American Journal of Psychotherapy

Official Journal of AAP Founded in 1939

Volume 67 Number 3 2013

Highlights

COGNITIVE REGULATORY CONTROL THERAPIES

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PRAGMATIC PSYCHODYNAMIC PSYCHOTHERAPY FOR A PATIENT WITH DEPRESSION AND BREAST CANCER: FUNCTIONAL MRI EVALUATION OF TREATMENT EFFECTS

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Dedicated to the Transtheoretical Practice and Research of Psychotherapy

Cognitive Regulatory Control Therapies

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Cognitive regulatory control processes play an essential but typically unappreciated role in maintaining mental health. The purpose of the current paper is to identify this role and demonstrate how cognitive-behavioral and related techniques can compensate for impairments. Impaired cognitive regulation contributes to the overly intense emotional states present in anxiety disorders, depression, and personality disorders; progression of adaptive hypomania to mania; expression of psychosis in the conscious and awake state; dominance of immature defense mechanisms in borderline and other personality disorders. A wide variety of standard (monitoring, reappraisal, response inhibition, relaxation training) and more novel (suppression therapy, willful detachment, cost-benefit analysis, normalization, mature defense mechanism training) cognitive-behavioral and related techniques can be applied to compensate for cognitive regulatory control impairments, and their success probably aligns with this capacity.

KEYWORDS: cognitive regulation; cognitive behavioral therapy; psychosis; mania; mood disorders; anxiety disorders; personality disorders

INTRODUCTION

Regulation is essential to healthy functioning. Cells free of regulation transform into cancer, uncontrolled immune system responses fail against pathogens and attack the host in the case of autoimmune conditions, and well-regulated homeostatic mechanisms are required to maintain physiological parameters such as temperature and blood pressure within the range required for survival. While the role of regulatory control is well established in regards to biological functioning, its application to psychological functioning is very limited. Given that regulatory control is essential for biological functioning, and that psychological and biological activity are tightly linked, it follows that such control must play a crucial role in maintaining the integrity of psychological functioning. Mental illness to a great extent can be conceptualized as the failure of cognitive regulatory

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control processes to maintain psychological homeostasis. For example, there is solid evidence that amygdala based fear/anxiety responses are regulated by prefrontal cortex (PFC)-amygdala connectivity, typically ensuring that exaggerated anxiety reactions are attenuated to promote adaptive functioning (Goldin, 2009; Goldin, Manber, Halimi, Canli, & Gross, 2009; Larson, Schaefer, Siegle, Jackson, Anderle, & Davidson, 2006). The PFC represents the brain's master controller accounting for a third of the human cortex, and with uncontrollable acute and chronic stress its functioning is diminished impairing regulation (Arnsten, 2009; Arnsten, 2011). When cognitive regulatory control functioning is inadequate, psychological disturbances arise.

Cognitive-behavioral techniques are well established in the treatment of many mental health conditions, including anxiety disorders, depression, and to a lesser extent, psychosis (Barlow, 2005; Beck, 2005). These techniques lend themselves well to the task of compensating for inadequate cognitive regulation because they focus on dysfunctional thoughts and the behaviors following from these thoughts. Their effectiveness probably equates with how well they compensate for impaired cognitive regulatory control, although these techniques are not typically framed in this fashion. In the present paper the role that deficient cognitive regulatory control plays in mental health issues will be discussed, and the ability of cognitive-behavioral and related techniques to compensate for deficits will be shown. Some of these techniques are highly familiar, forming the basis of cognitive-behavioral therapy (CBT), while others are more unique, being designed to address specific cognitive regulatory control deficits.

ANXIETY DISORDERS

Cognitive activating appraisals underlie emotional responses (Lazarus, 1991; Lazarus, 1984; Clore & Ortony, 2000). For at least the primary emotions (fear, sadness, anger, happiness, disgust, surprise, interest, and possibly shame) a cognitive deep structure is present (Clore & Ortony, 2000). Threat or danger represents the cognitive deep structure for fear (Finlay-Jones & Brown 1981; Eley & Stevenson 2000). Hence, whenever conscious or unconscious thought processes detect threat or danger, fear arises. Several primary emotions, such as fear, clearly preceded the evolution of Homo sapiens. For example, rodents and mammals demonstrate clear fear responses. With the evolution of our much more advanced level of intelligence, the cognitive appraisals underlying fear and the other primary emotions became more intensive, extensive, and extended in time (Bowins, 2004; Bowins, 2006). Consequently the emotions following from

these cognitive activating appraisals are amplified (Bowins, 2004; Bowins, 2006). This amplification effect accounts for why we have been described as the most emotional of all species (Hebb, 1949), and it plays a major role in our propensity for anxiety and depression (Bowins, 2004; Bowins, 2006). Anxiety entails amplified fear and depression amplified sadness.

Anxiety disorders are characterized by excessive reactions to threat relevant stimuli; normal reactions to threat do not constitute a mental health problem. In many instances this excessive reactivity involves mutually reinforcing interactions between cognitions and emotional reactions (Beck & Clark, 1997; Beck, 1991; Rachman, 1998). For example, cognitive activating appraisals perceiving threat trigger feelings of fear that in turn generate an emotional climate conducive to the perception of further threats (Beck and Clark, 1997). This has been termed ex-consequentia reasoning, referring to the process by which a person deduces a threat from the fact of feeling anxious (Rachman, 1998). The mutually reinforcing nature of these threat oriented cognitions and emotional responses further amplifies anxious thoughts and affective states (Beck, 1991).

The affective component of anxiety derives from the amygdala and limbic system more generally, and substantial evidence points to excessive activity of these structures playing a key role in anxiety disorders (Goldin, 2009; Goldin, Manber, Halimi, Canli, & Gross, 2009; Larson, Schaefer, Siegle, Jackson, Anderle, & Davidson, 2006). For example, Goldin et al. (2009) found that patients with Social Anxiety Disorder experience greater limbic system reactivity to social threat stimuli than do healthy controls. Likewise, those with phobias experience exaggerated amygdala responses to specific threats (Larson, Schaefer, Siegle, Jackson, Anderle, & Davidson, 2006). A certain degree of amygdala and more general limbic system reactivity to threat relevant stimuli is adaptive, but excessive responses impair functioning and perpetuate the negative cognitive and emotional cycles characterizing anxiety disorders. It currently is unclear why the limbic system is excessively reactive to threat stimuli in some people, but it is feasible that mutually reinforcing threat oriented cognitions and emotional responses play a role by repeatedly triggering emotional reactions. In addition, genetic vulnerabilities favoring a high degree of emotional reactivity, combined with learning influences such as trauma and exposure to reactive models, might foster the development of very robust limbic system responses to threat oriented stimuli.

Exerting regulatory control over these excessive amygdala and limbic system responses is the PFC and possibly the frontal cortices more generally, with top down connectivity attenuating excessive emotional

reactions to restore adaptive functioning (Goldin, Manber, Halimi, Canli, & Gross 2009; Hartley & Phelps, 2010; Kim & Hamann, 2007; Arnsten, 2011; Arnsten, 2009; Klein, Lewinsohn & Seeley, 1996; Schardt et al., 2010; Strauss, 1969). This cognitive regulatory control appears to be essential in countering the mutually reinforcing negative cognitive-emotional cycles, and impairment to this regulation contributes to anxiety disorders (Goldin, Manber, Halimi, Canli, & Gross 2009; Arnsten, 2011; Arnsten, 2009). For instance, patients with Social Anxiety Disorder show reduced cognitive regulation related neural responses in the medial and dorsolateral prefrontal cortex to social threat stimuli compared to healthy subjects (Goldin, Manber, Halimi, Canli, & Gross 2009).

Defective cognitive regulatory control is also evident in regards to a so-called fear circuit whereby information from sensory systems is relayed directly from the thalamus to the amygdala without first being processed by the higher cortical centers (Ledoux, 1994). This circuit enables rapid responses in dangerous situations, thereby promoting survival and evolutionary fitness. However, as this immediate processing is unfolding the thalamus relays the information to higher cortical centers that can then influence amygdala responses (Ledoux, 1994). For example, you turn a corner and encounter an attack dog, resulting in an instantaneous flight/fight response based on the thalamus-amygdala fear circuit. A second or two later you settle down as higher cortical centers analyze this to be a friendly dog based on the wagging tail, and dampen the amygdala based reaction. Excessive anxiety responses to potentially threatening stimuli (as occurs in panic attacks) likely involve deficient higher cortical regulation of these fear-circuit based responses.

Cognitive-behavioral techniques commonly applied for treating anxiety disorders enhance cognitive regulatory control over amplified anxiety responses. Patients with social anxiety who complete cognitive behavioral therapy demonstrate greater PFC responses and reduced amgdala activation (Goldin, 2009). Even when a genetic polymorphism contributes to heightened amygdala responses to fear-inducing pictures, a cognitive behavioral strategy consisting of willful detachment from emotions arising out of exposure to these pictures, attenuates the fear response (Schardt et al., 2010). This effect is mediated by increased functional connectivity between the ventromedial/ventrolateral prefrontal cortices and the amygdala, reducing responses of the latter (Schardt et al., 2010). Metacognitive therapy, using attention shifts to produce detachment from the fear stimulus, has shown promise for anxiety disorders (Wells, 2008). Prefrontal cortex and amygdala connectivity also regulates phobic reac-

tions derived from increased amygdala activation (Hermann, Schafer, Walter, Stark, Vaitl, & Schienle, 2007). Amygdala responses to positive emotions also seem to be regulated by the prefrontal cortex, but in a fashion that increases rather than decreases them (Kim & Hamann, 2007).

Prefrontal cortex-amygdala connectivity down-regulates excessive negative emotional responses, a process that involves increased PFC activity to reduce excess limbic system activity (Hartley & Phelps, 2010; Salzman & Fusi, 2010; Schardt et al., 2010; Arnsten 2011; Arnsten, 2009). Cognitive-behavioral techniques facilitate this top-down emotional regulation by either cognitive reappraisal of the situation, dissociating from adverse stimuli, changing the stimulus-response association, or activating psychological defensive processes (Ochsner, Bunge, Gross, & Gabrieli, 2002; Salzman & Fusi, 2010; Schardt et al., 2010; Wells, 2008; Bowins, 2004), and can occur unconsciously or consciously (Van Gaal, Lamme, Fahrenfort, & Ridderinkhof, 2011). As an example cognitive reappraisal replaces thoughts entailing threat-oriented cognitive activating appraisals producing fear/anxiety responses with thoughts that do not involve these cognitive activating appraisals. In some instances thoughts involving cognitive activating appraisals for positive emotions are introduced. Mutually reinforcing cycles of negative cognitions and emotional responses are interrupted, and at times replaced by positive cycles. For example, instead of being viewed as a threat a new job is seen as a chance to grow, producing feelings of interest and related thoughts that mutually reinforce one another.

Suppression therapy defensively blocks adverse memories associated with traumatic occurrences (Anderson & Levy, 2009), dissociating the patient from the event. Traumatic memories then lose their ability to elicit anxiety. Willful detachment relies on attention shifts to dissociate an individual from stressful input (Schardt et al., 2010). Absorption in a more positive focus or placing the experience in a psychological compartment enhances adaptive dissociation toning down anxiety responses (Bowins, 2012). Exposure and response prevention therapy replaces anxiety responses with non-anxious responses by altering the stimulus-response linkage. For example, an individual assumes that failure to hand wash will produce deadly contamination, but by inhibiting the anxious response the person learns that no such event occurs. Interestingly, compulsive behaviors might actually limit excessive anxiety generated by obsessions: Obsessions involve recurrent intrusive thoughts generating anxiety, while compulsions are capable of reducing anxiety associated with the obsession (Storch, Abramowitz, & Goodman, 2008). The amplified threat percep-

tion inherent in the obsession produces an adverse cognitive and emotional state, and by performing the mental and/or physical behavior (the compulsion) the adverse state is at least temporarily terminated (Abed & de Pauw, 1998/1999; Stein et al., 1992). By reducing anxiety the compulsive behavior is negatively reinforced (Abed & de Pauw, 1998/1999; Stein et al., 1992).

Cognitive-behavioral and related techniques can restore healthy psychological defensive functioning, an important component of top-down regulation of excessive negative emotions. Healthy psychological functioning involves a positive bias, such as placing a self-enhancing spin on experiences (Beck, 1991; Beck & Clarke, 1997; Bowins, 2004). With a shift to anxiety or depression the positive bias is neutralized, and as mental illness develops, a negative bias ensues (Beck, 1991; Beck & Clarke, 1997; Bowins, 2004). Cognitive therapy identifies negative automatic thoughts and underlying schemata associated with the negative bias. The person learns to question and test these negative cognitions and generate positive replacements. Through this process the negative bias of mental illness is shifted to a healthy self-enhancing positive bias, thereby restoring the natural psychological defensive capacity protecting against anxiety and depression.

DEPRESSION

The root emotion of depression is sadness. The deep structure of sadness is loss (Finlay-Jones & Brown, 1981; Shrout, Link, Dohrenwend et al., 1989; Eley & Stevenson, 2000; Beck, 1991). When cognitive activating appraisals detect a loss, feelings of sadness arise that are often amplified due to how human intelligence makes the cognitive activating appraisals more intensive, extensive, and adds a temporal dimension (Bowins, 2004; Bowins, 2006). For example, a person is let go from a good job and intensifies the loss by thinking of associated losses such as not seeing friendly coworkers anymore. Thoughts such as "I might never be able to find a job as good as this one" extend the loss. These cognitive activating appraisals can repeat over days, weeks, months, and even years. This negative loss-oriented perspective contributes to depression and constitutes a key component of the negative automatic thoughts and schemata that are at the core of depression. Negative thoughts and feelings of sadness/depression mutually reinforce each other, the loss-oriented thoughts producing an emotional climate conducive to the perception of further losses, which in turn generate more feelings of sadness and depression (Beck, 1976; Beck, 1991).

Much as with anxiety disorders, depression appears to entail too little cognitive regulatory control over excessive limbic system based feeling of sadness and related negative emotions (Beauregard, Paquette, Levesque, 2006; Brambilla, Glahn, Balestrieri, & Soares, 2005; Philippot & Brutoux, 2008; Salzman & Fusi, 2010; Arnsten, 2011; Arnsten, 2009). For example, compared to non-depressed controls, depressed individuals show impaired down-regulation of sad feelings, likely making it more difficult for them to overcome depression-related emotions (Beauregard, Paquette, Levesque, 2006). Neuroimaging studies reveal enhanced activation in limbic and emotion-related structures and attenuated activity in frontal regions associated with emotion regulation and higher cognitive functions, indicating a lack of inhibition by higher-order cognitive systems on limbic and emotion-related structures (Hamilton et al., 2012; Cusi et al., 2012; Arnsten, 2011; Arnsten, 2009). Furthermore, deficits pertaining to cognitive control might even increase with each depressive episode and persist after remission suggesting a "scar' on cognitive control processes (Vanderhasselt & DeRaedt, 2009).

Cognitive-behavioral techniques can help compensate for impaired cognitive regulatory control over the excessive limbic system based emotional responses characterizing depression. Cognitive reappraisal, a powerful therapeutic strategy for depression, alters negative automatic thoughts and underlying schemata in such a way to remove or reduce the perception of loss. Consequently, cognitive activating appraisals for sadness are eliminated or decreased. For example, loss of a partner who was truly incompatible with the individual is shifted to an opportunity for a more compatible relationship. Mutually reinforcing cycles of negative thoughts with a loss theme and feelings of sadness are then terminated, and in some cases replaced with mutually reinforcing positive cycles. This is in effect restoring the natural defensive positive spin on events that characterizes non-depressive mental health (Beck, 1991; Beck & Clarke, 1997; Bowins, 2004). Consistent with this process cognitive reappraisal has been found to improve emotional self-regulation (Beauregard, 2007)

BIPOLAR DISORDERS

Depression constitutes a key component of bipolar disorder, and the cognitive-behavioral principles used to treat unipolar depression appear to apply to bipolar depression, producing similar outcomes for the most part (Mansell, Colom, & Scott, 2005). In addition to depression, hypomania and mania are essential components of bipolar disorder (depression-hypomania bipolar II [BPII], and depression-mania bipolar I [BPI]).

Currently the relationship between depression, hypomania, and mania is not well understood (Bowins, 2008). The prevailing assumption is that hypomania is quite rare and frequently leads to mania. This perception is based in part on the DSM-IV-TR criteria requiring hypomania to persist for four days (First, Frances, & Pincus, 2002), thereby increasing the perception that it progresses to mania more often than it does, as only the more extreme instances are included. Evidence indicates that the modal timeframe for hypomania is really only one to three days (Akiskal & Pinto, 1999; Benazzi & Akiskal, 2001; Wicki & Angst, 1991).

Hypomania is vastly more common than mania and rarely progresses to mania, particularly in the absence of antidepressant treatment (Akiskal, 1996; Akiskal, 2005; Akiskal et al., 2003; Cassano, Akiskal, Savino, Musetti, & Perugi, 1992; Coryell et al., 1989; Klein, Lewinsohn, & Seeley, 1996; Weinberger, Berman, & Zec, 1986). Hypomania occurs on a one-to-one ratio with depression and is estimated to affect 3% to 6% of the general population, whereas mania is much less common affecting 1% (Akiskal, 1996; Akiskal, 2005; Weissman, Berman, & Zec, 1986). Subthreshold hypomanic symptoms are almost certainly more commonplace. Even in those who have full-blown manic episodes, 50% to 60% will also experience milder hypomanic episodes not progressing to mania (Cassano, Akiskal, Savino, Musetti & Perugi, 1992; Coryell et al., 1989). Amongst those who experience cyclothymia only 6% develop mania (Akiskal, 1996). Hence, most of those with hypomania remain stable and do not progress to mania (Akiskal, 1996; Klein, Lewinsohn & Seeley, 1996).

Another important misperception regarding hypomania is that it is maladaptive, an understandable perspective if only more severe expressions that lead to mania are included. Hypomania is actually a very adaptive state providing increased mental, physical, and social behavior that typically enhances overall functioning, and certainly relative to depression (Akiskal & Pinto, 1999; Jamison et al., 1980; Himmelhoch, 1998). Furthermore, it appears to represent a natural defensive compensation for depression and various forms of anxiety, in particular social anxiety (Perugi et al., 1999; Akiskal & Pinto 1999; Jamison et al., 1980; Himmelhoch, 1998; Bowins, 2008). This defensive function seems to consist of temporarily overriding or interrupting depression to restore adaptive functioning (Bowins, 2008). Expressed in an ongoing fashion as a personality type, hyperthymia-hypomania frequently results in enormous success and serves as a natural defense against the onset of depression (Chapman & Chapman, 1986; Akiskal, 1996; Akiskal & Pinto, 1999). Those with

hyperthymic personality who eventually succumb to depression usually only do so in later life (Akiskal & Pinto, 1999).

Hypomania is so adaptive and so rarely leads to mania that it should not even be considered a mental health problem (Bowins, 2008). It really has no place in a diagnostic manual beyond indicating its defensive and compensatory role for depression and social anxiety. In contrast to hypomania, mania is typically maladaptive, due to the unsustainable lack of sleep and energy, unfocused activity, and disconnect with reality. A spectrum exists from subthreshold hypomania, hypomania, subthreshold mania, to full-blown mania (Bowins, 2008). There is no absolute cut-off point distinguishing mania, but at a certain level of intensity adaptive and limited hypomania shifts to maladaptive and out-of-control mania. The question arises: Why does this shift occur in some individuals while seemingly not being an issue in the vast majority of people experiencing hypomania? A related question is why hypomania in a given individual will often not progress to mania, but in some instances does? It appears that there is a mechanism designed to down-regulate and terminate the hypomania defense when costs exceed benefits (Bowins, 2008). Excessive costs, such as marked sleep impairment, inability to concentrate, and reduced contact with reality activate the cognitive regulatory control mechanism, preventing the development of full-blown mania.

In many individuals this cognitive regulatory control process is intact and hypomania never progresses to full-blown mania. In others it is impaired (at least under certain conditions), and hypomania progresses to mania (Bowins, 2008). The effective operation of this cognitive regulatory control process likely provides for BPII, while impairment to it constitutes BPI (Bowins, 2008). In susceptible individuals psychoactive substances such as antidepressants, alcohol, and illicit drugs seem capable of impairing the relevant cognitive regulatory control mechanism. This mechanism can also be impaired due to genetic vulnerabilities, at least in interaction with environmental insults. In bipolar disorder emotional dysregulation and cognitive control deficits have been observed, although to date this represents a novel line of investigation (Novak & Sprah, 2010).

Given that deficient cognitive regulatory control processes seem to play a major role in the progression to mania, and cognitive-behavioral and related techniques can compensate for impaired cognitive regulatory control processes, it follows that there is potential for these techniques in bipolar disorder beyond dealing with the depressive component. The first step consists of monitoring mood and behavior for signs of dysfunction. Common warning signs include markedly reduced sleep, excessive energy,

unfocused thoughts shifting rapidly from one topic to another, pronounced irritability or euphoria, the temptation to spend money or engage in atypical behaviors, increased alcohol or drug use, and odd thoughts. Of particular significance are triggers for past manic episodes. For instance, in some people greatly increased stress can trigger a manic state perhaps by impairing sleep. Once signs of impending mania are detected through monitoring, behavioral strategies can be implemented to prevent a progression to mania. For example, distractions can be minimized prior to sleep, and relaxation strategies, such as meditation and Yoga, can be engaged in to produce a more settled psychological state. Manic episodes can often be halted early in their development by getting adequate sleep. If alcohol triggers the progression to mania, then consumption should be terminated and alcohol removed from the dwelling. The person should always be advised to contact his/her therapist immediately when monitoring suggests a potential problem.

Assuming that impaired cognitive regulation allows adaptive hypomania to progress to maladaptive mania, consciously applying a cost-benefit analysis to behavior might compensate for deficient regulation. A case example will illustrate the process. A patient, who will be named Maria, has a lengthy history of bipolar illness. She has frequent instances of hypomania during which she becomes mentally, physically, and socially active, thus enabling her to be more productive (relative to her dysfunctional state when depressed). She has experienced milder subthreshold mania and less frequently, full-blown manic episodes. Maria has tried several medications but limited effectiveness and excessive side effects have made her very resistant to being medicated, and she prefers to work with the problem in a "natural fashion" without pharmaceuticals.

In treatment Maria has learned to monitor her mood state and behavior, and is well aware of her current mental state pertaining to depression, hypomania, and mania. She is also aware that when crossing into the manic realm she is highly tempted to spend well beyond her means and to "party" with alcohol and drugs. With guidance she has learned to consciously weigh the costs and benefits of different actions, and to intervene when the costs exceed the benefits. For instance, when she is tempted to spend she stops and evaluates the situation using concrete examples provided in therapy. She asks herself whether the item is really needed; the answer is almost always no. She then considers how the benefit of temporarily feeling better will be vastly outweighed by the cost of financial debt and depression resulting from the loss of money and control. Discussing the issue with her parents and friends helps clarify any doubts. If tempted to party the

costs both financial and of disruption to her routine is readily seen as greatly exceeding the benefit of immediate gratification.

In effect Maria is restoring the impaired cost-benefit analysis that is hypothesized to play a key role in the bipolar cognitive regulatory control mechanism. Behavioral strategies including meditation and relaxation exercises are then engaged in to ensure that the developing manic state is cut off or at least does not exert such a cost on her. Regarding spending, she gives her credit card, bank card, and excessive cash to her parents, and avoids the trigger of stores by remaining away from them. She has largely replaced her "party friends" with others not oriented to such behavior, and resists calling those who enjoy partying to prevent an escalation of mania. Maria uses sedative and antipsychotic medication that we have contracted for her to use on an as-needed basis only, and she takes them if the other strategies are not limiting the progression to mania. Thus far this cost-benefit strategy has been successful in preventing mania from developing. Mania frequently involves psychosis, another condition influenced by cognitive regulatory control processes.

PSYCHOSIS

Deficient cognitive regulation appears to play a major role in psychosis (Carhart-Harris et al., 2012; Arnsten, 2011; Arnsten, 2009; Bowins, 2011). Psychosis, also referred to as positive symptoms in the context of schizophrenia, consists of extreme alterations in thought content (delusions), thought form, and sensory perceptual experiences (hallucinations). The evolution of human intelligence has provided for an extensive range of these cognitive capacities (Bowins, 2011). Thought content alterations involve cognitive distortions with milder versions slightly altering our perception of reality, such as by placing a self-enhancing or diminishing spin on experience. Moderate cognitive distortions consist of excessive fantasy involvement, magical thinking, and over-valued ideas. More extensive cognitive distortions represent actual psychosis, namely delusions (Bowins, 2004; Bowins, 2006). In regards to thought form a natural range exists from highly logical thinking to loose associations, circumstantiality, tangentiality, fragmentation, blocking, and derailment. People vary in terms of how tightly or loosely structured their thoughts typically are. Furthermore, the thought form of even a tight thinker can become circumstantial, tangential, fragmented, or blocked at times, underscoring the potential range of expression. Within the general population the intensity and quality of sensory experiences also vary, such as seeing concrete images in abstract forms like clouds, illusions, and hallucinatory

experiences at the border of sleep (Choong, Hunter, & Woodruff, 2007). Evidence supports psychotic experiences as being on the extreme end of a normal continuum (Fowler, Garety, & Kuipers, 1998; Kingdon & Turkington, 2005).

Given that extreme cognitive distortions, thought form variants, and sensory perceptual experiences occur on a natural cognitive continuum, why do we not routinely experience them? Actually, we do in sleep with bizarre ideation, very loose and fragmented thought form, and false sensory experiences. However, in the conscious and awake state these experiences are the exception rather than the rule. The reason appears to be that cognitive regulatory control processes block these experiences from the conscious and awake state to facilitate reality congruency, which is necessary for adaptive functioning under the vast majority of circumstances (Bowins, 2011). Psychotic experiences involve a loss of contact with reality and hence impair functioning within reality. For example, if you believe that all other employees and your boss are conspiring to kill you, and hear voices discussing how this will occur, it is very unlikely that your performance will even be marginally adequate. You will probably lose your job and end up being hospitalized. During sleep, when reality congruency is not an issue, the cognitive regulatory controls are relaxed and extreme cognitive distortions, thought form variants, and sensory perceptual experiences are routinely expressed. Cognitive regulatory controls can also be relaxed to facilitate psychological defensive functioning. For example, when someone close to you passes away, it is common to experience auditory, visual, and general feeling hallucinations of that person (Olson, Suddeth, & Peterson, 1985). These experiences appear to restore the lost sensory and related emotional stimulation (Bowins, 2011).

Psychotic manifestations form a common component of schizophrenia. It has been hypothesized that the neural damage associated with the negative symptoms of schizophrenia impair or damage the cognitive regulatory control processes, allowing extreme cognitive distortions, thought form variants, and sensory perceptual experiences to persistently intrude into the conscious and awake state (Bowins, 2011). To a large extent schizophrenia consists of impaired regulation of impulses, urges, wishes, and emotional reactions, producing bizarre behavior. The cognitive regulatory control processes normally preventing psychotic equivalents from intruding into the conscious and awake state are likely centered in the prefrontal cortex—The PFC plays an important role in cognitive control generally (Morsella & Krieger, & Bargh, 2010; Dumontheill et al., 2010; Savine & Braver, 2010; Brass et al., 2005), and inhibitory control

over inappropriate or maladaptive emotional and cognitive behaviors (Dietrich, 2003; Lhermitte, Pillon, & Serdaru, 1986). There is evidence that the prefrontal cortex undergoes extensive changes in schizophrenia that could damage or impair the functioning of cognitive regulatory control processes residing there (Fuster, 2001; Molina et al., 2005; Morice & Delahunty, 1996; Ragland et al., 2009; Weinberger, Berman, & Zec, 1986; Woo & Crowell, 2005; Arnsten, 2009; Arnsten, 2011). Furthermore, impaired cognitive regulatory control appears to be mediated, at least in part, by sustained dopamine levels in the PFC (Arnsten, 2009, Arnstend, 2011), with the dopamine system of the PFC being unusually vulnerable to dysfunction or alteration (Diamond, 2011).

Supporting the role of the PFC in regulating psychotic level thoughts and sensory perceptual experiences, this structure is less active during dreaming when psychotic equivalents are expressed (Solms & Turnbill, 2002), and the bizarreness of a dream is directly related to the degree of hypofunction of the prefrontal cortex (Hobson, Pace-Schott, & Stickhold, 2000). In addition, creative idea generation involves reduced cognitive control, particularly of the PFC (Chrysikou & Thompson-Schill, 2011). Psychedelic drugs were commonly thought to work by increasing brain activity but experiments with psilocybin, a psychedelic in magic mushrooms, has demonstrated reduced activity in control centers such as the PFC, thalamus, and anterior and posterior cingulated cortices (Carhart-Harris et al., 2012). The greater the reduction in activity within these control regions the more intense the self-reported psychedelic experiences (Carhart-Harris et al., 2012). Conceivably the link between marijuana use and psychosis might work in the same way. Excessive activation of the limbic system has been associated with schizophrenia and psychosis in particular (Andreasen et al., 1994; Suddath et al., 1989). Impairment to cognitive regulatory control processes that normally block over-activity of the limbic system might allow for the emergence of psychosis (Arnsten, 2011; Arnsten, 2009; Modinos, Ormel, & Aleman, 2010). In support of this supposition, cognitive control of at least delayed emotional responses is impaired in those with schizophrenia (Carter, Ursu, Mizzenberg, Yoon, Solomon, & Ragland, 2011; Ursu, Kring, Gard, Minzenberg, Yoon, & Ragland, 2011).

If impaired cognitive regulatory control is central to psychosis, then psychotherapeutic strategies designed to compensate might have a role to play in treatment. Cognitive-behavioral strategies have been successfully applied to treat psychosis (Tarrier & Wykes, 2005; Turkington et al., 2008; Kuller & Bjorgvinsson, 2010; Landa et al., 2006; Valmaggia et al., 2008),

and they likely work by enhancing deficient cognitive regulation. A hallmark of the CBT approach to psychosis is normalization (Kingdon & Turkington, 1994; Landa et al., 2006). Psychotic manifestations are seen as being on the extreme end of a normal continuum, and the purpose of therapy is to bring the distortions back to a more moderate level (Kingdon & Turkington, 1994; Landa et al., 2006). Cognitive-behavioral techniques for psychosis are almost entirely focused on the thought content component (Beck et al., 2009). Even for hallucinations, beliefs regarding the sensory perceptual experience are seen as the key factor generating distress, and attempts are made to alter these beliefs such that the hallucination is no longer disturbing (Kuller & Bjorgvinsson, 2010). This approach is supported by how people experiencing hallucinations during grieving (or as a normal state of affairs) are often not distressed, and if anything more worried about how others will view them (Kuller & Bjorgvinsson, 2010).

Cognitive-behavioral strategies can be used to normalize psychotic level beliefs (Kingdon & Turkington, 1994; Landa et al., 2006). The delusion is never directly challenged to ensure a good therapeutic alliance. One strategy involves encouraging the individual to provide evidence for the belief (Landa et al., 2006; Kuller & Bjorgvinsson, 2010; Beck et al., 2009). Typically, there is no concrete evidence for the delusion, only the drawing of tenuous connections between largely unrelated events. Frequently, the person will maintain that this evidence is sufficient, but in some instances will begin to doubt the belief. An important assumption of CBT for psychosis is that delusions are not necessarily fixed but vary in degree of adherence (Landa et al., 2006; Kuller & Bjorgvinsson, 2010; Beck et al., 2009). Even without antipsychotic medication some people have doubts about their delusions, while others rigidly adhere to them. With the right input, firmly held beliefs can be altered, or the strength of adherence weakened.

Another cognitive-behavioral strategy consists of generating alternative perspectives (Landa et al., 2006; Kuller & Bjorgvinsson, 2010; Beck et al., 2009). Very often those with delusional beliefs assume that their view is the only right one, and by considering possible alternatives the delusional belief can be weakened. It has been found that group CBT can be particularly effective for managing persecutory delusions (Landa et al., 2006). In a group setting an individual with a persecutory belief can see how others adhere rigidly to their beliefs even when clearly wrong, and the social support assists in generating alternative perspectives (Landa et al., 2006). When appropriate, experiments can be set up to compare their

explanation against alternatives. Through the various cognitive-behavioral interventions mentioned, an individual learns to normalize delusional thinking. Cognitive regulatory control over reality incongruent cognitions and the behaviors arising from these thoughts is then improved. Teaching an individual to inhibit overt behavior that is clearly maladaptive, such as aggressive and sexual displays, can further enhance control over the behavioral component. Combining psychotherapy with antipsychotic medication typically improves the overall outcome.

To illustrate the use of these techniques a case example is helpful. A male patient, who will be called Ted, developed schizophrenia in his twenties derailing his (until that point) successful premed studies. He developed the delusion that while at a party where he indulged in illicit drugs, a child was murdered and he was involved. A delusional system grew containing the narrative that his accomplices are after him to ensure that he does not talk about the crime, and at times the FBI and other authorities check up on him as well. When I encountered Ted, he had moved far away from the supposed crime scene and interpreted sounds at night as evidence that someone was trying to get him. Without challenging the delusion, we went over alternative explanations for events. For example, even though it was possible that the sounds at night might be coconspirators trying to break in, they might possibly arise from the wind blowing tree branches against the house or raccoons rummaging at garbage. He accepted these alternative explanations and was able to generate others. Regarding the supposed crime, he accepted that it was feasible that the drugs he took could have produced a hallucination of the murder, particularly if spiked with another substance. We went on to look for evidence and to test his views. He was unable to provide evidence for his perspective and he was aware that drugs could trigger hallucinations. Although too frightened to consult newspaper stories for murders of children in the area where the party occurred, Ted was able to experiment with the causes of the sounds he heard at night, even getting out of bed to see what was really causing them; most often it was the wind. With the addition of antipsychotic medication he was able to understand that the whole event was illogical, because if true someone would have reported the murder and the investigation would have uncovered the guilty parties. The highly distorted delusional beliefs were then normalized, compensating for deficient cognitive regulatory control.

Another component of CBT for psychosis involves the stress-diathesis model, maintaining that stress can elicit psychosis in those who are vulnerable (Kingdon & Turkington, 2005). The early identification of

stressors producing psychosis can lead to actions that attenuate it. For example, if an individual is aware that relationship stress produces disturbing beliefs, the individual can try to remedy the relationship or withdraw from it if a solution is not possible. Ted experienced his father as very stressful because he could not accept Ted's illness and viewed his son as being lazy or depressed. This stress certainly did not help with Ted's psychosis and appeared to worsen it when the criticisms escalated. Ted brought his father to a couple of sessions where I explained the nature of the illness but to no avail. Ted then went to live with his more understanding mother. By removing the stress of his father's criticisms Ted's efforts to normalize his own thinking advanced.

While psychosis is integral to schizophrenia, it is also common in mania, and BPI overlaps with schizophrenia. From a cognitive regulatory control perspective this relationship is understandable. Accounting for the presence of psychosis in mania, neural damage impairing the cognitive regulatory processes preventing the conversion of hypomania to mania might also impair those blocking the expression of extreme cognitive distortions, thought form variants, and sensory perceptual experiences in the conscious and awake state. The neural damage associated with negative symptoms of schizophrenia could impair the cognitive regulatory processes relevant to both psychosis and mania. Mania is not typically present in schizophrenia though, in part because clinical depression, and hence hypomania, does not always occur in schizophrenia. In addition, the neural damage underlying negative symptoms might impair the capacity for a hypomanic defensive response to depression. The latter is feasible given that negative symptoms involve an absence of activity, and hypomania is a very active response to depression providing increased mental, physical, and social activity.

PERSONALITY DISORDERS

The psychotic spectrum personality disorders (paranoid, schizoid, and schizotypal) involve inadequate regulation of extreme cognitive distortions, thought form variants, and sensory perceptual experiences. The cognitive-behavioral techniques applied to psychosis can be used for these disorders. Overly intense negative emotions, suggesting inadequate regulation of limbic system emotional responses, are found in many personality disorders. Intense anxiety occurs with avoidant, dependent, obsessive-compulsive, histrionic, and borderline personality disorders (BPD). With BPD a range of poorly regulated emotional states is the norm and intense sadness/depression and anger/aggression are common (Schulze et al.,

2011). Much as with anxiety disorders and depression, evidence supports a combination of heightened limbic system reactivity, and attenuated activation of the frontal cortex impairing cognitive regulatory control of emotional responses in BPD (Schulze et al., 2011).

A major factor contributing to the inadequate control of emotions and behavior in BPD and some other personality disorders is impaired regulation of defense mechanisms (Finzi-Dottan & Karu, 2006; Bowins, 2010). With BPD immature defenses, such as acting out, idealization/devaluation, and splitting are the norm (Kernberg, 1976; Bond, Paris, & Zweig-Frank; 1994). These immature defenses are applied to deal with a wide range of stressors leading to overly intense emotional reactions. For example, if perceptions of the therapist shift back and forth from perfect to horrible, emotional reactions in therapy will necessarily be extreme, and with the use of the acting out defense they will be expressed in an unfiltered form. Acting out demonstrates the poorly regulated control of impulses seen in this disorder (Kernberg, 1976). Impulse problems in this and other personality disorders entail an obvious deficiency in cognitive regulatory control.

In contrast to immature defenses, mature defenses provide for solid regulation of emotions and behavior. For example, sublimating negative emotional energy into constructive activities turns a negative state into a positive one. Suppression enables neurotic impulses to be inhibited, thereby providing an opportunity to evaluate the situation more fully and act in a constructive fashion. Formal suppression therapy can help block adverse memories associated with traumatic occurrences commonly experienced in BPD (Anderson & Levy, 2009). Anticipating problems and taking proactive steps to manage them attenuates circumstances that give rise to negative emotions. Placing a humorous spin on a negative event limits adverse emotions and increases positive ones. Many successful comedians have turned this defense into a career. Progress in therapy involves replacing immature defenses with mature ones (Perry & Bond, 2012).

Therapeutic strategies to shift from immature to mature defenses can be applied to assist those with BPD and other personality disorders. The starting point is psycho-education pertaining to types of defense mechanisms, their role in regulating emotions and behavior, and the ones preferentially used by the individual. Self-monitoring is initiated to assess the defenses applied, and understand both the contributing circumstances and outcome of their use. Guided instruction is to be given regarding the application of mature defenses, with practice in sessions using patient-

therapist interactions as a platform in individual therapy, and interactions with other group members and the facilitator in group therapy. Monitoring is then encouraged to assess the outcome of applying more mature defenses in terms of both emotional reactions and overall functioning. A very important aspect of successful therapy for BPD involves modeling mature and adaptive defense mechanism regulation starting with how to manage situations in the therapeutic relationship. Providing a stable, professional, and supportive therapy structure with clear limits and boundaries helps the person with this condition regulate defense mechanisms and relevant behavior (Bowins, 2010).

CONCLUSION

Impaired cognitive regulation plays a major role in mental health conditions including anxiety disorders, depression, mania, psychosis, and personality disorders. Excessive limbic system activity related to deficient cognitive regulatory control allows dysfunctional emotional and behavioral states to be expressed. Standard cognitive-behavioral techniques, such as monitoring, reappraisal, response inhibition, and relaxation training can compensate for impaired cognitive regulatory control. More novel techniques, such as suppression therapy and willful detachment in anxiety disorders, consciously applying a cost-benefit analysis in bipolar disorder, normalization for psychosis, and guided application of mature psychological defenses in borderline and other personality disorders, offer a way to treat the respective conditions by improving cognitive regulatory control functioning. The success of cognitive-behavioral and related psychotherapy techniques probably equates with their ability to compensate for deficient cognitive regulatory control.

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