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Anxiety mediates the association between anxiety sensitivity and coping-related drinking motives in alcoholism treatment patients

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Abstract

Anxiety sensitivity (AS), the tendency to interpret feelings of anxiety as dangerous, is a core dispositional trait in a well articulated and extensively studied cognitive model of proneness to anxiety disorder. In recent years, there has been an increasing body of findings that also links AS to the tendency to use alcohol in general and the tendency to use alcohol as a means of coping with negative affect in particular. We expand on this empirical base by proposing and testing a theoretical model in which anxiety symptoms mediate the association between AS and alcohol use. That is, we propose that AS promotes anxiety symptoms, which, in turn, promote alcohol use aimed at coping with anxiety and other negative affect states. Over a 1-year data collection period, we assessed 82 alcohol-dependent individuals shortly after they began an intensive alcoholism treatment program. Self-reported anxiety symptoms associated with distinct anxiety syndromes were obtained with reference to the month period preceding their entry into the treatment program. Other information, including the presence of withdrawal symptoms, was obtained via interview. We found that syndrome-related anxiety symptoms and Trait Anxiety, but not State Anxiety or withdrawal symptoms, mediated the significant association between AS and the self-reported tendency to use alcohol as a means of controlling anxiety symptoms. Demonstrating a similar pattern of findings, but much less robustly so, were tests of these mediator models using alcohol use aimed at coping with negative affect (vs. coping with anxiety per se) as an outcome. In discussing these findings, we attempt to further develop a coherent model that incorporates AS, anxiety symptoms, and drinking motives. Our findings suggest that these relationships may differ for negative affect not specifically related to anxiety. We also discuss the

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possible associations of AS to withdrawal symptoms implied by our findings.

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1. Introduction

Anxiety sensitivity (AS) is an individual difference variable that is based on the clinical concept "fear of fear" (Reiss, 1987). Specifically, AS refers to the extent to which an individual interprets otherwise benign anxiety symptoms as dangerous (Reiss & McNally, 1985). For example, an individual with high AS might interpret a rapid heart rate as indicating an impending heart attack. Such catastrophic misinterpretations would obviously compound or amplify an initially low-level anxiety/stress response, potentially resulting in panic-level fear reactions. Thus, AS is conceptually unique from anxiety per se, a view that is buttressed by studies showing that AS can be differentiated empirically from the general tendency to respond fearfully to a wide range of stimuli (i.e., trait anxiety; e.g., Taylor, Koch, & Crockett, 1991).

Although distinct constructs, AS and anxiety responding are thought to be causally related. Early theoretical treatments of the fear of fear concept tended to assign causal priority to anxiety in an essentially Pavlovian framework (i.e., AS as a conditioned response; e.g., Goldstein & Chambless, 1978; Klein, 1981). However, over the last 20 years, evidence and theoretical treatments have pointed to AS as a dispositional variable reflecting beliefs that somatic manifestations of anxiety are indicative of danger (e.g., Reiss, 1987). Consistent with this view and opposed to the conditioning model, high AS has been found to exist in the absence of anxiety disorder and has been found to predict the future development of pathological fears and anxiety reactions (e.g., Reiss, Peterson, Gursky, & McNally, 1986). In short, modern AS theory holds that AS is a cognitive dispositional trait that serves as an anxiety accelerant capable of amplifying low-level anxiety feelings to clinical-level anxiety episodes.

AS has also been linked to alcohol use and problems (cf. Stewart, Samoluk, & MacDonald, 1999). For example, those with high AS report more drinking to cope with negative affect than do those with low AS, while those with low AS report more social-affiliative motives for drinking than do those with high AS (Stewart & Zeitlin, 1995). This finding is especially relevant in that coping-related drinking may place individuals at greater risk for the development of significant alcohol use problems (Cooper, Russell, Skinner, & Windle, 1992). In another study, Stewart, Peterson, and Pihl (1995) found that women with higher AS drank more overall and drank to excess more often than did those with low AS. Stewart and Pihl (1994) found that alcohol dampened anticipatory anxiety more among those with high AS than those with low AS. These findings led Stewart and Pihl to speculate that those with anxiety will be more motivated to drink if they also have high AS because this group experiences greater alcohol-induced anxiety dampening (see similar argument made by McNally, 1996). However, an equally compelling interpretation is that high AS promotes high levels of anxiety, which, in turn, promote greater alcohol use (e.g., Kushner, Abrams, & Borchardt, 2000).