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Why Should Clinical Researchers Care About Cognitive Affective Neuroscience?

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We are living in a time of unprecedented advancements in the methods and technologies that are available to study the neural basis of psychological disorders. Just over a century ago, physicians and researchers relied heavily on studying brain lesions resulting from devastating illnesses and injuries in order to gain insight into the particular function of a neural region and the extent to which it might contribute to normal and abnormal human behavior. As one classic example, by observing the erratic behavior of Phineus Gage following his railway injury, it became clear that an intact prefrontal cortex is crucial for effective emotion regulation and rational decision making (Harlow, 1868). Fortunately, researchers of today can now rely not on unfortunate happenstance, but on quick and noninvasive assessments using functional neuroimaging in order to pinpoint the neural correlates of even the most complex clinically relevant psychological processes, including cognition and emotion. One of the most important technological developments that has spawned new insights into the

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revised INSTRUCTIONS *for* AUTHORS

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neural correlates of cognitive and affective processes is functional magnetic resonance imaging (fMRI), which has the spatial and temporal resolution necessary to investigate task-related changes in neural functioning within millimeters of neural structures of interest (e.g., Bandettini, 2007).

In parallel with these technological advances, entirely new interdisciplinary research domains have emerged, including affective neuroscience (Davidson & Sutton, 1995), and more broadly, social cognitive affective neuroscience (SCAN; Ochsner & Gross, 2008; Ochsner & Lieberman, 2001). SCAN research is predicated on the assumption that multilevel models that incorporate a variety of factors, including behavioral, environmental, and neurobiological variables, should be employed in order to best explain psychological functions (Ochsner & Gross). Such multilevel modeling necessarily requires an interdisciplinary approach to research design and interpretation. Using cross-disciplinary approaches, researchers in these fields have contributed greatly to our knowledge regarding the neural correlates of both normal and abnormal cognitive and affective functioning (the interested reader is referred to recent literature reviews that cover cognitive and affective neuroscience research in both healthy and clinical samples; e.g., Dillon & Pizzagalli, 2007; Luna, Padmanabhan, & O'Hearn, 2010; Mitterschiffthaler et al., 2006; Sweet, 2011; Taylor & Liberzon, 2007; Walter, Berger, & Schnell, 2009).

Despite the advancements in our basic knowledge of the neural correlates of clinically relevant psychological processes, this increased knowledge has yet to be accompanied by significant improvements in mental health outcomes (Gould & Manji, 2004; Insel, 2009). Indeed, psychiatric disorders remain highly prevalent, as evidenced by data from the National Comorbidity Survey Replication suggesting that nearly half of all Americans will meet diagnostic criteria for a psychological disorder at some point in their lifetime (Kessler, Berglund, et al., 2005). Moreover, despite increased rates of mental health treatment access over the 10-year period from 1992 to 2002, there was no evidence of decreased disability during the same time period, and lost income due to mental health disability has increased over time (Kessler, Demler, et al., 2005; Kessler et al., 2008). As such, early speculations among researchers regarding the potential for neuroimaging methods to identify biomarkers that would inform the development of novel and effective treatments for psychological disorders do not appear to have come to fruition.

There are a number of plausible explanations for why advancements in functional neuroimaging have not led to significant reductions in the prevalence or severity of psychiatric illness thus far. First, cognitive affective neuroscience is a field in its infancy. Indeed, many functional neuroimaging findings are new and yet to be replicated, and in some cases the findings directly contradict one another across studies, thus complicating attempts to apply fMRI findings to the development or application of clinical interventions. Moreover, there is a historical precedence for a temporal lag between paradigm shifts in the field of psychology and the actual integration of novel theoretical developments into clinical practice. For example, although learning theorists including Hull and Skinner began theorizing about behavioral principles as early as the 1930s, the application of behavioral learning principles, including operant conditioning and shaping, into psychological interventions was gradual, with behaviorism ultimately surpassing psychoanalysis in the 1970s as the dominant clinical orientation (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008). Second, clinical researchers and clinicians alike may question the utility of considering and incorporating expensive neuroscience methods into their research and clinical assessments and interventions unless they provide significantly more explanatory power than more affordable and accessible self-report and behavioral assessments. However, there is ample evidence that the interactions between neurobiological measures and individual difference and/or environmental factors may account for more variance in mental health outcomes than the main effects of any one factor alone (Beauchaine et al., 2008). Finally, the limited progress in integrating neuroscience into clinical research and practice may be due in part to a lack of interdisciplinary collaboration, leading to difficulties in translating basic neurobiological knowledge about mental illness into the development and dissemination of efficacious cognitive and behavioral treatments. By forging these interdisciplinary relationships and integrating the use of cognitive affective neuroscience methods into clinical research, the impact of basic research on treatment development and dissemination, as well as mental health outcomes, may be greater in the decades to come.

To be clear, we are not arguing that neurobiological factors are the sole contributors to the development of psychopathology, and that studying these factors in isolation will be sufficient to reduce mental and emotional suffering among those diagnosed with psychiatric diseases. Indeed, ample clinical research is available to support the role of social and environmental mechanisms in psychiatric disease liability. On this basis, some researchers have argued that studying biological correlates of psychiatric disease may actually distract from social and environmental factors that are also central to the development of psychopathology, and may reduce the likelihood that clinical research will yield effective preventions and interventions for mental illness (e.g., Albee & Joffee, 2004); however, we clearly disagree with this stance. Further, a rapidly growing cohort of researchers have argued that understanding the biological basis of psychopathology, as well as the interaction between biological and environmental factors, is absolutely crucial for reducing the morbidity and mortality of psychiatric disease (e.g., Beauchaine et al., 2008; Insel, 2009). The National Institute of Mental Health (NIMH), one of the largest funding bodies in psychiatric research, clearly agrees, as evidenced by the addition of funding priorities highlighting the need for scientific inquiry that aims to translate basic neurobiological findings into clinical practice (Insel). As such, the field's interest and focus on neural mechanisms underlying psychiatric illness and treatment is already evident and is likely to expand even further in the coming years.

Despite growing interest in and emphasis on integrating functional neuroimaging methods into the study and treatment of psychiatric disorders, the gap between our basic knowledge of psychiatric neurobiology and clinical outcomes raises the question of whether clinical researchers are routinely considering neurobiological models when developing novel cognitive and behavioral treatments and when administering extant empirically supported treatments in practice. As such, the current article aims to highlight the potential value of using cognitive affective neuroscience research, including fMRI assessments in particular, to augment clinical research and inform clinical practice. We will draw specifically from the field of addiction to provide examples of clinically relevant research questions that can be answered with cognitive affective neuroscience, as well as clear examples of extant and ongoing research that is already working at this intersection between clinical research and neuroscience. We will begin with an overview of current neurobiological models

of addiction, provide examples of extant research that is drawing on neurobiological knowledge and methods to answer clinically relevant research questions, and conclude with suggestions for future research. This review is not meant to be exhaustive; however, it is hoped that the examples provided herein will spark broader interest in the potential utility of interdisciplinary research that draws from cognitive affective neuroscience in order to better understand the etiology, progression, and treatment of psychopathology.

Neurobiological Models of Addiction

Turning to substance use disorders (SUDs) specifically, numerous complex and overlapping neurobiological models of addiction have been developed (e.g., Everitt & Robbins, 2005; Koob & Le Moal, 2001, 2008; Li & Sinha, 2008; Robinson & Berridge, 2001; Volkow & Li, 2004), leading to a deeper understanding of the neural mechanisms underlying the core clinical features of addiction, including craving, compulsive drug seeking, and loss of control over drug use (American Psychiatric Association [*DSM-IV-TR*], 2000). Yet, this improved basic understanding has not been translated into the dissemination of more effective prevention and intervention programs. Alcohol and other substance use disorders remain among the most highly prevalent psychiatric disorders diagnosed in the United States today, surpassed only by major depressive disorder (Kessler & Wang, 2008; World Health Organization, 2002). Further, despite the availability of no less than 10 behavioral therapies that have been classified as "efficacious" for the treatment of SUDs (Carroll & Onkin, 2005; Chambless & Ollendick, 2001), recovery is still incredibly difficult, as evidenced by estimated relapse rates of up to 90% within 4 years following substance use treatment (e.g., Monti, Rohsenow, Michalec, Martin & Abrams, 1997; Morgenstern, Blanchard, Morgan, Labouvie, & Hayaki, 2001; Office of Applied Studies, 2009). The intractable nature of addiction is not surprising given neural models that emphasize complex and interrelated changes that occur across several neural circuits in the brain over the course of chronic drug use. Thus, a more thorough overview of the neurobiological basis of addiction is warranted in order to better understand the potential targets for novel cognitive and behavioral treatments for addiction.

Across neurobiological models, addiction theorists have emphasized the role of motivational circuitry localized in corticolimbic regions, which are also implicated in the regulation of emotions and behavior. As one component of this circuitry, human neuroimaging studies show that acute exposure to nearly all drugs of abuse is associated with increased activation in the mesocorticolimbic dopamine pathway extending from the midbrain ventral tegmental area to the prefrontal cortex, and that this activation correlates with subjective ratings of high or euphoria and craving, thus providing the neural substrate by which addictive drug use is positively reinforced (e.g., Di Chiara et al., 2004; Wise, 1996). However, chronic drug administration and acute withdrawal is associated with alterations in this pathway, leading to blunted dopaminergic functioning and reduced activation in these regions (for detailed reviews see Koob & Le Moal, 2008; Sinha, 2008; Volkow, Fowler, & Wang, 2002). Compulsive drug use also leads to alterations in brain stress circuits, including the hypothalamic-pituitary-adrenal (HPA) axis and the extended amygdala, resulting in hyperactivation in these regions, which is associated with increased negative affect and greater stress reactivity (Koob & Le Moal; Sinha). These neuroadaptive changes in corticolimbic functioning are in line with theories suggesting a shift over the course of chronic substance use from positive reinforcement–based drug use motivation to negative reinforcement as the primary motivator (e.g., Baker et al., 2004).

Additionally, neurobiological models of addiction frequently emphasize impaired functioning in prefrontal regions, including the anterior cingulate cortex, which leads to reduced regulatory control over emotions and behavior (e.g., Goldstein & Volkow, 2002; Li & Sinha, 2008; Volkow & Li, 2004). This dysfunction in top-down regulatory functioning contributes even further to the execution of habitual drug seeking by reducing one's ability to inhibit this behavior. Overall, neurobiological models of addiction emphasize abnormalities in the neural circuits underlying both positive and negative reinforcement-based drug use motivation, as well as executive control over craving and drug-seeking behavior, suggesting that abnormalities in these circuits may serve as biomarkers to identify individuals at greatest risk of developing and relapsing to addiction.

Clinical Research Questions That Can Be Addressed With Cognitive Affective Neuroscience

Preventing SUD Development and Relapse

Of great importance to clinical researchers and treatment providers alike is the ability to reliably identify individuals who are at the greatest risk for developing a psychiatric disorder in order to create more targeted and efficient prevention programs, and to identify individuals at the greatest risk for relapse, for whom the most prolonged and intensive treatment may be required. As such, the following sections will focus specifically on the potential for cognitive affective neuroscience research to inform prevention and intervention efforts aimed at reducing SUD vulnerability and relapse following cessation.

How Can Cognitive Affective Neuroscience Inform SUD Prevention?

In no area is the application of cognitive affective neuroscience to clinically relevant research questions potentially more fruitful than in the area of prospective prediction of addiction vulnerability prior to the development of an SUD (e.g., Rutherford, Mayes, & Potenza, 2010). The early identification of individuals at risk for future substance use and the application of effective targeted prevention programs are of paramount importance for reducing addiction morbidity, as early substance use is associated with neurobiological changes that increase the risk for future addiction (Volkow & Li, 2005). Surprisingly, there has been little if any research examining the neural correlates of addiction vulnerability among drug-naïve individuals. On the other hand, there is a growing body of neuroimaging work examining differences in neural structure between adolescents with and without existing substance use disorders. For instance, it has been shown that prefrontal cortex (PFC) volume is significantly reduced among adolescents with alcohol use disorders and that this volume reduction is not related to age of alcohol use onset, suggesting that PFC volume reductions may have predated alcohol use initiation among these individuals (De Bellis et al., 2005). Although the null relationship between PFC volume and age does not entirely rule out the possibility that PFC volume reductions could be a consequence of alcohol use, these findings provide initial support for the hypothesis that abnormalities in the PFC, which is implicated in executive function-

ing, decision making, and impulse control, may be a risk factor for the development of SUDs. As such, targeted prevention efforts that aim to improve functions mediated by the PFC, such as decision making and inhibitory control, particularly among individuals exhibiting reduced PFC volume, may be a fruitful area for SUD prevention research. Moreover, there may be utility in examining neurobiological factors, such as PFC volume, as potential moderators of the relationship between executive functioning-focused prevention efforts and substance use outcomes in order to identify individuals who are most likely to benefit from such treatments, or as possible neural mechanisms underlying treatment effects.

Additional evidence has emerged implicating dopamine receptor availability as a predictor of substance use vulnerability. Rats who are highly impulsive on an impulsive choice task display reduced D2/3 receptor binding in the ventral striatum, a brain region implicated in reward processing, as well as more rapid escalation in cocaine self-administration relative to nonimpulsive rats (Everitt et al., 2008). In human adults with no history of drug use, lower D2 receptor levels in the striatum are associated with increased subjective ratings of pleasure following administration of the stimulant methylphenidate (Volkow et al., 1999). These findings suggest that individuals evidencing lower levels of dopamine receptor availability may be more impulsive, may experience more subjective pleasure from substances of abuse as opposed to natural reinforcers, and may be at risk for rapid escalation in drug-seeking behavior. As such, researchers have suggested the potential utility of prevention and intervention strategies that aim to replace risk-taking behaviors, such as substance use, with other stimulating and novel behaviors that can be experienced as rewarding (Rutherford et al., 2010). To this end, behavioral activation (Lejuez et al., 2011) approaches, which aim to increase engagement in alternative reward-based activities, may be useful for preventing the future development of SUDs. Indeed, behavioral activation has been shown to be an effective intervention for chronic substance use (e.g., Daughters et al., 2008; Magidson et al., 2011), and an effective prevention of alcohol-related problems among healthy college students (Reynolds et al., 2011). As such, behavioral activation treatments may be useful for preventing SUDs, particularly among individuals exhibiting low dopamine receptor availability. However, more research is needed in order to specifically examine the relationship between dopamine receptor availability and substance use outcomes following completion of behavioral activation–based prevention programs in order to test the extent to which these treatments might be particularly well suited for individuals exhibiting low dopamine receptor density.

Clearly, the identification of neurobiological risk factors for substance use using functional neuroimaging methods is an area that is ripe for discovery. The identification of the neural correlates of substance use vulnerability is particularly important from a clinical perspective given that neural circuits implicated in psychopathology are often particularly malleable in childhood (e.g., Raine et al., 2001), and some researchers have suggested that key neural networks, such as the PFC, may be modified through targeted preventions and interventions (e.g., Beauchaine et al., 2008; Rueda, Rothbart, Saccomanno, & Posner, 2007). Given the dearth of neuroimaging research among drug-naïve individuals, an important next step will be to incorporate neuroimaging methods into longitudinal substance use outcome research. In doing so, it may be possible to identify clear neural markers that predict future drug use among drug-naïve individuals, which would provide further support for a more integrated approach to prevention that incorporates fMRI methods into targeted efforts to prevent addiction.

How Can Cognitive Affective Neuroscience Inform Relapse Prevention?

A field of inquiry that is gaining more attention in addiction research is the use of cognitive affective neuroscience methods to identify neural processes associated with relapse following substance use treatment. Early examples of work in this domain involved fMRI paradigms that exposed individuals to drug-related cues to assess the relationship between cue-induced neural activation and subsequent treatment outcome. In one study of this kind, Grüsser and colleagues (2004) showed alcohol-related images and neutral images to a sample of abstinent alcoholics and healthy controls, and found that greater cue-induced activation in regions involved in emotion regulation and reward, including the medial prefrontal cortex, the striatum, and anterior cingulate cortex (ACC), was associated with an increased risk of relapse in the 3 months following the scan. A subsequent study found that among cocaine-dependent individuals, neural activation in response to co-

caine-related videos was related to subsequent relapse during a 10-week outpatient treatment, such that greater activation in the sensory association cortex, motor cortex, and the posterior cingulate cortex was associated with relapse to cocaine abuse (Kosten et al., 2005). Despite differences in the specific neural regions identified, the abovementioned studies provided some of the first evidence that functional neuroimaging could be used to identify individuals who are at a heightened risk of relapse following substance use treatment.

Research in this domain has since expanded to include assessments of neural activation in response to stress and stress-induced drug craving to predict substance use treatment outcomes. For example, in line with theories suggesting alterations in brain stress circuits, Sinha, Lacadie, Li, Skudlarski, and Kosten (2005, as cited in Sinha & Li, 2007) exposed cocaine-dependent individuals to personalized stress imagery scripts and neutral scripts in the fMRI at the beginning of an inpatient treatment stay, and then conducted a follow-up interview with participants at 90 days posttreatment. Findings revealed that greater stress-induced activation in the medial prefrontal cortex, which has been associated with suppression of negative affect while viewing aversive stimuli, was associated with shorter time to cocaine relapse and with a higher number of days of cocaine use during the 90-day follow-up period. Further, greater stress-induced activation in limbic regions, including the insula, was associated with a higher number of days of cocaine use, while increased activation in the posterior cingulate was associated with the amount of cocaine used per occasion. The authors suggested that enhanced stress-induced activation in these limbic regions may serve as neural risk factors for binge episodes or loss of control over cocaine intake (Sinha & Li).

In line with growing efforts to identify stress-related neurobiological indicators of relapse risk, our own group has modified two previously validated distress tolerance tasks for use in the fMRI environment. Low distress tolerance, defined as the inability to persist in goal-directed behavior in the face of negative affect, is consistently associated with poor substance use outcomes across drug classes (e.g., Brown, Lejuez, Kahler, & Strong, 2002; Brown, Lejuez, Kahler, Strong, & Zvolensky, 2005; Daughters, Lejuez, Bornovalova, et al., 2005; Daughters, Lejuez, Kahler, et al., 2005); however, the neural correlates of distress tolerance have yet to be identified. Initial pilot work with a healthy control sample revealed that individuals with low distress tolerance showed reduced stress-induced activation in regions involved in inhibitory control, executive functioning and working memory, including the PFC and subgenual ACC. Low distress tolerance was also associated with increased activation in regions involved in emotional processing, including the dorsal and ventral ACC (Daughters et al., 2010). The next step in this ongoing project is to examine the neural correlates of distress tolerance in a sample of cocaine users and determine whether stress-induced neural activation on these tasks might predict substance use treatment outcomes.

Continued treatment outcome research that integrates neuroimaging assessments into testing batteries are certainly needed in order to replicate and extend existing work; however, the available findings are exciting in that they mark a clear area in the field where the integration of cognitive affective neuroscience and clinical research is already providing new insights into the neurobiological basis of addiction treatment response and relapse to substance use.

Treatment Mechanisms and Development of Innovative Treatments

Despite what is known about the neural basis of addiction vulnerability and relapse, few if any cognitive behavioral treatments for addiction were developed with these neurobiological factors in mind. Yet, a growing number of effective behavioral treatments for SUDs have been developed, including contingency management, motivational interviewing, cognitive behavioral approaches (e.g., functional analysis, relapse prevention), and families and couples counseling (Carroll & Onken, 2005). Although these available treatments are based largely on behavioral and cognitive theories, they have also been hypothesized to target the neural correlates of reinforcement and regulatory control that are emphasized in neurobiological models of addiction (Potenza, Sofuoglu, Carroll, & Rounsaville, 2011). For example, motivational interviewing has been hypothesized to impact brain regions in the prefrontal cortex involved in decision making, cognitive control, and planning, while contingency management approaches have been hypothesized to target both cortical and subcortical regions involved in decision making and reward (Potenza et al., 2011). Unfortunately, however, these hypotheses remain largely untested. As such, the following sections will highlight the limited

published work that has already begun to tackle questions regarding the neural targets of established addiction treatments, followed by a discussion of novel cognitive behavioral treatments, informed by neurobiological models of addiction, which are currently being developed and evaluated.

How Do Current Empirically Supported Treatments Affect the Neural Correlates of Addiction?

Cognitive affective neuroscience methods have recently been employed to examine the neural mechanisms underlying an established SUD treatment strategy, namely cognitive craving regulation (Kober et al., 2010). Specifically, while lying in an fMRI machine, a sample of cigarette smoking participants were shown pictures of smoking cues and instructed to either focus on the short-term pleasurable aspects of tobacco use or to use the cognitive strategy of focusing on the long-term consequences of cigarette smoking. Findings revealed that focusing on the long-term consequences of smoking was associated with reductions in self-reported cigarette craving. Moreover, focusing on the long-term consequences of smoking was associated with increased activation in cortical regions including the dorsomedial, dorsolateral, and ventrolateral PFC, as well as decreased activation in regions previously implicated in craving and reward, such as the ventral striatum, subgenual cingulate, amygdala, and ventral tegmental area. Further, decreased self-reported craving while using this cognitive strategy was associated with decreased activity in the ventral striatum, and this relationship was fully mediated by increased activation in the dorsolateral PFC. That is, activation in a region of the PFC that was previously associated with effective cognitive emotion regulation appears to play an equally important role in regulating drug craving by exerting control over activation in the ventral striatum, a brain region implicated in reward. These findings suggest that cognitive strategies can be used by smokers to effectively regulate cigarette craving, and that these reductions in craving are driven by recruitment of cognitive control circuits that function to overpower midbrain reward functioning.

Although the study reviewed above yielded promising findings, there is clearly a need for much more work of this kind in order to enhance our understanding of neural mechanisms underlying other substance use treatment strategies beyond cognitive craving regulation. The more we understand about the neural mechanisms underlying substance use treatment, the more questions we will be able to answer. For example, given standard neural models of effective addiction treatment strategies, such as cognitive craving regulation, future researchers may be able to test the extent to which other cognitive and behavioral treatments for addiction are useful for inducing functional changes in core neural networks implicated in successful craving regulation. Further, fMRI assessment paradigms such as the one developed by Kober and colleagues (2010) could be used to assess individual differences in neural functioning at baseline, and also to track treatment progress over time.

How Can Clinical Researchers Use Cognitive Affective Neuroscience to Inform Treatment Development?

Based on findings from studies of the kind outlined above, cognitive affective neuroscience can also inform the development of more efficient cognitive and behavioral treatments by pointing to specific neurobiological targets for the prevention and treatment of addiction pathology. Indeed, novel cognitive and behavioral approaches are already receiving increased attention, with some researchers arguing that these approaches may more effectively target the cognitive, emotional, and behavioral deficits characteristic of addiction, as well as their underlying neural correlates (e.g., Potenza et al., 2011). As one example, cognitive remediation strategies involving computerized exercises that aim to improve working memory, attention, and other executive functions, are believed to strengthen neural circuits, particularly in the PFC, that have consistently been implicated in neurobiological models of addiction and relapse. Working memory training has already gained support among clinical researchers following the emergence of evidence for its utility in the treatment of impulse control disorders such as ADHD (e.g., Klingberg, Forssberg, & Westerberg, 2002; Klingberg et al., 2005). Given that substance abuse is also associated with deficits in executive functions including impulse control (Di Sclafani, Tolou-Shams, Price, & Fein, 2002; Rapeli et al., 2006; Verdejo-Garcia, Rivas-Perez, Vilar-Lopez, & Perez-Garcia, 2007), similar strategies are now being tested among individuals with substance use disorders. Specifically, a recent report by Bickel, Yi, Landes, Hill, and Baxter (2011) showed that working memory training in stimulant addicts improved delayed discounting, a measure of impulsivity. Clearly, the next step is to examine whether this improvement in impulse control can also improve substance use treatment outcomes.

To address this next step, our group is currently testing the utility of working memory training as a strategy to target the cognitive dysfunction associated with addiction, with the overall aim of improving substance use treatment outcomes. We have piloted a working memory training program in substance-dependent individuals and found that they are indeed able to do the training, and by doing so, are able to increase their working memory capacity, as measured by digit ordering and N-back performance compared to patients who trained on the control training condition (i.e., a computerized task with no working memory content). A larger follow-up study is planned to randomize a group of substance-dependent patients in residential substance use treatment to either several weeks of daily working memory training using an N-back task, or an equal amount of time on a control task. It is hypothesized that training on the working memory task will be associated with improvements in inhibitory control, delayed discounting, and working memory capacity itself, as well as improvements in behavior such as impulse control, and compliance with treatment program requirements as evaluated by the participants' treatment team. Additionally, the plasticity of circuits underlying changes in executive function in response to training will be assessed among participants using fMRI assessments of delayed discounting, inhibitory control, and working memory both before and after treatment. The influence of each of these processes on long-term substance use abstinence rates will also be examined.

Mindfulness- and acceptance-based treatments, which have already established an empirical base in the treatment of mood, anxiety, and personality disorders (e.g., Hayes, Strosahl, & Wilson, 1999; Linehan, 1993), may also be useful for targeting the neural correlates of stress-induced relapse in addiction. At a theoretical level, it has been argued that mindfulness- and acceptance-based treatments may be particularly effective at simultaneously improving executive control functions and reducing stress reactivity and distress intolerance (Brewer, Bowen, Smith, Marlatt, & Potenza, 2010). Empirically, there is evidence that mindfulness training can actually strengthen white matter tracts connecting regions involved in emotion regulation and cognitive control

(Tang et al., 2010), and mindfulness-based treatments for substance use yield comparable rates of treatment retention and abstinence, as well as superior reductions in self-reported and physiological stress reactivity as compared to standard addiction treatments (Brewer et al., 2009). Similarly, a novel mindfulness- and acceptance-based treatment for substance users with low distress tolerance was recently tested in a residential substance use treatment center. Findings revealed that individuals who received the mindfulness- and acceptance-based treatment showed significantly greater increases in behavioral distress tolerance from pre- to posttreatment as compared to individuals assigned to time-matched supportive counseling and treatment as usual (Bornovalova, Gratz, Daughters, Hunt, & Lejuez, 2011). As such, novel applications of mindfulness-based treatments appear to be effective for the targeted treatment of the behavioral, physiological, and neural mechanisms underlying addictive behavior.

Beyond treatments that are already available or in development, studies that integrate neuroscience and clinical research could also contribute to the development of novel applications of real-time fMRI itself for the treatment of substance use disorders. Indeed, the combination of real-time fMRI and biofeedback represents a nascent, yet promising, avenue for the treatment of a variety of psychiatric illnesses, including substance use disorders. Specifically, this method involves providing individuals with continuous information about activation in a particular brain region, and asking them to alternately increase and decrease the activation level in their brain as they continuously receive feedback about their neural functioning in real time (deCharms, 2008). To date, most real-time fMRI studies have focused on participant-driven modulation of blood oxygenation level dependent (BOLD) signal in cortical regions, including the somatomotor cortex (deCharms et al., 2005), auditory cortex (Yoo et al., 2007), insular cortex (Caria et al., 2007), medial frontal cortex (Phan et al., 2004), and ACC (deCharms et al., 2004; deCharms et al., 2005), and have largely included healthy participants between the ages of 18 and 45 years old. However, a smaller body of research has focused on subcortical structures, including the amygdala (Posse et al., 2003). Moreover, preliminary studies are emerging in support of real-time fMRI and participant-driven signal modulation as potential treatments for clinical disorders, including epilepsy (Fernandez et al., 2001; Kesavadas et al., 2007) and chronic pain (deCharms, 2007; deCharms et al., 2005). Given the extensive evidence reviewed herein, it can be concluded that addiction is indeed a disorder of the brain, and as such, may be amenable to treatment through real-time feedback and modulation of neural functioning by participants in the neural circuits underlying addiction using fMRI methods. This proposition is speculative and we are not aware of any studies to date that have examined the use of real-time fMRI as a treatment for addiction; however, this possibility is yet another example of how functional neuroimaging methodology may inform the development of novel treatments for addiction.

Based on the nascent findings reviewed above, it is clear that cognitive affective neuroscience can have a significant impact on the prognosis of individuals suffering from addiction by fostering innovative

treatment approaches for targeting neurobiological impairments that are characteristic of addiction vulnerability and relapse. Larger scale treatment outcome studies that integrate fMRI assessments into the study designs will be needed to examine not only the effects of these innovative treatments on substance use treatment outcomes, but also the extent to which the targeted neural mechanisms are indeed driving treatment effects.

Summary and Future Directions

As reviewed above, today's clinical researchers are operating in a time when collaborative, interdisciplinary approaches are becoming paramount to understanding the core mechanisms underlying psychiatric illness and treatment outcomes. Therefore, it is important to consider how clinical researchers can begin to draw on the work of cognitive affective neuroscience and strive to incorporate functional neuroimaging methods into their own work. One step includes collaborating directly with cognitive affective neuroscientists to develop and integrate functional neuroimaging paradigms into the assessment batteries that are administered in clinical research studies. In this way, cognitive affective neuroscience data can serve as an additional level of analysis to aid clinical researchers in the development, evaluation, and modification of clinical theories of psychopathology and treatment, thus providing new insights into the biological mechanisms through which cognitive and behavioral treatments actually do work, and for whom these treatments may be most effective. Translational work of this kind presents a number of challenges; however, collaboration across disciplines can be an effective way to overcome many of the challenges, and may even produce synergistic effects that will compound the knowledge gained even further.

Regardless of the challenges, the integration of cognitive affective neuroscience methods into clinical research is indeed feasible as evidenced by the studies reviewed above, and this work will likely increase in feasibility as technology continues to advance. As new tools, methods, and techniques become available, an even wider range of clinical questions will likely be tackled using these methods. For example, future researchers may gain additional insight into the common and unique neural mechanisms underlying comorbid psychological disorders, which may point to new targets for the treatment of co-occurring psychopathology. Additionally, as new knowledge emerges about normal and abnormal neural development, researchers may be able to identify critical neurodevelopmental periods when interventions will be most effective. Also of great importance will be future research efforts to identify neurobiological moderators of treatment outcome in order to determine which cognitive and behavioral treatments are likely to work best for whom. This is indeed an exciting time to be a clinical researcher, as the integration of cognitive affective neuroscience methods will surely help us to find new answers to these critically important clinical research questions.

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Clinical Forum

Male Sexual Desire and Function

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There are two myths that dominate public and professional discourse about gender and sexuality. The first is that female hypoactive sexual desire disorder (HSDD) causes nonsexual marriages and second is that the proerection medications have totally resolved erectile dysfunction (ED). What should CBT clinicians and researchers know about the scientific and clinical reality? Leiblum's (2010) book on sexual desire emphasizes female desire problems as a very common problem. However, using the operational definition of a nonsexual marriage—being sexual less than 10 times a year (McCarthy & McCarthy, 2003)—when couples totally stop being sexual it is the man's decision in the great majority of cases (Lindau et al., 2007). In essence, he has lost comfort and confidence with erections, intercourse, and orgasm. He is stuck in the pattern of anticipatory anxiety, tense and performance-oriented sex (intercourse as the pass-fail test), frustration, embarrassment, and avoidance of intimacy. His internal cognition is, "I don't want to start something I can't finish." He avoids not just intercourse, but sensual, playful, and erotic touch. Typically, this decision is made unilaterally and conveyed nonverbally.

The proerection medications (Viagra, Cialis, and Levitra) can be valuable resources to promote male sexual function as he confronts illness, side effects of medications, and, with aging, a less efficient and resilient vascular system. However, for erectile medications to work in an optimal manner they need to be integrated into the couple style of intimacy, pleasure, and eroticism (McCarthy & Fucito, 2005). Just as important, the man and couple have to maintain positive, realistic sexual expectations. Rather than expecting the pill to be a stand-alone intervention that will return him to totally predictable erections of his teens and 20s, they need to embrace a "new sexual normal." The best estimate is that when a man takes an erection medication the couple will have successful intercourse in 65% to 85% of encounters (Brock et al., 2002). This is a positive, realistic expectation, but marketing ads and medical overpromising leads the man and couple to feel like "sex failures." The dropout rate is quite high (40% to 80%), leading to demoralization and sexual avoidance. CBT clinicians and researchers need to reengage in this important field, which has a major impact on the psychological well-being of men, women, and couples (McCarthy & Breetz, 2010).

Traditional Male Sexual Socialization

The great majority of males learn sexual desire and erection as spontaneous, in their control, totally predictable, and most important, autonomous. In other words, teenage and young adult men can experience desire, erection, intercourse and orgasm as an autonomous function; they need nothing from their partner. Cultural images and male folklore reinforce that this is "first-class male sex" and anything else is "settling" or second-class. In reality, approximately 10% of men do not experience autonomous sexual desire and function (McCarthy & McDonald, 2009). Primary male-inhibited sexual desire is usually caused by a sexual secret—a variant arousal pattern, greater confidence with masturbatory sex (with or without pornography), a history of sexual trauma, or conflicts about sexual orientation. The anxieties for most young males involve sex performance factors such as concerns about penis size, number of partners, and ability to "give" the partner an orgasm during intercourse. These concerns do not cause male inhibited sexual desire.

This overemphasis on perfect individual sex performance—being able to have sex in any situation regardless of mood or stress, valuing sex quantity over sexual quality, and especially, the emphasis on totally predictable, autonomous sex function—is an unrealistic demand on the man as he ages and is in a serious or marital relationship (McCarthy & Metz, 2008). This model of male sexuality sets the stage for sex dysfunction, inhibited desire, and sexual avoidance.

Male Sexual Desire

For the majority of males, spontaneous erections are the cue for sexual desire. Typically, boys learn sexual response through masturbation beginning between ages 10 to 14. Partner sex to orgasm typically begins between ages 15 to 20. Although there is often awkwardness and anxiety about premature ejaculation, desire and erections remain strong and reliable.

The key for sexual desire is to maintain positive anticipation with the cognition that he deserves to enjoy sex at this time and in this relationship. In a serious or marital relationship the primary challenge for the man is to balance his "sexual voice" with being an intimate, erotic team. The second challenge is to integrate intimacy and eroticism into this relationship. The couple needs to confront the stereotype of male sexuality being simple while female sexuality is complex. Maintaining a strong, resilient sexual desire in serious relationships is a complex psychosocial challenge for the man, woman, and couple (Metz & McCarthy, 2010).

Erectile Comfort and Confidence

Once a sensitizing event occurs (i.e., the man does not have an erection sufficient for intercourse), he does not return to the unself-conscious total confidence of his youth. Whether this occurs at 25, 35, 45, or not until 65 (the latter is rare), this changes the man's approach to sex. Ideally it would allow him to be a more aware, better lover—in other words, focus on enhancing intimacy, pleasuring, and eroticism both for himself, his partner, and their relationship. Unfortunately, the more typical outcome is he becomes sensitized to anticipatory and performance anxiety. As Zilbergeld (1999) noted, the man no longer views his penis as a "friend," but as an unreliable performance tool.

When the man uses a proerection medication, he naïvely hopes that this will return him to the totally predictable, autonomous erections of his youth. He is disappointed, frustrated, and embarrassed when this unrealistic expectation is dashed.

Metz and McCarthy (2007) argue for a comprehensive, integrative approach to regaining comfort and confidence with erections whether an erection medication is used on a consistent basis, intermittent basis, or not at all. Medication cannot be a stand-alone intervention, nor can it return the man to totally predictable, autonomous erections. What can, and ideally does, occur is the man's commitment to rebuild sexual anticipation and desire based on sharing pleasure and eroticism. Sexual desire and function is not contingent on perfect intercourse performance. He learns to view the

woman as his intimate, erotic friend rather than someone to perform for. If he chooses to use a proerection medication he needs to be sure it is integrated into the couple style of intimacy, pleasuring, and eroticism. Just as important, the man and couple need to develop positive, realistic expectations about erections and intercourse. The self-defeating approach is to view the pill as a stand-alone magic solution. The "Good Enough Sex" (GES) guideline is that 85% of sexual encounters will flow to intercourse. When that does not occur the man, without apologizing or panicking, is able to comfortably transition to an erotic, nonintercourse scenario or a sensual, cuddly scenario (Metz & McCarthy, 2004). This approach presents a challenge for the man, woman, and couple. The clinician helps them carefully process emotions of rejection, failure, or not feeling loved and replace these with positive, realistic sexual expectations. The GES approach confronts negative attributions and emotions and replaces them with feelings of being intimate and erotic allies.

For some couples, Viagra is the preferred drug because it serves to give the man confidence and serves as a cue to initiate sex. Proerection medications do not cause desire or arousal, but do serve as a placebo to reduce anticipatory and performance anxiety as well as enhance vascular function so that when aroused the erection maintains and is resilient. For other couples, Cialis is the preferred drug because it provides more degrees of freedom of when and how to initiate a sexual encounter. In other words, the proerection medications have different psychosexual dimensions that have a stronger influence over sexual effectiveness rather than simply the biological impact of the medication.

The most important issue for the man and couple is to break the avoidance cycle and rebuild the cycle of desire (positive anticipation), pleasure (receiving and giving pleasure-oriented touching), eroticism (taking emotional and sexual risks to enhance sexual vitality), and satisfaction (couple bonding and meaning; Metz & Epstein, in press).

Acceptance Versus Conditional Approach to Sexual Function and Dysfunction

Both the man's youthful sexual experiences and the media (including drug advertisements) emphasize a totally predictable model of erection, intercourse, and orgasm. This is a powerful and seductive message that sets an unrealistic model of the way men "should" function sexually. This model is not the reality for most men after 40 or 50; very few men can meet this criterion in their 60s. In reality, the variable, flexible GES model is preferable for males after 50 (and ideally before that). A favorite cognitive intervention is to ask the man whether he will cling to the traditional model, in which case he is likely to stop being sexual in his 50s or 60s, or whether he will be a "wise" man, embrace the GES model, and enjoy sexuality in his 60s, 70s, and 80s.

The issue is what "first-class" male sexuality means. Is it autonomous sexuality with totally predictable erections and perfect intercourse performance? Or is it intimate, interactive couple sexuality focused on giving and receiving pleasure-oriented touching, integrating intimacy and eroticism, valuing sensual, erotic, and intercourse scenarios, and viewing the woman as his intimate sexual partner to share with rather than perform for. Women are more likely to embrace the GES model both for themselves as well as their relationship. It is a much greater challenge for the man. He receives no support from male peers who brag/lie about sexual performance. Whether it is transitioning to erotic sex if the encounter does not flow to intercourse, accepting that at times her arousal and orgasm is easier than his, being comfortable in using self-stimulation during partner sex, or valuing sensual scenarios, the rigid male sex culture says that only firm erections, intercourse, and orgasm qualify as acceptable sex. The man is not willing to admit he "failed" the performance test even if to do so would enhance male and couple desire and satisfaction.

Michael and Laura[1]

Sixty-seven-year-old Michael had fallen into the traditional male sex trap. Over the past 2 years, he had lost his comfort and confidence with erections and intercourse. Instead of *enjoying* the sexual experience, he was *relieved* when he ejaculated inside Laura.

Couple sex occurred less than once a month and usually ended with frustration when Michael lost his erection. Michael envied the sexual vitality of his two adult sons, and increasingly felt old and "over the hill." He loved Laura (who is 61) and viewed her as attractive, although not as sexually appealing as the 25-year-old women he saw on the street or on TV. Michael masturbated 2 to 4 times a month using Internet porn. This reassured him that he could still have orgasms, although he felt his erections were less strong and his ejaculations less satisfying. He felt guilty about ignoring Laura and using porn, but rationalized that is was the only way for him to be sexual.

Laura felt confused and resentful about Michael's sexual avoidance. She had viewed sexuality as a strength in the marriage, especially before the boys were born and after the last one left for college. Laura wanted to enjoy the "empty nest" chapter of her life. She had won an award as a master teacher at the community college, felt connected to her son's families—especially the grandchildren—felt her marriage was good, and believed that since both Michael and she were healthy and he had cut back to a 30-hour work week, they would travel and enjoy life more. However, the lack of intimacy, touching, and sexuality felt like a big hole in her life and a drain on their relationship.

When Laura tried to talk to Michael about the lack of sex, he became defensive and counterattacked about her reluctance to be sexual when they were raising their sons. He bewailed the missed sexual opportunities during those years. Laura, feeling defensive, informed Michael that she had been drained by going to graduate school part-time, teaching, and being the primary parent. Michael complained that Laura wanted to argue and put him down about the past. She felt ignored and that he put up a wall against emotional intimacy and sexual touching. They enjoyed each other more when they were around other people and involved in family activities.

What brought the intimacy issue to a crisis point were plans for their 35th wedding anniversary. Laura wanted to take a 4-day trip as a couple to a historic hotel in the mountains, but Michael wanted a long weekend at the beach with the whole family. It was clear that this wasn't about the mountains versus the beach; it was about Michael not wanting to be alone with Laura in a potential sexual situation.

One daughter-in-law was a clinical social worker. She suggested to Laura that they consult a couple therapist with a specialty in intimacy issues with older couples. Laura was very enthusiastic, but Michael was concerned he'd be viewed as the "bad guy." He was relieved to learn that the therapist was a man who rejected "male bashing." However, Michael was still anxious. Underneath, Michael felt ashamed that he wasn't able to perform like a "real man." Two weeks before the scheduled appoint-

[1] Details about the case have been modified to protect the identity of the client.

ment, he bought 10 Viagra pills on the Internet. Without saying anything to Laura, he took 2 pills washed down by 2 beers (Michael felt he needed "liquid courage"). Although he did have an erection, Michael tried to rush intercourse and lost his erection during intromission, which was very frustrating and embarrassing for him. Laura was totally bewildered by what had happened and why. She felt frustrated by Michael's unwillingness to talk with her at the time or afterward.

When they appeared for their initial therapy appointment they were a tense and tentative couple. Conducting the first session as a couple is the preferred strategy because it provides the opportunity to define the problem as a couple issue and to start them thinking, talking, and feeling as an intimate team (McCarthy & Thestrup, 2008).

Rather than blaming or shaming Michael, the clinician was empathic and respectful. He did challenge Michael's assumption about what constituted "first-class" male sexuality after 60. Michael was surprised to learn (as was Laura) that when couples stopped being sexual it is almost always the man's decision. Michael was reassured to learn that very few men experienced the perfect, predictable erections portrayed in the medication ads. In addition, his alcohol use interfered with the Viagra by weakening vascular response. Michael would learn to utilize relaxation and mindfulness techniques to reduce anticipatory anxiety.

Psychologically, the major issue for Michael was to accept the challenge of GES, especially erections that developed from touching rather than easy, autonomous erections. Biologically, Michael adopted the strategy of using daily doses of Cialis. Relationally, he learned to view Laura as his intimate, erotic friend. In terms of psychosexual skills, two key factors were use of multiple stimulation before and during intercourse and Laura guiding intromission so Michael could focus on the pleasuring. These were not easy for Michael to accept and implement, but with Laura's support and involvement they experienced significant sexual growth.

Laura found both the therapy sessions and the psychosexual skill exercises easier and more inviting than did Michael. The fact that Michael needed her sexually was a powerful aphrodisiac for Laura. She identified with the concept that his "grown-up" erections were better than his youthful "show-up" erections. Although Laura very much valued intercourse sex, like many women of her age she found erotic scenarios and techniques very satisfying. When she was moderately aroused she enjoyed receiving oral sex (if she experienced low levels of arousal, oral sex was counterproductive because it increased her self-consciousness rather than facilitated erotic flow). Often Laura would be orgasmic with oral stimulation and then Michael used a vaginal lubricant to facilitate intercourse. He preferred orgasm inside of her.

The process of couple sex therapy, especially where there is male inhibited sex desire, is seldom easy or linear. More often it is a "two steps forward, one step back" process. Michael longed for the past pattern of being the initiator, predictable erections, and reliable intercourse and orgasm. Laura found it much easier to embrace variable, flexible GES because it was congruent with her sexual socialization and experiences. Michael had an internal argument about whether he was "settling" and whether GES was really first-class male sexuality. The biggest psychological "trap" for men is to view sex and aging as "loss" rather than "challenge."

The psychobiosocial model of understanding and changing the couple's sexual script was the organizing concept of therapy. All three factors need to be carefully assessed and addressed. Just as important is to replace the "poisons" that subvert healthy couple sexuality with positive psychological, biological, and relational strategies and techniques. Sexuality is a "team sport" focused on pleasure rather than an individual sex performance with intercourse as the pass-fail test.

For Michael, the empowering psychological cognitions were being a "wise" man and "taking pride in beating the odds." Michael's "trap" had been to obsess about his erection and intercourse. The new male and couple mantra is "desire, pleasure, eroticism, and satisfaction." Sexually, especially after 50, desire is the premier issue, not performance or orgasm. These concepts were reinforced throughout the couple therapy sessions (and during occasional individual sessions with Michael). Between sessions Michael and Laura were assigned psychosexual skill exercises using the format of "read, talk, and do." In a typical gender split, Laura valued the reading and talking with its concepts of being an intimate, erotic team, nondemand pleasuring, the GES approach, and couple bonding and satisfaction. Michael emphasized implementing psychosexual skills and engaging sexually. Laura was more accepting of variable, flexible sexual scenarios than was Michael. The key psychological change for Michael was how much better he felt about himself and the relationship when they shared orgasmic experiences whether during the pleasuring/eroticism phase, during intercourse, or in afterplay.

Biologically, the most helpful intervention was a couple consultation with their internist. When the couple attend together and make it clear to the physician that they want to be active, involved patients, focus on health behaviors, and deal with side-effects of medications (not ask the physician to do sex counseling), it brings out the best in the physician. In the couple consultation, Michael wanted reassurance that he was a reasonably healthy 67-year-old man who was capable of enjoying sexuality, although not in the same way he had 30 years ago. The physician's explanation of vascular and cardiology changes was particularly useful for Michael. In addition, the physician suggested altering medication doses and, even more importantly, changing the times he took his medications so sexual side-effects were reduced.

Relationally, Michael needed Laura to be both his intimate and erotic ally. Michael's receptivity and responsivity to Laura's sexual expressiveness was powerfully motivating for her. Desire is the core issue in couple sexuality and the combination of Laura feeling wanted by Michael and the freedom to have her own sexual initiatives and preferences enhanced couple sexuality. An involved, aroused partner is the best aphrodisiac.

Improved psychosexual skills and adopting a variable, flexible sexual repertoire enhanced Michael's sexual desire. Accepting that it is better to transition to intercourse at high levels of erotic flow, allowing Laura to initiate the transition to intercourse and to guide intromission, and to enjoy giving and receiving multiple stimulation during intercourse enhanced sexual desire and function for Michael. The biggest challenge for Michael was to accept that when sex did not flow to intercourse they could seamlessly transition (without panicking or apologizing) to a mutual erotic scenario (Michael did not enjoy a sexual scenario where he was only the giving or receiving partner, nor did he appreciate sensual scenarios). Although Laura was fine with asynchronous and sensual scenarios, she respected Michael's preferences.

Relapse Prevention Strategies and Techniques

Sexuality, especially sexual desire, cannot be treated with benign neglect. Michael

and Laura were followed for 2 years with 6-month follow-up sessions. The format was to process what was going well, address psychological, relational, or sexual concerns, and set a new intimacy, pleasuring, or eroticism goal for the next 6 months. The theme was to continue to grow their sexual relationship. In addition, if there were any concerns or problems, they were urged to call for a "booster" session.

For Michael to maintain sexual desire, the prime challenge was to embrace GES as "first-class." Laura's embracing of the GES approach and her obvious enjoyment of variable, flexible sexual expression (both her own and Michael's) strongly reinforced his desire. He understood he would not get support or validation from his male peers, but the cognition "We're going to beat the odds and enjoy couple sexuality in our 60s, 70s, and 80s" was highly motivating.

Michael found the 6-month follow-up sessions helped him remain accountable. He especially enjoyed the goal of developing a new sexual scenario during the next 6 months. His favorite new scenario was to take turns manually and orally stimulating each other to orgasm. Laura was very pleased that Michael had learned to value asynchronous as well as mutual sexual scenarios.

Summary

Conceptually, clinically, and empirically, the issue of male sexual desire is poorly understood. It is crucial to develop a model for valuing and maintaining male sex desire in serious relationships with the aging of the man and aging of the relationship.

Both primary and secondary HSDD needs to be carefully assessed and treated. Particularly challenging is developing the commitment to GES as the most appropriate model for male and couple sexual desire and function. Confronting the traditional male sex performance model of totally predictable erections, intercourse, and orgasm and replacing it with the variable, flexible, pleasure-oriented GES model is key to male sexual desire with aging. This new model of male and couple sexuality is a challenge for CBT couple therapists and for the public.

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Call *for* Papers President's New Researcher

ABCT's 2011–2012 President, Robert K. Klepac, Ph.D., ABPP, invites submissions for the 34th Annual President's New Researcher Award. The winner will receive a certificate and a cash prize of $500. The award will be based upon an early program of research that reflects factors such as: consistency with the mission of ABCT; independent work published in high-impact journals; and promise of developing theoretical or practical applications that represent clear advances to the field. While nominations consistent with the conference theme are particularly encouraged, submissions will be accepted on any topic relevant to cognitive behavior therapy, including but not limited to topics such as the development and testing of models, innovative practices, technical solutions, novel venues for service delivery, and new applications of well-established psychological principles. Submissions must include the nominee's current Curriculum Vita and one exemplary paper. Eligible papers must (a) be authored by an individual with five years or less posttraining experience (e.g., post-Ph.D. or post-residency); and (b) have been published in the last two years or currently be in press. Submissions will be judged by a review committee consisting of Robert Klepac, Ph.D., Debra A. Hope, Ph.D., and Stefan Hofmann, Ph.D. (ABCT's President, Immediate Past-President, and President-Elect). **Submissions must be received by Monday, August 6, 2012, and must include four copies of both the paper and the author's vita and supporting letters if the latter are included**. Send submissions to ABCT President's New Researcher Award, 305 Seventh Ave., 16th floor, New York, NY 10001.

A Reflection on Mentorship (and Why YOU Should Join the ABCT Mentorship Directory)

Jennifer Lundgren, ***University of Missouri-Kansas City***

I have thought a lot about mentorship over the past year since assuming the role of training director for an APA-accredited clinical psychology doctoral program. This is a sometimes challenging, but often rewarding role that has presented me with the opportunity to interact with many current and prospective doctoral students. Frequently, as I meet with prospective graduate students, I am reminded of just how fortunate I was to have great undergraduate mentorship (thank you, Sue Orsillo and Brian Marx) when I applied to graduate school and graduate mentorship (thank you, Drew Anderson) to help me navigate my way through the graduate training process.

I can vividly remember standing in the lobby of the ABCT conference hotel waiting to "interview" with my future doctoral mentor. Later in my training we enjoyed a laugh while he shared with me the look of panic that must have come over my face when I was introduced to him. It was at this same meeting that I had the pleasure of interacting with other potential doctoral mentors during the poster session (and the anxiety of navigating my way through Washington, DC, for the first time so that I could take the Psychology GRE, because it was the last time it was offered before applications were due!). It was also at this meeting that I experienced firsthand the role that ABCT plays in the academic training and professional development of so many cognitive-behaviorally oriented health-care professionals.

As many *tBT* readers know, applying to graduate school can be an overwhelming process, filled with anxiety about GRE scores, having the "right" type of research experience, and what it is, exactly, that admissions committees are looking for in a "personal statement." Having served on the graduate admissions committee in my program several times, I am quick to remind students that an equally important part of the application is the student/mentor match. As a student, you devote several years of your life to graduate training and often to the work of your mentor. It is important, therefore, that you put as much effort into finding the right mentor as you put into finding the right program (or the one that is closest to the beach!). As a mentor, it is equally important that we put effort into selecting students who best fit our training program, research team needs, and interpersonal style.

Throughout one's career, finding the right mentor is an often necessary, but not always sufficient, ingredient for professional success. Workplace mentorship research has shown that mentored versus nonmentored workers fare better in both objective and subjective outcomes, including more compensation and promotions, more commitment to one's career, and greater job and career satisfaction (Allen, Eby, Poteet, Lentz, & Lima, 2004). In the context of graduate training in psychology, Forehand (2008) reported that successful mentorship (a) begins with the selection of doctoral students who are a good fit with the mentors, (b) involves instrumental behaviors on the part of the faculty member (e.g., hold research meetings, help student develop research goals), and (c) requires that the faculty mentor possess certain characteristics that facilitate the mentorship relationship (e.g., engaged with students, open to student ideas).

In the spirit of Forehand's (2008) first criterion for successful mentorship, the ABCT Academic Training Committee, along with the ABCT central office staff, recently launched an online Mentorship Directory to help facilitate the graduate student-mentor match. The Mentorship Directory is modeled after the ABCT Find-a-Therapist directory, and it has two goals: (a) help future graduate students locate and connect with cognitive-behaviorally oriented graduate mentors and (b) provide members of ABCT an opportunity to recruit graduate students with an interest in CBT.

The directory is searchable based on several variables, including geographic location, program training model (e.g., scientist practitioner, clinical scientist, scholar practitioner), populations served/studied, specialty area (e.g., depression, eating disorders, anxiety disorders), and whether or not a mentor plans to accept a new graduate student in the current application cycle. Mentors (ABCT members) can easily join the directory by visiting the Mentorship Directory home page (http://www.abct.org/Mentorship/?m=mMentorship&fa=meMain) on the ABCT website (www.abct.org). To join or update your information, you need to enter your ABCT number (which you can obtain by contacting the ABCT central office) and your last name. Anyone, including the general public, can search the directory for free by following the same link.

On behalf of the ABCT Academic Training Committee, I encourage all graduate program mentors to join the directory and to invite your ABCT colleagues to join as well. It is important for the future of CBT and ABCT that the next generation of mental health professionals have the opportunity to connect with the many outstanding members of ABCT and to develop lasting professional relationships that will benefit the field for years to come.

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Nominations for ABCT Officers: Get in on Choosing the Leaders

Raymond DiGiuseppe, *Chair, Leadership and Elections Committee*

We are approaching the nominations period for ABCT's elected leadership positions. Professional organizations are as strong as their members' participation. ABCT belongs to all of us and the selection of leaders represents the single most important task that members accomplish. Please take ownership of your association and participate in the leadership selection process. Make this the year you guide your professional home and make a contribution by running for office or take an active role in selecting our leaders. If you ask members who have previously served in a leadership role in ABCT why they participated, they all share similar reasons for participating in the leadership: They wanted to make a difference, and they did. Will you or someone you know run for office? In addition to the inherent satisfaction achieved from contributing to ABCT, you have the opportunity to develop new friendships while reconnecting with old ones.

This coming year we need nominations for three elected positions: President-Elect, Secretary-Treasurer, and Representative-at-Large. Those members who receive the most nominations will appear on the ballot. In April, members in good standing vote for the candidates of their choice to serve for 3 years. The President-Elect will serve in that function from 2012–2013, then as President from 2013–2014, and then as Past President from 2014–2015. The Secretary-Treasurer, one of the most important roles in the organization, serves as the Chief Financial Officer. Reviewing annual income and expense, reviewing budget projections, and making recommendations for our investment policies allow us to prosper as an organization. The Board approves the budget each year and the Secretary-Treasurer has oversight.

Each Representative-at-Large serves as a liaison to one of the branches of the Association. The representative position up for election this year serves as the liaison to the Convention and Education Issues Coordinator. Our convention is one of the most important things we do. This Representative-at-Large serves a crucial role ensuring that we maintain high-quality conventions. All full members in good standing are eligible to be nominated, and there is no limit to the number of members you can nominate for any of the positions.

Electioneering starts at the Annual Convention. So if you are interested in running for office, or if you have a candidate in mind, start the campaign now with the nominations and go to the Annual Convention and start making your case to the electorate.

Nominating a candidate is very easy. Nomination ballots will be posted on our web page, printed in several issues of *the Behavior Therapist* (see below), and will be available at the convention.

How to Nominate: Three Ways

- ➔ Mail the form to the ABCT office (address below)
- ➔ Fill out the nomination form by hand and fax it to the office at 212-647-1865
- ➔ Fill out the nomination form by hand and then scan the form as a PDF file and email the PDF as an attachment to our committee: membership@abct.org.

The nomination form with your original signature is required, regardless of how you get it to us.

Good governance requires participation of the membership. ABCT needs you participation to insure good governance and to continue to thrive as an organization. ■

NOMINATE the Next Candidates for ABCT Office

I nominate the following individuals:

PRESIDENT-ELECT (2012–2013)

_____

_____

REPRESENTATIVE-AT-LARGE (2012–2015)

_____

_____

SECRETARY-TREASURER

_____

_____

NAME (printed)

_____

SIGNATURE (required)

_____

2012 Call for Nominations

Every nomination counts! Encourage colleagues to run for office or consider running yourself. Nominate as many full members as you like for each office. The results will be tallied and the names of those individuals who receive the most nominations will appear on the election ballot next April. **Only those nomination forms bearing a signature and postmark on or before February 1, 2012, will be counted.**

Nomination acknowledges an individual's leadership abilities and dedication to behavior therapy and/or cognitive therapy, empirically supported science, and to ABCT. When completing the nomination form, please take into consideration that these individuals will be entrusted to represent the interests of ABCT members in important policy decisions in the coming years. Contact the Leadership and Elections Chair for more information about serving ABCT or to get more information on the positions.

Please complete, sign, and send this nomination form to Raymond DiGiuseppe, Ph.D., Leadership & Elections Chair, ABCT, 305 Seventh Ave., New York, NY 10001.

18th Annual Awards & Recognition

Call *for* Award Nominations

The ABCT Awards and Recognition Committee is pleased to announce the 2012 awards program. Nominations are requested in all categories listed below. Please visit our website in December for specific submission instructions. Please note that award nominations may not be submitted by current members of the ABCT Board of Directors.

Outstanding Contribution by an Individual for Educational/Training Activities

Eligible candidates for this award should be members of ABCT in good standing who have provided significant contributions toward educating and training behavior therapists. Past recipients of this award include Gerald C. Davison in 1997, Leo Reyna in 2000, Harold Leitenberg in 2003, Marvin R. Goldfried in 2006, and Philip C. Kendall in 2009. **Please check ABCT's website in December for instructions on submitting nominations.**

Outstanding Mentor

This year we are seeking eligible candidates for the Outstanding Mentor award who are members of ABCT in good standing who have encouraged the clinical and/or academic and professional excellence of psychology graduate students, interns, postdocs, and/or residents. Outstanding mentors are considered those who have provided exceptional guidance to students through leadership, advisement, and activities aimed at providing opportunities for professional development, networking, and future growth. Appropriate nominators are current or past students of the mentor. The first recipient of this award was Richard Heimberg in 2006, followed by G. Terence Wilson in 2008, and Richard J. McNally in 2010. **Please check ABCT's website in December for instructions on submitting nominations.**

Student Dissertation Awards:

- **Virginia A. Roswell Student Dissertation Award ($1,000)**
- **Leonard Krasner Student Dissertation Award ($1,000)**
- **John R. Z. Abela Student Dissertation Award ($500)**

Each award will be given to one student based on his/her doctoral dissertation proposal. The research should be relevant to behavior therapy. Accompanying this honor will be a monetary award (see above) to be used in support of research (e.g., to pay participants, to purchase testing equipment) and/or to facilitate travel to the ABCT convention. Eligible candidates for this award should be student members who have already had their dissertation proposal approved and are investigating an area of direct relevance to behavior therapy, broadly defined. A student's dissertation mentor may complete the nomination. Self-nominations are also accepted. Nominations must be accompanied by a letter of recommendation from the dissertation advisor. **Please check ABCT's website in December for instructions on submitting nominations.**

Distinguished Friend to Behavior Therapy

Eligible candidates for this award should NOT be members of ABCT, but are individuals who have promoted the mission of cognitive and/or behavioral work outside of our organization. Applications should include a letter of nomination, three letters of support, and a curriculum vitae of the nominee. Past recipients of this award include Jon Kabat-Zinn, Nora Volkow, John Allen, Anne Fletcher, Jack Gorman, Art Dykstra, Michael Davis, Paul Ekman, and The Honorable Erik K. Shinseki. **Please check ABCT's website in December for instructions on submitting nominations.**

Career/Lifetime Achievement

Eligible candidates for this award should be members of ABCT in good standing who have made significant contributions over a number of years to cognitive and/or behavior therapy. Applications should include a letter of nomination, three letters of support, and a curriculum vitae of the nominee. Past recipients of this award include Albert Ellis, Leonard Krasner, Steven C. Hayes, David H. Barlow, G. Alan Marlatt, and Antonette M. Zeiss. **Please check ABCT's website in December for instructions on submitting nominations.**

NOMINATIONS FOR THE FOLLOWING AWARD ARE SOLICITED FROM MEMBERS OF THE ABCT GOVERNANCE:

Outstanding Service to ABCT

Please check ABCT's website in December for instructions on submitting nominations.

We appreciate your patience as the Awards & Recognition Committee transitions to a new leadership: full details on submitting nominations will be available on ABCT's website in December.

Nominate on line: www.abct.org

Deadline for all nominations: March 1, 2012

Full Members

Robin L. Aupperle
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Samuel Cooper
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Emily Justine Dunn
Alexis Elmore
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Thomas J. Flanagan
Brett M. Goshe
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Stephanie Kerrigan
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Neha Mitragotri
Nicholas Perry
Kristen Reinhardt
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Miya L. Barnett
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Call for

Continuing Education Sessions

46th Annual Convention | November 15–18, 2012

National Harbor, MD

Workshops

Workshops cover concerns of the practitioner / educator / researcher. Workshops are 3 hours long, are generally limited to 60 attendees, and are scheduled for Friday and Saturday.

Jillian Shipherd, *Workshop Committee Chair*
workshops@abct.org

Institutes

Institutes, designed for clinical practitioners, are 5 hours or 8 hours long, are generally limited to 40 attendees, and are scheduled for Thursday.

Risa Weisberg, *Institute Committee Chair*
institutes@abct.org

Master Clinician Seminars

Master Clinician Seminars are opportunities to hear the most skilled clinicians explain their methods and show taped demonstrations of client sessions. They are 2 hours long, are limited to 40 attendees, and are scheduled Friday through Sunday.

L. Kevin Chapman, *Master Clinician Seminar Committee Chair*
masterclinicianseminars@abct.org

Please send a 250-word abstract and a CV for each presenter. For submission requirements and information on the continuing education session selection process, please see the Frequently Asked Questions section of the ABCT Convention page at **www.abct.org**.

Submission deadline: February 1, 2012

ABCT AMBASSADORS

are needed to:

messenger — ***Articulate ABCT's vision, purpose, and identity to encourage membership***

guide — ***Mentor individuals through the process of presenting at the annual convention or transitioning into leadership positions***

signify — ***Act as the eyes and ears of the association locally***

ABCT's Ambassador program is a brand-new initiative promoting leadership, participation, and membership in ABCT.

ABCT Ambassadors are easily recognized at the annual meeting by their special ribbons. They also receive a certificate of recognition and are featured on our website and in tBT.

For more information, contact Lisa Yarde at ABCT's central office (lyarde@abct.org)

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