



Thought suppression, impaired regulation of urges, and Addiction-Stroop predict affect-modulated cue-reactivity among alcohol dependent adults

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ABSTRACT

Abstinent alcohol dependent individuals commonly employ thought suppression to cope with stress and intrusive cognitions about alcohol. This strategy may inadvertently bias attention towards alcohol-related stimuli while depleting neurocognitive resources needed to regulate urges, manifested as decreased heart rate variability (HRV) responsivity to alcohol cues. The present study tested the hypothesis that trait and state thought suppression, impaired regulation of urges, and alcohol attentional bias as measured by the Addiction-Stroop would have significant effects on the HRV responsivity of 58 adults in residential treatment for alcohol dependence (mean age = 39.6 ± 9.4 , 81% female) who participated in an affect-modulated cue-reactivity protocol. Regression analyses controlling for age, level of pre-treatment alcohol consumption, and baseline HRV indicated that higher levels of trait thought suppression, impaired regulation of alcohol urges, and attentional fixation on alcohol cues were associated with lower HRV responsivity during stress-primed alcohol cue-exposure. Moreover, there was a significant state \times trait suppression interaction on HRV cue-responsivity, such that alcohol dependent persons reporting high levels of state and trait suppression exhibited less HRV during cue-exposure than persons reporting low levels of state and trait suppression. Results suggest that chronic thought suppression taxes regulatory resources reflected in reduced HRV responsivity, an effect that is particularly evident when high trait suppressors engage in intensive suppression of drinking-related thoughts under conditions of stress. Treatment approaches that offer effective alternatives to the maladaptive strategy of suppressing alcohol urges may be crucial for relapse prevention.

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1. Introduction

Alcohol dependent persons in recovery confront numerous challenges to their resolve to remain abstinent. Exposure to socioenvironmental stressors may render such individuals vulnerable to relapse, as stress promotes habitual behaviors that are resistant to changes in outcome contingencies while inducing deficits in executive function (Dias-Ferreira et al., 2009). Hence, it is plausible that stress enhances relapse risk by evoking automatic appetitive responses and impairing functions involved in the regulation of alcohol urges (Garland et al., 2011). Although the relationship between stress and alcohol use has been studied for decades (e.g., Conger, 1951; Cappell and Herman, 1972), less is known about how the neurocognitive mediators of the stress

response promote relapse among recovering alcohol dependent individuals.

Recently, we proposed a theoretical model that specifies feedback circuits between stress, implicit cognition, self-regulation attempts, and the drive to consume alcohol (Garland et al., 2011). According to this model, repeated alcohol misuse in the context of stress and negative affect may establish automatic alcohol-use action schemas, memory structures that coordinate and compel behaviors involved in alcohol consumption. Hypothetically, these schemas are evoked in a conditioned response to present stressors (Tiffany, 1990), which may bias attention towards alcohol (Field and Powell, 2007) and increase the urge to drink as a means of palliative coping. However, among alcohol dependent persons in recovery, such urges are often ego-dystonic, i.e., perceived as intrusive and incongruent with the goal of abstinence (Soutullo et al., 1998). In an attempt to regulate mounting alcohol urges, such individuals may employ “willpower” to suppress the urge to drink. Indeed, the attempt to suppress intrusive thoughts of drinking is common among persons in treatment for alcohol use disorders, and is prospectively associated with greater frequency of alcohol-related thoughts and craving episodes (Kavanagh et al., 2009).

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Ironically, *thought suppression* may be a key component in the cognitive-mediation of stress-precipitated relapse insofar as this strategy inadvertently further *biases attention towards alcohol-related cognitions and affective reactions*. These so-called “rebound effects” are suggested by research demonstrating that attempted suppression often results in an increased rate of the thoughts and emotions it is directed against (Wegner et al., 1987; Wenzlaff and Wegner, 2000). For example, when asked to suppress urges following alcohol cue exposure, alcohol dependent adults exhibited speeded reaction times to alcohol-related statements relative to control phrases (Palfai et al., 1997). Similarly, abstinent alcohol dependent adults experienced greater Stroop interference for the word “alcohol” after they had initially attempted to suppress alcohol-related thoughts compared to individuals who expressed such thoughts (Klein, 2007). Rebound effects have also been identified for appetitive behavior. Experimental induction of suppression of thoughts of smoking (Erskine et al., 2010) and eating (Erskine and Georgiou, 2010) leads to greater enactment of such behaviors. These findings may be explained by Wegner’s *ironic process theory* (1994), which asserts that suppression involves two processes: (a) a conscious search for cognitive contents consistent with the desired mental state, and (b) an implicit monitoring process that searches continually for cognitions that are inconsistent with the desired state. When attention is automatically deployed in search of undesirable mental content to be replaced, the ensuing positive feedback loop leads to hyperaccessibility of unwanted cognitions (Wegner and Erber, 1992), amplifying their frequency and intensity under conditions of stress (Nixon et al., 2009; Williams and Moulds, 2007). As a result, the intrusive and distressing nature of the target thoughts is magnified by the very process employed to avoid them (Abramowitz et al., 2001).

Hypothetically, suppression may exhaust the capacity for self-regulation, which Baumeister and colleagues have characterized as a limited resource that is depleted through repeated acts of self-control (Baumeister, 2003; Muraven and Baumeister, 2000). Integrating notions drawn from Wegner’s and Baumeister’s theories, we propose that suppression undermines cognitive control (Pu et al., 2010), resulting in *impaired regulation of alcohol urges*, increased stress, and relapse. These deleterious effects may be exacerbated when persons whose self-regulatory resources have already been depleted due to chronic thought suppression (Wegner and Zanakos, 1994) engage in acute or state suppression of alcohol-related thoughts triggered by stressful circumstances.

Although suppression is intended to reduce emotional experience, instead, it often leads to sympathetic arousal, indicated by changes in electrodermal response, finger pulse amplitude, pulse transmission time, and skin temperature (Gross and Levenson, 1993; Roberts et al., 2008). The physiological effects of suppression may be illuminated by the neurovisceral integration model (Thayer and Lane, 2000), which proposes that a network of central (e.g., prefrontal and anterior cingulate cortices) and autonomic (e.g., vagal nerve) structures regulate attention and emotion by exerting inhibitory influences over perturbations to visceral homeostasis, such as those experienced by abstinent alcohol dependent persons when confronted with stressors and/or alcohol cues. Regulation of attentional and emotional responses by this central autonomic network may be indicated by increases in high-frequency heart rate variability (HRV), i.e., the beat-to-beat variation in heart rate modulated by parasympathetic outflows of the vagus on the pacemaker of the heart (Thayer and Lane, 2009). Increased HRV is positively correlated with the ability to flexibly regulate attention and emotion (Friedman, 2007; Thayer et al., 2009). Conversely, decreased HRV is observed during inflexible cognitive-emotional states associated with heightened and prolonged sympathetic arousal (Brosschot et al., 2006), such as attentional hypervigilance (Hansen et al., 2003) and perseverative cognition (Brosschot, 2010; Key et al., 2008).

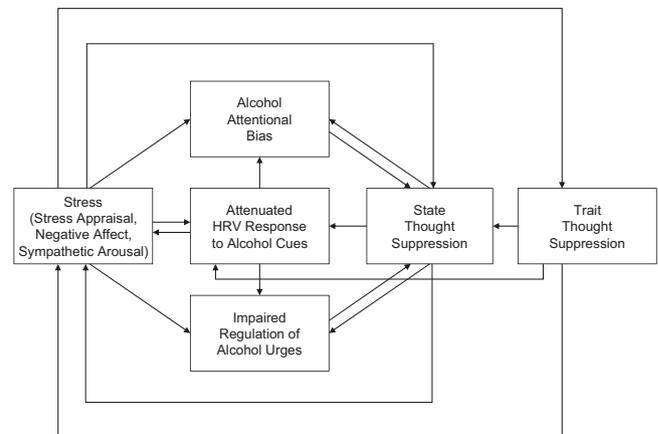


Fig. 1. Theoretical model depicting relationships between stress, trait and state thought suppression, impaired regulation of alcohol urges, alcohol attentional bias, and attenuated HRV response to alcohol cues.

Hence, the neurovisceral integration model predicts that alcohol dependent individuals would exhibit attenuated HRV responsivity coupled with a rigid focus of attention on alcohol cues (indexed by greater alcohol attentional bias) when they employ thought suppression to regulate stress and urges.

Such predictions accord with results from Ingjaldsson et al.’s (2003) study of 49 alcohol dependent subjects, which found that chronic thought suppression was inversely associated with heart rate variability (HRV) responsivity to an imaginal alcohol cue-exposure script. In addition, individuals who were less able to regulate alcohol urges exhibited reduced HRV responses to alcohol cues compared to persons reporting greater ability to regulate urges, who evidenced elevated HRV in response to alcohol cues. Hence, thought suppression among alcohol dependent individuals is linked with impaired regulation of alcohol urges by the central autonomic network.

Although thought suppression and impaired ability to regulate drinking urges were associated with an attenuated HRV response, Ingjaldsson et al. (2003) did not examine these relationships simultaneously to determine the extent to which these factors independently contributed to HRV responsivity to alcohol cues among alcohol dependent persons. Moreover, no behavioral measures of attention to alcohol cues were evaluated, leaving relationships between alcohol attentional bias (AB), thought suppression, impaired regulation of urges, and HRV responsivity unspecified. Lastly, Ingjaldsson et al. (2003) did not discriminate chronic thought suppression, that is, the trait-like tendency to suppress thoughts in general in everyday life, from state suppression of alcohol-related thoughts during the cue-reactivity paradigm. The present investigation sought to provide a partial test of our theoretical model (Fig. 1) and extend the work of Ingjaldsson et al. (2003) by examining these relationships in the context of an affect-modulated cue-reactivity paradigm intended to induce stress prior to alcohol cue-exposure (Cheetham et al., 2010). The use of this paradigm was crucial, given the centrality of stress to our conceptual framework, and the body of work demonstrating that stress often precipitates relapse (Sinha, 2007) through its effects on cognitive and affective processes in addiction (Garland et al., 2011). We hypothesized that trait suppression, impaired regulation of urges, and alcohol AB would be significantly inversely associated with HRV responsivity to stress-primed alcohol cues, and that higher levels of trait suppression and impaired regulation of alcohol urges would predict elevated subjective alcohol cue-reactivity. We also hypothesized that greater state suppression of thoughts of drinking during stress cue-exposure would be associated with increases in subjective reactivity to subsequent alcohol cue-exposure. Finally,

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