


8-2014

Effects of Parental Modeling on the Development of Panic-Relevant Escape and Avoidance among Adolescents: An Experimental Study Using a Biological Challenge Procedure

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Effects of Parental Modeling on the Development of Panic-Relevant Escape and Avoidance
among Adolescents: An Experimental Study Using a Biological Challenge Procedure

Effects of Parental Modeling on the Development of Panic-Relevant Escape and Avoidance
among Adolescents: An Experimental Study Using a Biological Challenge Procedure

A dissertation submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy in Psychology

by

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ABSTRACT

Escape and avoidance behaviors play a prominent role in the maintenance of panic-spectrum difficulties and may also be implicated in the development of such problems. Although the current state of the literature regarding the etiology of these emotion-regulation strategies is relatively underdeveloped, it appears that learning experiences involving parental modeling of such behaviors may be instrumental in the development of panic-relevant escape and avoidance. Given that no tests of such a conceptualization exist at this time, the current study proposed an initial experimental examination of whether parental modeling of defensive reactivity during a well-established biological challenge could impact panic-relevant escape and avoidance among adolescents. Random assignment, stratified by adolescent gender, was utilized to assign 50 adolescents to observe their parents either: a) complete participation in a 3-min voluntary hyperventilation exercise (no escape modeling group); or b) prematurely terminate a similar procedure (escape modeling group). It was predicted that adolescents who observed their parents prematurely terminate participation in the biological challenge would evidence greater escape and avoidance of panic-relevant sensations compared to adolescents who observed their parents complete participation in the same biological challenge. Results were partially consistent with hypotheses. Relative to adolescents in the no escape modeling group, adolescents in the escape modeling group demonstrated a stronger escape response, but not a stronger avoidance response, in relation to the voluntary hyperventilation exercise. Although preliminary, these results suggest that parental behaviors may play an important role in the development of panic-relevant defensive reactivity.

ACKNOWLEDGMENTS

I would like to express my deepest gratitude to my graduate mentor and dissertation director, Dr. Matthew Feldner, for providing me with the instrumental guidance that fostered my development as a research scientist, clinician, educator, and person. Indeed, primarily as a result of mentorship that I received from Dr. Feldner in his roles as research advisor, clinical supervisor, and course instructor, I am now a better informed scholar and a wiser human being.

I would like to thank my masters and dissertation committee members, Dr. Ellen Leen-Feldner and Dr. Lindsay Ham, for helping me develop my research program over the past six years. I would like to thank Dr. Jeffrey Lohr for helping me appreciate how science, as a candle in the dark, should perpetually be applied to improve our understanding of the human condition. I would like to thank Dr. Ana Bridges and Dr. Jennifer Veilleux for their supervision, guidance, and support during the past year. I am also very grateful for the undergraduate mentorship provided by Dr. Douglas Woods, Dr. Michael Himle, and Dr. James Moyer.

I would like to thank the graduate students and the members of our research laboratory who have provided me with incredible support throughout graduate school. To this end, special thanks are in order for Heidemarie Blumenthal, Marie Karlsson, and Christal Badour.

I would like to thank all of the clients and research participants who have made my graduate training possible.

Lastly, I would also like to offer a very special thank you to Vanessa Gaspard and Argus. Without the two of you life during graduate school would have been much less fulfilling.

DEDICATION

This dissertation is dedicated to my parents, Andrei L. Bunaciu and Zoea Bunaciu, for sacrificing everything to give me the opportunity to be successful in life.

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I. INTRODUCTION

Panic attacks are sudden periods (peaking within 10 minutes) of intense fear or discomfort highlighted by somatic (e.g., tachycardia, dyspnea, paresthesia) and cognitive symptoms [e.g., fear of dying; *Diagnostic and Statistical Manual of Mental Disorders–Fourth Edition-Text Revision* (DSM-IV-TR) American Psychiatric Association (APA), 2000]. Although frequently reported by non-treatment seeking populations, outside of psychopathology [i.e., nonclinical panic attacks; see Norton, Cox, and Malan (1992) for a review], panic attacks also occur in the context of many psychological conditions (e.g., disorders of anxiety, mood, and substance misuse; Kessler et al., 2006). To this end, panic attacks play a central role in relation to panic disorder (PD), a common anxiety disorder characterized by the repeated occurrence of “out of the blue” panic attacks that are followed by concern or worry about future attacks, their consequences, or a significant change in behavior associated with the panic attacks. Panic attacks are also relevant to agoraphobia (AG), or the fear and avoidance of places from which escape may not be possible in the event of a panic attack (APA, 2000).

Panic-spectrum problems (i.e., panic attacks, PD, AG; Barlow, 2002) are relatively common occurrences. The *National Comorbidity Survey Replication* (NCS-R) reported lifetime prevalence rates of 22.7% for panic attacks, 3.7% for PD, and 1.1% for AG (Kessler et al., 2006). Moreover, PD with and without AG represents a disabling condition accompanied by high levels of impairment, suffering, and societal costs (Batelaan et al., 2007; Kessler, Chiu, Demler, & Walters, 2005; Markowitz, Weissman, Oullette, Lish, & Klerman, 1989; Mendlowicz & Stein, 2000), that is frequently comorbid with other psychological disorders (Kessler et al., 2006; Zvolensky, Bernstein, Marshall, & Feldner, 2006). For these reasons, research has emphasized the importance of understanding variables involved in the etiology and maintenance

of panic-spectrum problems. After all, advancing knowledge in this domain may prove invaluable for the treatment and prevention of these difficulties and may also have benefits in terms of human suffering and economic costs (Zvolensky, Schmidt, Bernstein, & Keough, 2006). Prior to addressing the role of escape and avoidance as key variables in this area, a review of a contemporary theoretical model of the etiology and maintenance of panic-spectrum problems is necessary.

A. THEORETICAL MODEL OF ETIOLOGY AND MAINTENANCE FOR PANIC-SPECTRUM PROBLEMS

Modern theories on the development of PD and related problems (e.g., AG) have converged on the postulation that panic attacks are learned fear responses to bodily arousal that, in part, involve activation of an individual's 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., stressful events) causes, the unconditioned fear that accompanies the attack becomes associated (via classical conditioning) with early cues of the panic attack (Wolpe & Rowan, 1988). These early cues include initially neutral interoceptive somatic sensations (e.g., breathlessness, elevations in heart rate) that occur just prior to the onset of the panic attack (Goldstein & Chambless, 1978), as well as benign exteroceptive stimuli that are present in the surrounding context at the time of the panic attack. As these associations are strengthened via repeated pairings occurring with subsequent panic attacks, the low-grade, interoceptive somatic sensations alone may come to elicit panic attacks that can appear to occur from "out of the blue" (Bouton et al., 2001). On the other hand, situational panic attacks may be elicited by the exteroceptive cues that were also paired with the initial panic attacks. Importantly, given that panic attacks are intense, negatively

valenced, emotional experiences, some individuals become chronically anxious about the possibility of experiencing additional panic attacks in anticipation of potentially relevant triggers. As a result, they make substantial efforts to reduce this anxiety and the likelihood of experiencing future attacks by escaping and avoiding situations or stimuli that appear to have triggered previous attacks (APA, 2000; Barlow, 2002), which frequently are characterized by relatively elevated bodily arousal. For example, a person who experienced a panic attack after running up a flight of stairs may avoid aerobic exercise to reduce the likelihood of experiencing a subsequent panic attack, despite possibly not being able to report that the bodily arousal experienced while running up the stairs was the actual trigger for the attack. Escape and avoidance behaviors that appear to decrease anxiety and reduce the likelihood of unwanted panic attacks are negatively reinforced, thus increasing the rate of such behaviors and preventing exposure to conditioned interoceptive and exteroceptive cues for panic attacks. Such behaviors limit extinction learning that would result from repeated and prolonged exposure to conditioned cues in the absence of panic and feared consequences, thereby maintaining conditioned fear of bodily arousal. Taken together, this well-established model suggests that panic-relevant fear (and subsequent anxiety) is acquired via classical conditioning, and is in turn broadly maintained by behaviors that reduce or prevent panic-relevant anxiety via operant conditioning (Mowrer, 1947).

B. ESCAPE AND AVOIDANCE AS RISK FACTORS FOR PANIC DISORDER MAINTENANCE AND DEVELOPMENT

Functionally, escape and avoidance represent two classes of negatively reinforced operant behaviors as they result in the modification of aversive stimuli or events, and as a consequence, their rate of responding increases. Escape responses occur when made in the

presence of a punishing stimulus, whereas avoidance responses are considered escape behaviors moved forward in time as they prevent the occurrence of the punishing stimulus altogether. Accordingly, these two classes of behavior represent distinct endpoints on a spectrum of negative reinforcement. Although routinely observed in day-to-day life, as aforementioned, escape and avoidance behaviors, in all of their forms (e.g., behavioral, cognitive, experiential), are widely considered hallmark maintaining variables for all anxiety disorders (Barlow, Allen, & Choate, 2004; Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). With regards to PD, it is well-established that people suffering from panic-spectrum problems 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 directly targeted via empirically-supported psychosocial interventions (Craske & Barlow, 2008).

Some scholars have recently proposed that defensive responses to various forms of distress may also function as essential contributing factors in the development of anxiety disorders (Craske, 2003). It is therefore possible that panic-relevant escape and avoidance may represent not only maintenance factors, but also risk factors for PD, particularly if they occur before and/or immediately in response to an initial, clinically-relevant, panic attack. Although such a conceptualization stands in contrast to predominant theoretical models (Bouton et al., 2001), the presence of escape and avoidance before the formal onset of psychopathology is a likely prospect. After all, 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 can take many forms aside from a full-blown panic attack (e.g., perspiration, nausea, a brief dizzy spell, an elevated heart beat, shortness of breath), and is likely experienced by most

individuals throughout their lives. Accordingly, many opportunities exist for individuals to respond to and form associative relations between exteroceptive and interoceptive stimuli and bodily arousal even in the absence of psychopathology. Given that support has been found for the presence of panic-relevant escape and avoidance among individuals without histories of panic-spectrum difficulties (i.e., nonclinical samples; Unnewehr, Schneider, Margraf, Jenkins, & Florin, 1996; Wilson & Hayward, 2006), it is possible that such defensive responses may increase risk for the development of PD. For example, repeated escape from and avoidance of developmentally normative elevations in bodily arousal may limit habituation and/or extinction that would likely result from continued exposure to panic-relevant sensations. As a result, avoidance of benign experiences involving panic-relevant bodily arousal from an early age may minimize mastery of such situations, and ultimately enhance fearful responding in the event of a later panic attack, thereby possibly leading to the development of PD and other related psychopathology (e.g., AG).

Panic development is influenced by a combination of genetic (Hettema, Neale, & Kendler, 2001), environmental (Craske, 2003), and cognitive (McNally, 2002) variables. Thus far, research has identified a number of prominent risk factors (e.g., anxiety sensitivity; Olatunji & Wolitzky-Taylor, 2009) that influence the classical conditioning process described earlier, thereby aiding in the acquisition of conditioned fear of bodily arousal and the subsequent development of PD (please see Bouton et al. [2001] and Zvolensky, Schmidt et al. [2006] for extensive reviews). However, despite the wide-ranging implications of panic-relevant escape and avoidance, minimal efforts have been made to elucidate variables enhancing the likelihood that panic-spectrum problems will be maintained (via operant conditioning) if they are acquired. Indeed, there has been a paucity of empirical research examining factors related to the

development of these emotion-regulation strategies among either clinical or nonclinical samples. More specifically, in the context of PD, minimal research has examined why only some people who experience panic attacks are inclined to start engaging in negatively reinforced behaviors by escaping or avoiding, rather than approaching, stimuli and situations that are largely (presumed) neutral prior to their conditioned association with initial panic attacks. Similarly, few investigations have addressed why panic-relevant escape and avoidance develop among relatively healthy individuals without histories of panic-spectrum difficulties.

C. RISK FOR PANIC-RELEVANT ESCAPE AND AVOIDANCE

Existing evidence suggests that gender and anxiety sensitivity (AS) are two variables most closely linked to panic-relevant escape and avoidance. Specifically, among samples of people who are already suffering from PD, women appear to be at greater risk than men to develop panic-related complications in the form of agoraphobic avoidance (please see Clum and Knowles [1991] for a review). Similar findings have also been noted among nonclinical populations where women, from an early age, appear to engage in escape and avoidance in the context of panic-relevant biological challenges more often than men (Bunaciu, 2009; Marshall-Berenz, Gonzalez, Leyro, & Zvolensky, 2011). It also appears that individuals who are highly fearful of anxiety-related sensations due to their perceived negative consequences are at elevated risk for escaping and avoiding activities that elicit bodily arousal. Indeed, significant positive associations between AS and panic-relevant avoidance have been documented among individuals diagnosed with PD (Berle et al., 2008; White, Brown, Somers, & Barlow, 2006), as well as persons without a history of psychopathology (Bonn-Miller, Zvolensky, & Bernstein, 2009; Gregor & Zvolensky, 2008; Wilson & Hayward, 2006). Importantly, although female gender and heightened AS are risk factor candidates likely implicated in the development of panic-

relevant escape and avoidance, it must be noted that most of the research referenced herein has employed designs that are correlational and cross-sectional in nature, which prevents any causal or temporally-oriented inferences to be drawn between these variables. It is likely however that other vulnerabilities exist, but have not yet been evaluated. Before addressing such potential variables in the area of panic, it may be beneficial to first review how escape and avoidance behaviors are posited to develop.

D. ETIOLOGY OF ESCAPE AND AVOIDANCE

It has long been accepted that learning experiences can enhance anxiety vulnerability via classical conditioning (Watson & Rayner, 1920). Indeed, etiological models for most fear-based disorders (e.g., posttraumatic stress disorder; Keane, Zimering, & Caddell, 1985) emphasize the importance of classical conditioning, and as outlined earlier, contemporary theories of PD development (Bouton et al., 2001) also consider this process instrumental in the acquisition of panic-relevant fear and anxiety. Importantly, escape and avoidance are also often presumed to develop as a result of direct classical conditioning experiences. Consistent with Mower's (1947) two-factor theory, escape and avoidance responses are believed to first be emitted when an unconditioned stimulus elicits an unconditioned response that occurs 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 noxious unconditioned (escape) or conditioned (avoidance) internal and external fear-eliciting stimuli (Dinsmoor, 2001). Although such two-factor accounts for the acquisition of escape and avoidance behaviors via an interaction of respondent and operant conditioning have received notable empirical support from

behavioral (Cain & LeDoux, 2007; Dinsmoor, 2001) and neurobiological research (Lazaro-Munoz, LeDoux, & Cain, 2009), direct conditioning-based explanations have been criticized as being unable to account for problems that develop in the absence of distinct classical conditioning events (Davey, 1992; Menzies & Clarke, 1993). To this end, a number of explanations for the indirect acquisition of anxiety-related difficulties in the absence of such episodes have been proposed. Rachman (1977) for example suggested that fears may be learned vicariously and by verbal transmission of negative information, two pathways that recently have been conceptualized to rely on the same associative learning principles underlying classical conditioning (please see Field [2006] for an extensive review). More specifically, as part of the vicarious learning and information transmission pathways, a model's fearful response or transmitted verbal information are posited to represent unconditioned stimuli that can become associated with a variety of neutral stimuli that later may be capable of eliciting a conditioned fearful reaction that can include escape and avoidance responses.

Although most models of anxiety and avoidance acquisition make reference to classical conditioning phenomena, anxiety vulnerability can also be enhanced by primarily operant processes. Here, too, escape and avoidance behaviors are likely to be fostered via direct (Ollendick, Vasey, & King, 2001) and indirect pathways (Dymond & Roche, 2009). For example, escape and avoidance learning may be promoted by selective positive reinforcement of fearful responses during the course of one's developmental history. Positive reinforcement likely also plays a vital role during vicarious learning where a model's anxious behavior can not only elicit a fearful response but also set the occasion for an escape or avoidance response to be emitted if it has previously resulted in reinforcement from the model or the environment. Importantly, following a lengthy history of being intermittently reinforced for imitating certain

anxious behaviors, individuals are likely to derive rule-governed behaviors and emit novel responses through the process of generalized imitation even though the newly emitted behaviors have never been modeled or reinforced in the past (Baer & Sherman, 1964). Escape and avoidance responses that have been reinforced in the context of nonspecific distress may therefore similarly be emitted in response to novel experiences that are panic-relevant (e.g., elevated bodily arousal, uncued panic attack). In a similar vein, behavior-analytic models that rely primarily on verbal processes (e.g., derived relational responding, transformation of functions) have demonstrated that escape and avoidance behaviors can also develop via symbolic pathways without ever being directly paired with either aversive consequences or reinforcement (Augustson & Dougher, 1997; Dymond, Roche, Forsyth, Whelan & Rhoden, 2008; Dymond, Schlund, Roche, De Houwer & Freegard, 2012).

E. PARENTAL BEHAVIORS AND THE ACQUISITION OF PANIC-RELEVANT CONDITIONED FEAR

Scholars have suggested that most of the reviewed pathways to anxiety-relevant escape and avoidance acquisition are likely to be strengthened by parents, who generally provide their children with numerous learning experiences throughout development (Askew & Field, 2008; Fisak & Grills-Taquechel, 2007; Ollendick et al., 2001). To this end, multiple studies have documented that parents can increase fearful reactivity to panic-relevant bodily arousal in their offspring by modeling or reinforcing anxious behaviors in the context of panic-relevant symptoms (Ehlers, 1993; Leen-Feldner, Blumenthal, Babson, Bunaciu, & Feldner, 2008; Stewart et al., 2001; Watt, Stewart, & Cox, 1998; Whitehead, Busch, Heller, & Costa, 1986). For example, among a sample of undergraduate students, learning experiences involving parental modeling or reinforcement of panic-relevant symptoms were predictive of panic attack frequency

(Stewart et al., 2001). Also, relative to individuals without psychopathology, adults with PD retrospectively report more childhood experiences during which they observed their parents model fearful reactions in response to somatic symptoms (Ehlers, 1993). Moreover, parental reinforcement of somatic complaints appears to predict fearful responding to a panic-relevant biological challenge (e.g., voluntary hyperventilation) among healthy individuals (Leen-Feldner et al., 2008). In sum, an emerging literature supports the notion that parental behaviors may enhance acquisition of panic-relevant fear among offspring.

F. PARENTAL BEHAVIORS AND THE ACQUISITION OF PANIC-RELEVANT ESCAPE AND AVOIDANCE

A notable relation between parental behaviors and the development of escape and avoidance has been documented in recent years in the areas of test anxiety (Burstein & Ginsburg, 2010), social anxiety (de Rosnay, Cooper, Tsigaras, & Murray, 2006; Murray et al., 2008), and specific phobia (Dubi, Rapee, Emerton, & Schniering, 2008; Egliston & Rapee, 2007; Gerull & Rapee, 2002; Remmerswaal, Muris, & Huijding, 2013). To this end, the vicarious learning pathway that appears to enhance risk for the acquisition of conditioned fear also appears to play a role in the development of avoidance. For example, upon examining the effects of parental modeling on the development of phobia-relevant responses of toddlers, Gerull and Rapee (2002) demonstrated that after observing maternal fear responses in the presence of a novel stimulus, toddlers responded with both fear and behavioral avoidance to the novel stimulus. Similarly, Burstein and Ginsburg (2010) found that parental modeling of anxious behaviors in anticipation of a short spelling test leads to elevated anxiety and a greater desire to avoid such a task among young adolescents. Further still, de Rosnay and colleagues (2006) demonstrated that upon observing mothers modeling anxiety during a social interaction with a stranger, toddlers

responded with both fear and avoidance of the stranger. In sum, an emerging body of work suggests that a relation exists between parental modeling of anxiety and anxious responding among offspring. Accordingly, it would appear likely that the development of panic-relevant escape and avoidance may similarly be fostered via parental modeling. Unfortunately, no empirical tests to date have examined this possibility.

G. SYNTHESIS AND OVERVIEW OF THE CURRENT STUDY

Taken together, escape and avoidance behaviors play notable roles in the maintenance and possibly the development of panic-spectrum difficulties. Unfortunately, the state of the literature regarding the etiology of these emotion-regulation strategies is gravely underdeveloped particularly in areas with panic relevance. Although multiple variables likely play an important role (e.g., behavioral inhibition; Rosenbaum et al., 1988) parental behaviors may be particularly instrumental in this domain. Despite theoretical promise, empirical tests of this conceptualization have not yet been proposed. Accordingly, the aim of this investigation was to examine whether parental modeling during a biological challenge (i.e., voluntary hyperventilation) can impact the development of panic-relevant escape and avoidance among adolescents undergoing an identical challenge shortly after their parents. The use of a voluntary hyperventilation challenge (VH) in this study was important for a number of reasons. First, such procedures safely produce abrupt increases in panic-relevant bodily arousal in real-time (Bouton et al., 2001; Zvolensky & Eifert, 2001). In doing so, they cause a decrease in end-tidal carbon dioxide, elevate blood pH, and increase plasma epinephrine and lactate levels (Fried & Grimaldi, 1993). Lastly, these challenges discriminate between clinical and nonclinical populations (Rapee, Brown, Antony, & Barlow, 1992) and have been demonstrated to be safe to use with both adults and adolescents (e.g., Leen-Feldner, Feldner, Bernstein, McCormick, & Zvolensky,

2005). In sum, employing such a procedure in this study allows for closely examining the development of escape and avoidance in the context of a well-established analogue model of panic.

Multiple hypotheses regarding escape and avoidance were tested in the current study. Specifically, relative to adolescents who observed their parents complete participation in a VH (i.e., no escape modeling group), it was expected that adolescents who observed their parents prematurely terminate participation in an identical challenge (i.e., escape modeling group) would: 1) evidence a longer delay time to initiating the VH; 2) discontinue the VH more quickly; 3) evidence a lower respiration rate; and 4) be less willing to complete an additional proposed VH. These predicted results would suggest that adolescents in the escape modeling group were more avoidant of and more likely to escape panic-relevant bodily arousal elicited by the VH compared to adolescents in the no escape modeling group.

II. METHOD

A. PARTICIPANTS

Fifty adolescents (28 females) between the ages of 10-14 years ($M_{age} = 11.58$, $SD = 1.21$) and their parents (39 females) who ranged in age between 27-61 years ($M_{age} = 40.04$, $SD = 7.84$) participated in this study. Racial background of adolescents was Caucasian ($n = 43$, 86%), Asian ($n = 2$, 4%), Biracial ($n = 2$, 4%), African American ($n = 1$, 2%), and American Indian ($n = 1$, 2%). In terms of ethnicity, 12% ($n = 6$) of adolescents identified themselves as Hispanic. Data for ethnicity and racial background were missing for one case (2%). In relation to educational level, 14% ($n = 7$) of adolescents reported being enrolled in second grade, 22% ($n = 11$) reported being enrolled in third grade, 28% ($n = 14$) reported being enrolled in fourth grade, 16% ($n = 8$) reported being enrolled in fifth grade, 12% ($n = 6$) reported being enrolled in sixth grade, and 8%

($n = 4$) reported being enrolled in seventh grade. The majority of parents were currently married or cohabitating ($n = 37$, 74%) and most ($n = 43$, 86%) noted being the primary caregivers of adolescents who participated in this study. In general, parents were well educated, with 88% at least partially completing college requirements. Parents endorsed an average household income of \$54,000. Please see Table 1 for additional demographic characteristics.

Age of offspring served as the only inclusionary criterion in this study. Adolescent exclusionary criteria included: 1) chronic cardiovascular or respiratory illness (e.g., asthma, history of heart attacks); 2) acute respiratory illness (e.g., bronchitis); 3) pregnancy possibility (by self-report and parental report); 4) active psychosis; 5) history of epilepsy or seizures; 6) active suicidality; 7) current or past diagnosis of PD; 8) inability to read and write; and 9) limited mental competency and inability to give informed, written assent to participate. Parental medical and psychological difficulties were not used as exclusionary criteria given that parents did not fully undergo the biological challenge. For families with multiple eligible children, only the child whose age was closest to the middle of the recruitment age range (i.e., 12 years of age) was selected to participate in the study. Upon completion of initial phone screening, four potential participants were excluded due to meeting one or more of the aforementioned exclusionary criteria.

B. MEASURES

Screening Measures. Screening for exclusionary criteria was conducted with structured interviews. Offspring medical conditions precluding participation in this study were examined using interviews that were administered to both parents and adolescents. These interviews have been successfully employed in prior biological challenge work with adults (Bunaciu, Feldner, Babson, Zvolensky, & Eifert, 2011) and adolescents (Leen-Feldner et al., 2005). The Anxiety

Disorders Interview Schedule-IV: Child Version (ADIS-C; Silverman & Albano, 1996) was administered to adolescents to assess for the presence of PD and suicidality. This diagnostic interview has adequate psychometric properties.

Pre-challenge Measures. The *Child Anxiety Sensitivity Index* (CASI; Silverman, Fleisig, Rabian, & Peterson, 1991) was employed to measure AS, a panic vulnerability factor formally defined as the fear of anxiety-related sensations due to their perceived negative consequences (Reiss & McNally, 1985). This well-established 18-item instrument that has been validated for children and adolescents asked participants to indicate on a three-point Likert scale (1 = *none* to 3 = *a lot*) their degree of concern about the possible consequences of anxiety-related symptoms. Example items included: “It scares me when my heart beats fast,” “Unusual feelings in my body scare me,” and “When my stomach hurts, I worry that I might be really sick.”

The negative affectivity subscale (NA) of the *Positive and Negative Affect Schedule for Children* (PANAS-C; Laurent et al., 1999) was used to assess the intensity of different feelings and emotions (e.g., sad, miserable, frightened) experienced by adolescents in the weeks prior to the experiment. This subscale is comprised of 15 items that were rated on a five-point Likert scale (1 = *very slightly or not at all* to 5 = *extremely*). Preliminary research has supported the psychometric properties of this measure (Laurent et al., 1999).

The *Behavioral Inhibition/Activation Scale* (BIS/BAS; Carver & White, 1994) was employed to assess sensitivity of the behavioral inhibition system that has been posited to alert individuals of danger and motivate avoidance behavior (Gray, 1987). The BIS subscale of this measure contains 7 items (e.g., “I am very fearful compared to my friends”, “Criticism or scolding hurts me quite a bit”, “I feel worried when I think I have done poorly at something”) that were rated on a four-point Likert scale (1 = *very true* to 4 = *very false*).

The *Revised Child Anxiety and Depression Scale* (RCADS; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000) is a psychometrically-sound, 47-item self-report measure of symptoms associated with anxiety and mood disorders. Adolescents rated on a 4-point Likert scale (0 = *never* to 3 = *always*) the frequency with which each item applied to them. Only scores from the 9-item panic/agoraphobia subscale (e.g., “When I have a problem, my heart beats really fast”, “I suddenly start to tremble or shake when there is no reason for this”) were employed in the analyses.

After observing their parents appearing to undergo a VH, adolescents provided ratings of their parents’ performance by answering the following question on a 0 (*not well at all*) to 100 (*extremely well*) visual analogue scale: “How well do you think your parent followed the instructions of their breathing exercise until they stopped?”. This measure’s aim was to evaluate whether adolescents were aware of the experimental manipulation. Adolescent anticipatory anxiety was also indexed using a visual analogue scale that was administered directly before they completed the VH. This scale was represented by a horizontal line on which adolescents noted their anxiety level. Scores on this scale ranged from 0 (*no anxiety*) to 100 (*extreme anxiety*).

Outcome Measures. Four measures of panic-relevant escape and avoidance were employed in this study. *Delay time* (sec) before starting the VH was measured via an electronic timer that was turned on after the experimenter left the laboratory room and was turned off when adolescents initiated the VH. In this case, longer delay times were deemed indicative of greater behavioral avoidance of the biological challenge. *Task duration* represented the primary measure of panic-relevant escape that was employed in this study and was indexed by the total duration of time (sec) that adolescents engaged in the VH before terminating the experiment. The maximum time period that participants could continue with the VH was 3 min, with shorter

durations being indicative of stronger escape responses from the bodily arousal elicited by the VH. Similar methods to assess laboratory-based escape and avoidance in the context of distressing tasks have been employed elsewhere (Dannecker & George, 2009). A BioNomadix Respiration Transducer and a RSP100C amplifier from Biopac Systems, Inc. were used to digitally record respiration which was monitored continuously throughout the VH with a respiration belt that was attached below the participant's diaphragms. The respiration waveform was resampled to 31 samples per sec to make Band Pass digital FIR filter transformations less computationally intensive. Average *respiration rate*, calculated in breaths per minute (bpm) during the first 30 sec of the VH served as the primary physiological variable that was computed. To this end, lower respiration rates were deemed indicative of greater behavioral avoidance of the bodily arousal elicited by the VH. Immediately after adolescents completed the VH, their *willingness* to undergo a second proposed VH was evaluated using a visual analogue scale with scores ranging from 0 (*not at all*) to 100 (*absolutely*). Participants expressing minimal willingness to undergo the second proposed challenge were considered avoidant of the panic-relevant bodily arousal elicited by the VH. This index of panic-relevant avoidance has been successfully utilized in past biological challenge studies (Eifert & Heffner, 2003; Gregor & Zvolensky, 2008). Of note, adolescents did not undergo this proposed challenge.

C. PROCEDURE

Parents and children were recruited from the Northwest Arkansas community using a number of recruitment strategies (e.g., flyers, radio and internet announcements). Parents contacted the laboratory, at which time they were informed about the nature of the study and its requirements. Interested parents with an eligible adolescent underwent a brief phone screening to assess for initial exclusionary criteria. Afterwards, parents and adolescents were scheduled to

attend a two-hour laboratory visit at the University of Arkansas psychology department. At this time, verbal and written informed consent and assent were collected from parents and adolescents, respectively, who were informed about the purposes of the study, limits to confidentiality, and of the option to withdraw from the study at any time without penalty.

After informed consent and assent were collected, medical and psychological screening interviews were administered. Parents and adolescents were escorted to private rooms in the laboratory where they were asked to provide necessary demographic information. Adolescents then completed a battery of self-report questionnaires (e.g., CASI, PANAS-C, BIS/BAS-C, RCADS), while at the same time, parents completed questionnaires that were a part of a larger data collection. All self-report questionnaires were counterbalanced to control for possible order effects.

D. BIOLOGICAL CHALLENGES

Random assignment, stratified by adolescent gender, was utilized to allocate parents to one of two experimental conditions. Without adolescents being aware, one group of parents was instructed on how to prematurely discontinue a VH after 15 sec, whereas the second group of parents was instructed on how to complete participation in a VH that lasted 3 min. Parents in both conditions underwent their respective challenges with their backs facing a one-way observation mirror behind which their offspring were asked to observe them (alongside an experimenter). This procedural detail was implemented to not only mask study procedures, but also to ensure that fear-relevant information was not being conveyed. The duration of time that parents engaged in the VH was monitored via an electronic timer as an adherence check for the manipulation.

After parents completed the VH, they were escorted to another room in the laboratory. Adolescents were equipped with the respiration belt and provided ratings of their parent's performance and their anticipatory anxiety before being asked to engage in a VH themselves. Situated behind a one-way observation window, an experimenter monitored adolescent's delay time before initiating the VH and the total task duration. Importantly, parents in both conditions were asked to only appear to engage in the VH, and not actually partake in the breathing exercise. Moreover, they were instructed to refrain from making any verbal statements during the VH. These details were emphasized to ensure that the information being conveyed to adolescents was entirely related to the experimental manipulation, and was not influenced by the parent's own fearful responding to the VH. In contrast, adolescents were instructed to engage in and continue with the breathing exercise until they chose to discontinue (up to 3 min). Importantly, the VH procedures for parents and adolescents were identical in all other respects. For example, to ensure standard breathing rates of 30 respiratory cycles/min during the VH, a pre-recorded audiotape guided all participants through the breathing challenge. In addition, an experimenter read the following directional script to both parents and their offspring:

“You will now be guided through a deep breathing exercise that will ask you to breathe in and breathe out very deeply at a rate that is faster than you normally breathe. In a little bit, I will leave the room. Once I close the door, you can press the green “play” button on this audio tape at any time. The instructions on this audio tape will tell you when you should breathe in and when you should breathe out. Simply inhale when asked to “breathe in” [experimenter demonstrated], and then exhale when asked to “breathe out” [experimenter demonstrated] – making each breath in as deep as possible and each breath out as forceful as possible. This exercise will last for several minutes and can make you feel dizzy and sweaty, and it can create other feelings in your body. If you feel too uncomfortable to keep going, you can stop at any time. Just hold up this “stop” sign [provided by experimenter], press red “stop” button on the audio tape, and breathe normally again.”

Directly after adolescents completed the VH, they were asked about their willingness to participate in an additional VH. After a brief positive mood induction task was completed with

the adolescents, parents and offspring were debriefed, thanked for their participation, and provided compensation for their time (\$40/dyad).

III. RESULTS

A. MANIPULATION CHECKS

Random Assignment. Differences between groups (i.e., escape modeling, no escape modeling) on theoretically relevant, dichotomous (i.e., adolescent gender) and continuous variables (i.e., CASI, BIS, RCADS, PANAS-C, anticipatory anxiety) were examined to ensure the efficacy of random assignment. A chi-square analysis revealed no significant group differences with regards to adolescent gender [$\chi^2(1) = .41, p > .05$].

A series of independent samples *t*-tests were planned to examine possible group differences on the continuous self-report measures. However, the distribution of data from these measures was notably skewed. In spite of screening for possible outliers and conducting data transformations, results from Shapiro-Wilk tests suggested that scores for the escape modeling and/or no escape modeling groups from the CASI [$W(27) = .85, p < .01$; $W(22) = .89, p < .05$], BIS [$W(25) = .95, p > .05$; $W(18) = .90, p = .05$], RCADS [$W(24) = .75, p < .01$; $W(21) = .79, p < .05$], PANAS-C [$W(27) = .81, p < .01$; $W(22) = .79, p < .01$], and anticipatory anxiety [$W(26) = .93, p > .05$; $W(23) = .91, p < .05$] were significantly non-normally distributed. Given that the normality assumption required for conducting parametric statistics was not met, possible group differences on the continuous self-report measures were examined using non-parametric procedures (i.e., Mann-Whitney tests). These analyses revealed no significant group differences with regards to the CASI [$U = 251, p > .05$], BIS [$U = 152, p > .05$], RCADS [$U = 222.5, p > .05$], PANAS-C [$U = 272, p > .05$], or anticipatory anxiety [$U = 294, p > .05$]. Given that

random assignment appeared to be effective, none of these variables were entered as covariates in the primary analyses delineated below. Please see Table 1 for related descriptive statistics.

Modeling Manipulation. Parents' adherence to the experimental instructions was examined via direct observation by the experimenter. Results from qualitative analyses of parent's behavior during the VH suggested that all parents followed the experimental directions by: 1) engaging in the VH for the required duration (i.e., 15 sec or 3 min); 2) remaining with their backs facing the observation room in which their offspring were located; and 3) refraining from making any statements during the VH. Furthermore, no significant group differences emerged regarding adolescents' interpretation of their parents' performance. Indeed, adolescents in the escape modeling group ($Mdn = 82.0$) and adolescents in the no escape modeling group ($Mdn = 93.0$) reported that their parents engaged in the VH equally well [$U = 232, p > .05$]. Taken together, results from qualitative and quantitative analyses suggest that the modeling manipulation was effectively implemented.

B. ZERO-ORDER CORRELATIONS

Table 2 includes the means, standard deviations, and zero order correlations among the theoretically-relevant pre-challenge variables. Although no significant relations emerged with regards to adolescents' anticipatory anxiety or their interpretation of their parents' performance, CASI, PANAS-C, BIS, and RCADS scores were all significantly related to one another. Table 3 includes the means, standard deviations, and zero order correlations among the dependent variables. Contrary to expectation, dependent variables were not consistently correlated with each other. To this end, task duration was only significantly and positively related to willingness, whereas delay time was only associated with respiration rate, but not task duration or willingness.

C. PRIMARY ANALYSES

Four dependent variables were measured in this study as indices of escape from or avoidance of panic-relevant sensations: 1) *delay time* before starting the VH; 2) *task duration*; 3) *respiration rate* during the first 30 sec of the VH; and 4) *willingness* to undergo a second proposed VH. To test the hypotheses that adolescents who observed their parents prematurely terminate participation in the VH challenge (i.e., escape modeling group) would evidence greater escape from and avoidance of panic-relevant sensations compared to adolescents who observed their parents complete participation (i.e., no escape modeling group) in the same biological challenge, a multivariate analysis of variance (MANOVA) was planned. However, a MANOVA was not conducted for two primary reasons.

First, recommendations have been made against conducting MANOVAs when the dependent variables are minimally correlated or uncorrelated (Tabachnick & Fidell, 2001), as is the case with the present data. Second, the distribution of data for the dependent variables was notably skewed. In spite of screening for possible outliers and conducting data transformations, results from Shapiro-Wilk tests suggested that scores from the escape modeling and/or no escape modeling groups for delay time [$W(27) = .41, p < .01$; $W(23) = .74, p < .01$], task duration [$W(27) = .91, p < .05$; $W(23) = .21, p < .01$], respiration rate [$W(26) = .89, p < .05$; $W(21) = .86, p < .01$], and willingness [$W(27) = .81, p < .01$; $W(23) = .78, p < .01$] were significantly non-normally distributed. Finally, a Levene's test revealed significant group differences in variance with regards to task duration [$F(1, 48) = 28.03, p < .01$]. Given that the assumptions of normality and/or homogeneity of variance required for conducting parametric statistics were not met, the primary hypotheses were examined using non-parametric procedures. For the purposes of this investigation, Pearson's r was selected as the index of effect size (Cohen, 1988) with

small, medium, and large effect sizes being represented by r -values of .10, .30, and .50 respectively.

A series of Mann-Whitney tests were conducted with parental modeling (i.e., escape, no escape) as the between-groups variable, and panic-relevant escape and avoidance indices as the dependent measures. First, no significant differences emerged between the two groups in terms of delay time before initiating the VH [$U = 261, p > .05$]. Indeed, adolescents in the escape modeling group ($Mdn = 2$ sec) and the no escape modeling group ($Mdn = 2$ sec) evidenced a comparable delay time to starting the biological challenge. Second, consistent with prediction, significant differences were observed with regards to task duration [$U = 75, p < .001, r = .70$], such that adolescents in the escape modeling group ($Mdn = 96$ sec) discontinued the VH notably sooner than adolescents in the no escape modeling group ($Mdn = 180$ sec). Third, as evidenced by nearly identical respiration rates between adolescents in the escape modeling group ($Mdn = 27.28$ bpm) and adolescents in the no escape modeling group ($Mdn = 27.40$ bpm), no significant differences emerged in terms of respiration rate [$U = 272, p > .05$]. Finally, adolescents in the escape modeling group ($Mdn = 81$) were not significantly less willing to complete the second proposed VH relative to adolescents in the no escape modeling group [$Mdn = 89; U = 272, p > .05$]. Please see Table 1 for additional descriptive statistics.

IV. DISCUSSION

Escape and avoidance behaviors are widely implicated in the maintenance of panic-spectrum difficulties and they may also play an important role in the development of such problems. Although a burgeoning literature suggests that parental behaviors are instrumental in the development of such emotion regulation strategies in the context of other anxiety difficulties (e.g., Burstein & Ginsburg, 2010; Dubi et al., 2008; Remmerswaal et al., 2013), empirical tests

have yet to examine the pathways via which parents may exert such risks in areas with panic relevance. Accordingly, the current study proposed an initial experimental examination of whether parental modeling of defensive reactivity could influence panic-relevant escape and avoidance among offspring. Healthy adolescents were asked to engage in a VH shortly after being randomly assigned to observe their parents either: a) complete participation in a VH (no escape modeling group) or b) prematurely terminate a similar procedure (escape modeling group). Panic-relevant escape and avoidance behaviors among adolescents were indexed using a multimodal assessment. It was hypothesized that relative to adolescents in the no escape modeling group, adolescents in the escape modeling group would respond with greater escape from, and avoidance of, panic-relevant sensations. Results were partially consistent with hypotheses. Indeed, although a significant difference was noted with regards to escape responding, the two groups responded similarly in terms of delay time to starting the VH, respiration rate, and willingness to engage in another breathing exercise.

Consistent with hypotheses, adolescents in the escape modeling group discontinued the VH much sooner than adolescents in the no escape modeling group. Offspring who observed their parents terminate the VH prematurely discontinued their own challenge on average, more than 70 sec sooner compared to offspring who observed their parents complete the VH. Indeed, 22 of the 27 adolescents in the escape modeling group (cf., 1 out of 27 in the no escape modeling group) prematurely discontinued the VH, and 70% of them endorsed doing so specifically as a result of experiencing panic-relevant sensations (e.g., dyspnea, parasthesia, tachycardia). This effect was large in magnitude ($r = .70$) and especially noteworthy given that VH procedures typically elicit relatively minimal levels of fear-based responding among healthy adolescents (Leen-Feldner et al., 2006) and adults (Rapee et al., 1992). Importantly, this effect cannot be

accounted for by group differences in demographic characteristics (i.e., gender), theoretically-relevant pre-challenge variables (i.e., anxiety sensitivity, negative affectivity, behavioral inhibition, and anticipatory anxiety), or parental adherence to experimental instructions. In addition, these differences in task duration are not attributable to a failure on the part of adolescents to adhere to the biological challenge instructions. After all, the respiration rate for both groups was not only almost identical, but also very comparable to the standard respiration rate (i.e., 30 bpm) expected to be achieved in such biological challenge work. Instead, the noted difference in task duration appears to be in general agreement with previous research demonstrating the impact of parental modeling on the fearful and avoidant responses of offspring (e.g., Burstein & Ginsburg, 2010; de Rosnay et al., 2006; Gerull & Rapee, 2002). In addition, this finding provides a noteworthy extension to the literature as it represents the first experimental demonstration of the role of parental behaviors on panic-relevant offspring responding broadly, and escape behaviors specifically.

Interestingly, in contrast to prediction, there were no significant group differences in terms of any of the panic-relevant avoidance indices. Importantly, given the nearly identical responding with regards to delay time before initiating the VH ($M_{Escape} = 3.11$, $M_{No\ escape} = 4.26$), respiration rate ($M_{Escape} = 27.58$, $M_{No\ escape} = 27.97$), and self-reported willingness to engage in another VH ($M_{Escape} = 73.59$, $M_{No\ escape} = 74.69$), it is not likely that insufficient methodological power resulted in the absence of statistically significant group differences on these measures. These null findings are interesting not only because they appear to run contrary to existing evidence linking parental modeling to anxious offspring responding, but also because they are inconsistent with the aforementioned pattern of results with respect to escape acquisition.

The measures of panic-relevant avoidance employed in this study must be briefly discussed as they represent one potential reason for the conflicting set of results. The three measures of avoidance were not correlated with each other as predicted, thereby suggesting poor convergent validity. To this end, when considering each of these measures, both the lack of relation between these variables and the lack of group differences are not particularly surprising. First of all, neither delay time nor respiration rate have previously been employed as indices of avoidance in the context of a VH. Accordingly, in the absence of appropriate psychometric investigations, the reliability and validity of these measures are questionable. Second, given that a large majority of adolescents in the escape modeling group discontinued the challenge prematurely, respiration rate could only be calculated for the first 30 sec of the exercise, thereby possibility limiting variability needed to document any potential group-based differences. Finally, while some studies have noted that expressed willingness to engage in a second biological challenge is associated to both post-challenge anxiety ratings (Gregor & Zvolensky, 2008) and actual willingness to engage in such exercises (Eifert & Heffner, 2003), evidence for the validity of this measure is not uniform (Dannecker & George, 2009). Importantly, although the measures employed in this study may account for the documented null results, another likely explanation centers on the nature of the manipulation employed.

Prior relevant research that has evaluated the effect of parental modeling on offspring fear and avoidance acquisition has primarily relied on two pathways: vicarious learning and verbal transmission of information, both of which are firmly grounded in the same associative learning principles underlying classical conditioning (Field, 2006). Typical experimental manipulations in this area have required parents to specifically model fear-eliciting emotions by using facial expressions or vocalizations in the context of novel tasks (Burstein & Ginsburg,

2010), stimuli (Gerull & Rapee, 2002), or strange persons (de Rosnay et al., 2006). In contrast, the present study aimed to limit learning opportunities occurring via such channels by explicitly urging parents to refrain from making any vocalizations or visible facial expressions during the VH. Indeed, rather than asking parents to convey fearful emotions, parents in the escape modeling group were asked to only model fear-relevant escape behaviors. As a result, it is possible that group differences in terms of avoidance responding did not emerge because the modeling manipulation specifically targeted escape responses and was also devoid of any fear and avoidance-eliciting stimuli. It is therefore possible that if avoidance responses had been modeled in the context of the VH, the corresponding hypothesized group-based differences may have also emerged.

As aforementioned, the large effect documented herein with regards to panic-relevant escape cannot be attributed to the parent's affective responses during the VH. Indeed, given that no such responses were emitted during the course of the manipulation, the current effect was likely exerted through other channels. The process of operant conditioning provides one such possibility. As noted earlier, escape and avoidance learning may be fostered directly by positive reinforcement of fearful responses during the course of one's developmental history (Ollendick et al., 2001). However, positive reinforcement likely also plays a vital role during observational learning whereby a model's behavior can set the stage for an imitative response to result in reinforcement. To this end, it is well-established that children are from an early age prompted by parents and other individuals to imitate a variety of behaviors. When correct imitative responses are emitted, reinforcement is provided, whereas when incorrect responses are emitted, reinforcement is withheld. In this fashion, children imitate numerous modeled behaviors that have typically resulted in reinforcement. Importantly, after many such episodes, children can

also emit novel imitative behaviors that have never before been modeled or reinforced, and it is this concept of generalized imitation (Baer & Sherman, 1964) that may be most relevant to understanding the results of the current investigation. Indeed, despite that none of the present participants had any prior experience with VH procedures, it is relatively safe to assume that adolescents had a lengthy history of being reinforced for imitating their parent's behavior. Accordingly, in spite of never having previously observed their parents engage in a VH procedure, adolescents may have responded in accordance with their parents' behavior because doing so had on numerous other occasions resulted in reinforcement.

Regardless of whether generalized imitation does indeed underlie the current pattern of results, the present study not only represents an important first step en route to forwarding our understanding of variables involved in the intergenerational transmission of panic-related difficulties, but also has important implications for etiological models of PD. After all, if over the course of development children learn to repeatedly escape situations associated with panic-relevant bodily arousal, they would likely fail to appreciate the benign nature of such experiences and the fact no defensive responses are typically required in their presence. By doing so, they would likely be at increased risk for developing PD by misinterpreting somatic sensations as signs of impending danger (Clark, 1986; Reiss, 1991) and ultimately responding fearfully in the event of a later full-blown panic attack (Bouton et al., 2001). Importantly, although the current study uniquely extends the existing literature implicating parental behaviors in the development of panic-related offspring problems, a variety of research questions remain to be addressed.

First of all, future research should evaluate the robustness of the documented effect of parental modeling on panic-relevant behavior by conducting additional VH procedures either in the context of the same laboratory visit, or over the course of several weeks. Doing so would

allow for evaluation of retention effects with regards to task duration, and it would likely also provide a more appropriate avenue for indexing avoidance responding. Another interesting area of research pertains to whether parental modeling can serve not only as a risk factor, but also as a protective factor that interferes with offspring responding. After all, children whose mothers modeled positive behaviors in the context of a fear-eliciting stimulus were found to be at decreased risk for exhibiting fear and avoidance in response to the fear-eliciting stimulus (Egliston & Rapee, 2007). An additional domain that deserves further exploration pertains to the model engaging in panic-relevant behaviors. More specifically, while the current study documented that parents in general play a notable role on offspring panic-relevant responding, future research should also examine whether parental gender moderates this effect, as has been demonstrated in some previous work (Burstein & Ginsburg, 2010). To this end, some time could be spent evaluating whether individuals outside of the family (e.g., siblings, peers, strangers) can also serve as adequate models for fearful offspring responding. Given the design of the current study, the impact of parental behaviors on challenge-related anxiety could not be examined in the present investigation. This aspect of fearful responding, along with more refined evaluations of avoidance, deserves further exploration. Future research could also usefully evaluate whether panic-relevant offspring responding can be impacted by parents via additional pathways (e.g., emotional or verbal transmission of information). Lastly, while this particular study explicitly evaluated the impact of parental behaviors on offspring responding, a similarly interesting idea would be to evaluate the bidirectional relation that likely exists between parenting behaviors and fearful responding among offspring.

At least two limitations must be noted to assist with interpretation of the current results.

First of all, the sample employed in this study consisted primarily of healthy, middle-class,

Caucasian individuals, thereby notably limiting the generalizability of these results to those who share these demographic characteristics. For this reason, future work in this area must aim to apply the results of this investigation to a more heterogeneous population. Second, the current study employed a well-established biological challenge procedure as a means to elicit panic-relevant-responding in “real-time.” Although highly relevant at this stage in the research process, it must be noted that such challenges cannot speak directly to the development of psychopathology. Accordingly, longitudinal research that examines the relation between parental modeling and offspring fearful responding is necessary alongside such laboratory-based methodologies.

In conclusion, despite the aforementioned limitations, the present study sheds important light on processes involved in the intergenerational transmission of anxiety-related difficulties. Indeed, the findings outlined herein uniquely advance the existing research base by providing the first experimental demonstration that parental modeling can affect panic-relevant escape responding among offspring via pathways that do not rely on classical conditioning-based explanations. Although multiple hypotheses were not supported and a plethora of questions remain, these findings leave researchers well positioned to better understand parenting-related behaviors involved in the development of prominent emotion-regulation strategies. Importantly, an improved understanding in this area may ultimately have noteworthy implications in the development of future prevention programs targeting parents and at risk youth.

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Table 1
 Descriptive Data for Demographic Variables, Theoretically-Relevant Pre-Challenge Variables,
 and Dependent Variables as a Function of Group

	Group	
	Escape M or n (SD or %)	No Escape M or n (SD or %)
<i>Adolescent Demographics</i>		
Age	11.44 (1.08)	11.73 (1.35)
Gender	14 (51.9%)	14 (60.9%)
Racial Background		
Caucasian	25 (96.2%)	18 (78.3%)
Asian	0 (0%)	2 (8.7%)
Biracial	0 (0%)	2 (8.7%)
African American	0 (0%)	1 (4.3%)
American Indian	1 (3.8%)	0 (0%)
Ethnicity		
Hispanic	2 (7.7%)	4 (17.4%)
Not Hispanic	24 (92.3%)	19 (82.6%)
Education Completed		
Second grade	5 (18.5%)	2 (8.7%)
Third grade	6 (22.2%)	5 (21.7%)
Fourth grade	9 (33.3%)	5 (21.7%)
Fifth grade	4 (14.8%)	4 (17.4%)
Sixth grade	2 (7.4%)	4 (17.4%)
Seventh grade	1 (3.7%)	3 (13.0%)
<i>Parent Demographics</i>		
Age	41.07 (8.27)	38.82 (7.30)
Gender	23 (85.2%)	16 (69.6%)
Family Income	\$ 57,615	\$ 50,272
<i>Theoretically-Relevant Variables</i>		
CASI	26.0 (6.62)	27.18 (6.26)
PANAS-C	26.40 (11.06)	23.72 (7.79)
BIS	16.92 (4.50)	14.66 (4.44)
RCADS	2.54 (3.23)	2.95 (3.18)
Anticipatory Anxiety	37.69 (28.40)	36.52 (27.79)
Ratings of Parents' Performance	77.70 (19.13)	85.73 (16.77)
<i>Dependent Variables</i>		
Delay Time	3.11 (4.61)	4.26 (4.39)
Task Duration	104.07 (54.93) **	174.91 (24.39) **
Respiration Rate	27.58 (2.93)	27.97 (2.56)
Willingness	73.59 (29.23)	74.69 (31.08)

Note: Total $n = 50$. n for gender reflects women. CASI = Child Anxiety Sensitivity Index; PANAS-C = Negative Affect Scale of the Positive and Negative Affect Schedule for Children; BIS = Behavioral Inhibition Scale; RCADS = Panic/Agoraphobia Scale of the Revised Child Anxiety and Depression Scale. ** $p < .01$

Table 2
Descriptive Data and Zero-Order Relations between Pre-Challenge Variables

	M (SD)	1	2	3	4	5	6
1. Rating of Parents' Performance	81.40 (18.35)	--	.03	-.10	-.25	.03	-.19
2. Anticipatory Anxiety	37.14 (27.83)		--	.23	.04	.17	.27
3. CASI	26.53 (5.88)			--	.50**	-.51**	.72**
4. PANAS-C	25.20 (9.72)				--	-.36*	.49**
5. BIS	15.97 (4.56)					--	-.31*
6. RCADS	2.73 (3.17)						--

Note. $n = 50$.

CASI = Child Anxiety Sensitivity Index; PANAS-C = Negative Affect Scale of the Positive and Negative Affect Schedule for Children; BIS = Behavioral Inhibition Scale; RCADS = Panic/Agoraphobia Scale of the Revised Child Anxiety and Depression Scale.

* $p < .05$; ** $p < .01$

Table 3
Descriptive Data and Zero-Order Relations between Dependent Variables

	M (SD)	1	2	3	4
1. Delay Time	3.64 (4.50)	--	.11	-.00	.02
2. Task Duration	136.66 (56.03)	.30	--	-.18	.52*
3. Respiration Rate	27.75 (2.75)	.49**	.34	--	.19
4. Willingness	74.10 (29.79)	.26	.31	.01	--

Note. $n = 50$. Correlations for adolescents in the escape modeling group are presented above the diagonal, and correlations for adolescents in the no escape modeling group are presented below the diagonal. Means and standard deviations for the entire sample are presented.

* $p < .05$; ** $p < .01$.

Figure Captions

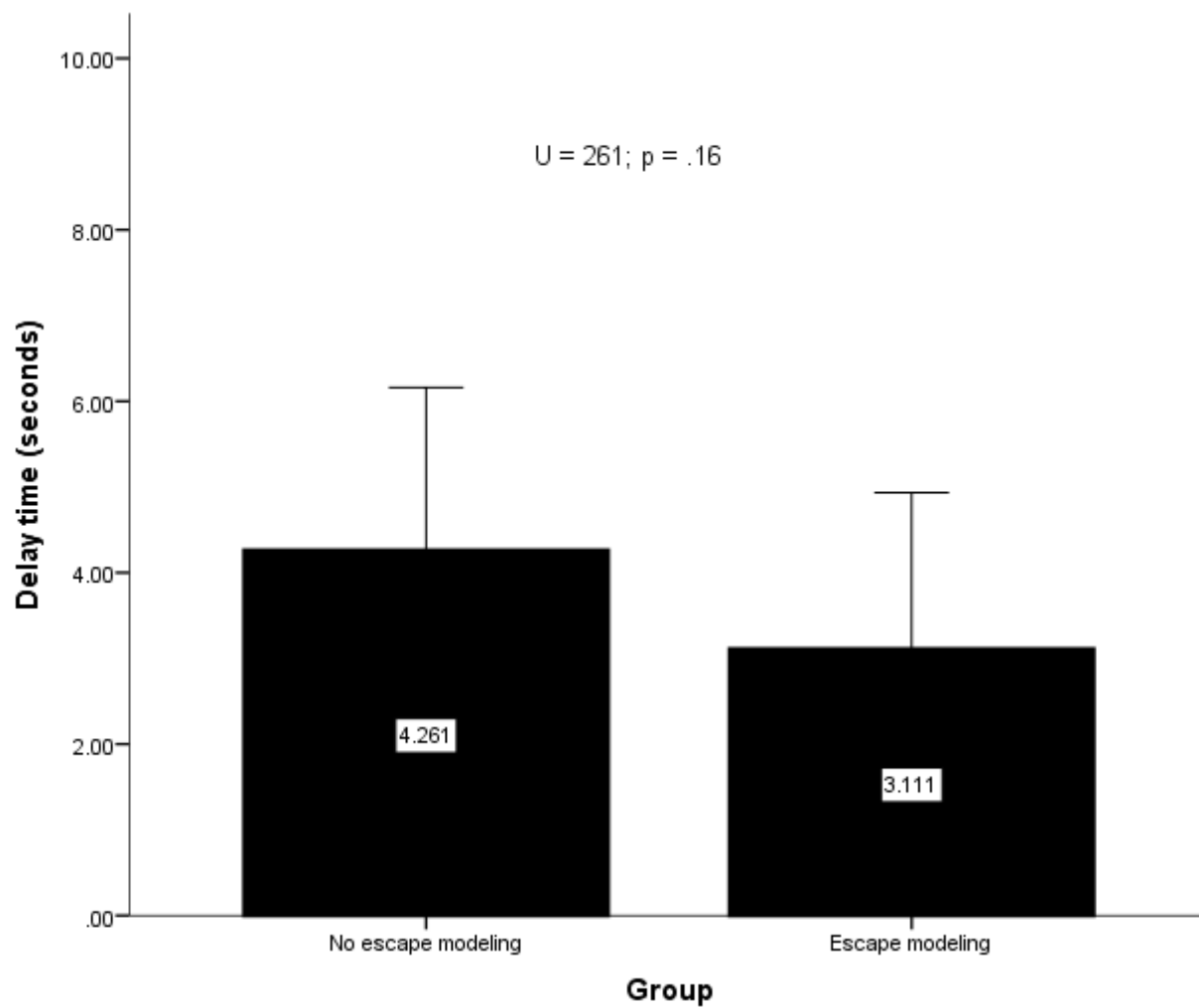
Figure 1. Mean delay time before adolescents started the voluntary hyperventilation challenge.

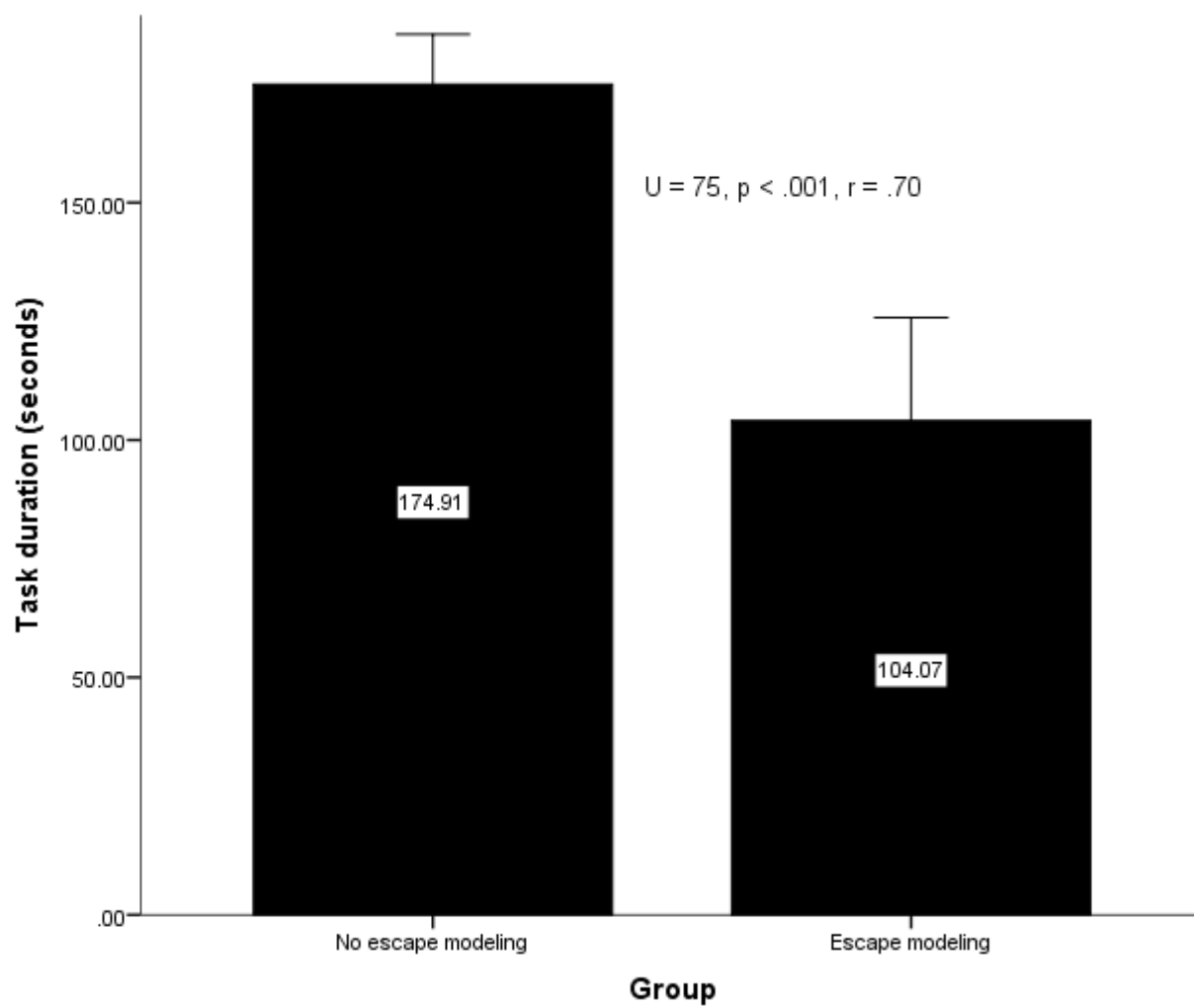
Error bars reflect 95% confidence intervals.

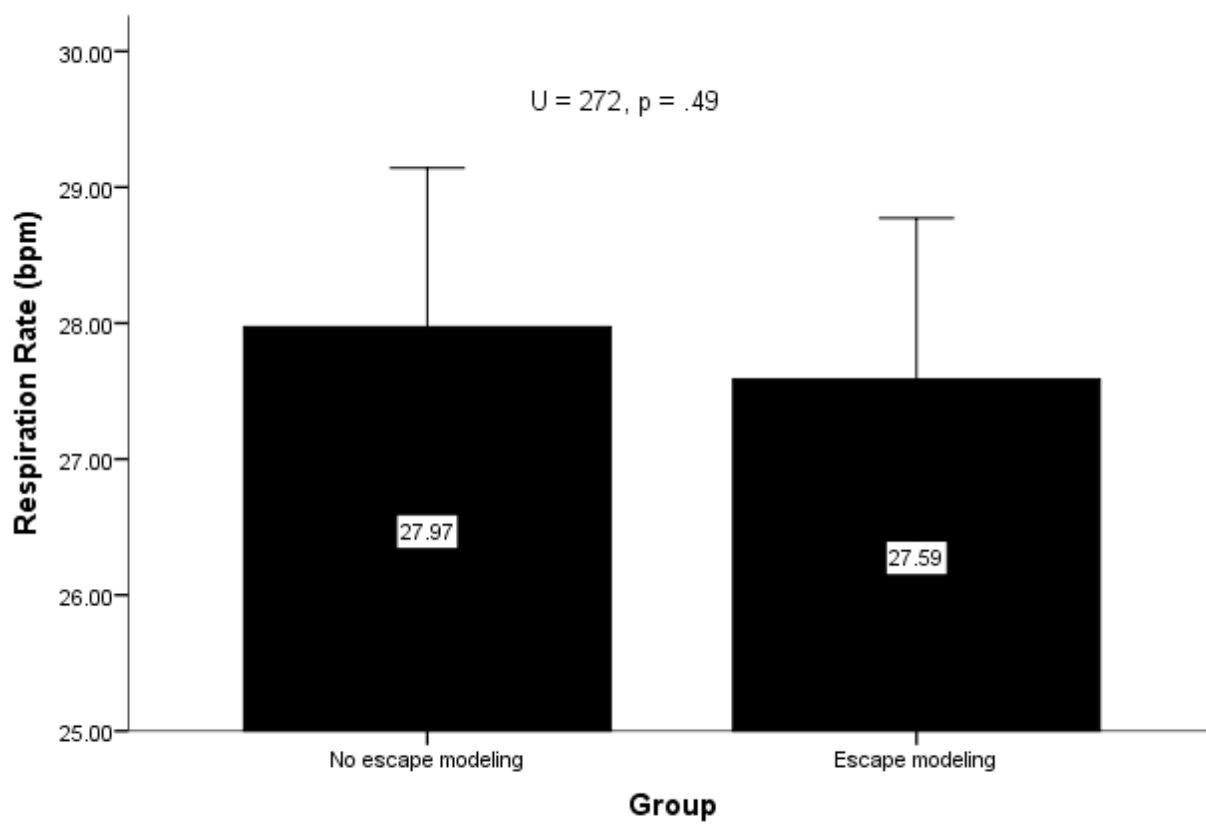
Figure 2. Mean task duration for the adolescents. Error bars reflect 95% confidence intervals.

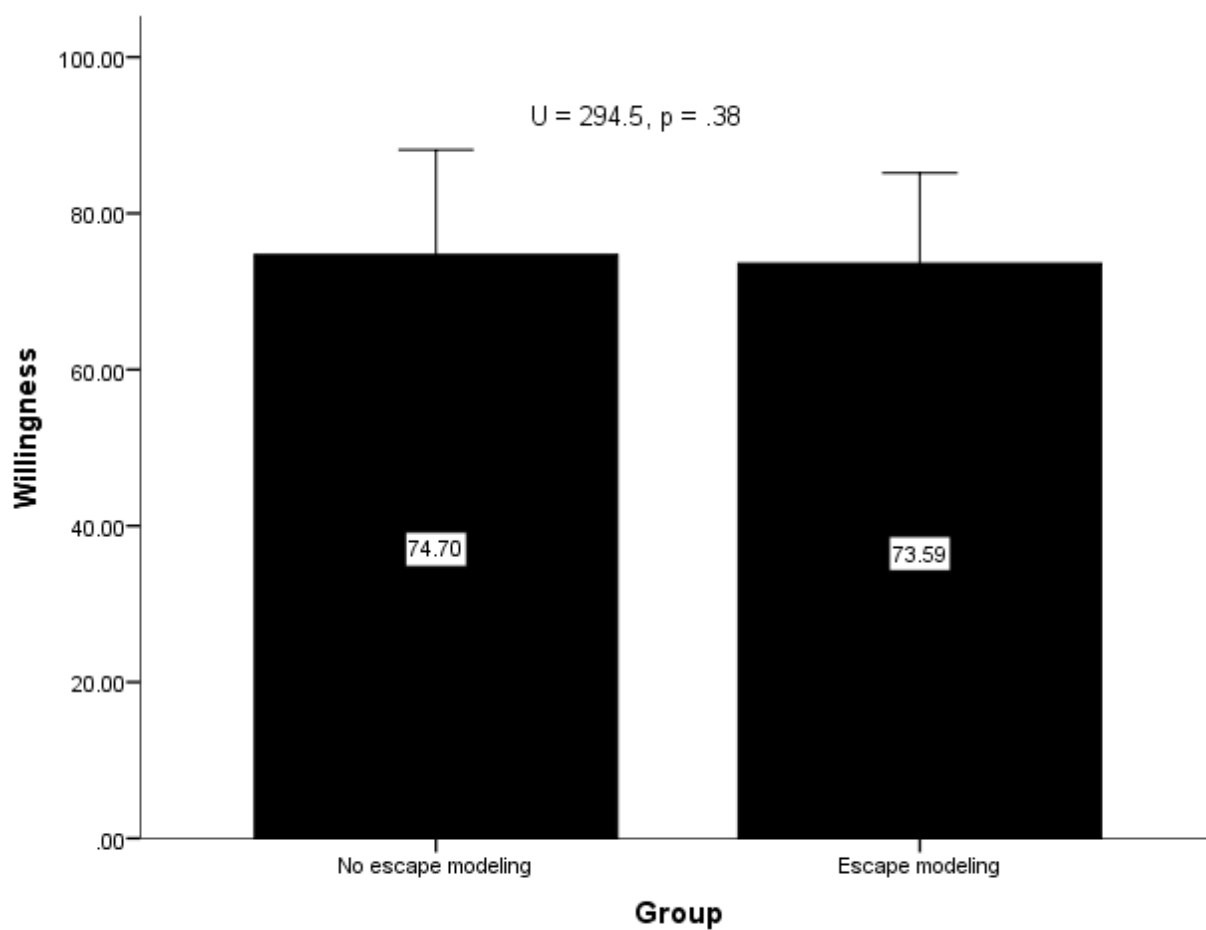
Figure 3. Mean respiration rate during the first 30 sec of the voluntary hyperventilation challenge. Error bars reflect 95% confidence intervals.

Figure 4. Mean expressed willingness to participate in a second voluntary hyperventilation challenge. Error bars reflect 95% confidence intervals.









December 22, 2011

MEMORANDUM

TO: Liviu Bunaciu
Matthew Feldner

FROM: Ro Windwalker
IRB Coordinator

RE: New Protocol Approval

IRB Protocol #: 11-12-331

Protocol Title: *Parent-Adolescent Modeling*

Review Type: EXEMPT EXPEDITED FULL IRB

Approved Project Period: Start Date: 12/16/2011 Expiration Date: 12/08/2012

Your protocol has been approved by the IRB. Protocols are approved for a maximum period of one year. If you wish to continue the project past the approved project period (see above), you must submit a request, using the form *Continuing Review for IRB Approved Projects*, prior to the expiration date. This form is available from the IRB Coordinator or on the Research Compliance website (<http://vpred.uark.edu/210.php>). As a courtesy, you will be sent a reminder two months in advance of that date. However, failure to receive a reminder does not negate your obligation to make the request in sufficient time for review and approval. Federal regulations prohibit retroactive approval of continuation. Failure to receive approval to continue the project prior to the expiration date will result in Termination of the protocol approval. The IRB Coordinator can give you guidance on submission times.

This protocol has been approved for 80 participants. If you wish to make *any* modifications in the approved protocol, including enrolling more than this number, you must seek approval *prior to* implementing those changes. All modifications should be requested in writing (email is acceptable) and must provide sufficient detail to assess the impact of the change.

If you have questions or need any assistance from the IRB, please contact me at 210 Administration Building, 5-2208, or irb@uark.edu.