Questionnaire: concurrent and predictive correlates. Int J Behav Dev 15:227–245, 1992.
67. JR Séguin, L Arseneault, B Boulerice, PW Harden, RE Tremblay. Response perseveration in adolescent boys with stable and unstable histories of physical aggression: the role of underlying processes. J Child Psychol Psychiatry 43(4):481–494, 2002.
68. F Vitaro, M Brendgen, RE Tremblay. Reactively and proactively aggressive children: antecedent and subsequent characteristics. J Child Psychol Psychiatry 43(4): 495–505, 2002.
69. JM Halperin, KE McKay, JH Newcorn. Development, reliability and validity of the children's aggression scale-parent version. J Am Acad Child Adolesc Psychiatry 41(3):245–252, 2002.
70. K McKay, JM Halperin, R Grayson, S Hall, N Peracchio, JH Newcorn. The children's aggression scale: parent and teacher versions. Sci Proc Annu Meeting Am Acad Child Adolesc Psychiatry 9, 1993.
71. SC Yudofsky, JM Silver, W Jackson, J Endicott, D Williams. The Overt Aggression Scale for the objective rating of verbal and physical aggression. Am J Psychiatry 143:35–39, 1986.
72. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. Washington: Author, 1994.
73. JM Silver, SC Yudofsky. Documentation of aggression in the assessment of the violent patient. Psychiatr Ann 17(6):375–384, 1987.
74. RP Malone, J Luebbert, M Pena-Ariet, K Biesecker, MA Delaney. (1994). The Overt Aggression Scale in a study of lithium in aggressive conduct disorder. Psychopharmacol Bull 30:215–218.
75. RP Malone, MA Delaney, JF Luebbert, J Cater, M Campbell. A double-blind placebo-controlled study of lithium in hospitalized aggressive children and adolescents with conduct disorder. Arch Gen Psychiatry 57(7):649–654, 2000.
76. RP Malone, JF Luebbert, MA Delaney, KA Biesecker, BL Blaney, AB Rowan, M Campbell. Nonpharmacological response in hospitalized children with conduct disorder. J Am Acad Child Adolesc Psychiatry 36(2):242–247, 1997.
77. P Sorgi, J Ratey, D Knoedler, R Markert, M Reichman. Rating aggression in the clinical setting: a retrospective adaptation of the overt aggression scale. Preliminary results. J Neuropsychiatry Clin Neurosci 3:S52–S56, 1991.
78. JN Constantino, M Liberman, M Kincaid. Effects of serotonin reuptake inhibitors on aggressive behavior in psychiatrically hospitalized adolescents: results of an open trial. J Child Adolesc Psychopharmacol 7(1):31–44, 1997.
79. D Schwartz, L Chang, JM Farver. Correlates of victimization in Chinese children's peer groups. Dev Psychol 37(4):520–532, 2001.
80. JA Hubbard. Emotion expression processes in children's peer interaction: the role of peer rejection, aggression, and gender. Child Dev 72(5):1426–1438, 2001.
81. K McBurnett, BB Lahey, PJ Rathouz, R Loeber. Low salivary cortisol and persistent aggression in boys referred for disruptive behavior. Arch Gen Psychiatry 57(1):38–43, 2000.
82. LR Huesmann, LD Eron, NG Guerra, VB Crawshaw. Measuring children's aggres-

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sion with teachers' predictions of peer nominations. Psychol Assess 6(4):329–336, 1994.
83. H Abikoff, M Courtney, WE Pelham, HS Koplewicz. Teachers' ratings of disruptive behaviors: the influence of halo effects. J Abnorm Child Psychol 21(5):519–533, 1993.
84. PJ Frick. Laboratory and performance-based measures of childhood disorders: introduction to the special section. J Clin Child Psychol 29(4):475–478, 2000.
85. PJ Frick, BR Loney. The use of laboratory and performance-based measures in the assessment of children and adolescents with conduct disorders. J Clin Child Psychol 29(4):540–554, 2000.
86. DR Cherek. Effects of smoking different doses of nicotine on human aggressive behavior. Psychopharmacology 75:339–349, 1981.
87. DB Huang, DR Cherek, SD Lane. Laboratory measurement of aggression in high school age athletes: provocation in a nonsporting context. Psychol Rep 85:1251–1262, 1999.
88. CD Casat, DA Pearson, MJ Van-Davelaar, DR Cherek. Methylphenidate effects on a laboratory aggression measure in children with ADHD. Psychopharmacol Bull 31(2):353–356, 1995.
89. DR Cherek, W Schnapp, FG Moeller, DM Dougherty. Laboratory measures of aggressive responding in male parolees with violent and nonviolent histories. Aggress Behav 22:27–36, 1996.
90. DR Cherek, FG Moeller, W Schnapp, DM Dougherty. Studies of violent and nonviolent male parolees. I. Laboratory and psychometric measurements of aggression. Biol Psychiatry 41:514–522, 1997.
91. MS Atkins, DM Stoff, ML Osborne, K Brown. Distinguishing instrumental and hostile aggression: does it make a difference? J Abnorm Child Psychol 21:355–365, 1993.
92. KA Dodge, RR Murphy, K Buchsbaum. The assessment of intention-cue detection skills in children: implications for developmental psychopathology. Child Dev 55: 163–173, 1984.
93. D Schwartz, KA Dodge, JD Coie, JA Hubbard, AH Cillessen, EA Lemerise, H Bateman. Social-cognitive and behavioral correlates of aggression and victimization in boys' play groups. J Abnorm Child Psychol 26(6):431–440, 1998.
94. JE Lochman, KA Dodge. Social-cognitive processes of severely violent, moderately aggressive, and nonaggressive boys. J Consult Clin Psychol 62(2):366–374, 1994.
95. KA Dodge, JM Price, J Bachorowski, JP Newman. Hostile attributional biases in severely aggressive adolescents. J Abnorm Psychol 99(4):385–392, 1990.
96. P Juujärvi, L Kooistra, J Kaartinen, L Pulkkinen. An aggression machine v. determinants in reactive aggression revisited. Aggress Behav 27(6):430–445, 2001.

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16

Psychosocial Interventions

Anger Disorders

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INTRODUCTION

Dysfunctional anger is an issue faced frequently by clinicians. Diagnostically, however, clinicians are in a conundrum. As noted in Chapter 7, there are no anger-based diagnostic categories to guide their assessment, conceptualization, and treatment planning. Anger-based diagnoses would facilitate not only clinical intervention, but also clinical science because protocols and findings would be linked to a common set of criteria describing patient populations. Since such diagnoses are not available, research on anger reduction has tended to define an anger-involved population (e.g., angry medical patients with hypertension, angry veterans, angry drivers) and then evaluate interventions designed for that population. Until agreed-upon anger-based diagnoses are available, researchers should continue to carefully describe demographics, background factors, how anger is experienced and expressed, how anger affects patients and others around them, and other psychosocial correlates and risk factors. Such information will help researchers understand more clearly the nature of the patient population, and will

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assist practitioners in making informed choices regarding what interventions may be appropriate for which angry patients.

Lacking research participants defined by common diagnostic criteria, what is the target of anger reduction interventions? Conceptually, dysfunctional anger is a syndrome (1) comprising emotional experience (e.g., feeling furious), physiological arousal (e.g., short, rapid breathing; hot sensations across face; clenched jaw), and cognitive processes (e.g., hostile attributions, images of revenge and retaliation, inflammatory labeling), which are distressing to the individual and/or lead to significant adverse consequences (e.g., damaged relationships, impaired work performance, legal involvement). Dysfunctional anger also may be related to dysfunctional responding when angry (e.g., aggression, inappropriate withdrawal, drug involvement), but is separable from such behavior. The way the person responds may require separate therapeutic attention. Dysfunctional anger is elicited by various external and/or internal cues or triggers. Interventions for anger reduction target different aspects of the cognitive-emotional-physiological experience, the relationship of anger to its triggers, and/or behaviors thought to directly lower anger. Interventions reviewed in this chapter will focus on interventions for adults and will not include those that focus primarily on dysfunctional behavior such as intimate partner violence as these are covered in other chapters.

Conclusions regarding the status of psychosocial interventions for anger reduction are no better than the research upon which they are based. Conclusions in this chapter will be based on two sources of data. First, they will be based on findings from four meta-analyses (2–5), some of which calculated effect sizes for specific types of interventions and different types of measures. Second, reference will be made to studies which employed multiple measures of anger and compared the therapeutic protocol to a no treatment, attentional, or placebo control. Case studies and quasi-experimental designs will be cited occasionally, but only as examples of suggestive interventions with different populations, rather than evidence of empirical support for the intervention. Absence of adequate controls limits their value as empirical support for the treatment, but they provide useful hypotheses about interventions for different groups.

GENERAL STATUS OF PSYCHOSOCIAL INTERVENTIONS FOR ANGER REDUCTION

At the most general level, research findings support the effectiveness of psychotherapy for anger reduction. However, this conclusion is too broad and overinclusive. Although there are some notable exceptions such as anger-focused, process-oriented group therapy derived from Yalom's model (6), most research is based on a cognitive-behaviorial model. Meta-analyses show that cognitive-behavioral treatment effects differ significantly from 0.0, documenting positive effects for

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cognitive-behavioral interventions, and that the average cognitive-behaviorally treated patient fared better than 76% of control subjects (2). Meta-analyses report considerable variability in effects with effect sizes ranging from 0.0 to 2.9 with the average effect size in the 0.7–1.2 range for anger measures and slightly lower for measures assessing constructs other than anger. Some studies employed other measures of effect size such as eta square which have revealed moderate to large effects for anger reduction interventions, as defined by Cohen's criteria (7) for effect size. Other research employed a rather stringent criterion for clinically significant change, namely Jacobson and Truax's (8) reliable clinical change index. To meet this criterion, a patient's postintervention score must be at least 2 standard deviations below the pretreatment mean for all participants and at least 1.96 standard error of measurement below his/her own pretreatment score. Approximately 40–50%, occasionally more, of cognitive-behaviorally treated participants met this criterion, whereas 0–5% of controls did so. Taken together, these research findings suggest at least moderate empirical support for cognitive-behavioral interventions, but other types of interventions await sufficient data from which to draw tentative conclusions.

STATUS OF SPECIFIC INTERVENTIONS FOR ANGER REDUCTION

There are several promising cognitive-behavioral approaches. However, there simply are too few studies with specific patient populations to break them down by patient group. Findings will therefore be collapsed across patient groups and summarized by intervention modality. For each intervention, the target, logic, and general clinical procedures will be outlined, followed by a review of the empirical support and range of applications. Conclusions regarding absolute treatment effectiveness will be based on meta-analyses and studies employing a no treatment control, a placebo, attentional control, or some other minimal intervention such as a self-monitoring condition. Establishment of absolute effectiveness will be followed by a discussion of relative effectiveness (i.e., whether one intervention is superior to others based on comparison of two or more interventions).

Relaxation Interventions

Relaxation protocols target emotional and physiological elements of the anger syndrome. The logic is that if patients reliably applied relaxation when angered, they would be able to calm down, face provocation and frustration, and access calmer thinking, problem solving, assertion, conflict management, and other skills they posses with which to address sources of anger.

Although specific procedures vary somewhat, they tend to involve five general steps. First, patients are taught a basic relaxation response. Although proce-

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dures such as biofeedback may be used, progressive relaxation is the most common mode of developing relaxation. Second, patients are trained in brief methods for triggering relaxation (e.g., relaxation without tension, relaxation imagery, breathing-cued relaxation, cue-controlled relaxation, etc.). Third, they are trained in becoming more aware of the situations that elicit anger and the ways in which they respond. For example, they may keep self-monitoring logs in which they record situations that elicit anger and their emotional, physiological, cognitive, and behavioral responses. They may be trained to pay more attention to areas of tension during relaxation training, as these often correspond to areas of physical tension during anger arousal. They are asked to attend the nature of anger aroused during relaxation application training. The goal of this enhanced awareness is for the patient to be able to apply relaxation whenever and wherever he/she notices anger arousal. Fourth, patients are trained in application of relaxation for anger reduction within therapy. For example, patients might visualize a situation which previously elicited anger, experience anger for 30–60 sec, and then initiate relaxation to lower anger arousal. Initially, the anger-arousing capacity of scenes is moderate, and the level of therapist assistance in relaxation retrieval high. As patients gain skill and success in relaxation retrieval across sessions, therapist assistance is reduced and level of the anger arousal increased. Roleplays and simulations may also be used as part of training. A provocative situation is enacted with the patient signaling the presence of anger and practicing relaxation to lower anger during the enactment. Such procedures provide in-session management of anger arousal. The final step involves transfer of relaxation skills to the external environment. Early in therapy patients might practice relaxation in nonangering situations (e.g., while watching television or riding on a bus). This provides practice applying relaxation in vivo under low stress conditions. As patients gain in-session experience in applying relaxation for anger reduction, assignments are given for application of relaxation to any angering situation and/or application in specific contracted situations (e.g., a difficult interaction with a spouse or coworker). Interventions such as lengthening the interval between final sessions, continued self-monitoring of applications sent to the therapist, and planned booster sessions may be employed to extend and strengthen relaxation skills for in vivo application.

Novaco's landmark study (9) comparing relaxation to cognitive, cognitive-relaxation, and attentional control conditions demonstrated relatively weak effects for relaxation compared to cognitive and cognitive-relaxation conditions. However, subsequent research (e.g., 10–17) reveals greater effects for relaxation interventions, effects that were generally as strong as other cognitive-behavioral interventions. Meta-analyses report relaxation effect sizes in the 0.8–1.2 range. Similar effect sizes have been found for emotional and physiological measures, suggesting that relaxation interventions impact domains to which they are theoretically targeted. In general, effects for relaxation are as strong as those for other

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interventions, and long-term follow-up studies (e.g., 11,13,15,16) showed maintenance of relaxation effects over time. Additionally, studies that assess effects other than anger reduction (e.g., trait anxiety) reveal at least modest transfer effects to other problem areas (e.g., 11,13,15,16,18). Relaxation interventions have been applied to a wide range of anger-involved individuals such as generally angry college students (11,13-15), angry community samples (9), incarcerated individuals (17), angry drivers (12,16,18,19), and individuals with elevated blood pressure (10,20,21) and chronic heart disease (22). In summary, relaxation is effective for anger reduction in a variety of populations, and effects appear to maintain over time and show at least modest transfer to other issues.

Cognitive Interventions

Cognitive interventions target anger-engendering information processing. These include hostile appraisals and attributions, angry self-dialogue, ineffective problem solving, rigid expectations and demands, overgeneralized and catastrophic thinking, aggression supportive expectancies and attitudes, and the like. The logic is that if individuals person can recode events, themselves, and their coping capacities in more realistic, less demanding, more benign ways, and if they can cognitively guide themselves through provocative situations in more calm, task-oriented ways, intense anger will not be elicited, and they will be able to cope more effectively. That is, as events are construed in less anger-engendering ways and patients can self-instruct through situations in less angry ways, anger is lowered, and they are less likely to react impulsively and aggressively as their access competencies typically associated with calmer thinking, feeling, and reflection.

Cognitive protocols vary in therapeutic format, but retain features in common. Like relaxation interventions, cognitive patients often engage in activities that make them more aware of their anger and the external and internal conditions (e.g., rumination or negative feelings) that trigger anger. Cognitively treated patients are also likely to focus on images, self-dialogue, and memory fragments that are part of their experience as these are often targets of change. As they become more aware of their cognitive processes, patients explore patterns in and validity of anger-engendering cognitions. For example, they might assess the validity of dichotomous thinking (e.g., I am strong or a wimp) or overinclusive labeling (e.g., they all hate me or have it in for me). They might be asked for alternative explanations or ways of thinking about an event or for evidence supporting their beliefs and attributions. They might be asked to define their terms and see the silly humor in them (e.g., generating the image of a retarded burro to define "dumb ass"). They might be asked to interview several people for their interpretations of an event to generate alternative perspectives to juxtapose to the patient's view. Whatever the intervention, the goals are to open automatic information processing to inspection and review and to generate alternative ways

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of thinking about and approaching frustrating or provocative events. Insight and understanding, however, are rarely sufficient. Patients are assisted in developing alternative, less hostile self-dialogue, imagery, self-guidance, and problem-solving strategies. These are rehearsed within sessions, perhaps during exposure to angry imagery, and then transferred to and maintained in the external environment through strategies like those outlined for relaxation interventions.

Novaco's component analyses of stress inoculation training demonstrated that the self-instructional, cognitive component was effective for anger reduction (9). Subsequent studies have supported this conclusion. In this cognitive intervention, patients identify anger-engendering engendering imagery and self-dialogue, and develop and rehearse alternative internal dialogue to guide themselves through angering events. The content of altered self-dialogue varies from patient to patient, but likely includes things such as rational reappraisal of provocative events, decatastrophizing, framing things in terms of personal desires and preferences rather than rigid rules and demands, challenging of highly negative or threatening attributions, coping self-statements, task-oriented problem-solving, and self-reinforcement of gains. Generally, new self-instructions are rehearsed within sessions (e.g., in response to anger imagery or roleplays), and the rehearsal format may follow stages of coping (i.e., preparing for a provocation, coping with a provocation, coping with overwhelming feelings of anger, and dealing with the aftermath of provocation). When anger reduction is achieved within sessions, homework and other assignments focus on transfer and maintenance. This intervention has achieved effect sizes of ~ 1.0 on self-report anger measures and slightly lower on other measures. The few studies that have included generalization measures and long-term follow-up have nontargeted effects and long-term anger reduction (15,23). Self-instructional training has shown positive effects with generally angry college students (14,15,23,24), angry community volunteers (9), incarcerated individuals (25), juvenile offenders (26), and patients with elevated blood pressure (10), suggesting a range of application as well.

There are other, less well researched cognitive interventions. A promising intervention is an adaptation of rational-emotive therapy (27–29). Angry community volunteers experienced prolonged exposure to hostile, denigrating, disparaging comments made to and about the patient and rehearsed self-dialogue based on rational-emotive therapy. The rational-emotive therapy condition was effective compared to no treatment control and equal to or slightly superior to exposure alone, or rehearsal of neutral responses. Another cognitive intervention is the cognitive component of Aaron Beck's cognitive therapy (2). Although it may seem odd to refer to the cognitive component of a therapy that is specifically labeled cognitive, Beck's cognitive therapy has a strong behavioral as well as cognitive focus (e.g., Socratic exploration and behavioral experiments focus on alternative behavior as well as cognitive change). A partial component analysis of Beck's cognitive therapy with generally angry college students showed the cognitive com-

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ponent alone was as effective as the full protocol, and both were more effective than an untreated control (30). Effect sizes were moderate to large, and 47% of clients in the cognitive only condition met the reliable clinical change index, compared to 0% of controls. The intervention also lowered a cognitive measure of anger, suggesting it influenced the theoretical target of change. A final promising cognitive intervention is problem-solving training (24). This approach assumes angry individuals have anger-related problem-solving deficits. Clients are taught specific steps of problem solving, which are rehearsed in relation to angering events. This intervention was effective compared to an untreated control, generally as effective as other interventions, and had effect sizes ranging from 0.3 to 1.3.

In summary, cognitive interventions are effective for anger reduction in a range of populations such as generally angry college students and community samples, incarcerated individuals, and individuals with medical problems such as hypertension.

Social/Communication Skills Interventions

Most anger is experienced in an interpersonal context. Individuals with problematic anger tend to rush to judgment, be abrasive and abrupt in their communication, and react impulsively and antagonistically toward others. Anger and interpersonal conflict escalate as the person reacts in such a manner and expresses him/herself in less controlled or constructive ways. Social-skills interventions target these interpersonal communication styles and behaviors. The logic is that when angry individuals develop and deploy more effective communication and conflict management skills, they communicate effectively and reduce or abort anger as they acquire the skills to prevent escalating cycles of anger.

Not every skill development program focuses on the same skills, but they tend to include becoming aware of the impact of one's behavior on others, basic listening skills such as listening without interruption and paraphrasing to clarify understanding, assertive expression of thoughts, feelings, and preferences; skills in giving positive and negative feedback to others; interpersonal negotiation and compromise; and taking a time out. Protocols typically involve assessment of component skill deficits. One or two behaviors (e.g., talking in a calmer tone, not using profanity, listening without interruption) are specified for rehearsal. The behavior is discussed, modeled, and rehearsed. The patient and therapist then provide feedback regarding the behavior, and that behavior or new behavior is added and rehearsed again. Homework assignments are given to transfer and solidify the behavior in the external world.

Three studies employed these behavioral strategies to increase assertive responding in the face of provocation in angry college students (24,31,32). Positive effects were found for anger reduction with effect sizes of 0.9 – 1.2 and for altered behaviors such as increased empathy and appropriate requests and de-

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creased inappropriate behavior such as profanity and making of threats with effect sizes ranging from 0.6 to 2.9. Somewhat similar training procedures were employed to increase listening and feedback skills and interpersonal assertion and negotiation skills in generally angry college students (33,34). These studies showed somewhat similar effect sizes for anger reduction (0.3 – 1.3 effect sizes) and for forms of anger expression and social skills (0.4 – 1.3 effect sizes). Modest treatment effects (effect sizes of 0.3 - 0.5) were also evidenced for trait anxiety reduction, and anger and anxiety reduction were maintained in long-term follow-ups (35,36). An interpersonal skill enhancement program also lowered outward negative expression of anger and increased controlled expression of anger in juvenile offenders (26). Another skill-based program trained adolescents and their parents in listening and conflict management skills also increased communication and problem-solving skills and lowered conflict (37). Social/communication skills programs reviewed thus far tend to be relatively structured with the therapist introducing sequentially the skills to be addressed. An alternative format is to follow a more Socratic, inductive style in which therapists encourage clients to identify effective communication for conflict (34,38). Patient-identified behaviors are then refined and rehearsed during visualizations or roleplays, and behavioral tryouts are negotiated to extend communication strategies externally. Compared to a no-treatment condition, this patient-centered social skills training format lowered anger, increased controlled, socially appropriate forms of anger expression, led to reductions of trait anxiety, and revealed maintenance in long-term follow-ups (34,36,38).

In summary, social/communication skills interventions appear effective for anger reduction and for skill enhancement. Such interventions were effective with angry college students, incarcerated individuals, and angry, conflicted parents and adolescents, again suggesting a possible range of effects in addressing anger in interpersonal contexts.

Multicomponent Interventions

Some treatment protocols integrate the logic and procedures of two or more interventions and target multiple parts of the anger syndrome. For example, a cognitive-relaxation intervention simultaneously targets the cognitive, emotional, and physiological elements of anger. This intervention has perhaps the greatest number of studies supporting its effectiveness. Novaco's study (9) documented the effectiveness of the cognitive-relaxation condition compared to an attentional control. Studies with generally angry college students (6,13–15,23,33,34,38) showed that the cognitive-relaxation condition lowered anger with average effect sizes of 1.0 for measures of general anger and 0.8 for other measures. Several studies employed the clinically reliable change index and showed that 40–50% of cognitive-relaxation clients met this criterion, whereas 0–5% of untreated con-

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trols did. Long-term follow-ups (6,13,15,23,35,36) revealed long-term maintenance of anger reduction and short- and long-term anxiety reduction (39). Recently, a series of studies with angry drivers (12,16,18,19) reported anger reduction for the cognitive-relaxation condition. Reduction of driving anger was also maintained at long-term follow-up (16). Gerina and Drummond (40) adapted the cognitive-relaxation intervention for anger reduction in police officers. Anger and anxiety reduction were reported by the officers, and their fellow officers, who were unaware of treatment, also reported that officers receiving treatment were significantly less angry. Thus, a combination of cognitive and relaxation interventions appears effective for anger reduction.

Another combined focus is on the cognitive and skill aspects of anger. As noted earlier, Beck's cognitive therapy has such a focus. Two studies with generally angry college students showed Beck's cognitive therapy lowered anger and outward negative anger expression and improved controlled anger expression (30,41). Treatment effect sizes (eta square) were moderate to large, and 40–70% of cognitive therapy clients met the reliable clinical change criterion, whereas none of the controls did. One study (41) revealed reduction of trait anger, whereas the other (30) did not. Treatment effects were maintained through 1-month (30) and 15-month (41) follow-up. Kogan et al. (19) also showed that cognitive therapy lowered anger and aggression on the road for angry drivers. Whiteman and colleagues (42) demonstrated cognitive therapy effective in reducing anger in parents at risk for child abuse.

Still other interventions have focused on cognitive, emotional, physiological, and skill elements of anger. Such cognitive-relaxation-skill approaches lowered anger in generally angry college students (43) and Type A community samples (44). Long-term follow-ups revealed maintenance of treatment gains (43,44). Chemtob et al. (46) implemented such a program with angry veterans suffering Posttraumatic stress disorder (PTSD) and found short- and long-term anger reduction compared to veterans receiving standard psychological and medical treatment. An adaptation of this kind of program to angry, retarded individuals revealed reduced anger, aggression, and depression compared to a control, and 6- and 12-month follow-ups revealed maintenance of anger reduction (47). Another combined intervention program lowered anger in adolescent inpatients with poor anger control (48). When a parent-adolescent conflict management skill program was combined with anger management, it was found to be as effective as the conflict management program at lowering conflict and improving communication, but led to greater anger reduction (37). Finally, multicomponent programs also have been effective with medically involved patients. For example, a combined intervention was effective in lowering hostility and improving constructive verbal expression of anger, which in turn was related to a decrease in resting blood pressure (49). Somewhat similar effects with hypertensive individuals were achieved with another multicomponent anger reduction program (50).

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In summary, interventions that target multiple aspects of anger arousal are effective, and there is some evidence of long-term maintenance of anger reduction and generalization to other areas of the patient's life. Moreover, combined interventions are effective with a range of anger-involved clients, suggesting considerable applicability and flexibility as well.

RELATIVE EFFECTIVENESS OF INTERVENTIONS FOR ANGER REDUCTION

There are several promising interventions for anger reduction, but little evidence that any one is superior to others. Three sources of information converge to support this conclusion.

First, there is a box score count of direct statistical comparisons between active treatments. A few studies have shown some differences. For example, Novaco (9) found that although cognitive-relaxation and cognitive-only conditions tended not to differ, they were superior to relaxation on some measures. Deffenbacher et al. (34) found greater effects for the inductive, client-developed social skills intervention on one measure. Deffenbacher et al. (12) reported that relaxation intervention led to greater anger reduction on some measures, but the cognitive-relaxation measure was superior at reducing risky behavior. Moon and Eisler (24) reported differences between cognitive restructuring, problem-solving, and social-skill interventions on some measures. Finally, Stern (37) reported greater anger reduction for an intervention combining anger and conflict management than the conflict management intervention alone.

Such statistical differences could be taken as support of differences between interventions; however, they should not be overgeneralized and taken out of context. Within these studies, treatments did not differ on most measures, which cautions against highlighting a few statistically significant differences and ignoring or minimizing the many equivalent outcomes. Moreover, the vast majority of studies do not show any between-intervention differences at all; that is, in most studies intervention effects were found when active treatments were compared to control conditions, but failed to show differences between treatments. Thus, the overall box score count does not favor between-treatment effects.

Finding differential treatment effects, however, has been limited by the sample sizes involved in most studies. Many employed samples of 10–25 participants per condition. If interventions compared are moderately effective, the statistical power needed to discriminate between-condition differences is relatively low. Large sample sizes address this problem, but efforts must be placed in to context of the history of clinical science. Anger reduction research is in its infancy compared to more established areas such the treating anxiety or depression. The resources and logistics needed to recruit, assess, treat, and retain angry individuals were simply not present for the larger samples needed for the documentation of

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potential differences between active treatments. Were the constraints of sample size imposed, many studies may not have been undertaken, and anger reduction research would have lost important information about the absolute effectiveness of interventions. Nonetheless, statistical power must be acknowledged, and differences between treatments may be discovered as the field moves to multisite collaborative studies in the future.

Second, meta-analyses address some of the problems of a box score analysis. Some meta-analyses calculated effect sizes for different interventions. Most treatments have approximately the same average effect size, suggesting that they are roughly equivalent in effects. However, tempting as it is, care should be taken in relying too much on such comparisons. First, measures across studies may have quite different measurement characteristics that introduce a source of measurement confounding, which may artificially inflate or deflate effect sizes and could influence conclusions drawn from the meta-analysis. Second, meta-analytic comparisons are often based on relatively small number of studies of any modality. Studies may not be comparable in other ways that could influence conclusions from the meta-analyses.

Third, there is a lack of replication of between-group differences across studies. Simply, several studies suggesting differences between conditions have not replicated. For example, Novaco (9) reported relatively weak effects for relaxation compared to cognitive and cognitive-relaxation conditions. A subsequent study by Deffenbacher et al. (11) reported stronger effects for relaxation. Several subsequent studies also found greater effects for relaxation and showed relaxation to be as effective as cognitive (14,15), cognitive-relaxation (13,14,16–18), and cognitive-behavioral (19) interventions. For example, differences favoring relaxation or cognitive-relaxation conditions for driving anger reduction (12) were not replicated in subsequent studies (16,18). The difference favoring client-developed social skills training (34) was not found in another evaluation of the two skill-training formats (38). Quite simply, if there are clinically meaningful differences between interventions, they should stand the test of time and be replicated in other studies. However, this has not been the case. Differences found in one study tend not to be replicated, suggesting they are not reliable, substantive differences between interventions.

In summary, the anger reduction research is young. There is an accumulating research literature documenting positive outcomes for several interventions, but there are few replicable data suggesting a gold standard, that one intervention is superior to others. Rather than despair over the lack of between-intervention differences and conclude that it does not make any difference which intervention is employed, it is suggested that a more appropriate approach is to conclude that several interventions have empirical support and that findings should be used intelligently to match them to critical elements of anger in specific populations and guide the treatment planning for that population.

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CONSIDERATIONS IN PROTOCOL DESIGN

Although research on anger reduction is far from being able to answer which intervention is most effective for which individual with which kind of anger issue and constellation of background factors, it can offer several empirically anchored themes to inform intervention design.

Carefully Tailor Protocol Design to the Characteristics of the Angry Population

The first level of individualization is targeting the intervention to the problematic aspects of the patient's anger. The triggers of anger and the salient aspects of the experience and expression of anger should be carefully mapped. Then, empirically supported treatments should be integrated into a protocol targeting those elements. For example, interventions for young angry drivers, middle-aged patients with anger-related hypertension, and parents with explosive tempers with their children may be appropriately dissimilar, because the triggers, experience, and expression are markedly different.

The nature of anger and anger expression is important, but these are not the only characteristics that should be considered. Problematic anger exists with a context of personal attributes, social systems, and cultural contexts that may support or interfere with anger management. These too should be taken into account if interventions are to make sense and appeal to patients. For example, anger reduction in family systems should identify and incorporate the religious, family, and cultural values in which anger is embedded. For example, interventions for individuals who are mandated to attend should address the anger and resentment stemming from being mandated, plus any individual and culturally sanctioned attitudes supportive of anger and aggression. Inattention to such matters may compromise or lessen treatment effectiveness.

More Is Not Necessarily Better

There are several empirically supported interventions, and clinicians and researchers may be tempted to include several different intervention strategies, hoping to maximize treatment effects. However, this may be unwise. First, there is little empirical support for making this choice. In general, interventions with a singular focus fared as well as interventions that combine several intervention components. Admittedly, research designs have not been employed which would disentangle the confounding of therapeutic exposure and a true implementation of all treatment components in combined interventions, but there is little evidence to date of synergy and greater effects in more complex programs. Second, at least in some populations, a treatment program with fewer components may be easier for patients to understand and implement. Having a singule focus may also

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provide patients with greater opportunities for overlearning and practice, which may increase their sense of self-efficacy faster. Third, if an intervention is time limited, multicomponent programs may inadvertently exclude important activities. Adding more treatment components typically means that time for personalizing applications and in-session rehearsal are reduced, if not eliminated.

However, this rehearsal and personalization may be critically important aspects of treatment for angry patients, and adding treatment components may eliminate key ingredients. If multiple components are to be employed (and there may be good reasons to do so), the protocol should be developed so that adequate time and therapeutic attention are devoted to all components so that they have a chance of being effective. For example, session number and/or length might be increased to include all treatment components. Number of sessions is also often important in this context so that patients have time to practice and implement different treatment strategies. Finally, not all angry patients, even those with similar presenting problems, need all treatment components. For example, two highly angry, verbally abusive parents might present for treatment. An intervention including cognitive and relaxation strategies for anger reduction, information and discussion of developmentally appropriate expectations, and behavioral rehearsal of effective parenting skills might be appropriate. However, as one of the patients learned to manage his/her anger, no further treatment might be necessary. As he/she lowered anger and was able to think more calmly, it might be found that he/she possessed adequate parenting and conflict management skills that were not accessed when highly angered. The other patient might need the additional components to address continuing parenting skill deficits and aggressive behavior. Attention to such possible patient differences should be addressed in assessment activities and in the sequencing of intervention components in multicomponent protocols.

More May Be Necessary

This suggestion may seem exactly counter to the prior suggestion, but it is not. Care must be taken not to overgeneralize findings from one population to another. For example, it may be tempting to generalize an eight-session protocol effective with angry college students to other groups. The intervention may not be effective or only somewhat effective when implemented with another population. For example, while some components may be effective, patients with intermittent explosive disorder or patients who are highly aggressive, court-mandated drivers may need additional treatment components and/or greater time spent in rehearsal and transfer activities. Admittedly, there may be legal, managed care, or other time constraints, but therapists and researchers should be realistic about what can be achieved in a given amount of time with a given population. Sometimes more is necessary to achieve desired outcomes, and necessary time and resources

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should be devoted to these activities. Time and energy may be needed to be expended in educating and setting realistic expectations in various external constituencies, or interventions may prove less than fully effective, which ultimately benefits neither clinical research, patients, or social systems in search of anger reduction.

A corollary of this point is to make sure that the overall protocol targets all of the important elements of angry patients' problems. Not all aspects of the anger syndrome are highly correlated and therefore necessarily responsive to the same intervention. In many cases, anger management strategies may be helpful but insufficient. For example, anger management may have a place as one part of an overall treatment for angry, impulsive aggressive individuals. However, treatment should also target the impulsive aggressive behavior specifically. As another example, interventions for driving anger have not reliably altered risky behavior on the road, even though risky behavior is correlated with anger and aggression behind the wheel (19). Protocols should design interventions to address reducing risky behavior directly. Anger management strategies may be of considerable value in the overall treatment planning, but should be integrated with other interventions if treatment is to be effective.

Consider a Group

Although there may be some legal or therapeutic reasons not to do so, if there is a group of angry individuals, then consider a group format. Many of the empirically supported anger reduction interventions have been delivered in groups. Although there has not been sufficient research comparing individual and group delivery of the same protocol, there is evidence that groups work. Second, groups are more efficient and perhaps more cost-effective than individual therapy with the same intervention. Third, groups can have some added benefits. For example, group members provide a range of alternative cognitions and perspectives that can be incorporated into therapy: they provide a range of positive models and different ways of handling angering situations; they provide a greater range of possibilities and flexibility in roleplays and behavioral rehearsal activities; and they can provide the possibility of partnered external assignments, to name just a few. So, if it fits the treatment environment and population, consider a group. In many cases, mixed-gender groups have been led by a single therapist. In some cases, this can be a well-trained and supervised masters-prepared therapist, as these were the therapists employed in much of the anger reduction research.

Consider a Cognitive-Relaxation Intervention for General Audiences

A cognitive-relaxation intervention may have the greatest utility when treating a group with diverse anger problems or doing an anger management group or

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class. First, the cognitive-relaxation interventions have considerable empirical support with a wide variety of angry individuals. Second, cognitive and relaxation interventions are easily combined and can be rehearsed together to lower anger aroused by visualization of anger scenes. Third, cognitive and relaxation interventions are likely to address some of the individual differences within a diverse audience. That is, relaxation may appeal more to some patients and cognitive interventions more to others, but the single intervention addresses both client preferences. Fourth, relaxation can precede cognitive interventions and reduce resistance that might occur to cognitive interventions alone. Fifth, cognitive-relaxation interventions address anger in both interpersonal and non-interpersonal contexts, whereas social skill interventions address only anger in interpersonal contexts. This can be important in general audiences because from 20% to 25% of anger occurs in an impersonal context (e.g., anger at inanimate objects and equipment, natural events such as the weather, anger at onesself, situations without close interpersonal contact such as driving, delays as in air travel, lines, and unavoidable obstructions). For these reasons, consider a cognitive-relaxation intervention for general anger groups.

Address Potential for Violence

Patient potential for violence should be assessed on an ongoing basis. Legal and other interventions to prevent harm to others should be initiated. However, there is also a concern about violence directed toward the therapist. This is a real concern, and there are documented cases of assault and violence toward the therapist. Where potential is high, adequate precautions and prevention efforts should be implemented. Nevertheless, the anger reduction literature does not suggest that this is a large problem. Many angry individuals have participated with very few reported problems. In fact, in several studies (27–29), the treatment protocol involved the therapist forcefully presenting hostile, personal barbs to the patient. These patients were angry males, many of whom had documented histories of physical assault and altercations, and aggression toward the therapist was a concern. However, no violence toward the therapist was experienced. Many patients even suggested ways of making the barbs even more negative and anger-arousing. Thus, potential violence toward the therapist should be assessed and addressed, but it should not be accepted automatically as a reason for not working with angry patients.

Address Resistance

Angry patients often perceive they are being told that they are the problem, that their thoughts, feelings, and behavior are wrong, and that they, not others, must change. They often react angrily and defensively, discounting therapy and the therapist, resisting change, and in some cases prematurely terminating therapy.

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To address and hopefully overcome this resistance and therapeutic impasses, treatment protocols should consider several things:

1. Therapists should attend to the therapeutic relationship and alliance. Angry patients may be threatening, abrasive, and intimidating, and they may reveal attitudes and behaviors which the therapist does not like or accept. Nevertheless, therapists attempt to listen carefully to and actively communicate an understanding of the patient's losses and perceived injuries, wrongs, and injustices, much as they would listen to an anxious or depressed patient. Angry patients deserve no less respect and attention.
2. The Socratic, collaborative approach of Beck's cognitive therapy should be considered, because it appears to reduce resistance engendered in some more active, directive approaches (15,23).
3. Wherever possible, time should be taken to develop the intervention collaboratively with the rationale and procedures linked repeatedly to the patient's anger issues as necessary.
4. If relaxation interventions are part of the treatment plan, implement relaxation early. Relaxation fits well with many patients' conceptualization of anger problems (i.e., heightened emotional and physiological arousal) and with ways of reducing anger (i.e., by calming down). Relaxation thus strengthens the therapeutic alliance while it does not involve a great deal of therapist challenging at the beginning of therapy. That is, relaxation is not only an effective intervention in its own right, but appears to reduce resistance to some other interventions as well (39).

Address Readiness for Anger Reduction

Readiness for change is an important, often overlooked issue. Anger reduction interventions reviewed in this chapter are action-oriented interventions. These interventions assume a patient who, to a considerable degree, is aware of his/her anger problems and is actively seeking to reduce his/her anger and associated problems. However, many people with anger problems, no matter how others see them, are not aware of their anger problems and/or do not accept that they have a problem for which they should seek change. They may be sent to or brought to therapy by others (e.g., employers, courts, schools, spouses), but they do not truly see themselves as having a problem. For example, they may totally externalize their anger. That is, if others did not behave as they did or do the things they did, then the person would not react as angrily and aggressively as he/she does. The source of the problem is others, not the person experiencing anger.

Such individuals are denying and minimizing anger problems and are at the precontemplative or perhaps contemplative stages of change. They are not ready for or accepting of action-oriented anger reduction interventions, and proceeding with such interventions may be doomed to failure or at the very least

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fraught with many therapeutic false starts and impasses. For example, in their research with angry drivers, Deffenbacher and colleagues (12) noticed a group of high-anger drivers who were not interested in therapy. Subsequent research showed this group to be at as much risk as the high-anger drivers who saw their anger as a personal problem and sought counseling for it. Although this group of high-anger drivers was offered treatment free of charge, they were not interested. These interventions were irrelevant to them, and they were not at a stage of change to accept or take advantage of counseling. Quite simply, if anger reduction interventions are to be successful, they must start where the patient is. Readiness for change should be assessed and interventions should be developed which address patients at the precontemplative and contemplative stages of change. Such interventions would probably focus on an increased awareness of the person's anger and the personal consequences of anger, and enhance motivation for change. If successful, then action-oriented anger reduction strategies may become relevant, but not likely until then.

CONCLUSIONS AND FUTURE DIRECTIONS

Although outcome research on anger reduction has lagged far behind other areas, there are some promising interventions. It is suggested that clinical researchers use this information intelligently to adapt and tailor interventions to their anger-involved population. Small clinical trials can be conducted from which to assess initial effects and to make modifications to enhance treatment involvement and outcome. These trials provide the basis from which to conduct larger comparative trials and establish the most effective interventions. In conjunction with these efforts, it is suggested that clinical researchers address readiness for and resistance to change and develop and evaluate interventions to address these important issues so that patients can become involved in and benefit more from efforts to lower their anger. Finally, the issue of maintenance and relapse prevention should receive greater attention now that there are some effective interventions. Long-term follow-up studies are necessary to assess maintenance generally and specifically for these strategies for maintaining and improving outcomes over time.

REFERENCES

1. Averill, J. R. (1982). Anger and Aggression: An Essay on Emotion. New York: Springer-Verlag.
2. Beck, R., Fernandez, E. (1998). Cognitive-behavioral therapy in the treatment of anger: a meta-analysis. Cogn Ther Res, 22, 63–74.
3. Edmondson, C. B., Conger, J. C. (1996). A review of treatment efficacy for individuals with anger problems: conceptual, assessment, and methodological issues. Clin Psychol Rev, 10, 251–275.

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4. Tafrate, R. C. (1995). Evaluation of treatment strategies for adult anger disorders. In H. Kassinove (Ed.), Anger Disorders: Definition, Diagnosis, and Treatment (pp 109–130). Washington: Taylor and Francis.
5. Tafrate, R. C., DiGiuseppe, R. (2000). A meta-analysis of anger reduction interventions. (Unpublished.)
6. Deffenbacher, J. L., McNamara, K., Stark, R. S., Sabadell, P. M. (1990). A comparison of cognitive-behavioral and process oriented group counseling for general anger reduction. J Couns Dev, 69, 167–172.
7. Cohen, J. (1988). Statistical Power Analysis for the Behavioral Sciences. 2nd ed. Hillsdale, NJ: Lawrence Erlbaum.
8. Jacobson, N. S., Truax, P. A. (1991). Clinical significance: a statistical approach to defining meaningful change in psychotherapy research. J Consult Clin Psychol, 59, 12–19.
9. Novaco, R. W. (1975). Anger Control. Lexington, MA: Heath.
10. Achmon, J., Granek, M., Golomb, M., Hart, J. (1989). Behavior treatment of essential hypertension: a comparison between cognitive therapy and biofeedback of heart rate. Psychosom Med, 51, 152–164.
11. Deffenbacher, J. L., Demm, P. M., Brandon, A. D. (1986). High general anger: correlates and treatment. Behav Res Ther, 24, 481–489.
12. Deffenbacher, J. L., Huff, M. E., Lynch, R. S., Oetting, E. R., Salvatore, N. F. (2000). Characteristics and treatment of high anger drivers. J Couns Psychol, 47, 5–17.
13. Deffenbacher, J. L., Stark, R. S. (1992). Relaxation and cognitive-relaxation treatments of general anger. J Couns Psychol, 39, 158–167.
14. Dua, J. K., Swinden, M. L. (1992). Effectiveness of negative-thought-reduction, meditation, and placebo training treatment in reducing anger. Scand J Psychol, 33, 135–146.
15. Hazaleus, S. L., Deffenbacher, J. L. (1986). Relaxation and cognitive treatments of anger. J Consult Clin Psychol, 54, 22–226.
16. Richards, T. L., Deffenbacher, J. L., Filetti, L. B., Lynch, R. S., Kogan, L. (2001). Short- and long-term effects of interventions for driving anger reduction. Presented at the 109th Annual Convention of the American Psychological Association, San Francisco, August 2001.
17. Schlichter, K. J., Horan, J. J. (1981). Effects of stress inoculation on the anger and aggression management skills of institutionalized juvenile delinquents. Cogn Ther Res, 5, 359–365.
18. Deffenbacher, J. L., Filetti, L. B., Lynch, R. S., Dahlen, E. R., Oetting, E. R. (2002). Cognitive-behavioral treatment of high anger drivers. Behav Res Ther, 40, 895–910.
19. Kogan, L. R., Richards, T. L., Deffenbacher, J. L. (2001). Effects of relaxation and cognitive therapy for driving anger reduction. Presented at the 109th Annual Convention of the American Psychological Association, San Francisco, August 2001.
20. Davison, G. C., Williams, M. E., Nezami, E., Bice, T. L., DeQuattro, V. L. (1991). Relaxation, reduction in angry articulated thoughts, and improvements in borderline hypertension and heart rate. J Behav Med, 14, 453–468.
21. Haaga, D. A. F., Davison, G. C., Williams, M. E., Dolezal, S. L., Haleblian, J.,

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Rosenbaum, J., Dwyer, J. H., Baker, S., Nezami, E., DeQuattro, V. (1994). Mode-specific impact of relaxation training for hypertensive men with Type A behavior pattern. Behav Ther, 25, 209–223.

22. Bhat, K. N. (1999). The role of biofeedback-assisted anger control in reversing heart disease. Dissertation abstracts international. Section B. The sciences and engineering, Vol 60(5-B), December 1999, p2326. Ann Arbor, MI: University Microfilms International.
23. Deffenbacher, J. L., Story, D. A., Brandon, A. D., Hogg, J. A., Hazaleus, S. L. (1988). Cognitive and cognitive-relaxation treatments of anger. Cogn Ther Res, 12, 167–184.
24. Moon, J. R., Eisler, R. M. (1983). Anger control: an experimental comparison of three behavioral treatments. Behav Ther, 14, 493–505.
25. Diaz, L. A. (2000). A comparison of cognitive restructuring and systematic desensitization techniques for anger reduction with an inmate population. Dissertation abstracts international. Section B. The sciences and engineering, Vol 61(2-B), August 2000, p1078. Ann Arbor, MI: University Microfilms International.
26. Escamilla, A. G. (2001). Effects of self-instructional cognitive-behavioral techniques on anger management in juveniles. Dissertation abstracts international. Section B. The sciences and engineering, Vol 61(8-A), March 2001, p3117. Ann Arbor, MI: University Microfilms International.
27. Mcvey, M. E. (2000). Exposure and response prevention versus rational self-statements in the treatment of angry men. Dissertation abstracts international. Section B. The sciences and engineering, Vol 61(6-A), January 2000, p2197. Ann Arbor, MI: University Microfilms International.
28. Tafrate, R. C., Kassinove, H. (1998). Anger control in men: barb exposure with rational, irrational, and irrelevant self-statements. J Cogn Psychother, 12, 187–211.
29. Terracciano, S. (2000). Effects of barb exposure and rational statement rehearsal on anger and articulated thoughts in angry married men: extinction or cognitive restructuring. Dissertation abstracts international: Section B: The sciences and engineering, Vol 61(6-B), January 2000, p3294. Ann Arbor, MI: University Microfilms International.
30. Dahlen, E. R., Deffenbacher, J. L. (2000). A partial component analysis of Beck's cognitive therapy for the treatment of general anger. J Cogn Psychother, 14, 77–95.
31. Fehrenbach, P. A., Thelen, M. H. (1981). Assertive-skills training for inappropriately aggressive college males: effects on assertive and aggressive behavior. J Behav Ther Exp Psychiatry, 12, 213–217.
32. Rimm, D. C. Hill, G. A., Brown, N. N., Stuart, J. E. (1974). Group assertive training in treatment of expression of inappropriate anger. Psychol Rep, 34, 791–798.
33. Deffenbacher, J. L., Story, D. A., Stark, R. S., Hogg, J. A., Brandon, A. D. (1987). Cognitive-relaxation and social skills interventions in the treatment of general anger. J Couns Psychol, 34, 171–176.
34. Deffenbacher, J. L., Thwaites, G. A., Wallace, T. L., Oetting, E. R. (1994). Social skills and cognitive-relaxation approaches to general anger reduction. J Couns Psychol, 41, 386–396.

Marcel Dekker, Inc.
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35. Deffenbacher, J. L. (1988). Cognitive-relaxation and social skills treatments of anger: a year later. J Couns Psychol, 35, 234–236.
36. Deffenbacher, J. L., Oetting, E. R., Huff, M. E., Thwaites, G. A. (1995). A fifteen-month followup of social skills and cognitive-relaxation approaches to general anger reduction. J Couns Psychol, 42, 400–405.
37. Stern, S. B. (1999). Anger management in parent-adolescent conflict. Am J Fam Ther, 27, 181–193.
38. Deffenbacher, J. L., Oetting, E. R., Huff, M. F., Cornell, G. R., Dallager, C. J. (1996). Evaluation of two cognitive-behavioral approaches to general anger reduction. Cogn Ther Res, 20, 551–573.
39. Deffenbacher, J. L., Lynch, R. S. (1998). Cognitive/behavioral intervention for anger reduction. In V. E. Caballo (Ed.), Manual para el Tratamiento Cognitivo-Conductual de los Trastornos Psicologicos, Vol 2 (pp 639–674). Madrid: Siglo XXI.
40. Gerina, M. A., Drummond, P. (2000). A multimodal cognitive-behavioural approach to anger reduction in an occupational sample. J Occup Org Psychol, 73, 181–194.
41. Deffenbacher, J. L., Dahlen, E. R., Lynch, R. S., Morris, C. D., Gowensmith, W. N. (2000). An application of Beck's cognitive therapy to general anger reduction. Cogn Ther Res, 24, 689–697.
42. Whiteman, M., Fanshel, D., Grundy, J. F. (1987). Cognitive-behavioral interventions aimed at anger of parents at risk for child abuse. Soc Work, 32, 469–474.
43. Deffenbacher, J. L., McNamara, K., Stark, R. S., Sabadell, P. M. (1990). A combination of cognitive, relaxation, and behavioral coping skills in the reduction of general anger. J Coll Student Dev, 31, 351–358.
44. Thurman, C. W. (1985). Effectiveness of cognitive-behavioral treatments in reducing Type A behavior among university faculty. J Couns Psychol, 32, 74–83.
45. Thurman, C. W. (1985). Effectiveness of cognitive-behavioral treatments in reducing Type A behavior among university faculty—one year later. J Couns Psychol, 32, 445–448.
46. Chemtob, C. M., Novaco, R. W., Hamada, R. S., Gross, D. M. (1997). Cognitive behavioral treatment for severe anger in posttraumatic stress disorder. J Consult Clin Psychol, 65, 184–189.
47. Rose, J., West, C., Clifford, D. (2000). Group interventions for anger in people with intellectual disabilities. Res Dev Disabil, 21, 171–181.
48. Snyder, K. V., Kymissis, P., Kessler, K. (1999). Anger management for adolescents: efficacy of brief group therapy. J Am Acad Child Adolesc Psychiatry, 38, 1409–1416.
49. Davidson, K., MacGregor, M. W., Stuhr, J., Gidron, Y. (1999). Increasing constructive anger verbal behavior decreases resting blood pressure: a secondary analysis of a randomized controlled hostility intervention. Int J Behav Med, 6, 268–278.
50. Larkin, K. T., Zayfert, C.(1996). Anger management training with mild essential hypertensives. J Behav Med, 19, 415–433.

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Psychosocial Interventions for Intimate-Partner Violence

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INTRODUCTION

We now recognize intimate-partner violence (IPV) to be one of the most common forms of interpersonal aggression. It occurs in both heterosexual and homosexual, married and unmarried couples across the socioeconomic and age spectra, and within every ethnic, racial, and cultural group. It is perpetrated almost equally by males and females (1), although it is considered to be more damaging and malignant when the male is the aggressor. In recognition of this, the term *batterer* is almost exclusively applied to male perpetrators. Historically, society has tolerated, sanctioned, and, at times, even prescribed the use of aggression by men toward their female partners. The legal system has been unresponsive and batterers have been spared any legal consequences for their behavior. As a result, it was only through the efforts of the battered-women's movement that IPV has been criminalized, and battered women have been accorded the same protections and rights as victims of other violent crimes. The sequelae of this process have been manifold and include dramatic increases in the rates of arrest and prosecu-

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tion of batterers, the development and rapid proliferation of intervention, and, more subtly, the splitting off of IPV from other forms of human aggression. In other words, the extensive body of literature on the etiology and treatment of human aggression has been largely ignored with respect to domestic violence, which is too often characterized as being solely attributable to the male's need to assert power and control over his female partner.

The dominance of the power and control paradigm has guided the development of interventions for batterers such that it is almost universally included and, in the case of the profeminist approaches, may be the sole focus of treatment. The relevance of power and control issues in IPV is generally accepted; however, empirical support for its utility is lacking, and intervention programs based on power and control models have generally produced small treatment effects (2). The politics of IPV have not only influenced the nature of intervention but also constrained it. Unlike other areas of treatment, in which the state regulates practitioners through licensure but otherwise maintains a laissez-faire policy with respect to intervention strategies, many states have adopted certification standards that dictate the content of treatment and inhibit the development of novel, innovative strategies.

Another consequence of the power and control zeitgeist is perpetuation of the fallacy that IPV is unidimensional and that batterers are a homogeneous population. The popularity of subtyping strategies coupled with the notoriety of "one size fits all" intervention models testifies to the heterogeneity of the battering population and the inadequacy of one dimensional treatment approaches. This is also reflected in the application of the transtheoretical model (3) to batterers' intervention. Proponents of this model suggest that interventions must be matched to the batterer's stage of readiness to change. Stage matching and motivational interviewing are increasingly proposed as either alternatives or adjuncts to subtype-specific intervention strategies. Although there is much disagreement on specific subtypes, and prescriptive matching of subtypes to interventions is more theoretical than real, there is little debate that the field is heading in this direction.

An important unanswered question is whether batterers' treatment is effective. Outcome research, flawed though it is, is less than encouraging (4–7). As with therapeutic approaches to other problems, empirical validation is necessary. Unlike most other treatments, however, legal and safety issues complicate randomized designs, especially with respect to waitlist or minimal contact control groups. Another methodological complication concerns the selection of valid outcome measures. Victim report, generally regarded as the gold standard among outcome indices, introduces both safety considerations and sample selection biases. In this chapter, we will examine the issues and controversies that surround batterers' treatment, the psychosocial intervention strategies most commonly employed in its remediation, and the evidence for its efficacy, and make some suggestions regarding future directions.

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Among the many controversies that abound in the IPV area is whether programs dealing with batterers should be classified as intervention or treatment. Initially, the term "batterers' treatment" predominated, but more recently the trend toward the criminalization (and consequent depathologizing) of IPV is reflected in the growing popularity of the term "batterers' intervention" to describe this enterprise. Intervention proponents are prone to view themselves as part of the criminal justice system and their mission as protection of the victim by monitoring the behavior of batterers and communicating with victims, the courts, and the police (8). There are legitimate concerns that viewing such intervention in therapeutic terms will support legal attempts to excuse battering, shift sympathy to the batterer, and impose confidentiality requirements that might interfere with victim protection. Equally realistic, however, are concerns that failing to approach batterers therapeutically will diminish the effectiveness of the treatment and thereby increase the risk for victims.

Supporters of therapeutic approaches are most likely to come from the ranks of the mental health professions and to subscribe to the confidentiality requirements of their respective disciplines. In most cases their actions would be guided by the Tarasoff decision (9), which would require the therapist to compromise confidentiality only in the event of a threat made toward a named, intended victim. Therapeutic programs take the position that the batterer is the patient and that effective treatment of the batterer is the primary goal. This is consistent with the widely held belief that patients will not engage in therapy nor can they be successfully treated unless the treater is viewed as an advocate. In fact, confidentiality is seen as essential to the creation of a safe environment where the patient can discuss his/her problems, and the therapist can offer suggestions for avoiding such problems in the future, and/or referrals for additional treatment. For therapeutic programs, protection of the victim is achieved as a byproduct of the successful treatment of the batterer.

Despite these confidentiality considerations, many therapeutic programs employ victim contacts, either to provide safety information to victims or to solicit feedback regarding the batterers' progress or the lack thereof, but only with the consent of the batterer. In some cases, a signed release is a precondition for participation in therapy, effectively negating confidentiality constraints. Therapeutic programs are also less likely to provide ongoing feedback to the courts/probation departments, especially for reasons other than attendance. It should be noted that there is great variability among programs with respect to this issue. Also, victim contacts are not risk free, and many programs agonize over how to use victim feedback in treatment without jeopardizing victim safety. Nevertheless, the batterers' treatment standards in many states require victim contacts (e.g., Massachusetts). One important function of victim contact is to inform the victim that participation in a batterers' treatment program is no assurance that the batterer will improve or that she is now safe to continue relating to him. She

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can also be given information regarding local shelters and services for women, and encouraged to have him arrested if he behaves aggressively toward her.

The impact of a program's confidentiality policy on its effectiveness has not been empirically evaluated; however, we can speculate that the fewer the protections, the less likely participants will be to disclose aggressive impulses and behaviors, alcohol, substance abuse, and other potentially reportable behavior. The more informational/educational a program is, the less they may be concerned about the batterer's reluctance to make such disclosures. Process-oriented programs might view this as a serious impediment to a successful outcome. The objective outcome, however, is independent of how the program views it. In either case, the limits of confidentiality should be clearly disclosed to participants at the initial contact with the program.

Batterers are most commonly treated in closed-ended, gender-specific groups, although couples counseling and couples groups have also been described in the literature (e.g., 10,11). Joint counseling for couples in which there is aggression has long been controversial. Many states either discourage or prohibit treating the dyad, at least as an initial or primary approach to batterers' treatment. Couples approaches have been criticized for shifting responsibility for aggression from the batterer to the couple, and for communicating the belief that relationships in which there is aggression can and should be preserved (12,13). There are also safety concerns about bringing the couple together to discuss hot-button topics and loaded issues and then allowing them to leave together.

Couples counseling for aggressive couples does, however, have some advantages. In many cases, the aggression is mutual and it is important to eliminate aggression by both partners (14). Strategies such as the time-out are also more easily implemented in a couples format, and having the victim present offers better opportunities for feedback and monitoring of progress. Empirical evaluations of programs employing a couples format have found them to be equally effective and safe as gender-specific groups (5,15); however, couples were carefully screened and more seriously violent couples were excluded from this research. Conjoint couples counseling is most often used as a follow-up to gender-specific group treatment of the perpetrator, and then only if both partners have committed to continuing in the relationship and are voluntarily participating. The remainder of this chapter focuses on gender-specific, psychoeducational batterers' treatment groups because these are the dominant paradigm for treatment of IPV.

OPEN- VS. CLOSED-ENDED GROUP FORMATS

There are advantages and disadvantages to each format. Groups serving court-mandated batterers are often closed ended and time limited, as judges might be reluctant to require batterers to complete programs of indeterminate length. This

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format allows for better development of group processes by providing a safe and familiar environment that facilitates trust and disclosure of fears, weaknesses, and past traumas. In a closed-ended group, the curriculum can be presented in a meaningful order, such that each session can build on previous sessions. For example, identifying cues that one is becoming angry is followed by the time-out. It is also easier to track the progress of each group member. Open-ended groups suffer from frequent disruptions as new members are added. The group has to readjust and become comfortable with new members on an ongoing basis. It is much harder for the group to form and for participants to become familiar with details of each other's lives. Another consequence of the open-ended format is that group members learn the content in a different order. It is possible that the order of presentation of the material impacts outcome, although this has not yet been empirically demonstrated.

An important advantage of open-ended groups is that the more experienced group members are often effective therapeutic agents for the newer members. A common dynamic in batterers' treatment groups is the tendency of group members to discount the advice of group leaders because they (the leaders) may be perceived as having come from more advantaged home environments, do not have the same financial concerns, wear better clothing, are older, are better educated, are from a different culture, or are otherwise "not like us." Group leaders are frequently confronted with, "You do not understand because. . . ." This inclusion/exclusion strategy is intended to invalidate the leaders, but also represents the participant's fear that the group leaders do not fully understand the demands of their environments. The presence of men who are further along in the program and who can support the group leaders is helpful in combating this problem and enabling the men to change.

The decision regarding closed/open-endedness is often made on the basis of pragmatic, rather than therapeutic, considerations. Many programs lack an insufficient referral base for the closed-ended format to be economically feasible. Others have concerns about waitlisting potentially violent participants even for a short time and open-ended groups allow batterers to enter group more quickly. There are no empirical studies comparing open-ended with closed-ended formats.

TREATMENT DURATION

What is the ideal length of a batterers' treatment program? There is considerable interprogram variability regarding group, and session, length. There are descriptions in the literature of groups of as few as 10 weeks up to 12 months or more (16,17). The California state standards require that groups be of at least 12 months' duration. Programs employed in research designs may be briefer than those developed solely for clinical purposes. Programs differ as to whether they describe their length in number of sessions or by number of hours of treatment.

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The Massachusetts standards require that certified programs provide at least 80 hours of treatment (18). Specifying the number of hours of treatment offers a convention that more clearly describes program length. There is also variability regarding how many sessions a participant may miss and still be considered to have completed the program. In our own program (The Men's Workshop), participants may miss no more than two sessions and must make up any missed sessions. Advocates of longer programs argue that battering involves long-standing patterns of behavior and deep-seated beliefs and attitudes regarding women and the nature of relationships with women (19). They feel that there is usually resistance to treatment and that breaking down resistance, changing belief systems and attitudes, and working with personality disorders, which include antisocial, borderline, and dependent traits, requires more than a "quick fix." Proponents of management/control types of programs also contend that batterers need to have their behavior monitored for as long as possible (8).

Proponents of briefer interventions contend that whatever benefits may result from longer interventions are offset by their lower completion rates. Of course, this too might be remedied by more rigorous attention by probation officers. In fact, Rosenbaum et al. (20) found that a court order was the best indicator of treatment completion. Longer programs are usually more expensive for the participant and are therefore differentially accessible and punitive to lower-SES participants. Perhaps their strongest argument, however, is the absence of empirical evidence that programs of longer duration produce more favorable outcomes. In fact, what evidence there is, suggests a point of diminishing returns beyond a critical length, which may be as few as 15–20 sessions (20). Although there is a dearth of evidence supporting the superiority of longer programs, the "longer programs" in most published investigations have not approached 52 weeks in length. Further empirical assessment is clearly warranted. Unfortunately, it is difficult to isolate program length as a variable, as longer programs may also differ on other variables, such as content, philosophy, victim, and legal system (courts, police, and probation) involvement.

COMPLETION CRITERIA

Time-limited, closed-ended groups usually set completion criteria that are attendance, rather than performance, related. A participant who completes the requisite number of sessions is considered to have completed the program. This avoids the need for the program to determine whether the batterer has made any progress or achieved the program objectives. In principle, a batterer could sit through the required number of sessions, have learned nothing, be continuing to abuse his partner, and still get a letter of completion which would satisfy his probation requirement. Alternatively, programs might set some attitudinal or behavioral criteria for termination and attempt to assess them either directly or via victim report. Setting such criteria might promote either a) impression management

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whereby the batterer either withholds relevant information or learns to parrot the sought-after responses, or b) "persuasion" of the victim to do the same.

TWO-TIERED GROUPS

Increasingly common is the use of a two-tiered format in which batterers first participate in a time-limited, short-term (8-12 weeks), highly structured, educational, or informational group. Sometimes referred to as an "intake" group, it is designed to raise consciousness, provide information, and, in some cases, teach behavioral strategies, such as the time-out. This is often an open-ended group, and participants are added as they enter the program. The objective is primarily educational, so there is less concern about any disruption caused by new members' joining the group. Upon completion of the intake group, participants join a process group, on equal footing with respect to their knowledge of relationship aggression and the strategies that may be employed to eliminate it.

The process component may be much longer, or even of indeterminate length, and typically deals with more complicated issues, such as exposure to violence in the family of origin, self-esteem, relationship issues, or their own sexual victimization. Programs vary in terms of whether the process component is also closed- or open-ended (21).

LEADERSHIP

A male-female coleader team is considered by many to be the ideal. Groups led by a single male or single female leader are also common. Dual-gender teams present opportunities for the coleaders to model egalitarian power relationships between males and females, bring in a female point of view, and reduce the likelihood that the group will deteriorate into a "good old boys" club. On the other hand, it takes a great deal of work and vigilance to maintain the ideal power balance and avoid falling into gender-typed patterns of behaving (e.g., the male coleader protecting the female coleader from group criticism, the female group member deferring to the male, or the male assuming leadership of the group), which can disrupt the normal therapeutic behavior of group leaders and the natural flow of the group. We have also noted that having a female coleader stifles the expression of misogynistic attitudes and language that might otherwise be expressed and treated. Outcome research comparing the various group leadership configurations is unavailable.

COMMON INTERVENTION STRATEGIES

Just as programs vary in philosophy, structure, and duration, there is substantial variability in the program content. Yet despite this, there is also a surprising

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amount of agreement regarding curriculum. What follows are descriptions of the more commonly included elements of treatment, as based on published program descriptions.

Almost all programs emphasize power and control issues. This is based on the core belief of the profeminist model that coercive control and the assertion of male power underlies all male-to-female domestic aggression. The power and control wheel developed by Pence and Paymer (19) is usually employed to acquaint batterers with the many forms of aggression and coercive strategies and encourage them to consider the way they use these strategies in their intimate relationships. Batterers, like most men, have been brought up believing it is wrong to hit women but do not consider restricting activities, monitoring behavior, economic control, or asserting male privilege to be abusive. The equality wheel, also adapted from the Duluth program (10), is often used to promote discussion of alternatives to power and control.

Profeminist models posit that battering is not angerrelated, and some state certification standards specifically exclude anger management as a primary intervention for batterers (e.g., Massachusetts). Nevertheless, many programs include some elements of anger management. It has been suggested that batterers may fail to appreciate their state of emotional arousal until they become so highly aroused that their ability to modulate their emotions or to think clearly regarding alternatives and consequences is impaired (22). Teaching batterers to identify physical, behavioral, and psychological cues that they are becoming angry offers several benefits, including increasing the probability that they can implement strategies taught in group, e.g., cognitive strategies to short-circuit the buildup of anger and reduce aggressive behavior. They can also be taught to identify situational cues, dangerous places and times, and "hot-button" topics which might help them to avoid repeating past problems. Concrete suggestions such as remaining seated during arguments, avoiding affect-laden topics while riding in automobiles, and carefully choosing times and places for working out disagreements are often included.

Most programs employ some variation of the time-out procedure. Once they have been taught to identify cues that their anger level is rising, batterers are instructed to remove themselves (physically) from the argument until they have calmed down sufficiently to continue the interaction. The combination of identifying cues and using time-out are the foundations of anger management. This strategy can also be combined with cognitive techniques including the use of calming cognitions that defuse, rather than inflame, them while in a time-out. We have had success with the strategy of having the men ask themselves, "How do I want this to turn out?" While in a time-out, they are instructed to remain out of the situation until they are able to safely resume the discussion with their partner. They are also instructed to call their partner before returning to make sure that she is in agreement that he should return. Many batterers report that

their partners try to prevent them from taking a time-out. In our program, we advise the men to discuss this strategy with their partners at a time when they are not in conflict and to promise that they will not use the time-out as a way of avoiding or terminating the discussion, that they will not drink, drug, or drive during the time-out, and that they will return to the discussion as soon as they can do so calmly and nonaggressively.

Because men are traditionally brought up to believe that emotions such as fear and sadness are feminine, they may be constricted in their emotional expression and left with anger as the only "acceptable" masculine emotion (23). The anger funnel is a metaphor for the idea that men are socialized to suppress all emotions except anger, and thus anger becomes the only "acceptable" way to express feelings and a proxy for these other painful emotions. Teaching batterers to identify the actual emotions that underlie anger can help to reduce the frequency with which they express anger. By expressing the actual emotions he is feeling, he gives his partner the chance to respond to his true feelings instead of responding to his anger with anger of her own.

Unfortunately, aggression in relationships is often immediately reinforced by the batterer getting his way, feeling powerful, punishing someone he perceives as having wronged him, etc. To counteract these reinforcers, programs often focus on the price one pays for being aggressive toward intimates. The so-called "costs of aggression" include financial costs, effects on their own and their partner's health, effects on children, effects on career, damage to the relationship, loss of intimacy, loss of freedom, loss of status in the community, shame, and embarrassment. Focusing on these long-term costs increases the likelihood that batterers will learn that the negative consequences of using aggression outweigh any benefits. This is particularly true with respect to the effects on children. Having often come from dysfunctional families where they often witnessed interparental aggression, were themselves abused by parents, or both, batterers are often motivated to change so that they do not become "like their fathers" or so that their own children do not hate them as they do their own fathers.

Although as Kantor and Straus (24) noted, although alcohol is neither necessary nor sufficient for the occurrence of relationship aggression, the two frequently co-occur. Recognizing the linkage between substance use, especially alcohol, and aggression, programs often include an alcohol and substance abuse component in which they discuss the relationship between alcohol and aggression, raise consciousness about the problems associated with alcohol and substance use, and provide information and referral. In our own program, we discuss the defining characteristics of alcoholism and then suggest that batterers consider that they have an alcohol problem if they get into trouble when under the influence, regardless of whether they meet DSM-IV criteria. We provide lists of alcohol treatment facilities and AA meetings in the area, and offer referrals to those men whose problems require more intensive interventions. We also focus on the

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fact that alcohol neither causes nor excuses aggression, and combat any attempts by the men to use alcohol as a way of diminishing their responsibility for their behavior.

Batterers may have poor communication skills, may not be good listeners, tend to be defensive in discussions, view disagreements as battles to be won, and magnify the importance of winning the argument, perhaps because they are "traditional" men who associate masculinity with being right and having their way (23). They may lack good communication skills and be less articulate than their partners, sometimes resorting to violence as a conflict resolution strategy. Although communication is a dyadic issue which may be more effectively addressed in a couple-counseling format, some groups include a communication component. In our program, we encourage the men to listen to their partners, ask questions, use "I" language, and paraphrase. We focus on teaching batterers about the different elements of communication, such as tone, posture, facial expression, volume, gestures, content, and the fact that they may send mixed messages, for example, if the tone and content are inconsistent. Batterers and other aggressive individuals are often unaware of their impact on others, including their partners and children, and express surprise that others are afraid of them. It can be useful to approach this from the perspective of how they communicate with others.

Many programs are self-described as having a cognitive-behavioral orientation. The cognitive model suggests, in part, that batterers anger themselves with inflammatory cognitions in the absence of factual information. Presented with the scenario that "your partner is 2 hours late in coming home and she has not called to let you know where she is," a batterer typically makes some negative attribution, for example, that she is cheating on him. Pressed for alternatives, the batterer is often unable to come up with more benign possibilities (e.g., maybe she had a flat tire or is stuck in traffic). Nonbatterers, in contrast, will more often conjure noninflammatory, or even empathetic, possibilities (25). Batterers may be taught to identify this pattern and to replace the inflammatory cognitions with more calming ones, or at the least, to suspend judgment until they have more information.

Relaxation and stress reduction strategies may be included because batterers report a variety of stressors including financial, occupational, legal, parenting, and family stress. If they see themselves as helpless in the face of these stressors, they will not take steps to alleviate them. Our program teaches a problem-solving approach to reducing stress. Batterers are taught to identify the specific stressors, generate possible solutions, evaluate pros and cons of each, select the most reasonable alternative, and try it out. Brief relaxation protocols (deep muscular relaxation, breathing exercises, mental imagery) are used to provide the men with skills they can use to reduce tension and/or anger. These skills may be used in conjunction with other techniques taught in group. For example, when angered,

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the men may be taught to take a time-out, relax, identify inflammatory cognitions, and replace them with calming ones.

Batterers often have children, sometimes with several different partners. Some of the reasons for this may be that they confuse sex with intimacy, view women as sexual objects, eschew birth control because it reduces their own pleasure, or want to reduce the woman's options by impregnating her. Batterers may have difficulty caring for, disciplining, relating to, and interacting with, their children. If they have poor self-esteem, they may be excessively sensitive to disrespect and view their children's misbehavior as a personal failure and/or an embarrassment. Many report being subjected to excessive physical discipline as children, and are frustrated by recent changes in the way child protective laws are enforced. Batterers can be taught empathy with the child, the importance of developing a positive parent-child relationship, and both the value and methods of nonphysical child management, such as the time-out, the use of consistent consequences, and positive reinforcement.

As noted above, batterers are often brought up with "traditional" male attitudes and beliefs. These include that they must be strong, independent, and in charge, and that the man is the head of the household and the woman is subservient to him. In addition to some of the negative consequences that this has for the partner, it also puts a great deal of pressure on the man himself. He may have difficulty asking his partner for help or sharing responsibilities with him. He may also have difficulty admitting he is wrong, giving and receiving compliments, and refusing requests by others, which are traits usually labeled unassertive. There is research indicating that batterers have difficulty with spouse-specific assertion, that is, asserting themselves in their relationships (26,27). Teaching proper assertiveness is therefore included in many batterers' treatment programs.

Developing empathy and compassion is an excellent way of reducing aggression both inside and outside relationships. Objectification of the enemy through the use of derogatory or slang nicknames serves the function of objectifying the enemy and facilitating aggression toward them. Using misogynistic terms for women serves a similar function. Programs typically focus on the importance of using nonsexist, nonaggressive language. The development of empathy may also be facilitated by having the men remember what it felt like to be abused themselves and then projecting those feelings onto their partners and/or children.

Films, videotapes, or other media may be used to convey some of the program content. The Duluth program, for example, has developed videotapes specifically for their program, but they are only available to individuals who have completed their training program. In our own program we use several films, including the videos "Battered Women" (28) and "Alcohol and Cocaine: The Secret of Addiction" (29), and the "Time-Out" series (O.D.N. Productions, Inc.). Other

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films and videotapes made specifically for batterers treatment are also available, e.g., "Shadows of the Heart" (30). Intermedia Productions [see (30) for contact information] offers a selection of these materials. These films are used to promote discussion about important issues. They present issues in a dramatic and often poignant fashion, and offer a break from the usual discussion format.

Handouts are popular and reinforce the teaching that goes on in group. They also serve as reminders of the things the men have been taught. Geffner and Mantooth (10) published a workbook containing a variety of worksheets culled from many different programs throughout the United States. The workbooks themselves may be purchased by, or for, batterers, and programs may choose to reproduce those sheets they find most appropriate for distribution to group participants. At the end of our program we give the men a review booklet that summarizes each of the topics covered in group and includes a list of important resources and telephone numbers.

DOES BATTERERS' TREATMENT WORK?

The success (or failure) of batterers' interventions is of paramount importance to those involved in the development of these programs as well as to the criminal justice system and society as a whole. Understandably, courts will be reluctant to mandate batterers into such programs without evidence that they reduce domestic aggression. Thus, it is not surprising that there has been increasing pressure to evaluate the effectiveness of batterers' intervention programs. Evaluation of these outcomes has not been an easy task, however. The gold standard of treatment outcome research involves randomized designs comparing treatments to each other and/or to no-treatment control groups. Ideally, participants are randomly assigned to the various treatment conditions, and outcome is assessed at different time periods, such as pre-, post-, and extended follow-up. Although optimal, this study design poses significant methodological and ethical problems in the case of batterers' intervention programs.

These issues have been extensively discussed in other places (c.f. 20) and will be only briefly summarized here. Legal and victim safety issues preclude the use of no treatment control groups, and certification standards in many states prohibit assignment of batterers to any but state-certified programs. State standards often proscribe couples counseling, individual psychotherapy, and psychodynamic approaches, or prescribe only the better known profeminist model programs such as Duluth or Emerge, further restricting research possibilities. The selection of an appropriate outcome measure is also problematic. Victim report is generally viewed as the most valid indicator; however, contacting victims is difficult and can compromise her safety. Studies utilizing victim report as an outcome measure generally report high refusal (or unable to contact) rates, injecting a self-selection bias into the results. Methodological weaknesses must be

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borne in mind when assessing the batterers' treatment outcome literature. Despite concerns, outcome research has been conducted (e.g., 2,5). Given the fact that batterers' intervention programs are a relatively recent development, the information gained from this research may be most valuable in guiding the direction of future research.

EFFICACY

Early studies of batterers' interventions indicated that between 50% and 80% of participants reported a decrease in aggression (as reported by their partners) at 6 months to 1 year following program completion (e.g., 16,31–33). In a review of this earlier research, Gondolf (2) indicated 11 primary limitations of these studies including: small sample size, single-site design, representativeness of the sample, low response rates, lack of measurement of batterers' characteristics, short follow-up periods, inclusion of program completers only, consideration of only initial partners, lack of a control group, research conducted by program staff that may have been biased toward successful outcome, and difficulties in random assignment.

More recent studies of batterers' interventions have attempted to address these limitations as well as evaluate other key issues in the treatment of batterers. Harrell (34) conducted a treatment outcome study in which arrested batterers were randomly assigned to a 3-month batterers' program, probation, or a suspended prosecution without treatment. No significant difference was found between treatment and no-treatment in this study; however, the batterer's intervention in the study was shorter, more disorganized, and not representative of batterers' treatment programs (2).

In a comparison of gender-specific treatment and conjoint (couples) treatment (15), volunteer couples were assigned to 14 weeks of either gender-specific or couples' group treatment. Results indicated significant decreases in psychological and physical aggression for husbands across time, but no differences as a function of treatment condition. However, 22 of the husbands continued to be aggressive following treatment, and only 39% ceased their aggressive behavior completely. The results of this study are important because they address the questions of safety and efficacy of dyadic interventions, which had been the subject of much speculation but little empirical research. They are somewhat limited by subject selection criteria which excluded couples who reported incidents of aggression that resulted in injury and inclusion of couples who reported only two incidents of aggression in the past year. Interpretation is further complicated by the fact that participating couples were volunteers (rather than court-mandated) and a study attrition rate of 51%.

A major study conducted by Dunford (5) examined the relative impact of three 12-month interventions for 861 U.S. Navy couples with a history of hus-

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band-to-wife physical aggression. Couples were randomly assigned to a control group, men's group, conjoint group, or a rigorous monitoring group. A cognitive-behavioral model and similar aggression-reduction strategies (i.e., time-out, cognitive restructuring, communication skills) were used in both the men's group and the conjoint group with the exception being that conflict resolution strategies were also taught in the conjoint group. The rigorous monitoring group was described as individual counseling conducted as deemed appropriate by the case manager at the Navy Family Advocacy Program. Results of the study revealed no significant differences between groups on victim- or perpetrator-reported incidents of continued abuse. In addition, there was no significant difference between any of the experimental groups and the control group on the outcome measures, nor were there any significant differences between groups on prevalence of new arrests following treatment. However, limitations in the study also advise caution in interpreting the results. The undefined nature of the rigorous monitoring intervention, and the fact that all participants were under military scrutiny and the low participation rate of wives in the conjoint treatment (2:5, women to men), are of concern.

Two additional studies (35,36) examined the effect of batterers' programs in New York and Florida. Both evaluated Duluth model programs and found no significant differences between treatment and control groups. As with previous studies, however, limitations mar the interpretability of the findings. In the study by Davis et al. (35), low response rate of partners during follow-up ($\sim$ 50%) and problems in random assignment may have affected study results. In the Feder and Ford study (36), an even lower response rate (21%) and inequality between response rates across experimental groups which led to the reliance on probation violations as an indicator of program effect might have also negatively skewed study results (2).

In a multisite evaluation of batterers' interventions, Gondolf (2) studied four programs in several cities. The programs met specific inclusion criteria and ranged from 3 to 9 months in duration. In addition, follow-up was assessed via phone interviews with men and women every 3 months. Women's reports were verified by using police incident reports. Researchers obtained information not only about assault and other forms of abuse but also regarding the women's overall quality of life as indicators of treatment effectiveness. Response rates for the study were reported as 68% for the 15-month follow-up period, 67% for the 30-month period, and 58% for the 48-month period. Results of the study indicated a "moderate" treatment effect for the batterer's programs (2,37).

There are a number of literature reviews and meta-analyses (4,7,32,33, 38,39) examining the effectiveness of batterers' intervention. The reviews have been inconclusive with regard to treatment effect.

Most recently, Babcock et al. (6) conducted a meta-analysis of the existing batterers' treatment outcome literature in an effort to assess the effect size for

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batterers' intervention. Of the 68 studies that were originally identified, 22 (i.e., quasi-experimental and experimental) were included. The findings indicated that batterers' treatment had a "small" effect on recidivism (6). Furthermore, Duluth-type and cognitive-behavioral programs were not significantly different in effect size. The authors concluded that when compared with the standards for psychotherapy in general, these findings suggest that batterers' intervention has room for considerable improvement.

As the researchers noted, meta-analytic studies are only as sound as the individual studies included. Although only the most rigorous studies were entered into this meta-analysis, many of the studies in the batterers' treatment outcome literature suffer from the various limitations that have been discussed. Given these limitations, it is not surprising that the overall effect size for batterers' interventions was in the "small" range (6).

Empirical outcome evaluations may be uninspiring but they have also raised many important questions that may ultimately move the field in a positive direction. Most of the studies examined, for example, included programs based on Duluth, or other pro-feminist, model programs. Many of these interventions fail to protect confidentiality, are confrontative, and are nontherapeutic. Brooks (23), writing about "traditional" men, concludes that confrontative approaches are ineffective with this population. Rather than conclude that batterers' treatment has a negligible effect, we might more accurately suggest that confrontational programs may not be effective. More productive would be to suggest that we turn our attention to discovering which components of treatment are effective and which are not.

Batterers' treatment as a "work in progress," however, is inconsistent with the trend toward the establishment of state certification standards which, all too often, pretend that we know exactly how to treat batterers. Existing standards (e.g., Massachusetts') are so restrictive as to preclude the development of new, innovative, possibly more effective interventions. The pros and cons of state certification standards are more fully discussed in a special issue of the Journal of Aggression, Maltreatment, and Trauma (40).

Another recent trend is the application of the transtheoretical model (3) to batterers' treatment (41,42). This model suggests that batterers are in various stages of acceptance regarding their problem and the need to change. Many are in the precontemplative stage where they have not yet taken responsibility for their behavior or made the decision to change. This model suggests that different interventions are required depending on the stage of readiness for change of any particular batterer. Techniques such as motivational interviewing are beginning to be applied to this population, and the idea that different types of batterers may require different interventions is catching on.

Another popular trend, which also acknowledges the diversity of the battering population, is subtyping. Recognizing that batterers are a heterogeneous

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group, numerous attempts have been made to identify and describe subtypes. The best-known exemplar of this strategy is the typology developed by Holtzworth-Munroe and Stuart (43), which identifies three subtypes: family only, generally violent/antisocial, and dysphoric borderline. While there is research supporting the existence of these subtypes, the idea that they are differentially responsive to treatment has not been evaluated. Saunders (44), however, did demonstrate differential response to treatment based on batterer characteristics. Specifically, he found that antisocial batterers did better in a cognitive behavioral batterers' program, whereas dependent batterers did better in a psychodynamic program. These lines of research suggest that in addition to stage matching, we might also need to match batterer subtypes to specific intervention strategies.

Finally, batterer intervention has been almost exclusively psychosocial in content. Although the potential for employing pharmacological agents as adjuncts to treatment has been proposed, their utility has not been demonstrated. Serotonin-reuptake inhibitors, anticonvulsants, mood stabilizers, and β-blockers have all been identified as candidates for treating this population; at present, however, there is no support for their efficacy. In part, this may be due to resistance to medicalizing this problem, and thereby reducing the responsibility of the batterer for his behavior. On the other hand, battering is such a significant problem that we should leave no stone unturned in our efforts to find effective solutions.

REFERENCES

1. Straus, M., Gelles, R. (1990). Physical Violence in American Families: Risk Factors and Adaptations to Violence in 8,145 Families. New Brunswick, NJ: Transaction.
2. Gondolf, E. (2002). Batterer Intervention Systems: Issues, Outcomes, and Recommendations. Thousand Oaks, CA: Sage.
3. Prochaska, J. O., DiClemente, C. C. (1984). The Transtheoretical Approach: Crossing Traditional Boundaries of Change. Homewood, IL: Dow Jones, Irwin.
4. Davis, R., Taylor, B. (1999). Does batterer treatment reduce violence? A synthesis of the literature. Women Crim Justice, 10, 69–93.
5. Dunford, F. W. (2000). The San Diego navy experiment: an asessment of interventions for men who assault their wives. J Consult Clin Psychol, 68, 468–476.
6. Babcock, J., Greeen, C., Robie, C. Does batterer's treatment work? A meta-analytic review of domestic violence treatment. Clin Psychol Rev. (In press.)
7. Levesque, D. A., Gelles, R. J. (1998, July). Does treatment reduce recidivism in men who batter? A meta-analytic evaluation of treatment outcome. Presented at the Program Evaluation and Family Violence Research: An International Conference. Durham, NH.
8. Adams, D. (1994). Treatment standards for abuser programs. Violence Update, 5, 1, 5–11.
9. Tarasoff v. Regents of the University of California, 131 Cal. Rptr. 14, 551 P.2d 334 (1976).

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

10. Geffner, R., Mantooth, C. (2000). Workbook to Accompany Ending Spouse/Partner Abuse: A Psychoeducational Approach for Individuals and Couples. New York: Springer.
11. Neidig, P. H., Friedman, D., Collins, B. S. (1985). Domestic conflict containment: a spouse abuse treatment program. Soc Casework J Contemp Soc Work, 66, 195–204.
12. McMahon, M., Pence, E. (1996). Replying to O'Leary. J Interpers Violence, 11, 452–455.
13. O'Leary, K. D. (1996). Physical aggression in intimate relationships can be treated within a marital context under certain circumstances. J Interpers Violence, 11, 450–452.
14. Vivian, D., Heyman, R. E. (1996). Is there a place for conjoint treatment of couple violence? In Session, 2, 25–48.
15. O'Leary, K. D., Heyman, R. E., Neidig, P. H. (1999). Treatment of wife abuse: a comparison of gender-specific and conjoint approaches. Behav Ther, 30, 475–505.
16. Gondolf, E. (1997). Patterns of reassault in batterers programs. Violence Vict, 12, 373–387.
17. Palmer, S. E., Brown, R. A., Barrera, M. E. (1992). Group treatment program for abusive husbands: a long-term evaluation. Am J Orthopsychiatry, 62, 276–283.
18. Commonwealth of Massachusetts Executive Office of Health and Human Services. (1995). Massachusetts Guidelines and Standards for the Certification of Batterer Intervention Programs. Boston: Massachusetts Department of Public Health.
19. Pence, P., Paymar, M. (1993). Education Groups for Men Who Batter: The Duluth Model. New York: Springer.
20. Rosenbaum, A., Gearan, P., Ondovic, C. (2001). Completion and recidivism among court-referred and self-referred batterers in a psychoeducational group treatment program. J Agress Maltreat Trauma, 5, 2, 199–220.
21. Rosenbaum, A., Leisring, P. A. (2001). Group intervention programs for batterers. J Aggress Maltreat Trauma, 2, 57–71.
22. Gottlieb, M. (1999). The Angry Self: A Comprehensive Approach to Anger Management. New York: Zeig, Tucker & Theisen.
23. Brooks, G. R. (1998). A New Psychotherapy for Traditional Men. San Francisco: Jossey-Bass.
24. Kantor, G., Straus, M. A. (1990). The drunken bum theory of wife beating. In M. A. Straus, R. J. Gelles (Eds.), Physical Violence in American Families (pp. 203–224). New Brunswick, NJ: Transaction Press.
25. Gearan, P., Rosenbaum, A. (1997, July). Cognitive differences between batterers and nonbatterers. Presented at the 5th Annual International Family Violence Research Conference, Durham, NH.
26. Rosenbaum, A., O'Leary, K. D. (1981). Marital violence: characteristics of abusive couples. J Consult Clin Psychol, 49, 63–71.
27. O'Leary, K. D., Curley, A. D. (1986). Assertion and family violence: correlates of spouse abuse. J Marital Fam Ther, 12, 281–290.
28. Crowley, C. (Director). (1976). Battered Women: Violence Behind Closed Doors [film]. (Available from J. Gary Mitchell Film Company, P.O. Box 2438, Sebastopol, CA 95473-2438.)

MARCEL DEKKER, INC.
270 Madison Avenue, New York, New York 10016

29. Siedor, C. (Executive Producer), Sykes, M. (Producer). (1990). Alcohol and Cocaine: The Secret of Addiction. (Film, available from Drystar Television, Atlanta.)
30. Stosny, S. (Writer), Garcia, M. (Director). (1991). Shadows of the Heart: A Treatment Tape for Male Batterers [film]. (Available from Intermedia, 1700 Westlake Ave. N. Suite 724, Seattle, WA 98109.)
31. Edelson, J. (1996). Controversy and change in batterers' programs. In J. Edelson, Z. Eisikovits (Eds.), Future Interventions with Battered Women and Their Families (pp. 154–169). Thousand Oaks, CA: Sage.
32. Rosenfield, B. (1992). Court-ordered treatment of spouse abuse. Clin Psychol Rev, 12, 205–226.
33. Tolman, R. Bennett, L. (1990). A review of quantitative research on men who batter. J Interpers Violence, 5, 87–118.
34. Harrell, A. (1991). Evaluation of Court-Ordered Treatment for Domestic Offenders. Washington: Urban Institute.
35. Davis, R., Taylor, B., Maxwell, C. (1998). Does batterer treatment reduce violence? A randomized experiment in Brooklyn. Final report to the National Institute of Justice, Washington.
36. Feder, L., Forde, D. (2000). A test of the efficacy of court-mandated counseling for domestic violence offenders: the Broward experiment. Final report to the National Institute of Justice, Washington.
37. Gondolf, E., Jones, A. (2001). The program effect of batterer programs in three cities. Violence Vict, 16, 693–704.
38. Hamberger, K., Hastings, J. (1993). Court-mandated treatment of men who assault their partner: issues, controversies, and outcomes. In N. Z. Hilton (Ed.), Legal Responses to Wife Assault (pp. 188–229). Newbury Park, CA: Sage.
39. Babcock, J., LaTaillade, J. (2000). Evaluating interventions for men who batter. In J. Vincent, E. Jouriles (Eds.), Domestic Violence: Guidelines for Research-Informed Practice (pp. 37–77). Philadelphia: Jessica Kingsley.
40. Geffner, R., Rosenbaum, A. (2001). Domestic violence offenders: treatment and intervention standards. J Aggress Maltreat Trauma, 5, 1–10.
41. Levesque, D. A., Gelles, R. J., Velicer, W. F. (2000). Development and validation of a stages of change measure for battering men. Cogn Ther Res, 24, 175–199.
42. Begun, A., Shelley, G., Strodthoff, T., Short, L. (2001). Adopting a stage of change approach for individuals who are violent with their intimate partners. J Aggress Maltreat Trauma, 5, 105–127.
43. Holtzworth-Munroe, A., Stuart, G. L. (1994). Typologies of male batterers: three subtypes and the differences among them. Psychol Bull, 3, 476–497.
44. Saunders, D. G. (1996). Feminist-cognitive-behavioral and process-psychodynamnic treatments for men who batter: Interaction of abuser traits and treatment models. Violence Vict, 11, 393–414.

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18

Psychopharmacological Interventions

Neuroleptics and Lithium

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INTRODUCTION

Aggression is one of the more common problems psychiatrists are called upon to treat, especially in children and adolescents (1). Unfortunately, despite the availability of a variety of treatment approaches, including psychopharmacologic and behavioral (2), there is no well-established and agreed-upon standard treatment for aggression. The purpose of this chapter is to review some of the better data that exist regarding the use of psychopharmacologic agents for reducing aggression. While the neuroleptics and lithium have been used to decrease aggression in adults, the best data have come from studies in children and adolescents. After considering some of the basic concepts concerning diagnosis and assessment, there will be a review of published double-blind and placebo-controlled studies of neuroleptics, lithium, and anticonvulsant agents.

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BASIC CONCEPTS

Aggression is a behavior that can be normal, but serious aggression is part of the symptomotology of a variety of psychiatric disorders, including those usually seen in childhood. For example, in conduct disorder, aggressive acts are included in nine of the 15 DSM-IV criteria (3). Likewise, oppositional defiant disorder includes behaviors related to aggression such as hostility and anger. Psychopharmacologic treatment studies in childhood aggression usually involve children diagnosed with one of the disruptive disorders—oppositional defiant disorder, conduct disorder, and, at times, children diagnosed with attention-deficit/hyperactivity disorder. Studying children with these diagnostic disorders is consistent with findings from developmental studies which suggest that childhood aggression is associated with hyperactivity, oppositional behavior, and defiance from early in life (4,5). Another important diagnosis to consider in treatment studies of aggression is intermittent explosive disorder (IED). While IED is less well studied in children, the criteria for this disorder focus on aggressive behavior that is explosive. Future studies of childhood aggression should explore the inclusion of intermittent explosive disorder as a diagnosis when investigating the efficacy of psychopharmacologic agents for reducing aggression in children and adolescents.

The focus of this chapter is psychopharmacologic treatments that have been studied for reducing severe aggression, particularly in children and adolescents diagnosed with conduct disorder and other disruptive disorders. Some of the studies reviewed do not necessarily use the conduct disorder nomenclature. However, the descriptions of the subjects are such that it seems reasonable to assume that most subjects would be diagnosed with conduct disorder or another, related disruptive disorder.

Aggression can also be an associated feature of other psychiatric disorders, including schizophrenia, depression, and bipolar disorder. Also, various types of destructive behavior can be demonstrated in individuals with mental retardation and pervasive developmental disorders. The appropriateness of any psychopharmacologic treatment aimed at reducing aggression will depend on the presence of any underlying psychiatric disorder. As a general approach, treatment for the underlying psychiatric disorder should be optimized before considering the addition of another psychopharmacologic agent aimed at reducing the aggression alone. For example, if a patient with schizophrenia is aggressive as a result of delusions and hallucinations, treatment for schizophrenia should be optimized before adding a separate drug treatment regimen to reduce aggression. A similar approach should be used when the aggression is determined to be related to other psychiatric disorders including major depressive disorder, mania, etc. However, there are disorders for which drug treatment would only be considered for the purpose of reducing aggression. For example in conduct disorder, drug could be

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considered as a treatment for reducing aggression, but not for other symptoms of the disorder such as lying, stealing, or truancy.

CLINICAL DECRIPTIONS

Aggression is difficult to assess both during the course of clinical treatment and as an outcome in treatment studies. Aggression is a rare behavior even in aggressive individuals. A child who has one or two physical fights a week would be considered an aggressive child, yet capturing those relatively few incidents may be difficult if not impossible because the aggression may never or rarely be observed. By way of comparison, children who are hyperactive are hyperactive for much of the day, and it is often possible to observe their hyperactivity across several settings, including in the examiner's office.

Furthermore, not all aggression is the same; a number of subtypes have been described (6). For example, aggression can be predatory, protective, provoked, unprovoked, planned and controlled, explosive—or contain some combination of these and other characteristics. It is likely that the effectiveness of treatments, including psychopharmacologic treatments, will depend on the frequency and severity of the aggression regardless of the subtype. Additionally, the occurrence of aggression may vary depending on the setting; a child may be aggressive at home and school but not on a structured inpatient unit. It is important to recognize that placebo treatments have been associated with reduced aggression in children, and apparently aggressive children can respond to changes in the environment (7,8).

SUMMARY OF INTERVENTION AND HYPOTHESIZED MECHANISMS OF THERAPEUTIC ACTION

The safety and effectiveness of a number of psychotropic agents have been investigated under controlled conditions, including stimulants, anticonvulsant agents, serotonergic agents, neuroleptics, and lithium (9). This chapter will focus on the neuroleptics and lithium; other chapters in the book will discuss serotonergic and anticonvulsive agents in depth. In addition to the agents mentioned above, a number of other psychopharmacologic agents including clonidine have been reported to be useful in treating aggression based on case reports and small open studies (10).

Neuroleptics

Apart from their use in schizophrenia, the neuroleptics are commonly used to treat the aggression associated with a number of psychiatric disorders. More recently, the atypical neuroleptics have been investigated for their antiaggressive

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potential in borderline personality disorder (11), dementia (12), and mental retardation (13,14). In fact, a key aim of a large NIMH multisite study is to investigate and compare the effectiveness of several atypical neuroleptics as treatments for reducing severe agitation in Alzheimer's disease (15).

Since shortly after they were introduced to the market, the neuroleptics have been used to reduce aggression in children. Several of the conventional neuroleptics—chlorpromazine, thioridazine, and haloperidol—have labeled indications that include the treatment of aggression in children with disruptive behaviors (16). By and large, controlled studies have shown that neuroleptics are more effective than placebo in reducing aggression in children and adolescents (see Table 1).

However, one of the exceptions regarding efficacy is of particular interest. It illustrates the difficulties involved in treating aggression and assessing treatment response. Molling and associates (8), in a double-blind placebo-controlled study involving children aged 10–15 years, investigated the efficacy of perphenazine as a treatment for reducing a variety of disruptive behaviors including aggression. There were three study groups—a perphenazine-treated group, a placebo-treated group, and a control group that received neither drug nor placebo. The perphenazine and placebo groups (n = 28 combined) were treated under double-blind conditions, but everyone knew that the control group (n = 28) received neither drug nor placebo. It was reported that the perphenazine-treated and the placebo-treated group showed equal improvement with clear reductions in aggression. On the other hand, the open control group had a marked increase in symptoms. The authors speculated that because the staff knew the open control was not receiving treatment, as compared to the drug and placebo groups who were treated blindly, the open control group was perceived as worsening in symptom severity. This finding underscores both the necessity for having a placebo control in studies of aggression in children—both drug and placebo groups improved—and the possibility that staff (and perhaps families) will think that drug treatment is a necessity, or there will be a worsening of symptoms.

By and large, however, controlled trials with neuroleptics suggest that drug is effective for reducing aggression, including both conventional and newer atypical agents (see Table 1). Haloperidol, when administered at relatively low dosages, has been shown effective for reducing aggression. In a study comparing haloperidol, lithium, and placebo in 61 children aged 5.2– 12.9 years, Campbell and associates (17) reported that both haloperidol and lithium were superior to placebo, but also found that the haloperidol group had more untoward effects than the lithium group and that hospital staff reported that the haloperidol group appeared to be more "drugged" than the lithium group. Side effects reported for haloperidol in this study included sedation and extrapyramidal side effects. A concern with long-term administration of conventional neuroleptics such as haloperidol is the risk of tardive dyskinesia. A long-term study of haloperidol (in

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Table 1 Neuroleptics: Representative Controlled Trials for the Treatment of Aggression in Disruptive Children

Drug	Dosage range	Sample size	Age range (years)	Design	Outcome	Ref.
Perphenazine	≤16 mg/day	56	10–15	DB, PC parallel	Drug = placebo	8
Haloperidol	0.05 mg/kg/day	10	4–15	DB, PC crossover	Effective	88
Haloperidol	1–3 mg/day	12	7–12	DB, PC crossover	Effective	89
Haloperidol	1–6 mg/day	61	5.2–12.9	DB, PC parallel	Both drugs effective	17
Lithium	500–2000 mg/day					
Molindone	Mean = 26.8 mg/day	31	6–11	DB, PC parallel	Both effective	90
Thioridazine	Mean = 169.9 mg/day					
Risperidone	0.75–1.5 mg/day	20	6–14	DB, PC parallel	Effective	32

DB, double-blind; PC, placebo-controlled.

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autism) reported that dyskinesias (mostly withdrawal dyskinesias) occurred in 33.9% of children (18).

In recent years, the use of atypical neuroleptics has increased in children (19). This increased usage may be due to the perception that the atypical agents are less likely to cause dyskinesias. While this may be true, the administration of atypical agents has been associated with weight gain in children which is emerging as more of a concern with atypical agents than with conventional agents (20–30). Moreover, there are reports of dyskinesias with long-term risperidone administration (27).

There are reports of studies using risperidone as a treatment for reducing aggression in children, though most of the data was obtained in children who were cognitively impaired or mentally retarded (31, for additional review). It is not clear that such patients would truly have the same qualitative problems in disregarding social norms or that treatment response would be the same as in children with conduct disorder who have normal cognitive abilities.

In a study involving children with apparently normal cognitive functioning, Findling and associates (32) reported on a double-blind placebo controlled trial in children aged 6–14 years of age (mean 9.2 $\pm$ 2.9 years) and diagnosed with conduct disorder. Of the 20 subjects enrolled in the study, nine completed the trial. The findings indicated that risperidone was significantly more effective than placebo at dosages ranging from 0.75 to 1.5 mg/day. Side effects of risperidone included sedation, increased appetite, and weight gain. No child had extrapyramidal symptoms. The reports on risperidone that include children with impaired cognitive functioning (33–35) and mental retardation (36) similarly found that the drug was effective for reducing aggression but had additional side effects including extrapyramidal side effects, sialorrhea, and nausea. While there are no controlled trials of olanzapine for the treatment of aggression, a study in children aged 4.8–11.8 years of age (mean = 7.8 $\pm$ 2.1) diagnosed with autism found that olanzapine (mean dose 7.9 $\pm$ 5 mg/day) was as effective as haloperidol (mean dose 1.4 $\pm$ 0.7 mg/day) in reducing disruptive symptoms (26). Controlled trials of other atypical neuroleptic for reducing aggression in children have not been reported.

Lithium

Lithium has putative effects on the neurotransmitter systems associated with aggression, and is reported to reduce aggression in laboratory animals and humans (37,38). A substantial body of research implicates abnormalities in the serotonin system in aggression (39–43), including in children (44,45). Additionally, heightened noradrenergic and dopaminergic activity have been associated with aggression (for review, see 46,47). The presumptive mechanism for the antiaggressive

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effect of lithium is via the serotonin system (48,49). Lithium may reduce aggression by decreasing dopamine and norepinephrine release (50).

Lithium is among the most critically assessed treatments for reducing aggression in children and adolescents with conduct disorder (see Table 2) (for review, 51). Since its introduction in 1949 (52), lithium has been one of the first-line treatments for mania, a disorder in which patients often display verbal and physical aggression. Sheard conducted a series of placebo-controlled studies demonstrating the efficacy of lithium in chronically impulsive and aggressive young adult prisoners, employing both single-blind (49,38) and double-blind designs (53). The largest double-blind placebo-controlled study employed parallel groups and random assignment to lithium or placebo. The subjects were 66 young male prisoners who ranged in age from 16 to 24 years and received treatment for 1–3 months. In this study, the primary outcome was the number of fights reported by prison staff. Using this measure, it was found that lithium was superior to placebo for reducing aggression. Apart from studies involving prison populations, lithium has been reported to decrease aggression in adult subjects with personality disorders (54,55).

The antiaggressive effects of lithium have been more extensively studied in children and adolescents. In all, there have been five double-blind and placebo-controlled studies of lithium as a treatment for aggression in children with conduct disorder (Table 2) (17,56–59). The most recently completed study, that of Malone and associates (58), was a 6-week double-blind placebo-controlled trial that used parallel group design with random assignment to treatments. Following a 2-week single-blind placebo baseline period, subjects were randomized to 4 weeks of double-blind treatment with either lithium or matching placebo. Unlike many other studies in this population, this study employed a specific measure of aggression, the Overt Aggression Scale (OAS) (60,61). All were diagnosed with conduct disorder (DSM-III-R criteria) with the diagnoses confirmed employing the Diagnostic Interview for Children and Adolescents. To be randomized to treatments, subjects were required to meet a specific aggression criterion during the 2-week baseline period.

In all, 86 children were enrolled into the study. All had been admitted to the hospital with histories of severe aggression. Of these 86, only 40 subjects were randomized to treatments (20 to lithium, 20 to placebo), including 33 males and 7 females, aged 9.5–15.9 years (mean 12.5 $\pm$ 1.6). Of the 46 subjects who were dropped from the study after the baseline period, 40 were dropped for not meeting the aggression criteria required for randomization, a key finding with relevance for clinical care. That is, while all of the children enrolled had histories of severe aggression, almost half were not aggressive while receiving placebo during the baseline phase. If, in fact, they had been admitted to a clinical service and active drug was commenced shortly after admission, the improvement in

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TABLE 2 Lithium: Representative Controlled Trials for the Treatment of Aggression in Disruptive Children

Lithium dosage range (mg/day)	Serum lithium levels (mEq/L)	Sample size	Age range (years)	Design	Outcome	Ref.
500–2000		61	5.2–12.9	DB, PC parallel	Effective	17
600–1800		50	5.1–12.0	DB, PC parallel	Effective	56
Not given	0.6–1.2	33	12–17	DB, PC parallel	Not effective	59
Not given	0.6–1.2	35	6–15	DB, PC parallel	Not effective	57
900–2100	0.78–1.55	40	9.5–15.9	DB, PC parallel	Effective	58

DB, double-blind; PC, placebo-controlled.

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aggression could have incorrectly been attributed to drug effect for subjects who did not display aggression during the baseline period. This would also mean that many children would have been exposed to possible adverse reactions to drug without need. Thus, this result suggests that children should not be started on drug shortly after admission to an inpatient psychiatric service. To start drug early may obscure the clinician's ability to judge the drug effectiveness (7,62).

The primary efficacy outcome measures for the study were the OAS, the Clinical Global Impressions (CGI) (63), and the Global Clinical Judgments (Consensus) Scale (GCJCS) (17). The OAS is a specific measure of aggression that measures the frequency and severity of four forms of aggression: verbal aggression, aggression against objects, aggression against others, and aggression against self. The CGI and the GCJCS were used to measure global improvement. The researchers completed the CGI. The GCJCS was completed based on the consensus of the ward staff. There were significantly more responders in the lithium group than in the placebo group on both the CGI (14 lithium vs. 4 placebo responders; $P = .004$) and the GCJCS (16 lithium vs. 6 placebo responders; $P = .004$). Thus, both the research team and the ward staff, under double-blind conditions, agreed that lithium was more effective than placebo using global measures. Lithium was also found to be significantly superior to placebo on the OAS, a specific measure of aggression (mixed-model ANOVA, $F_{1,119} = 4.14$; $P = .04$). Thus, lithium was superior to placebo on both the global and specific measures of aggression.

Campbell and associates (17,56) reported on two major double-blind placebo-controlled studies with lithium, employing random assignment, conducted in an inpatient setting. In the first study, lithium was compared to haloperidol in 61 aggressive, conduct-disordered subjects aged 5.2–12.9 years (mean 8.9 years). Following a 2-week baseline period, subjects were randomized to lithium (500–2000 mg/day; optimal serum levels of 0.32–1.51 mEq/L, mean = 0.993 mEq/L), haloperidol (1– 6 mg/day; mean = 2.95 mg/day), or placebo for 4 weeks. Overall, lithium and haloperidol were significantly superior to placebo, and except for side effects, the drugs did not significantly differ from each other. Notably, haloperidol had significantly more side effects than lithium. In the second study (56), lithium and placebo were compared in 50 aggressive, conduct-disordered subjects aged 5.1–12 years (mean 9.4 years). They were treated with the study drug for a period of 6 weeks. Optimal daily dosages of lithium ranged from 600 to 1800 mg (median = 1248 mg, mean serum level = 1.12 mEq/L). In this second study, lithium was again found to be effective in reducing aggression.

The findings of Campbell and associates (17,56) and Malone and associates (58), conducted at a different sites and across different age ranges, found that lithium was an effective and safe treatment for hospitalized children and adolescents with conduct disorder. These findings, however, were not replicated in two studies, one conducted by Rifkin and associates (59) and the other by Klein (57).

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However, a number of factors may explain the disparate outcomes among these studies. Rifkin and associates (59) conducted a double-blind, placebo-controlled trial of lithium in 33 hospitalized conduct-disordered adolescents between the ages of 12 and 17 years who exhibited aggression. As in the study of Malone and associates (58), aggression was rated using the OAS (61), and therapeutic lithium levels (0.6-1.0 mEq/L) were quickly reached employing the test-dose method of Cooper and Simpson (64-66). No treatment effect for reducing aggression was found. However, the treatment period for the study drug was only 2 weeks, whereas it was 4 weeks in the study of Malone and associates (58) and Campbell and associates (17) and 6 weeks in the second study of Campbell and associates (56). By comparison, studies in mania have shown that with lithium administration, initial improvement may not occur until week 4 of treatment (67). Therefore, the clinical trial of Rifkin and associates (59) likely used an insufficient treatment period to draw conclusions about the efficacy of lithium.

In the other negative study, Klein (57) reported on 35 outpatients, aged 6–15 years. They were randomized to lithium (serum levels ranging from 0.6 to 1.2 mEq/L), methylphenidate (up to 60 mg/day), or placebo for a period of 5 weeks. Lithium was found ineffective as a treatment for aggression. This disparate finding may be attributed to the different aims of the studies. The aim of Malone and associates (58) and Campbell and associates (17,56) was to investigate whether lithium reduced explosive aggression, whereas the aim of Klein (57) was to investigate whether lithium reduced antisocial behavior. In the Klein study, few subjects who were enrolled displayed serious aggression. Another, perhaps critical, difference in the studies was the treatment setting. The studies of Malone and associates (58) and Campbell and associates (17,56) were conducted in the inpatient setting, while that of Klein was conducted in the outpatient setting.

Thus, excluding the study of Rifkin and associates (59), all of the inpatient studies (17,56,58) have found that lithium is effective for reducing aggression in children and adolescents with conduct disorder. However, there are no studies demonstrating efficacy in the outpatient setting. Clearly, demonstrating that lithium is effective and safe in the outpatient setting is a critical step because most patients are treated clinically as outpatients.

Long-Term Studies of Lithium

Less is known about the long-term safety and efficacy of lithium when it is used as a treatment to reduce aggression. Delong and Aldershof (68) reported on their clinical experience with 196 children and adolescents who had a variety of diagnoses and were treated with lithium for periods ranging from 1 to 10 years. Lithium was reported to be safe when used long term. One child developed hypothyroidism, which reversed when the medication was discontinued.

There is only one published long-term controlled study of lithium as a

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treatment for aggression in children with conduct disorder (69; for review, see 51). In this study, 11 children who responded to a short-term inpatient trial of lithium were randomized to 6 months of outpatient treatment with lithium or placebo under double-blind conditions. Treatment response was measured on the Children's Psychiatric Rating Scale (CPRS) and the Clinical Global Impressions and (CGI) (70). Aggressive symptoms decreased in both the lithium and placebo groups. Because of the small sample size, the results were inconclusive. Importantly, lithium was found to be safe as a long-term treatment. Obviously, more research is required to establish the efficacy and safety of lithium as a long-term treatment for aggression in children and adolescents.

Safety

In general, lithium has been a safe drug including in children and adolescents when used at the serum levels in the above-reviewed studies. The side effects of lithium in children and adolescents have been well documented (17,51,56,59,71,72). Campbell and associates (71) pooled data from three controlled studies involving 48 hospitalized children ranging in age from 5.08 to 12.92 years (mean = 9.23 years) and found that side effects were more common in the younger children. Interestingly, the half-life of lithium may be shorter in children than in adults (73).

Anticonvulsants

While these agents are not the focus of this chapter, a brief review is given to put these agents in perspective with the efficacy of lithium and neuroleptics in the treatment of aggression. There are few controlled studies regarding the use of anticonvulsants for the treatment of aggression, though drugs of this class are used in this population (74). Three anticonvulsants have been studied for their safety and effectiveness as a treatment to reduce aggression in children: diphenylhydantoin, carbamazepine, and divalproex.

Diphenylhydantoin

Early open reports with diphenylhydantoin indicated that this agent decreased aggression and other disruptive behaviors in children (75,76). However, later controlled trials found that diphenylhydantoin was no more effective than placebo (77–79) and that it may cause cognitive impairment (80). The study with the largest sample and longest treatment period was that of Lefkowitz (79). In this study, 50 males aged 13–16 years (mean = 14.11) were treated with diphenylhydantoin at a dosage of 100 mg BID for 76 days. While the authors found that there was no significant difference between the placebo and diphenylhydantoin groups on a number of measures, the placebo group actually had significantly more improvement on some measures of aggressiveness. Other controlled trials

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had either smaller samples (78) or shorter treatment periods (2 weeks) (78), but they likewise found that diphenylhydantoin was ineffective.

Carbamazepine

Carbamazepine has been used to treat a number of psychiatric conditions, including those in children (81,82). Early double-blind placebo controlled studies examining the effectiveness of carbamazepine for the treatment of aggression and disruptive behavior include those of Groh (83) and Puente (84). Though these studies reported positive results, it was unclear whether they actually reduced aggression specifically. The study of Cueva and associates (81) was designed to more critically assess the efficacy of carbamazepine in reducing aggression in children who displayed explosive aggression and were diagnosed with conduct disorder. This double-blind placebo-controlled study used parallel group design. Subjects who met a specific aggression criterion (based on the OAS) during the 2-week baseline were randomized to 6 weeks of treatment with carbamazepine or placebo. In all, 41 subjects were enrolled in the study. Of these 41 subjects, 14 did not meet the aggression criterion for randomization and were dropped from the study after baseline—again emphasizing the need to avoid commencing drug treatment soon after admission to avoid incorrectly attributing improvement to drug effect. Twenty-two children completed the study, including 20 males and two females, who ranged in age from 5.33 to 11.7 years (mean = 8.97). Optimal carbamazepine dosages ranged from 400 to 800 mg/day (mean = 683) with corresponding carbamazepine levels ranging from 4.98 to 9.1 μg/mL (mean = 6.8). Carbamazepine was not found to be significantly better than placebo for reducing aggression.

Divalproex

The anticonvulsant divalproex is labeled for the treatment of mania associated with bipolar disorder (16). Additionally, there are reports concerning the efficacy of divalproex in a number of psychiatric disorders (for review, see 85) including for the treatment of aggression (for review, see 86). However, apart from the studies on mania, there are few controlled studies examining efficacy in other psychiatric disorders. One exception is a report by Donovan and associates (87; also see Chapter 20, this volume) on divalproex for the treatment of aggression in children and adolescents. This was a double-blind placebo-controlled trial that used a crossover design. The subjects were children aged 10–18 years (mean age = 13.8 ± 2.4) diagnosed with conduct disorder or oppositional defiant disorder. Of the 20 subjects enrolled, 18 completed phase 1 and 15 completed phase 2. Subjects were randomized to treatment with either divalproex or placebo for 6 weeks of treatment and switched to the alternate treatment during the second phase of the crossover for 6 weeks. Of note, four subjects continued to receive treatment with stimulant medication during the trial. Overall, divalproex was

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found to be a significantly better treatment than placebo on several specific measures of aggression. The authors state that increased appetite was the only significant side effect but did not specify how side effects were assessed or what other side effects were encountered.

SUGGESTED GUIDELINES FOR CLINICIANS

1. The first step in considering psychopharmacologic treatments for aggression is to assess for the presence of a psychiatric disorder. A number of psychiatric disorders can have aggression as an associated symptom, including depression, mania, and schizophrenia. Treatment for the primary psychiatric disorder should be maximized before adding a treatment for aggression alone.
2. When considering a psychopharmacologic intervention for aggression, it is important to carefully document the baseline frequency and severity of aggression. Ideally, there should be at least two separate assessments before beginning drug treatment. Data from inpatient studies (58,81) indicate that despite history, aggression may not be demonstrated during a careful baseline assessment. If drug treatment is started before completing a careful assessment, a number of children will receive drug needlessly (6).
3. The findings of Molling and associates (8) suggest that some staff (and perhaps some parents) will have a strong bias that medication should be used, and, if not, they will rate the child's behavior as worsening in severity. To some degree, this makes it difficult to assess the need for treatment and for treatment effect.
4. There is no well-established drug treatment for severe aggression. In children and adolescents diagnosed with conduct disorder, the better-studied agents include the neuroleptics and lithium. However, further studies will be needed before recommending any drug(s) for standard treatment. Importantly, the safety and efficacy of polypharmacy have not been studied, and should be employed judiciously.
5. The finding that aggression seems to be reduced in the inpatient setting suggests that clinicians must be aware that environmental factors may sustain or alter aggressive behavior. Treatment should not be purely pharmacologic but should include other therapeutic approaches, including psychosocial treatments.

SUMMARY

Aggression is a difficult symptom to assess and is influenced by many factors including the environment. There are a variety of subtypes of aggression, and different subtypes may require diferrent treatments. Limited evidence suggests drug may be most efficacious in treating the explosive type of aggression. Though

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drugs may be commonly used to treat aggression, there are few studies to guide the clinician as to the efficacy and safety of these agents in children. Data suggest that the neuroleptics and lithium may be effective for treating severe aggression including that in children with conduct disorder.

REFERENCES

1. AE Kazdin. Conduct Disorders in Childhood and Adolescence, Vol 9: Developmental Clinical Psychology and Psychiatry Series. Newbury Park, CA: Sage Publications, 1987.
2. JS Werry, JP Wollersheim. Behavior therapy with children and adolescents: a twenty year overview. J Am Acad Child Adolesc Psychiatry 28:1–18, 1989.
3. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 4th ed (DSM-IV). Washington: American Psychiatric Association, 1994.
4. R Loeber. The stability of antisocial and delinquent child behavior: a review. Child Dev 53:1431–1446, 1982.
5. R Loeber. Development and risk factors of juvenile antisocial behavior and delinquency. Clin Psychol Rev 10:1–41, 1990.
6. RP Malone, DS Bennett, JF Luebbert, AB Rowan, KA Biesecker, BL Blaney, MA Delaney. Aggression classification and treatment response. Psychopharmacol Bull 34:41–45, 1998.
7. RP Malone, JF Luebbert, MA Delaney, KA Biesecker, BL Blaney, AB Rowan, M Campbell. Nonpharmacological response in hospitalized children with conduct disorder. J Am Acad Child Adolesc Psychiatry 36:242–247, 1997.
8. PA Molling, AW Lockner, RJ Sauls, L Eisenberg. Committed delinquent boys. Arch Gen Psychiatry 7:96–102, 1962.
9. RP Malone. Assessment and treatment of abnormal aggression in children and adolescents. In: M Crowner, ed. Understanding and Treating Violent Psychiatric Patients. Washington: American Psychiatric Association Press, 2000, pp 21–47.
10. JP Kemph, CL DeVane, GM Levin, R Jarecke, RL Miller. Treatment of aggressive children with clonidine: results of an open pilot study. J Am Acad Child Adolesc Psychiatry 32:577–581, 1993.
11. American Psychiatric Association Practice Guidlines. Practice guidelines for the treatment of patients with borderline personality disorder. Am J Psychiatry 158(10 suppl):1–52, 2001.
12. SS Kindermann, CR Dolder, A Bailey, IR Katz, DV Jeste. Pharmacological treatment of psychosis and agitation in elderly patients with dementia: four decades of experience. Drugs Aging 19(4):257–276, 2002.
13. SA Cohen, K Ihrig, RS Lott, JM Kerrick. Risperidone for aggression and self-injurious behavior in adults with mental retardation. J Autism Dev Disord 28(3):229–233, 1998.
14. CJ McDougle, JP Holmes, DC Carlson, GH Pelton, DJ Cohen, LH Price. A double-blind, placebo-controlled study of risperidone in adults with autistic disorder and other pervasive developmental disorders. Arch Gen Psychiatry 55:633–641, 1998.
15. LS Schneider, PN Tariot, CG Lyketsos, KS Dagerman, KL Davis, S Davis, JK

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

Hsiao, DV Jeste, IR Katz, JT Olin, BG Pollock, PV Rabins, RA Rosenheck, GW Small, B Lebowitz, JA Lieberman. National Institute of Mental Health clinical antipsychotic trials of intervention effectiveness (CATIE): Alzheimer disease trial methodology. Am J Geriatr Psychiatry 9(4):346–360, 2001.
16. Physician's Desk Reference. Montvale, NJ: Medical Economics Company, 2002.
17. M Campbell, AM Small, WH Green, SJ Jennings, R Perry, WG Bennett, L Anderson. Behavioral efficacy of haloperidol and lithium carbonate: a comparison in hospitalized aggressive children with conduct disorder. Arch Gen Psychiatry 41:650–656, 1984.
18. M Campbell, JL Armenteros, RP Malone, PB Adams, ZW Eisenberg, JE Overall. Neuroleptic-related dyskinesias in autistic children: a prospective, longitudinal study. J Am Acad Child Adolesc Psychiatry 36:835–843, 1997.
19. RP Malone, R Sheikh, JM Zito. Novel antipsychotic medications in the treatment of children and adolescents. Psychiatr Serv 50:171–174, 1999.
20. DB Allison, DE Casey. Antipsychotic-induced weight gain: a review of the literature. J Clin Psychiatry 62(suppl 7):22–31, 2001.
21. JL Armenteros, AH Whitaker, M Welikson, DJ Stedge, J Gorman. Risperidone in adolescents with schizophrenia: an open pilot study. J Am Acad Child Adolesc Psychiatry 36:694–700, 1997.
22. J Horrigan, LJ Barnhill. Risperidone and explosive, aggressive autism. J Autism Dev Disord 27:313–323, 1997.
23. DL Kelly, RR Conley, RC Love, DS Horn, CM Ushchak. Weight gain in adolescents treated with risperidone and conventional antipsychotics over six months. J Child Adolesc Psychopharmacol 8:151–159, 1998.
24. S Kumra, LK Jacobsen, M Lenane, BI Karp, JA Frazier, AK Smith, J Bedwell, P Lee, CJ Malanga, S Hamburger, JL Rapoport. Childhood-onset schizophrenia: an open-label study of olanzapine in adolescents. J Am Acad Child Adolesc Psychiatry 37:377–385, 1998.
25. PJ Lombroso, L Scahill, RA King, KA Lynch, PB Chappell, BS Peterson, CJ McDougle, JF Leckman. Risperidone treatment of children and adolescents with chronic tic disorders: a preliminary report. J Am Acad Child Adolesc Psychiatry 34:1147–1152, 1995.
26. RP Malone, J Cater, RM Sheikh, MS Choudhury, MA Delaney. Olanzapine vs. haloperidol in children with autistic disorder: an open pilot study. J Am Acad Child Adolesc Psychiatry 40:887–894, 2001.
27. RP Malone, G Maislin, MS Choudhury, C Gifford, MA Delaney. Risperidone treatment in children and adolescents with autism: short- and long-term safety and effectiveness. J Am Acad Child Adolesc Psychiatry 41:140–147, 2002.
28. MW Mandoki. Risperidone treatment of children and adolescents: increased risk of extrapyramidal side effects? J Child Adolesc Psychopharmacol 5:49–67, 1995.
29. DM Taylor, R McAskill. Atypical antipsychotics and weight gain—a systematic review. Acta Psychiatr Scand 101:416–432, 2000.
30. DA Wirshing, WC Wirshing, L Kysar, MA Berisford, D Goldstein, J Pashdag, J Mintz, SR Marder. Novel antipsychotics: comparison of weight gain liabilities. J Clin Psychiatry 60:358–363, 1999.
31. SB Schur, L Sikich, R Findling, RP Malone, ML Crismon, A Derivan, JC MacIntyre

MARCEL DEKKER, INC.
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II, E Pappadopulos, L Greenhill, N Schooler, K Van Orden, PS Jensen. Treatments for aggression in children and adolescents: a review. J Am Acad Child Adolesc Psychiatry 42(2):132–144, 2003.
32. RL Findling, NK McNamara, LA Branicky, MD Schluchter, E Lemon, JL Blumer. A double-blind pilot study of risperidone in the treatment of conduct disorder. J Am Acad Child Adolesc Psychiatry 39:509–516, 2000.
33. JK Buitelaar, RJ van der Gaag, P Cohen-Kettenis, CTM Melman. A randomized controlled trial of risperidone in the treatment of aggression in hospitalized adolescents with subaverage cognitive abilities. J Clin Psychiatry 62:239–248, 2001.
34. M Van Bellinghen, C De Troch. Risperidone in the treatment of behavioral disturbances in children and adolescents with borderline intellectual functioning: a double-blind, placebo-controlled pilot trial. J Child Adolesc Psychopharmacol 11:5–13, 2001.
35. RL Findling, MG Aman, GD D'Smedt, A Derivan. Risperidone in children with significant conduct problems and sub-average intellectual functioning. Scientific Proceedings of the 39th Annual Meeting of the American College of Neuropsychopharmacology, San Juan, Puerto Rico, 2000, p 224.
36. MG Aman, RL Findling, A Derivan, U Merriman, Conduct Group USA. Risperidone versus placebo for severe conduct disorder in children with mental retardation (abstr). 40th Annual Meeting NCDEU, Boca Raton, Poster Presentation, 2000, p 73.
37. Eichelman. Neurochemical and psychopharmacologic aspects of aggressive behavior. In: Meltzer HY, ed. Psychopharmacology: The Third Generation of Progress. New York: Raven Press, 1987, p 697–704.
38. MH Sheard. Lithium in the treatment of aggression. J Nerv Ment Dis 160:108–118, 1975.
39. GL Brown, FK Goodwin, JC Ballenger, PF Goyer, LF Major. Aggression in humans correlates with cerebrospinal fluid amine metabolites. Psychiatry Res 1:131–139, 1979.
40. GL Brown, MH Ebert, PF Goyer, DC Jimerson, WJ Klein, WE Bunney, FK Goodwin. Aggression, suicide, and serotonin: relationships to CSF amine metabolites. Am J Psychiatry 139:741–746, 1982.
41. EF Coccaro, LJ Siever, HM Klar, G Maurer, K Cochrane, TB Cooper, RC Mohs, KL Davis. Serotonergic studies in patients with affective and personality disorders. Correlates with suicidal and impulsive aggressive behavior. Arch Gen Psychiatry 46:587–599, 1989.
42. EF Coccaro, RJ Kavoussi, YI Sheline, JD Lish, JG Csernansky. Impulsive aggression in personality disorder correlates with tritiated paroxetine binding in the platelet. Arch Gen Psychiatry 53:531–536, 1996.
43. M Linnoila, M Virkkunen, M Scheinin, A Nuutila, R Rimond, FK Goodwin. Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from non-impulsive violent behavior. Life Sci 33:2609–2614, 1983.
44. MJ Kruesi, JL Rapoport, S Hamburger, E Hibbs, WZ Potter, M Lenane, GL Brown. Cerebrospinal fluid monamine metabolites, aggression, and impulsivity in disruptive behavior disorders of children and adolescents. Arch Gen Psychiatry 47:419–426, 1990.
45. DM Stoff, L Pollock, B Vitiello, D Behar, WH Bridger. Reduction of (3)H-imipra-

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mine binding sites on platelets of conduct-disordered children. Neuropsychopharmacology 1:55–62, 1987.
46. EF Coccaro, LJ Siever. The neuropsychopharmacology of personality disorders. In: FE Bloom, DJ. Kupfer, eds. Psychopharmacology: The Fourth Generation of Progress. New York: Raven Press, 1995, pp 1567–1579.
47. JJ Mann. Violence and aggression. In: FE Bloom, DJ Kupfer, eds. Psychopharmacology: The Fourth Generation of Progress. New York: Raven Press, 1995, pp 1919–1928.
48. MH Sheard, GK Aghajanian. Neuronally activated metabolism of brain serotonin: effect of lithium. Life Sci 9:285–290, 1970.
49. MH Sheard. Effect of lithium on human aggression [letter]. Nature 230:113–114, 1971.
50. RJ Baldessarini, M Vogt. Release of ^{3}H-dopamine and analogous monoamines from rat striatal tissue. Cell Mol Neurobiol 8:205–216, 1988.
51. M Campbell, V Kafantaris, JE Cueva. An update on the use of lithium carbonate in aggressive children and adolescents with conduct disorder. Psychopharm Bull 31:93–102, 1995.
52. JFJ Cade. Lithium salts in the treatment of psychotic excitement. Med J Aust 36: 349–352, 1949.
53. MH Sheard, JL Marini, CI Bridges, E Wagner. The effect of lithium on impulsive aggressive behavior in man. Am J Psychiatry 133:1409–1413, 1976.
54. P Links, M Steiner, I Boiago, D Irwin. Lithium therapy for borderline patients: preliminary findings. J Personal Discord 4:173–181, 1990.
55. A Rifkine, F Quitkin, C Carrillo, AG Blumberg, DF Klein. Lithium carbonate in emotionally unstable character disorder. Arch Gen Psychiatry 27(10):519–523, 1972.
56. M Campbell, PB Adams, AM Small, V Kafantaris, RR Silva, J Shell, R Perry, JE Overall. Lithium in hospitalized aggressive children with conduct disorder: a double-blind and placebo-controlled study. J Am Acad Child Adolesc Psychiatry 34:445–453, 1995.
57. RG Klein. Preliminary results: lithium effects in conduct disorders. CME Syllabus and Proceedings Summary, Symposium 2. The 144th Annual Meeting of the American Psychiatric Association, New Orleans, 1991, pp 119–120.
58. RP Malone, MA Delaney, JF Luebbert, J Cater, M Campbell. A double-blind placebo-controlled study of lithium in hospitalized aggressive children and adolescents with conduct disorder. Arch Gen Psychiatry 57:649–654, 2000.
59. A Rifkin, B Karajgi, R Dicker, E Perl, V Boppana, N Hasan, S Pollack. Lithium treatment of conduct disorders in adolescents. Am J Psychiatry 154:554–555, 1997.
60. RP Malone, J Luebbert, M Pena-Ariet, K Biesecker, MA Delaney. The Overt Aggression Scale in a study of lithium in aggressive conduct disorder. Psychopharmacol Bull 30:215–218, 1994.
61. SC Yudofsky, JM Silver, W Jackson, J Endicott, D Williams. The Overt Aggression Scale for the objective rating of verbal and physical aggression. Am J Psychiatry 143:35–39, 1986.
62. RP Malone, GM Simpson. Use of placebos in clinical trials involving children and adolescents. Psychiatr Serv 49:1413–1415, 1998.

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63. W Guy. ECDEU Assessment manual for psychopharmacology-revised (DHEW Publ No. ADM-76-338). Rockville, MD: U.S. Department of Health, Education and Welfare, 1976.
64. TB Cooper, PE Bergner, GM Simpson. The 24-hour serum lithium level as a prognosticator of dosage requirements. Am J Psychiatry 130:601–603, 1973.
65. TB Cooper, GM Simpson. The 24-hour serum lithium level as a prognosticator of dosage requirements: a 2-year follow-up study. Am J Psychiatry 133:440–443, 1976.
66. RP Malone, MA Delaney, J Luebbert, M White, KA Biesecker, TB Cooper. The lithium test dose prediction method in aggressive children. Psychopharmacol Bull 31:379–381, 1995.
67. JR Calabrese, C Bowden, MJ Woyshville. Lithium and the anticonvulsants in the treatment of bipolar disorder. In: FE Bloom, DJ Kupfer, eds. Psychopharmacology: The Fourth Generation of Progress. New York: Raven Press, 1995, pp 1099–1111.
68. GR Delong, AL Aldershof. Long-term experience with lithium treatment in childhood: correlation with clinical diagnosis. J Am Acad Child Adolesc Psych 26:389–394, 1987.
69. RR Silva, NM Gonzalez, V Kafantaris, M Campbell. Long-term use of lithium in aggressive conduct disorder children (abstr). Scientific Proceedings. 38th Annual Meeting of the American Academy of Child and Adolescent Psychiatry, San Francisco, 1991, p 74.
70. National Institute of Mental Health. Special feature: rating scales and assessment instruments for use in pediatric psychopharmacology research. Psychopharmacol Bull 21, 1985.
71. M Campbell, RR Silva, V Kafantaris, JJ Locascio, NM Gonzalez, D Lee, NS Lynch. Predictors of side effects associated with lithium administration in children. Psychopharmacol Bull 27:373–380, 1991.
72. RR Silva, M Campbell, RR Golden, AM Small, CS Pataki, CR Rosenberg. Side effects associated with lithium and placebo administration in aggressive children. Psychopharmacol Bull 28:319–326, 1992.
73. B Vitiello, D Behar, P Ryan, R Malone, MA Delaney, PJ Ryan, G Simpson. Pharmacokinetics of lithium in pre-pubertal children. J Clin Psychopharmacol 8:355–359, 1988.
74. DF Conner, KR Ozbayrak, RH Harrison, R Melloni. Prevalence and patterns of psychotropic and anticonvulsant medication use in children and adolescents referred to residential treatment. J Child Adolesc Psychopharmacol 8:27–38, 1998.
75. B Pasamanick. Anticonvulsant drug therapy of behavior problem children with abnormal electroencephalograms. Arch Neurol Psychiatry 65:752–766, 1951.
76. CF Walker, BB Kirkpatrick. Dilantin treatment for behavior problem children with abnormal electroencephalograms. Am J Psychiatry 103:484–492, 1947.
77. A Looker, CK Conners. Diphenylhydantoin in children with severe temper tantrums. Arch Gen Psychiatry 23:80–89, 1970.
78. CK Conners, R Kramer, GH Rothschild, L Schwartz, A Stone. Treatment of young delinquent boys with diphenylhydantoin sodium and methylphenidate. Arch Gen Psychiatry 24:156–160, 1971.

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79. MM Lefkowitz. Effects of diphenylhydantoin on disruptive behavior, study of male delinquents. Arch Gen Psychiatry 20:643–651, 1969.
80. SS Dikmen, NR Temkin, B Miller, J Machamer, R Winn. Neurobehavioral effects of phenytoin prophylaxis of posttraumatic seizures. JAMA 265:1271–1277, 1991.
81. JE Cueva, JE Overall, AM Small, JL Armenteros, R Perry, M Campbell. Carbamazepine in aggressive children with conduct disorder: a double-blind and placebo-controlled study. J Am Acad Child Adolesc Psychiatry 35:480–490, 1996.
82. H Remschmidt. The psychotropic effect of carbamazepine in non-epileptic patients, with particular reference to problems posed by clinical studies in children with behavioural disorders. In: W Birkmayer, ed. Epileptic Seizures–Behaviour–Pain. Bern, Switzerland: Hans Huber, 1976, pp 253–258.
83. C Groh. The psychotropic effect of tegretol in non-epileptic children, with particular reference to the drug's indications. In: W. Birkmayer, ed. Epileptic Seizures–Behaviour–Pain. Bern, Switzerland: Hans Huber, 1976, pp 259–263.
84. RM Puente. The use of carbamazepine in the treatment of behavioural disorders in children. In: W Birkmayer, ed. Epileptic Seizures–Behaviour–Pain. Bern, Switzerland: Hans Huber, 1976, pp 243–247.
85. LL Davis, W Ryan, B Adinoff, F Petty. Comprehensive review of the psychiatric uses of valproate. J Clin Psychopharmacol 20(suppl 1):1S–17S, 2000.
86. JP Lindenmayer, A Kotsaftis. Use of sodium valproate in violent and aggressive behaviors: a critical review. J Clin Psychiatry 61:123–128, 2000.
87. SJ Donovan, JW Stewart, EV Nunes, FM Quitkin, M Parides, W Daniel, E Susser, DF Klein. Divalproex treatment for youth with explosive temper and mood lability: a double-blind, placebo-controlled crossover design. Am J Psychiatry 157:818–820, 2000.
88. P Barker, IA Fraser. A controlled trial of haloperidol in children. Br J Psychiatry 114:855–857, 1968.
89. MA Cunningham, V Pillai, WJ Rogers. Haloperidol in the treatment of children with severe behaviour disorders. Br J Psychiatry 114:845–854, 1968.
90. LL Greenhill, M Solomon, R Pleak, P Ambrosini. Molindone hydrochloride treatment of hospitalized children with conduct disorder. J Clin Psychiatry 46(8 pt 2): 20–25, 1985.

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19

Treatment of Aggression

Serotonergic Agents

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INTRODUCTION

This chapter focuses on psychopharmacological treatments of impulsive aggression with a serotonergic mechanism of action. The psychopharmacological treatment of impulsive, rather than premeditated, aggression will be the focus of this discussion because of the preponderance of evidence liking serotonergic dysfunction to impulsive, rather than premeditated, aggression (1).

Treatment studies to date have focused on impulsive aggression as a dimensional, as opposed to a categorical, variable. This is because no diagnostic category exists that can reliably include individuals with impulsive aggression. Current DSM-IV criteria for intermittent explosive disorder (IED), for example, would exclude many antisocial and borderline personality disordered individuals who would otherwise meet criteria for IED. Although not all individuals with borderline personality disorder would meet revised research criteria for IED (IED-R: criteria that do not exclude borderline or antisocial personality disorder; see Chap. 9), many do meet such criteria (2). Thus, treatment studies have included subjects with antisocial and borderline personality disorder in addition to subjects who would meet criteria

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for DSM-IV IED. This is consistent with data from pharmacochallenge and neuroimaging studies that have found impulsive aggression correlated with serotonergic abnormalities regardless of the presence of any particular axis II personality disorder (1,3). Note that impulsive aggression is encountered in other patient populations, including affective disorders, psychotic disorders, mental retardation, brain trauma, and mental retardation, and some attention will be given to these patient populations where evidence is available.

SEROTONERGIC SYSTEM AND RECEPTOR SUBTYPES

There are two central 5-HT subsystems: a rostral division with cell bodies in the midbrain and rostral pons with connections to the forebrain, and a caudal division in the medulla oblongata with descending connections to the spinal cord and brainstem nuclei (4). 5-HT is synthesized through a two-step process from the essential amino acid tryptophan. The rate-limiting step in this process is the availability of tryptophan to be converted to 5-hydroxytryptophan (5-HTP) by tryptophan hydroxylase; 5-HTP is then rapidly dehydroxylated to 5-HT. 5-HT is then incorporated into presynaptic vesicles by the vesicular monoamine transporter type-2 and stored until its release. Cell depolarization induces the opening of voltage-sensitive calcium channels and Ca^{2+} entry into the cell. The intracellular Ca^{2+} increase links depolarization and exocytotic secretion. The amount of serotonin released is subject to receptor-dependent regulation. After being released into the extracellular space, or synapse, 5-HT binds to postsynaptic receptors and presynaptic receptors. A general description of these receptors is provided in the next paragraph.

Serotonin in the synapse must be removed to be inactivated. The serotonin transporter is responsible for the reuptake of serotonin from the synapse to enable its intracellular breakdown. Inside the cell, the enzyme monoamine oxidase-A catalyzes the breakdown of 5-HT. Although many 5-HT subtypes are present in the human brain, the $5\text{-}HT_{1A}$, $5\text{-}HT_{2A}$, and $5\text{-}HT_{2C}$ receptors have been best described in relationship to the mechanism of antidepressant drug action and the neuropsychopharmacology of impulsive aggression. $5\text{-}HT_{1A}$ receptors can be pre- or post-synaptic. Presynaptic $5\text{-}HT_{1A}$ receptors are located on the soma and dendrites of 5-HT neurons in the dorsal and medial raphe nuclei. Activation reduces the firing rate of serotonergic neurons and suppresses 5-HT synthesis, turnover, and release. Hence the presynaptic $5\text{-}HT_{1A}$ receptor plays an important role as a serotonergic autoreceptor (5). Postsynaptic $5\text{-}HT_{1A}$ receptors are found in the terminal fields of 5-HT neurons in the hippocampus, lateral septum, cortex, amygdala, and hypothalamus.

Stimulation of postsynaptic $5\text{-}HT_{1A}$ receptors leads to the release of neurohormones in response to serotonergic agents such as m-CPP and ipsapirone. The $5\text{-}HT_{1A}$ receptors differ from most of the other 5-HT receptor subtypes in that

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they are more similar to adrenergic receptors. This explains their cross-reactivity with certain β-adrenergic blockers such as pindolol. 5-HT_{2A} receptors are postsynaptic G protein–linked receptors. Their highest density is in the neocortex, but they are widely distributed in the brain. 5-HT_{2A} receptors have activating effects on phosphoinositide hydrolysis, c-AMP-response element-binding protein and brain-derived neurotrophic factor (BDNF). Many drugs with 5-HT_{2A} agonist properties have euphoria-inducing effects. This mood-altering property may represent one component of the antidepressant activity of serotonergic agents such as selective serotonin reuptake inhibitors (SSRIs) (6). The 5-HT_{2C} receptor is also a postsynaptic G protein–linked receptor that activates guanylyl cyclase and phosphoinosidtide hydrolysis (7). The 5-HT_{2C} receptor may mediate the prolactin response to fenfluramine, which has been found to be negatively correlated with measures of impulsive aggression (1).

SEROTONIN AND IMPULSIVE AGGRESSION

The relationship between abnormalities in serotonergic function and impulsive aggression is described elsewhere so will be reviewed only briefly. Deficits in serotonergic function have been associated with suicide in depressives (8); aggression toward self and others (9); impulsive, rather than premeditated aggression (10); self-injurious behavior in depressives (11); and self-injurious behavior in personality-disordered subjects (12). Neuropsychopharmacological challenge studies with the serotonergic agent fenfluramine (both D,l- and D-stereoisomer forms) have found blunted prolactin responses in association with impulsive aggression, implicating reduced serotonergic function, in borderline (1) and antisocial personality-disordered subjects (13). Using PET in borderline personality disordered subjects with histories of impulsive aggression, Siever and associates (3) found blunted metabolic responses to d,l-fenfluramine in orbital frontal, adjacent medial, and cingulate cortex when compared to matched controls, findings that were partially replicated by Soloff and associates (14).

These brain regions rely heavily on serotonergic neurotransmission and are also implicated in the functional neurobiology of major depressive disorder. Hypofunction in these regions induced by a reduction in available serotonin may predispose persons with affective disorders to relapse. This overlap of abnormalities in highly serotonergic brain regions may partially explain the frequent comorbidity of mood and impulsive aggressive personality disorders by a shared dysfunction of prefrontal serotonin function.

Serotonergic Agents

Currently available serotonergic agents may be divided into three major categories based on their primary effect on brain serotonin function: 1) direct and indirect acting 5-HT receptor agonist/antagonists such as buspirone and pindolol; 2)

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monoamine oxidase (MAO) inhibitors, which interfere with the breakdown of 5-HT; and 3) inhibitors of 5-HT reuptake such as the serotonin reuptake inhibitors. Evidence for the efficacy and hypothesized mechanism of action of each category will be presented next.

5-HT_{1A} Partial Agonists

Mechanism of Action. Sustained administration of 5-HT_{1A} partial agonists is associated with desensitization of 5-HT_{1A} autoreceptors (15). This process may be responsible for the antianxiety and antidepressant actions of 5HT_{1A} partial agonists. Desensitization of 5-HT_{1A} autoreceptors has been shown to preferentially affect the somatodendritic autoreceptor over the postsynaptic receptor (16,17). Desensitization has been demonstrated as early as three days into treatment. The percentage of desensitized neurons reaches 60–80% at $\sim$ 21 days, which parallels the time of onset of antidepressant effect (18). Imaging studies suggest that pindolol is relatively selective for 5-HT_{1A} autoreceptors in the dorsal raphe nuclei compared to 5-HT_{1A} receptors in corticolimbic areas (19). This preferential occupancy might stem from the presence of more 5-HT_{1A} receptors in the dorsal raphe neulei being configured in a high affinity state. As a partial agonist, pindolol is sensitive to the state of the receptor and is modulated by aspects of second messenger systems such as G protein coupling (20) and the presence of GTP or its analog guanylylimidodiphosphate (21). PET studies of 5-HT_{1A} occupancy reveal that at doses used in treatment trials of depression of pindolol (as an augmenting agent: 2.5 mg TID), pindolol occupancy of 5-HT_{1A} receptors is only moderate and is highly variable (19). This would suggest that higher doses than 2.5 mg TID would be necessary to effectively block the 5-HT_{1A} autoreceptor, although higher doses would also incur more side effects due to effects on other receptor systems. Buspirone, also, acts as a partial agonist at the 5-HT1A receptor (22). Prolactin response to buspirone is reduced in impulsive aggressive personality disordered subjects compared to controls (23) and violent compared to nonviolent parolees (24). While these findings most likely reflect bupsirone's properties as a 5-HT probe, a relationship among these variables and buspirone's antidopaminergic activity cannot be ruled out.

Treatment Studies. Neither pindolol nor buspirone has been tested in controlled trials in the treatment of impulsive aggression in personality disordered subjects. However, two controlled studies of pindolol have been reported in different patient groups. A double-blind study of the effect of pindolol on behavioral disturbances in patients with organic brain disease found that treatment with 40–60 mg/day pindolol was associated with significant decreases in assaultive behavior as documented by a decrease in the frequency of incident reports of assault or attempted assault.

Hostility, as measured by a clinical, behavioral rating was also significantly reduced. These effects were not due to generalized sedation, as no effect was

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seen on ratings of lethargy, and positive changes were noted for measures of communicativeness and uncooperativeness. The doses used in this study were not associated with hypotension or orthostasis (25). Pindolol's beneficial effect on assaultive behavior in this study may have been secondary to either β-adrenergic blockade and/or serotonin 5-HT_{1A} effects. A double-blind crossover randomized study of pindolol in thirty male schizophrenic inpatients with histories of aggressive behavior found that treatment with pindolol (5 mg TID) was associated with reductions in Overt Aggression Scores (OAS) scores for number of aggressive incidents against objects and others. No significant change in PANSS scores were found, indicating that the antiaggressive effect occurred independently of major effects on psychosis. Concomitant neuroleptic medications were maintained at steady doses throughout the study. OAS scores were reduced in frequency of aggressive incidents towards objects and other persons during pindolol treatment (0.59 vs. 1.46 and 1.96 vs. 3.23). OAS scores were also significantly reduced in severity of incidents (0.89 vs. 3.58). Three patients were discontinued due to syncope, low blood pressure, or bronchospasm (26).

No controlled trials have been conducted with buspirone in the treatment of impulsive aggression. A single open trial in 25 prepubertal anxious and moderately aggressive inpatients found that 6 weeks of maintenance treatment did not lead to a clear pattern, with four subjects dropping out of the study owing to clinically increased aggressive behavior. Only three subjects improved sufficiently to continue buspirone after the study, although in the group as a whole, there was a significant reduction in scores on the Measure of Aggression, Violence, and Rage in Children (27). Case reports suggest efficacy in mentally retarded subjects but double-blind placebo-controlled studies are absent (28,29).

Monoamine Oxidase Inhibitors

Mechanism of Action. Acute administration of selective MAO-A or nonselective MAO-A/MAO-B inhibitors produces an immediate inhibition of 5-HT catabolism leading to an increase in 5-HT available for release. The subsequent increase in extraceullular 5-HT results in an activation of 5-HT_{1A} autoreceptors (30,31) and a reduction in 5-HT neuron firing activity (32). With prolonged administration of MAOIs, however, brain 5-HT is increased after sustained blockade of MAO-A (33). Following sustained administration MAOIs, desensitization of 5-HT_{1A} autoreceptors occurs, causing a decrease in the inhibitory influence of autoreceptors on 5-HT neuron firing activity. After 14–21 days of treatment, 5-HT neurons resume their normal firing frequency (4) and intracellular availability of 5-HT increases (32), possibly owing to desensitization of 5-HT_{1A}-mediated inhibition of adenylyl $cyclase_{34}$, or desensitization of G protein function (35).

Treatment Studies. MAO inhibitors (MAOIs), useful in the treatment of atypical depression, have been studied in personality disorder patients with the hopes that they would be able to address some of the atypical mood symptoms

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such as the mood reactivity of histrionic personality disorder, rejection sensitivity in dependent and avoidant personality disorder, and depressive symptoms in borderline personality disorder. The serotonergic effects of the MAO inhibitors also make them candidates for the treatment of impulsive-aggressive behavior.

Early reports suggested that MAOIs might be effective for a range of symptoms in persons with Cluster B personality disorders (36,37). Cowdry and Gardner's (38) double-blind, placebo-controlled crossover comparison of tranylcypromine (40 mg/day), alprazolam, and carbamazepine found efficacy in mood symptoms in a group of treatment-resistant subjects with borderline personality disorder. These subjects had significant histories of behavioral dyscontrol, and did not have comorbid depressive disorder. Although improvement was seen in observer and self-rated suicidality and impulsivity, this was not matched by a corresponding decrease in proneness to behavioral dyscontrol. The authors concluded that the improvement seen was multidimensional and mood related, with little or no effect on impulsivity.

In a double-blind, placebo-controlled comparison of phenelzine and haloperidol in DSM-III-R borderlines, Soloff and associates found phenelzine 60 mg/day to be modestly effective for symptoms of depression, anxiety, and hostility compared to placebo. Hostility was measured by the Buss-Durkee Hostility Inventory total score (39). The magnitude of change for all arms of the study were relatively low, leaving the patients still substantially impaired at the end. In a 16-week continuation study, 38 patients received phenelzine, 36 patients received haloperidol, and 34 received placebo (40). Phenelzine demonstrated minor efficacy in comparison to placebo for treatment of the mood symptoms of irritability as rated by the Buss-Durkee Hostility Inventory. This small effect occurred without improvement in atypical depressive symptoms. In these subjects, phenelzine had an activating effect, showing increased, rather than decreased mood reactivity, although the authors note that activation was an improvement in the overall well-being of the patients suffering from anergia, and did not represent an increase in impulsive aggression. These small improvements in irritability over long-term treatment must be weighted against the weight gain and risk of hypertensive crisis.

Selective Serotonin Reuptake Inhibitors

Mechanism of Action

Selective serotonin-reuptake inhibitors (SSRIs) increase synaptic 5-HT concentrations acutely by binding to the 5-HT transporter and inhibiting presynaptic 5-HT uptake (41). Feedback inhibition due to increased 5-HT levels (41) on both presynaptic terminal and somatic-dendritic 5-HT autoreceptors initially prevents increased "net" 5-HT neurotransmission. Increased "net" 5-HT neurotransmission does not occur until weeks later, when receptor downregulation and desensi-

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tization of both somatodendritic and terminal autoreceptors occurs (16,42), possibly owing to desensitization of 5-HT_{1A}-mediated inhibition of adenylyl cyclase (34) and sensitization of G protein function (35,43). Downregulation of the terminal autoreceptor function leads to an increase in the release of 5-HT per nerve impulse, a function that is regulated by the terminal autoreceptor (44). At 14–21 days of treatment, serotonergic neurons resume their normal firing frequency (4,45).

Fluoxetine's receptor binding profile has been studied with respect to 5-$HT_{1A/-2A/-2C}$ and other non-5-HT receptors. Of all receptors studied, the only receptor within an order of magnitude of fluoxetine's affinity for the 5-HT transporter is the 5-HT_{2C} receptor (46). Fluoxetine functions as an antagonist at 5-HT_{2C} receptors (47,48). This suggests that 5-HT_{2C} receptor antagonism may play a relevant role in the therapeutic effects of fluoxetine. However, while chronic (though not acute) fluoxetine reduces 5-HT_{2C}-mediated behavioral effects in animal studies (49,50), this effect is more likely due to the global increase of synaptic 5-HT associated with 5-HT transporter inhibition by fluoxetine rather than blockade of 5-HT_{2C} receptors by fluoxetine. This is because this effect is also seen with other SSRIs such as paroxetine (50) that have little affinity for the 5-HT_{2C} receptor (46). Fluoxetine's direct effects on other 5-HT receptors are less clear, and receptor binding for the other 5-HT receptors may be increased, decreased, or unchanged depending on the study (46). One recent study (51) notes no change in 5-$HT_{1A/B}$ binding sites but reductions in 5-HT_{1A} mRNA levels in the anterior raphe and increased 5-HT_{1B} mRNA levels in the striatum and cerebral cortex of rats, indicating that changes in receptor function may occur on a transcriptional level.

Imaging studies of patients treated with SSRIs for major depressive disorder give additional clues regarding the regional brain effect of these agents. To examine the effect of paroxetine treatment on 5-HT_{2A} receptor function in depressives, Meyer and associates (6) used the high-affinity and high-specificity 5-HT_{2A} antagonist [^{18}F] setoperone. Nineteen patients scanned with [^{18}F] setoperone PET before and after treatment with paroxetine were matched to 19 age-matched healthy subjects. In patients under the age of 30, decreases in 5-HT_{2A} receptor binding potential were found globally in the cortex. These decreases attenuated with increasing age, and were not correlated with degree of improvement of depressive symptoms. Since paroxetine does not have significant 5-HT_{2A} antagonist properties, these changes in 5-HT_{2A} receptor function were not due to direct action on the receptor by paroxetine (52). Downregulation in receptor binding could be attributed to either decreased receptor density or decreased affinity. Evidence from ex vivo studies suggests that changes in receptor density may account for decreased receptor binding seen with long term agonist administration (53). These results are consistent with paroxetine causing a global increase in serotoninergic activity on the postsyntaptic 5-HT_{2A} receptor.

In a PET study of depressives examining changes in brain metabolism be-

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fore, at 1 week, and at 6 weeks treatment with fluoxetine, improvement in responders was associated with a reciprocal pattern of limbic-paralimbic decreases (subgenual cingulate [BA 25] cortex, hippocampaus, insula) and brainstem and dorsal cortical increases (prefrontal [Bas 46/9], anterior cingulate [BA 24b], inferior parietal [BA 40], and posterior cingulate [Bas 31/23] cortices (54). These changes represented correction of pretreatment cortical abnormalities as well as suppression of pretreatment levels of activity in limbic and paralimbic brain regions (subgenual cingulate and hippocampus). These findings overlap partially with results of a PET study examining regional brain metabolism before and after extended paroxetine treatment in depressed subjects. Successful antidepressant effect of paroxetine was associated with increases in metabolic activity in dorsolateral, ventrolateral, and ventral prefrontal areas, as well as dorsal medial prefrontal, anterior cingulate, and inferior parietal regions, predominantly on the left side. Decreases in metabolism were found in the right hippocampus and parahippocampus, and left and right anterior insula. Results from these and other regional brain imaging studies are of interest because they demonstrate that 5-HT uptake inhibitors may have effects on regional brain function in depressives. Their significance remains unclear, however, given that studies have produced different findings. In addition, available evidence does not establish that these changes in regional brain metabolism are the direct effect of the administration of serotonin reuptake inhibitors given that the presence of major depression is itself associated with abnormalities in prefrontal cortex function. However, they are of interest here because these same brain regions are part of the emotional circuit hypothesized to underlie impulsive aggression (anterior cingulate cortex, orbitofrontal cortex, dorsolateral prefrontal cortex) (55) and to overlap with regions found to be hypoactive after administration of the serotonin releasing agent fenfluramine in subjects with impulsive aggression (3).

Treatment Studies

Preliminary evidence that SSRIs are effective in reducing impulsive aggression in personality-disordered patients comes from several open trials of fluoxetine (56–59) and one of sertraline (60). In the open trial of sertraline, marked improvement occurred in both overaggression and irritability as measured by the OAS-M, but improvement in overt aggression showed improvement beginning at week 2 while mean OAS-M irritability scores were not significantly lower than baseline until week 4. Although this study did not have a placebo control group, the pattern of delayed improvement is consistent with that seen in controlled treatment trials of depression and the placebo-controlled trials described below (see Chap. 7 for a detailed discussion on the treatment of depressed patients with "anger attacks," this volume). Impulsive aggression did not increase in any of the open or controlled trials conducted.

Salzman and associates (61) compared fluoxetine (mean dose 40 mg/day)

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to placebo in a 13-week double-blind study of subjects with borderline disorder diagnosed by DSM-III-R criteria. Patients in this study were mildly to moderately symptomatic volunteers with no history of psychiatric hospitalization, recent suicidal behavior, or self-mutilation, limiting the generalizability of findings to less severely impaired individuals. Significant improvement was found in anger and depression on the Profile of Mood States (POMS). There were trends for improvement in OAS and Hamilton Depression Inventory (HAM-D) scores. Computation of odds ratios for PDRS measures of anger and depression revealed that patients receiving fluoxetine were seven times as likely to have decreased anger compared with patients receiving placebo.

Coccaro and Kavoussi (62), in a double-blind, placebo-controlled trial of fluoxetine (20–60 mg), treated 40 nondepressed DSM-III-R personality-disordered individuals with impulsive aggressive behavior with fluoxetine or placebo. To ensure that subjects had trait problems with impulsive aggressive behavior, subjects were required to score high scores on at least one of the Anger (Irritability or Labile Anger) and at least one of Aggression (Direct Physical, Indirect Physical, and Verbal) subscales of the self-report Anger, Irritability, and Aggression Questionnaire (AIAQ) for their lifetime as an adult. The study found significant decreases in Observed Aggression Scale-Modified (OAS-M) irritability beginning at week 6, and OAS-M verbal aggression and aggression against objects subscale decreases between weeks 10 and 12 of treatment and for all subjects at endpoint (Fig. 1). Curiously, the effect of fluoxetine was mainly on verbal aggression and aggression against objects, with most subjects responding at in the 20–40 mg/day dose range. Physical aggression against others was too rare to analyzed in this study. Consistent with other treatment studies of impulsive aggression (63,68), no significant subjective improvement was found, indicating that patient's perception of their aggressiveness may differ from that of their observers. No patients worsened while on fluoxetine. Patients in the study had an average of 1.8 ± 1.1 axis II personality disorders, with the three most common diagnoses being borderline personality disorder (33%), paranoid personality disorder (25%), and obsessive-compulsive personality disorder (23%).

Results from these two controlled studies are consistent with the notion that serotonergic dysfunction contributes to traitlike impulsive aggression. Laboratory measures of impulsive aggressive behavior (Point Subtraction Aggression Paradigm) respond accordingly to paroxetine administration (64) and manipulation of the serotonin precursor tryptophan (65). If SSRIs in treatment responders were ameliorating a "serotonin deficit," it might be hypothesized that patients with abnormalities in peripheral measures of serotonergic function would be the most likely to respond to fluoxetine. However, in the study by Coccaro and Kavoussi (62), 10 of the 14 patients who completed 12 weeks of treatment with fluoxetine had undergone a d-fenfluramine challenge prior to the start of the treatment trial (66). Among these subjects, a statistically significant positive correlation was

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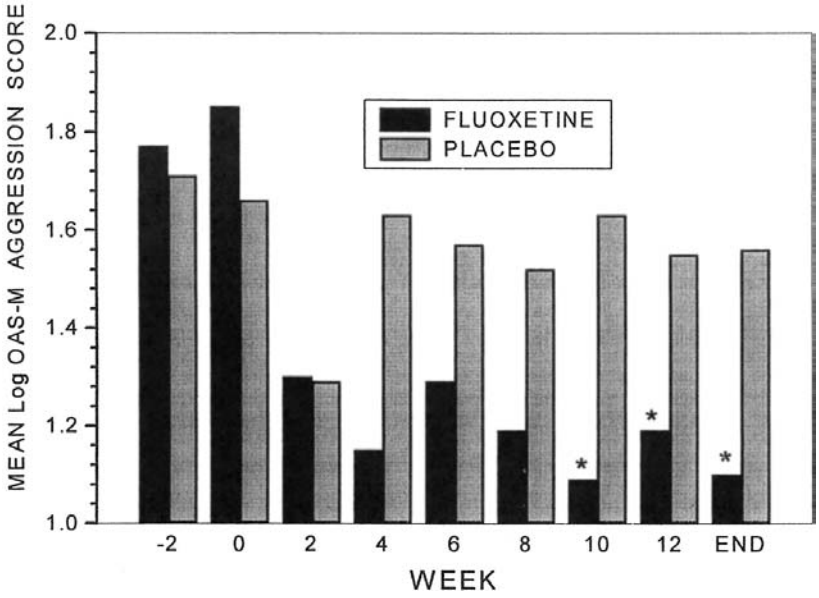

FIGURE 1 Visitwise mean OAS-M scores (log transformed) for IED subjects randomized to fluoxetine or placebo. Study began with 27 subjects randomized to fluoxetine (14 by week 12) and 13 subjects randomized to placebo (9 by week 12). (See Ref. 62 for more details.)

observed between the pre-treatment prolactin response to d-fenfluramine challenge (PRL [d-FEN]) and the percent improvement in the Overt Aggression Scale-Modified (OAS-M) score. Subjects with smaller physiological responses to the 5-HT challenge pretreatment showed less improvement with SSRI treatment. Accordingly, these results suggest that impulsive aggressive subjects with the most severe 5-HT abnormalities may be the least likely to benefit from SSRIs. This finding may be due to blunted 5-HT_{2C} receptor responsiveness and/or reduced presynaptic availability of 5-HT, both of which would render 5-HT uptake inhibitors less potentially effective. Similarly, it is possible that the most severely aggressive individuals (who, according to the 5-HT hypothesis of aggression, should have the greatest 5-HT deficit) may be less responsive to treatment with SSRIs. Support for this hypothesis is provided in a recent reanalysis of these data. In this analysis, subjects were divided into "highly aggressive" (i.e., Life History of Aggression [LHA] scores >17) and "moderately aggressive" (i.e., LHA scores ≤ 17) subjects and the treatment outcome data was revisited. Fluoxetine efficacy was only present in "moderately aggressive" subjects and not at all

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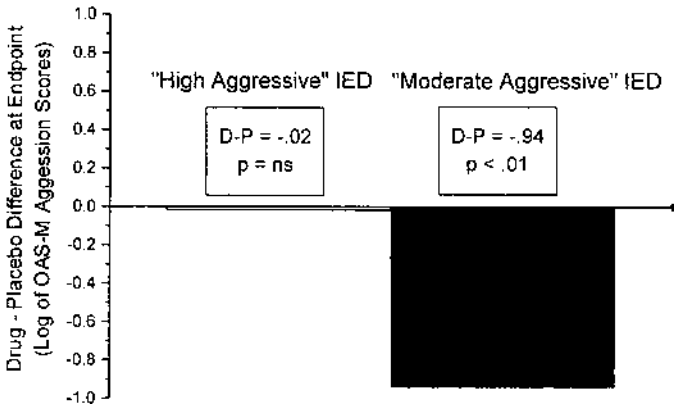

FIGURE 2 Drug-placebo difference in OAS-M Aggression score at endpoint in "highly aggressive" vs. "moderately aggressive" IED subjects.

present in "highly aggressive" subjects (Fig. 2). If so, SSRI agents may be less effective in such subjects. Other agents (e.g., mood stabilizers; see Chaps. 18 and 20) may need to be considered to treat the impulsive aggressive behavior manifested by individuals who are "highly aggressive" over their lifetime.

OTHER ANTIDEPRESSANTS

Given that other neurotransmitter abnormalities have been found in subjects with impulsive aggression—e.g., reduced CSF levels of catecholamines metabolites (9,10)—it follows that antidepressants with less specifically serotonergic mechanisms of action may be helpful in the treatment of impulsive aggression. Evidence from treatment trials of borderline personality-disordered patients, however, suggests that tricyclic antidepressants may worsen impulsive aggression in a subgroup of patients with histories of aggression. In a 5-week controlled study of hospitalized borderline and schizotypal personality disordered patients, Soloff and associates (67) found modest beneficial effects on mood with amitriptyline treatment compared to placebo. Notably, a subgroup of patients on amitriptyline showed increases in ratings of impulsive and aggressive behavior. These patients were characterized as having initially higher levels of aggression, psychoticism, negativism, impulsivity, and schizotypal symptoms. At least two other studies (68,69) also reported that a subgroup of patients with borderline or schizotypal personality disorder worsened after treatment with tricyclic antidepressants with respect to suicidality and physical assaultiveness. This may be due to the norad-

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renergic actions of these agents, which could increase the likelihood of acting on aggressive impulses or increase their intensity.

No controlled studies are yet available on new, "atypical" antidepressants such as mirtazepine, nefazadone, venlafaxine, or buproprion in the treatment of impulsive aggression. These agents, which also have effects on norepinephrine, may hypothetically have tricycliclike activating effects in patients with impulsive aggression, although empirical evidence for this is lacking.

CONCLUSION

Evidence for the efficacy of the treatment of impulsive aggression with serotonergic agents is consistent with biological studies finding serotonergic deficits in impulsive and aggressive personality disorder subjects. Neuroimaging studies in subjects with major depression suggest that serotonin reuptake inhibitors alter regional brain metabolism in areas such as the prefrontal cortex, and that these changes are correlated with symptomatic improvement in mood. While results from studies in the treatment of affective disorders may not be fully applicable to the treatment of impulsive aggression, they leave open the possibility that serotonergic agents are able to enhance function in those serotonergically ennvervated areas of the brain which are implicated in the neurobiology of impulsive aggression.

Treatment trials to date have found positive results for SSRIs and MAOIs. The limited efficacy and problematic side effects of available MAOIs limit their role in the first line treatment of impulsive aggression in personality-disordered subjects. The SSRI fluoxetine has been found to be more effective than placebo in controlled studies in the treatment of impulsive aggression. Open trials of other serotonin reuptake inhibitors have also had positive results. Controlled trials of these agents are needed to verify their efficacy. Given the propensity of tricyclic antidepressants to worsen impulsive aggression in vulnerable individuals, controlled trials of newer antidepressants that have effects on both serotonin and norepinephrine are needed.

Results from clinical trials reveal limits to the efficacy of serotonergic agents. People with the highest amounts of aggression and/or the most pronounced serotonergic deficits may be less likely respond to SSRIs. Also, in those who do respond, the "residual" amount of aggression may still be clinically significant. In real-world terms, this means that persons taking fluoxetine may still face the consequences of continuing impulsive aggression at home, in the workplace, or in the midst those around them. Future research is needed to see whether these subjects benefit from other psychopharmacologic agents such as mood stabilizers or the addition of serotonin-enhancing agents such as pindolol or buspirone. It is also unknown how psychopharmacological treatment might interact with psychosocial interventions such as cognitive-behavioral therapy.

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REFERENCES

1. Coccaro EF, Siever LJ, Klar HM, Maurer G, Cochrane K, Cooper TB, Mohs RC, Davis KL: Serotonergic studies in patients with affective and personality disorders. Correlates with suicidal and impulsive aggressive behavior. Arch Gen Psychiatry 1989, 46:587–599.
2. Coccaro EF, Kavoussi RJ, Berman ME, Lish JD. Intermittent explosive disorder-revised: development, reliability, and validity of research criteria. Compr Psychiatry 39:368–376.
3. Siever LJ, Buchsbaum MS, New AS, Spiegel-Cohen J, Wei T, Hazlett EA, Sevin E, Nunn M, Mitropoulou V. d,l-Fenfluramine response in impulsive personality disorder assessed with [^{18}F]flourodeoxyglucose positron emission tomography. Neuropsychopharmacology 1999, 20:413–423.
4. Pineyro G, Blier P. Autoregulation of serotonin neurons: role in antidepressant drug action. Pharmacol Rev 1999, 51:533–591.
5. Jolas T, Haj-Dahmane S, Kidd EJ, Langlois X, Lanfumery L, Fattaccini CM. Central pre- and postsyntaptic 5-HT1A receptors in rats treated chronically with a novel antidepressant, cericlamine. J Pharmacol Exp Ther 1994, 268:1432–1443.
6. Meyer JH, Kapuur S, Eisfeld B, Brown GM, Houle S, DaSilva J, Wilson AA, Rafi-Tari S, Mayberb HS, Kennedy SH. The effect of paroxetine on 5-HT2A receptors in depression: an [^{19}F]setoperone PET imaging study. Am J Psychiatry 2001, 158: 78–85.
7. Kaufman MJ, Hartig PR, Hoffman BJ. Serotonin 5-HT2C receptor stimulates cyclic GMP formation in choroid plexus. J Neurochem 1995, 64:199–205.
8. Åsperg M, Traskman L, Thoren P. 5-HIAA in the cerebrospinal fluid. A biochemical suicide predictor? Arch Gen Psychiatry 1976, 33:1193–1197.
9. Brown GL, Goodwin FK, Ballenger JC, Goyer PF, Major LF. Aggession in humans correlates with cerebrospinal fluid amine metabolites. Psychiatry Res 1979, 1:131–139.
10. Linnoila M, Virkkunen M, Scheinin M, Nuutila A, Rimon R, Goodwin FK. Low cerebrospinal fluid 5-hydroxyindolacetic acid concentration differentiates impulsive from nonimpulsive violent behavior. Life Sci 1983, 33:2609–2614.
11. Lopez-Ibor JJ, Saiz-Ruiz J, Perez de los Cobos JC. Biological correlates of suicide and aggressivity in major depressions (with melancholia).Neuropsychobiology 1985, 14:67–74.
12. New AS, Trestman RL, Mitropoulou V, Benishay DS, Coccaro EF, Silverman J, Siever LJ. Serotonergic function and self-injurious behavior in personality disorder patients. Psychiatry Res 1997, 69:17–26.
13. O'Keane V, Moloney E, O'Neill H, O'Connor A, Smith C, Dinan TG. Blunted prolactin responses to d-fenfluramine in sociopathy: evidence for subsensitivity of central serotonergic function. Br J Psychiatry 1992, 160:643–646.
14. Soloff PH, Meltzer CC, Greer PJ, Constantine D, Kelly TM. A fenfluramine-activated FDG-PET study of borderline personality disorder. Biol Psychiatry 2000, 47:540–547.
15. Blier P, De Montigny C. Electrophysiological investigations on the effect of re-

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peated zimelidine administration on serotonergic neurotransmission in the rat. J Neurosci 1983, 6:1270–1278.
16. Blier P, De Montigny C. Electrophsiological investigations on the effect of repeated zimelidine administration on serotonergic neurotransmission in the rat. J Neurosci 1987, 3:1270–1278.
17. Jolas T, Haj-Dahmane S, Lanfumey L, Fattaccini CM, Kidd EJ, Adrien J, Gozlan H, Guardiola-Lemaitre B, Hamon M. (-) Tertatolol is a potent antagonist at pre- and postsynaptic serotonin 5-HT1A receptors in the rat brain. Naunyn Schmiedebergs Arch Phramacol 1993, 347:453–463.
18. Artigas F, Romero L, de Montigny C, Blier P. Acceleration of the effect of selected antidepressant drugs in major depression by 5-HT1A antagonists. Trends Neurosci 1996, 19:378–383.
19. Martinez D, Mawlawi O, Dah-Ren Hwang, Justine Kent, Simpson N, Parsey RV, Hashimoto T, Slifstein M, Huang Y, Heertum RV, Abi-Dargham A, Caltabiano S, Malizia A, Cowley H, Mann JJ, Laruelle M. Positron emission tomography study of pindolol occupancy of 5-HT1A receptors in humans: preliminary analysis. Nuclear Med Biol 2000, 27:523–527.
20. Harrington MA, Peroutka SJ. Modulation of 5-hydroxytryptamine1A receptor density by nonhydrolyzable GTP analogues. J Neurochem 1990, 54:294–299.
21. Mongeau R, Welner SA, Quirion R, Suranyi-Cadotte BE. Further evidence for differential affinity states of the serotonin1A receptor in rat hippocampus. Brain Res 1992, 590:229–238.
22. Peroutka SJ. Selective interactions of novel anxiolytics with 5-HT1A receptors. Biol Psychiatry 1985, 20:971–979.
23. Coccaro EF, Astill JL, Herbert JL, Schut AG. Fluoxetine treatment of impulsive aggression in DSM-III-R personality disorder patients. J Clin Psychopharmacol 1990, 10:373–375.
24. Cherek DR, Moeller FG, Khan-Dawood F, Swann A, Lane SD. Prolactin response to buspirone was reduced in violent compared to nonviolent parolees. Psychopharmacology (Berl) 1999, 142:144–148.
25. Greendyke RM, Kanter DR. Therapeutic effects of pindolol on behavioral disturbances associated with organic brain disease: a double-blind study. J Clin Psychiatry 1986, 47:423–426.
26. Caspi N, Modai I, Barak P, Waisbourd A, Zbarsky H, Hirschmann S, Ritsner M. Pindolol augmentation in aggressive schizophrenic patients: a double-blind crossover randomized study. Int Clin Psychopharmacol 2001, 16:111–115.
27. Pfeffer CR, Jiang H, Domeshek LJ. Buspirone treatment of psychiatrically hospitalized prepubertal children with symptoms of anxiety and moderately severe aggression. J Child Adolesc Psychopharmacol 1997, 7:145–155.
28. Ratey J, Sovner R, Parks A, Rogentine K. Buspirone treatment of aggression and anxiety in mentally retarded patients: a multiple-baseline, placebo lead-in study. J Clin Psychiatry 1989, 50:382–384.
29. Verhoeven WM, Tuinier S. The effect of buspirone on challenging behaviour in mentally retarded patients: an open prospective multiple-case study. J Clin Psychopharmacol 1994, 14:126–130.

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30. Sharp T, Hjorth S. Application of brain microdialysis to study the pharmacology of the 5-HT1a autoreceptor. J Neurosci Methods 1990, 34:93–80.
31. Artigas F. 5-HT and antidepressants: new views from microdialysis studies. Trends Pharmacol Sci 1993, 14:262.
32. Blier P, de Montigny C. Serotoninergic but not noradrenergic neurons in rat central nervous system adapt to long-term treatment with monoamine oxidase inhibitors. Neuroscience 1985:16:949–955.
33. Blier P, De Montigny C, Azzaro AJ. Modification of serotonergic and noradrenergic neurotransmissions by repeated administration of monoamine oxidase inhibitors: electrophysiological studies in the rat central nervous system. J Pharmacol Exp Ther 1986, 237:987–994.
34. Newman ME, Lerer B, Shapira B. 5-HT1A receptor-mediated effects of antidepressants. Prog Neuro-Psychopharmacol Biol Psychiatry 1993, 17:1–19.
35. Lesch KP, Maji HK. Signal-transducing G proteins and antidepressant drugs: evidence for modulation of alpha subunit gene expression in rat brain. Biol Psychiatry 1992, 32:549–579.
36. Hedberg DL, Houck JH, Glueck BC. Tranylcypromine-trifluoperazine combination in the treatment of schizophrenia. Am J Psychiatry 1971, 127:1141–1146.
37. Parsons B, Quitkin FM, McGrath PJ. Phenelzine, imipramine, and placebo in borderline patients meeting criteria for atypical depression. Psychopharmacol Bull 1989, 25:524–534.
38. Cowdry RW, Gardner DL. Pharmacotherapy of borderline personality disorder. Alprazolam, carbamazepine, trifluoperazine, and tranylcypromine. Arch Gen Psychiatry 1988, 45:111–129.
39. Soloff PH, Cornelius J, Anselm G, Nathan S, Perel JM, Ulrich RF. Efficacy of phenelzine and haloperidol in borderline personality disorder. Arch Gen Psychiatry 1993, 50:377–385.
40. Cornelius JR, Soloff PH, Perel JM, Ulrich RF. Continuation pharmacotherapy of borderline personality disorder with haloperidol and phenlzine. Am J Psychiatry 1993, 50:1843–1848.
41. Wong DT, Reid LR, Bymaster FP, Threlkeld PG. Chronic effects of fluoxetine, a selective inhibitor of serotonin uptake, on neurotransmitter receptors. J Neural Transm 1985, 64:251–269.
42. Blier P, de Montigny C, Chaput Y. Electrophysiological assessment of the effects of antidepressant treatments on the efficacy of 5-HT neurotransmission. Clin Neuropharmacol 1988, 11(suppl 2):S1–S10.
43. Li Q, Muma NA, Battaglia G, Van de Kar LD. A desensitization of hypothalamic 5-HT1A receptors by repeated injections of paroxetine: reduction in the levels of G(i) and G(o) proteins and neuroendocrine responses, but not in the density of 5-HT1A receptors. J Pharmacol Exp Ther 1997, 282:1581–1590.
44. Moret C. Pharmacology of the serotonin receptor. In: Neuropharmacology of Serotonin, ed. Green RA. Oxford: Oxford University Press, 1985.
45. Aghajanian GK, Wang RY, Baraban J. Serotonergic and non-serotonergic neurons of the dorsal raphe: reciprocal changes in firing induced by peripheral nerve stimulation. Brain Res 1978, 153:169–175.

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46. Sanchez C, Hyttel J. Comparison of the effects of antidepressants and their metabolites on reuptake of biogenic amines and on receptor binding. Mol Neurobiol 1999, 19:467–489.
47. Palvimaki EP, Roth BL, Majasuo H, Laakso A, Kuoppamaki M, Syvalahti E, Hietala J. Interactions of selective serotonin reuptake inhibitors with the serotonin 5-HT2c receptor. Psychopharmacology (Berl) 1996, 126:234–240.
48. Ni YG, Miledi R. Blockage of 5HT2C serotonin receptors by fluoxetine (Prozac). Proc Natl Acad Sci USA 1997, 94:2036–2040.
49. Maj J, Moryl E. Effects of fluoxetine given chronically on the responsiveness of 5-HT receptor subpopulations to their agonists. Eur Neuropsychopharmacol 1993, 3:85–94.
50. Kennett GA, Lightowler S, de Biasi V, Stevens NC, Wood MD, Tulloch IF, Blackburn TP. Effect of chronic administration of selective 5-hydroxytryptamine and noradrenaline uptake inhibitors on a putative index of 5-HT2C/2B receptor function. Neuropharmacology 1994, 33:1581–1588.
51. Le Poul E, Boni C, Hanoun N, Laporte AM, Laaris N, Chauveau J, Hamon M, Lanfumey L. Differential adaptation of brain 5-HT1A and 5-HT1B receptors and 5-HT transporter in rats treated chronically with fluoxetine. Neuropharmacology 2000, 39:110–122.
52. Seeman P. Receptor Tables, Vol 2: Drug Dissociation Constants for Neuroreceptors and Transporters. Tornoto: Schizophrenia Research, 1993, Section 26.
53. Leysen J, Paulwels P. 5-HT2 receptors, roles and regulation. Ann NY Acad Sci 1990, 600:183-191.
54. Mayberg HS, Brannan SK, Tekell JL, Silva A, Mahurin RK, McGinnis S, Jerabek PA. Regional metabolic effects of Fluoxetine in major depression: serial changes and relationship to clihnical response. Biol Psychiatry 2000, 48:830–843.
55. Best M, Williams JM, Coccaro EF. Evidence for a dysfunctional prefrontal circuit in patients with an impulsive aggressive disorder. Proc Natl Acad Sci USA 2002, 99:8448–8453.
56. Corneilius JR, Soloff PH, Perel JM, Ulrich RF. Continuation pharmacotherapy of borderline personality disorder with haloperidol and phenelzine. Am J Psychiatry 1993, 150:1843–1848.
57. Norden MJ. Fluoxetine in borderline personality disorder. Prog Neuropsychopharmacol Biol Psychiatry 1989, 13:885–893.
58. Markovitz PJ, Calabrese JR, Schulz SC, Meltzer HY. Fluoxetine in the treatment of borderline and schizotypal personality disorders. Am J Psychiatry 1991, 148: 1064–1067.
59. Coccaro EF, Gabriel S, Siever LJ. Buspirone challenge: preliminary evidence for a role for central 5-HT1a receptor function in impulsive aggressive behavior in humans. Psychopharmacol Bull 1990, 26:393–405.
60. Kavoussi RJ. Open trial of sertraline in personality disorders with impulsive aggression. J Clin Psychiatry 1994, 55:137–141.
61. Salzman C, Wolfson AN, Schatzberg A, Looper J, Henke R, Albanese M, Schwartz J, Miyawaki E. Effect of fluoxetine on anger in symptomatic volunteers with borderline personality disorder. J Clin Psychopharmacol 1995, 15:23–29.

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62. Coccaro EF, Kavoussi RJ. Fluoxetine and impulsive aggressive behaviour in personality-disordered subjects. Arch Gen Psychiatry 1997, 54:1081–1088.
63. Sheard MH, Marini JL,Bridges CI, Wagner E. The effect of lithium on impulsive aggressive behavior in men: Am J psychiatry 1976, 33:1409–1413.
64. Cherek DR, Lane SD, Pietras CJ, Steinberg JL. Effects of chronic paroxetine administration on measures of aggressive and impulsive responses of adult males with a history of conduct disorder. Psychopharmacology 2002, 159:266–274.
65. Marsh DM, Dougherty DM, Moeller FG, Swann AC, Spiga R. Laboratory-measured aggressive ebehavior of women: acute tryptophan depletion and augmentation. Neuropsychopharmacology 2002, 26:660–671.
66. Coccaro EF, Kavoussi RJ, Trestman RL, Gabriel SM, Cooper TB, Siever LJ. Serotonin function in human subjects: intercorrelations among central 5-HT indices and aggressiveness. Psychiatry Res 1997, 73:1-14.
67. Soloff PH,George A, Swami Nathan R, Schulz PM, Perel JM. Behavioral dyscontrol in borderline patients treated with amitriptyline. Psychol Bull 1987, 23:177–181.
68. Klein DF. Psychiatric diagnosis and a typology of clinical drug effects. Psychopharmacology 1968, 13:359–386.
69. Rampling D. Aggression: a paradoxical response to tricyclic antidepressants. Am J Psychiatry 1978, 135:117–118.

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20

Pharmacological Interventions

Anticonvulsants

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INTRODUCTION

Anticonvulsants are used to control tantrums, violence, and agitation in children (1), adolescents (2), adults (3), and the elderly (4). Some reviews of psychopharmacology of aggression include a section on anticonvulsants, and some reviews of anticonvulsant use include a section on aggression (3,5). While aggression is typically regarded as a target symptom, there have been efforts to use pharmacological response to classify aggression (6). Here, we extend these efforts and elaborate a tentative, clinically derived nosology of aggression with two major subtypes and four divisions within the second subtype (Table 1). After delineating this classification, we use it to address four clinical issues: when to use an anticonvulsant, which anticonvulsant to use, at what dose and duration, and what to do when treatment response is inadequate.

Pharmacological treatment of aggression elicits the criticism that this endeavor psychologizes a social and political set of issues (7,8). However, wars and most violent crimes are instrumental, willful acts that presumably arise through social learning (9). Pursuit of wicked policies and goals is totally consis-

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TABLE 1 Proposed Nosology of the "Aggressive Disorders"

Division	Subdivision	Second subdivision	Medication implications
Predatory	None	None	None
Affective	Cognitive	Impulsive	Stimulant
	Cognitive	Paranoid	Low-dose antipsychotics
	Mood	Mixed irritable (outer- and inner-directed; also called depressive-irritable)	Serotonin uptake inhibitors
	Mood	Outer-directed irritable (also hostile mood)	Anticonvulsants and mood stabilizers

tent with having a normal brain (7) and a normal mind (10). Something more is involved when aggression is associated with significant psychopathology.

PRELIMINARY ISSUES

Nature of Aggression

Aggression presents formidable definitional problems that are addressed elsewhere in this volume. The classificatory terms used in this chapter come from Affective Neuroscience (9), a theory of evolutionary neuroanatomy. Briefly, this theory posits that in animals and humans, normal aggression is "predatory" (reward driven, as in the thrill of the hunt) or "affective" (as in fighting off an intruder) (9). Both types of aggression are consistent with a normal neurobiology. Both have been adaptive throughout evolution. Each has a separate brain circuitry controlling its modulation and expression in mammals (9), and either could be subject to dysregulation leading to significant pathology. *Affective Aggression* indicates that there is a high level of arousal, display, and reactivity, not necessarily the presence of a particular mood (although mood does play the main role in two of four proposed subtypes of the pathological form of this phenomenon).

Dysregulation of Aggressive Mechanisms

Psychopathology is defined as "a disorder" in the DSM-III, III-R, and IV system (11–13). By definition, a disorder means that something is wrong [a "harmful dysfunction" (14,15), some propose] in an individual that cannot be explained by social factors alone. Its best conceptualization may lie in a brain, a mind, or an in-between model, but the intuition is that the problem is "inside" an individual and social factors alone do not explain it.

Is serial killing a set of wicked acts or psychiatric disorder? On the one hand, it is reward driven, and therefore presumably voluntary, and, in addition, there is no psychiatric treatment for it. On the other hand, there is a sense that something is dysfunctional in a serial killer that is not explained by social factors alone, perhaps in the attachment system. We could call this a disorder of predatory aggression, and it would be a "harmful dysfunction" of a normal adaptive evolutionary repertoire, i.e., hunting.

The point is easier to make with affective aggression. Affective aggression, with its emphasis on display, clearly has an adaptive role in evolution as a means of scaring enemies and rivals, usually within the same species (9). This normal repertoire can become harmful and dysfunctional when a full rage response follows minimal, imaginary or no provocation. This maladaptive response is not explained by social factors alone. The low threshold and high amplitude of response tells us that it is "inside" the individual.

Of the two types of aggression (predatory and affective), only pathological affective aggression is the target of psychopharmacology. Pathological affective aggression entails a subjective experience of anger lacking in reward seeking behavior (predation).

Although there is general agreement that only nonpredatory aggression is the target of psychopharmacology, the literature is confusing regarding the generic term for pathological nonpredatory aggression, i.e., what we call pathological affective aggression. This literature often uses "impulsive aggression" (16,17) as the generic term. This creates a practical problem because paranoid fear, irritable mood swings, depression, and probably other psychological states are also associated with nonpredatory aggression. "Impulsivity" connotes dysfunctional neuroanatomy (primarily in the frontal lobe). It is not clear that paranoia, irritability and depression primarily involve frontal lobe dysregulation. These states seem quite distinct from impulsivity seen in Attention-Deficit/Hyperactivity Disorder (ADHD). In this chapter, we avoid "impulsive aggression" as a generic term and restrict it to aggression seen in persons with ADHD spectrum problems. The purpose is to facilitate a nosological approach founded on the psychological states (paranoia, irritability, depression, and impulsivity per se) associated with pathological affective aggression.

In summary, for nosologic clarity, we prefer "pathological affective aggression" as the general term for pathological manifestations of nonpredatory aggression. This term suggests "harmful dysfunction" of an evolutionary useful mechanism (affective aggression). It also suggests mechanisms underlying normal function lie in distinct brain circuits. For a discussion of the philosophical justifications for this approach to psychopathology in general and aggression in particular, we recommend the work of Wakefield on harmful dysfunction (14) and Panksepp on affective neuroscience (9). It is important to reiterate that "affective" in this context does not equate with mood, although some affective aggression is

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motivated by mood, at least in the model we propose. "Affective," as used here, simply calls attention to the high level of arousal, display, and reactivity that characterizes nonpredatory aggression.

Pathological Affective Aggression in the Context of DSM-IV

While intoxication, epilepsy, dementia, medical illnesses, and various psychiatric conditions can present with pathological affective aggression, these conditions are not the main concerns of this chapter. Our main concern is *idiopathic* pathological affective aggression. When known physical/psychiatric conditions do not adequately explain why a patient has rage outbursts, the DSM-IV diagnosis is Intermittent Explosive Disorder (IED) (13) (Table 2). This diagnosis facilitates communication about pathological affective aggression (18). However, while its high "prevalence" ($\sim$ 5%) in outpatient psychiatric settings (19,20) indicates that it is a serious clinical problem, it may be too nonspecific to always be clinically useful (18,21).

Rationale for the Use of Anticonvulsants in the Treatment of Pathological Affective Aggression

Explosions of rage and epileptic seizures have superficial similarities. Both are sudden and seem superimposed on a more or less normal background. An epileptoid model of aggression was first developed by Monroe and colleagues (22). It was based on the discovery of abnormal electrophysiological discharges in the limbic areas of some chronically aggressive patients, discharges undetectable with surface electrodes. This epileptoid theory was echoed in the DSM-III and III-R criteria for IED (18). The explosions were not supposed to be associated with any particular background psychological state. The expectation was that the aggression would be sudden and unprovoked with little evidence of aggressive spectrum pathology between episodes.

However, the DSM-IV task force found little evidence for such a disorder.

TABLE 2 DSM-IV Criteria for the IED

A.	Several discrete episodes of failure to resist aggressive impulses that result in serious assaultive acts or destruction of property.
B.	The degree of aggressiveness expressed during the episodes is grossly out of proportion to any psychosocial stressor.
C.	The aggressive behaviors are not better accounted for by another mental disorder and are not due to the direct physiological effect of a substance or general medical condition.

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They noted that when aggression occurs without warning, it is usually associated with another medical or psychiatric condition. When idiopathic, aggression is usually associated with some psychological state between episodes. Clinicians were using the diagnosis to communicate about episodic violent behavior not explainable by another medical or psychiatric diagnosis. The task force noted these facts and changed the criteria for IED to allow for interepisode pathology (13). This action conformed to the way clinicians thought about IED, but created a nonspecific collection of patients with only episodic violent behavior in common.

This DSM-IV change vitiated hidden epileptoid limbic discharges as a model for idiopathic affective aggression. It did not rule out all theories implicating abnormal electrophysiology in aggression, however, and Barrett has proposed abnormal electrophysiological gating of stimuli leads to frontal lobe disinhibition. This theory allows for characterologic failure to inhibit impulses (i.e. interepisode pathology) and intermittent overt aggression (6). It is therefore consistent with the clinical observations that lead to the changes in DSM-IV.

Diffuse neuronal excitability is another electrophysiological model. It has been proposed as a point of contact between the antiseizure and mood stabilizing properties of anticonvulsants (23). Many, perhaps all anticonvulsants have mood stabilizing properties. Irritability, a global state of impatience and intolerance, has a close relation to poor anger control and overt aggression. It is a mood and it can fluctuate with normal mood. In some patients, it presents as a form of mood instability (1,2). It is also a cardinal feature of Bipolar Disorder, especially Bipolar II disorder, a condition characterized by mood swings (13). Thus, there may be a connection among abnormal electrophysiology, mood instability, and aggression mediated by the concept of irritability.

In closing this section on theories of anticonvulsant use for aggression, it is important to note that the mood-stabilizing properties of anticonvulsants need not be related to abnormal electrophysiology. Many argue that they are not related, that we must look to the properties anticonvulsants share with lithium, the paradigmatic mood stabilizer, for a theory of the mood-stabilizing properties of anticonvulsants. Significantly, raising the seizure threshold is not a property of lithium (24).

Subdivisions of Pathological Affective Aggression

Disorders of Affective Aggression is the proposed generic concept for all pathological affective aggression. Pathological affective aggression is associated with a range of psychological states (impulsivity, paranoia, terror, depression, and irritability). It is also associated with a range of very different pharmacological treatments (25–28). If the differences in psychology are linked to different pharmacological treatments, we have a tentative classification system with clinical relevance built into it.

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Two cognitive subtypes of Aggressive Disorders might be an impulsive type and a paranoid type. Psychostimulants are the first choice when aggression is associated with impulsivity (hitting without thinking), particularly in children (29). Antipsychotics are the first choice when aggression is associated with suspicion and poor reality testing (30).

There may also be two distinct mood subtypes of Aggressive Disorders. As noted, irritable mood has a close connection with overt aggression. Although irritability is a common term in psychiatry, it is rarely defined. Following Snaith (31,32), one of the few researchers who has looked into the problem of systematically defining this concept, we believe that irritability can be purely outer-directed or it can be a mixture of outer- and inner-directed pathology. By outer-directed, we mean the impatience and intolerance is directed only at the environment. By inner-directed, we mean the same attitude is also expressed toward the self. The point of this distinction is that it clarifies the nosology and it may have treatment implications. We now turn to these matters in greater detail.

Patients with inner-directed irritability endorse statements such as "I get angry at myself and call myself names" and "I feel like harming myself." Those with outer-directed irritability endorse statements like "people upset me so much, I feel like slamming doors and banging around." Snaith quantified these ideas in a scale, the IDA Scale (Irritability-Depression-Anxiety) and found that outer-directed irritability was a distinct construct in adults, independent of anxiety and depression (32). Thus the phenomenology of irritability is heterogeneous. Snaith found that outer-directed irritability is a freestanding condition, whereas inner-directed irritability occurs in combination with other affective states.

Biological studies also support the heterogeneity of irritability. Low serotonin is associated with inner-directed aggression (e.g., suicide) (33). Some adults with irritable aggression respond to a serotonin uptake inhibitor (34), and some do not but do respond to divaloproex, an anticonvulsant/mood stabilizer (35). Thus normalizing serotonin is therapeutic in some aggressive patients but not in others. Corroborating this, we found no inner-directed aggression in 20 irritable aggressive youth with a high divalproex response and a low placebo response (1). Adapting the Snaith IDA Scale to our adolescent patients, we found very high scores on outer directed irritability, little inner-directed irritability and normal levels of anxiety and depression (36).

It would be useful to examine whether these observations generalize. Do irritable-impulsive-aggressive patients who do not improve on a serotonin uptake inhibitor but do improve on divalproex differ in direction of their symptoms (34,35)? If so, this may indicate a separation on the biological level between a serotonin-based irritable system and another biological system (e.g. GABA, or glutamate). Among irritable-aggressive patients, we suspect serotonin uptake inhibitor responders have a mix of inner-directed and outer-directed irritability, while anticonvulsant responders may have only outer-directed irritability. To

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summarize the argument thus far: we propose two cognitive subtypes of Aggressive Disorders, an impulsive and a paranoid type, and two mood subtypes, a mixed irritable type and an outer-directed irritable type.

Earlier in this chapter, we noted the problems using "impulsivity" as the generic term for nonpredatory aggression. Since important work on anticonvulsant responsive aggression is reported using the term impulsivity, we review two papers with the above framework in mind.

Barratt and Stanford completed two double-blind, placebo-controlled studies with phenytoin, demonstrating efficacy in reducing aggression, impulsivity, and hostile mood (37,38). The studies presented electrophysiological measures that normalized with clinical improvement on phenytoin. The focus was on cognitive problems, the lack of planning prior to an aggressive act, and the sense that after treatment there was delay between the impulse and the discharge. However, these patients also had hostile mood, and this also improved on phenytoin.

Hostile mood and outer-directed irritability may be the same or closely related. The population in one study, explosive prisoners, is known from previous studies to be lithium responsive (39). Lithium is a mood stabilizer. Our outcome measure of irritability consisted of the six Hostility Subscale items from the SCL-90 (40). These items measure hostile mood, the same mood assessed on the Profile of Mood States, the scale used in the phenytoin study. Taken together, these facts suggest that outer-directed irritability and the impulsivity described in these patients may be different aspects of the same construct.

We think of outer-directed irritability as a mood, but moods have effects on cognition and can alter frontal lobe electrophysiological parameters, as our group has found with atypical depression and melancholia (41). If what Barrett and Stanford call impulsivity is the same as what Snaith calls outer-directed irritability, and they are viewing the same phenomenon from different perspectives, this would be important, because this construct, whatever one calls it, would then predict anticonvulsant response in three double-blind, placebo-controlled studies.

CLINICAL ISSUES

Indications for the Use of an Anticonvulsant in Aggression

It follows from the above discussion that, using our terminology, patients with pathological affective aggression driven by "outer-directed irritability" are likely to improve on an anticonvulsant. As noted, subject to the previous argument, three double-blind, placebo-controlled studies corroborate this point, our own and two phenytoin studies. In addition, our primary outcome measure was [in addition to a modified form of the OAS (42)] the six Hostility items from the Hopkins Symptom Checklist (SCL-90) (40). We found these six items very sensi-

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TABLE 3 Hostility Items from the SCL-90 (SCL-6) (To be read aloud to patient and in the presence of an coinformant)

This past week, how much were you bothered by? (to informant, how much did he/she seem bothered by?)					
1. Feeling easily annoyed or irritated	1	2	3	4	5
2. Temper outbursts that you (he/she) could not control	1	2	3	4	5
3. Having urges to beat, injure, or harm someone	1	2	3	4	5
4. Having urges to break or smash things	1	2	3	4	5
5. Getting into frequent arguments	1	2	3	4	5
6. Shouting or throwing things	1	2	3	4	5

To allow computation of percent reduction, add scores and subtract 6 to arrive at the zero-based score. It is important to emphasize when reading 3 and 4 aloud that the item is referring to urges: "not that you [he/she] did it; urges."

tive to medication effects, both in response and relapse. The items are listed in Table 3. We advocate its use in patients placed on an anticonvulsant for outer-directed irritability. It is easy to administer to multiple informants. We would read the items aloud to the patient and at least one other informant. We found consensus is easily reached on these items between patient and informant. The two informants should be together (in person or on speakerphone) in order to prod each other's memories as to the events of the previous week.

In addition to patients with prominent outer-directed irritability, aggressive patients with abnormal EEGs, head injury, and mental deficiency may benefit from anticonvulsants (43). When irritability is accompanied by significant internalizing symptoms, it is not clear that anticonvulsants are the first choice over serotonin uptake inhibitor. For patients with unequivocal inattention, impulsivity, and hyperactivity, as well as aggression, i.e., ADHD, stimulants are the first choice. If a stimulant is to be used, it is important to make sure that the rage outbursts are not driven by paranoia, as this may worsen on stimulants. Aggression and rage in the context of paranoia might best be treated with antipsychotics.

Rationale for Type of Anticonvulsant to Use in the Treatment of Aggression

It follows from the previous section that the only double-blind placebo-controlled studies for outer-directed irritability used valproic acid and phenytoin. The side effects and the potential for lethal overdose limit the use of phenytoin despite evidence of efficacy. Valproic acid is the easiest to use in outpatient settings. If that fails, most clinicians then move to other medications with mood-stabilizing

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properties, such as lithium or olanzapine, or another anticonvulsant. Rationale for this practice can be found in the antiaggressive properties of mood stabilizers as a class. Lithium has well-documented antiaggressive properties (39,44). In our study, patients did have unstable mood. The swings were from normal to irritable, rather than elation to depression, but there were distinct shifts. Several, if not all, anticonvulsant medications have known or putative mood-stabilizing effects. In addition to phenytoin (45) and divalproex, carbamazapine, topiramate, lamotrigine, and gabapentin (24) have all been used to stabilize mood. Thus, there is a range of choices with little rigorous study to recommend one over the other in the treatment of aggression.

It is unclear that mood stabilization and/or antiaggressive properties of anticonvulsants are related to antiseizure mechanisms (46). For example, unlike valproate and carbamazapine, phenytoin's antiseizure activity is mediated only by its effect on sodium channels in the membrane. It does not work on the synapse (46). If it has mood-stabilizing properties, then either it has synaptic effects relevant to mood stabilization but not to seizure control, or mood stabilization is connected to neuronal excitation in general.

Nature of an Adequate Trial

An adequate trial of a medication is the dose and duration of treatment after which further clinical improvement is unlikely. If a patient has failed an adequate trial, it is time to switch or modify treatment. Two factors are involved—the dose of the medication, and the duration of treatment on that dose. For most antidepressants, an adequate trial is generally 3–4 weeks after achieving a therapeutic dose. The evidence is that even with no dose increase, more new responses tend to occur on antidepressant than on placebo between weeks 3 and 6 of a 6-week trial. There is a latency period between the last dose increase and full clinical response of 2–3 weeks (47).

The aggression literature does not address adequate anticonvulsant dose and treatment duration. Regarding dose, it is not clear that antiseizure blood levels are needed for psychiatric effects. The closest analog comes from loading-dose studies in bipolar illness, which suggest that an antiseizure blood level is desirable. The same studies suggest a delay of several days between a) giving doses large enough to achieve anticonvulsant levels and b) clinical response (48,49). The studies do not indicate the proportion of unresponsive patients who improve after a longer trial at the same blood level, information crucial to the question of duration of treatment before deciding that further improvement is unlikely. Our divalproex study allowed 2 weeks to reach a dose of 10 mg/lb, a dose that invariably placed the patient's blood level in the anticonvulsant range (50–100 μ/mL). Patients were continued 4 additional weeks. Clinically, we felt 10 mg/

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lb could have been achieved more quickly. We also observed that clinical response could be delayed 1–2 weeks after achieving a steady anticonvulsant blood level.

Our own practice is to assume an antiseizure level of the medication is necessary, and then to maintain the patient on that level for 3 weeks. After that time, it is important to distinguish nonresponders from partial responders (see below). This overall approach assumes that the response pattern of anticonvulsants for aggression is analogous to that of antidepressants for depression. Our experience indicates that the entire dose of valproate can be given at 6 PM. The advantage is that if taken with dinner, the medication will be absorbed 5 hours later, and side effects will occur mostly during the night. During the study, the medication was very well tolerated using this approach. The delayed release form of the medication may be even more useful in simplifying dosing, decreasing side effects and increasing compliance.

Differential Diagnosis of Treatment Failure on an Anticonvulsant Among "Aggressive Disorder" Patients

There are many examples from clinical experience in which patients with the same ostensible diagnosis respond to a medication of the same class as the one that just failed. For example, some children improve on dextroamphetamine after deriving no benefit from methylphenidate. Nonetheless, we would suggest dividing the nonresponders into partial responders (minimally improved) and nonresponders (unimproved).

For partial responders, raising the dose or adding another anticonvulsant (e.g., topiramate to divalproex) or adding lithium is a reasonable action. For nonresponders, it is useful to reexamine the diagnosis before trying another mood stabilizer/anticonvulsant. For a patient who appears irritable and aggressive but unresponsive, we suggest looking for evidence of paranoia. Someone who is irritable feels everything in the world is "annoying," but the person with paranoid rage feels targeted, humiliated, ridiculed. Patients with borderline personality disorder often present this dilemma in that they appear to be irritable, and often do benefit from mood stabilizers, but when they do not it is often because of paranoid states. Switching to a medication with antipsychotic properties, such as olanzapine or risperidol, is a logical choice. The antiaggressive properties of these medications are supported in the literature (17).

If paranoia is not a plausible explanation for medication failure, then depressive symptoms need to be explored. In our experience, aggressive patients with inner-directed irritability have dysthymic symptoms, and this is usually but not always detectable on initial exam. Studies in irritable conduct disordered children support the existence of depressive-irritability (50).

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Issue of Substance Abuse, Especially Marijuana

The broader differential diagnosis includes DSM-IV diagnoses not apparent at the start of treatment. Unrecognized Bipolar II Disorder, with consequent depressive or hypomanic cycling is always possible, especially if depressive symptoms emerge following acute treatment with a mood stabilizer. Schizophrenia and Schizoaffective Disorders are unlikely to be missed. Substance abuse and withdrawal are real possibilities [see review by Lavine (51)]. Amphetamine and cocaine withdrawal produce irritability.

A key substance abused by adolescents and adults with temper outbursts and irritable mood is marijuana (52). Many of the subjects we treated reported consuming enormous quantities of marijuana in an attempt to "chill out" (calm down) (53). While there is no evidence that marijuana is a mood stabilizer, there is a good deal of evidence that it has anticonvulsant properties (54). At the same time, marijuana also has pro-convulsant properties (54). The net effect of smoking marijuana on overall neuronal excitability is suggested by a careful study of first-onset seizures. All first-onset seizures entering the emergency room at Harlem Hospital over a 3-year period were ascertained. The investigators found that the amount of alcohol consumed was a risk factor for a first-onset seizure (55), and marijuana was a protective factor for a first-onset seizure (56). This suggests that marijuana is a weak anticonvulsant. Whether it is also a weak mood stabilizer, as many of the adolescents and adults we treat claim, remains to be documented. If so, this would be the first instance we know of self-medication with a gateway drug. Thus marijuana is a significant comorbidity in youth and perhaps adults with irritable aggression. It is also part of the differential diagnosis of treatment failure, since marijuana withdrawal induces irritability (57) and irritable aggression (58).

CONCLUSION

The totality of what we call pathological affective aggression is sometimes labeled impulsive aggression. We believe that label is misleading because only some of the nonpredatory aggression that improves on medication is impulsive. We limit the term Impulsive Aggression to designate the aggressivity of patients with ADHD. The word "affective" is meant to suggest arousal, emotion, and reactivity, not necessarily that a disturbance in mood is driving the aggression. Our perspective is that pathological affective aggression is heterogeneous, and comes in a) primarily cognitive and b) primarily mood forms. The cognitive form is driven by either impulsivity or paranoia. Impulsivity involves disinhibition. Paranoia involves distorted perceptions of other people's intentions, but remains affective (nonpredatory) in that the explosions are defensive within the reality of the individual.

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The mood form of pathological affective aggression exists as variants of irritability. Serotonin-based studies frame aggression in terms of impulsivity, but a dominant symptom is a mood—irritability. If SSRI-responsive irritability is a mixture of outer- and inner-directed symptoms, while anticonvulsant responsive irritability is outer-directed only, as we found in our study, then considerable nosologic clarity is possible. If phenytoin-sensitive aggression involves outer-directed irritability, this would further clarify the issue. None of these questions are settled, however, and the advantage of an "Aggressive Disorders" heuristic is that it provides a clinically relevant context for a discussion of these issues. Finally, we note that many abused substances can induce aggression, but marijuana abuse may have a deeper clinical meaning. Just as anxiety and social fears increase risk for alcohol abuse, so irritability seems to have an intimate relation to marijuana abuse.

The future of aggression research and especially therapeutics depends on a clinically relevant nosology. This chapter is an attempt to motivate such a discussion and provide a tentative solution.

REFERENCES

1. Donovan, S.J., et al. Brief report: divalproex treatment for youth with explosive temper and mood lability: A double-blind, placebo-controlled crossover design. Am J Psychiatry, 2000; 157:818–820.
2. Donovan, S.J., et al. Divalproex treatment of disruptive adolescents: a report of 10 cases. J Clin Psychiatry, 1997; 58:12–15.
3. Lindenmayer, J.-P., Kotsaftis, A. Use of sodium valproate in violent and aggressive behaviors: a critical review. J Clin Psychiatry, 2000; 61(2):123–128.
4. Raskind, M.A. Evaluation and management of aggressive behavior in the elderly demented patient. J Clin Psychiatry, 1999; 60(suppl 15):45–49.
5. Young, J.L., Hillbrand, M. Carbamazepine lowers aggression: a review. Bull Am Acad Psychiatry Law, 1994; 22(1):53–61.
6. Barratt, E.S. The use of anticonvulsants in aggression and violence. Psychopharmacol Bull, 1993; 29(1):75–81.
7. Richters, J.E. Disordered views of aggressive children: a late twentieth century perspective. Ann NY Acad Sci, 1996; 794:208–223.
8. Richters, J.E., Hinshaw, S.P. The abduction of disorder in psychiatry. J Abnorm Psychol, 1999; 108(3):438–445.
9. Panksepp, J. Affective Neuroscience: The Foundation of Human and Animal Emotions. Series in Affective Science, ed. R.J. Davidson, P. Ekman, K. Scherer. New York: Oxford University Press, 1998, p 466.
10. Arendt, H. Eichmann in Jerusalem: A Report of the Banality of Evil, revised and enlarged edition. New York: Penguin Books, 1977.
11. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 3rd ed. Washington: American Psychiatric Publishing, 1980.

Marcel Dekker, Inc.
270 Madison Avenue, New York, New York 10016

12. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 3rd ed., revised. Washington: American Psychiatric Publishing, 1987.
13. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. Washington: American Psychiatric Publishing, 1994.
14. Wakefield, J.C. Evolutionary versus prototype analyses of the concept of disorder. J Abnorm Psychol, 1999; 108(3):374–399.
15. Klein, D.F. Harmful dysfunction, disorder, disease, illness, and evolution. J Abnorm Psychol, 1999; 108(3):421–429.
16. Coccaro, E.F., Siever, L.J. Pathophysiology and treatment of aggression. In: Neuropsychopharmacology: The Fifth Generation of Progress. Davis, K.L., et al., eds. American College of Neuropsychopharmacology, 2002.
17. Moeller, F.G., et al. Psychiatric aspects of impulsivity. Am J Psychiatry, 2001; 158(11):1783–1793.
18. Bradford, J., et al. Impulse control disorders. In: DSM-IV Sourcebook. Widiger, T.A., et al., eds. Washington: American Psychiatric Publishing, 1996 p 1195.
19. Zimmerman, M., Mattia, J. Prinicipal and additional DSM-IV disorders for which outpatients seek treament. Psychiatr Serv, 2000; 51:1299–1304.
20. Coccaro, E.F. Intermittent explosive disorder. Curr Psychiatry Rep, 2000; 2:67–70.
21. Kavoussi, R., Armstead, P., Coccaro, E. The neurobiology of impulsive aggression. Psychiatr Clin North Am, 1997; 20(2):395–403.
22. Monroe, R.R. Episodic Behavior Disorders: A Psychodynamic and Neurophysiologic Analysis. Cambridge: Harvard University Press, 1970.
23. Post, R.M., et al. The place of anticonvulsant therapy in bipolar illness. Psychopharmacology, 1996; 128:115–129.
24. Lenox, R.H., Frazer, A. Mechanism of action of antidepressants and mood stabilizers. In: Neuropsychopharmacology: The Fifth Generation of Progress. Davis, K.L., et al., eds. American College of Neuropsychopharmacology, 2002.
25. McElroy, S.L. Recognition and treament of DSM-IV intermittent explosive disorder. J Clin Psychiatry, 1999; 60:12–16.
26. Pabis, D.J., Stanislav, S.W. Pharmacotherapy of aggressive behavior. Ann Pharmacother, 1996; 30(3):278–287.
27. Stoewe, J.K., Kruesi, M.J.P., Lelio, D.F. Psychopharmacology of aggressive states. Psychiatr Clin North Am, 1995; 4(2):359–397.
28. Donovan, S.J. A new approach to disruptive behavior disorders and the antisocial spectrum. Medscape Mental Health, June 1998.
29. Klein, R.G., et al. Clinical efficacy of methylphenidate in conduct disorder with and without attention deficit hyperactivity disorder. Arch Gen Psychiatry, 1997; 54: 1073–1080.
30. Campbell, M., Gonzalez, N.M., Silva, R.R. The pharmacologic treatment of conduct disorders and rage outbursts. Psychiatr Clin North Am, 1992; 15(1):69–85.
31. Snaith, R.P., et al. A clinical scale for the self-assessment of irritability. Br J Psychiatry, 1978; 132:164–171.
32. Snaith, R.P., Taylor, C.M. Irritability: definition, assessment and associated factors. Br J Psychiatry, 1985; 147:127–136.
33. Linnoila, V.M., Virkkunen, M. Aggression, suicidality, and serotonin. J Clin Psychiatry, 1992; 53(suppl):46–51.

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34. Coccaro, E.F., Kavoussi, R.J. Fluoxetine and impulsive aggressive behavior in personality disordered subjects. Arch Gen Psychiatry, 1997; 54:1081–1088.
35. Kavoussi, R.J., Coccaro, E.F. Divalproex sodium for impulsive aggressive behavior in patients with personality disorder. J Clin Psychiatry, 1998; 59(12):676–680.
36. Donovan, S.J., Nunes, E.V., Stewart, J.W. Absence of internalizing symptoms in divalproex-responsive irritable disruptive youth. AACAP Scientific Proceedings, 2001.
37. Barratt, E.S., et al. The effects of phenytoin on inpulsive and premeditated aggression: a controlled study. J Clin Psychopharmacol, 1997; 17:341–349.
38. Stanford, M.S., et al. A double-blind placebo controlled crossover study of phenytoin in individuals with impulsive aggression. Psychiatr Res, 2001; 103:193–203.
39. Sheard, M.H., et al. The effect of lithium on impulsive aggressive behavior in man. Am J Psychiatry, 1976; 133(12):1409–1413.
40. Derogatis, L.R., Lipman, R.S., Covi, L. SCL 90: an outpatient psychiatric rating scale—preliminary report. Psychopharmacol Bull, 1973; 9:13–28.
41. Bruder, G.E., et al. Outcome of cognitive-behavioral therapy for depression: relation to hemispheric dominance for verbal processing. J Abnorm Psychol, 1997; 106(1): 138–144.
42. Yudofsky, S.C., Silver, J.M., Jackson, W. The Overt Aggression Scale for objective ratings of verbal and physical aggression. Am J Psychiatry, 1986; 143:25–29.
43. Wilcox, J. Divalproex sodium in the treatment of aggressive behavior. Ann Clin Psychiatry, 1994; 6(1):17–20.
44. Sheard, M.H. Lithium in the treatment of aggression. J Nerv Ment Dis, 1975; 160(2-1):108–118.
45. Mishory, A., et al. Phenytoin as an antimanic anticonvulsant: a controlled study. Am J Psychiatry, 2000; 157(3):463–465.
46. McNamara, J.O. Drugs effective in the therapy of the epilepsies. In: Goodman & Gilman's The Pharmacologic Basis of Therapeutics. Hardman, J.G., et al., eds. New York: McGraw-Hill, 1996, pp 461–486.
47. Donovan, S.J., et al. Duration of antidepressant trials: clinical and research implications. J Clin Psychopharmacol, 1994; 14(1):64–66.
48. McElroy, S.L., et al. Valproate as a loading treatment in acute mania. Neuropsychobiology, 1993; 27(3):146–149.
49. Keck, P.E. Jr., et al. Valproate oral loading in the treatment of acute mania. J Clin Psychiatry, 1993; 54(8):305–308.
50. Simic, M., Fombonne, E. Depressive conduct disorder: symptom patterns and correlates in referred children and adolescents. J Affect Disord, 2001; 62:175–185.
51. Lavine, R. Psychopharmacological treatment of aggression and violence in the substance using population. J Psychoact Drugs, 1997; 29(4):321–329.
52. Donovan, S.J., Nunes, E.V. Treatment of comorbid affective and substance use disorders: therapeutic potential of anticonvulsants. Am J Addict, 1998; 7(3):210–220.
53. Donovan, S.J., Susser, E.S., Nunes, E.V. Divalproex sodium for use with conduct disordered adolescent marijuana users [letter]. Am J Addict, 1996; 5:181.
54. Karler, R., Turkanis, S.A. Cannabis and epilepsy. In: Marijuana's Biological Effects: Advances in the Biosciences. Proceeding of the Satellite Symposium of the 7th International Congress of Pharmacology, Paris. Oxford: Pergamon Press, 1976.

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55. Ng, S.K., Hauser, W.A., Brust, J.C. Alcohol consumption and withdrawal in new-onset seizures. N Engl J Med, 1988; 319:666–673.
56. Ng, S.K., et al., Illicit drug use and the risk of new-onset seizures. Am J Epidemiol, 1990; 132:47–57.
57. Haney, M., et al. Abstinence symptoms following smoked marijuana in humans. Psychopharmacology, 1999; 141:395–404.
58. Kouri, E.M., Pope, H.G.J., Lukas, S.E. Changes in aggressive behavior during withdrawal from long-term marijuana use. Psychopharmacology, 1999; 143:302–308.

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About the Editor

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