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An Experimental Test of the Effects of Parental Modeling on Panic-Relevant Escape and Avoidance Among Early Adolescents

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Escape and avoidance behaviors play a prominent role in the maintenance and possibly development of panic disorder, yet the literature regarding the etiology of these emotion-regulation strategies is relatively underdeveloped. The current study experimentally tests hypotheses that parental modeling of escape during a well-established panic-relevant biological challenge increases panic-relevant escape and avoidance among offspring. Fifty physically and psychologically healthy early adolescents (28 females; M_{age} = 11.58; 86% Caucasian), stratified by gender, were randomly assigned to observe one of their parents (39 females; M_{age} = 40.04): either (a) model completing a 3-min voluntary hyperventilation exercise (no escape modeling group) or (b) model premature termination of a similar procedure (escape modeling group). Offspring in the escape modeling group demonstrated a stronger escape response by discontinuing their own challenge sooner than those in the no-escape modeling group (r = .70). No group differences emerged in terms of avoidance responding, as indexed by nearly identical responding in terms of delay time before initiating the challenge, respiration rate, and self-reported willingness to engage in a second proposed challenge. Results suggest that parental behaviors may play an important role in the development of some forms of panic-relevant responding. These preliminary findings may have important implications for future prevention programs targeting parents and at-risk youth.

Keywords: escape; avoidance; modeling; panic; voluntary hyperventilation

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MODERN THEORIES OF PANIC DISORDER (PD) suggest panic attacks are learned fear responses to bodily

arousal that, in part, involve activation of the fight-or-flight system (Bouton, Mineka, & Barlow, 2001; Gorman, Kent, Sullivan, & Coplan, 2000). During an initial panic attack that may be triggered by organic (e.g., substance use, medical conditions) or psychological (e.g., trauma) causes, unconditioned fear accompanying the attack becomes associated with early cues of the attack (Wolpe & Rowan, 1988). These cues include initially neutral somatic sensations (e.g., dyspnea, perspiration) that occur just prior to the onset of the attack (Goldstein & Chambless, 1978), and benign stimuli and settings (e.g., movie theatres, driving) relevant at the time of the attack. As these associations are strengthened via additional attacks, low-grade sensations and external cues alone may come to elicit "out-of-the-blue" panic attacks and situational panic attacks (Bouton et al., 2001). Importantly, given panic attacks are intense, negatively valenced, emotional experiences, some individuals become chronically anxious about experiencing additional attacks in anticipation of relevant triggers. As a result, they attempt to reduce this anxiety and the likelihood of future attacks by escaping and avoiding stimuli or situations that appear to have triggered past attacks (Barlow, 2002), which usually are characterized by elevated bodily arousal. Escape and avoidance behaviors that reduce contact with anxiety-eliciting stimuli are negatively reinforced, thus increasing their rate of occurrence and preventing exposure to conditioned cues for panic attacks. As a result, extinction learning that would result from repeated and prolonged exposure to conditioned cues in the absence of panic and feared outcomes is limited, thereby maintaining conditioned fear of bodily arousal. Taken together, this model suggests that panic-relevant fear (and subsequent anxiety) is acquired via classical conditioning, and is in turn broadly maintained by operant conditioning (Mowrer, 1947).

Negatively reinforced behaviors increase in frequency due to reducing or preventing contact with aversive environmental cues. Escape occurs when an organism withdraws from or increases proximity between itself and aversive stimuli, whereas avoidance prevents engagement with aversive stimuli altogether. Escape and avoidance, in all forms (e.g., behavioral, cognitive, experiential), are considered hallmark maintaining variables for anxiety disorders (Barlow, Allen, & Choate, 2004). With regard to PD, people suffering from panic-related difficulties report elevated levels of avoidance (Feldner, Zvolensky, & Leen-Feldner, 2004), and contemporary approaches regard panic-relevant escape and avoidance behaviors as prime maintaining factors that must be targeted via empirically supported interventions (Craske & Barlow, 2008).

Scholars have suggested that such responses to distress (e.g., negative affectivity, somatic experiences) also may contribute to the etiology of PD if they occur before an initial, clinically relevant, episode (e.g., panic attack; Craske, 2003). Consistent with this perspective, learning opportunities that engender protective (e.g., latent inhibition) and risk-enhancing phenomena (e.g., super learning) occur throughout one's developmental history and not just during clinically relevant episodes. Indeed, panic-relevant bodily arousal takes many forms aside from a panic attack (e.g., nausea), and is experienced by most individuals throughout their lives. Accordingly, pre-clinical opportunities exist for people to respond to, and form associative relations between external and internal stimuli and bodily arousal. In fact, panic-relevant escape and avoidance has been documented in nonclinical samples (Unnewehr, Schneider, Margraf, Jenkins, & Florin, 1996; Wilson & Hayward, 2006), which may increase risk for PD. For example, repeated escape from and avoidance of normative elevations in bodily arousal limits habituation and/or (subclinical) extinction learning that would result from exposure to panic-relevant sensations. Escape from, and avoidance of, benign experiences involving bodily arousal from an early age may minimize mastery of such situations and lead to the onset of panic-related problems by gradually increasing fearful conditioning and setting the stage for panic attacks.

Escape and avoidance are typically presumed to develop following direct conditioning experiences by being first emitted when unconditioned stimuli elicit unconditioned responses not only at an autonomic level, but also at a musculoskeletal level (Solomon & Wynne, 1954). As a result of multiple such pairings, previously neutral stimuli come to elicit conditioned responses while also functioning as occasion-setters for defensive behaviors to be emitted. Defensive behaviors are then reinforced by fostering safety and terminating contact with unconditioned (escape) or conditioned (avoidance) internal and external fear-eliciting stimuli (Dinsmoor, 2001). Although such two-factor accounts of the acquisition of escape and avoidance via an interaction of classical and operant conditioning have received support from behavioral (Cain & LeDoux, 2007; Dinsmoor) and neurobiological research (Lazaro-Munoz, LeDoux, & Cain, 2010), direct conditioning-based explanations have been criticized as unable to account for problems that develop in the absence of distinct classical conditioning events (e.g., Menzies