



Phenomenology of Impulsive Aggression and Intermittent Explosive Disorder

Emil F. Coccaro^{*}, Michael S. McCloskey[†]

^{*}Clinical Neuroscience & Psychopharmacology Research Unit, Department of Psychiatry and Behavioral Neuroscience, Pritzker School of Medicine, University of Chicago, Chicago, IL, United States

[†]Mechanisms of Affect Dysregulation Laboratory (MAD LAB), Department of Psychology, Temple University, Philadelphia, PA, United States

Contents

Aggression as a Behavioral Dimension	38
Relevance of Anger to Impulsive Aggression	39
Intermittent Explosive Disorder	40
Diagnostic Criteria for IED in DSM-5	40
Relevance of the A ₁ Criteria for DSM-5 IED	41
Relevance of the A ₂ Criteria for DSM-5 IED	41
Community Survey Studies of IED	42
Nature of IED Diagnosis in Community Survey Studies	43
Prevalence of IED in Community Survey Studies	43
Other Characteristics of IED Explored in Community Survey Studies	46
Age of Onset (AOO)	46
Type of Aggression	46
Severity of IED	46
Persistence of IED	46
Impairment Associated With IED	47
Association With Suicidal Behavior	47
Treatment Seeking in IED	47
Relationship With Age	47
Relationship With Biological Sex	48
Ethnic Differences in IED	49
Relationship With Education	49
Relationship With Household Income	49
Clinical Research Studies of IED	50
Prevalence of IED in Clinical Research Studies	50
Biological Sex and Ethnic Differences in IED	51
Type of Aggression in IED	51
Psychometric Characteristics of Aggression in IED	52
Association With Suicidal and Self-Injurious Behavior	53
Subjective Experience of Impulsive Aggressive Outbursts in IED	53

Psychosocial Consequences	55
Treatment Seeking in IED	55
Psychological Correlates	55
Criticisms of IED and Commentary	57
IED is an Unnecessary "Medicalization" of Human Aggression	57
IED is Just "Bad Behavior"	58
IED is Due to the Presence of Other Disorders and Conditions	58
IED is Due to "Psychopathy"	59
Data on IED are Unreliable Because Those With Impulsive Aggressive Outbursts are Inherently Unreliable	59
Summary	60
References	61
Further Reading	64



Aggression as a Behavioral Dimension

Human aggression occurs when individual assaults/attacks another in the context of defense or in the context of securing access to resources needed for survival. As such, aggression represents a fundamental aspect of human behavior. Over the course of civilization, however, human aggression has become less advantageous, with the use of aggression, for reasons other than self-defense, generally viewed as unacceptable.

Aggressive behavior can vary in both form and type. With respect to form, aggression can be verbal (e.g., snapping, name-calling, arguments, and threats) and/or physical (e.g., throwing things, to breaking things, to pushing/hitting to physically injuring someone). The aggression can also be direct (yelling at/hitting a person) or indirect (gossiping about a person, damaging an object). Indirect aggression aimed at damaging a person's interpersonal relationships is called relational aggression (Murray-Close, Ostrov, Nelson, Crick, & Coccaro, 2010). What all forms of aggression have in common is intent to harm and/or to forcibly defend oneself in the setting of a perceived social threat, though the function or "type" of aggression may differ.

Aggression can be socially sanctioned (e.g., soldiers, law enforcement) or medically related. Neither type of aggression is typically the focus of aggression interventions, as in the former case the aggression is not seen as problematic and in the latter there is clear medical cause for the behavior. Other, more "primary" types of aggression are premeditated (i.e., instrumental, proactive) aggression, and impulsive (e.g., affective, angry, or reactive) aggression (Dodge, Bates, & Pettit, 1990; Barratt, Stanford, Felthous, & Kent, 1997).

The critical difference between these types of aggression is that in premeditated aggression, the harm caused by the aggressive behavior is a merely a means to an end (e.g., punching someone to steal their wallet), whereas in the case of impulsive aggression the desire to harm to another, in response to a social threat, or frustration, is a primary goal of the aggressive act.

Though aggressive acts can include both aspects instrumental and impulsive facets, the large majority of aggression is predominately impulsive (Fanning, Coleman, Lee, & Coccaro, 2019). Furthermore, primary aggression can be viewed as both along a continuum (i.e., dimensionally) and as a dichotomy between normative and pathological aggression (i.e., diagnostically). In this chapter we will examine the nature and phenomenology of primary aggression reflected by the clinical diagnosis of Intermittent Explosive Disorder.



Relevance of Anger to Impulsive Aggression

There are at least three, related, dysphoric affects: depression, anxiety, and anger. While the first two have been the subject of much study, anger has been less extensively studied. Built into the definition of impulsive aggression is the idea that the aggressive behavior is manifest in the context of anger. That said, dimensional measures of depression and anxiety may also be related to aggressive behavior through their relationship with anger. In examining our own clinical research data of more than 300 study participants, we found that measures of these three trait dysphoric affects are highly correlated with each other (i.e., depression with anger: $r=0.50$; anxiety with anger: $r=0.60$; depression with anxiety: $r=0.73$; all $P<.001$). However, when entered into the same regression model, trait anger emerges as the strongest correlate of lifetime aggression ($\beta=0.55$, $P<.001$) compared with trait anxiety ($\beta=0.19$, $P=.003$) and trait depression ($\beta=0.10$, $P=.114$). Thus attention to the presence of anger in psychiatric patients is critical in predicting aggression at some time in the future.

Not surprisingly, the presence of anger in psychiatric patients appears to be far more prevalent and meaningful than previously thought. For example, a recent study by Judd, Schettler, Coryell, Akiskal, and Fiedorowicz (2013) reported that more than half of the acutely depressed patients had at least a “mild” degree of overt anger (i.e., quick to express annoyance) during their current or worst week of depression. More importantly, the presence of anger was associated with increased depressive severity, longer duration of the index major depressive episode, poorer impulse control, a more chronic

and severe long-term course of illness, higher rates of lifetime comorbid substance abuse and anxiety disorder, antisocial personality disorder, greater psychosocial impairment before evaluation and during follow-up, and reduced life satisfaction. A more recent study (Genovese, Dalrymple, Chelminski, & Zimmerman, 2017) reported that nearly half of a large ($n=3800$) general sample of psychiatric outpatients reported moderate-to-severe levels of current subjective anger, and that one-fifth reported moderate-to-severe levels of current overt aggression. The frequency of anger was similar to the frequencies of depressed mood and psychic anxiety. Anger and aggression were elevated across all diagnoses except adjustment disorder. Anger and aggression were most elevated in patients with major depressive disorder, panic disorder with agoraphobia, posttraumatic stress disorder, intermittent explosive disorder, and cluster B personality disorders.



Intermittent Explosive Disorder

When sufficiently frequent and severe, individuals with expressed anger and impulsive aggression can meet the DSM-5 diagnosis of Intermittent Explosive Disorder (IED; Coccaro, 2012). While impulsive aggression is also observed in other diagnostic conditions, where aggression is not directly due to another psychiatric disorder, a diagnosis of IED may be made. In this way, IED represents a disorder of primary impulsive aggression while other behavioral disorders in which aggression may occur (e.g., psychosis, mania, etc.) can be referred to disorders of secondary aggression.

Diagnostic Criteria for IED in DSM-5

The essence of recurrent, problematic, impulsive aggression has been in the DSM since its first edition. Despite this, criteria for IED did not unequivocally require impulsive aggression as its hallmark characteristic until its fifth edition (DSM-5). IED in the DSM-5 is characterized by one, or both, types of impulsive aggressive outbursts (Coccaro, 2011). First (Criterion A), frequent, but low intensity, outbursts occurring twice weekly for at least 3 months (A_1 Criterion) and/or infrequent, but high intensity, outbursts occurring three times per year (A_2 Criterion). Second (Criterion B), outbursts are out of proportion to stressors and, thus, do not reflect normative reactions to extreme provocation (i.e., otherwise healthy individuals would not respond to the “stressor” with an aggressive outburst). Third (Criterion C), aggressive outbursts are impulsive (i.e., not premeditated), anger based in nature,

and not displayed in order to reach some tangible goal (i.e., not instrumental). Fourth (Criterion D), aggressive outbursts are associated with distress and impairment. Fifth (Criterion E), individuals are 6 years old or older before the diagnosis is made. Sixth (Criterion F), aggressive outbursts are not better accounted for by another disorder (i.e., aggressive outbursts do not occur only during the presence of another disorder).

Relevance of the A₁ Criteria for DSM-5 IED

In preparation for the update of DSM-IV IED criteria for DSM-5, we conducted a series of analyses to see if individuals with recurrent, problematic, impulsive aggression (i.e., IED) could be effectively identified if both high frequency but low intensity impulsive aggressive outbursts (A₁ in DSM-5) were used to define IED [in addition to low frequency but high intensity impulsive aggressive outbursts (A₂ in DSM-5) that was already part of the DSM-IV criteria for IED]. We found that those who met (at the time) research diagnostic criteria for IED due only to A₁ outbursts did not differ from those with only A₂ outbursts as a function of aggression severity measures, presence of familial IED, biological characteristics (e.g., platelet binding of 5-HT Transporters, hormonal responses to 5-HT pharmacochallenge), and treatment response to fluoxetine (Coccaro, 2012). Considering all with a lifetime diagnosis of IED in our early studies we found that about 70% met both A₁ and A₂ criteria, 20% met A₂ criteria only, and 10% met A₁ criteria only for IED (Coccaro, 2011). We reexamined this issue in our present studies of individuals with current DSM-5 IED and found that, while a similar proportion met both A₁ and A₂ criteria for IED, the proportion meeting A₁-Only and A₂-Only was reversed. Overall, it appears that most individuals who meet current DSM-5 criteria for IED have both forms of the “A” criteria for aggressive behavior.

Relevance of the A₂ Criteria for DSM-5 IED

We also examined our data regarding how to best operationalize the A₂ criteria. In this analysis we looked at those with current DSM-5 IED who had reported physical aggression that was not destructive or injurious, physical aggression that was moderately destructive/injurious (i.e., damage to items worth less than \$50, bodily injury not requiring medical attention), and physical aggression that was more seriously destructive/injurious (i.e., damage to items worth \$50 or more, bodily injury requiring medical attention). Compared with healthy controls, psychiatric controls, and those

meeting the A_1 criteria only (i.e., no reported physical aggression), we found a stepwise increase in aggression severity that was statistically significant only for those with serious physical aggression. These data suggest that, perhaps, only serious physical aggressive behavior should meet criteria for A_2 aggressive outbursts. If so, this would increase the proportion of those meeting the A_1 criteria alone. Regardless of how individuals met the DSM-5 criteria for IED, all study participants with IED (i.e., A_1 -Only, A_2 -No Damage/Injury, A_2 -Moderate Damage/Injury, A_2 -Serious Damage/Injury) had significantly higher aggression scores compared with both healthy and psychiatric controls. Thus even those with less than serious physical aggression are more aggressive than other individuals without recurrent, problematic, impulsive aggressive behavior.



Community Survey Studies of IED

Though early versions of the DSM characterized IED as a “rare” disorder (DSM-I through DSM-III-R), recent studies indicate that IED is much more common than previously thought up through the publication of the DSM-IV in 1994. Since the mid-2000s a number of community surveys have been conducted that included an assessment of DSM-IV IED. These include a small “add-on” IED assessment to an Epidemiological Catchment Area follow-up study in Baltimore (ECA-B; [Coccaro, Schmidt, Samuels, & Nestadt, 2004](#)); the National Comorbidity Study—Replication (NCS-R; [Kessler et al., 2006](#)); the National Latino and Asian American Study (NLAAS; [Ortega, Canino, & Alegria, 2008](#)); the NCS-R Adolescent Supplement Study ([McLaughlin et al., 2012](#)); the Army STAARS Study ([Knock et al., 2014](#)); the National Survey of American Life, Adolescent Supplement Study (NSAL-AS; [Oliver et al., 2016](#)) which reported on black adolescents; the Mental Health Surveillance Study (MHSS; [Karg et al., 2014](#)); as well as a series of other community surveys conducted in a number of non-US countries included in a recent summary of 17 national surveys in the World Mental Health Survey (WMHS; including the United States; [Scott et al., 2016](#)). Below, we review these data in terms of nature of diagnosis, prevalence (lifetime and 12 month), age of onset, male/female ratio, type of aggressive behavior in those with IED, persistence of IED over time, impairment, and severity. Comorbidity data from these studies will be reviewed in the next chapter of this volume.

Nature of IED Diagnosis in Community Survey Studies

No community survey has yet employed DSM-5 criteria for IED due to the fact that the DSM-5 has been available only since May 2013. While all community studies employed DSM-IV criteria, one small study also included the Research Criteria for IED (Coccaro et al., 2004). For all community surveys using DSM-IV criteria, the diagnosis of IED used either a “broad” and/or a “narrow” definition of IED. The broad definition simply required cases to have at least three “anger attacks” over the course of their life. These anger attacks had to be out of proportion to provocation and not due to medical or pharmacologic reasons for aggressive behavior. The narrow definition was similar but required the presence of three anger attacks in any given year. Twelve-month prevalence required that there be at least one anger attack in the past year. This distinction is important because, on most relevant parameters, those diagnosed IED by the narrow definition are more severely affected compared with diagnosed IED by the broad definition only in terms of severity, persistence, and impairment related to IED (Coccaro, 2012). In addition, the narrow definition is much closer to the definition outlined in DSM-5 which requires at least three aggressive outbursts in a single year rather than across the lifetime.

Prevalence of IED in Community Survey Studies

The first comprehensive survey including IED as a disorder of interest was the NCS-R study including 9282 adult individuals in the United States (Kessler et al., 2006). This study reported a lifetime and 12-month prevalence of “Broad IED” of 7.3% and 3.9%, respectively, and 5.4% and 2.7% for “Narrow IED,” respectively. An independent analysis of the NCS-R study data, performed by our group, including the new DSM-5 criteria (i.e., presence of A_2 outbursts, presence of substantial anger during aggressive outbursts, presence of distress and/or psychosocial impairment, and age of onset ≥ 6 years) reported that the prevalence of IED in the United States may be somewhat lower at 4.0% and 2.6% for lifetime and 12-month prevalence, respectively. That said, the true prevalence of IED by DSM-5 cannot be known based on existing data because DSM-5 criteria for IED require either low intensity (but high-frequency) impulsive aggressive outbursts (A_1 Criteria) or high intensity (but low frequency) outbursts (A_2 Criteria). Since the data instrument used in the NCS-R (Kessler et al., 2006) and the multinational (Scott et al., 2016) study only sought

information primarily about A_2 , not A_1 , type of aggressive outbursts, the prevalence of those meeting only the A_1 criteria in any of the existing community survey studies is unknown at this time. This absence of data referable to A_1 outbursts is important because data from our own studies suggest that more than one-fifth of those with current DSM-5 IED meet the A_1 criteria only (i.e., have frequent verbal and/or nondestructive/injurious outbursts only) but not the A_2 criteria for DSM-5 IED. If so, our revised numbers for the prevalence of DSM-5 IED likely constitute an underestimate.

In the NCS-AS study of 6493 adolescents (McLaughlin et al., 2012), lifetime and 12-month prevalence of “Broad” IED were somewhat lower at 5.3% and 1.7%, respectively. Among 1170 Black adolescents in the NSAL study (Oliver et al., 2016), lifetime and 12-month prevalence of “Narrow IED” were in a similar range at 6.2% and 4.1%, respectively. Among 2554 Latino adults (NLAAS study; Ortega et al., 2008), lifetime and 12-month prevalence of “Narrow IED” were 5.8% and 4.1%, respectively. Notably, IED was more prevalent in Latinos born in the United States compared with those born outside the United States (9.5% vs. 3.2% for lifetime; 6.7% vs. 2.3% for 12 months). This was also true for those whose parents were born in the United States compared with those born outside the continental United States (9.4%–9.7% vs. 3.2%), suggesting that there may be something about the culture of the United States that facilitates the expression of impulsive aggression. Finally, the Army STAARS study of 5428 nondeployed army soldiers (Knock et al., 2014) reported a lifetime pre- and post-enlistment diagnosis of “Broad IED” of 15.7% and 4.8%, respectively. If so, it appears that the prevalence of IED among those entering the military in the United States may be twice that of the general population, suggesting that those who seek military service may already be more impulsive aggressive than the general population. The Army STAARS study, however, did not apply hierarchical rules (as did NCS-R) to the diagnosis of IED and, thus, its prevalence estimate is, naturally, higher compared with the results from other community surveys. That said, if one takes a rate of 4.0%, as representing the prevalence of individuals who met the DSM-IV “A” and “B,” but not “C,” for IED from the NCS-R study, one could estimate the preenlistment rate of DSM-IV “Broad IED,” in Army STAARS as 11.7%, a figure still higher than that in the general US population as assessed in the NCS-R study (7.4%).

In contrast to the earlier are results from the MHSS study (Karg et al., 2014) involving 5653 US adults and from the WMHS survey (Scott et al., 2016) involving 88,063 adults in 16 countries (including the

NCS-R study in the United States). Unlike the earlier studies, the 12-month prevalence of “Narrow IED” was 0.4% in the MHSS study. This may have been due to the fact that this study used the Structured Clinical Interview for DSM Disorders (SCID; [First, Williams, & Gibbon, 1997](#)) which contained a very limited module for IED compared with the Composite International Diagnostic Interview (CIDI; [Kessler & Üstün, 2004](#)) used for most of the community surveys including the NCS-R, NCS-R-AS, and the WMH surveys, which included more than 30, compared with three, questions regarding anger attacks and that the MHSS study employed telephone rather than face-to-face interviews. The WMHS survey ([Scott et al., 2016](#)), involving 16 countries, also reported a 12-month prevalence of 0.4% (0.8% for lifetime). In this study, 12 of the individual surveys reported a lifetime prevalence of “Narrow IED” of less than 1.0%, while four reported a lifetime prevalence of 1.1 (Northern Ireland, Ukraine) or 1.2 (Columbia, South Africa), and one reported a lifetime prevalence of 2.7% (USA). Reasons for these cross-national differences are not known but are likely related to variation in general risk factors, cultural factors that impact on willingness to disclose information about one’s own psychopathology, and other methodological factors. That said, even the prevalence of DSM-IV “Narrow IED” in the United States, which was initially published as 5.4% and 2.7% for lifetime and 12-month prevalence, respectively, was reported at 2.7% and 1.5%, respectively. This is because the WMHS survey algorithm for DSM-IV IED included impairment as a criterion (based on a single CIDI item) and this reduced prevalence estimates by 50% for lifetime and by 56% for 12-month prevalence. In our own reanalysis of the NCS-R data ([Coccaro, Fanning, Fisher, Couture, & Lee, 2017](#); [Coccaro, Fanning, & Lee, 2017](#)), we included impairment and distress as a criterion (DSM-5 Criterion D) based on eight, rather than one, CIDI item and found the lifetime and 12-month prevalence to be 4.0% and 2.6%, respectively.

Even if the worldwide lifetime prevalence is truly 0.8%, IED by “Narrow” DSM-IV criteria would not be considered rare because “rare disorders” (in the United States) are defined as affecting less than 1 in 1500 individuals ($<0.066\%$; Rare Disease Act of 2002). For the United States, however, the 12-month prevalence of IED (2.7% in [Kessler et al., 2006](#); 1.5% in [Scott et al., 2016](#); 2.6% in our NCS-R reanalysis) is relatively common, especially in comparison to the 12-month prevalence estimates for schizophrenia (0.3%; [McGrath, Saha, Chant, & Welhan, 2008](#)), autism spectrum disorder (1.5%; [Christensen et al., 2016](#)), and all forms of bipolar disorder (2.6%; [Kessler, Chiu, Demler, Merikangas, & Walters, 2005](#)).



Other Characteristics of IED Explored in Community Survey Studies

Age of Onset (AOO)

Across community survey studies, the mean AOO is reported as early to middle adolescence. In the NCS-R study (Kessler et al., 2006), mean age of onset (AOO) was similar for both “Broad IED” (14.8 years) and “Narrow IED” (13.5 years). In the NCS-AS study (McLaughlin et al., 2012) mean AAO was 12.5 years and in the ECA-B study (Coccaro et al., 2004) mean AAO was 18.3 years. In the NSAL study (Oliver et al., 2016) mean AAO was 10 years.

Type of Aggression

In the NCS-R study (Kessler et al., 2006), type of aggression was similar between “Broad” and “Narrow” IED with about two-thirds of anger attacks involving physical aggression with the rest involving threat, but not acts, of physical aggression.

Severity of IED

No IED-specific severity measures have been used in the community surveys conducted to date. That said, the NCS-R study reported that those with “Narrow IED” had nearly 10 times the number of anger attacks in the previous year compared with those with “Broad IED” (11.8 vs. 1.3 incidents); this was also true for number of weeks with at least one anger attack (19.6 vs. 1.5 weeks). In addition, the NCS-R study noted that the mean value of property damage was three times greater for those with “Narrow IED” compared with “Broad IED” (\$1603 vs. \$447). This was also true for the mean number of times in the lifetime that someone was physically hurt and required medical attention (233 vs. 37 incidents).

Persistence of IED

Persistence of a disorder can be assessed by dividing the 12-month prevalence by the lifetime prevalence. For the NCS-R study (Kessler et al., 2006), the persistence figure for “Narrow IED” was 64% compared with 24% for “Broad IED.” For the WMHS survey (Scott et al., 2016), the persistence figure was 50% for all countries and the persistence figure for the NLAAS Latino study was 71% (Ortega et al., 2008). For adolescents,

the persistence figure for the NSAL Black Adolescent study (Oliver et al., 2016) was 66%, though the persistence figure for the NCS-R-AS study (McLaughlin et al., 2012) was 32%.

Impairment Associated With IED

In the studies utilizing the CIDI, impairment was assessed using the Sheehan Disability Scale (SDS; Sheehan, Harnett-Sheehan, & Raj, 1986). In the NCS-R study, reported SDS scores associated with IED were twice as high among those with “Narrow IED” compared with those with “Broad IED” (40.4 vs. 19.6).

Association With Suicidal Behavior

In our reanalysis of the NCS-R data set, we found that history of a suicide attempt (SA) was fourfold higher in those with lifetime DSM-5 IED compared with those without IED [19.0% vs. 4.5%; Odds Ratio: 4.69 (95% CI: 3.51–6.29), $P < .001$]. Adding relevant comorbid diagnoses (i.e., mood, anxiety, substance use, and posttraumatic stress disorders) lowered the odds ratio for history of a past SA to about twofold, but still at a highly statistically significant level [Odds Ratio: 2.11 (95% CI: 1.53–2.91), $P < .001$]. Thus the relationship between suicidal behavior and IED is present even after accounting for the presence of other relevant disorders.

Treatment Seeking in IED

In the NCS-R study, only 13.2% of those with “Narrow” IED in the past year received treatment for their impulsive aggressive behavior compared with 33.2% for treatment of any psychiatric disorder. This trend was similar for those with lifetime “Narrow” IED (32.4% for treatment for IED and 61.6% for treatment of any psychiatric disorder) indicating that IED is far less recognized for treatment than other psychiatric disorders.

Relationship With Age

The inverse relationship between age and aggression is well known. Accordingly, it is important to explore the prevalence of IED as a function of age (Olweus, 1979). The NCS-R study reported a significantly higher prevalence of lifetime “Broad IED” in individuals 59 years and younger compared to individuals 60 years and older and revealed a stepwise reduction in the odds ratios going from the youngest to the oldest of individuals [OR for 18–29 years of age: 4.3 (2.1–9.0); OR for 30–44 years of age: 2.9 (1.3–6.3);

OR for 45–59 years of age: 1.6 (0.8–3.5)]. Our reanalysis of these data using DSM-5 criteria revealed a similar finding for past year IED but with much larger odds ratios for the younger age groups compared to the oldest age group [OR for 18–29 years of age: 15.9 (7.4–34.6); OR for 30–44 years of age: 9.2 (4.2–20.1); OR for 45–59 years of age: 6.0 (2.7–13.5)]. Thus IED emerges early in life with the greatest prevalence before 30 years of age and becomes increasing less prevalent approaching late middle age.

Relationship With Biological Sex

Clinical studies (McElroy, 1999) have suggested that males are much more likely to have IED than females. While recent community survey data suggests the same this ratio is lower than previously thought. This is consistent with observations that females are generally less physically, though not verbally, aggressive than males (Eagly & Steffen, 1986). However, in our clinical research studies, we have found that such sex differences are modest in magnitude: between a small- ($d=0.10$) and medium- ($d=0.30$) sized effect. For example, in our studies, males in our studies have a significantly greater degree of lifetime history of aggression (Life History of Aggression: LHA; Coccaro, Berman, & Kavoussi, 1997); a greater degree of a disposition to physical, but not verbal, aggression (Buss-Perry Aggression Questionnaire: BPA); and a greater degree of aggressive responding on a laboratory assessment of physical aggression (Taylor Aggression Paradigm: TAP). The effect sizes for these differences range from small ($d=0.09$ for Buss-Perry Verbal Aggression) to medium ($d=0.23$ for mean shock in the TAP which is a type of physical aggression). In contrast, females have higher trait anger scores than males on the BPA ($d=0.21$). Given that trait anger is a very strong correlate of IED, the lower rate of IED in females suggests that other factors must be working to constrain females from behaving aggressively. Such factors may be both biological and cultural.

In the NCS-R study, the sex ratio was reported for lifetime “Broad IED” only and this ratio was 1.66–1.00 suggesting a 66% greater prevalence of IED in males compared with females. In our reanalysis of the NCS-R data, we found a 1.52–1.00 ratio for past year DSM-5 IED. In contrast, the NCS-AS study found no significant difference in the prevalence of IED as a function of sex while other community studies report mixed results. The NLAAS (Ortega et al., 2008) and NSAL (Oliver et al., 2016) studies report a 1:35–1.00, and a 1.94–1.00, male-female ratio, respectively, while the

MUSS study (Karg et al., 2014) reports a threefold increase in prevalence among males compared with females. In summary, while IED is more prevalent in males than in females the difference in prevalence of IED as a function of sex is not as large as previously thought.

Ethnic Differences in IED

The NCS-R study reported only modest effects for differences in ethnicity as a function of lifetime “Broad IED”; note that the published data did not break this down as a function of “Narrow IED.” Our reanalysis of these data using DSM-5 criteria for lifetime IED revealed no significant difference in prevalence of IED as a function of ethnicity (i.e., White, Black, Latino, Other); for past year IED, however, those with “Other” ethnicity had a greater prevalence of IED compared to those who self-identified as non-Latino-White.

Relationship With Education

Similar to age, the NCS-R study reported a significantly higher prevalence of lifetime “Broad IED” in individuals with less than a full college education compared with those with a college education or more [OR for “less than high school”: 2.0 (1.4–3.0); OR for “high school graduates”: 1.4 (1.0–1.8); OR for “partial college”: 1.6 (1.2–2.2)]. Our reanalysis of these data revealed a similar finding for past year DSM-5 IED with odds ratios of about two (2) for each comparison [OR for “less than high school”: 2.2 (1.4–3.6), $P=.001$; OR for “high school graduates”: 1.7 (1.1–2.5), $P=.022$; OR for “partial college”: 1.46 (0.9–2.2), $P=.083$]. Accordingly, the presence of higher education (i.e., 16 plus years) may be a protective factor in the case of IED. This may be partly accounted for by the higher intelligence of those who go on to graduate college (Ceci & Williams, 1997), and the development of cognitive skills that reduce the risk of aggression since greater intelligence is associated with problem-solving skills that limit aggressive responding in social interaction (Huesmann, Eron, & Yarmell, 1987).

Relationship With Household Income

Also, similar to age, the NCS-R study reported a significantly higher prevalence of lifetime “Broad IED” in individuals with lower incomes

compared with those with “high” incomes [OR for “low income”: 1.5 (1.4–3.6); OR for “low average income”: 1.3 (1.3–3.0); OR for “high average income”: 1.3 (1.2–2.9)] though only the comparison for “low” vs. “high income” was statistically significant. While our reanalysis of these data found a similar odds ratio, comparing the prevalence of past year IED for the “low income” group with the “high income” group [OR: 1.3 (0.9–1.9), $P = .209$], group differences did not reach statistical significance ($P = .119$).



Clinical Research Studies of IED

Clinical research studies are quite different from community/epidemiologic studies in that study participants are drawn from the community either through public service announcements regarding the participation in research studies of IED (“symptomatic volunteers”) or through clinical referral for treatment. An advantage of clinical research studies is that investigators are able to collect much more data on study participants and the assessments are done by research psychiatrists and/or clinical psychologists. The disadvantage is that the study participants may not reflect all the characteristics of those with IED in the community because study participants are not necessarily representative of the full population with the disorder of interest.

The first such clinical research study in IED was performed in the DSM-III era and reported that very few individuals reporting “anger issues” actually met DSM-III criteria for IED (Felthous, Bryant, Wingerter, & Barratt, 1991). This result was due to the fact that the “B” criterion of DSM-III IED prohibited the diagnosis of IED in individuals with interoutburst aggressiveness and/or impulsivity and because of the exclusionary “C” criteria which prohibited the diagnosis in those with antisocial and/or borderline personality disorder. The DSM-IV revised IED criteria so that generalized aggressiveness/impulsiveness was not exclusionary for diagnosing IED and that impulsive aggressive individuals with AsPD/BPD could be given the diagnosis if the impulsive aggressive behavior was not explained, simply by the presence of AsPD and/or BPD.

Prevalence of IED in Clinical Research Studies

For the DSM-IV diagnosis of IED, we found a lifetime and current prevalence of IED of 6.6% and 3.1%, respectively, in a university outpatient clinic

population of 1800 individuals (Coccaro et al., 2005). An updated study adding another 2000 individuals to this data set reported a similar current prevalence of DSM-IV IED at 3.6% (Genovese et al., 2017); lifetime prevalence was not reported. The most recent study (Gelegen & Tamam, 2018) reported on the prevalence of IED by DSM-5 criteria in 406 patients consecutively evaluated in a Turkish outpatient psychiatry clinic and found a lifetime and 12-month prevalence of IED of 16.7% and 11.3%. To our knowledge, no other clinical studies have been conducted as a consecutive series of individuals coming for treatment in which the presence of IED was assessed.

Biological Sex and Ethnic Differences in IED

In a clinical survey study of psychiatric disorders in which the presence of DSM-IV IED was assessed (Coccaro et al., 2005), no significant differences were observed for the male-to-female ratio for current (1.17–1.00) or lifetime IED, though the sex ratio for lifetime IED (1.40–1.00) was in the same range as that reported in many of the community surveys. In contrast to this result, a recent study in Turkey (Gelegen & Tamam, 2018) reported a much higher male-to-female ratio for lifetime DSM-5 IED (2.97–1.00). In the first clinical survey study (Coccaro et al., 2005), the prevalence of DSM-IV IED, as a function of ethnicity, was significantly higher for nonwhites compared to whites for both current (2.7–1.0) and lifetime DSM-IV IED (1.9–1.0); subgroups of nonwhites did not differ from each other. These findings should be taken with caution, however, because the number of those with IED (current: 40; lifetime: 82) was relatively small. The recent clinical survey of Turkish psychiatric outpatients did not report data on ethnicity.

Type of Aggression in IED

The frequency of verbal assault (>100 events/year), during aggressive outbursts, is an order of magnitude greater than that for physical assault (>10 events/year (Kulper, Kleiman, McCloskey, Berman, & Coccaro, 2015); these frequencies are much greater than that seen in psychiatric (<5.0, and 0.1, events/year), or healthy (<0.1, and <0.01, events/year), controls. Like both psychiatric and healthy controls, the vast majority of aggressive behavior in those with is impulsive, rather than instrumental or premeditated, in nature (Fanning, Coleman et al., 2019).

Psychometric Characteristics of Aggression in IED

Examination of all our clinical research data comparing study participants with IED to those with nonaggressive psychiatric disorders (Psychiatric Controls) and nonaggressive Healthy Controls, document clear separation between the three groups with respect to both state (Overt Aggression Scale—Modified) and trait (LHA, BPA, and laboratory assessments of aggression such as the Taylor Aggression Paradigm and the Point Subtraction Aggression Paradigm) measures of aggression. While separation from Healthy Controls is expected, it is important to note that the high levels of aggression in IED are not due to the presence of general psychopathology since the IED and Psychiatric Control groups share that feature (Table 1).

Table 1 Mean Aggression and Impulsiveness Scores in Healthy Controls (HC), Psychiatric Controls (PC), and in Current IED Study Participants

	HC	PC	Current-IED	Group Differences
State Measures (±SD)				
OAS-M Total Aggression	1.8 ± 3.9	12.1 ± 26.1	101.1 ± 136.9	IED > PC > HC
OAS-M Global Anger and Aggression	0.9 ± 1.1	2.0 ± 2.5	5.9 ± 1.8	IED > PC > HC
Trait Measures (±SD)				
LHA Aggression	4.6 ± 3.4	7.7 ± 5.1	18.5 ± 4.0	IED > PC > HC
BPAQ Aggression	27.7 ± 8.9	32.0 ± 9.8	47.9 ± 11.2	IED > PC > HC
BPAQ Anger	12.0 ± 4.7	14.9 ± 6.0	25.1 ± 6.0	IED > PC > HC
LHIB Impulsivity	24.3 ± 16.6	34.5 ± 19.9	52.9 ± 19.7	IED > PC > HC
BIS Impulsivity	54.3 ± 8.9	61.9 ± 11.5	68.2 ± 11.1	IED > PC > HC
Laboratory Measures (±SD)				
Taylor Aggression (Mean Shock)				
(Low Provocation)	2.34 ± 2.13	2.44 ± 2.56	3.43 ± 3.39	IED > PC = HC
(High Provocation)	4.99 ± 3.85	5.23 ± 4.38	7.35 ± 5.04	IED > PC = HC
Point Subtraction Aggression (% Aggressive/All Presses)	4.98 ± 9.24	6.55 ± 9.53	10.02 ± 11.31	IED > PC = HC

Association With Suicidal and Self-Injurious Behavior

Analysis of our clinical research data set revealed that history of a SA and history of self-injurious behavior (SIB) was two- to fourfold higher in those with lifetime DSM-5 IED compared with those without IED [SA: 21.3% vs. 5.6%; Odds Ratio: 3.46 (95% CI: 2.4–5.0), $P < .001$; SIB: 16.4% vs. 5.7%; Odds Ratio: 2.5 (95% CI: 1.7–3.7), $P < .001$]. Adding relevant comorbid diagnoses (i.e., mood, anxiety, substance use, and posttraumatic stress disorders) lowered the odds ratio for both by about half (as was observed in the reanalysis of the NCS-R data set, see previously) to an Odds Ratio of 1.66 (95% CI: 1.1–2.5); $P < .015$) for SA and 1.4 (95% CI: 0.9–2.2, $P < .09$) for SIB. Adding Life History of Aggression (LHA) scores, however, completely eliminated the relationship between SA and SIB with DSM-5 IED [Odds Ratio for SA: 1.2 (95% CI: 0.7–2.1), $P = .839$; Odds Ratio for SIB: 1.1 (95% CI: 0.6–1.9), $P = .924$] indicating that the relationship between IED and risk of SA (and SIB) is fully explained by history of aggression.

Subjective Experience of Impulsive Aggressive Outbursts in IED

As part of our initial studies in IED we developed a semistructured research module to assess for the presence of IED as well as other aspects regarding the emotions and sensations felt before and during impulsive aggressive outbursts (IED-Module; IED-M). In an analysis comparing those with IED to healthy and psychiatric controls (Kulper et al., 2015; Fig. 1), we found that the vast majority of IED and psychiatric controls reported being angry (or irritated) compared with healthy controls. Much larger differences between the groups were seen for “feeling enraged” with those with IED endorsing this feeling compared with both psychiatric and healthy controls. Though less prevalent, nearly 40% of those with IED reported “feeling detached/unreal” or “feeling fearful/panicky” before/during outbursts, though this was also true for psychiatric, but not healthy, controls. IED and psychiatric controls also reported being less “clear-headed” before/during outbursts compared with healthy controls. For physical sensations, about three-quarters of IED and psychiatric controls reported having a racing heart, about half of both groups reported feeling “tremulous,” and up to a third of both groups reported “hot flashes,” before/during outbursts, proportions that were significantly greater compared with healthy controls. Other sensations, such as shortness of breath, did not differ among the groups. In contrast, feelings after impulsive aggressive outbursts

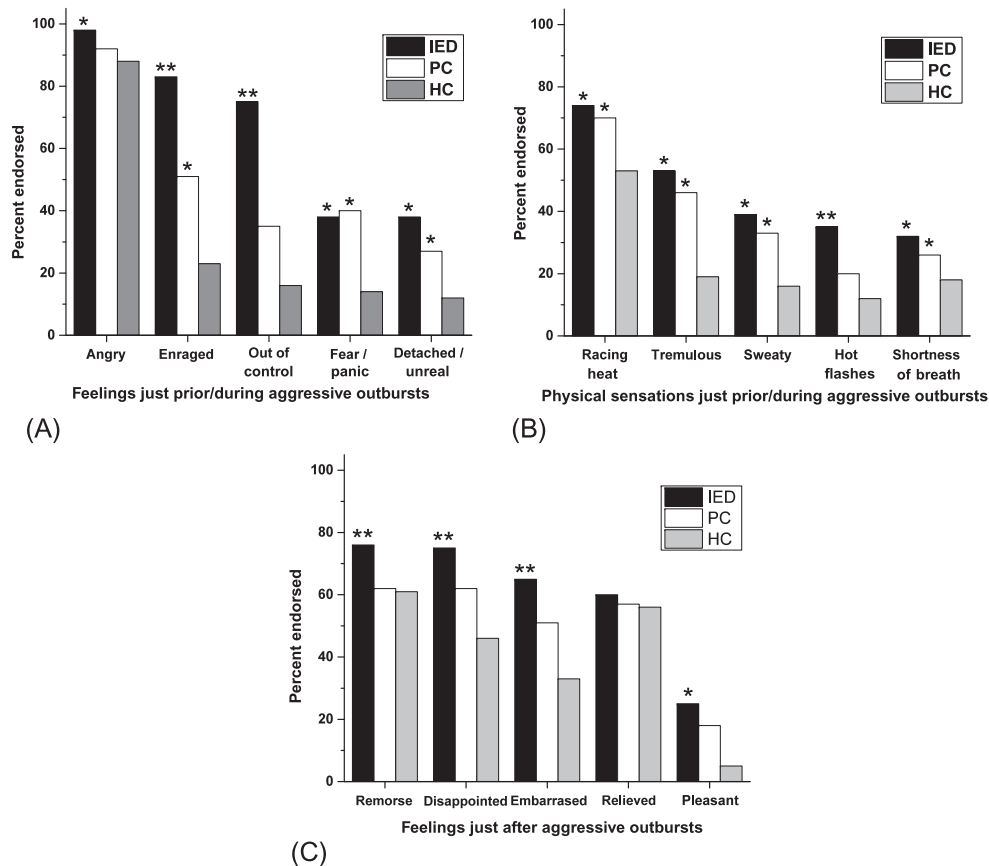


Fig. 1 Percent endorsing the emotions/sensations prior to and after aggressive outbursts as a function of group. *HC*, healthy control; *PC*, psychiatric control; *IED*, Intermittent Explosive Disorder. Asterisks indicate statistically significant differences from other groups.

distinguished those with IED from both psychiatric and healthy controls in most cases. Nine of ten of those with IED reported being “upset” after outbursts compared with about half for psychiatric controls and a third for healthy controls. Three-quarters of those with IED felt “remorseful” or “disappointed” after outbursts compared with about two-fifths to three-fifths for the control groups. Two-thirds of those with IED reported feeling “embarrassed” after outbursts compared to half for psychiatric controls and a third for healthy controls. Finally, one-quarter of those with IED reported feeling “pleasure” after outbursts compared with less than a fifth for psychiatric and healthy controls.

Psychosocial Consequences

Not surprisingly, individuals with IED experience significant psychosocial consequences as a result of recurrent, impulsive, aggressive behavior. First, global psychosocial function is reduced in IED compared with both healthy and psychiatric controls as is quality of life experience, even when differences in global psychosocial function are accounted for (Rynar & Coccaro, 2018). Examined more closely (Kulper et al., 2015), individuals with IED report large differences with psychiatric and healthy controls with regard to dysfunction with family (82% vs. 23% vs. 13%), with friends (75% vs. 24% vs. 6%), at work (56% vs. 16% vs. 4%), and in legal matters (39% vs. 11% vs. 0%). A second study in this area (Rynar & Coccaro, 2018) reported that those with IED had been more episodes of unemployment (1.57 vs. 0.96 vs. 0.93), and had been fired due to aggressive behavior in the workplace more often (17.5% vs. 7.0% vs. 4.0%), compared with psychiatric and healthy controls.

Treatment Seeking in IED

In a clinical survey study of psychiatric disorders in which the presence of DSM-IV IED was assessed (Coccaro et al., 2005), only 20% of those with IED were primarily in treatment to reduce their impulsive aggressive behavior, though an additional 60% would have been interested in such treatment. The remaining individuals were either unsure or uninterested in treatment.

Psychological Correlates

Consistent with the broad and significant impairments associated with the disorder, individuals with IED also demonstrate deficits across several psychological areas. Not surprisingly, those with IED report higher rates of

relational aggression (Murray-Close et al., 2010). They also endorse greater levels of immature defensive styles (i.e., acting out) and less use of mature defensive styles (i.e., sublimation; Puhalla, McCloskey, Brickman, Fauber, & Coccaro, 2016), which may contribute to the reduced emotional intelligence found among those with IED (Coccaro, Solis, Fanning, & Lee, 2015). Further, several studies have found cognitive-affective deficits among those with IED including a hostile attribution bias and related socio-emotional information-processing deficits (e.g., Coccaro, Fanning, Keedy, & Lee, 2016, also see the chapter on Social Cognition). Another area of focus has been impulsivity, as IED is often characterized as a clinical manifestation of “impulsive” aggression. This was supported by several studies in which those with IED self-reported greater impulsivity relative to non-psychiatric and psychiatric comparison groups (e.g., McCloskey, Lee, Berman, Noblett, & Coccaro, 2008). However, studies using behavioral measures of impulsivity were more equivocal (e.g., Coccaro, 1998). A recent study that may shed light on this discrepancy (Puhalla, Ammerman, Uyeji, Berman, & McCloskey, 2016) showed that, though individuals with IED reported greater impulsivity across several domains relative to a nonpsychiatric comparison group, only the impulsivity domain of negative urgency (the tendency to act out impulsively when experiencing a negative emotion) discriminated between those with IED and those with other psychiatric disorders. Furthermore, negative urgency uniquely predicted both increased anger and problems with aggression control among those with IED (Puhalla, McCloskey, et al., 2016). Thus it seems that impulsivity problems in IED may be limited to “hot” periods of negative affect (e.g., anger). This is also consistent with research showing that IED is associated with poor emotion regulation that extends beyond anger problems to global problems with increased affect lability and negative affect intensity, though anger dysregulation is most prominent (Fettich, McCloskey, Look, & Coccaro, 2015). These emotion regulation problems may be linked to dysregulation of corticolimbic brain circuitry, as individuals with IED demonstrate increased amygdala and, to a lesser extent, decreased orbitomedial activation to threatening stimuli (i.e., angry faces; Coccaro, McCloskey, Fitzgerald, & Phan, 2007; McCloskey et al., 2016). Difficulties with emotion regulation and impulsivity may also be facilitated by the tendency of individuals with IED to engage in angry rumination (Fahlgren, Puhalla, Sorgi, & McCloskey, 2019). Finally, though individuals with IED may have difficulty identifying their own emotion, early evidence suggests they do not have problems identifying and/or empathizing with the

emotions of others (Fahlgren et al., 2019). Thus IED appears to be linked to socioemotional deficits that both facilitate, and are exacerbated by, negative affect, rather than the lack of empathy often found in psychopathy (Coccaro, Lee, & McCloskey, 2014).



Criticisms of IED and Commentary

IED is an Unnecessary “Medicalization” of Human Aggression

There is no doubt that impulsive aggressive behavior is dimensional in nature and that individuals manifest aggression along dimensions of severity and frequency. If so, what are the reasons to have a categorical disorder of impulsive aggression? First, distributions of aggression scores are highly skewed to the left. In a population-based study we conducted (Coccaro & Jacobson, 2006), the vast majority of individuals (80.9%) had life history of aggression (LHA; Coccaro et al., 1998) scores in the normative range (e.g., LHA scores ≤ 11), while a smaller proportion (15.7%) had aggression scores in the moderate range (LHA scores from 12 to 17), and a much smaller proportion (3.5%) had aggression scores in the high range (LHA scores from 18 to 25), suggesting the possibility of a discontinuity in aggression scores. Second, in the context of medical models, therapeutic intervention is guided by the identification of a “disorder” as a target for treatment. While one could use the score on an aggression assessment in making this decision, valid cutoff scores on rating scales are difficult to establish and, more importantly, do not necessarily reflect on the degree of distress and/or impairment due to aggressive behavior. Third, sophisticated taxometric analyses (Meehl & Yonce, 1994) have shown that IED (Ahmed, Green, McCloskey, & Berman, 2010), unlike many other behavioral disorders (Haslam, Holland, & Kuppens, 2012), is categorical (taxonic) rather than dimensional in nature. This is in contrast to 80% of behavioral disorders in which aggression is a feature (Haslam et al., 2012). In a community study of over 20,000 individuals (Alegria, Jackson, Kessler, & Takeuchi, 2007), Ahmed et al. (2010) mapped seven behavioral indicators (derived from 28 IED-related items) onto both DSM-IV and Research Criteria for IED (Coccaro, 1998). Analysis yielded a statistical profile consistent with a taxonic, rather than dimensional, structure for IED. Taxon group membership was also associated with treatment seeking, family history of aggressive outbursts, lower age of onset of these outbursts, and male gender, factors that characterize individuals with IED. Recently, we replicated this finding

for DSM-5 IED in a clinical research sample (unpublished data). In this sample of those with IED ($n=344$) and healthy ($n=174$) and psychiatric ($n=295$) controls, a taxon for IED was identified in both the whole sample and in the sample after removing healthy controls. Among the IED and psychiatric control groups, the taxon displayed a sensitivity of 89.8% and a specificity of 81.4% (and a positive predictive value of 84.5% and a negative predictive value of 87.3%) for the DSM-5 diagnosis of IED. Not surprisingly, members of the taxon group had significantly higher scores on assessments of aggression, impulsivity, and related measures. This is in contrast to other disorders, in which aggression is frequently observed, such as post-traumatic stress disorder (Forbes, Haslam, Williams, & Creamer, 2005), anti-social personality disorder (Marcus, Ruscio, Lilienfeld, & Hughes, 2008), and borderline personality disorder (Fossati, Raine, Borroni, & Maffei, 2007; Rothschild, Cleland, Haslam, & Zimmerman, 2003).

IED is Just “Bad Behavior”

There has been increasing concern that the field of psychiatry is medicalizing “normative” behavior, especially, in the context of aggressive behavior. However, recurrent, problematic, impulsive aggressive behavior, as defined by DSM-5 IED, does not represent a variant of normal human behavior. Data from both community surveys and clinical research studies clearly indicate that the impulsive aggressive behavior of those with IED is aberrant when compared with both healthy controls and others with nonaggressive psychiatric disorders (psychiatric controls). Not only are dimensional measures of aggression, anger, and impulsivity higher in those with IED compared with controls, but this behavior is associated with objective assessments of distress and impairment. Further, those with IED display significant differences from controls on a number of biological parameters, including indices of serotonin function, gray matter volume in fronto-limbic circuits, shape of subcortical structures, amygdala activation to anger faces, and white matter integrity in selected brain areas (see relevant chapters in this volume). Accordingly, it is difficult to claim that there is “nothing there” when it comes to the validity of IED.

IED is Due to the Presence of Other Disorders and Conditions

In general, IED begins early in life (see the Age of Onset sections), typically earlier than the onset of many other psychiatric disorders such as mood, anxiety, and substance use disorders. [The one exception being that of phobic (social, and simple, phobic) disorders for which the onset precedes that of

IED by a few years in most individuals with both types of disorders.] Accordingly, it is difficult to argue that IED is due to other comorbid disorders. Rather, it is likely that IED increases the risk of other psychiatric disorders rather than the other way round. A more detailed discussion regarding comorbidity in IED is the focus of the next chapter in this volume.

IED is Due to “Psychopathy”

Psychopathy is a critical, though often misunderstood, cognitive-behavioral concept related to, but not identical to, antisocial behavior. Research over several decades has shown that there are at least two components to psychopathy. First, “callous-unemotionality” (i.e., superficial emotionality, grandiosity, lack of remorse, lack of empathy, deceitfulness, denial of responsibility) and second, “social deviance” (i.e., antisocial behavior, impulsivity, lack of clear goals, lack of behavioral controls, general irresponsibility). When we examine the presence of psychopathy in those with IED (Coccaro et al., 2014) we find that while dimensional scores of this factor are higher among those with IED, compared with psychiatric and healthy controls, only a small proportion of those with IED are likely to be psychopathic (20% total: 14% possibly psychopathic and 6% highly likely to be psychopathic) using the Psychopathy Checklist-Screening Version (Hart, Cox, & Hare, 2003). More importantly, scores for “social deviance” were significantly higher than those of “callous/unemotional” scores by about 71% in those with IED without psychopathy and by about 35% in those with IED with possible psychopathy. Accordingly, IED appears to be far less related to “callous-unemotionality” than to “social deviance” in general. This is also consistent with the fact that the form of aggression observed in IED is impulsive rather than premeditated in nature (Fanning, Coleman, et al., 2019).

Data on IED are Unreliable Because Those With Impulsive Aggressive Outbursts are Inherently Unreliable

This critique is driven by the assumption that those with recurrent, problematic, impulsive aggressive outbursts are in denial about their behavior and/or deliberately seek to minimize it. While there is little data regarding this specific issue, it is likely that those who seek evaluation and treatment for IED are different from those who are forced into to evaluation and treatment. When we examine our own data we have two separate findings of relevance (Steakley-Freeman, Lee, McCloskey, & Coccaro, 2018). First, compared with healthy and psychiatric controls, those with DSM-5 IED

score significantly lower on measures of social desirability (Social Desirability Scale) and deceptive self-reporting (Lie scale on the Eysenck Personality Questionnaire), and score significantly higher on “readiness to change” their aggressive behavior. Second, the use of first degree family informants did not increase the proportion of those meeting DSM-5 criteria for IED. In fact, out of 70 study participants in a family study of DSM-5 IED, only one study participant was identified by a family member as having IED when the study participant denied it. These data suggest that it is unlikely that denial, or the minimalization, of impulsive aggressive behavior is an important factor when studying those interested in evaluation and/or treatment of impulsive aggressive/angry behavior.



Summary

Anger is a common human emotion and, consequently, many behavioral disorders show some elevation in levels of anger and corresponding aggression that is secondary to the focus of that disorder. However, when excessive levels of anger and aggression are the core pathology, then IED is appropriate diagnosis. A disorder of primary aggression, IED is defined by excessive/disproportionate aggressive acts either in the form of frequent minor aggression (i.e., twice a week for 3 months or more) or more severe, damaging acts of aggression that can occur less frequently (i.e., three or more times a year), though most individuals with IED will show both over their lifetime. Research shows that IED is common (~3%–4%), persistent (typically beginning in adolescence and continuing throughout much of adulthood), and highly impairing across most domains of functioning. Though highly comorbid, IED tends to precede other psychiatric diagnoses. IED occurs in both men and women, though slightly more often in men, across racial and ethnic groups. Aggressive outbursts in IED are differentiated by their extreme level of anger, rage, and lack of subjective control, as well as the strong negative (and to some extent positive) feeling after the outbursts. This feeling of dyscontrol is also reflected in several cognitive-affective deficits among those with IED including hostile attributional biases, poor emotion regulation, and increased impulsivity when experiencing negative affect. However, unlike psychopathy, those with IED demonstrate a normal capacity for empathy, and despite the common symptom of aggression, the two disorders are not highly comorbid. In sum, IED is a distinct and common disorder of primary aggression that is poorly understood (or even known) by much of the public as well as by many practitioners, resulting in the undertreatment of this serious disorder.

References

- Ahmed, A., Green, B., McCloskey, M. S., & Berman, M. E. (2010). Latent structure of intermittent explosive disorder in an epidemiological sample. *Journal of Psychiatric Research*, *44*, 663–672.
- Alegria, M., Jackson, J. S., Kessler, R. C., & Takeuchi, D. (2007). *Collaborative Psychiatric Epidemiology Surveys (CPES), 2001–2003 [United States]*. Computer file ICPSR20240-v5. Ann Arbor, MI: Institute for Social Research, Survey Research Center.
- Barratt, E. S., Stanford, M. S., Felthous, A. R., & Kent, T. A. (1997). The effects of phenytoin on impulsive and premeditated aggression: a controlled study. *Journal of Clinical Psychopharmacology*, *17*, 341–349.
- Ceci, S. J., & Williams, W. M. (1997). Schooling, intelligence, and income. *The American Psychologist*, *52*(10), 1051–1058.
- Christensen, D. L., Baio, J., Van Naarden Braun, K., Bilder, D., Charles, J., Constantino, J. N., et al. (2016). Centers for disease control and prevention (CDC). *MMWR Surveillance Summaries*, *65*(3), 1–23.
- Coccaro, E. F. (1998). Impulsive aggression: a behavior in search of clinical definition. *Harvard Review of Psychiatry*, *5*, 336–339.
- Coccaro, E. F. (2011). Intermittent explosive disorder: development of integrated research criteria for Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. *Comprehensive Psychiatry*, *52*(2), 119–125.
- Coccaro, E. F. (2012). Intermittent explosive disorder as a disorder of impulsive aggression for DSM-5. *The American Journal of Psychiatry*, *169*, 577–588.
- Coccaro, E. F., & Jacobson, K. C. (2006). PennTwins: A population-based cohort for twin studies. *Twin Research and Human Genetics*, *9*(6), 998–1005.
- Coccaro, E. F., Berman, M. E., & Kavoussi, R. J. (1997). Assessment of life history of aggression: Development and psychometric characteristics. *Psychiatry Research*, *73*(3), 147–157.
- Coccaro, E. F., Fanning, J. R., Fisher, E., Couture, L., & Lee, R. J. (2017). Social emotional information processing in adults: development and psychometrics of a computerized video assessment in healthy controls and aggressive individuals. *Psychiatry Research*, *248*, 40–47.
- Coccaro, E. F., Fanning, J. R., Keedy, S. K., & Lee, R. J. (2016). Social cognition in intermittent explosive disorder and aggression. *Journal of Psychiatric Research*, *83*, 140–150.
- Coccaro, E. F., Fanning, J. R., & Lee, R. (2017). Intermittent explosive disorder and substance use disorder: analysis of the National Comorbidity Study–Replication sample. *The Journal of Clinical Psychiatry*, *78*, 697–702.
- Coccaro, E. F., Lee, R., & McCloskey, M. S. (2014). Relationship between psychopathy, aggression, anger, impulsivity, and intermittent explosive disorder. *Aggressive Behavior*, *40*, 526–536.
- Coccaro, E. F., McCloskey, M. S., Fitzgerald, D. A., & Phan, K. L. (2007). Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. *Biological Psychiatry*, *62*, 168–178.
- Coccaro, E. F., Posternak, M. A., & Zimmerman, M. (2005). Prevalence and features of intermittent explosive disorder in a clinical, setting. *The Journal of Clinical Psychiatry*, *66*(10), 1221–1227.
- Coccaro, E. F., Schmidt, C. A., Samuels, J. F., & Nestadt, G. (2004). Lifetime and 1-month prevalence rates of intermittent explosive disorder in a community sample. *Journal of Clinical Psychiatry*, *65*(6), 820–824.
- Coccaro, E. F., Solis, O., Fanning, J., & Lee, R. (2015). Emotional intelligence and impulsive aggression in intermittent explosive disorder. *Journal of Psychiatric Research*, *61*, 135–140.
- Dodge, K. A., Bates, J. E., & Pettit, G. S. (1990). Mechanisms in the cycle of violence. *Science*, *250*(4988), 1678–1683.

- Eagly, A. H., & Steffen, V. J. (1986). Gender and aggressive behavior: a meta-analytic review of the social psychological literature. *Psychological Bulletin*, 100(3), 309–330.
- Fahlgren, M. K., Puhalla, A. A., Sorgi, K. M., & McCloskey, M. S. (2019). Emotion processing in intermittent explosive disorder. *Psychiatry Research*, 273, 544–550.
- Fanning, J. R., Coleman, M., Lee, R., & Coccaro, E. F. (2019). Subtypes of aggression in intermittent explosive disorder. *Journal of Psychiatric Research*, 109, 164–172.
- Felthous, A. R., Bryant, S. G., Wingerter, C. B., & Barratt, E. (1991). The diagnosis of intermittent explosive disorder in violent men. *Bulletin of the American Academy of Psychiatry and the Law*, 19(1), 71–79.
- Fettich, K. C., McCloskey, M. S., Look, A. E., & Coccaro, E. F. (2015). Emotion regulation deficits in intermittent explosive disorder. *Aggressive Behavior*, 41(1), 25–33.
- First, M. B., Williams, J. B. W., & Gibbon, M. (1997). *Structured clinical interview for DSM-IV patient edition (SCID-P)*. Washington, DC: American Psychiatric Press.
- Forbes, D., Haslam, N., Williams, B. J., & Creamer, M. (2005). Testing the latent structure of posttraumatic stress disorder: a taxometric study of combat veterans. *Journal of Traumatic Stress*, 18(6), 647–656.
- Fossati, A., Raine, A., Borroni, S., & Maffei, C. (2007). Taxonic structure of schizotypal personality in nonclinical subjects: issues of replicability and age consistency. *Psychiatry Research*, 152(2–3), 103–112.
- Gelegen, V., & Tamam, L. (2018). Prevalence and clinical correlates of intermittent explosive disorder in Turkish psychoatric outpatients. *Comprehensive Psychiatry*, 83, 64–70.
- Genovese, T., Dalrymple, K., Chelminski, I., & Zimmerman, M. (2017). Subjective anger and overt aggression in psychiatric outpatients. *Comprehensive Psychiatry*, 73, 23–30.
- Hart, R. D., Cox, D. N., & Hare, R. D. (2003). *The hare psychopathy checklist-screening version*. Toronto, ON, Canada: Multi Heath Systems.
- Haslam, N., Holland, E., & Kuppens, P. (2012). Categories versus dimensions in personality and psychopathology: a quantitative review of taxometric research. *Psychological Medicine*, 42(5), 903–920.
- Huesmann, L. R., Eron, L. D., & Yarmell, P. W. (1987). Intellectual function and aggression. *Journal of Personality and Social Psychology*, 52(1), 232–240.
- Judd, L. L., Schettler, P. J., Coryell, W., Akiskal, H. S., & Fiedorowicz, J. G. (2013). Overt irritability/anger in unipolar major depressive episodes: Past and current characteristics and implications for long-term course. *JAMA Psychiatry*, 70(11), 1171–1180.
- Karg, R. S., Bose, J., Batts, K. R., Forman-Hoffman, V. L., Liao, D., Hirsch, E., et al. (2014). Past year mental disorders among adults in the United States: Results from the 2008–2012 Mental Health Surveillance Study. CBHSQ Data Review. Rockville (MD): Substance Abuse and Mental Health Services Administration (US).
- Kessler, R. C., & Üstün, T. B. (2004). The World Mental Health (WMH) Survey Initiative Version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI). *International Journal of Methods in Psychiatric Research*, 13(2), 93–121.
- Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62(6), 617–627.
- Kessler, R. C., Coccaro, E. F., Fava, M., Jaeger, S., Jin, R., & Walters, E. (2006). The prevalence and correlates of DSM-IV intermittent explosive disorder in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 63, 669–678.
- Knock, M. K., Stein, M. B., Heeringa, S. G., Ursano, R. J., Colpe, L. J., Fullerton, C. S., et al. (2014). Prevalence and correlates of suicidal behavior among soldiers: results from the army study to assess risk and resilience in servicemembers (Army STAARS). *JAMA Psychiatry*, 71(5), 514–522.
- Kulper, D. A., Kleiman, E. M., McCloskey, M. S., Berman, M. E., & Coccaro, E. F. (2015). The experience of aggressive outbursts in Intermittent Explosive Disorder. *Psychiatry Research*, 225(3), 710–715.

- Marcus, D. K., Ruscio, J., Lilienfeld, S. O., & Hughes, K. T. (2008). Converging Evidence for the Latent Structure of Antisocial Personality Disorder. *Criminal Justice and Behavior*, 35(3), 284–293.
- McCloskey, M. S., Lee, R., Berman, M. E., Noblett, K. L., & Coccaro, E. F. (2008). The relationship between impulsive verbal aggression and intermittent explosive disorder. *Aggressive Behavior*, 34, 51–60.
- McCloskey, M. S., Phan, K. L., Angstadt, A., Fettich, K. C., Keedy, S., & Coccaro, E. F. (2016). Amygdala hyperactivation to angry faces in intermittent explosive disorder. *Journal of Psychiatric Research*, 79, 34–41.
- McElroy, S. L. (1999). Recognition and treatment of DSM-IV intermittent explosive disorder. *The Journal of Clinical Psychiatry*, 60, 12–16. Suppl 15.
- McGrath, J., Saha, S., Chant, D., & Welhan, J. (2008). Schizophrenia: a concise overview of incidence, prevalence, and mortality. *Epidemiologic Reviews*, 30, 67–76.
- McLaughlin, K. A., Green, J. G., Hwang, I., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2012). Intermittent explosive disorder in the National Comorbidity Survey Replication Adolescent Supplement. *Archives of General Psychiatry*, 69, 1131–1139.
- Meehl, P. E., & Yonce, L. J. (1994). Taxometric analysis: I. Detecting taxonicity with two quantitative indicators using means above and below a sliding cut (MAMBAC procedure). *Psychological Reports*, 74, 1059–1274.
- Murray-Close, D., Ostrov, J. M., Nelson, D. A., Crick, N. R., & Coccaro, E. F. (2010). Proactive, reactive, and romantic relational aggression in adulthood: measurement, predictive validity, gender differences, and association with Intermittent Explosive Disorder. *Journal of Psychiatric Research*, 44(6), 393–404.
- Oliver, D. G., Caldwell, C. H., Faison, N., Sweetman, J. A., Abelson, J. M., et al. (2016). Prevalence of DSM-IV intermittent explosive disorder in Black adolescents: Findings from the National Survey of American Life, Adolescent Supplement. *American Journal of Orthopsychiatry*, 86(5), 552–563.
- Olweus, D. (1979). Stability of aggressive reaction patterns in males: A review. *Psychological Bulletin*, 86(4), 852–875.
- Ortega, A. N., Canino, G., & Alegria, M. (2008). Lifetime and 12-month intermittent explosive disorder in Latinos. *American Journal of Orthopsychiatry*, 78(1), 133–139.
- Puhalla, A. A., Ammerman, B. A., Uyeji, L. L., Berman, M. E., & McCloskey, M. S. (2016). Negative urgency and reward/punishment sensitivity in intermittent explosive disorder. *Journal of Affective Disorders*, 201, 8–14.
- Puhalla, A. A., McCloskey, M. S., Brickman, L. J., Fauber, R., & Coccaro, E. F. (2016). Defense styles in Intermittent Explosive Disorder. *Psychiatry Research*, 238, 137–142.
- Rothschild, L., Cleland, C., Haslam, N., & Zimmerman, M. (2003). A taxometric study of borderline personality disorder. *Journal of Abnormal Psychology*, 112(4), 657–666.
- Rynar, L., & Coccaro, E. F. (2018). Psychosocial impairment in DSM-5 intermittent explosive disorder. *Psychiatry Research*, 264, 91–95.
- Scott, K. M., Lim, C. C., Hwang, I., Adamowski, T., Al-Hamzawi, A., Bromet, E., et al. (2016). The cross-national epidemiology of DSM-IV intermittent explosive disorder. *Psychological Medicine*, 46(15), 3161–3172.
- Sheehan, D. V., Harnett-Sheehan, K., & Raj, B. A. (1986). The measurement of disability. *International Clinical Psychopharmacology*, 11, 89–95. Suppl, 3.
- Steakley-Freeman, D. M., Lee, R. J., McCloskey, M. S., & Coccaro, E. F. (2018). Social desirability, deceptive reporting, and awareness of problematic aggression in intermittent explosive disorder compared with non-aggressive healthy and psychiatric controls. *Psychiatry Research*, 270, 20–25.

Further Reading

- Brown, G. L., Ebert, H., Goyer, F., Jimerson, D. C., Klein, W. J., Bunney, W. E., et al. (1982). Aggression, suicide, and serotonin: relationships to CSF amine metabolites. *The American Journal of Psychiatry*, 139, 741–746.
- Brown, G. L., Goodwin, F. K., Ballenger, J. C., Goyer, P. F., & Major, L. F. (1979). Aggression in humans correlates fluid amine metabolites with cerebrospinal. *Psychiatry Research*, 1, 131–139.
- Coccaro, E. F. (2010). A family history study of intermittent explosive disorder. *Journal of Psychiatric Research*, 44, 1101–1105.
- Coccaro, E. F., Bergeman, C. S., Kavoussi, R. J., & Seroczynski, A. D. (1997). Heritability of aggression and irritability: a twin study of the buss-durkee aggression scales in adult male subjects. *Biological Psychiatry*, 41(3), 273–284.
- Coccaro, E. F., Fitzgerald, D. A., Lee, R., McCloskey, M., & Phan, K. L. (2016). Frontolimbic morphometric abnormalities in intermittent explosive disorder and aggression. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 1, 32–38.
- Coccaro, E. F., Lee, R., & Kavoussi, R. J. (2009). Inverse relationship between numbers of 5-HT transporter binding sites and life history of aggression and intermittent explosive disorder. *Journal of Psychiatric Research*, 44, 137–142.
- Coccaro, E. F., Lee, R., & Kavoussi, R. J. (2010). Aggression, suicidality, and intermittent explosive disorder: serotonergic correlates in personality disorder and healthy control subjects. *Neuropsychopharmacology*, 35, 435–444.
- Coccaro, E. F., Lee, R., McCloskey, M., Csernansky, J. G., & Wang, L. (2015). Morphometric analysis of amygdala and hippocampus shape in impulsively aggressive and healthy control subjects. *Journal of Psychiatric Research*, 69, 80–86.
- Coccaro, E. F., Siever, L. J., Klar, H. M., Maurer, G., Cochrane, K., Cooper, T. B., et al. (1989). Serotonergic studies in patients with affective and personality disorders. *Archives of General Psychiatry*, 46, 587–599.
- Duke, A. A., Bègue, L., Bell, R., & Eisenlohr-Moul, T. (2013). Revisiting the serotonin-aggression relation in humans: a meta-analysis. *Psychological Bulletin*, 139, 1148–1172.
- Fanning, J. R., Meyerhoff, J. J., Lee, R., & Coccaro, E. F. (2014). History of childhood maltreatment in intermittent explosive disorder and suicidal behavior. *Journal of Psychiatric Research*, 56, 10–17.
- Frankle, W. G., Lombardo, I., New, A. S., Goodman, M., Talbot, P. S., Huang, Y., et al. (2005). Brain serotonin transporter distribution in subjects with impulsive aggressivity: a positron emission study with [¹¹C]McN 5652. *The American Journal of Psychiatry*, 162, 915–923.
- Haslam, N. (2003). Categorical versus dimensional models of mental disorder: the taxometric evidence. *The Australian and New Zealand Journal of Psychiatry*, 37(6), 696–704.
- Lee, R., Arfanakis, K., Evia, A. M., Fanning, J., Keedy, S., & Coccaro, E. F. (2016). White Matter Integrity Reductions in Intermittent Explosive Disorder. *Neuropsychopharmacology*, 41(11), 2697–2703.
- Lee, R., Meyerhoff, J., & Coccaro, E. F. (2014). Intermittent explosive disorder and aversive parental care. *Psychiatry Research*, 220, 477–482.
- Linnoila, M., Virkkunen, 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 Sciences*, 33, 2609–2614.
- Miles, D. R., & Carey, G. (1997). Genetic and environmental architecture of human aggression. *Journal of Personality and Social Psychology*, 72(1), 207–217.
- Montalvo-Ortiz, J. L., Zhang, H., Chen, C., Liu, C., & Coccaro, E. F. (2018). Genome-wide DNA methylation changes associated with intermittent explosive disorder: a gene-based functional enrichment analysis. *The International Journal of Neuropsychopharmacology*, 21(1), 12–20.

- New, A. S., Trestman, R. F., Mitropoulou, V., Goodman, M., Koenigsberg, H. H., Silverman, J., et al. (2004). Low prolactin response to fenfluramine in impulsive aggression. *Journal of Psychiatric Research*, 38, 223–230.
- Nickerson, A., Aderka, I. M., Bryant, R. A., & Hofmann, S. G. (2012). The relationship between childhood exposure to trauma and intermittent explosive disorder. *Psychiatry Research*, 197, 128–134.
- Rosell, D. R., Thompson, J. L., Slifstein, M., Xu, X., Frankle, W. G., New, A. S., et al. (2010). Increased serotonin 2A receptor availability in the orbitofrontal cortex of physically aggressive personality disordered patients. *Biological Psychiatry*, 67, 1154–1162.
- Seroczynski, A. D., Bergeman, C. S., & Coccaro, E. F. (1999). Etiology of the impulsivity/aggression relationship: genes or environment? *Psychiatry Research*, 86, 41–57.
- Siever, L. J., Buchsbaum, M. S., New, A. S., Spiegel-Cohen, J., Wei, T., Hazlett, E. A., et al. (1999). d,l-fenfluramine response in impulsive personality disorder assessed with [18F] fluorodeoxyglucose positron emission tomography. *Neuropsychopharmacology*, 20, 413–423.
- Tremblay, R. E. (2013). Early development of physical aggression and early risk factors for chronic physical aggression in humans. In K. Miczek & A. Meyer-Lindenberg (Eds.), *Vol. 17. Neuroscience of Aggression. Current Topics in Behavioral Neurosciences*. Berlin, Heidelberg: Springer.
- van de Giessen, E., Rosell, D. R., Thompson, J. L., Xu, X., Girgis, R. R., Ehrlich, Y., et al. (2014). Serotonin transporter availability in impulsive aggressive personality disordered patients: a PET study with [11C]DASB. *Journal of Psychiatric Research*, 58, 147–154.
- Virkkunen, M., De Jong, J., Bartko, J., Goodwin, F. K., & Linnoila, M. (1989). Relationship of psychobiological variables to recidivism in violent offenders and impulsive fire setters. A follow-up study. *Archives of General Psychiatry*, 46, 600–603.
- Virkkunen, M., Nuutila, A., Goodwin, F. K., & Linnoila, M. (1987). Cerebrospinal fluid monoamine metabolite levels in male arsonists. *Archives of General Psychiatry*, 44, 241–247.