Biopsychosocial Assessment of a Mindfulness-Oriented Cognitive Intervention for Alcohol Dependent Adults

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ABSTRACT

ERIC GARLAND: Biopsychosocial Assessment of a Mindfulness-Oriented Cognitive Intervention for Alcohol Dependent Adults
(Under the direction of Matthew Howard)

A biopsychosocial approach is needed to comprehend the complex pathogenic processes implicated in alcohol dependence. The following three papers employ such an approach to explore key research questions: (a) How might stress precipitate alcohol misuse, dependence, and relapse, and (b) How can targeted psychosocial interventions influence this process? The first paper presents a novel conceptual framework integrating formerly discrete theories of stress appraisal, neurobiological allostasis, automatic cognitive processing, and addictive behavior to explain how alcohol misuse and dependence is maintained and re-activated by stress. This theoretical framework underpins the measurement model and intervention that are the focus of the second and third papers. The second paper explores relationships between baseline dispositional mindfulness and alcohol attentional bias among a sample of recovering alcohol dependent adults, relationships that are critical to our argument in support of mindfulness training as a treatment for alcohol dependence. The third paper in this series is a report of a randomized controlled pilot trial comparing the effects of a mindfulness-oriented intervention to those of an alcohol dependence support group. Results from this study provide tentative support for the proposed theoretical framework and for the use of mindfulness as a treatment for alcohol dependence.
To my grandparents, Murray and Norma Garlitsky, who lived near the edge of poverty and whose family name was surrendered to avoid the savage inequities of ethnic discrimination; and to my mother, Eileen Garland, who would have loved to read this work and witness her son become the next Dr. Garland.
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TARGETING COGNITIVE-AFFECTIVE RISK MECHANISMS IN STRESS-PRECIPITATED ALCOHOL MISUSE, DEPENDENCE, AND RELAPSE:
AN INTEGRATED, BIOPSYCHOSOCIAL MODEL OF
ALLOSTASIS, AUTOMATICITY, AND ADDICTION

ABSTRACT

This review presents a conceptual integration of formerly discrete theories of stress appraisal, neurobiological allostasis, automatic cognitive processing, and addictive behavior to explain how alcohol misuse and dependence is maintained and re-activated by stress. We outline a risk chain in which psychosocial stress initiates physiological arousal, perseverative cognition, and negative affect that in turn triggers automatized schema to compel alcohol consumption. This implicit cognitive process then leads to attentional biases towards alcohol, subjective experiences of craving, paradoxical rebound effects of urge suppression, and palliative coping through drinking. When palliative coping relieves distress, it results in negative reinforcement conditioning that perpetuates the cycle by further sensitizing the system to future stressful encounters. This conceptual framework has implications for development and implementation of innovative behavioral interventions such as mindfulness training that may disrupt cognitive-affective mechanisms underpinning stress-precipitated dependence on alcohol.
Alcohol dependence remains prevalent despite a century of intervention efforts. Even with apparently efficacious behavioral and pharmacological treatments, relapse following treatment is the norm, and long-term recovery rates are low. According to the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), 28.4% of persons ever treated for alcohol problems remain dependent on alcohol and 19.1% continue to exhibit alcohol abuse or subclinical dependence symptoms over the past year (Dawson, Grant, Stinson et al., 2005); hence, certain risk chains leading to the development and maintenance of alcohol dependence may be intractable to extant interventions. One such pathway may involve positive feedback loops between stress appraisal, emotion dysregulation, physiological arousal, implicit cognition, and palliative coping with alcohol. As components of this stress-initiated risk chain may be malleable to novel behavioral therapies targeting cognitive-affective mediators of pathogenic gene-environment interactions, further explication of the pathways underpinning stress-precipitated alcohol dependence appears warranted.

The stress reaction appears to be a key mechanism underlying alcohol dependence, intensifying alcohol consumption and precipitating relapse; indeed, persons who drink alcohol to cope with stress and negative affect exhibit significantly higher rates of lifetime and current alcohol dependence symptoms than persons who drink for other reasons (Schroder & Perrine, 2007), and increases in stress can precipitate a shift towards heavy and more frequent alcohol consumption (Dawson, Grant, & Ruan, 2005). Although the causal associations between stress and alcohol dependence have yet to be fully specified, clinical experience and empirical research suggest that alcohol is often used to “self-medicate” averse cognitive-emotional and psychophysiological sequelae of
the stress response (Khantzian, 1997). NESARC data have provided epidemiological data on stress and drinking behavior. Among adult past-year drinkers, 72.5% reported experiencing at least one stressful life event in the past year, and 23.2% had experienced 3 to 5 such stressors (Dawson, Grant, & Ruan, 2005). Annual drinkers who reported experiencing six or more stressful life events had consumed more than three times the amount of daily ethanol and evidenced more than thrice the frequency of heavy drinking compared to drinkers who had not experienced life stressors in the past year (Dawson, Grant, & Ruan, 2005). Each experience of a past-year stressful life event increased the frequency of heavy drinking by 24% for men and 13% for women, and increases in stress were associated heavier patterns of alcohol consumption (Dawson, Grant, & Ruan, 2005).

Congruent with these findings, an event-history analysis of 1786 urban, young adults found that both cumulative distal and proximal exposure to stressful life events significantly predicted risk of alcohol dependence onset in a linear and additive fashion even after controlling for socioeconomic status and history of psychiatric disorder, implicating a causal role for life stress in the etiology of alcohol use disorders (Lloyd & Turner, 2008). Clearly, life stress is prevalent among alcohol users, and is an important predictor of heavy drinking and alcohol dependence.

The risk chain linking the experience of stress to alcohol dependence may be explicated via the integration of transactional stress-coping theory (Lazarus & Folkman, 1984) with the allostatic model of alcohol dependence (Koob, 2003; Koob & Le Moal, 2001), the cognitive processing model of craving and compulsive alcohol use (Tiffany, 1990; Tiffany & Conklin, 2000), the affective processing model of negative reinforcement (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004), and the second-order
cybernetic model of stress, metacognition, and coping (Garland, 2007). According to this integrated framework, alcohol dependence involves implicit cognitive operations (Wiers et al., 2006; Wiers, Teachman, & De Houwer, 2007) and attentional biases towards alcohol-relevant stimuli (Field, 2006) that organize and drive the appetitive, motivational states and maladaptive drug-seeking behaviors that characterize this disorder. In brief, repeated alcohol misuse establishes automatic alcohol-use action schemas that impel continued abuse of the substance through automatized sequences of context-dependent behavior (Tiffany, 1990). Nonautomatic inhibition of alcohol-use action plans manifests in the subjective experience of craving (Tiffany, 1990, 1999; Tiffany & Conklin, 2000), a factor which appears to drive continued alcohol use (de Wit, 2000; Flannery et al., 2001; Monti, Rohsenow, & Hutchison, 2000). Psychosocial stress and negative affect evoke these automatic and nonautomatic cognitive operations implicated in alcohol dependence (Field & Powell, 2007), leading to increased motivation to imbibe alcohol as a means of palliative coping (Olff, Langeland, & Gersons, 2005). In turn, such palliative coping through alcohol is sustained through negative reinforcement conditioning (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004). Continued operation of this self-perpetuating cycle, which may be conceptualized as a feedback loop, leads to an ever-deepening dependence on alcohol fueled by an increasingly heightened sensitivity to stress, a self-destructive pattern that afflicts vulnerable members of society.

Given the biopsychosocial dimensions of this risk chain, an integrated conceptual framework is needed to explicate the causal pathways leading to stress-precipitated alcohol misuse, dependence, and relapse. The present manuscript presents a conceptual integration of formerly discrete theories of stress appraisal, neurobiological allostasis,
automatic cognitive processing, and addictive behavior to explain how alcohol
dependence is maintained and re-activated by stress. This conceptual framework has
implications for development and implementation of innovative behavioral interventions
that disrupt mechanisms underpinning stress-exacerbated dependence on alcohol.

An Integrated Biopsychosocial Framework of Stress-Precipitated Alcohol Misuse,
Dependence, and Relapse

The etiology of alcohol use disorders is complex and multifactorial, involving
interactions between genetic, environmental, interpersonal, and intrapersonal factors.
Over time, as alcohol consumption becomes compulsive, automatic appetitive reactions
begin to supersede controlled, volitional use (Wiers & Stacy, 2006). Once patterns of
alcohol dependence are established, self-regulatory mechanisms are hijacked by the
addictive process, and consumption of alcohol is maintained despite willful intent to
abstain. Even repeated exposure to aversive consequences (e.g., loss of a spouse,
termination from a job, legal and health problems) may not be enough to discourage the
alcohol dependent individual from further drinking, and attempts to remain abstinent
often eventuate in relapse. The question of why alcohol consumption persists in spite of,
and perhaps, due to, adversity has been the subject of theory and scientific investigation.

Early motivational theories posited a relation between alcohol consumption and
stress. The tension reduction hypothesis, originating from animal experiments (Conger,
1951, 1956), claims that stressful life circumstances motivate alcohol consumption, and
under such aversive or conflict-laden conditions, alcohol decreases anxiety, which then
reinforces subsequent alcohol consumption (Cappell & Herman, 1972). Despite its initial
popularity, this theory lost favor because there was little agreement regarding the
conditions under which alcohol dampens the stress response, and some aversive conditions actually decrease alcohol consumption (e.g. Caplan & Puglisi, 1986). Inconsistent evidence of tension reduction-related drinking motivations in humans has been attributed to differences in alcohol expectancies, that is, beliefs about alcohol’s supposed ameliorative effect on distress (Young, Oei, & Knight, 1990).

In an influential paper that demonstrated the stress-response-dampening effects of alcohol in humans, Levenson et al. (1980) raised the possibility that cognitive factors might mediate the pharmacological effects of alcohol on physiological reactivity. Sons of male alcoholics have been shown to exhibit heightened autonomic stress responses that are dampened by the effects of alcohol (Pihl, Finn, & Peterson, 1989); such stress-response dampening has been shown to be highly correlated with executive function deficits indicative of prefrontal cortical dysfunction in descendants of alcoholic probands (Peterson, Finn, & Pihl, 1992). Pihl, Peterson, and Finn (1990) hypothesized that persons who drink alcohol to dampen stress have neurocognitive tendencies towards misattributing threatening significance to novel stimuli, resulting in augmented arousal, while exhibiting attenuated responses to stimuli that require sustained attention for processing.

The relationship between attentional factors and stress-response-dampening was addressed in Steele and Joseph’s attention-allocation model (1988; 1990). This model posited that drinking reduces stress via alcohol myopia, that is, a pharmacologically-induced impairment in controlled cognitive processing coupled with a narrowed attentional focus onto immediate internal and external cues. Such myopia is hypothesized to reduce capacity for stressful cognition in the face of a demanding task, as well as limit
attention to immediately present stimuli rather than future threats. Hence, this model predicts that alcohol consumption will result in stress-response-dampening when attention to stressors is restricted or divided by task demands, a prediction that has been supported by several studies (Curtin, Lang, Patrick, & Stritzke, 1998; Curtin, Patrick, Lang, Cacioppo, & Birbaume, 2001; Josephs & Steele, 1990; Steele & Josephs, 1988). However, this model has received critical refutation from evidence that suggests that, even without attentional manipulations, moderately high doses of alcohol can robustly reduce negative emotion (Donohue, Curtin, Patrick, & Lang, 2007). Recent research has helped to reconcile this incongruity: using a social stress induction, alcohol was shown to exert direct stress-response-dampening effects on heart rate, galvanic skin response, and subjective anxiety, but the effects of drinking on stress-induced skin conductance responses were partially mediated by differences on a sustained attention task (Sher, Bartholow, Peuser, Erickson, & Wood, 2007). Because drinking appears to exert effects on stress reactivity through its influence on attention, attentional mechanisms and related cognitive processes may be an important link in the association between stress and alcohol consumption.

Building on such earlier work, we argue that alcohol dependence is maintained, in part, by automatic and implicit cognitive processes which subvert and bypass the conscious desire to abstain from alcohol. Stress and negative affect play a large role in activating the appetitive automaticity and attentional biases underpinning alcohol dependence maintenance and relapse. The proposed conceptual framework of risk mechanisms implicated in stress-precipitated maintenance of and relapse to alcohol dependence integrates a transactional stress-coping model (Lazarus & Folkman, 1984;
Olff et al., 2005) with an allostatic model of alcohol dependence (Koob, 2003; Koob & Le Moal, 2001), a cognitive processing model of craving and compulsive alcohol use (Tiffany, 1990; Tiffany & Conklin, 2000), and an affective processing model of negative reinforcement (Baker et al., 2004). This integrated framework, which builds upon a conceptual model of stress, metacognition, and coping (Garland, 2007), describes a cybernetic, informational circuit; as such, the causal flow loops back upon itself, with the output of the circuit (i.e., relapse) becoming its own input (i.e. a stressor) in further iterations of the cycle. Figure 1 (below) depicts this circuit, which is then detailed in the text that follows.

Figure 1. An integrated, biopsychosocial framework of stress-precipitated alcohol misuse, dependence, and relapse
Stress and cognitive appraisal activate the risk chain. Though some models of stress and addiction treat stress exposure as a monolithic concept, stress is a multicomponent process modulated by biopsychosocial factors. Among the numerous factors that influence the stress process, cognitive appraisal may be viewed as a central governor of the system. Although the stress concept derived from the physical sciences, biological organisms subjected to stressors are quite unlike inorganic objects which deform predictably and systematically under the external force of a load. Organisms actively construct their phenomenological experiences by coupling with the medium of the environment according to their own self-organizing structure (Maturana & Varela, 1987; Varela, Thompson, & Rosch, 1991); in other words, an animal with “a nervous system perceives the world according to its own linkages and activities, not as a readout of some objective reality” (Lewis, 2001). Thus, humans encounter environmental stimuli and interpret the meaning of events and situations of their lives according to their perceived relevance to self and others, a perception which is shaped by the historical, sociocultural, and environmental context in which the individual is embedded.

This self-organizing interpretive or evaluative process, known as appraisal, may fundamentally modulate the physiological stimulus-response relationship, allowing for substantial behavioral variation in the organism’s adaptation to the environment. Indeed, although an extensive range of diverse stimuli may activate the common set of cortical, sub-cortical, neuroendocrine, and autonomic systems involved in the stress response, appraisal accounts for qualitative and quantitative differences in stress reactivity within and between individuals (Lazarus & Folkman, 1984). Exposed to the same stressor, one
individual may respond with depression and helpless apathy, another with violence and rage, and a third with optimism and constructive, prosocial action.

A wide array of life events may initiate the stress process. Compared to the environment of our ancestors where the scenario of fleeing from a wild beast was more commonplace, the usual context of most industrial and information-age societies less frequently presents humans with immediately life-threatening stressors. On the other hand, there are still many countries today where violence and death are encountered frequently due to war, crime, or famine. Nevertheless, we more often face stress from our attribution of symbolic meaning to psychosocial events deemed relevant to well-being (Rosmond, 2005). Persons from the middle- and upper-classes are threatened by possible loss of financial security, marital strife, and the downsizing of companies. In contrast, persons from the marginalized strata of society experience insidious, pervasive, constant stress in the form of deprivation, discrimination, and intimidation. Among persons confronted with the inequities of poverty and oppression, who already face chronic, environmental stress from the load of multiple economic, health, and social disparities, stressors such as the loss of a job present a very real danger that may initiate sequelae deleterious to mind and body.

Modern society places demands upon the individual that require one to cope with often high levels of stress. In the face of war, economic recession, and ecological crisis, individuals and communities bear the brunt of societal and global catastrophes, compounded by the daily hassles of living. Vulnerable populations who are less able to cope due to a lack of social, economic, or cognitive-emotional resources evidence the adverse consequences of stress through mental and physical illness. Hence, stress-
induced pathology presents a serious problem for a society increasingly subjected to both
global and local strains, especially so for its most vulnerable members.

Just as stress aggravates other forms of pathology, psychosocial stressors may
exacerbate alcohol misuse. Individuals who experience social, legal, or work-related
stressors report significantly more frequent heavy drinking days than those who do not
face such stressors (Dawson, Grant, & Ruan, 2005). Given the association between stress
and heavy drinking and the higher prevalence of social, legal, and job-related stressors
among poor persons, it follows that poverty is a significant correlate of higher levels of
alcohol consumption (Dawson, Grant, & Ruan, 2005). Indeed, past-year prevalence of
alcohol dependence was highest among persons making less than $20,000 a year in 2002,
and the odds of meeting criteria for alcohol dependence in the past year and over one’s
lifetime are greatest for those with lower incomes (Hasin et al., 2007). Additionally,
among poor persons, job-related stress was found to increase alcohol consumption
quantity and frequency (Dawson, Grant, & Ruan, 2005). These findings echo those from
a study conducted nearly two decades ago: among a sample of 501 problem drinkers who
had been recruited at two large medical centers, problem drinkers reported having
significantly more financial and neighborhood-related stressors and significantly fewer
financial resources than non-problem drinkers, and financial stress was a significant
predictor of drinking problems (Brennan & Moos, 1990). On the other hand, heavy
drinking may impede social and occupational functioning, leading to socioeconomic
disadvantage. Although survey research cannot rule out the possibility of reciprocal
causation, it appears as if socioeconomic forces exert strain upon individuals that may
result in stress-precipitated alcohol consumption.
Whether confronted by psychosocial or physical stressors, the stress process initiates with a primary appraisal of stimuli for risk value. When a given stimulus is cognitively appraised as challenging, harmful, or threatening, an activation of physiological systems involved in the stress response co-occurs with the subjective experience of distress (Lazarus & Folkman, 1984). Subsequently, a cognitive process of secondary appraisal determines the sufficiency of available resources and coping options to meet the demands of the actual or potential harm. Appraisals may be automatic, executed without intention and performed without conscious deliberation (Bargh & Chartrand, 1999); for example, meta-analysis has shown that predictions about the intent and future behaviors of others are typically made in less than 30 seconds (Ambady & Rosenthal, 1992). Such rapid and unconscious appraisals may utilize hardwired reflexes, nondeclarative memory, and implicit cognitive operations, in contrast to intentional appraisal processes that rely upon declarative memory and propositional reasoning (Ellsworth & Scherer, 2002; Scherer, Schorr, & Johnstone, 2001). Implicit appraisals of threatening stimuli (e.g., angry facial expressions, loud sounds that might herald the approach of a predator, poisonous animals such as snakes and spiders) facilitate survival and thus may have been naturally selected for during human evolution (Ohman & Wiens, 2002).

From a biological perspective, appraisal may involve relaying visual, auditory, and somatic information about a stimulus from the thalamus to sensory processing areas of the cortex, activating affective processing circuits involving the amygdala, medial temporal lobe, and medial prefrontal and orbitofrontal cortices (LeDoux, 2002). This neural circuitry appears to compute the hedonic or threat value of the stimulus according
to previously established stimulus-reinforcement contingencies. For example, based on past experiences (e.g., previous encounters with strangers), a stimulus (e.g., facial expression of a passer-by on the street) is judged to be threatening, innocuous, or even rewarding. This computation may be modulated by prefrontal-amygdala circuits involving the anterior cingulate, prelimbic, and medial prefrontal cortices, which appear to temper and regulate stress reactivity through cognitive processing. In addition, inputs from the hippocampus may provide the amygdala with information about the stimulus context, allowing for differentiation between stimuli that in one context would be appraised as benign and in another, as dangerous. Stress appraisals discriminating threatening from benign stimuli (e.g., snakes from flowers) can be made within as few as 50 milliseconds (Ohman & Wiens, 2002). Backwards masking experiments, involving brief presentation of a target stimulus immediately followed by a mask of random noise, show threat appraisals can occur completely without consciousness via implicitly conditioned, subcortical thalamic-amygdala pathways (LeDoux, 2002; Ohman, 2005; Ohman, Carlsson, Lundqvist, & Ingvar, 2007)

Triggered by such appraisals, the stress reaction is instantiated through spreading neural activity from the amygdala to the hypothalamic-pituitary-adrenal (HPA) axis, the locus coerulesus, and the autonomic nervous system. Stimulus-evoked activation of this neural circuitry regulates physiological parameters through the multifarious feedforward and feedback processes of allostasis. Perception of threat triggers a neuroendocrine cascade from HPA axis, initiated by the central amygdala signaling the paraventricular nucleus of the hypothalamus to release corticotrophin-releasing hormone (CRH), stimulating the pituitary to secrete adrenocorticotropicin (ACTH), beta-endorphin, and
other peptides which in turn trigger release of cortisol from the adrenal cortex (Brosschot, Gerin, & Thayer, 2006; Kaye & Lightman, 2005). Cortisol exerts effects on nearly every cell of the body to redirect regulatory processes to meet the perceived challenge; these changes include mobilizing cellular energy resources via induction of liver enzymes, decreasing digestion, modulating the trafficking of immune cells, and influencing inflammatory processes through cytokine production (Chrousos & Gold, 1992; Kudielka & Kirschbaum, 2007). Cortisol also facilitates the encoding of fear-based memories by influencing neurotransmission between the amygdala and hippocampus (McEwen, 2007). Such hormonal regulation is comparatively slow, occurring over periods of several minutes, hours, or days (Janig, 2002).

In addition to these slower hormonal responses, stress appraisal activates the locus coeruleus (LC) in the brainstem to release the catecholamines nonadrenaline and adrenaline, which increase heart rate, blood pressure, and blood flow to skeletal muscles and the brain during the “fight-or-flight response” (Cannon, 1929; Chrousos & Gold, 1992). This stress-evoked survival response is also mediated through the rapid response (occurring within seconds) of sympathetic and parasympathetic neurons of the autonomic nervous system, consisting of reciprocally interconnected neural circuits between prefrontal cortex, amygdala, brainstem, viscera, and periphery that innervate muscle groups, drive and modulate the pacemaker of the heart, effect gastric contractions, stimulate sweat gland activity, and regulate shifts in body temperature (Janig, 2002). Under typical conditions, this system is tonically inhibited by prefrontal cortical regions such as anterior cingulate, orbitofrontal, ventromedial, and insular cortices, but is
disinhibited during threat perception to mobilize the body into defensive action (Thayer & Ruiz-Padial, 2006).

If, during this complex cognitive process of appraisal, available resources (e.g., individual, familial, or communal) are deemed insufficient to address the challenge presented by the threatening stimulus, then biopsychosocial consequences of stress may result. Prolonged or repeated stress activation may lead to an allostatic state, a chronic deviation of self-regulatory mechanisms from their normal mode of operation that leads to heightened sensitivity to threat and vulnerability to future stressors (McEwen, 1998). Among allostatic mechanisms at work is a feed-forward cycle between the amygdala and the HPA axis, whereby amygdala-triggered release of cortisol from the adrenals impairs hippocampal function while sensitizing the amygdala, leading to greater cortisol release during repeated exposures to the stressor (LeDoux, 2002; McEwen, 2007; Neville, Stutz, Lee, Davis, & Rosbash, 1997). Release of stress hormones also impairs function of the prefrontal cortex, which inhibits successful emotion regulation and heightens future stress reactivity (Arnsten, 1998; Arnsten & Goldman-Rakic, 1998). This state of hyperarousal results in allostatic load, a “wear and tear” on the body involving consequences such as include hippocampal atrophy (McEwen, 2003; 2007), as well as neuroendocrine (McEwen, 2007) and cardiovascular (Brosschot, Pieper, & Thayer, 2005; McEwen, 2006) dysregulation. Deleterious effects of stress can be moderated by effective coping. The appraisal process is dynamic and mutable; new data from the changing environment coupled with information about the effect of one’s behavioral responses to the threat may initiate a reappraisal, in which the original evaluation is changed as a result of feedback.
Problem- and emotion-focused coping ameliorate stress. Once an event is appraised as stressful, the individual may utilize problem- and emotional-focused coping efforts to deal with the stressor. Problem-focused coping consists of strategic attempts to manage or resolve the stressful event by gathering information, making decisions, and resolving conflict. Positive emotion can be generated when, as a result of successful coping efforts, the stressor event is resolved favorably; however, biopsychosocial distress intensifies when coping attempts are unsuccessful and the stressor is not resolved (Lazarus & Folkman, 1984). Lack of a favorable resolution may lead to deployment of emotion-focused coping efforts to manage the distress itself (e.g., positive reappraisal, a cognitive-affective regulatory strategy of re-interpreting the stressor event as benign or meaningful) (Folkman, 1997). Positive reappraisal, which appears to engage the prefrontal cortex and anterior cingulate to inhibit activation of the amygdala (Ochsner & Gross, 2005), is thought to attenuate negative emotions via the re-construal of the stressor event as meaningful and growth-promoting (Folkman & Moskowitz, 2000).

Positive reappraisal is an active coping strategy (Folkman, 1997), rather than a defense mechanism used to repress or deny. Unlike suppression of negative emotions which can cause increased sympathetic nervous system activation (Gross & Levenson, 1997), positive reappraisal does not typically lead to physiological or psychosocial complications (Gross, 2002; Ochsner, Bunge, Gross, & Gabrieli, 2002). In addition, positive reappraisal is often the first step toward a reengagement with the stressor event. For instance, a person stricken with a non-fatal heart attack might positively reappraise the event as an opportunity to change their lifestyle and subsequently begin to make changes in diet and exercise behaviors. Alternatively, a person who has recovered from
cancer might view their survival of the disease as evidence of their strength and resilience, and they might decide to dedicate their life to helping others make similar recoveries. Hence, positive reappraisal is often an adaptive rather than an avoidant strategy. However, in the absence of adaptive coping, stress leads to perseveration.

**Perseverative cognition exacerbates stress.** The stress response often results in perseverative cognition, a maladaptive process of fruitlessly maintaining a cognitive representation of the stressor in the absence of implementing adaptive coping behaviors (Brosschot et al., 2006). Such perseveration may involve activation of working memory circuits including the prefrontal cortex, hippocampus, and extended amygdala, whereby computations about present environmental stimulus contingencies are colored by past aversively conditioned relations stored in explicit memory systems (LeDoux, 2002). Regions in prefrontal cortex that appear to provide top-down governance of the amygdala during stress appraisal may become impaired during anxiety states, leading to amplified threat perception (Rauch, Shin, & Wright, 2003). Perseverative cognitive styles such as catastrophizing, the exaggeration of the threat value of a stimulus, or rumination, the experience of repetitive, intrusive negative thoughts about an event, result in runaway positive feedback loops between cognitive stress-appraisal processes, negative affect, and sustained activation of both the autonomic nervous system and its visceral efferents. Protracted activation of this pathway disrupts homeostasis of body systems through cortisol- and catecholamine-mediated stress-responses (Thayer & Brosschot, 2005). In the case of an alcohol dependent individual early in the process of recovery, this activation compound the physiological distress of conditions such as alcohol withdrawal.
Stress primes impulsive alcohol consumption via allostasis. Allostatic load from chronic, cognitively-driven, negative affective states may dysregulate stress and reward neurocircuitry within the extended amygdala, moving the brain reward set point from its normal level, resulting in decreased sensitivity to reward and increased sensitivity to punishment or aversive states (Koob & Le Moal, 2001). Stress-induced dysregulation of hedonic processing may be particularly pernicious among alcohol dependent individuals, who tend to favor immediate gratification and discount delayed rewards. This cognitive process of impulsive decision-making may be mediated by alterations in HPA-axis functioning (Mitchell, Fields, D'Esposito, & Boettiger, 2005), increased activation in posterior parietal cortex, dorsal prefrontal cortex, and parahippocampal gyrus regions (Boettiger et al., 2007), or a combination of decreased activity and structural abnormalities in orbitofrontal cortex (Dom, Sabbe, Hulstijn, & van den Brink, 2005). Given their tendency toward impulsivity exacerbated by a possible neurobiological shift of the reward set point, recovering alcohol dependent individuals under stress may turn to alcohol consumption to achieve hedonic allostasis (Koob, 2003) despite of potential future consequences of use. In this case, the impulse to drink may be subserved by automatic cognitive processes.

Automatic alcohol use schemata regulate addictive behavior. Perseverative cognition induces intense affective experiences of worry and dysphoria that may evoke alcohol use action schema, automatized, associative networks within which are encoded information for the nonvolitional execution of alcohol-use behaviors (Tiffany, 1990). Frequent drinking in response to stressors initially leads to the formation of behavior-outcome associations (Elsner & Hommel, 2001) as the palliative effects of alcohol
negatively reinforce drinking behaviors. At first such stress-precipitated drinking may stem from explicit expectancies that alcohol will provide relief from stress (Cooper, Frone, Russell, & Mudar, 1995; Stewart, Hall, Wilkie, & Birch, 2002) based on past experience of the rewarding and hedonic effects of alcohol. Over time, repeated drinking under stressful circumstances can lead to stimulus-response habits which may not be affected by aversive consequences. For example, among rats, self-administration of alcohol is rendered undeterred or insensitive to conditioned aversion (e.g. illness due to alcohol being contaminated with lithium chloride) (Dickinson, Wood, & Smith, 2002). This finding from basic science parallels observations of intractable drinking in the face of severe, stress-inducing consequences such as loss of a spouse or job.

Such schemata may arise out of a history of repeated alcohol consumption in much the same way that other overlearned behavioral repertoires become automatized. Stimulus-response habits are formed through repetition. After hundreds of repetitions of consistent responses to a given stimulus, attending and responding to that stimulus becomes automatic, leading to rapid processing in neural circuits involved in response execution (Chein & Schneider, 2005; Schneider & Chein, 2003). Automaticity requires the consistent training of associations without varying stimulus-response relationships (Schneider & Shiffrin, 1977; Shiffrin & Schneider, 1977). During formation of automatic habits, a neurobiological shift occurs in which behaviors that were originally guided by associative networks involving prefrontal cortical regions become controlled by sensorimotor cortico-basal ganglia networks (Yin & Knowlton, 2006). Addictive consumption of alcohol appears to derive, in part, from an automatized stimulus-response habit.
Automaticity underlying compulsive drinking may be compounded by changes in brain reward circuits caused by repeated alcohol consumption, dopaminergic neuroadaptations in the nucleus accumbens and ventral striatum that appear to result in sensitization to the rewarding effects of alcohol and alcohol-related cues (Robinson & Berridge, 1993, 2000, 2008). Such heightened incentive salience may impart compulsivity to alcohol-seeking behaviors, motivating the alcohol dependent person to drink despite countervailing reasons to remain abstinent (Robinson & Berridge, 2003). Thus, cues such as the sight of a bar, an advertisement in a magazine, or a familiar “drinking buddy” can reflexively trigger the desire to consume alcohol, long after one has gone through withdrawal and even after extended periods of abstinence. Once alcohol has been obtained, it may be consumed automatically, guided by implicit cognitive schemata.

Alcohol use action schemata may be subserved by neural circuits between the dorsal cingulate cortex, hippocampus, amygdala, striatum, and nucleus accumbens where conditioning and context-encoding neural projections motivate appetitive behavior (Everitt & Robbins, 2005). The rapid, automatic processing of addiction-related stimuli (including negative affective states and environmental-contextual stimulus configurations) via implicit schema may trigger conditioned appetitive and behavioral responses without deployment of conscious decision-making processes. Hence, the alcohol dependent person may find him or herself consuming alcohol without consciousness of the motive or intent to drink, in much the same way as other complex thought-action repertoires such as goal-pursuit can be engaged without conscious volition by conditioned contextual cues (Bargh & Chartrand, 1999).
Automatic cognitive processes appear to exert a significant influence on drinking. Indeed, implicit memory associations of alcohol with positive outcomes (e.g., providing relief from stress) are among the strongest predictors of future drinking behavior, even after controlling for lifetime alcohol use, explicit alcohol expectancies, and sociodemographic and personality variables (Stacy, 1997). Automatic alcohol approach associations were correlated with urge to drink after exposure to alcohol (Palfai & Ostafin, 2003). In contrast to problem drinkers who reported low levels of psychiatric distress, among problem drinkers high in psychiatric distress, negative affective words primed responses (i.e., speeded reaction times) to alcohol words (Zack, Toneatto, & MacLeod, 1999). Using the addiction Stroop task, Stewart and colleagues found that persons who drank alcohol to cope with stress exhibited priming effects to negative mood cues on alcohol words (Stewart et al., 2002).

The initiation of alcohol use action schema may be indexed by the addiction-Stroop paradigm, which, like the emotional Stroop paradigm used with affectively salient stimuli, is thought to index activation of automatic cognitive operations (Algom, Chajut, & Lev, 2004). The addiction-Stroop is an adaptation of the classic Stroop task wherein participants are tasked to identify the color of alcohol-related and neutral words; meta-analysis of 17 studies evidenced that relative to controls, alcohol dependent, alcohol abusing, and heavy-drinking individuals are slower to name the color of alcohol-related words than neutral words (Cox, Fadardi, & Pothos, 2006). For example, in the study with the strongest effect size (d = 2.07), 128 subjects comprised of 64 persons undergoing outpatient alcohol treatment and 64 controls participated in an alcohol Stroop task (Lusher, Chandler, & Ball, 2004). Relative to controls, alcoholics have significantly
slower color-naming responses to alcohol-related words. This finding suggests that alcohol-related stimuli may be automatically processed by persons with alcohol use disorders. However, slowed reaction times to alcohol cues on the addiction Stroop task may alternately index exogenous engagement of attention, capture of cognitive resources, or elicitation of subjective craving; all three processes may result in cognitive load which may impede goal-directed behavior (Field & Cox, 2008). Whether such disruption of cognitive resources contributes to alcoholics’ self-reported difficulty in using coping skills to resist alcohol cravings remains the subject of future empirical tests.

*Alcohol attentional bias is linked to craving.* Engagement of alcohol use action schemata may result in automatic processing of salient stimuli, manifested as an involuntary attentional bias towards alcohol cues. On visual probe tasks, heavy drinkers compared to light social drinkers preferentially attend to alcohol-relevant stimuli, evidencing decreased reaction times to probes replacing alcohol photographs relative to those replacing neutral photos (Field et al., 2007; Field & Eastwood, 2005; Field, Mogg, & Bradley, 2005; Field et al., 2004). In heavy drinkers, this bias occurs for alcohol cues presented for durations of 500 ms and 2000 ms, and not for stimuli presented only for 200 ms (Field et al., 2004). By contrast, abstinent alcohol abusers evidenced an attentional bias for alcohol-related photos presented for 50 ms, but showed no bias for photos presented for 500 ms (Noel et al., 2006). For comparison, on alcohol-unrelated perceptual tasks, participants typically require 50 ms to shift attention to a visual cue (Duncan, Ward, & Shapiro, 1994), while requiring 150 ms to disengage attention from one cue and shift it to another location in space (Theeuwes, 2005). Hence, alcohol
attentional biases may be measured during maintenance/disengagement of attention as well as during initial orienting processes.

Alcohol attentional bias is robustly and positively correlated with craving (Field, Mogg, & Bradley, 2005; Field, Mogg, Zetteler, & Bradley, 2004; Field & Powell, 2007). The relation between alcohol attentional biases and subjective craving may be causal; persons trained to attend to alcohol cues for 500 ms with a modified visual probe task experienced increased cravings and consumed significantly more beer compared to persons trained to attend to neutral stimuli (Field & Eastwood, 2005). Thus, it appears to be attentional hold rather than attentional orienting to alcohol cues that seems to influence alcohol seeking behaviors in heavy drinkers. Although the processes by which addiction-related attentional biases influence alcohol dependence have not been adequately detailed, it is evident that subjective urges to drink and drinking behavior itself are modulated by attention. Among persons who drink alcohol to cope, stress intensifies alcohol attentional bias and concomitant experience of craving (Field & Powell, 2007).

Across several studies, attentional bias appears to be proportional to the frequency and quantity of alcohol consumed by drinkers (Field & Cox, 2008). Additionally, alcohol attentional bias as measured by the addiction Stroop task predicted relapse in alcohol abusers (Cox, Hogan, Kristian, & Race, 2002) and alcohol consumption at a 6-month follow-up (Cox, Pothos, & Hosier, 2007). Due to ambiguity in interpreting results from the addiction Stroop task, it is unclear how to account for this predictive relationship. Nevertheless, whether through the invocation of automatic, conditioned responses, preferential attending to alcohol cues, or diversion of cognitive resources away from
maintenance of normal daily activity and thought processes, alcohol-related attentional biases may foster addictive behavior and impede recovery in alcohol dependent persons.

**Craving results in dysphoria and autonomic arousal.** There appears to be a positive feedback loop between alcohol attentional biases and the experience of craving, such that preferential attending to alcohol cues drives craving, which then magnifies the attentional bias (Field & Powell, 2007). Craving itself is a multiplex phenomenon, involving cognitive processes, negative affect, neurobiological circuits involved in withdrawal and reward, contextual learning, and socially-driven alcohol expectancies. Theorists debate whether craving is the subjective correlate of classically conditioned alcohol withdrawal (A. M. Ludwig & Wikler, 1974), the cognitive interpretation of alcohol cue-related physiological arousal (Melchior & Tabakoff, 1984), the expectation or anticipation of the rewarding effects of alcohol (Marlatt, 1985), or the cognitive, affective, and physiological reactivity resulting from impeded automatized alcohol-use sequences (Tiffany, 1990). According to Tiffany (1999), alcohol dependent persons in recovery experience craving when they attempt to block or inhibit an automatic impulse to consume alcohol triggered by external (e.g., the sight of one’s favorite drink) or internal (e.g., an emotional state) cues.

Given that prefrontal cortical areas such as the orbitofrontal cortex and anterior cingulate are implicated in cognitive control and volitional inhibition of urge impulses (Knoch & Fehr, 2007), cognitive processing models of craving (Tiffany & Conklin, 2000) would predict increased activity in these brain regions when alcohol dependent individuals attempt to abstain from drinking in the face of alcohol cues. In fact, craving has been associated with increased activity in orbitofrontal cortex (Dom et al., 2005;
Kalivas & Volkow, 2005; Risinger et al., 2005), while the attempt to inhibit addictive urges has also been shown to evoke anterior cingulate activity (Brody et al., 2007). This finding supports the notion that heightened activation of prefrontal cortex in abstinent alcohol dependent persons during exposure to alcohol-related stimuli (Park et al., 2007) may stem in part from inhibition of craving-related neural activity in the ventral striatum (Heinz et al., 2004). Craving also correlates with metabolic increases in dorsolateral prefrontal cortex and the amygdala, suggesting that addictive urges are subserved by activation of integrated cognitive-emotional memory circuits (S. Grant et al., 1996). A cascade of autonomic responses co-occur with subjective craving, including decreased heart-rate variability and increased salivation (Ingjaldsson et al., 2003), as well increased blood pressure and salivary cortisol levels (Fox, Bergquist, Hong, & Sinha, 2007).

Nearly two decades ago, Tiffany (1990) astutely recognized the inherent difficulty in teasing apart physiological correlates associated with activation of automatized alcohol use schema from those associated with withdrawal and the nonautomatic cognitive processes concomitant with craving; this constellation of physiological responses is a relatively-undifferentiated aggregate of generalized autonomic arousal that co-occurs with the dysphoria of craving.

Suppressing the urge to drink intensifies craving and alcohol-related cognitions. In response to the disturbing thoughts and feelings that accompany craving, alcohol dependent persons in recovery may attempt to suppress the urge to drink as a expression of “willpower” (Bateson, 1971). Unwittingly, such efforts may only serve to enhance the availability of alcohol-related cognitions and affective reactions to consciousness, as a body of research indicates that attempted suppression often results in an increased rate of
the very thoughts and moods it is directed against, as well as heightened psychophysiological reactivity (Wegner, Schneider, Carter, & White, 1987; Wegner & Zanakos, 1994; Wenzlaff & Wegner, 2000). Heavy drinkers exhibited faster reaction times to alcohol expectancy items than control phrases after having been asked to suppress drinking urges subsequent to visual and olfactory alcohol cue-exposure (Palfai, Monti, Colby, & Rohsenow, 1997). Among alcoholics presented with an imaginal alcohol exposure script, thought suppression was inversely associated with tonic heart rate variability, indicative of impaired inhibitory control of perseverative cognition (Ingjaldsson, Laberg, & Thayer, 2003). Hence, it appears as if the attempt to suppress the urge to drink paradoxically increases the autonomic arousal and intrusive alcohol-related cognitions characteristic of craving, resulting in magnified distress and enhanced drive to drink alcohol.

Palliative coping through alcohol consumption is negatively reinforcing. For negative affect subtypes of alcohol dependence, alcohol consumption may be an attempt to allay the autonomic distress and negative affective states that co-occur with stress and craving. In this light, alcohol use is a form of palliative coping (Olff et al., 2005). This alcohol-mediated coping response may be twofold in nature: via anxiolytic depressant effects that reduce sympathetic arousal (Stritzke, Lang, & Patrick, 1996), and via acutely rewarding effects that compensate for dysphoric emotions via endogenous opioid release and dopamine release (Kalivas & Volkow, 2005). Although the opioid and dopaminergic effects of alcohol consumption are positively reinforcing, alcohol’s psychopharmacological reduction of negative affect is theorized to exert negative reinforcement conditioning (Baker et al., 2004). Through both forms of reinforcement,
alcohol consumption may become a conditioned response to endogenously-generated negative affect and exogenously encountered stressor stimuli.

When the addictive process is established, withdrawal from alcohol generates negative emotions and physiological distress, motivating the addict to imbibe more alcohol to relieve the discomfort. Ultimately, the negative reinforcement obtained from such palliative coping efforts augments associative networks between stress, perseverative cognition, negative affect, and alcohol use action schemata, such that when reactivated by subsequent stressors, these cognitive-affective stimulus configurations initiate and guide ensembles of automatized alcohol consumption behaviors. This pattern drives relapse into a self-destructive, downward spiral fueled by an increasing sensitivity and vulnerability to stressful life events.

Conclusion

Stress appraisals coupled with perceived lack of problem-solving resources result in neurophysiological arousal, perseverative cognition, and negative affect. This reactivity may in turn trigger automatized schemata to deploy sequences of maladaptive cognitive-behavioral processes, including attentional biases towards affectively-charged stimuli, the urge to alleviate distress, and palliative coping attempts to avoid the stressor or allay its impact through impulsive behavior. When palliative coping relieves distress, it results in negative reinforcement conditioning that perpetuates the cycle by further sensitizing the system to future stressful encounters.

Hypothetically, this stress-initiated risk chain may undergird multiple forms of psychopathological self-regulation failure, ranging from the various forms of alcohol and drug dependence to sex and gambling addiction, obsessive-compulsive disorder, eating
disorders, and mood disorders. These diverse conditions appear to share a common structure of stress-precipitated, automatic allostasis, where dysfunctional attempts to self-regulate in response to stressors perpetuate a system of runaway positive feedback loops that result in continued dysregulation. Whether an individual becomes adapted to exogenously obtained chemicals or those generated within the nervous system during a particular mood, in either case that individual acclimates to a particular state of the *milieu interior*, and thereby becomes entrenched in a self-perpetuating cycle.

Thus, stress-precipitated addiction is a form of adaptation, a learning process whereby a number of interlocking variables are maximized, resulting in runaway growth of the system and the disruption of homeostasis. The essential systemic dependency of organism and environment, involving the organismic relationship to food, water, air, and other basic units of survival, is disrupted via the acquisition of a new dependency (i.e., the dependence on drugs or alcohol). This acquisition involves a systemic change, an acclimation of the system to a new functional state. However, in the escalating symmetrical process of stress-precipitated addiction, the adaptation of the system does not solve but instead exacerbates the perceived problem, resulting in a positive feedback loop of increased addictive behavior, leading to a runaway state. The intake of the psychoactive substance “resets the bias - changes the structure,” leading to a recalibration of the system into an allostatic mode (Bateson & Bateson, 1987, p. 314).

In this sense, addiction may be seen as a self-organizing system, operating to maximize and maintain its own organization without assistance from an external regulator (Bickel & Potenza, 2006). Like other self-organizing or autopoietic systems, stress-precipitated addiction is a multivariate process whose components (neurobiological
and sociocognitive) dynamically interact to produce and preserve its internal coherence.
Out of this dynamic interaction arises the emergent phenomenon of addiction itself: that is, the self-maintaining, continually recalibrating process relayed in the proverb: “first the man takes a drink, then the drink takes the drink, then the drink takes the man” (Ludwig, 1988). Self-organizing systems maintain homeostasis through overarching negative feedback processes, and hence, only change as a result of perturbation from an outside source (Bateson, 1972; Bickel & Potenza, 2006). If the calamitous social, occupational, and health consequences of “hitting bottom” are of sufficient intensity, they may serve to perturb the otherwise stable, self-perpetuating system of addiction, eventuating in the dissolution of the autopoeitic unity of the addictive system. Similarly, if therapies target critical links of the risk chain, the resultant perturbation of the addictive system may lead to an adaptive reconfiguration. Whether precipitated by hitting bottom or fostered through treatment, the disassembly of behavioral routines, cognitive processes, and physiological responses underpinning addiction may ultimately lead to the shift towards sobriety.

The integrated biopsychosocial model of stress-precipitated alcohol dependence has implications for targeted forms of treatment, guiding the design of interventions that may disrupt the risk chain at multiple points. Therapies can be aimed at initial stress appraisal processes, leading to more accurate perception of situational demands and valid self-efficacy estimations. Clarified appraisals may attenuate subsequent stress reactions, interrupting preservative cognitive processes like catastrophizing and rumination, thereby preventing or lessening stress-precipitated alcohol consumption. Similarly, interventions could reduce the psychophysiological arousal and negative affective states triggered by
stress appraisal. By inducing a parasympathetic “relaxation response,” stress-precipitated activation of the nervous system can be countered, preventing elicitation of downstream addictive processes. Emotion regulation through affect labeling, attentional refocusing, cognitive reappraisal, or metacognitive decentering from affectively-charged stimulus evaluations can attenuate the influence of emotionally-distressing stimuli on biobehavioral responses (Ochsner & Gross, 2005, 2008). These forms of self-regulatory cognitive control mechanisms can be developed over time as a means of coping with distress (Posner & Rothbart, 1998), and appear to be subserved by the interaction of prefrontal cortical structures (e.g., anterior cingulate cortex, dorsolateral and medial PFC) with the amygdala and insula (Levesque et al., 2003; Ochsner, Bunge, Gross, & Gabrieli, 2002; Ochsner et al., 2004). Thus, interventions that promote clarification of appraisals, disrupt perseverative cognition, and facilitate emotion regulation may prevent stressful encounters from precipitating or exacerbating the consumption of alcohol.

In addition, interventions could target alcohol use action schema and the ensembles of maladaptive cognitive-behavior processes that lead to addictive consumption of alcohol. Therapies may increase attention to drinking triggers and the presence of urges, enabling a skillful deployment of coping strategies. If, as Rohsenow and colleagues (1994) observed, inattention to alcohol cues is correlated with increased drinking behaviors, then alcohol consumption may be decreased by enhancing attention to alcohol dependence triggers. Concomitantly, interventions could enable awareness of the engagement of alcohol use action schema when triggered by alcohol cues or negative affect, thereby allowing for the disruption of automatized drinking processes with a controlled coping response. Abstaining from use of alcohol requires the deployment of
cognitive control mechanisms in stressful situations where affect regulation is needed (Wiers et al., 2006). Psychosocial interventions might strengthen top-down cognitive control, thereby facilitating inhibition of alcohol use urges in the face of stress triggers. Additionally, stress-precipitated engagement of alcohol use action plans may also be interrupted by disengagement of attention from alcohol-relevant cues to allow for focus on neutral or health-promoting stimuli. Ultimately, repetitively engaging, disengaging, and moving attention away from alcohol-use triggers (including interoceptive data stemming from affective responses) toward innocuous or beneficial stimuli may weaken associative networks of alcohol use action schema. Lastly, treatment may help the alcohol dependent person to learn how to tolerate alcohol-related cognitions and craving without engaging in thought suppression. In so doing, alcohol cue-exposure may occur without the added burden of the post-suppression rebound effect, leading to the eventual extinction of conditioned appetitive responses.

Although a number of cognitive-behavioral therapies might leverage some of the aforementioned therapeutic mechanisms, one such intervention, mindfulness training, holds promise for being especially well-suited as a treatment for stress-precipitated alcohol dependence. Mindfulness-based interventions have recently gained prominence in the psychological and medical literatures. Mindfulness involves self-regulation of a metacognitive form of attention: a nonreactive, non-evaluative monitoring of moment-by-moment cognition, emotion, perception, and physiological state without fixation on thoughts of past and future (Lutz, Slagter, Dunne, & Davidson, 2008). A growing body of research suggests that mindfulness impacts stress, implicit cognition, and attentional processes (Brefczynski-Lewis, Lutz, Schaefer, Levinson, & Davidson, 2007; Jha,
Mindfulness training, which originates from Buddhist traditions but has been co-opted by and translated for secular, Western clinicians, has been shown to exert significant, salutary effects on stress-related, biobehavioral conditions (for reviews, see Baer & Krietemeyer, 2006; Grossman, 2004; Ludwig & Kabat-Zinn, 2008). Research suggests that mindfulness-based treatments may improve clinical outcomes in substance-abusing populations. Bowen et al. (2006) employed a nonrandomized pre-test post-test comparison group design to test the efficacy of a mindfulness meditation intervention for adults incarcerated within a minimum security correctional facility and found that mindfulness reduced substance use and substance-related problems to a greater extent than treatment as usual (TAU). Several other small pilot studies have examined the effects of mindfulness meditation on substance use disorders, including one which identified post-intervention attenuation of physiological stress (Marcus et al., 2003), and another which found significant decreases in heavy drinking days, stress, anxiety, depression, and the cytokine interleukin-6, a stress-responsive biomarker predictive of poor health outcomes (Zgierska et al., 2008). Although the four studies of mindfulness-based treatments reviewed above suggest that the intervention may ameliorate factors related to stress-induced maintenance and relapse to alcohol dependence, the lack of random assignment presents serious threats to the validity of findings. Well-controlled studies are needed to strengthen the evidence base for this nascent therapy.
Hypothetically, mindfulness training may prevent stress-precipitated alcohol dependence relapse through a number of means, as delineated below. Mindfulness has been conceptualized as an awareness of stimuli without distortions and reactivity related to emotional valence (Brown, Ryan, & Creswell, 2007); hence, mindfulness training may increase the accuracy of primary and secondary stress appraisals (Garland, 2007) as well as facilitate cognitive reappraisal (Garland, Gaylord, & Park, 2009), attenuating exaggerated stress reactions stemming from perseverative cognitive processes such as catastrophizing or rumination. Given that mindfulness training has been shown to decrease negative affective reactions (e.g., Evans et al., 2007; Ma & Teasdale, 2004; Teasdale et al., 2002), such improved emotion regulation may reduce the risk of stress-induced relapse. Second, mindfulness training may disrupt alcohol use action schema by increasing awareness of the presence of urges, enabling a skillful deployment of coping strategies. Mindfulness training may alert the individual to the engagement of alcohol use action schema when triggered by alcohol cues or negative affect, thereby disrupting automatized drinking processes with controlled cognitive operations. Third, mindfulness may facilitate exposure to alcohol-related cognitions and cravings without being subject to the paradoxical effects of urge suppression. Mindful exposure to alcohol cue-reactivity may prevent post-suppression rebound effects on the accessibility of alcohol-related thoughts (Palfai et al., 1997). Indeed, changes in thought suppression have been shown to partially mediate the effects of mindfulness training on alcohol use and drinking consequences (Bowen, Witkiewitz, Dillworth, & Marlatt, 2007). Fourth, because mindfulness training has been shown to potentiate attentional orienting functions (e.g., Jha, Krompinger, & Baime, 2007), mindfulness-based interventions may facilitate
disengagement of attention from alcohol-relevant cues, weakening alcohol attentional biases to allow for focus on neutral or health-promoting stimuli.

The stress reaction and its addictive consequences, then, are not eventualities, for with sufficient intervention and training threat appraisals can give way to reappraisals of self-efficacy, acceptance, or a sense of coherence even in the face of grave adversity. The encounter with the stressor can be met with a sense of resourcefulness or with an attitude of benefit-finding, and in so doing, what would have otherwise been perceived as threatening becomes a navigable and meaningful challenge. Through the generative cognitive process of re-attributing the meaning of ambiguous stimuli, the individual can attend to constructions of reality wherein they have the wherewithal to adapt to and solve the problems in their lives. Surely, it is this ability that is articulated in the addict’s supplication for “the serenity to accept the things I cannot change, to change the things I can, and the wisdom to know the difference.” The person recovering from alcohol dependence cannot avoid the ubiquity of stressors, but may be able to use problem-focused coping to manage them. Where problem-solving approaches fall short, emotion-focused strategies such as reappraisal and decentering can be employed, attenuating the affective reaction to the stressor. For the seriously and chronically dependent individual, attentional biases and cravings may automatically arise during the stress response, driving the impulse to relieve distress with alcohol. But herein may lay the power of volition, not in repressing the urge to drink, but in mindfully observing the addictive impulse as it arises, abides, and ceases without triggering alcohol consumption. In so doing, each moment of unanswered craving in the face of a stressor becomes an instance of extinction
learning that can break the chains of risk and ultimately fuel the recovery process with a sense of empowerment.
References


MINDFULNESS IS INVERSELY ASSOCIATED WITH ALCOHOL ATTENTIONAL BIAS AMONG RECOVERING ALCOHOL-DEPENDENT ADULTS

ABSTRACT

Although mindfulness training is a promising intervention for alcohol dependence, its therapeutic mechanisms are at present unspecified. Because mindfulness training may facilitate attentional disengagement (Jha, Krompinger, & Baime, 2007), we hypothesized that mindfulness would be inversely associated with attentional bias toward visual alcohol cues. Fifty-four alcohol-dependent adults (mean age = 39.2, SD = 9.2) residing in a therapeutic community (TC) located in the southeastern U.S. completed self-report questionnaires and a cognitive task. A majority of participants were male (85.2%), African American (53.7%), and had earned < $20,000 in the year before entering the TC (55.6%). The mean number of lifetime DSM-IV alcohol dependence criteria met by participants was 6.5 (SD = 1.0), and the mean number of standard alcoholic drinks consumed per day in the year before entering treatment was 18.8 (SD = 10.9). Alcohol attentional bias was assessed with a dot probe task. Questionnaires were used to evaluate self-reported mindfulness, craving, and stress. Recovering alcohol-dependent individuals high in mindfulness exhibited less alcohol attentional bias (AB), stress and craving than their counterparts low in mindfulness. Multiple linear regression analysis indicated that mindfulness was more predictive of alcohol AB than either stress or craving. Among recovering alcohol-dependent adults mindfulness is associated with alcohol AB.
Alcohol dependence involves implicit cognitive processes (Wiers et al., 2006) and attentional biases (AB) toward alcohol-relevant stimuli (Field and Cox, 2008) that drive appetitive states and compulsive drinking. Recurrent alcohol misuse is thought to establish schemata that compel alcohol consumption through automatized sequences of context-dependent behavior (Tiffany, 1990). Engagement of alcohol use action schemata may result in implicit processing of salient stimuli, manifested as an involuntary AB towards alcohol cues. Such bias is evident in visual probe tasks, in which heavy drinkers preferentially attend to alcohol cues, resulting in decreased reaction times (RTs) to probes replacing alcohol photographs compared to probes replacing neutral photographs (e.g., Field et al., 2004). Alcohol AB has been linked to subjective craving; persons experimentally manipulated to attend to alcohol stimuli experience increased cravings and consume more beer than persons trained to attend to neutral stimuli (Field and Eastwood, 2005). Although the causal relationship between AB and alcohol consumption is unknown, data suggest that drinking urges and behaviors can be modulated by attention. Among persons who drink to cope with negative affect, stress increases alcohol AB and craving (Field and Powell, 2007). Whether through invocation of automatic, appetitive responses or displacement of cognitive resources, alcohol AB may foster and/or maintain alcohol dependence, impeding recovery in alcohol dependent persons.

Given that automaticity and AB may be integral components of alcohol dependence, interventions affecting attention and implicit cognition may hold promise for its treatment. One such intervention, mindfulness meditation, has in recent years gained prominence for its apparent efficacy in treating stress-related, biobehavioral conditions (Ludwig and Kabat-Zinn, 2008). Mindfulness involves self-regulation of a metacognitive
form of attention: a nonreactive, non-evaluative monitoring of moment-by-moment cognition, emotion, and physiological state without fixation on thoughts of past and future (Lutz et al., 2008) that negatively correlates with factors such as thought suppression, alexithymia, and emotion dysregulation (Baer et al., 2006). Although there are significant individual differences in dispositional mindfulness, this metacognitive faculty can be fostered by training (Carmody and Baer, 2008). Recent research suggests that mindfulness-based treatments may improve clinical outcomes in substance-misusing populations (e.g., Bowen et al., 2006). While there is evidence that the salutary effect of mindfulness training on substance use is partially mediated by reductions in thought suppression (Bowen et al., 2007), it is plausible that this effect may also be mediated by the influence of mindfulness on attentional processes implicated in addiction.

At present, the processes by which mindfulness may promote recovery from alcohol dependence are unknown. Thus, we examined relationships between mindfulness, alcohol AB, stress, craving, and thought suppression among alcohol-dependent adults residing in a therapeutic community. Because mindfulness training may facilitate purposive disengagement of attention on attentional tasks (Jha et al., 2007; Slagter et al., 2007; Tang et al., 2007), we hypothesized that individuals with higher levels of mindfulness would evidence less AB toward visual alcohol cues relative to individuals with lower mindfulness.

Methods

Sample characteristics and study design

Study participants were 54 alcohol-dependent adults who had resided for at least 18 months (M = 21.9, SD = 2.5, range = 18 to 28 months) in a therapeutic community.
In this program, after 18 months individuals obtain employment outside of the TC and thus become at higher risk for relapse due to increased access to alcohol. Potential study participants were administered the Alcohol Use Disorders Identification Test (AUDIT) and completed a semi-structured diagnostic interview conducted by a Master’s-level social worker to ensure that all participants met lifetime DSM-IV criteria for alcohol dependence. A majority of participants were men (85.2%); 53.7% were African American and 40.7% were Caucasian. With regard to income in the year before entering treatment, 55.6% had earned < $20,000, and 29.6% had earned $20,000-$40,000. The mean age of the sample was 39.2 (SD = 9.2). The mean number of DSM-IV alcohol dependence criteria met by participants was 6.5 (SD = 1.0), and the mean total AUDIT score for the sample was 33.3 (SD = 4.9). The mean number of standard alcoholic drinks consumed per day in the year before entering treatment was 18.8 (SD = 10.9). All participants reported having continuously abstained from use of psychoactive substances during their residence in the TC. Reports of abstinence were corroborated by random urinalyses conducted at the TC on an as needed basis, as well as through daily observation by program staff. During a single, hour-long assessment period conducted on premises at the TC, study participants first completed several standardized psychosocial instruments, including the *Five Facet Mindfulness Questionnaire* (Baer et al., 2006), *Perceived Stress Scale* (Cohen et al., 1983), and *Penn Alcohol Craving Scale* (Flannery et al., 1999). Next, participants were engaged in a computer-based dot probe task (MacLeod, et al, 1986) as a measure of alcohol AB. All measures were administered in the same order across participants in a single session. This measurement protocol was part of a baseline assessment of a randomized controlled pilot trial of a mindfulness-
oriented treatment for alcohol dependence. After this baseline assessment, participants were randomly assigned to either a mindfulness-promoting treatment or social support group for alcohol dependence. The results of this clinical intervention study are reported in Chapter III.

Table 1. Sample Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>M or %</th>
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<tbody>
<tr>
<td>Length of stay in residential program (M, SD)</td>
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<tr>
<td>Gender N (%)</td>
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<td>Female</td>
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<td>Race N (%)</td>
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<td>Age (M, SD)</td>
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</tr>
<tr>
<td>DSM-IV Dependence Criteria (M, SD)</td>
<td>6.5 (1.0)</td>
</tr>
<tr>
<td>Income before entering TC N (%)</td>
<td></td>
</tr>
<tr>
<td>&lt;$20,000</td>
<td>30 (55.6)</td>
</tr>
<tr>
<td>$20,000-40,000</td>
<td>16 (29.6)</td>
</tr>
<tr>
<td>$41,000-60,000</td>
<td>5 (9.3)</td>
</tr>
<tr>
<td>$61,000-80,000</td>
<td>1 (1.9)</td>
</tr>
<tr>
<td>&gt;$80,000</td>
<td>2 (3.7)</td>
</tr>
<tr>
<td>Drinks per day (M, SD)</td>
<td>18.8 (10.9)</td>
</tr>
<tr>
<td>AUDIT (M, SD)</td>
<td>32.3 (4.9)</td>
</tr>
<tr>
<td>DAST (M, SD)</td>
<td>21.4 (4.6)</td>
</tr>
</tbody>
</table>

Measures

Mindfulness. The Five Facet Mindfulness Questionnaire (FFMQ, α = .81), comprised of 39 likert-type items, was used to measure self-reported mindfulness. The FFMQ yields a total score and scores for five internally consistent mindfulness factors each with their own convergent and predictive validity: nonreactivity to inner experience (tapped by items such as “I watch my feelings without getting lost in them”), observing
and attending to experience ("I pay attention to sensations, such as the wind in my hair or the sun on my face"), describing and discriminating emotional experiences ("I’m good at finding words to describe my feelings"), nonjudging of experience ("I tell myself I shouldn’t be feeling the way that I am feeling"), and acting with awareness ("I find myself doing things without paying attention") (Baer, Smith, Hopkins, Krietemeyer, & Toney, 2006).

*Alcohol attentional bias.* A dot probe task (MacLeod, et al, 1986) created in E-Prime 1.2 (PST Inc., Pittsburgh, PA) and presented on an IBM T60 laptop with a 15” screen was used to measure alcohol AB. In each trial, two grayscale images appeared, one on each side of a fixation cross (+); one image was neutral in content, the other was alcohol-related. The pair of images was presented for either 200 ms or 2000 ms. Left/right position of the alcohol images and presentation duration were both randomized and counterbalanced across 20 practice trials and 160 trials. Following a 50 ms inter-stimulus interval (ISI), a target probe (two dots) replaced one of the images and a distracter probe (one dot) replaced the other image; probes appeared for 100 ms. Participants were instructed to fixate on the cross between the images and indicate the location of the target probe by responding with a left or right button press on a keypad. Target probes randomly replaced alcohol and neutral images with equal frequency.

Some parameters of the task employed here vary slightly from tasks used previously to study addiction-related AB. Two notable differences are: (1) the fixation point remains on the screen throughout the duration of each trial, and (2) dots appear in both probe locations, requiring participants to discriminate between the location with one dot and the location with 2 dots. This task design was chosen to eliminate confounding
contributions of automatic, reflexive attention that are not related to the image cues. In particular, both sudden onsets and offsets have been found to capture attention (Hopfinger and Maxwell, 2005), and the sudden offset of the fixation cross, or the sudden onset of a target probe at only one location, could interfere with any spatial shift in attention caused by the pictorial cues, especially in trials with the short (200ms) stimulus onset asynchrony (SOA). More specifically, if the fixation cross were to disappear when the dot probes appeared, attention would likely be drawn to the middle of the screen reducing the power to detect attentional allocation shifts due to the alcohol image cues. Additionally, if a probe appeared in only location, participants’ attention would be reflexively captured by the probe. Including a place marker in the opposite target probe location, requires the participant’s attention to be directed to the spatial location of the target probe and ensures that response selection cannot be based on detection of the probe through peripheral vision. Moreover, use of two probes requires greater attentional resources than detection of a single probe and thus this design may have more power to resolve attentional shifts elicited by alcohol cues.

Alcohol stimuli included 13 photographs of alcoholic drinks (i.e., liquor, beer, etc), as well as 7 photos of persons drinking alcohol. Neutral stimuli included 13 photos of kitchen items and 7 photos of persons in kitchen scenes. Stimulus sets were analyzed with respect to their spatial frequency content to ensure that they did not differ in terms of basic visual properties, which could elicit reflexive attentional capture irrespective of image content. On measures of spectral peak (Neutral: 0.0180, Alcohol: 0.0176, $t_{(38)}=0.383, p=0.704$) and spectral width (Neutral: 59.20, Alcohol: 59.29, $t_{(38)}=-0.027, p=0.979$), the two stimulus sets were not significantly different.
Alcohol craving and perceived stress. Subjective alcohol craving was assessed with Penn Alcohol Craving Scale (PACS, $\alpha = .91$) (Flannery et al., 2001). Participants use a 7-point scale to indicate craving frequency and intensity over the past week on items like “How often have you thought about drinking or about how good a drink would make you feel?” and “At its most severe point, how strong was your craving?” The 10-item Perceived Stress Scale (PSS-10, $\alpha = .85$) was used to assess on a 5-point scale how often (0 = never, 4 = very often) in the past month participants found their lives unpredictable, uncontrollable, and overwhelming (Cohen, Kamarck, & Mermelstein, 1983), and includes items such as “How often have you felt nervous and ‘stressed’?” and “How often have you felt that you were on top of things?”

Data analysis

For AB data, trials with extreme RTs, defined as those with RTs longer than $+3$ SD above the individual mean (c.f., Field et al., 2004), were discarded as outliers (mean = 2.5±1.5 per participant); error trials were also discarded. For each participant, AB scores were calculated by subtracting their mean RT to target probes replacing alcohol photos from their mean RT to target probes replacing neutral photos, such that positive bias scores indicate an AB toward visual alcohol cues. All data are reported as means ± SD unless otherwise noted.

Bivariate correlations, t-tests, and multiple linear regression analyses were performed with SPSS 16.0. Multiple linear regression was used for hypothesis testing, in which age, gender, number of DSM-IV alcohol dependence criteria endorsed, mindfulness, craving, and perceived stress were entered simultaneously, to examine the percentage of variance in alcohol AB explained by these variables. Potential
multicollinearity issues were screened by examining the variance inflation factor (VIF) of each variable. There were no variables with VIF > 1.5.

Results

Dot probe task

Mean accuracy on the dot probe task was 97.2% ± 0.4%. Mean RT to target probes replacing alcohol photos presented for 2000 ms was 587.7 ± 120.8 ms, whereas mean RT for neutral photos was 585.3 ± 123.9 ms. Mean RT to target probes replacing alcohol photos presented for 200 ms was 588.10 ± 119.5 ms, whereas mean RT for neutral photos was 584.73 ± 121.9 ms. Paired t-tests revealed nonsignificant differences between RTs to alcohol and neutral photos for each SOA: for 200 ms, \( t(53) = 1.45, p = .15 \), and for 2000 ms, \( t(53) = .96, p = .34 \).

Despite that both 2000 and 200 ms AB data were approximately normally distributed (both yielding non-significant Kolmogorov-Smirnov tests for normality), approximately one-half of participants exhibited 2000 and 200 ms AB towards probes replacing alcohol photos while the other half exhibited AB towards probes replacing neutral photos. Given that AB towards alcohol cues is theorized to represent appetitive conditioning (Field & Cox, 2008), whereas AB away from alcohol cues is thought to represent disengagement from or avoidance of the alcohol (Townshend & Duka, 2007), we analyzed data from participants with these two apparently distinct types of AB separately. Thus, for each presentation duration (2000 ms and 200 ms) we divided participants into two groups: those with an AB > 0 (i.e., the alcohol approach group) and those with an AB < 0 (i.e., the alcohol disengagement group). Mean 2000 ms AB for the approach group (\( N = 24 \)) was = 13.1 ± 8.9 ms, whereas the mean 2000 ms AB for the
disengagement group (N = 30) was -14.8 ± 13.3 ms. Mean 200 ms AB for the approach group (N = 25) was 10.9 ± 8.4 ms, whereas the mean 200 ms AB for the disengagement group (N = 29) was -15.8 ± 12.1 ms.

**Bivariate relationships between mindfulness, AB, craving, and stress**

Correlation coefficients for variables of interest are reported in Table 1. Mindfulness was significantly negatively associated with 2000 ms alcohol AB, subjective alcohol craving, perceived stress, and thought suppression. *Nonreactivity to inner experience* and *describing experience with words* were the only mindfulness factors significantly inversely correlated with 2000 ms alcohol AB. With regard to 200 ms AB, although overall mindfulness was uncorrelated with this variable, the mindfulness subscale of *nonjudging of experience* was significantly negatively correlated with this attentional bias for short (200 ms) duration alcohol stimuli.
Table 2: Bivariate Correlations Between Mindfulness, Craving, Stress, Thought Suppression, and Attentional Bias in Abstinent Alcohol Dependent Adults (N = 54)

<table>
<thead>
<tr>
<th></th>
<th>FMQ</th>
<th>NONR</th>
<th>OBS</th>
<th>DES</th>
<th>NONJ</th>
<th>ACT</th>
<th>AB200</th>
<th>AB2000</th>
<th>ACS</th>
<th>PSS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total mindfulness (FMQ)</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonreactivity to inner experience (NONR)</td>
<td>.46***</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observing/attending to experience (OBS)</td>
<td>.61***</td>
<td>.29*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Describing experience with words (DES)</td>
<td>.76***</td>
<td>.31*</td>
<td>.34*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonjudging of experience (NONJ)</td>
<td>.38**</td>
<td>.03</td>
<td>.13</td>
<td>-.06</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acting with awareness (ACT)</td>
<td>.67***</td>
<td>-.10</td>
<td>.03</td>
<td>.48***</td>
<td>.17</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol attentional bias 200 ms (AB200)</td>
<td>.13</td>
<td>.15</td>
<td>.14</td>
<td>-.04</td>
<td>-.27*</td>
<td>-.02</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol attentional bias 2000 ms (AB2000)</td>
<td>-.39**</td>
<td>-.27*</td>
<td>-.20</td>
<td>-.37**</td>
<td>-.07</td>
<td>-.26</td>
<td>.02</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol craving scale (ACS)</td>
<td>-.38**</td>
<td>-.28*</td>
<td>-.49***</td>
<td>-.32*</td>
<td>.12</td>
<td>-.18</td>
<td>-.17</td>
<td>.13</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Perceived stress scale (PSS)</td>
<td>-.37**</td>
<td>-.41**</td>
<td>-.19</td>
<td>-.27</td>
<td>-.13</td>
<td>-.19</td>
<td>-.15</td>
<td>.02</td>
<td>.17</td>
<td>1</td>
</tr>
</tbody>
</table>

*p < .05  **p < .01  ***p ≤ .001
Relationship between mindfulness and alcohol approach/disengagement bias

Given the set of observed significant correlations, the dichotomized 2000 ms attentional bias variable (approach and avoidance) was used to predict variation in mindfulness. One-way ANOVA showed that participants with 2000 ms alcohol approach biases had significantly lower mindfulness, $F(1, 52) = 5.09, p<.05$, than those with disengagement biases.

Multivariate analysis of alcohol AB

When age, sex, number of DSM-IV alcohol dependence criteria, mindfulness, craving, and perceived stress were entered simultaneously into a multiple linear regression model predicting 2000 ms alcohol AB, mindfulness was the sole, statistically significant predictor, indicating that mindfulness scores accounted for a significant portion of variation in alcohol AB [$\beta = -.37, p = .029$] after controlling for the influence of the demographic and clinically-relevant variables listed above. Despite that the relationship between mindfulness and 2000 ms alcohol AB was statistically significant, the model only explained 14% of the variance in alcohol AB. Regression parameters are reported in Table 3.
Table 3: Summary of Simultaneous-Entry Multiple Linear Regression Analysis for Variables Predicting 2000 ms Alcohol AB Among Alcohol Dependent Patients (N = 54)

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-.07</td>
<td>.26</td>
<td>-.04</td>
</tr>
<tr>
<td>Sex</td>
<td>-1.36</td>
<td>6.63</td>
<td>-.03</td>
</tr>
<tr>
<td># DSM-IV dependence criteria</td>
<td>1.91</td>
<td>2.50</td>
<td>.10</td>
</tr>
<tr>
<td>Mindfulness</td>
<td>-.43</td>
<td>.19</td>
<td>-.37*</td>
</tr>
<tr>
<td>Craving</td>
<td>-.13</td>
<td>.51</td>
<td>-.04</td>
</tr>
<tr>
<td>Perceived stress</td>
<td>-.18</td>
<td>.42</td>
<td>-.07</td>
</tr>
</tbody>
</table>

Note. $R^2 = .14$. * $p < .05$.

Discussion

Mindfulness was significantly inversely correlated with alcohol AB, perceived stress, and subjective alcohol craving among recovering alcohol dependent adults. Mindfulness was the sole significant predictor of alcohol AB after controlling for the influence of stress and craving, variables which have been linked with alcohol AB in prior studies (Field & Powell, 2007). To our knowledge, this is the first report identifying significant associations between self-reported mindfulness and alcohol dependence-related factors.

Among the present sample of alcohol dependent adults in a long-term TC, mindfulness was more closely related to alcohol AB than measures of stress or craving. Indeed, low mindfulness may reflect enhanced risk for addictive urges, automatic appetitive responses, and/or attentional fixation onto alcohol cues. Persons in recovery
with low levels of mindfulness may be especially susceptible to relapse. Of course, given the modest r-squared of our model, mindfulness only accounts for a portion of the variance in alcohol AB among alcohol dependent individuals, and other important factors that were omitted by the present investigation may contribute to attentional fixation on alcohol cues.

Because mindfulness was inversely correlated with attentional bias towards visual alcohol cues presented for 2000 ms but not for 200 ms, it appears as if mindfulness relates to attentional hold or maintenance of attention rather than initial orienting processes. It may be that relative to those who are low in mindfulness, recovering alcohol dependent persons reporting high levels of mindfulness are less fixated on alcohol cues or perhaps are better able to disengage and shift attention away from alcohol cues. Given that mindfulness training appears to potentiate attentional re-orienting functions (Jha et al., 2007), mindfulness-promoting interventions may facilitate disengagement of attention from alcohol to allow for a focus on neutral or health-promoting objects, persons, and experiences. Ultimately, repetitively engaging, disengaging, and moving attention away from alcohol cues toward innocuous or beneficial stimuli may weaken associative networks of alcohol use action schemata and strengthen an alcohol dependent person’s ability to avoid relapse. However, it should be noted that the present study is correlational in nature, and thus the identified relationship between mindfulness and alcohol AB should not be taken as evidence in support of a potential effect of mindfulness training on addiction-related attentional processes.

Among mindfulness factors, nonreactivity to internal experience and describing/differentiating cognitive-emotional experience appear to be related to
decreased alcohol approach bias. These findings are interesting in light of the relationship between emotion dysregulation and alcohol dependence. A large corpus of research has associated stress reactivity, negative affect, and the appetitive drive to consume alcohol. Additionally, alexithymia has predicted poor outcomes among alcohol-misusing inpatients (Loas, Fremaux, Otmani, Lecercle, & Delahousse, 1997). The observed inverse relationship between mindfulness facets and alcohol AB may reflect self-regulatory competence among recovering alcohol dependent persons reporting high levels of mindfulness. Thus, mindfulness may reduce attentional fixation on alcohol cues by promoting awareness of and detachment from experiences of negative affect or addictive urges.

Although overall mindfulness was uncorrelated with 200 ms AB, the mindfulness facet of nonjudging was significantly associated with this shorter duration AB, suggesting that recovering alcohol dependent adults who experience greater attentional capture by alcohol cues also tend to be less judgmental of their thoughts and feelings. It is possible that persons with strong, automatic appetitive responses towards alcohol may be unaware of such responses, and therefore do not judge or attempt to inhibit these responses. Given the fact that alcohol pictures presented for 200 ms elicit implicit attentional biases, they may represent an unconscious desire to approach alcohol that is typically inaccessible to conscious self-regulatory efforts. As a contrasting explanation, the tendency to negatively judge alcohol-related cognitions and cravings may lead to aversive implicit alcohol associations, resulting in decreased appetitive reactions towards alcohol among persons in recovery from addiction. Because the correlational nature of the data precludes causal inference, experimental research is needed to clarify these relationships further.
Despite reports that heavy drinkers have alcohol approach biases on visual probe tasks (cf., Field, 2006), research has identified attentional disengagement from alcohol cues among abstinent alcohol dependent persons in recovery (Stormark, Field, Hugdahl, & Horowitz, 1997; Townshend and Duka, 2007). In contrast, Noel et al. (2006) found an absence of AB in alcohol dependent inpatients at 1,250 ms stimulus durations but an AB for alcohol pictures presented for 50 ms. Although the mean 2000 and 200 ms AB for our sample was not statistically significantly different than zero, closer inspection of individual differences revealed that the sample was comprised of persons with significant alcohol approach and alcohol disengagement biases. This finding is understandable given that one might expect significant heterogeneity of responses to long-term participation in a TC; many residents might successfully learn to avoid temptation by directing their attention away from alcohol while others might still harbor covert urges to drink indexed by alcohol approach AB. Alternatively, the lack of a significant mean AB in this study may stem from our use of a modified dot probe task using a stationary fixation cross, which might decrease visual scanning of the pictures and thereby limit task sensitivity. A third explanation for the lack of significant mean alcohol AB across the sample may be due to the relatively unusual nature of the sample itself. Study participants were recruited from a long-term TC, and thus on the whole alcohol AB may have been extinguished after an average of nearly 22 months of residing in a therapeutic milieu. Certainly, the long length of treatment may have led to higher levels of mindfulness than may be observed in typical alcohol dependent populations. In spite of this caveat, the present study suggests that degree of mindfulness may predict the extent to which the attention of alcohol dependent individuals is biased towards alcohol cues.
In summary, the present data provide novel evidence associating mindfulness and factors linked to onset and maintenance of alcohol dependence. Whether mindfulness training can impact alcohol AB over time remains a question to be explored by future research.
References


MINDFULNESS TRAINING AFFECTS COGNITIVE, EMOTIONAL, AND PHYSIOLOGICAL MECHANISMS IMPLICATED IN ALCOHOL DEPENDENCE: RESULTS OF A RANDOMIZED CONTROLLED PILOT TRIAL

ABSTRACT

The risk chain of stress-precipitated alcohol dependence relapse may be malleable to targeted behavioral therapies. Data is presented from a pilot-test of a mindfulness-oriented recovery enhancement (MORE) intervention designed to disrupt cognitive, affective, and physiological mechanisms implicated in stress-precipitated alcohol dependence. Fifty-three alcohol-dependent adults (mean age = 40.3, SD = 9.4) recruited from a long-term therapeutic community were randomly assigned to either 10 weeks of mindfulness training or an alcohol dependence support group (ASG). A majority of participants were male (79.2%), African American (60.4%), and had earned <$20,000 in the year before entering the TC (52.8%). Repeated measures analyses of variance were used to explore the differential effects of treatment on clinical self-report measures, a psychophysiological cue-reactivity protocol, and a dot-probe task designed to measure alcohol attentional bias (AB). 37 participants completed the interventions. Relative to ASG (n = 18), MORE (n = 18) resulted in medium-large effect size reductions in perceived stress and thought suppression, increased HRV recovery from alcohol cues, and significant effects on alcohol AB. Mindfulness training appears to effect cognitive, affective, and physiological risk mechanisms implicated in alcohol dependence relapse.
Few social ills are as pernicious and persistent as alcohol dependence. Despite more than a century of intervention efforts, 28.4% of persons ever treated for alcohol problems remain dependent on alcohol and 19.1% continue to exhibit alcohol abuse or subclinical dependence symptoms in the past year (Dawson et al., 2005). Clearly, extant interventions are not effective for all alcohol misusers. Persons who drink to cope with stress have significantly higher rates of lifetime and current alcohol dependence symptoms than persons who drink for other reasons (Schroder & Perrine, 2007), and stress is known to increase risk of relapse (Sinha, 2007). Alcohol users have a comparatively high likelihood of experiencing serious life stressors: within the population of adult past-year drinkers, 72.5% reported experiencing at least one stressful life event in the past year, and 23.2% experienced 3 to 5 such stressors (Dawson, Grant, & Ruan, 2005). The experience of stressful life events significantly predicts quantity and frequency of alcohol consumption; for example, drinkers who reported experiencing six or more stressful events in the past year consumed, on average, more than three times the amount of ethanol per day than did alcohol users who denied experiencing any such stressors (Dawson, Grant, & Ruan, 2005). Congruent with such findings, an event-history analysis of 1786 young adults found that both distal and proximal exposure to stressful life events significantly predicted onset of alcohol dependence in a linear and additive fashion after controlling for socioeconomic status and history of psychiatric disorder, implicating a causal role for life stress in the etiology of the disorder (Lloyd & Turner, 2008). Convergent evidence suggests that stress is a common precipitant of alcohol misuse and may play an important role in the pathogenesis of formal alcohol use disorders.
The central risk chain of stress-precipitated alcohol misuse, dependence, and relapse involves cognitive-affective mechanisms that may be explicated by an integrated biopsychosocial framework (Garland, 2009). In brief, stress appraisals coupled with deficits in coping resources result in psychophysiological reactivity, perseverative cognition, and negative affect, which in turn trigger automatized schemata to deploy sequences of maladaptive cognitive-behavioral processes that result in compulsive alcohol consumption in spite of often severe consequences for drinking.

Stress-activated engagement of alcohol use action schemata may result in implicit processing of salient stimuli, manifested as an involuntary attentional bias (AB) towards alcohol cues. Such bias is evident in visual probe tasks, in which heavy drinkers preferentially attend to alcohol cues, resulting in decreased reaction times (RTs) to probes replacing alcohol photographs presented for 500 and 2000 ms compared to probes replacing neutral photographs presented for the same duration (Field, Mogg, Zetteler, & Bradley, 2004). Conversely, although alcohol dependent patients have been shown to exhibit an AB toward alcohol cues presented for 50 ms (Noel et al., 2006), they evidence AB away from alcohol cues presented for 500 ms (Stormark, Field, Hugdahl, & Horowitz, 1997; Townshend & Duka, 2007). Collectively, these findings suggest that alcohol dependent individuals evince treatment-induced attentional disengagement from or avoidance of alcohol cues presented for longer stimulus durations (which allow for conscious mediation of attention), but continue to exhibit implicit appetitive attentional responses for stimuli presented at subliminal durations. Alcohol AB been linked to subjective craving and alcohol consumption; in one study, persons manipulated to attend to alcohol stimuli experienced increased craving and consumed more beer than persons
trained to attend to neutral stimuli (Field & Eastwood, 2005). Although the causal relationship between AB and alcohol consumption is unknown, data suggest that drinking urges and behaviors can be modulated by attention. Among persons who drink to cope with dysphoria, stress enhances alcohol AB and craving (Field & Powell, 2007).

Alcohol dependent individuals exposed to visual or olfactory alcohol cues exhibit significant psychophysiological reactivity (Carter & Tiffany, 1999). When attention is fixated on such cues, increased craving may impel alcohol consumption as a means of reducing distress. Many persons recovering from alcohol use disorders attempt to suppress cravings, which, paradoxically, serves only to increase intrusive, automatic alcohol-related cognitions (Palfai, Monti, Colby, & Rohsenow, 1997), dysphoria, and autonomic arousal (Wenzlaff & Wegner, 2000). Indeed, among alcohol dependent persons, thought suppression is negatively correlated with vagally-mediated heart rate variability (Ingjaldsson, Laberg, & Thayer, 2003), a putative index of emotion regulation and parasympathetic inhibition of stress reactions (Thayer & Lane, 2000). As thoughts of drinking intensify and are coupled with psychobiological distress, the impulse to consume alcohol as a form of palliative coping may overcome depleted self-regulation strength (Muraven, Collins, & Nienhaus, 2002; Muraven & Shmueli, 2006) leading to relapse. The attempt to avoid distress or allay its impact through compulsive alcohol consumption results in negative reinforcement conditioning that may perpetuate this cycle by further sensitizing the brain to future stressful encounters via allostatic dysregulation of neuroendocrine systems (Koob, 2003). Components of this risk chain may be especially malleable to behavioral therapies that target cognitive mediators of pathogenic gene-environment interactions.
Given that negative affect, autonomic arousal, automaticity, and attentional biases appear to be integral components of the risk chain for stress-precipitated alcohol misuse, dependence, and relapse, interventions targeting these mechanisms may hold promise for its treatment. One such intervention, mindfulness training, which originates from Buddhist traditions but has been co-opted by Western clinicians, has recently gained prominence in the psychological and medical literatures for its salutary effects on stress-related biobehavioral conditions (Baer & Krietemeyer, 2006; Ludwig & Kabat-Zinn, 2008). Mindfulness involves self-regulation of a metacognitive form of attention: a nonreactive, non-evaluative monitoring of moment-by-moment cognition, emotion, perception, and physiological state without fixation on thoughts of past or future (Garland, 2007). A growing body of research suggests that mindfulness affects implicit cognition and attentional processes (Jha, Krompinger, & Baime, 2007; Lutz, Slagter, Dunne, & Davidson, 2008; Slagter et al., 2007; Slagter, Lutz, Greischar, Nieuwenhuis, & Davidson, 2009; Wenk-Sormaz, 2005) as well as heart rate variability indices of parasympathetic regulation (Tang et al., 2009). This evidence, coupled with knowledge that alcohol use disorders involve automaticity and attentional biases, suggests that mindfulness training may be an effective treatment for alcohol dependent persons coping with stress and dysphoria.

Research suggests that mindfulness treatments may enhance clinical outcomes in substance-abusing populations. Bowen et al. (2007) employed a nonrandomized pre-post comparison group design to test the efficacy of a mindfulness meditation intervention for adults incarcerated within a minimum security correctional facility and found that mindfulness reduced post-release substance use, substance-related problems, and
psychiatric symptoms to a greater extent than standard chemical dependency services offered at the prison. Several other smaller pilot studies have examined the effects of mindfulness meditation on addictive disorders and found significant reductions in variables such as perceived stress, negative affect, stress-related biomarkers, and substance use (Marcus, Fine, & Kouzekanani, 2001; Marcus et al., 2003; Zgierska et al., 2008). Despite the suggestive evidence provided by these studies that mindfulness interventions may ameliorate factors related to stress-precipitated alcohol misuse, their lack of random assignment of participants to mindfulness and comparison conditions presents serious threats to the validity of the findings. Although mindfulness meditation is a promising intervention for substance misusers, better-controlled research is needed with different populations of substance abusers to assess the efficacy of the approach and its clinical utility. In addition, performance-based, behavioral measures and psychophysiological assessments should be used as indices of intervention-related change. Lastly, to elucidate therapeutic mechanisms of action, intervention effects on potential mediators of alcohol misuse should be assessed and accounted for in subsequent analyses.

To that end, we conducted a pilot randomized controlled trial of a mindfulness intervention designed to disrupt cognitive, affective, and physiological risk mechanisms implicated in stress-precipitated relapse to alcohol consumption. A randomized, controlled design was used to compare the therapeutic effects of a mindfulness-oriented recovery enhancement (MORE) intervention to those of an evidence-based alcohol dependence support group (ASG) in a sample of low-income, primarily African
American alcohol dependent adults recruited from a long-term, modified therapeutic community (TC).

We hypothesized that MORE would result in significantly larger decreases in perceived stress, impaired alcohol response inhibition, craving for alcohol, psychiatric symptoms, and thought suppression and significantly larger increases in mindfulness than ASG. In addition, we hypothesized that mindfulness training, relative to ASG, would lead to increased heart rate variability (HRV) recovery from stress-primed alcohol cues. Lastly, we hypothesized that mindfulness training would reduce alcohol AB, a presumed mechanism of change, by the intervention midpoint and prior to changes in clinical outcomes, such that MORE would result in significantly larger decreases in alcohol AB than ASG for participants with pre-treatment alcohol approach bias but not for those with baseline alcohol disengagement bias. This hypothesis was justified on the rationale that participants with pre-treatment alcohol disengagement bias had already learned during their TC participation to disengage their attention from alcohol cues and would therefore exhibit a floor effect.

METHOD

Sample Characteristics

The sample was comprised of alcohol-dependent adults who were living in a TC serving persons with substance-use disorders. The TC serves approximately 400 individuals annually, 33% of whom are homeless prior to entering the program.

Potential participants met study inclusion criteria if they were ≥18 years old, met lifetime Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV) alcohol dependence criteria, and had resided in the TC for ≥18 months. In this program, after 18
months individuals begin to obtain employment outside of the TC and transition to independent living arrangements, thus becoming at higher risk for relapse due to increased access to alcohol. We targeted the intervention to this group given their increased exposure to high-risk situations. Although persons with active suicidal ideation or psychosis were excluded from this study, nearly all participants had misused other psychoactive substances, and many reported numerous psychiatric symptoms on the Brief Symptom Inventory (Derogatis & Melisaratos, 1983).

Table 4 presents sample characteristics. Study participants (N = 53) had resided in the TC for a mean of 22.3 ± 3.7 months. A majority of participants were male (79.2%); 60.4% were African American and 34.0% were Caucasian. With regard to income in the year before entering the TC, 52.8% had earned < $20,000, and 32.1% earned $20,000-$40,000 in 2008 U.S. dollars. The mean age of the sample was 40.3 ± 9.4, and ranged from 19 to 57 years old. Participants reported high rates of exposure to traumatic violence: for example, the mean number of times participants had been attacked with a knife or gun by someone trying to seriously hurt or kill them was 2.4 ± 2.0 times. The mean number of DSM-IV alcohol dependence criteria met by participants was 6.5 ± 1.0, and the mean total Alcohol Use Disorders Identification Test (AUDIT) score for the sample was 32.3 ± 5.6. The mean number of standard alcoholic drinks consumed per day in the year before entering treatment was 19.0 ± 10.9, and the mean number of drinking days in the month before entering the TC was 22.8 ± 9.9. Approximately 81% of the sample reported daily use of at least one psychoactive drug in addition to alcohol before entering the TC, with cocaine the most frequently used drug (M = 17.1 ± 12.5 days used in the month before entering the TC). All participants reported continuous abstinence
from psychoactive substance use during their residence in the TC, which ranged from 18 to 28 months. Reports of abstinence were corroborated by random urinalysis conducted at the TC, as well as through daily evaluation from program staff.

Table 4. Pre-intervention Characteristics of MORE and ASG Intervention Groups, and Total Study Participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>MORE (N=27)</th>
<th>ASG (N=26)</th>
<th>Total (N = 53)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length of stay in residential program (M, SD)</td>
<td>22.4 (2.6)</td>
<td>22.2 (4.6)</td>
<td>22.3 (3.7)</td>
</tr>
<tr>
<td>Gender N (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>22 (81.5)</td>
<td>20 (76.9)</td>
<td>42 (79.2)</td>
</tr>
<tr>
<td>Female</td>
<td>5 (18.5)</td>
<td>6 (23.1)</td>
<td>11 (20.8)</td>
</tr>
<tr>
<td>Race N (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>17 (62.9)</td>
<td>15 (57.7)</td>
<td>32 (60.4)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>7 (25.9)</td>
<td>11 (42.3)</td>
<td>18 (34.0)</td>
</tr>
<tr>
<td>Other</td>
<td>3 (11.1)</td>
<td>0</td>
<td>3 (5.6)</td>
</tr>
<tr>
<td>Age (M, SD)</td>
<td>39.9 (8.7)</td>
<td>40.7 (10.2)</td>
<td>40.3 (9.4)</td>
</tr>
<tr>
<td>DSM-IV Dependence Criteria (M, SD)</td>
<td>6.5 (0.9)</td>
<td>6.6 (0.9)</td>
<td>6.5 (1.0)</td>
</tr>
<tr>
<td>Income before entering TC N (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;$20,000</td>
<td>14 (51.9)</td>
<td>14 (51.9)</td>
<td>28 (52.8)</td>
</tr>
<tr>
<td>$20,000-$40,000</td>
<td>9 (33.3)</td>
<td>8 (30.8)</td>
<td>16 (32.1)</td>
</tr>
<tr>
<td>$41,000-$60,000</td>
<td>2 (7.4)</td>
<td>3 (11.5)</td>
<td>5 (9.4)</td>
</tr>
<tr>
<td>$61,000-$80,000</td>
<td>1 (3.7)</td>
<td>0</td>
<td>1 (1.9)</td>
</tr>
<tr>
<td>&gt;$80,000</td>
<td>1 (3.7)</td>
<td>1 (3.8)</td>
<td>2 (3.8)</td>
</tr>
<tr>
<td>Drinks per day prior to entering TC (M, SD)</td>
<td>21.4 (11.9)</td>
<td>16.6 (9.5)</td>
<td>19.0 (10.9)</td>
</tr>
<tr>
<td>AUDIT (M, SD)</td>
<td>32.8 (6.0)</td>
<td>31.8 (5.3)</td>
<td>32.3 (5.6)</td>
</tr>
<tr>
<td>DAST (M, SD)</td>
<td>20.7 (5.1)</td>
<td>21.2 (5.8)</td>
<td>21.0 (5.4)</td>
</tr>
<tr>
<td>Psychiatric symptoms:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>4.9 (4.6)</td>
<td>5.0 (4.4)</td>
<td>4.9 (4.4)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>5.8 (3.8)</td>
<td>5.0 (4.3)</td>
<td>5.4 (4.1)</td>
</tr>
<tr>
<td>Hostility</td>
<td>4.0 (3.6)</td>
<td>4.3 (3.8)</td>
<td>4.2 (3.6)</td>
</tr>
<tr>
<td>Interpersonal sensitivity</td>
<td>4.4 (3.8)</td>
<td>3.9 (3.4)</td>
<td>4.2 (3.6)</td>
</tr>
<tr>
<td>Trauma:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td># of times seen someone killed in person</td>
<td>3.0 (3.2)</td>
<td>2.7 (2.7)</td>
<td>2.9 (2.9)</td>
</tr>
<tr>
<td># of times hurt or in danger of being killed</td>
<td>6.0 (11.4)</td>
<td>6.7 (12.3)</td>
<td>6.4 (11.7)</td>
</tr>
<tr>
<td># of times hit by someone with intent to hurt</td>
<td>7.3 (6.9)</td>
<td>9.1 (11.4)</td>
<td>8.1 (9.3)</td>
</tr>
<tr>
<td># of times mugged</td>
<td>2.4 (2.9)</td>
<td>1.5 (1.5)</td>
<td>2.0 (2.3)</td>
</tr>
<tr>
<td># of times attacked by a weapon or by someone with intent to kill</td>
<td>2.5 (2.2)</td>
<td>2.3 (1.7)</td>
<td>2.4 (2.0)</td>
</tr>
</tbody>
</table>
**Intervention Groups**

**MORE.** The ten-session, manualized MORE intervention was adapted as a treatment for alcohol dependence from Mindfulness-Based Cognitive Therapy, an empirically-supported, mindfulness intervention designed to prevent depression relapse (Segal, Williams, & Teasdale, 2002). MORE involves mindful breathing and walking meditations, as well as experiential exercises relating general mindfulness principles to addiction-specific issues such as relapse triggers, craving, thought suppression, stress, and unconscious substance use behaviors. A Master’s level social worker (MSW) with experience in mindfulness meditation who was trained in cognitive-behavioral treatments for substance dependence delivered the MORE intervention.

**ASG.** To control for time, attention, credibility, and group process, the ten-session ASG condition consisted of MSW-led social support groups derived from the active, evidence-based treatment condition outlined in the Matrix Model intensive outpatient treatment manual (Rawson & McCann, 2006). These groups were led by a Master’s level social worker with comparable training in addictions treatment to that of the MORE clinician. Wherever possible, ASG topics were selected to roughly match corresponding themes of the MORE intervention. ASG participants were guided to disclose feelings and thoughts about group topics, as well as to provide advice and emotional support for their peers. Although the Master’s-level social worker facilitated discussion using client-centered counseling skills (Rogers, 2003), no prescriptions for change were given.

For fifteen minutes a day, MORE participants were asked to practice mindfulness exercises while ASG participants were asked to journal for 15 minutes per day on support
A brief session by session description of the study treatment groups is detailed in Table 5.

Table 5. *Session-Specific Description of MORE and ASG Interventions*

<table>
<thead>
<tr>
<th>Session</th>
<th>MORE</th>
<th>ASG</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Discussion of mindfulness and automatic drinking; mindfulness of craving; mindful breathing and body scan</td>
<td>Discussion of triggers for alcohol dependence relapse</td>
</tr>
<tr>
<td>2</td>
<td>Discussion of cognitive reappraisal; practice of mindful decentering and mindful breathing</td>
<td>Discussion of justifications for relapse and using alcohol to mask one’s emotions</td>
</tr>
<tr>
<td>3</td>
<td>Discussion of attentional re-orienting as means of coping with negative emotions and craving; mindfulness of perceptions &amp; sensations</td>
<td>Discussion of how participants cope with emotions of anger and sadness</td>
</tr>
<tr>
<td>4</td>
<td>Discussion of craving; practice of “urge surfing,” mindfulness of craving, and contemplation of negative consequences of drinking; imaginal alcohol cue-exposure; mindful breathing practice</td>
<td>Discussion of how participants cope with craving and managing compulsive behavior</td>
</tr>
<tr>
<td>5</td>
<td>Discussion of the relationship of the stress response to craving; imaginal stress exposure; mindful breathing</td>
<td>Discussion of stress and methods participants use to relax without alcohol</td>
</tr>
<tr>
<td>6</td>
<td>Discussion of thought suppression, aversion, and attachment; exercise in the futility of thought suppression; mindful breathing and acceptance</td>
<td>Discussion of the attempt to maintain control and loss of control</td>
</tr>
<tr>
<td>7</td>
<td>Discussion of the deleterious effects of alcohol on the body; mindful interoceptive awareness; mindful walking</td>
<td>Discussion of physical health promotion and ways to recreate without alcohol</td>
</tr>
<tr>
<td>8</td>
<td>Discussion of relational triggers for relapse; mindful communication; compassion meditation</td>
<td>Discussion of codependence relationships and the challenge of forming friendships without alcohol</td>
</tr>
<tr>
<td>9</td>
<td>Discussion of interdependence, meaning, and spirituality; meditation on interdependence; mindful breathing</td>
<td>Discussion of spirituality</td>
</tr>
<tr>
<td>10</td>
<td>Review; discussion of how to maintain mindfulness practice; development of mindful relapse prevention plan; imaginal rehearsal of mindful relapse prevention; mindful breathing</td>
<td>Reflection on the support group experience; discussion of the recovery process</td>
</tr>
</tbody>
</table>
Design and Procedure

All study procedures were approved by the University of North Carolina – Chapel Hill Institutional Review Board. Participants were recruited when they had resided at least 18 months at the TC through an informational presentation about the study made at the TC, as well as through flyers and direct referrals from TC staff. Interested potential participants contacted the researchers by way of TC staff, were verbally consented for screening, and completed a 20 minute face-to-face eligibility screen. Screening procedures included administration of the Alcohol Use Disorders Identification Test (AUDIT) and a semi-structured diagnostic interview conducted by a Master’s-level social worker to ensure that all participants met lifetime DSM-IV criteria for alcohol dependence. Participants were excluded if they scored < 16 on the AUDIT, or if they endorsed screening questions indicating active psychosis (Degenhardt, Hall, Korten, & Jablensky, 2005) or suicidality.

Following the informed consent protocol, eligible participants were scheduled for a pre-intervention assessment at the TC on a separate day. At completion of the assessment, participants were randomly assigned, using a computerized random number generator, to either ten weeks of MORE or ASG. No participants refused randomization. Participants received $25 for completion of each assessment and $5 for attending each intervention session, with a possible bonus of $25 for perfect attendance of all assessments and sessions. The maximum amount a study participant could earn was $175.

Measures
Study participants completed standardized psychosocial instruments and a
psychophysiological cue-reactivity protocol before and after 10 weeks of intervention, as
well as a computer-based measurement of alcohol AB at pre-intervention, intervention
midpoint (5 weeks), and post-intervention. Psychosocial instruments were verbally
administered in interviews conducted by a research assistant who was blind to group
assignment. All measures were administered in the same order across participants in a
single session. A follow-up report will examine three-month outcomes when those data
become available.

**Intervention credibility.** Perceived credibility of the MORE and ASG
interventions as treatments for alcohol dependence was assessed after session two using a
scale ($\alpha = .83$) based on the *Attitudes Towards Treatment* measure (Borkovec & Nau,
1972) which assesses patients’ expectations of benefit once treatment has been explained.
The scale has been found to distinguish between standard psychotherapy approaches and
illogical placebo treatments, is predictive of clinical improvement, and relatively
independent of symptom severity (Borkovec & Nau, 1972).

**Mindfulness.** The *Five Facet Mindfulness Questionnaire* (FFMQ, $\alpha = .81$),
comprised of 39 likert-type items, was used to measure self-reported mindfulness. The
FFMQ yields a total score and scores for five internally consistent mindfulness factors
each with their own convergent and predictive validity: nonreactivity to inner experience
(tapped by items such as “I watch my feelings without getting lost in them”), observing
and attending to experience (“I pay attention to sensations, such as the wind in my hair or
the sun on my face”), describing and discriminating emotional experiences (“I’m good at
finding words to describe my feelings”), nonjudging of experience (“I tell myself I
shouldn’t be feeling the way that I am feeling”), and acting with awareness (“I find myself doing things without paying attention”) (Baer, Smith, Hopkins, Krietemeyer, & Toney, 2006).

*Psychosocial factors related to alcohol-dependence.* The *Brief Symptom Inventory* (BSI, $\alpha = .96$) was use to measure to what degree participants were currently distressed by psychiatric symptoms (Derogatis & Melisaratos, 1983) on 5-point scale ($0 = $ not at all, $4 = $ extremely). The 53 items include symptoms such as “Feeling fearful” and “Feelings of guilt.” Subjective alcohol craving was assessed with *Penn Alcohol Craving Scale* (PACS, $\alpha = .91$) (Flannery et al., 2001). Participants use a 7-point scale to indicate craving frequency and intensity over the past week on items like “How often have you thought about drinking or about how good a drink would make you feel?” and “At its most severe point, how strong was your craving?” Self-reported ability to inhibit the urge to use alcohol was assessed with the *Impaired Alcohol Response Inhibition Scale* (IRISA, $\alpha = .79$) (Guardia, Trujols, Burguete, Luquero, & Cardus, 2007). Participants indicate on a 4-point scale with 14 items how often over the past week they would agree with statements including “When I have decided not to drink, I find it easy not to” and “If I thought about the possibility of drinking I think I could have resisted.” The 10-item *Perceived Stress Scale* (PSS-10, $\alpha = .85$) was used to assess on a 5-point scale how often ($0 = $ never, $4 = $ very often) in the past month participants found their lives unpredictable, uncontrollable, and overwhelming (Cohen, Kamarck, & Mermelstein, 1983), and includes items such as “How often have you felt nervous and ‘stressed’?” and “How often have you felt that you were on top of things?”. Chronic thought suppression, the maladaptive and counterproductive tendency to avoid or repress undesirable cognitions
and emotions, was assessed with the White Bear Suppression Inventory (WBSI, \( \alpha = .84 \)) (Wegner & Zanakos, 1994). Participants indicate agreement on a 5-point scale with 15 items, including “I always try to put problems out of mind” and “I often do things to distract myself from my thoughts.”

*Psychophysiological cue-reactivity.* Due to our interest in the effects of stress on alcohol dependence, a cue-reactivity protocol was used to measure autonomic reactivity to and recovery from stress-primed alcohol cues. First, electrocardiogram (ECG) sensors in a lead II configuration were attached. ECG was sampled at 500 Hz and recorded continuously throughout the protocol on a Biopac MP150 data acquisition system (Biopac Systems, Goleta, CA). Once electrodes were attached, participants were asked to rate their current level of distress on a 10-point visual analogue scale (VAS) (0 = not at all, 9 = extremely). Next, participants were instructed to remain motionless, silent, and “not think about anything in particular” for a 5-minute baseline. After this baseline period, participants again rated their current distress to assess whether any initial reactivity elicited by the unfamiliar experimental context had stabilized after the 5-minute rest period. Next, 30 aversive photographs from the International Affective Picture System (IAPS) were displayed on a 15” laptop screen for 10 seconds each in succession (total duration: 5 min). Participants were asked to fixate on the image stream while holding as still as possible. After this presentation, participants again rated their current level of distress. Next, 30 photographs of beer, wine, and distilled liquor (12 of which included individuals drinking or preparing to drink alcohol) were displayed for 10 seconds each in succession (total duration: 5 min), and participants were again instructed to fixate on the image stream while holding as still as possible. At the end of the alcohol
cue exposure period, participants were asked to rate their current level of distress. Lastly, participants were instructed, “Remaining silent and keeping your body still, use whatever methods you have learned to cope with or reduce your craving” for a 5-minute recovery period, after which they again rated their current level of distress. A single qualitative question, “What methods did you use to cope?”, identified coping strategies used.

Nevrokarad aHRV (Medistar, Stegne, Ljubljana, Slovenia) was used to automatically detect and mark R-waves. The R-wave file was visually inspected to remove incorrectly identified R-waves (i.e., a movement artifact) or score R-waves that were missed by the automated detection. Kubios 2.0 (Biosignal Analysis and Medical Imaging Group, University of Finland) was used for time-domain analysis of R-R intervals. The square root of the mean squared differences between successive R-R intervals (RMSSD) was selected as our estimate of vagally mediated HRV. This analysis focused on a particular planned contrast: HRV recovery, that is, changes in HRV between alcohol cue exposure and the recovery period.

Alcohol attentional bias. A dot probe task (MacLeod, Mathews, & Tata, 1986) created in E-Prime 1.2 (PST Inc., Pittsburgh, PA) and presented on an IBM T60 laptop with a 15” screen was used to measure alcohol AB. In each trial, two grayscale images appeared, one on each side of a fixation cross (+); one image was neutral in content, the other was alcohol-related. The pair of images were presented for either 200 ms or 2000 ms. Left/right position of the alcohol images and presentation duration were both randomized and counterbalanced across 20 practice trials and 160 trials. Following a 50 ms inter-stimulus interval (ISI), a target (two dots) replaced one of the images and a distracter probe (one dot) replaced the other image; probes appeared for 100ms.
Participants were instructed to fixate on the cross between the images and indicate the location of the target by responding with a left or right button press on a keypad. Target probes randomly replaced alcohol and neutral images with equal frequency.

Some parameters of the task employed here vary slightly from tasks used previously to study addiction-related AB. Two notable differences are: (1) the fixation point remains on the screen throughout the duration of each trial, and (2) dots appear in both probe locations, requiring participants to discriminate between the location with one dot and the location with 2 dots. This task design was chosen to eliminate confounding contributions of automatic, reflexive attention that are not related to the image cues. In particular, both sudden onsets and offsets have been found to capture attention (Hopfinger & Maxwell, 2005), and the sudden offset of the fixation cross, or the sudden onset of a target at only one location, could interfere with any spatial shift in attention caused by the pictorial cues, especially in trials with the short (200ms) stimulus onset asynchrony (SOA). More specifically, if the fixation cross were to disappear when the dot probes appeared, attention would likely be drawn to the middle of the screen reducing the power to detect attentional allocation shifts due to the alcohol image cues. Additionally, if a target appeared in only one location, participants’ attention would be reflexively captured by the target. Including a place marker in the opposite target location, requires the participant’s attention to be directed to the spatial location of the target and ensures that response selection cannot be based on detection of the probe through peripheral vision. Moreover, the use of two probes requires greater attentional resources than detection of a single probe and thus this design may have more power to resolve attentional shifts induced by the alcohol cues.
Alcohol stimuli included 13 photographs of alcoholic drinks (liquor, beer, etc), as well as 7 photos of persons drinking alcohol. Neutral stimuli included 13 photos of kitchen items and 7 photos of persons in kitchen scenes. Stimulus sets were analyzed with respect to their spatial frequency content to ensure that they did not differ in terms of basic visual properties, which could elicit reflexive attentional capture irrespective of image content. On measures of both the spectral peak (Neutral: 0.0180, Alcohol: 0.0176, \( t_{(38)}=0.383, p=0.704 \)) and spectral width (Neutral: 59.20, Alcohol: 59.29, \( t_{(38)}=-0.027, p=0.979 \)), the two stimulus sets were not significantly different.

**Data analysis**

Data were examined for outliers and to ensure they met distributional assumptions for normality. All outcome variables were approximately normally distributed, save for heart rate variability data (RMSSD) which was skewed and therefore log-transformed (\( \log_{10} \)). Bivariate correlations, t-tests, chi-square tests, and repeated-measures analyses of variance (ANOVA) were used to test hypotheses, compare group differences at baseline and over time, and explore individual differences in the change process.

For AB data, trials with extreme RTs, defined as those with RTs longer than +3 SD above the individual mean (c.f., Field et al., 2004), were discarded as outliers (mean = 2.5±1.5 per participant); error trials were also discarded. For each participant, AB scores were calculated by subtracting their mean RT to probes replacing alcohol photos from their mean RT to probes replacing neutral photos, such that positive bias scores indicate an AB toward visual alcohol cues. All data are reported as means ± SD unless otherwise noted.
RESULTS

Pre-intervention Group Differences

There were no significant pre-intervention differences between MORE and ASG participants on age, gender, race, income, number of months residing in the TC, alcohol dependence criteria, AUDIT, DAST, exposure to traumatic violence, craving, impaired alcohol response inhibition, psychiatric symptoms, mindfulness, thought suppression, or alcohol attentional bias. Similarly, there were no significant differences between MORE and ASG participants in HRV responses during the pre-intervention cue-reactivity protocol. The only difference approaching statistical significance ($p = .094$) was for number of drinks/day, such that participants randomized to MORE had marginally higher levels of alcohol consumption before entering the TC than those randomized to ASG. Similarly, there were no significant differences between intervention completers and drop outs on any of the variables listed above. In addition, there was no significant difference in intervention credibility between MORE ($M = 39.4$, $SD = 6.4$) and ASG participants ($M = 40.6$, $SD = 4.7$), $F(1, 41) = .497$, $p = .485$.

Intervention Compliance and Attrition

Approximately 69% ($N = 37$; MORE = 18, ASG = 19) of the total enrolled sample ($N = 53$) completed post-intervention assessments. Attrition did not significantly differ by intervention group. Similarly, the difference in attendance between the groups was nonsignificant. On average, MORE participants completed 8.0 ($SD = 2.1$) sessions, whereas ASG participants completed 7.3 ($SD = 3.5$) sessions.

Outcomes
Table 6. *Means (SD) in Clinical and Attentional Variables*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Perceived stress</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MORE (n=18)</td>
<td>15.6 (4.7)</td>
<td>10.8 (5.3)</td>
</tr>
<tr>
<td>ASG (n=19)</td>
<td>16.0 (7.6)</td>
<td>14.5 (5.8)</td>
</tr>
<tr>
<td><strong>Thought suppression</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MORE (n=18)</td>
<td>53.6 (8.7)</td>
<td>50.1 (7.9)</td>
</tr>
<tr>
<td>ASG (n=19)</td>
<td>50.9 (11.2)</td>
<td>53.5 (9.4)</td>
</tr>
<tr>
<td><strong>Mindfulness</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MORE (n=17)</td>
<td>108.3 (12.3)</td>
<td>109.8 (14.6)</td>
</tr>
<tr>
<td>ASG (n=19)</td>
<td>115.9 (15.1)</td>
<td>118.3 (17.9)</td>
</tr>
<tr>
<td><strong>Craving</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MORE (n=18)</td>
<td>4.7 (5.5)</td>
<td>4.6 (5.3)</td>
</tr>
<tr>
<td>ASG (n=19)</td>
<td>4.9 (4.4)</td>
<td>3.2 (3.6)</td>
</tr>
<tr>
<td><strong>Impaired alcohol response inhibition</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MORE (n=18)</td>
<td>7.8 (5.5)</td>
<td>4.9 (4.9)</td>
</tr>
<tr>
<td>ASG (n=18)</td>
<td>6.2 (4.9)</td>
<td>5.3 (4.2)</td>
</tr>
<tr>
<td><strong>Global psychiatric symptoms</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MORE (n=18)</td>
<td>42.7 (36.4)</td>
<td>19.6 (12.5)</td>
</tr>
<tr>
<td>ASG (n=19)</td>
<td>46.7 (33.0)</td>
<td>31.8 (21.4)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Mid</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>200ms approach AB</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MORE (n=9)</td>
<td>7.2 (6.9)</td>
<td>10.7 (13.9)</td>
<td>2.9 (21.9)</td>
</tr>
<tr>
<td>ASG (n=9)</td>
<td>13.5 (8.2)</td>
<td>-9.0 (19.9)</td>
<td>17.6 (17.2)</td>
</tr>
<tr>
<td><strong>2000ms approach AB</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MORE (n=7)</td>
<td>12.6 (12.1)</td>
<td>3.6 (23.7)</td>
<td>2.3 (21.9)</td>
</tr>
<tr>
<td>ASG (n=7)</td>
<td>12.9 (9.1)</td>
<td>-12.8 (10.9)</td>
<td>1.7 (10.4)</td>
</tr>
</tbody>
</table>
Table 6 summarizes changes in clinical and attentional variables over the course of both the MORE and ASG interventions.

**Intervention effects on self-report measures.** Both MORE and ASG led to significant reductions in perceived stress over time, $F(1, 35) = 18.11, p < .001, \eta^2 = .34$. Moreover, there was a significant intervention X time interaction on perceived stress, $F(1, 35) = 5.06, p = .03, \eta^2 = .13$, such that MORE led to significantly larger decreases in perceived stress over a 10-week period than ASG (see Figure 2).

*Figure 2.* Intervention group differences in perceived stress at baseline and 10-weeks post-intervention

Although there were no main effects of time on thought suppression, there was a significant intervention X time interaction on thought suppression, $F(1, 35) = 4.26, p = .04, \eta^2 = .11$, such that MORE led to significant decreases in thought suppression over a 10-week period, whereas an ASG led to increased thought suppression (see Figure 3).
Conversely, while there was a main effect of time on psychiatric symptoms, $F(1, 35) = 10.83, p = .002, \eta^2 = .24$, there was no significant intervention X time interaction on psychiatric symptoms; both MORE and ASG led to significant decreases in global psychiatric symptoms over time.

With regard to self-reported mindfulness, there was neither a main effect nor an intervention X time interaction effect on the total scale and subscale scores. Similarly, there was an absence of both main and interaction effects on craving and impaired alcohol response inhibition, suggesting that both interventions had negligible effects on alcohol urges and neither reduced craving more than the other.

*Intervention effects on alcohol attentional bias.* Despite that both 2000 and 200 ms AB data were approximately normally distributed (both yielding non-significant Kolmogorov-Smirnov tests for normality), approximately one-half of participants
exhibited 2000 and 200 ms AB towards probes replacing alcohol photos while the other half exhibited AB towards probes replacing neutral photos. Given that AB towards alcohol cues is theorized to represent appetitive conditioning (Field & Cox, 2008) while AB away from alcohol cues is thought to represent disengagement from or avoidance of the substance (Townshend & Duka, 2007), we analyzed data from participants with these two apparently distinct types of AB separately. Thus, for each presentation duration (2000 ms and 200 ms) we divided participants into two groups: those with an AB > 0, i.e., the 200 ms (N = 18) and 2000 ms (N = 15) alcohol approach groups, and those with an AB < 0, i.e., the 200 ms (N = 19) and 2000 ms (N = 21) alcohol disengagement groups.

Among the 200ms alcohol approach bias group, there was a significant intervention X time interaction on 200 ms AB, $F(2, 32) = 4.76, p = .03, \eta^2 = .23$, using a Greenhouse-Geisser correction due to violations of sphericity (See Figure 4). Within-subjects contrasts revealed a significant quadratic form to the intervention X time interaction, $F(1, 16) = 8.09, p = .01$. Repeated contrasts were used to clarify the group X time interaction. These indicated that when baseline 200 ms AB was compared with midpoint 200 ms AB, there was a significant group X time interaction, $F(1, 16) = 11.85, p = .003, \eta^2 = .43$. When midpoint 200 ms AB was compared with post-intervention 200 ms AB, there was also a significant group X time interaction, $F(1, 16) = 5.18, p = .04$.

For the 2000ms alcohol approach bias group, although there was a significant main effect for time such that the AB decreased over the course of both interventions, $F(2, 12) = 4.54, p = .02$, there was no significant intervention X time effect.
Intervention group differences in 200 ms AB at baseline, 5-week intervention midpoint, and 10-weeks post-intervention for participants with alcohol approach attentional bias.

**Figure 4.** Intervention group differences in 200 ms AB at baseline, 5-week intervention midpoint, and 10-weeks post-intervention for participants with alcohol approach attentional bias.

**Intervention effects on heart rate variability responses to stress-primed alcohol cues.** To examine the effects of stress-primed alcohol cues on HRV, we controlled for severity of alcohol dependence by covarying number of drinks consumed a day prior to entering the TC. We also covaried post-intervention level of perceived stress to examine the effects of the experimental stress-primed alcohol cue exposure protocol on HRV above and beyond the HRV effects of tonic levels of stress. Although the overall $F$-test for the intervention X experimental condition (baseline, stress exposure, alcohol exposure, recovery) interaction was nonsignificant, there was a significant intervention X condition interaction on the planned contrast between alcohol cue exposure and the recovery period, $F(1,30) = 5.30$, $p = .03$, $\eta^2 = .15$, indicating that the pattern of RSA from alcohol cue exposure to recovery varied as a function of intervention (see Figure 5). No such significant intervention X condition effect on HRV was found during the
baseline assessment after covarying prior level of alcohol consumption and pre-intervention perceived stress, lending support to the interpretation that the observed post-intervention between-groups differences in HRV recovery were the result of the MORE and ASG interventions.

Qualitative inquiry indicated that during the post-intervention cue-reactivity protocol, all MORE participants practiced mindfulness meditation during the recovery period while the most common strategies used by ASG participants included cognitive reappraisal (e.g., focus on the negative consequences of drinking and the positive benefits of sobriety) as well as suppression (e.g., tried to think about something else). With regard to subjective distress during the cue-reactivity protocol, after controlling for prior level of alcohol consumption and tonic post-intervention perceived stress, MORE participants had significantly larger reductions in distress from alcohol cue-exposure to the recovery period, $F(1,32) = 4.94, p = .03, \eta^2 = .13$.

Figure 5. Intervention group differences in post-treatment HRV (RMSSD, log$_{10}$)* during stress-primed alcohol cue-reactivity protocol.
Individual difference variables and changes in stress and addiction-related factors. To determine whether improvements in self-reported ability to inhibit alcohol responses were associated with individual differences in baseline stress, changes in impaired alcohol response inhibition were correlated with pre-intervention perceived stress. Among mindfulness participants, the greatest improvements in self-reported ability to inhibit alcohol responses were found among those with highest baseline levels of perceived stress, \( r = .52, p = .03 \). Among support group participants, there was no significant correlation between these variables, indicating that the relationship between high baseline stress and improvements in the ability to inhibit alcohol responses may be attributable to the MORE intervention. Thus, it appears that MORE is most effective at inhibiting the appetitive alcohol responses of persons who experience high levels of stress at baseline, i.e. persons who may be more likely to drink as a means of coping with stress.

Importantly, among MORE participants, changes in thought suppression were correlated with changes in AB200 \( (r = .49, p = .042) \), changes in impaired alcohol response inhibition \( (r = .48, p = .045) \), and changes in post-intervention HRV recovery, \( (r = .49, p = .045) \). MORE participants who experienced the larger decreases in thought suppression over the course of intervention also evidenced the larger decreases in AB200 and impaired alcohol response inhibition while evincing the greater HRV recovery. However, among ASG participants, there was no significant correlation between these variables, suggesting that the associations between change in thought suppression, 200ms AB, impaired alcohol response inhibition, and HRV may be attributable to the MORE intervention. In contrast, across the entire sample of persons who completed either
MORE or ASG intervention, greater decreases in stress were significantly correlated with larger decreases in impaired alcohol response inhibition ($r = .33, p = .046$), suggesting that regardless of the nature of the intervention, persons who experienced the largest reductions in stress over the course of intervention reported the greatest improvements in their ability to inhibit appetitive alcohol responses, and vice versa.

**DISCUSSION**

Results suggest that training in mindfulness may affect cognitive-affective risk mechanisms implicated in alcohol dependence relapse. Mindfulness training appeared to reduce stress and thought suppression to a significantly greater extent than an evidence-based active control intervention. Notably, mindfulness training seems to decrease the 200ms AB, an implicit cognitive process linked with alcohol dependence. In addition, MORE appears to be a feasible intervention for the prevention of stress-precipitated alcohol relapse based on the high perceived treatment credibility and acceptable attendance rates in this study, as well as absence of significant differences in attrition from an ASG, a commonly accepted standard of care. This finding of feasibility is notable given the low income and otherwise high risk nature of the study sample.

Importantly, MORE reduced perceived stress to a greater extent that ASG, which is noteworthy given that social support has been shown reduce stress reactivity and buffer deleterious effects of stressful life events (Christenfeld & Gerin, 2000; Kamarck, Manuck, & Jennings, 1990). The stress reduction effects of mindfulness training among nonclinical populations are well known in the literature (Grossman, Niemann, Schmidt, & Walach, 2004), but it is noteworthy that significant effects were obtained in a sample of clinically-disordered, alcohol-dependent adults with extensive trauma histories who
may be more vulnerable due to allostatic dysregulation of neural stress circuitry (Valdez & Koob, 2004). Among recovering alcohol-dependent individuals, mindfulness training appears to be a potentially effective stress reduction technique and therefore may be a useful component in the prevention of stress-precipitated relapse. Hypothetically, the stress-reduction effects of mindfulness training among recovering addicts may be mediated by the regulation of corticotrophin releasing factor and neuropeptide Y systems that had been dysregulated in the transition to alcohol dependence. Research by Marcus and colleagues (2003) demonstrating mindfulness-induced attenuation of waking salivary cortisol levels in alcohol- and drug-dependent individuals lends support to this hypothesis, but more work is needed to clarify the mechanisms of mindfulness’ stress-reduction effect in this population.

Like stress, thought suppression significantly decreased over the course of ten weeks of mindfulness training. In turn, decreases in thought suppression among MORE participants were significantly correlated with decreases in impaired alcohol response inhibition, which suggests that for those participants who improved their ability to regulate drinking urges, they may have done so via reductions in thought suppression. This finding partially replicates results reported by Bowen et al. (2007) within the context of our randomized controlled design, although limitations of statistical power in the present study preclude replication of their tests for mediation. Thought suppression appears to play a substantial role in psychopathology (Purdon, 1999), by increasing the rate, intensity, and accessibility of the very thoughts and moods it is directed against (Wegner, Schneider, Carter, & White, 1987), as well as intensifying sympathetic nervous system activity (Gross, 2002; Gross & Levenson, 1993; Wenzlaff & Wegner, 2000). In
the context of alcohol dependence, thought suppression seems to enhance the availability of alcohol-related cognitions and affective reactions to consciousness. MORE, with its emphasis on nonjudgmental, metacognitive awareness of present-moment experience, appeared to counter this deleterious cognitive strategy and therefore may have prevented post-suppression rebound effects from exacerbating negative affect and intrusive alcohol-related cognitions that could have otherwise led to relapse. Conversely, participants in the alcohol-dependence support group may have professed invulnerability to alcohol urges for the purposes of social conformity and maintaining an appearance of competence, thereby leading to the increased thought suppression observed in the ASG data. This behavior seems to parallel the willful hubris denounced in 12-step fellowships, which exhort the person in recovery to admit their powerlessness over addiction perhaps as a means of disrupting the paradoxical effects of urge suppression.

Contrary to our hypotheses, MORE did not lead to significant increases in self-reported mindfulness, nor did it result in significant decrements in craving. This counterintuitive finding may be explained by the nature of the participants and the mindfulness training itself. Prior to mindfulness training, the alcohol-dependent individuals sampled in this study may have overestimated their level of mindfulness due to their lack of understanding of the construct and their general lack of self-awareness, which was one of the intended targets of mindfulness training itself. After ten weeks of mindfulness practice reflecting on their own internal experience, MORE participants may have had a greater sense of the meaning of the FFMQ questions, and having encountered numerous challenges in their attempts to embody the principles of nonreactivity, nonjudgment, acting with awareness, etc., may have been able to more accurately
appraise their level of mindfulness. Similarly, the alcohol-dependent study participants residing in a therapeutic community where access and exposure to alcohol was limited may have lacked awareness of the extent to which they continued to have drinking urges, leading to an underreporting of craving at baseline. This lack of awareness of alcohol reactivity that has been shown to be predictive of future relapse (Rohsenow et al., 1994). Because MORE was, in part, designed to increase consciousness of craving and decrease urge suppression, ten weeks of the intervention may have heightened awareness of latent alcohol urges, thus resulting in an apparent lack of change in craving over time. Of course, such interpretations are speculative, and a number of alternative interpretations are possible, such as inaccurate reporting due to social desirability bias, ceiling/floor effects due to previous participation in a therapeutic community, and the possibility of true null effects of MORE on mindfulness and craving.

Both interventions led to statistically significant reductions in psychiatric symptoms. The fact that ASG exerted a treatment effect comparable to that of MORE indicates that the control condition used in this study was therapeutically active. This finding bolsters the body of research on the clinical utility of social support groups for the treatment of psychological problems (Dean & Lin, 1977; Laudet, Magura, Vogel, & Knight, 2000), as well as replicating the findings of Bowen et al. (2006) that mindfulness training decreases psychiatric symptoms among substance abusers. However, given that no significant differences were found between intervention conditions, it cannot be ruled out that the reductions in psychiatric symptoms observed were due to placebo effects.

In contrast, MORE and ASG had differential effects on HRV during a stress-primed alcohol cue-reactivity paradigm. Although there were no significant differences at
baseline, after 10 weeks of intervention, MORE participants, relative to ASG participants, exhibited high HRV during alcohol cue exposure which then decreased as they practiced mindfulness meditation. The pattern observed among MORE participants may be interpreted as evidence of engagement of emotion regulation processes during alcohol cue exposure followed by disengagement of cognitive resources from alcohol-related cognitions and cravings during the recovery period.

Greater HRV is linked to greater prefrontal inhibition of stress-induced sympathetic nervous system activation (Thayer & Lane, 2009). However, lower HRV may be related to decreased parasympathetic efference associated with focused, concentrated attention on the breath, as well as the rhythmic pacing of the breath that may stem from novice meditation techniques of breath focus and counting (Peressutti, Martin-Gonzalez, J, & Mesa, 2009). These techniques may work as a rhythmic stimulus that entrains the breath and reduces overall HRV. While highly experienced meditators, who describe the phenomenology of meditation as an effortless opening of awareness, may show increased HRV during mindfulness practice, novice meditators, who use primarily concentration meditation with the breath as the object of focus, exhibit decreased HRV during meditation practice (Peressutti et al., 2009).

This interpretation is consistent with findings of suppressed HRV during working memory and sustained attention tasks (Hansen, Johnsen, & Thayer, 2003). As Thayer and Lane (2000) assert in their neurovisceral integration model, “sustained attention is associated with vigilance and the suppression of HRV. The appropriate short-term suppression of HRV and the associated focusing of attention are important for effective self-regulation” (p. 207). For novices, meditation on the breath is effortful, and the
cognitive load and working memory demands inherent in intense attentional focus may lead to phasic vagal withdrawal from the heart.

Other converging evidence supports our interpretation. Indeed, the neurovisceral integration model (Thayer & Lane, 2000) posits that as CAN (central autonomic network) activity increases HRV increases, leading to increased braking activity on the heart. Thus, HRV should increase proportionally to the intensity of conscious processing of emotion and emotion regulation. Indeed, relative to ASG participants, MORE participants had higher post-intervention HRV during stress and alcohol cue exposure (despite having lower pre-intervention levels of HRV), perhaps indicative of marshalling greater cognitive control in service of regulating negative emotions triggered by aversive images and cravings elicited by alcohol cues. In contrast, the post-intervention ASG group evidenced phasic suppression of HRV during stress and alcohol cue exposure, a pattern that seems to indicate disruption of homeostasis, anxiety, and stress reactivity (Thayer & Lane, 2000).

Germane to this discussion are findings that high self-regulation efforts to resist temptation result in increased HRV (Segerstrom & Nes, 2007), and alcoholics who reported having a greater ability to regulate alcohol urges experienced increased HRV when exposed to alcohol cues relative to those who were reportedly less able to regulate alcohol-use compulsions (Ingjaldsson et al., 2003). In fact, Ingjaldsson et al. (2003) reported that high compulsive drinkers experienced a nonsignificant decrease in HRV from baseline to alcohol cue exposure. This differential pattern is similar to the pattern observed in our own data, where the response of MORE participants to alcohol cue
exposure parallels that of the low compulsive drinkers and the response of ASG participants parallels that of high compulsive drinkers.

After alcohol cue exposure, when emotion-regulatory efforts were no longer needed, MORE participants may have experienced vagal withdrawal leading to decreased HRV during the recovery period. During this period MORE participants may have shifted their attentional focus onto the breath, resulting in a state of cognitive simplicity that requires effortful engagement of working memory to maintain, thus, leading to suppressed HRV and increased HR. In contrast, ASG participants used emotion regulation strategies such as reappraisal and suppression during the recovery period. These strategies may have required a high level of PFC engagement, thus leading to the increased HRV observed among ASG participants during the recovery period, an interpretation supported research showing increased HRV during reappraisal and suppression (Butler, Wilhelm, & Gross, 2006). Hypothetically, this pattern of cardiac response may indicate the superior efficiency of mindfulness as an emotion regulation strategy relative to reappraisal, distraction, and suppression strategies, an inference supported by our self-report data that show that MORE led to greater reductions in subjective distress from alcohol cue-exposure to recovery.

Importantly, among MORE participants but not among ASG participants, larger pre-post reductions in thought suppression were correlated with greater post-intervention HRV recovery from alcohol cue exposure. Hence, we can speculate that those MORE participants who most successfully learned to employ mindful awareness of alcohol-related cognitions and craving were able to disengage from those mental phenomena more efficiently, as reflected by increased HRV recovery. In contrast, alcohol dependent
persons who continued to use thought suppression as a strategy may have remained
fixated on alcohol-related cognitions and cues due to the post-suppression rebound effect,
resulting in less HRV recovery which may be reflective of continued ruminative
engagement (Key, Campbell, Bacon, & Gerin, 2008).

Intervention effects on 200 ms alcohol AB may also index degree of
disengagement from alcohol-related cognitions. Given that mindfulness training has been
shown to impact attentional orienting and alerting processes (Jha et al., 2007), we
hypothesized that MORE would impact 200ms AB in persons with alcohol approach
biases at baseline. Although this hypothesis was supported by the data, we expected a
simple decreasing linear change trajectory and not the apparently u-shaped, quadratic
pattern observed. What could account for the pattern of results seen in the AB data?
During the first five weeks of the MORE intervention, participants were encouraged to
decenter from their cognitive-emotional experience and become aware of whatever
thoughts and feelings arose during the meditation sessions. Several mindfulness exercises
involved imaginal alcohol exposure in order to teach methods to deal mindfully with
craving. During these exercises, participants were encouraged to imagine having strong
alcohol cravings and to notice the resultant physiological and affective reactivity. In
addition, participants were asked to log daily experiences of craving and to carefully
attend to the psychophysiological concomitants of the craving experience. Participation
in these exercises may have activated alcohol use action schema, triggering implicit
positive alcohol expectancies through the imagination of conditioned, hedonic stimuli,
resulting in the deployment of a conditioned attentional response, i.e. increases in the 200
ms AB. As the mindfulness participants became more proficient at self-regulating

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attention and developing metacognitive awareness of automatic response patterns, this effect may have been attenuated by the post-intervention time point.

Conversely, by five weeks into the alcohol support group intervention, participants may have felt considerable pressure to conform to the culture of the group, which emphasized an abstinence-only attitude toward recovery from alcohol dependence. Group culture focused on the negative consequences of drinking, and the climate of the group was such that verbal pronouncements of one’s ability to resist the temptation to drink were reinforced. These discussions may have activated negative implicit alcohol expectancies and therefore attenuated the 200 ms AB. However, as social bonds increased throughout the latter half of the group, participants may have become more comfortable disclosing their “weaknesses” to their peers, and were more apt to admit and discuss cravings for alcohol. This may have led to the triggering of positive implicit alcohol expectancies, and without exercising the metacognitive deautomatization afforded by the mindfulness training, such triggering may have led to increased 200 ms AB.

What is the clinical significance of a small yet statistically significant change in 200 ms AB over time among alcohol dependent MORE participants with baseline approach biases? To answer this question, post-intervention craving was regressed onto pre-post change in 200 ms AB for MORE participants with alcohol approach biases (N = 9). Pre-post change in 200 ms AB significantly predicted post-intervention craving, $\beta = .79$, $p = .01$, R-squared = .62. Similarly, among MORE participants with baseline 200 ms approach bias, post-intervention 200 ms AB was significantly negatively correlated with post-intervention craving, $r = -.72$, $p = .03$. The finding that decreases in 200 ms AB over
the course of mindfulness intervention predicted higher craving at the end of the intervention is counterintuitive, as is the finding that smaller 200 ms AB is associated with higher subjective craving.

However, when this seemingly paradoxical finding is viewed from the lens of Tiffany’s Cognitive Processing Model of Addiction, a coherent interpretation emerges. Tiffany (1990) posited that conscious craving was the result of the inhibition of automatized alcohol use action schema. The 200 ms AB has been considered an automatic, unconscious bias in initial attentional orienting towards alcohol-related stimuli (Field & Cox, 2008). Stimuli presented for 200 ms are too fast for conscious deployment of attention, and thus, initial orienting to such stimuli may be driven by implicit appetitive processes that can detect and operate on nonconscious stimuli. According to Tiffany’s theory, when alcohol use action schemas operate in an unobstructed fashion, there is an absence of craving. However, when an alcohol dependent individual interrupts the automatic, appetitive cognitive-behavioral responses that subserve his or her addiction, the resultant negative affective state of craving arises.

Training in mindfulness meditation may lead to disruption of automatized behavior patterns. Deikman (1966) conceptualized the effect of meditation as a form of “deautomatization,” that is, an undoing of automatization whereby unconscious, habitual patterns of perception and motor behavior are reinvested with attention. It is plausible that during the course of the MORE intervention, automatized alcohol use action schema were deautomatized through formal and informal mindfulness practices, which involve the intentional and conscious direction of attention to cognitions, emotions, physiology, and behavior. Given that such conscious attentional processing disrupts automatic
processing (Lieberman, 2003), mindfulness training may have interrupted alcohol use action schema, which Tiffany’s (1990) theory would predict to result in an increase in conscious craving. In accord with this prediction, data from the present study show that decreases in 200 ms AB, an automatic orienting of attention towards alcohol cues, significantly predicted higher levels of craving among mindfulness participants in the baseline 200 ms alcohol approach bias group.

This finding also is sensible in the context of the findings of Rohsenow and colleagues (1994), who found that greater awareness of physiological reactivity during alcohol exposure predicted decreased drinking among alcohol dependent men at three months post-intervention. If mindfulness training disrupts automatic processing of alcohol cues, leading to increased awareness of craving, this effect may indeed be salutary. By Tiffany’s (1990) account, craving is the result of an inhibition of appetitive tendencies. Thus, greater awareness of craving-related reactivity may indicate that automatic addictive patterns have been disrupted, which might eventuate in decreased drinking over time. In fact, this was one of Rohsenow et al.’s interpretations of their finding: “greater attention to drinking related stimuli or responses may result in greater extinction of conditioned reactions” (1994, p. 624). It should be noted that the form of attention mentioned in this quotation is conscious attention, i.e., maintenance or re-orienting of attention and not the unconscious initial orienting indexed by the 200 ms AB. In further support of Tiffany’s contention that cravings are not necessary for the instigation of substance use, Rohsenow and colleagues (1994) found that urge to drink did not significantly predict post-intervention alcohol consumption.
The identified effects of MORE on thought suppression also support a
deautomatization interpretation of the correlation between mindfulness-induced
reductions in 200 ms AB and higher post-intervention craving, and notably, among
MORE participants, reductions in thought suppression were significantly correlated with
decreases in 200 ms AB. Over time, mindfulness training may reduce the tendency to
suppress one’s thoughts and feelings, thereby allowing cravings that had been previously
suppressed to surface to consciousness. In so doing, automatic appetitive schema
(indexed by 200 ms AB) which may have been operating unchecked by controlled
cognitive processing during active suppression may become accessible to explicit
cognitive control. Thus, as thought suppression decreases, controlled cognitive
processing can be more effectively deployed to inhibit and counter appetitive responding
(as evidenced by the significant correlation between decreases in thought suppression and
decreases in impaired alcohol response inhibition), resulting in decreased attentional
capture by alcohol cues coupled with the conscious experience of craving as alcohol use
action schema are disrupted. Through mindfulness, alcohol urges can be attended to in a
nonreactive, nonjudgmental fashion, obviating the need for suppression. In so doing,
cravings may extinguish over time, instead of intensifying as a result of suppression
attempts.

Alternatively, the counterintuitive relationship between decreased 200 ms AB and
increased craving may be explained by the methodological issues that plague self-report
measures of craving, such as lack of consensus on the operationalization of the construct,
the temporal scope of the craving measurement, and inconsistency between psychological
and physiological indices of craving (Monti, Rohsenow, & Hutchison, 2000; Tiffany,
Carter, & Singleton, 2000). If awareness of craving increased after ten weeks of mindfulness training, differences in pre-post craving scores may be biased, leading to spurious correlations between change in craving and 200 ms AB. In contrast, performance on a dot probe task with alcohol stimuli presented for 200 ms latencies may be a more valid indicator of appetitive tendencies towards alcohol than self-reports of craving.

In sum, the unwitting attempts of recovering alcohol dependent persons to suppress appetitive cognitive-emotional reactions towards alcohol may obscure these responses from consciousness only to perpetuate and intensify them within the cognitive unconscious. In the domain of unconscious mental life, automatic processes run smoothly and efficiently uninhibited by volitional control (Kihlstrom, 1987). Hence, by shunting appetitive reactions into the unconscious, the alcohol dependent individual may increase the very appetitive response towards alcohol he or she is trying to suppress and/or exacerbate psychophysiological reactivity. Mindfulness training may serve to undo the process of making conscious responses unconscious. Thus, practice of mindfulness may promote the recovery of alcohol dependent persons through: a) deautomatization of alcohol use action schema, resulting in diminished attentional bias towards subliminal alcohol cues and increased craving as a result of disrupted automaticity; and b) decreased thought suppression resulting in increased awareness of alcohol urges over time, increased HRV recovery from alcohol cue-exposure, and improved ability to inhibit appetitive responses.

The primary limitation of this study is its small sample size, which limits statistical power and generalizability of the findings. Generalizability of study findings
may also be limited by the sample selection and the absence of random sampling -
participants who had resided for at least 18 months in a TC self-selected into the study,
and thus had been exposed to a therapeutic milieu for an unusually long length of time
relative to a 28-day residential or intensive outpatient treatment format. Due to the
probable high motivation and competence of participants who had successfully
maintained sobriety for over 18 months while residing in the TC, MORE and ASG may
have had greater salutary effects in this sample than in persons with less commitment or
ability to change. Conversely, because persons who could not maintain sobriety for a
substantial length of time (i.e., those who were most severely impaired or dependent)
were not included in this study, intervention effects may have been underestimated for
individuals who might have shown the greatest benefit from MORE or ASG. Self-report
measures were administered during face-to-face interviews, which may have led to social
desirability bias in self-reported outcomes. Our interpretations of HRV data may be
confounded because we were unable to control for the effects of respiration (Grossman &
Taylor, 2007), although there is ample debate in the literature regarding the need for such
corrections (e.g., Denver, Reed, & Porges, 2007). Given the presence of counterintuitive
findings, this study needs to be replicated with a larger and more broadly representative
sample, and additional research is needed to explore the clinical significance of the effect
of mindfulness training on stress, thought suppression, HRV, and alcohol attentional bias.
To that end, we are collecting follow-up data and intend to examine how intervention-
related changes in these cognitive, affective, and physiological variables predict alcohol
relapse in a subsequent paper.
Despite evidence suggesting that stress appraisal and attentional biases are key components of alcohol dependence, the form of addictions treatment most available to poor and marginalized persons, social support groups, does not target these pathogenic mechanisms directly. In contrast, practice of mindfulness may attenuate stress reactivity and thought suppression while disrupting addictive automaticity, resulting in increased awareness of craving and greater ability to cope with alcohol urges in stressful contexts. Hence, mindfulness training may hold promise as an alternative, targeted treatment for stress-precipitated alcohol dependence among vulnerable members of society.
References


INTEGRATIVE SUMMARY

The convergence of theory, cross-sectional data, and clinical-experimental evidence detailed in this series of papers offers insights into the nature of stress-precipitated alcohol dependence and its treatment. Stress reactivity, implicit cognition, and emotional suppression appear to be key intervening mechanisms between the experience of stressful life events and the compulsive use of alcohol. Targeting the links between these processes may unhinge alcohol misuse and dependence, and prevent relapse. The construct of mindfulness may be an important consideration in treatment and prevention efforts.

What is the relationship between mindfulness and stress-precipitated alcohol dependence? To answer this question, we must organize the often complementary and sometimes incongruent findings of this work. Theory and past research suggests a number of pathways through which mindfulness, as a self-regulatory strategy, may disrupt alcohol dependence: emotion-regulation, decentering from perseverative cognition, deautomatization of alcohol use action schema, attentional disengagement from alcohol cues, awareness of craving, and cue-exposure without suppressing alcohol urges. Our present work has found evidence for a number of these therapeutic mechanisms.

Among recovering alcohol dependent adults, trait-level mindfulness was inversely correlated with stress, thought suppression, and attentional fixation on alcohol cues. Thus, persons endorsing higher levels of mindfulness exhibited lower levels of risk factors associated with alcohol dependence. In this manner, high levels of trait
mindfulness may reflect self-regulatory competence, and thus persons reporting higher levels of mindfulness may be less susceptible to relapse. It should be noted that the sample of alcohol dependent persons in this investigation were not treatment naïve; thus, levels of mindfulness reported by participants may reflect both dispositional tendencies towards mindfulness and the salutary effects of long-term residence in a therapeutic community, which may impart attitudes of nonjudgment, nonreactivity, emotional awareness, etc.

Our clinical trial data suggest that mindfulness training significantly decreases stress, thought suppression, and alcohol AB. The effects of mindfulness training on stress and thought suppression outcomes roughly parallel the pattern observed for trait mindfulness. On the contrary, mindfulness training appears to effect alcohol AB for alcohol cues presented for 200 ms, while dispositional mindfulness is associated with the 2000 ms AB. This incongruity deserves further exploration.

To resolve this incongruity, the construct of mindfulness must be parsed more carefully. Mindfulness is a term that describes a trait, state, and practice (Chambers, Gullone, & Allen, 2009). *Dispositional mindfulness* , as a trait, reflects the tendency towards equanimity, a present-centered temporal orientation, awareness of emotional and perceptual experience, and taking a nonjudgmental attitude towards one’s own thoughts and feelings. The *state of mindfulness* (which, as it unfolds over time, may actually be comprised of numerous stages each with their own distinct phenomenology) is characterized by a decentered, non-conceptual attentiveness, wherein thoughts, emotions, and sensations are experienced as ephemeral, insubstantial, and not colored by a prereflexive sense of ownership or belonging to the self. In metaphorical terms, when
immersed in the state of mindfulness, mental experiences become like “clouds passing in a clear blue sky” or “waves on the surface of the ocean.” The *practice of mindfulness* involves anchoring one’s attention onto an object (e.g., the breath, the sensation of one’s feet against the ground) from which one can metacognitively monitor moment-by-moment experience without judgment or reactivity. In so doing, one develops a recursive awareness of one’s own background state of consciousness (Chalmers, 2000; Damasio, 1999); that is, a global mode (e.g., awake, aroused, sleeping, dreaming) that encompasses and regulates the contents of consciousness (e.g., specific thoughts, feelings, perceptions, or memories).

Given the multidimensionality of the construct of mindfulness, it is perhaps unsurprising that dispositional mindfulness and mindfulness practice have differing relationships to alcohol AB. We used a self-report measure of trait mindfulness, and thus, participants rated their level of mindfulness according to their self-awareness of their own qualities. This measure was inversely correlated with 2000 ms alcohol AB, a bias that is thought to index conscious attending to alcohol cues. Both self-reported dispositional mindfulness and 2000 ms alcohol AB appear to involve explicit cognition. The ability to reflect on one’s own level of mindfulness requires declarative cognitive processes such as evaluation, interpretation of semantic meaning, and executive decision making. Some of these same processes may have been invoked during the 2000 ms participants were exposed alcohol and neutral photographs, at which time they may have assessed the personal implications of the images and their overall hedonic value (i.e., whether alcohol is perceived as positive and rewarding or negative and destructive). In turn, these evaluations may have led to fixation on the alcohol photos or to
disengagement from them. More mindful individuals appear to have disengaged more readily from alcohol cues presented for 2000 ms than their less mindful counterparts.

Conversely, dispositional mindfulness was unrelated to the 200 ms alcohol AB measure, a bias thought to index implicit attentional orienting to alcohol cues. This bias is considered to be driven by unconscious, automatic cognitive processes that presumably are not accessible to self-awareness. If evaluations of trait mindfulness require self-awareness of one’s own capacities, then it is possible that assessments of this trait might be unrelated to implicit processes such as those indexed by the 200 ms alcohol AB task. In other words, through self-reflection persons might evaluate themselves as being mindful but be unaware of the extent to which they remain affected by implicit appetitive responses towards alcohol.

In contrast, training in mindfulness meditation exerted significant effects on 200 ms alcohol AB task performance. The practice of meditation has been shown to impact automatic cognitive processes (e.g., Slagter et al., 2007; Wenk-Sormaz, 2005) and may lead to durable changes in brain function (Brefczynski-Lewis, Lutz, Schaefer, Levinson, & Davidson, 2007; Holzel et al., 2007; Lutz, Brefczynski-Lewis, Johnstone, & Davidson, 2008; Lutz, Greischar, Rawlings, Ricard, & Davidson, 2004). In turn, meditation-related changes in patterns of neural activation may result in neuroplastic alterations to brain circuitry instantiating attention, emotion regulation, interoception, and autonomic control (Garland & Howard, 2009; Holzel et al., in press; Holzel et al., 2008; Lazar et al., 2005; Luders, Toga, Lepore, & Gaser, 2009; Vestergaard-Poulsen et al., 2009). Hence, repeated, intentional engagement of mindful states appears to cause changes in attentional and neurophysiological processes that may be inaccessible to self-awareness. While
biobehavioral changes may result from mindfulness training (e.g., reduction in 200 ms alcohol AB and increased parasympathetic recovery from alcohol cues), only the downstream effects of such changes (e.g., the sense that one is more equanimous or no longer as “captivated by” alcohol cues) may be reportable via self-reflection. Although mindfulness training reduced 200 ms AB, it did not appear to increase dispositional mindfulness. Thus, after 10 weeks of training, MORE participants exhibited substantial changes in implicit attentional operations, while apparently being unaware of changes in self-reported mindfulness.

Recognizing the potential instability of the 200 ms AB data due to the small sample size and unknown reliability of the measure, these interpretations remain provisional, at best. Alternatively, the differential effects of dispositional mindfulness and mindfulness training may be explained by methodological limitations associated with the use of self-report instruments such as the Five Facet Mindfulness Questionnaire to assess mindfulness. The limitations inherent in self-report measures of mindfulness may be overcome through the use of performance-based, behavioral, and physiological measures, such as those employed in this series of investigations. Reconciling first- and third-person data remains the task of neurophenomenology (Varela, Thompson, & Rosch, 1991) and scholars dedicated to the study of the effects of mental experiences on biobehavioral outcomes.

In spite of and perhaps as a result of these unanswered questions, the rough outline of a future research agenda is taking shape. Mindfulness training appears to hold promise as an intervention for alcohol misuse and dependence, particularly as a form of relapse prevention. Full scale clinical trials are needed to establish the efficacy of this
promising treatment, as well as to explore the mediators and moderators of the treatment effect. Two important research questions deserve further exploration: (a) “How might mindfulness training disrupt the cognitive-affective mechanisms of alcohol dependence?”; and (b) “For whom is mindfulness training most effective?” In answering these questions, important insights into the nature of alcohol dependence and its remediation may be gleaned.

Ultimately, the study of stress, alcohol dependence, and mindfulness illuminates the age-old question of determinism. This question was empirically investigated through the ingenious work of Benjamin Libet and colleagues, which revealed a readiness potential, viz., a cerebral activation several hundred milliseconds prior to the onset of the experience of volition that precedes a willed motor act (Libet, Gleason, Wright, & Pearl, 1983; Libet, Wright, & Gleason, 1982). If acts of will are initiated by unconscious, neural impulses, how much more so must compulsive, habitual behaviors (i.e., those observed in alcohol dependence) be driven by conditioned circuits in the brain. Yet, if this process were inexorable and immutable, the prospect of liberating oneself from grips of addiction would be moot.

The work of Libet, however, also suggests the possibility of freedom from automatic and impulsive initiation of action. Libet et al. (1982, 1983) discovered a temporal gap between the experience of willing an action and the act itself, and in this gap, the individual was able to veto the execution of the action. Thus, in this case, an unconscious readiness potential would precede the volition, but would not ultimately manifest in motor action. There is, then, the possibility of selecting amongst possible courses of action, even if the impulses underlying these actions result from the
unconscious and autonomous function of the nervous system. Thus, both the “Yes” and “No” to act are presented to consciousness, and it is consciousness that selects amongst the options, leading to the eventual execution or inhibition of the action. The process of selection is the province of attention: that capacity for certain subsets of information to gain preeminence in the competitive processing of neural networks at the expense of other subsets of information (Desimone & Duncan, 1995). The selectivity of attention allows for the veto, in that the impulse to withhold the action is amplified relative to the impulse to act. According to models of attention as biased competition (e.g., Desimone & Duncan, 1995), attended stimuli govern behavior; thus, if attention is allocated to the inhibitory impulse instead of the addictive urge, processing resources will be biased towards impulse control, leading to its primacy in the competition between responses.

Neurocognitive resources are preferentially allocated to emotional stimuli through automatic and selective attentional mechanisms (Dolan & Vuilleumier, 2003), potentially enhancing the survival of the organism by highlighting the hedonic value of objects, events, and beings in the natural world. Although emotional stimuli (such as alcohol or the stressors potentially leading to its consumption) are especially salient, having privileged access to information-processing resources of the brain, modulatory effects of affect may compete with other sources of top-down control (Vuilleumier, 2005). Attentional operations such as the metacognitive shifts produced by mindfulness meditation are cognitive control processes that can attenuate the influence of affective stimuli on biobehavioral responses (Ochsner & Gross, 2005, 2008). Thus, the ability to self-regulate cognitive control mechanisms in the midst of emotional distress appears to be a key means of willfully reducing negative affect (Posner & Rothbart, 1998). The
intentional self-regulation of attention and cognitive appraisal processes resulting in an inhibitory veto of affectively-charged neural impulses may be seen as an instance of volition, *par excellence*.

Mindfulness training, then, may be a means of exercising human volition over habitual responding triggered by external stimuli, and is therefore congruent with the social work value of self-determination and commitment of the profession to empowering vulnerable persons to overcome problematic environmental forces. Thus, mindfulness training is a strengths-based intervention focused on the cultivation of personal empowerment. As a technique for developing self-regulatory capacity, mindfulness training enhances coping and thereby promotes resiliency. Mindfulness-based interventions such as MORE may be useful additions to the armamentarium of clinical social work, particularly as a treatment for addiction. Given the natural fit between mindfulness training and the overarching practice philosophy of the social work profession, it could advance practice significantly if a cadre of social work professionals were trained in the delivery of this promising intervention.
References


