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The Role of Early Experience and Cognitive Vulnerability: Presenting a Unified Model of the Etiology of Panic

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Abstract Cognitive vulnerability models have been developed to explicate the etiology of panic and other anxiety disorders. This study takes a step forward by presenting a unified vulnerability model that incorporates a continuum of proximal and distal factors involved in the etiology of panic. The present study tested distal elements of the model, including childhood histories of vicarious and instrumental learning, and cognitive constructs such as anxiety sensitivity and perceived control. Our study found that parental modeling of the dangerousness of anxiety symptoms accounted for more model variance than from direct experiences with arousal-reactive symptoms or from parental reinforcement of the child's own sick role behavior when experiencing arousal reactive symptoms. We found that parental modeling independently predicted model variance even when perceived control was included in the model. Our results indicated that low perceived anxiety control and anxiety about bodily symptoms uniquely accounted for variance in the model. Our findings add to the growing body of research and suggest that anxiety about bodily symptoms and low perceived control together may interact to create a stronger distal vulnerability construct for panic than either construct alone in

individuals whose childhood caregivers may have modeled fear of anxiety symptoms. The results of this study provide support for the inclusion of distal factors in unified cognitive vulnerability models of panic disorder as well as for future prospective research of these constructs.

Keywords Panic · Anxiety sensitivity · Perceived control · Cognitive vulnerability · Etiology · Learning models

Introduction

A large body of research has demonstrated the importance and implications of cognitive vulnerability in the field of depression and other mood disorders (e.g., Abramson et al. 2002; Beck 1964, 1987; Calvete et al. 2013; Cukor and McGinn 2006; Hamilton et al. 2013; Ingram et al. 1998; McGinn et al. 2005). Research has also been conducted to investigate cognitive vulnerability factors in the etiology and maintenance of anxiety disorders (e.g., Chorpita et al. 1998; McGinn et al. 2005, 2010; Riskind and Williams 2005; Riskind et al. 2013). Emerging research has focused on testing specific cognitive vulnerability models of panic disorder (PD; e.g., Bentley et al. 2013; Gregor and Zvolensky 2008; Li and Zinbarg 2007; Schmidt et al. 2006, 2010; Teachman 2005) although some previous studies examining specificity have failed to observe a link between panic disorder patients and individuals with other anxiety disorders (Hofmann et al. 2000).

Cognitive behavioral models of PD contain five main facets that help us understand vulnerability to PD (Barlow 1988, 2002; Beck 1987; Beck et al. 1985; Clark 1986, 1988; McNally 2001; Reiss 1991; Reiss and McNally 1985; Schmidt and Woolaway-Bickel 2005; Teachman

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2005). Taken together, these models include: (a) a proximal description of the sequence of events that lead to panic (e.g., catastrophic cognitions); (b) information-processing biases (attention, memory, and interpretive biases) that operate within the vulnerable individual; (c) pre-dispositional trait variables or beliefs that are hypothesized to confer distal cognitive vulnerability (anxiety sensitivity, low perceived control, general vulnerability to harm beliefs); (d) early environmental contributions that create a cognitive blueprint for later pathological functioning (e.g., experiences with unpredictability, uncontrollability); and (e) genetic and temperamental factors that confer a biological vulnerability.

All models stress the fear of bodily sensations of anxiety but vary with regard to the proximity of the espoused vulnerability factor to panic onset. Individually, these models emphasize some aspects over others. For example, Clark's cognitive model emphasizes proximal catastrophic cognitions while the expectancy model emphasizes the pre-dispositional trait of anxiety sensitivity (Clark 1986, 1988; Reiss 1991; Reiss and McNally 1985). Some models suggest that a conscious catastrophic misappraisal is necessary for panic onset (Clark 1988) while others propose that panic may occur without misappraisals (McNally 1994) or even conscious appraisals (Barlow 1988, 2001). Most of the models propose that biological and environmental factors make individuals vulnerable to developing panic but vary with regard to the types of factors they propose as bestowing vulnerability. Some propose environmental origins of pathological anxiety (Barlow 1988, 2002; Beck 1987; Beck et al. 1985) while others merely elaborate on the cognitive processes that confer vulnerability (Clark 1988).

Unified Vulnerability Model of Panic

Our unified vulnerability model of panic attempts to integrate the various cognitive vulnerability models proposed to explain the etiology and maintenance of PD (Barlow 2000, 2002; Beck 1987; Clark 1986, 1988; Cox 1996; Reiss and McNally 1985) (see Fig. 1). Centrally, our model incorporates Reiss and McNally's expectancy theory of anxiety sensitivity and Barlow's triple vulnerability model, which emphasizes both anxiety sensitivity and low perceived control as distal vulnerability factors in the development of panic. The unified vulnerability model also incorporates Clark's proximal model of panic, which suggests that catastrophic misappraisals of bodily sensations as threatening go on to trigger panic. It also derives from the general cognitive model of anxiety, which proposes that dysfunctional schemas arise from early childhood experiences and provide a template for processing future experiences (e.g., Beck 1976; Beck et al. 1985; Young 1999).

Our unified model builds on the integrated model proposed by Schmidt and Woolaway-Bickel (2005), which elegantly organizes the different models on a continuum of distal to proximal vulnerability factors, akin to the model first explicated by Ingram et al. (1998) in their description of diathesis–stress models of depression. Our model incorporates other distal influences and refinements on this vulnerability continuum. For example, our model incorporates into the continuum, distal influences proposed by earlier models such as evolutionary preparedness (sensitivity to certain stimuli or conditions/fight/flight response) and genetic/biological influences (e.g., genetic, temperament). Additionally, our unified model also suggests that informational biases (e.g., interpretive, attentional, or memory biases) arise from dispositional traits or beliefs (e.g., anxiety sensitivity; Teachman 2005), and hence are closer in proximity to panic onset.

Further, although Schmidt and Woolaway-Bickel (2005) implicate learning histories in the evolution of distal cognitive vulnerability constructs, our model explicitly incorporates both aversive events (e.g., a spontaneous panic attack) and other early experiences with anxiety (e.g., vicarious learning via parental response to panic) and places them into the theoretical continuum of vulnerability for panic onset. This continuum includes a range of early environmental experiences previously theorized to contribute to distal cognitive vulnerability factors, including adverse or unpredictable events, instrumental and/or vicarious learning with anxiety symptoms, or early incidents with over-control and over-protection. In sum, our unified vulnerability model presents a continuum of distal to proximal vulnerability factors segmented into six stages, which incorporate the different elements of panic vulnerability proposed by earlier theorists. Figure 1 depicts the vulnerability factors highlighted by our unified vulnerability model of PD as they lie on a distal to proximal continuum.

Empirical Basis for Anxiety Sensitivity in the Development of PD

Anxiety sensitivity has been the one of the most widely studied and reported to be one of the most robust predictors of panic onset (e.g., Plehn and Peterson 2002; Schmidt et al. 1997, 1999; Zinbarg et al. 2001). However, findings in the literature are mixed and anxiety sensitivity also appears to be predictive of broader anxiety pathology in general, suggesting that the relationship between anxiety sensitivity and panic may not be specific (Teachman 2005; Zvolensky and Forsyth 2002).

Fewer studies have examined related constructs, such as low perceived control, separately or in combination with other cognitive constructs such as anxiety sensitivity (Bentley et al. 2013; Schmidt et al. 2000). In support of

Fig. 1 Unified cognitive vulnerability model of panic—proximal to distal continuum of vulnerability factors

Barlow's triple vulnerability model, a recent study found that both perceived control (general psychological vulnerability) and anxiety sensitivity (specific psychological vulnerability) uniquely predicted panic and that anxiety sensitivity exerted greater effects on panic symptoms in individuals with lower perceived control (Bentley et al. 2013). However, more studies are needed to replicate these findings and to examine the unique or additive effects of other cognitive constructs (e.g., proximal catastrophic cognitions) theorized to lead to the development and maintenance of panic attacks.

Empirical Basis of Childhood Learning History as a Distal Vulnerability to PD

Although cognitive models emphasize the importance of the environmental factors in the development of anxiety disorders (e.g., Beck et al. 1985; Chorpita and Barlow 1998), and despite the fact that a number of studies have found evidence of family transmission of risk of anxiety disorders (Rapee 2002), only a small number of studies have tested these relationships in the context of PD (e.g., Schmidt et al. 2000). Despite the fact that different types of learning processes are believed to confer vulnerability (e.g., classical vs. instrumental paradigms), few studies have directly examined the types of learning experiences that may lead to a cognitive vulnerability or investigated the relationships between learning experiences, cognitive vulnerability factors, and PD (Ehlers 1993; Schmidt et al.

2000; Stewart et al. 2001; Watt and Stewart 2000; Watt et al. 1998; Zvolensky et al. 2005).

There have been only a handful of studies investigating childhood learning histories (CLH) as a distal cognitive vulnerability to panic. In an early study, Ehlers (1993) employed a modified version of the Learning History Questionnaire (LHQ; Whitehead et al. 1986) to investigate the relationship between child and adolescent learning experiences and the development of panic attacks in a clinical sample. Ehlers (1993) showed that in comparison to controls, panic groups reported more instrumental and vicarious learning from parental modeling of sick role behavior in response to anxiety symptoms, and reinforcement of the subject's own sick-role behavior in response to anxiety but not cold symptoms. Additionally, anxiety but not cold scales of the LHQ were significantly associated with scores on the Body Sensations Questionnaire (BSQ), a measure of the degree of fear of panic related bodily sensations, for the panic group. These results support the stressor-trait congruency aspect of the cognitive vulnerability model in that only CLH involving parental reinforcement and modeling of anxiety symptoms, but not other illnesses, were associated with BSQ scores.

Both Watt et al. (1998) and Stewart et al. (2001) found differences between high and low anxiety sensitivity participants in both the anxiety and cold symptoms scales of the LHQ whereas Ehlers' (1993) found that high and low anxiety sensitivity participants did not differ on the cold

scale. This discrepancy regarding the specificity of the origins of anxiety sensitivity may possibly be accounted for by the fact that the studies used different measures to assess current fear of anxiety symptoms. In other words, the BSQ used in the Ehlers' study may be a more precise measure of the fear of anxiety sensations whereas the ASI may measure a more general fear of somatic symptoms.

Objectives of Present Study

Building on previous research (Ehlers 1993; Stewart et al. 2001; Watt et al. 1998), the present study examines specific aspects of the unified vulnerability model by investigating: (a) the relationship of CLH to anxiety specific symptoms and panic severity in adulthood, and (b) anxiety related cognitions as mediators in this relationship. According to cognitive models of panic, a CLH involving the dangerousness of anxiety symptoms should lead to the acquisition of beliefs about those symptoms as dangerous, and in so doing, increase the frequency and severity of panic attacks (Barlow 2002; Ehlers 1993).

Parent modeling may increase the likelihood that children will experience physical symptoms of anxiety as threatening and believe they have low control over these symptoms. For example, parental transmission may include parent's expressing or modeling fears regarding bodily sensations, which in turn, gives rise to children believing that bodily sensations are a source of uncontrollable threat. In line with prior theories and findings (e.g., Bentley et al. 2013) encompassed within our unified model of panic, the present study proposes that anxiety sensitivity and low perceived control are integral in mediating the relationship between CLH and panic symptoms.

Study Hypotheses

Our unified vulnerability model integrated cognitive vulnerability constructs along the proximal–distal continuum pertaining to PD (see Fig. 1), from which we developed three specific hypotheses.

First, we predicted that a CLH of vicarious (parental modeling of anxiety symptoms) and instrumental learning (parental reinforcement of anxiety symptoms), both measured by the Learning History Questionnaire (LHQ), would be associated with adult panic symptoms as measured by the Panic Attack Questionnaire-Revised (PAQ-R).

Second, we anticipated that greater anxiety sensitivity (ASI), bodily sensations associated with anxiety (BSQ), and lower perceived control (ACQ) would be associated with adult panic symptoms as measured by the PAQ-R.

Third, we examined how ASI, BSQ, and ACQ may moderate the relationship between CLH and adult panic

symptoms. Specifically, we hypothesized that ASI, BSQ, and ACQ would each uniquely account for a significant proportion of model variance in the relationship between parental modeling and reinforcement of childhood anxiety and adult panic symptoms. We also examined two-way interactions to see if the relationship between CLH and adult panic symptoms differed depending on the level of the moderators.

Methods

Participants

We present data on 129 participants recruited from private business offices in the New York area. Volunteers ranged in ages between 18 and 77. Seventy-seven percent of the participants were female. The average age was 42.5 years ($SD = 13.2$). Twenty-two and a half percent of the respondents were single, 60.5 % married or cohabitating, and 14.7 % divorced, separated, or widowed. All participants who responded completed high school with years of education ranging from 12 to 21 years. The mean years of education was 15.3 ($SD = 2.6$). Demographic information from the present sample may be found in Table 1.

Procedure

Participants were contacted during the months of September, 2005 through March, 2006 via a contact person in each office who distributed the questionnaire to every employee in that business. Participants who agreed to participate filled out a packet of self-report measures that included a

Table 1 Sample Demographics ($N = 129$)

	<i>M (SD)</i>
Age (years)	42.5 (13.2)
	(%)
Gender	
Male	22.5
Female	76.0
Marital status	
Married/cohabitating	60.5
Single	22.5
Divorced/separated/widowed	14.7
Education	
High school graduate	16.3
Some college	25.0
College graduate	37.5
Some graduate education	18.8

cover page containing a description of the study, instructions on how to complete the questionnaires, and to return the completed packets to the designated contact person. Informed consent information was included in the cover sheet. Volunteers were told that they were participating in a study that investigates the relationship between childhood experiences and anxiety.

Measures

This study used the Learning History Questionnaire to examine the criterion construct of CLH. We measured the criterion construct of panic in adulthood with the Panic Attack Questionnaire. Last, three assessments were employed to examine the moderating variables of perceived control and anxiety sensitivity: the Anxiety Control Questionnaire, the Anxiety Sensitivity Questionnaire, and the Body Sensations Questionnaire (Table 2).

Predictor

Learning History Questionnaire-Modified Version (LHQ) The LHQ version that we used in the present study contained three of the five scales used in the Watt et al. (1998) study. We combined the three scales used separately by Watt et al. (1998) to assess the arousal non-reactive symptoms of pain, colds, and rashes into one scale for the purposes of shortening the length of the assessment packet. The LHQ version used in the present study contained a total of three scales. The first two scales reflect instrumental learning involving arousal reactive and arousal non-reactive symptoms, respectively, during childhood. The last scale reflects vicarious learning of arousal reactive symptoms. Internal consistency estimates for the LHQ (Cronbach's α) range from .90 for the 'Experience/Arousal-Reactive' scale to .92 for both the 'Experience/Arousal Non-Reactive' and 'Observation/Arousal-

Reactive' scales (Watt et al. 1998). Subjects' LHQ responses were significantly positively correlated with those of their parents for the Experience/Arousal-Reactive ($r = .26$) and Observation/Arousal-Reactive ($r = .35$) scales, but not the Experience/Arousal Non-Reactive ($r = .12$) scale (Watt et al. 1998).

Criterion

Panic Attack Questionnaire, Revised (PAQ-R; Cox et al. 1992) The PAQ-R was included to assess participants' history and severity of panic attacks. The PAQ-R provides a description of a panic attack according to the DSM-III-R (American Psychiatric Association 1987) criteria and then asks whether subjects have ever experienced a panic attack. The PAQ-R enables the determination of both lifetime and current panic attack history. The PAQ-R also allows for a quantifiable determination of the severity of panic symptoms. In the symptom severity section, 23 panic-related symptoms are listed and respondents are asked to indicate the severity of each during a typical panic attack on a five-point Likert scale ranging from 0 = "does not occur" to 4 = "severely." Cox et al. (1994) reported that the three reliable factors (dizziness symptoms, cardio-respiratory symptoms, and cognitive symptoms) had adequate reliability and validity.

Moderator

Anxiety Control Questionnaire (ACQ; Rapee et al. 1996) The Anxiety Control Questionnaire is a 30 item self-report inventory that assesses perceived control over anxiety disorder related events such as, emotional reactions and external threats. Statements are rated on a five-point Likert scale ranging from 0 = "strongly disagree" to 5 = "strongly agree." This measure is scored by summing

Table 2 Pearson correlations: childhood learning history, ASI, BSQ, and adult panic symptoms ($N = 52$)

	PAQ-R	ACQ	ASI	BSQ	LHQ-PR	LHQ-PM
DV						
PAQ-R	—					
Mod						
ACQ	-.44**	—				
ASI	.38**	-.63**	—			
BSQ	.40**	-.27*	.49**	—		
IV						
LHQ-PR	.31*	-.15	.22	.17	—	
LHQ-PM	.40**	-.27*	.21	.35**	.60**	—

PAQ-R Panic Attack Questionnaire-Revised, ASI Anxiety Sensitivity Index, ACQ Anxiety Control Questionnaire, BSQ Body Sensations Questionnaire, LHQ-PR Learning History Questionnaire-Parent Reinforcement, LHQ-PM Learning History Questionnaire-Parent Modeling

* Indicates correlation is significant at the $p \leq .05$ level (2-tailed)

** Indicates correlation is significant at the $p \leq .01$ level (2-tailed)

items from the two subscales, control over internal reactions and control over external events. The ACQ has good overall internal consistency, Chronbach's $\alpha = .87$, and good internal consistency for each of the subscales (internal subscale $\alpha = .79$ and external subscale $\alpha = .81$; Zebb and Moore 1999). The ACQ has been demonstrated to have good reliability and validity in both inpatient (Lang and McNiel 2006) and outpatient populations (Rapee et al. 1996).

Moderator

Anxiety Sensitivity Index (ASI; Peterson and Reiss 1992) The Anxiety Sensitivity Index is a 16 item self-report inventory where subjects rate the extent to which they agree or disagree with beliefs that anxiety symptoms are signs of harmful/aversive consequences, by endorsing one of five-points on a Likert scale. The scale ranges from 0 = "very little" to 4 = "very much." The total ASI score, which ranges from 0 to 64, is obtained by summing the item scores. Considerable evidence supports the good psychometric properties of the ASI, including evidence of its validity as a measure of fear of anxiety (Peterson and Reiss 1992; Taylor 1996). Internal consistency is good to excellent, ranging from .82 to .91 across numerous studies (Peterson and Reiss 1993). Test-retest reliability appears to be satisfactory with correlations ranging from .71 to .75 (Peterson and Reiss 1993).

Moderator

Body Sensations Questionnaire (BSQ; Chambless et al. 1984) The Body Sensations Questionnaire is a 17-item measure that indicates the degree to which respondents fear bodily sensations that typically occur while one is anxious or experiencing a panic attack. Each item is rated on a five-point Likert scale ranging from 1 = "not at all" to 5 = "extremely." Prior studies support the BSQ's good psychometric properties, including an internal consistency of .87, and adequate test-retest reliability (Chambless et al. 1984). The BSQ has been shown to discriminate between individuals with PD and agoraphobia, individuals with other anxiety disorders, and non-anxious people (Chambless and Gracely 1989).

Results

Statistical Analyses

Bivariate Pearson correlations were computed between: (a) CLH with modeled parental anxiety (LHQ for Modeled Parental) and anxiety reinforcement (LHQ Experience Reinforced Anxiety) with adult panic outcome measures

(PAQ-R for panic history, panic symptoms severity); (b) CLH with anxiety reinforcement and with modeled parental anxiety with moderator measures (ASI, BSQ, ACQ); and (c) moderator measures (ASI, BSQ, ACQ) and panic outcome measures (panic history, panic symptom severity on PAQ-R).

Hierarchical regression analyses were used to determine the association between CLH (total of LHQ Experience Arousal-Reactive and Observation Arousal-Reactive scales) and panic outcome measures (PAQ-R for panic history and panic symptom severity), using the cognitive vulnerability measures (ASI, BSQ, ACQ) as moderating variables. Interaction effects were not hypothesized. However, exploratory two-way interactions were conducted for significant moderators; this was factored into the alpha correction.

Results

Descriptive Statistics

Mean scores on the BSQ ($M = 38.5$, $SD = 12.9$) and ACQ ($M = 96.2$, $SD = 22.0$) were consistent with published norms for non-clinical samples. For the ASI, mean scores ($M = 20.5$, $SD = 11.1$) were also consistent with published norms for non-clinical respondents (Peterson and Reiss 1993). These means indicate that the sample as a whole had only a mild level of 'fear of fear' or conviction that anxiety symptoms are dangerous. A summary of all self-report scores can be found in Table 3.

Of the 129 total participants, 39.7 % ($n = 52$) reported experiencing at least one panic attack. Individuals with and without panic attacks did not differ with regard to age ($P = 42.3$ years; nonP = 42.7 years; $t = .039$, $df = 118$, $p = .969$), gender ($\chi^2 = 2.482$, $df = 1$, $p = .115$), level of education ($\chi^2 = 3.173$, $df = 6$, $p = .715$), or marital status ($\chi^2 = 3.70$, $df = 6$, $p = .717$). Respondents who experienced at least one panic attack completed the PAQ-R. The mean PAQ-R total score ($M = 14.28$, $SD = 20.6$) indicates that panic symptoms were in the mild range for individuals who reported a history of a panic attack.

Forty two percent ($n = 54$) of the overall sample reported CLH with anxiety symptoms. Within these subjects, 63 % reported personal experience with anxiety symptoms (instrumental learning), 63 % reported observing their parents or others in the household experience symptoms (vicarious learning) and 26 % reported both instrumental and vicarious learning experiences. Participants who reported CLH with anxiety symptoms did not differ from those who reported no CLH with regard to age (CLH = 42 years, no CLH = 42 years, $t = .549$, $df = 75$, $p = .584$), gender ($\chi^2 = .08$, $df = 1$, $p = .77$), level of education ($\chi^2 = 1.685$, $df = 3$, $p = .641$), or marital status ($\chi^2 = 1.684$, $df = 3$, $p = .641$).

Table 3 Mean self-report scores ($N = 52$)

Test	<i>M</i>	<i>SD</i>
LHQ-PR	.75	.68
LHQ-PM	1.25	1.88
ASI	26.46	11.44
ACQ	87.04	22.08
BSQ	44.26	12.13
PAQ-R	34.92	17.78
<i>LHQ-PR</i> Learning History Questionnaire-Parent Reinforcement, <i>LHQ-PM</i> Learning History Questionnaire-Parent Modeling, <i>ASI</i> Anxiety Sensitivity Index, <i>ACQ</i> Anxiety Control Questionnaire, <i>BSQ</i> Body Sensations Questionnaire, <i>PAQ-R</i> Panic Attack Questionnaire-Revised		

Hypothesis 1 *Parental modeling of anxiety symptoms and reinforcement of anxiety symptoms, measured by the Learning History Questionnaire (LHQ), would each be associated with adult panic symptoms (PAQ-R).* In keeping with our predictions, parental reinforcement of anxiety symptoms was significantly related to panic symptoms [$r(52) = .40, p = .005$]. Also as predicted, having a parent model fear reactions or anxiety symptoms was significantly related to panic symptoms [$r(52) = .31, p = .024$].

Hypothesis 2 *Greater anxiety sensitivity (ASI), bodily sensations associated with anxiety (BSQ), and lower perceived control (ACQ) would be associated with higher adult panic symptoms (PAQ-R).* As expected, the ASI and BSQ were significantly related to adult panic symptoms [ASI: $r(52) = .38, p = .006$; BSQ: $r(52) = .40, p = .003$], demonstrating that beliefs about the harmfulness of anxiety symptoms were significantly associated with panic pathology for the overall sample. The ACQ was also significantly related to adult panic symptoms [ACQ: $r(128) = -.45, p < .01$]. Complete correlational data may be found in Table 2.

Hypothesis 3 We examined how ACQ and ASI may moderate the relationship between CLH (parent modeling and parent reinforcement) and adult panic symptoms. The ASI was dropped from the model because bivariate correlations revealed that it was not significantly correlated with either CLH variable ($p > .05$; see Table 2).

In model one of our hierarchical regression (Cohen et al. 2013), we entered our primary independent variables CLH with parental modeling (LHQ-PM) and CLH with parent reinforcement (LHQ-PR). We were able to see how each of these independent variables was associated with adult panic. Multicollinearity statistics (tolerance/VIF) did not indicate a high degree of overlap in the variance accounted

for by these variables. The overall model was statistically significant, [$F(1, 51) = 9.85, p = .004, R^2 = .17$]. Independent of the order of entry, we found that parental modeling uniquely predicted variance in the model ($\beta = .33, p = .004$). However, parent reinforcement did not uniquely predict variance in the model ($p > .05$). See Table 4 for model summary.

In the second step, we added the moderators ACQ and BSQ. Multicollinearity statistics (tolerance/VIF) were again within acceptable limits for the variables. Overall, model two was statistically significant, [$F(1, 51) = 6.27, p = .001, R^2 = .34$]. The independent variable, parent modeling, still uniquely predicted model variance ($\beta = .21, p = .04$); parent reinforcement was not significant ($p > .05$). The moderators ACQ and BSQ each predicted unique model variance independent of order of entry (ACQ $\beta = -.25, p = .02$; BSQ $\beta = .35, p = .03$). For the two-way interactions, multicollinearity statistics exceeded acceptable limits (Tolerance = .048, VIF = 20.89); therefore, they could not be computed for the model. See Table 4 for model summary.

Discussion

Our results lend support to unified cognitive vulnerability models of panic (Barlow 2000, 2002; Reiss and McNally 1985; Schmidt and Woolaway-Bickel 2005). Consistent with more recent findings (Bentley et al. 2013; Schmidt and Lerew 2002), our study found that the relationship between CLH and adult panic symptoms was explained

Table 4 Summary of exploratory hierarchical regression analyses for variables predicting adult panic symptoms ($N = 52$)

Variable	Model 1			Model 2		
	<i>B</i>	<i>SE B</i>	β	<i>B</i>	<i>SE B</i>	β
LHQ-PM	3.11	1.55	.33**	2.01	1.56	.21*
LHQ-PR	2.95	4.26	.11	3.43	4.10	.13
ACQ				-.25	.10	-.32**
BSQ				.35	.19	.24**
Two-way interactions				n.s.	n.s.	n.s.
R^2	.17			.34		
<i>F</i> for R^2 change	9.85**			6.27**		

n.s. not significant; betas could not be computed for interactions due to high multicollinearity. *LHQ-PR* Learning History Questionnaire-Parent Reinforcement, *LHQ-PM* Learning History Questionnaire-Parent Modeling, *ASI* Anxiety Sensitivity Index, *ACQ* Anxiety Control Questionnaire, *BSQ* Body Sensations Questionnaire, *PAQ-R* Panic Attack Questionnaire-Revised

* $p \leq .05$; ** $p \leq .01$

more fully by accounting for moderators pertaining to the individual's perception, sensitivity, and physical experience of anxiety.

Results largely supported our hypothesis pertaining to the importance of childhood history of parental modeling of panic symptoms in the manifestation of panic in adulthood. Our results support etiological models that highlight CLH as a distal vulnerability towards the development of anxiety psychopathology (Barlow 2000, 2002; Schmidt and Woolaway-Bickel 2005). As predicted, our study demonstrates that retrospectively reported CLH of parental modeling of anxiety is linked to adult beliefs such as anxiety sensitivity and low perceived control, as well as increased bodily sensations pertaining to anxiety. Our finding for anxiety-related symptoms is strengthened by its specificity; a CLH with non-arousal symptoms (e.g., colds) was not associated with high anxiety sensitivity, low perceived control, or panic symptoms. Indeed, it seems that a history of CLH marked by arousal-reactive symptoms may be *uniquely* associated with panic.

Interestingly, directly experiencing symptoms of anxiety alone in childhood was not associated with panic symptoms or with high anxiety sensitivity and low perceived control beliefs in our analyses. By contrast, indirect learning experiences were related to distal beliefs and panic symptoms in adulthood. In keeping with our predictions and in line with previous investigations (Stewart et al. 2001; Watt et al. 1998), being reinforced for having anxiety symptoms (instrumental learning) or having a parent or significant other model fear reactions or otherwise convey the harmfulness of anxiety symptoms (vicarious learning) was significantly related to panic history and panic symptoms in adulthood.

However, when correlations were computed for each learning modality separately with regard to distal beliefs, only vicarious learning was found to be associated with beliefs about the dangerousness of anxiety sensations and lower perceptions of control. These findings are interesting in that they imply a far stronger learning effect from parental modeling of the dangerousness of anxiety symptoms than from direct experiences with arousal-reactive symptoms or from parental reinforcement of the child's own sick role behavior when experiencing arousal reactive symptoms.

Prior studies have found significant associations between CLH, beliefs about anxiety symptoms, and panic frequency regardless of learning modality (Watt et al. 1998). There may be a couple of ways to explain this discrepancy. First, previous studies employed a large college student sample whereas our study contained a smaller, community-based, older sample. Thus, the smaller sample or the different population surveyed may have contributed to the discrepant results. Second, a number of participants

in the present study who reported directly experiencing arousal-reactive symptoms in childhood spontaneously noted on their questionnaires that the symptoms were not due to anxiety but rather to other medical problems. Although fear of symptoms such as dizziness or heart palpitations could develop regardless of the origins of those symptoms, it is possible albeit speculative, that the participants were educated as to the medical nature of their problems and thus viewed them in a more appropriate and realistic context, which may have prevented the acquisition of dysfunctional beliefs regarding those symptoms. In contrast to their own experiences, it is conceivable that participants may not have been as aware regarding the symptoms they observed in adults in their environment, thereby possibly creating attributions of anxiety or panic symptoms as fear provoking and not within their control.

As predicted and consistent with prior research (Stewart et al. 2001; Watt et al. 1998), greater anxiety sensitivity and lower perceptions of control were significantly associated with a history of panic attacks and severity of panic symptoms. These findings add to the already established data implicating cognitive factors such as anxiety sensitivity beliefs as central figures in the relationship to panic (Chambless and Gracely 1989; Schmidt et al. 1997, 1999) and add to the smaller but growing body of data implicating low perceived control as a key distal belief in the development of panic (Bentley et al. 2013).

Congruent with recent findings (Bentley et al. 2013; Schmidt Lerew 2002), our study showed that anxiety sensitivity and low perceived control combined exerted more powerful effects than either construct alone.

Interestingly, when examined individually, anxiety sensitivity and low perceived control did not significantly mediate the relationship between CLH (or specifically between experiences with arousal-reactive symptoms) and adult panic pathology. These findings are incongruent with previous evidence that has shown that anxiety sensitivity mediates the relationship between CLH and panic (Stewart et al. 2001), and between childhood experiences and hypochondriacal concerns (Watt and Stewart 2000). A possible explanation for the lack of findings is the small size of our sample relative to the previous studies that found positive results. Of note, our results showed that each belief contributed a small (25 %) but unique portion of the variance in accounting for participant's severity of panic psychopathology. When arousal reactive experiences were examined separately, ASI scores predicted most of the 18 % of the variance in explaining panic history.

For many, the ASI replaced older measures of the 'fear of fear' construct thought to highlight the cognitive components of panic. Recent literature has established anxiety sensitivity as a distal cognitive vulnerability factor in the development of panic attacks and PD (Schmidt and

Woolaway-Bickel 2005). The anxiety sensitivity construct has had a long history of empirical support as a key factor in the etiology of panic. Indeed, the ASI has long been employed to measure people's beliefs about their fear of anxiety and its consequences. However, in light of recent findings that suggest a lack of specificity for anxiety sensitivity (Watt et al. 1998; Watt and Stewart 2000), attempts to determine if the construct (or the measure most used to measure it) taps a broader vulnerability to general anxiety or somatic concerns seemed indicated.

As predicted, the ASI was significantly correlated with CLH's involving arousal reactive symptoms but, contrary to predictions, the ASI was not found to be related to CLH's of non-arousal symptoms. This finding seems to conflict with some newer studies suggesting that the ASI is not specific to anxiety-related symptoms (Watt et al. 1998; Watt and Stewart 2000).

Thus, our study did not replicate Watt and colleague's (1998, 2000) findings that the ASI is associated with both a CLH with anxiety as well with more general non-arousal symptoms such as colds, rashes, and pain. Our findings seem to suggest that the learning history origins of anxiety sensitivity may be solely rooted in experiences involving anxiety symptoms. The present finding may be reconciled with other studies that have noted the relationship between the anxiety sensitivity and other distressing conditions such as menstrual distress (Sigmon et al. 1996), hypochondriasis (Watt and Stewart 2000), and back pain (Asmundson and Norton 1995). It is interesting to note that all the conditions mentioned have associations with anxiety. Thus, a possible explanation for our results is that the developmental origins of anxiety sensitivity may indeed stem from experiences with anxiety alone and confer a cognitive vulnerability to view somatic symptoms of anxiety as harmful. Beliefs about the harmfulness of anxiety sensations may then generalize to other somatic concerns about illness, pain, or other bodily conditions via associative learning. For example, an individual who has beliefs that shortness of breath is dangerous may exhibit high levels of anxiety sensitivity and thus may be more prone to fear symptoms of menstrual pain as dangerous via a process of associative learning.

Our study offers some support for the BSQ as a finer measure of vulnerability to panic. Like the ASI, the BSQ was significantly associated with a CLH of arousal reactive symptoms but not with non-arousal symptoms in the overall sample. These findings support previous research (Ehlers 1993) that showed a relationship between BSQ scores and panic frequency and confirm our speculation that the BSQ may measure a cognitive vulnerability that is specific to fears of anxiety or panic sensations. In further support of our hypothesis, within the subgroup of participants who reported a history of panic attacks, CLH with

arousal reactive symptoms was uniquely associated with the BSQ, but not with the ASI. This finding indicates that for adult individuals with panic, the BSQ may more accurately reflect beliefs regarding the harmfulness of anxiety symptoms acquired through childhood learning experiences than the ASI.

There are several limitations to this study. First, our study employed a non-clinical cross-sectional, correlational design. Thus, we cannot establish causality or direction of causality. Longitudinal research should further investigate the pathways to the development of the cognitive vulnerabilities and their relationship to panic psychopathology suggested by the present correlational analyses. Specifically, researchers could follow children over time to determine whether instrumental and vicarious learning experiences lead to the development of beliefs, which in turn increase risk for more severe panic psychopathology in adulthood. Future research investigating cognitive vulnerability to PD using larger samples may use a longitudinal design to study other general cognitive risk factors not included in the unified model, such as the 'looming maladaptive style' described by Riskind and Williams (2005, 2006). Additionally, future research should be conducted with a clinical sample. Caution is required in generalizing these findings to clinical samples, since the current study was comprised of non-clinical individuals.

Second, the present study investigated only a small portion of the development trajectory hypothesized to lead to the development of panic disorder (Barlow 2000; Clark 1986; Reiss and McNally 1985). By combining prior theories and findings, our unified model of panic offers a possible comprehensive etiological model for the development of PD. Future studies are needed to investigate other aspects of the model and to determine if and how they may influence each other and contribute to the development of panic. For example, future studies may examine the influence of other early experiences, such as parental over-control and over-protection or other adverse and uncontrollable events, on the development of distal beliefs and ultimate panic. Prior research, for example, has found a link between low warmth and over-controlling parenting style, cognitive style, and anxiety (McGinn et al. 2005). Examining the impact of various childhood experiences on the development of both distal and proximal cognitions, and ultimately panic, would also provide greater confirmation for the various theoretical models encompassed within the unified model of panic.

Retrospective designs have also been criticized for being susceptible to memory biases and distortions (Eysenck et al. 1987; Ross and Conway 1986). Some of these biases include: selective memory of past events; the influences of current attitudes, behaviors, and experiences; and demand characteristics of the study itself. For example, scores on

the LHQ scales may reflect the same misinterpretations of bodily sensations as tapped by the measures of present panic symptoms, rather than reflecting actual childhood experiences. However, Ehlers' (1993) notes that "it is possible that current behavior is more determined by remembered events (such as those assessed by the questionnaire) than by actual circumstances" (p. 71). Although prospective designs are preferred, it is worth noting that adult children's retrospective reporting on similar versions of the LHQ has been shown to significantly correlate with parental responses on the LHQ (Watt et al. 1998). Recent findings also indicate that participant's responses remain accurate over time provided that the events or information they are asked to remember occurred when they were sufficiently old enough (Brewin et al. 1993), but the results of this study should still be interpreted with caution given the retrospective nature of the LHQ.

Finally, although our findings speak to the specificity of our unified model in observing a link between both anxiety sensitivity and perceived control as variables involved in PD, our study does not preclude the possibility that this link may also be present in other anxiety disorders. Indeed, some prior studies have failed to find specificity between panic disorder and other anxiety disorders on variables such as anxiety sensitivity, the event rate of trauma, the proportion of individuals with events preceding the onset of the disorder, or other types of events (Hofmann et al. 2000; Teachman 2005; Zvolensky and Forsyth 2002).

Our results suggest that CLH is associated with beliefs that may play an important role in conferring cognitive vulnerability to panic. Taken together, our findings suggest that parental modeling of the dangerousness of anxiety symptoms may be even more powerful than parental reinforcement of the child's own sick role behavior or the mere experience of arousal reactive symptoms.

Our findings also add to the small body of research suggesting that anxiety sensitivity and low perceived control together may interact to create a stronger distal vulnerability construct for panic in individuals whose childhood caregivers may have modeled fear of anxiety symptoms.

Although neither cognitive vulnerability measure was individually found to be a full mediator between CLH and adult panic pathology, both the anxiety sensitivity and low perceived control contributed a small and unique portion of the variance accounting for panic history and severity. These results support the growing body of evidence that beliefs regarding the harmfulness of anxious arousal and low perceptions of control may serve as distal cognitive vulnerability factors for the onset of panic attacks.

However, because individual measures of beliefs did not mediate the relationship between CLH and panic, future

research should consider specific types of childhood learning experiences as unique distal vulnerability factors as well as assessing other cognitive mediators such as the looming style.

Treatment implications of our findings should also be examined. Prior studies have shown that low perceived control predicted better outcomes in cognitive therapy and worse outcomes using Capnometry-assisted respiratory training (CART; Meuret et al. 2010). Future studies could investigate moderating effects of low perceived control and high anxiety sensitivity (individually and combined) on outcomes of different types of treatments for PD.

Conflict of Interest L. K. McGinn, Ph.D., K. B. Noonan, J. Cohen and K. D. Leaberry declare that they have no conflicts of interest, financial disclosures, or grants related to this manuscript.

Informed Consent All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000 (5). Informed consent was obtained from all individual subjects participating in the study.

Animal Rights No animal studies were carried out by the authors for this paper.

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