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Causal modeling of relations among learning history, anxiety sensitivity, and panic attacks

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Abstract

We used structural equation modeling (SEM) to test the hypothesis that childhood instrumental and vicarious learning experiences influence frequency of panic attacks in young adulthood both directly, and indirectly through their effects on anxiety sensitivity (AS). A total of 478 university students participated in a retrospective assessment of their childhood learning experiences for arousal–reactive sensations (e.g., nausea, racing heart, shortness of breath, dizziness) and arousal–non-reactive sensations (i.e., colds, aches and pains, and rashes). SEM revealed that learning history for arousal–reactive somatic symptoms directly influenced both AS levels and panic frequency; AS directly influenced panic frequency; and learning history for arousal–non-reactive symptoms directly influenced AS but did not directly influence panic frequency. These results are consistent with the findings of previous retrospective studies on the learning history origins of AS and panic attacks, and provide the first empirical evidence of a partial mediation effect of AS in explaining the relation between childhood learning experiences and panic attacks in young adulthood. Implications for understanding the etiology of panic disorder are discussed.

Keywords: Structural equation modeling; Anxiety sensitivity; Panic attacks; Instrumental learning; Vicarious learning

Anxiety sensitivity (AS) is a fear of anxiety-related sensations, which arises from beliefs that these sensations have harmful physical, social, or psychological consequences (Reiss, 1991). To illustrate, people with high AS tend to fear sensations such as nausea, racing heart, shortness of

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breath, or dizziness. This is because they believe such symptoms portend imminent physical catastrophe, public embarrassment, or mental breakdown. In contrast, people with low AS regard such sensations as harmless.

AS tends to amplify anxiety reactions, and therefore may cause panic attacks and panic disorder (Reiss, 1991). In support of this conjecture, AS is higher in people with panic attacks, compared to normal controls (see Taylor, 1999). Longitudinal studies also have shown that AS is one of the best predictors of the development of panic attacks (e.g., Schmidt, Lerew & Jackson, 1997, 1999; see also review by Schmidt, 1999).

In a twin study examining the genetic and environmental contributions to AS, Stein, Jang, and Livesley (1999) found that AS has a heritable component, accounting for 45% of the variance in AS levels. Over half of the variance in AS levels was attributable to environmental influences. This raises the question of the potential role of childhood learning experiences in the development of elevated AS.

Instrumental and vicarious learning during childhood may influence AS. Instrumental learning refers to direct learning where behavior is acquired or eliminated by its consequences. Instrumental learning could contribute to a heightened sensitivity to anxiety symptoms (AS) if a child displayed fear of anxiety-related bodily sensations (e.g., racing heart, dizziness) and was rewarded in some way as a result. Rewards might include special attention (positive reinforcement) or being allowed to miss school (negative reinforcement). If a parent discouraged a child's display of fear of anxiety-related symptoms, AS would be theoretically less likely to develop (punishment). Vicarious learning refers to learning by watching and imitating others (Bandura, 1986). This could account for the development of high AS if a parent modeled, and was rewarded for, fear reactions to their own such bodily symptoms in the presence of their child, or verbally transmitted their beliefs about the harmfulness of such symptoms to the child.

In the first study of the learning history origins of AS, Watt, Stewart and Cox (1998) found high AS individuals, compared to those with lower AS, retrospectively reported more childhood instrumental and vicarious conditioning experiences involving somatic symptoms. Contrary to predictions, however, high AS individuals reported more of these learning experiences for both anxiety-related and cold symptoms prior to age 18 than individuals with lower levels of AS. This result was recently replicated and extended in a second study (Watt & Stewart, 2000) where the scale pertaining to instrumental learning of cold symptoms was replaced by instrumental and vicarious conditioning scales assessing parental responses to the child's and parent's own non-anxiety-related symptoms (i.e., lumps, stomach problems, pain, and tiredness). AS levels were once again found to be significantly positively correlated with retrospectively reported childhood learning experiences involving parental reinforcement and modeling of both anxiety-related and non-anxiety-related somatic symptoms (Watt & Stewart, 2000).

The lack of specificity to anxiety symptoms in these two previous studies on the origins of AS stands in contrast to the findings of studies investigating the roles of childhood learning history factors in the development of panic attacks and panic disorder. Ehlers (1993) reported significant differences between panickers and normal controls in terms of instrumental and vicarious learning experiences for anxiety symptoms. However, there were no group differences in non-specific parental encouragement in response to non-anxiety-related (i.e., cold) symptoms in childhood. A replication of Ehlers' (1993) analysis with the panickers vs non-panickers in the Watt et al. (1998) study yielded results identical to those reported by Ehlers (1993) for panickers vs normal controls.