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Stressful Life Events, Anxiety Sensitivity, and Internalizing Symptoms in Adolescents

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Anxiety sensitivity represents a robust risk factor for the development of anxiety symptoms among both adolescents and adults. However, the development of anxiety sensitivity among adolescents remains inadequately understood. In this study, the authors examined the role of stressful life events as a risk factor for the development of elevated anxiety sensitivity. Anxiety sensitivity was then examined in a longitudinal design as a mechanism linking stressful life events to changes in anxiety symptoms. Stressful life events, anxiety sensitivity, and internalizing symptoms were assessed in a diverse community sample of adolescents ($N = 1{,}065$) at 3 time points spanning 7 months. The results indicated that stressful life events were longitudinally associated with increases in anxiety sensitivity and that certain types of stressful life events, specifically events related to health and events related to family discord, were differentially predictive of increases in anxiety sensitivity. Moreover, anxiety sensitivity mediated the longitudinal relation between stressful life events and anxiety symptoms. Evidence was also found for the predictive specificity of anxiety sensitivity to symptoms of anxiety but not depression.

Keywords: anxiety sensitivity, stress, anxiety, depression, adolescence

Anxiety sensitivity refers to fear of anxiety symptoms, including bodily sensations, which results from beliefs about the harmful social, psychological, or physiological consequences of such symptoms (Reiss, 1991; Reiss & McNally, 1985). Anxiety sensitivity has consistently been identified as a risk factor for the onset of panic attacks, panic disorder, and other anxiety disorders in prospective studies of adults (Maller & Reiss, 1992; Schmidt, Lerew, & Jackson, 1997, 1999) and has increasingly emerged as a risk factor for the development of anxiety among children and adolescents. Specifically, anxiety sensitivity has been found to predict the development of panic attacks among adolescents (Hayward, Killen, Kraemer, & Taylor, 2000; Weems, Hayward, Killen, & Taylor, 2002) and has been linked to paniclike symptoms, trait anxiety, fears, and anxiety disorders across a range of community and clinical samples of children and adolescents (Kearney, Albano, Eisen, Allan, & Barlow, 1997; Lau, Calamari, & Waraczynski, 1996; Pollock et al., 2002; Weems, Hammond-Laurence, Silverman, & Ginsburg, 1998). It is important to note that anxiety sensitivity predicts anxiety symptoms above and beyond trait anxiety (Chorpita, Albano, & Barlow, 1996; Weems, Hammond-Laurence, Silverman, & Ferguson, 1997), demonstrating the incremental validity of this construct in children and adolescents.

Individuals who believe that symptoms of anxiety portend negative physical and social consequences are thought to be more vulnerable to the development of anxiety pathology for several reasons. Individuals with high anxiety sensitivity may be more likely to attend to bodily sensations that are associated with anxiety, such as sweating or increased heart rate, and to misinterpret these symptoms as dangerous or catastrophic. These interpretations of bodily sensations can lead to increased anxiety and can perpetuate a cycle of increased attention to and misinterpretation of bodily cues. This process may eventually lead to panic attacks, avoidance, or increased symptoms of anxiety (Maller & Reiss, 1992; Reiss, 1991).

Although a consistent body of literature has identified anxiety sensitivity as an etiologic factor in the pathogenesis of anxiety among children and adolescents, a number of important questions remain unanswered regarding the determinants of anxiety sensitivity and its relation to different types of internalizing symptoms. First, the developmental origins of anxiety sensitivity remain unclear. Retrospective evidence from an adult sample suggests a role for operant conditioning in the development of anxiety sensitivity related to parental reinforcement of sick-role behavior in response to anxiety symptoms (Watt, Stewart, & Cox, 1998). In this study, parental reinforcement of sick-role behavior in response to somatic symptoms of anxiety was thought to both increase children's focus on bodily sensations associated with anxiety and to reinforce children's beliefs that anxiety symptoms lead to negative physical or health consequences, thereby increasing their fear of those symptoms. Other types of events, such the experience of uncued panic, may also lead to the development of beliefs that innocuous bodily sensations, like sweating, are dangerous and lead to negative health consequences. Indeed, the experience of spontaneous panic attacks has been found to predict increases in anxiety sensitivity among young adults over 5-week (Schmidt, Lerew, & Joiner, 2000) and 1-year intervals (Li & Zinbarg, 2007), demonstrating some role for learning in the development of anxiety sensitivity. The experience of panic has been proposed to have a scarring effect that renders individuals more vulnerable to developing elevated anxiety sensitivity.

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Accumulating evidence in adult samples therefore suggests that learning processes may be related to the development of anxiety sensitivity for some individuals; however, improving our understanding of the etiology of anxiety sensitivity requires a developmental approach. To our knowledge, factors related to the development of anxiety sensitivity among adolescents has been examined in only one study with a longitudinal design (Weems et al., 2002). This investigation identified different developmental pathways in levels of anxiety sensitivity among adolescents. Through cluster analysis, two groups with stable levels of anxiety sensitivity were identified (stable low and stable high), as well as a group for whom anxiety sensitivity escalated over a 4-year period. The group of adolescents with escalating anxiety sensitivity was equally at risk for the development of panic, as compared with the group with stable high anxiety sensitivity. The occurrence of panic attacks was examined as a potential risk factor for increasing anxiety sensitivity over time. Contrary to previous evidence documenting this association among adults (Schmidt et al., 2000), the relation between panic attacks and subsequent increases in anxiety sensitivity was not replicated among adolescents (Weems et al., 2002). As such, risk factors for the development of elevated anxiety sensitivity among adolescents have yet to be identified.

What factors might be related to the development of high anxiety sensitivity among adolescents? As discussed, prior research with adults has suggested that learning may influence the development of anxiety sensitivity. Environmental events may also play a role in shaping beliefs about the consequences of anxiety symptoms. The experience of stressful life events, particularly stressors that are uncontrollable and unpredictable, represents one possibility. The experience of uncontrollable stressful life events has been consistently implicated in the pathogenesis of anxiety symptomatology among children and adolescents (Grant, Compas, Thurm, & McMahon, 2004). Moreover, there is a substantial literature documenting that stress leads to various forms of repetitive, self-focused thought, such as worry and rumination, which confer risk for depression and anxiety disorders (Nolen-Hoeksema, Larson, & Grayson, 1999; Watkins, 2008). Ruminative self-focus involves repetitive thought focused on the causes and consequence of symptoms of distress (Nolen-Hoeksema & Morrow, 1991). Stressful events may contribute to the development of anxiety sensitivity by setting in motion a process similar to rumination that involves increased self-focused attention to bodily sensations and to physical and cognitive symptoms of anxiety, as well as increased thought about the causes and consequences of those symptoms. Increases in these types of cognitions may in turn lead to the development of anxiety sensitivity, particularly if an individual focuses on the potential negative consequences of anxiety symptoms. Support for the hypothesis that stress plays a role in the development of anxiety sensitivity comes from a brief longitudinal study of adults in which high levels of reported anxiety during a period of heightened stress—basic combat training for military recruits—were related to increases in anxiety sensitivity over 5 weeks (Schmidt et al., 2000). However, the relation between unanticipated stressful life events and anxiety sensitivity has never been examined.

It is important to note that if anxiety sensitivity does increase following stressful life events, anxiety sensitivity may represent a mechanism linking stress to the development of anxiety symptoms among adolescents. Although stress represents an important determinant of adverse mental health among children and adolescents (Grant et al., 2004), there is a paucity of research addressing mediators of the stress–psychopathology relation, despite repeated calls for such research (Grant et al., 2003). As such, the second goal in this study was to determine whether anxiety sensitivity represents a mechanism linking stress to increases in anxiety symptoms over time.

In addition to identifying determinants of anxiety sensitivity, a second, unresolved issue regarding anxiety sensitivity among adolescents involves its specificity as a risk factor for the development of anxiety disorders, versus a more global risk factor for internalizing psychopathology (i.e., mood as well as anxiety disorders). In examining differential effects of anxiety sensitivity on anxiety and depression, it is important to take into account the multidimensional nature of anxiety sensitivity. Anxiety sensitivity is characterized by a hierarchical structure that involves a global fear of anxiety symptoms that can be further broken down into fears about specific types of symptoms (Lilienfeld, Turner, & Jacob, 1993). At least three lower order factors have been identified in samples adults (Li & Zinbarg, 2007; Taylor, Koch, Woody, & McLean, 1996; Zinbarg, Barlow, & Brown, 1997) and samples of children and adolescents (Muris, Schmidt, Merkelbach, & Schouten, 2001; Silverman, Ginsburg, & Goedhart, 1999; Silverman, Goedhart, Barrett, & Turner, 2003; van Widenfelt, Siebelink, Goedhart, & Treffers, 2002), which represent (a) fear of physical symptoms, (b) fear of cognitive or mental incapacitation, and (c) fear of publicly observable symptoms and/or social fears. The factor structure of anxiety sensitivity among children and adolescents has been found to be more differentiated, such that the physical concerns factor separates into two factors that tap fear of anxiety symptoms that could signify disease and fear of feeling unsteady or shaky (Muris et al., 2001; Silverman et al., 2003).

Several studies have addressed the extent to which anxiety sensitivity is uniquely associated with anxiety. Elevated anxiety sensitivity has also been found among adults with major depression (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995; Taylor et al., 1996), and a positive association between anxiety sensitivity and depressive symptoms among children and adolescents has been documented after accounting for concurrent anxiety symptoms in cross-sectional studies (Weems et al., 1997). However, other studies in both adolescent samples and adult samples have shown that the relation between anxiety sensitivity and depressive symptoms disappears when anxiety symptoms are included in the model (Pollock et al., 2002; Schmidt, Lerew, & Joiner, 1998). Thus, it remains unclear whether the relation between anxiety sensitivity and depression actually exists or whether it is accounted for by the overlap between symptoms of anxiety and depression. To date, the relation between anxiety sensitivity and depression has been examined in only one study with a longitudinal design (Schmidt et al., 1998), and in this study it was reported that among adults, the association between anxiety sensitivity and depression disappeared after anxiety symptoms were included in the predictive model. No prospective studies have examined this association among adolescents.

The different facets of anxiety sensitivity have been documented to have differential associations with symptoms of anxiety and depression. Specifically, fear of physical symptoms and fear of publicly observable symptoms have each been found to predict

anxiety symptomatology, whereas fear of mental incapacitation has been demonstrated to predict symptoms of depression (Taylor et al., 1996; Zinbarg, Brown, Barlow, & Rapee, 2001). To our knowledge, the predictive specificity of the different facets of anxiety sensitivity to symptoms of anxiety and depression has not been examined in longitudinal studies of children or adolescents. The third goal in this study, therefore, was to examine the longitudinal relation between anxiety sensitivity and each of its four facets and symptoms of both anxiety and depression among adolescents.

In sum, the purpose of the current investigation was to address several gaps in the literature on anxiety sensitivity, with prospective data from a large, diverse community-based sample of adolescents. We first aimed to identify factors related to the development of anxiety sensitivity by examining the impact of stressful life events on changes in anxiety sensitivity over time. We hypothesized that adolescents who experienced stressful life events would report subsequent increases in anxiety sensitivity. In addition, we evaluated whether certain types of stressful life events—events related to health and events related to family discord—were uniquely predictive of increased anxiety sensitivity. Next, we examined the role of anxiety sensitivity as a mechanism linking stressful life events to changes in anxiety symptoms over time. We predicted that anxiety sensitivity would mediate the longitudinal association between stressful life events and changes in symptoms of anxiety. It is important to note that we were able to apply a stringent test of mediation in a longitudinal design with three separate assessments (Maxwell & Cole, 2007). Finally, we examined the specificity of anxiety sensitivity as a risk factor for anxiety symptomatology among adolescents. The predictive specificity of anxiety sensitivity and each of its four facets to the development of symptoms of anxiety and depression was examined with a covariance strategy. We expected that fear of physical symptoms and social fears would be uniquely associated with anxiety, whereas fear of mental incapacitation would uniquely predict depression.

Method

Participants

The sample for this study was recruited from the total enrollment (approximately 1500 students) of two middle schools (Grades 6–8) from a school district in central Connecticut that agreed to participate in the study (students in self-contained special education classrooms and students in technical programs who did not attend school for the majority of the school day were excluded). The community in which the schools are located is a small, urban community (metropolitan population of 71,538). The school district was selected to participate in a larger investigation of mechanisms linking stress to psychopathology in adolescents. To ensure sufficient variability in stress exposure, we chose schools for the study on the basis of diversity in racial, ethnic, and socioeconomic composition, as well as their willingness to participate in the study.

The parents of all eligible students ($N = 1{,}567$) in the participating middle schools were asked to provide active consent for their adolescent to participate in the study. Parents who did not return written consent forms to the school were contacted by telephone to obtain consent. Twenty-two percent of parents did not return consent forms and could not be reached to obtain consent, and 6% of parents declined to provide consent for their adolescent to participate in the study. The overall participation rate in the study at baseline was 72%. Additional students who were not present at the baseline assessment period were added at Time 2 (March 2006; 71 students) and Time 3 (June 2006; 139 students). Some of these students were absent at the baseline assessment, and some had moved to the school district during the time interval separating the assessments. Two hundred twenty-one (20.8%) participants who were present at the baseline assessment did not participate at the Time 2 assessment, and 217 (20.4%) participants who were present at the baseline assessment did not participate at the Time 3 assessment. Some of these students were absent on the day of data collection, and some had moved from the district. It is important to note the transience of student enrollment in this district. Data from the school district indicate that over the 4-year period from 2000–2004, 22.7% of students had left the district (Connecticut Department of Education, 2006).

The baseline sample included 51.2% ($n = 545$) boys and 48.8% ($n = 520$) girls, resulting in 1,065 participants, all of whom provided assent before participating in the study. Participants were evenly distributed across grade level, with 31.8% ($n = 337$) of participants in the sixth grade, 33.9% ($n = 360$) in the seventh grade, and 34.3% ($n = 364$) in the eighth grade at the time of the study. Participant ages ranged from 11 years to 14 years. The race and ethnicity composition of the sample was as follows: 13.2% ($n = 141$) non-Hispanic White, 11.8% ($n = 126$) non-Hispanic Black, 56.9% ($n = 610$) Hispanic or Latino, 2.2% ($n = 24$) Asian or Pacific Islander, 0.2% ($n = 2$) Native American, 0.8% ($n = 9$) Middle Eastern, 9.3% ($n = 100$) biracial or multiracial, and 4.2% ($n = 45$) members of other racial and ethnic groups. A small percentage of participants, 1.3% ($n = 14$), declined to provide information on their racial and ethnic background. Twenty-seven percent ($n = 293$) of participants reported living in single-parent households. We did not ask the students to report on their family income because the validity of their reports was unlikely to be high. The community in which the participating middle schools reside is a uniformly lower SES community, with a per capita income of U.S.$18,404 (Connecticut Department of Education, 2006). School records indicated that 62.3% of students qualified for free or reduced lunch in the 2004–2005 school year. There were no differences across the two schools in demographic variables.

Measures

Stress. The Life Events Scale for Children (LES-C; Coddington, 1972) is composed of 25 items that each represent a stressful life event (e.g., *"Your parents got divorced"* and *"You were hospitalized for a serious illness"*). Participants are asked to indicate which events they had experienced in the prior 6 months. Life events checklists are the instruments most commonly used to assess adolescent stress (Grant et al., 2004), and the Life Events Scale is one of the two most commonly used checklists in the adolescent stress literature (Hammen, 2008). In addition to examining the effect of total stressful life events, we created two subscales of the Life Events Scale for Children in order to evaluate whether specific facets of stressful life events were uniquely associated with the development of anxiety sensitivity. The first

subscale included seven health-related stressful life events that involved the hospitalization of the participant or a family member for a serious illness or the death of a relative or close friend (e.g., "*One of your parents was hospitalized for a serious illness*"). The second subscale included five stressful life events that reflect family discord, such as parental separation, divorce, or increases in serious family conflict (e.g., "*Your parents were divorced*").

Anxiety sensitivity. Anxiety sensitivity was assessed with the Childhood Anxiety Sensitivity Index (CASI; (Silverman, Fleisig, Rabian, & Peterson, 1991), an 18-item self-report measure that measures the extent to which children believe that anxiety symptoms have negative consequences. Each item on the CASI is rated on a three-point scale ranging from *none* (1) to *a lot* (3). The CASI has been consistently found to be multifactorial (Silverman et al., 1999, 2003; van Widenfelt et al., 2002). Examination of the factor structure across community and clinical samples of children and adolescents supports, in addition to a general factor, the existence of four lower order factors (Anxiety Sensitivity–Total; Silverman et al., 2003): Disease Concerns (Anxiety Sensitivity–Disease; *"It scares me when my heart beats fast"*), Unsteady Concerns (Anxiety Sensitivity–Unsteady; *"It scares me when I feel faint"*), Mental Incapacitation Concerns (Anxiety Sensitivity–Mental; *"When I'm afraid, I worry that I might be crazy"*), and Social Concerns (Anxiety Sensitivity–Social; *"I don't want other people to know when I feel afraid"*). The CASI has good test–retest reliability, internal consistency, and convergent validity (Silverman et al., 1991). The CASI demonstrated good reliability in this sample ($\alpha = .88$). However, because the CASI is multifactorial, the meaning of Cronbach's alpha as a measure of scale reliability is not clear (Zinbarg, Revelle, Yovel, & Li, 2005). As such, we report the more appropriate omega_hierarchical statistic (ω_h) that provides an estimate of scale reliability that takes into account the factor structure of the CASI (Zinbarg, Yovel, Revelle, & McDonald, 2006). Omega_hierarchical was calculated by estimating the squared correlation between the general factor of the CASI and the CASI total score (i.e., the sum of the 18 individual items). At minimum, the general factor should account for 50% of the variance in total scores (Revelle, 1979). The reliability of the CASI in this sample was supported ($\omega_h = .64$).

Anxiety symptoms. The Multidimensional Anxiety Scale for Children (MASC; March, Parker, Sullivan, Stallings, & Conners, 1997) is a 39-item widely used measure of anxiety in children. The MASC assesses physical symptoms of anxiety, harm avoidance, social anxiety, and separation anxiety and is appropriate for children and adolescents aged 8 years to 19 years. Each item presents a symptom of anxiety, and participants indicate how true each item is for them or how frequently they experience that symptom on a 4-point Likert scale ranging from *never true* (0) to *very true* (3). The MASC has high internal consistency and test–retest reliability across 3-month intervals, has good convergent validity and divergent validity (Muris, Merckelbach, Ollendick, King, & Bogie, 2002), and differentiates among anxious children, control children, and children with other types of psychopathology (March, Sullivan, & Parker, 1999). The MASC demonstrated good reliability in this sample ($\alpha = .88$).

Depressive symptoms. The Children's Depression Inventory (CDI; (Kovacs, 1992) is the most widely used self-report measure of depressive symptoms in children and adolescents. The CDI is a 27-item self-report measure of depressive symptoms that has been standardized on children and adolescents aged 7 years to 17 years. Each item consists of three statements representing different levels of severity of a specific symptom of depression (e.g., dysphoric mood, sleep disturbance). The CDI has sound psychometric properties, including internal consistency, test–retest reliability, and discriminant validity (Kovacs, 1992; Reynolds, 1994). The item pertaining to suicidal ideation was removed from the measure at the request of school officials and the human subjects committee. The CDI demonstrated good reliability in this sample ($\alpha = .82$).

Procedure

Participants completed study questionnaires during their homeroom period on 2 consecutive days at Time 1 and Time 3. The Time 2 assessment consisted of fewer questionnaires and was completed in 1 day. All questionnaires used in the present analyses were administered at Time 1. Because of our hypothesis that anxiety sensitivity would mediate the longitudinal relation between stressful life events and increases in symptomatology over time, we assessed anxiety sensitivity at Time 2 and the symptomatology questionnaires at Time 3. Four months elapsed between the Time 1 (November 2005) and Time 2 (March 2006) assessments, and 3 months elapsed between Time 2 and Time 3 (June 2006) assessments. This time frame was chosen to allow the maximum time between assessments and to ensure that all assessments occurred within the same academic year. Given the transient nature of the school population, data collection within 1 academic year was necessary to avoid high attrition. Homeroom teachers and one member of the research team were present in the classroom during the assessment period. Participants were assured of the confidentiality of their responses and the voluntary nature of their participation.

Data Analysis

The role of stressful life events in the development of anxiety sensitivity was examined by conducting linear regression analyses examining stressful life events at Time 1 as a predictor of anxiety sensitivity at Time 2, including a covariate for Time 1 anxiety sensitivity. Separate regression equations were estimated for the four facets of anxiety sensitivity (disease concerns, unsteady concerns, fear of mental incapacitation, and fear of publicly observable symptoms). Next, we examined whether certain types of stressful life events were uniquely predictive of increased anxiety sensitivity. We examined the role of health-related stressful life events and events related to family discord as predictors of anxiety sensitivity.

The hypothesis that anxiety sensitivity mediated the longitudinal relation between stressful life events and symptoms of anxiety was evaluated with two mediation approaches. First, procedures outlined by Baron and Kenny (1986) were used: (a) Stressful life events, assessed at Time 1, were examined as a predictor of Time 3 symptoms of anxiety, when a covariate for baseline symptom levels was included; (b) Time 1 stressful life events were examined as a predictor of anxiety sensitivity assessed at Time 2, when a covariate for baseline anxiety sensitivity was included; (c) anxiety sensitivity at Time 2 was evaluated as a predictor of anxiety symptoms at Time 3, including a covariate for baseline symptom levels; (d) the full mediation model was tested in order to evaluate

the hypothesis that anxiety sensitivity mediated the relation between stressful life events and anxiety symptomatology. Second, the asymmetric distribution of products approach (MacKinnon, Fritz, Williams, & Lockwood, 2007) was used to determine whether significant mediation effects existed. The 95% confidence limits of the indirect effect were calculated with the PRODCLIN program. After examining the full mediation model, mediation effects were examined separately for each of the facets of anxiety sensitivity that were found to significantly increase as a result of stressful life events, to determine which facets of anxiety sensitivity were driving mediation effects.

The predictive specificity of anxiety sensitivity was examined with a covariance analysis strategy, consistent with previous investigations of the specificity of anxiety sensitivity to anxiety symptom development among adults (Schmidt et al., 1999, 1998). Separate regression equations were examined for symptoms of anxiety and depression. In the first regression analysis, anxiety symptoms were examined at Time 3 as the dependent variable. Time 1 anxiety symptoms were added at Step 1 to create residualized change scores, followed at Step 2 by symptoms of depression at Time 1 and Time 3 to account for changes in depression. Each of the four facets of Time 1 anxiety sensitivity was added at Step 3. This analysis strategy identified the impact of each facet of anxiety sensitivity on symptoms of anxiety after accounting for changes in depression over time (Schmidt et al., 1998). The effect of the general anxiety sensitivity factor was examined by subtracting the amount of variance accounted for by each of the four facets from the total amount of variance accounted for by the set (Li & Zinbarg, 2007; Zinbarg et al., 2001). The second set of regression equations examined depressive symptoms at Time 3 as the dependent variable and followed the same stepped approach as the anxiety analysis.

Results

Preliminary Analyses

Analyses were first conducted to determine whether participants who did not complete all three assessments differed from those who did complete the baseline and two follow-up assessments. Univariate analyses of variance were conducted for continuous outcomes, with attrition as a between-subjects factor and with demographic factors, each of the psychopathology outcomes, stressful life events, and anxiety sensitivity as dependent variables. Chi-square analyses were performed for dichotomous outcomes. These analyses revealed that participants who completed the baseline but not both follow-up assessments were more likely to be female, $\chi^2(1, N = 1065) = 6.85$, $p < .01$, but did not differ in grade level, in race and ethnicity, or in single-parent household background ($ps > .10$). Participants who did not complete at least one of the follow-up assessments did not differ from participants who completed all three assessments on baseline depression or anxiety symptoms, stressful life events, or anxiety sensitivity ($ps > 0.10$).

Next, we examined the factorial invariance of anxiety sensitivity over time (Cole & Maxwell, 2003; Horn & McArdle, 1992) to ensure that the factor structure, and therefore the interpretation, of the CASI did not change upon readministration. This analysis was conducted with AMOS 6.0 software (Arbuckle, 2005), with the full information maximum likelihood estimation method, which estimates means and intercepts to handle missing data. We conducted a confirmatory factor analysis on the continuously scored CASI, using the four previously identified lower order factors (Silverman et al., 2003). This model fit the data very well, $\chi^2(129, N = 921) = 341.04$, comparative fit index = .95, root-mean-square error of approximation = .04. We then constrained the factor loadings from Time 1 to be equal to the factor loadings at Time 2 and compared this model with the unconstrained model. No significant difference between these models emerged, $\chi^2(14, N = 886) = 22.62$, $p > .05$, demonstrating metric invariance of the CASI across the two administrations (Horn & McArdle, 1992).

Descriptive Statistics

Anxiety sensitivity increased from the Time 1 assessment to Time 2 assessment, $F(1, 674) = 13.92$, $p < .001$, $\eta^2 = .02$. Anxiety symptoms decreased across the two assessments, $F(1, 854) = 109.59$, $p < .001$, $\eta^2 = .11$, whereas depressive symptoms increased significantly over time, $F(1, 850) = 9.03$, $p = .003$, $\eta^2 = .01$. Table 1 displays the mean and standard deviation of Time 1 stressful life events, anxiety sensitivity at Time 1 and Time

Table 1
Correlations, Means, and Standard Deviation of Stressful Life Events, Anxiety Sensitivity, and Symptoms of Anxiety and Depression

Measure	1	2	3	4	5	6	7
1. Stressful life events Time1	—						
2. CASI anxiety sensitivity Time 1	.22**	—					
3. MASC anxiety Time 1	.10**	.50**	—				
4. CDI depression Time 1	.29**	.30**	.28**	—			
5. CASI anxiety sensitivity Time 2	.19**	.47**	.37**	.26**	—		
6. MASC anxiety Time 3	.14**	.39**	.53**	.24**	.42**	—	
7. CDI depression Time 3	.20**	.18**	.13**	.54**	.21**	.33**	—
M	5.10	10.30	40.20	9.67	11.44	34.80	10.63
SD	3.33	6.88	15.39	6.44	6.80	18.05	8.15

Note. CASI = Children's Anxiety Sensitivity Index; MASC = Multidimensional Anxiety Scale for Children; CDI = Children's Depression Inventory.
** $p < .01$.

2, and anxiety and depression symptoms at the Time 1 and Time 3 assessments, along with the zero-order correlations among variables. As expected, anxiety sensitivity demonstrated significant concurrent associations with symptoms of anxiety and depression both at Time 1 and at Time 3 (see Table 1).

Stressful Life Events and Anxiety Sensitivity

In linear regression analysis, Time 1 stressful life events predicted anxiety sensitivity (Anxiety Sensitivity–Total) at Time 2, after Time 1 anxiety sensitivity was included as a covariate ($\beta = .08$, $p < .05$). The four facets of anxiety sensitivity were then examined in separate regression analyses. Stressful life events predicted Time 2 disease concerns (Anxiety Sensitivity–Disease; $\beta = .08$, $p < .05$) and mental incapacitation concerns (Anxiety Sensitivity–Mental; $\beta = .13$, $p < .001$) when Time 1 levels were included as a covariate but did not predict Time 2 unsteady concerns (Anxiety Sensitivity–Unsteady; $\beta = .04$, $p > .05$) or social concerns (Anxiety Sensitivity–Social; $\beta = .03$, $p > .05$).

Next, we examined whether certain types of stressful life events were differentially predictive of increases in each of the facets of anxiety sensitivity. The impact of health-related stressful life events, involving serious illness or death, was examined first. Health-related stressful life events predicted Time 2 disease concerns (Anxiety Sensitivity–Disease; $\beta = .09$, $p < .05$) and mental incapacitation concerns (Anxiety Sensitivity–Mental; $\beta = .09$, $p < .05$) after Time 1 levels were included as a covariate but did not predict Time 2 unsteady concerns (Anxiety Sensitivity–Unsteady; $\beta = .04$, $p > .05$) or social concerns (Anxiety Sensitivity–Social; $\beta = .02$, $p > .05$). We then examined stressful life events reflecting family discord and found that these types of events predicted Time 2 unsteady concerns (Anxiety Sensitivity–Unsteady; $\beta = .08$, $p < .05$), mental incapacitation concerns (Anxiety Sensitivity–Mental; $\beta = .12$, $p < .001$), and social concerns (Anxiety Sensitivity–Social; $\beta = .10$, $p < .01$) when Time 1 levels were included as a covariate but did not predict Time 2 disease concerns (Anxiety Sensitivity–Disease $\beta = .07$, $p > .05$).

Mediation Analysis

Time 1 stressful life events were significantly associated with Time 3 anxiety symptoms, after Time 1 anxiety was included as a covariate ($\beta = .09$, $p < .01$). To ensure that the longitudinal association between stressful life events and anxiety is not an artifact of the association between anxiety sensitivity and anxiety, we added a covariate for Time 1 anxiety sensitivity to the model. Stressful life events remain a significant predictor of Time 3 anxiety symptoms ($\beta = .07$, $p = .03$). Time 1 stressful life events predicted anxiety sensitivity at Time 2, after Time 1 anxiety sensitivity was included as a covariate ($\beta = .08$, $p < .05$). Finally, Time 2 anxiety sensitivity was associated with Time 3 anxiety symptoms, when Time 1 anxiety was included as a covariate ($\beta = .30$, $p < .001$). In the final mediation model, stressful life events were no longer a significant predictor of Time 3 anxiety symptoms, when anxiety sensitivity was added to the model with Time 1 anxiety symptoms as a covariate ($\beta = .05$, $p = .161$). The 95% confidence limits of the indirect effect did not include zero (.25, .46), indicating a significant indirect effect of stressful life events on anxiety symptoms through anxiety sensitivity[1] (MacKinnon et al., 2007).

Disease concerns and mental incapacitation concerns, the two facets of anxiety sensitivity that increased as a result of stressful life events, were examined separately as mediators of the relation between stressful life events and increases in anxiety symptoms. Time 1 stressful life events were significantly associated with Time 3 anxiety symptoms, after including Time 1 anxiety as a covariate ($\beta = .09$, $p < .01$). Time 1 stressful life events predicted disease concerns at Time 2, after Time 1 disease concerns were included as a covariate ($\beta = .08$, $p < .05$). Finally, Time 2 disease concerns were associated with Time 3 anxiety symptoms, when Time 1 anxiety was included as a covariate ($\beta = .27$, $p < .001$). In the final mediation model, stressful life events were no longer a significant predictor of Time 3 anxiety symptoms, after Time 1 anxiety symptoms were included as a covariate, when disease concern was added to the model ($\beta = .06$, $p = .078$). The 95% confidence limits of the indirect effect did not include zero (0.36, 0.75), indicating a significant indirect effect of stressful life events on anxiety symptoms through the disease concerns facet of anxiety sensitivity (MacKinnon et al., 2007).

Mediation was also supported for the mental incapacitation facet of anxiety sensitivity. Time 1 stressful life events predicted mental incapacitation concerns at Time 2, after Time 1 mental incapacitation concerns were included as a covariate ($\beta = .13$, $p < .001$), and Time 2 mental incapacitation concerns were associated with Time 3 anxiety symptoms, when Time 1 anxiety was included as a covariate ($\beta = .24$, $p < .001$). In the final mediation model, stressful life events were no longer a significant predictor of Time 3 anxiety symptoms, after Time 1 anxiety symptoms were included as a covariate, when mental incapacitation concern was added to the model ($\beta = .05$, $p = .208$). The 95% confidence limits of the indirect effect did not include zero (0.27, 1.01), indicating a significant indirect effect of stressful life events on anxiety symptoms through the mental incapacitation facet of anxiety sensitivity (MacKinnon et al., 2007).

Predictive Specificity

As a set, the four facets of anxiety sensitivity accounted for significant variability in Time 3 anxiety symptoms after including covariates for Time 1 anxiety symptoms and changes in depressive symptoms in the model ($R^2 = .02$, $p < .001$; see Table 2). The general factor (Anxiety Sensitivity–Total) accounted for 1.2% of the variance in anxiety symptoms, Anxiety Sensitivity–Disease accounted for 0.1%, Anxiety Sensitivity–Unsteady accounted for 0.5%, Anxiety Sensitivity–Social accounted for 0.1%, and Anxiety Sensitivity–Mental accounted for virtually 0%. Thus, the association between anxiety sensitivity and anxiety symptoms was driven largely by the general anxiety sensitivity factor and, to a lesser degree, the facet reflecting concerns about feeling unsteady. See Table 2 for all beta weights and step statistics.

[1] We tested two alternative models in which Time 1 anxiety sensitivity (a) moderates the effect of stressful life events on anxiety sensitivity at Time 2 and (b) moderates the effect of stressful life events on Time 3 anxiety symptoms. We found no evidence for moderation of Time 1 stressful life events on anxiety sensitivity at Time 2 ($\beta = -.13$, $p > .05$) or on anxiety symptoms at Time 3 ($\beta = -.02$, $p > .05$) by Time 1 anxiety sensitivity.

Table 2
Longitudinal Associations Between Anxiety Sensitivity Facets and Symptoms of Anxiety and Depression

Predictors	Step statistics				Final statistics		
	ΔR^2	*b*	*SE b*	β	*b*	*SE b*	β
Time 3 MASC outcome							
Time 1 MASC	.29**	.65	.04	.54**	.52	.05	.44**
Time 1 CDI	.06**	−.17	.11	−.06	−.24	.11	−.07
Time 2 CDI		.61	.09	.27**	.60	.09	.27**
Time 1 AS-Disease	.02**				.28	.27	.05
Time 1 AS-Unsteady					.82	.39	.10*
Time 1 AS-Mental					−.11	.90	−.00
Time 1 AS-Social					.42	.40	.04
Total R^2	.36**						
Time 3 CDI outcome							
Time 1 CDI	.31**	.70	.04	.56**	.69	.04	.55**
Time 1 MASC	.05**	−.09	.02	−.17**	−.09	.02	−.18**
Time 2 MASC		.12	.02	.26**	.12	.02	.27**
Time 1 AS-Disease	.01				.06	.12	.03
Time 1 AS-Unsteady					−.18	.17	−.05
Time 1 AS-Mental					.42	.22	.08
Time 1 AS-Social					−.37	.18	−.08*
Total R^2	.36**						

Note. CDI = Children's Depression Inventory; MASC = Multidimensional Anxiety Scale for Children; AS-Disease = Anxiety Sensitivity, Disease Concerns; AS-Unsteady = Anxiety Sensitivity, Unsteady Concerns; AS-Mental = Anxiety Sensitivity, Mental Incapacitation Concerns; AS-Social = Anxiety Sensitivity, Social Concerns.
* $p < .05$. ** $p < .01$.

After accounting for changes in anxiety symptoms and Time 1 depression, the four anxiety sensitivity variables were not significantly associated with Time 3 depression ($R^2 = .01$, $p > .05$). Of the variability in depression that was accounted for by anxiety sensitivity, less than .01% was attributable to the general factor (Anxiety Sensitivity–Total), Anxiety Sensitivity–Disease, Anxiety Sensitivity–Unsteady, Anxiety Sensitivity–Mental, and Anxiety Sensitivity–Social. In sum, anxiety sensitivity was prospectively associated with symptoms of anxiety but not with depression.

Discussion

In the current investigation, we sought to address several gaps in the literature on anxiety sensitivity among adolescents. The first goal was to examine stressful life events as a contributor to increased anxiety sensitivity. As hypothesized, the experience of stressful life events was longitudinally associated with increases in anxiety sensitivity. To our knowledge, this study is the first to identify stressful life events as a factor associated with the development of anxiety sensitivity in adolescents. Although previous studies have identified uncued panic attacks as a factor predicting increased anxiety sensitivity among adults (Schmidt et al., 2000), this association has not been replicated in adolescent samples (Weems et al., 2002). As such, these findings provide novel information regarding the development of anxiety sensitivity among adolescents, providing evidence for the role of environmental factors.

What are the pathways that explain the association between stressful life events and the formation of negative beliefs about the consequences of anxiety symptoms? Prior research suggests that the experience of stressful events increases certain types of self-focused repetitive thought, such as rumination (Robinson & Alloy, 2008; Watkins, 2008). A similar process may lead to the development of elevated anxiety sensitivity, wherein stressful life events increase attention to bodily cues of anxiety and anxiety-related cognitions. This increased attention, paired with greater focus on the causes and consequences of anxiety symptoms, may lead to the development of negative beliefs about the consequence of anxiety symptoms. Anxiety sensitivity, in fact, shares many features with certain types of repetitive self-focused thought (Watkins, 2008). Both anxiety sensitivity and rumination involve thought about the meaning or consequences of negative affect and distress (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Reiss, 1991). These processes each may lead to similar cognitions and self-statements during periods of self-focus (e.g., "I won't be able to concentrate if I keep feeling this way"). Finally, both anxiety sensitivity (Maller & Reiss, 1992; Weems et al., 2002) and rumination (Calmes & Roberts, 2007; Nolen-Hoeksema, 2000; Nolen-Hoeksema & Morrow, 1991) are established risk factors for the development of anxiety. As such, it is likely that similar environmental events (i.e., stressors) may trigger the development of each of these types of self-focused thought.

We also examined the association between specific types of stressful life events—including those related to physical health and family discord—and the subsequent development of anxiety sensitivity. The results documented that these two types of stressors were differentially associated with certain facets of anxiety sensi-

tivity. In particular, health-related stressors were predictive of fears of disease and mental incapacitation, whereas stressors associated with family discord predicted fears of unsteadiness, mental incapacitation, and social concerns. To our knowledge, this is the first study to document associations between specific stressors and unique facets of anxiety sensitivity. Given the novelty of these results, little is known regarding the reasons underlying the differential associations between types of stress and anxiety sensitivity. However, existing research on stressful life events points to several possibilities. For example, the perception that stressors portend danger is uniquely associated with the development of anxiety disorders (Brown, 1993). Health-related stressors may be particularly likely to elicit feelings of impending danger, which could in turn lead to greater attention to, and fears surrounding, the anxiety symptoms that result from these stressors. These types of stressors may be particularly likely to increase attention to symptoms that appear to portend disease or health problems. In addition, the stress of family discord is associated with chronic emotional arousal and increased reactivity to stressors (for a review, see Repetti, Taylor, & Seeman, 2002), factors that may be particularly likely to produce anxiety sensitivity. Adolescents reared in adverse family environments also are likely to have low perceptions of control over their environment (Chorpita & Barlow, 1998), which may predispose them to the development of negative beliefs about losing control of physical and mental states, specifically, consistent with our findings regarding concerns about mental incapacitation and feeling unsteady. Moreover, stress associated with dysfunctional family relationships can disrupt the normative development of social competence (Repetti et al., 2002), which may render some adolescents more vulnerable to developing fears of the social consequences of anxiety that were evidenced in the current study. These hypotheses should be pursued in future studies examining relations between the type and meaning of stress and the development of anxiety sensitivity during adolescence.

These findings extend the literature on stress and adolescent psychopathology in several important ways. Adolescence represents an important period in which to examine mechanisms linking stress to the development of psychopathology. Adolescence is characterized by higher risk for the development of psychopathology (Hankin et al., 1998; Lewinsohn, Striegel-Moore, & Seeley, 2000; Twenge & Nolen-Hoeksema, 2002) than previous points in development, and stressful events become more closely linked to the emergence of negative affect during this period, rendering adolescents more emotionally vulnerable to the effects of stress (Larson & Ham, 1993; Larson, Moneta, Richards, & Wilson, 2002). The relation between stress and psychopathology among children and adolescents is well-established (Grant et al., 2004); however, to date, research examining the mechanisms by which stress leads to increased psychiatric morbidity has been lacking, hindering the development of effective, preventive interventions for children and adolescents exposed to stressful life events. Our findings suggest one intrapersonal mechanism linking stress to anxiety symptomatology. Specifically, elevated perceptions of anxiety symptoms as dangerous and portending negative physiological consequences served as a mechanism underlying the relation between stressful life events and anxiety symptoms. The global anxiety sensitivity factor, as well as concerns about disease and mental incapacitation, mediated the association between stress and the development of anxiety symptoms.

These results have potentially important treatment implications. In particular, they suggest that preventive interventions that focus on attenuating anxiety sensitivity may help to reduce stress-related psychiatric morbidity in adolescents. Evidence-based treatments such as cognitive-behavioral (Barlow, 2002) and emotion regulation (Mennin, 2004) interventions may be particularly effective in enabling adolescents to more adaptively challenge and manage their negative beliefs about the harmfulness of anxiety symptoms following stressful experiences. Stress inoculation training (Meichenbaum, 1985) represents an additional evidence-based intervention that may prove effective in decreasing adolescents' anxiety sensitivity following stress. This training teaches a variety of coping skills that are applied and practiced with target fears, which could include beliefs about the deleterious consequences of anxiety symptoms subsequent to life stressors. Finally, two recent prevention programs specifically targeting anxiety sensitivity showed promising results in reducing levels of anxiety sensitivity (Feldner, Zvolensky, Babson, Leon-Feldner, & Schmidt, 2009; Schmidt et al., 2007), thereby providing an important template for how future interventions can address anxiety sensitivity among adolescents who have been exposed to stressful life events.

A final contribution of this study was the examination of differential relations between anxiety sensitivity and depressive and anxious symptoms in adolescents. Prior research examining the predictive specificity of anxiety sensitivity has been inconsistent, with some research documenting that the association between anxiety sensitivity and depressive symptoms disappears after anxiety symptoms are added to the model (Pollock et al., 2002; Schmidt et al., 1998), and others reporting a positive association between anxiety sensitivity and depression (Otto et al., 1995; Taylor et al., 1996; Weems et al., 1997). Existing studies among adolescents have relied exclusively on cross-sectional data (Weems et al., 1997), which might obscure the true relations between anxiety sensitivity and depression during this developmental period and have failed to examine the impact of the different facets of anxiety sensitivity on symptoms of anxiety and depression. Anxiety sensitivity was associated longitudinally with the development of anxiety symptoms in our sample, and this association was explained largely by the general anxiety sensitivity factor. Concerns about feeling unsteady also contributed to the longitudinal relation between anxiety sensitivity and anxiety symptoms. In contrast, anxiety sensitivity was not associated longitudinally with symptoms of depression when covariates for anxiety were included in the model. These findings indicate that anxiety sensitivity serves as a unique vulnerability factor for anxiety, but not depressive symptoms, among adolescents.

This study had a number of important methodological strengths that contribute to our understanding of anxiety sensitivity among adolescents and that expand on the literature examining mechanisms linking stressful life events and adolescent psychopathology. In particular, the use of a longitudinal design allowed us to examine the role of stressful life events in the development of anxiety sensitivity, to examine anxiety sensitivity as a potential mechanism responsible for the association between stressful life events and increases in anxiety symptoms over time (Maxwell & Cole, 2007), and to determine whether anxiety sensitivity was associated with increases in depressive symptoms over time in addition to anxiety. A large sample with substantial racial and ethnic diversity participated. However, limitations of

the current study must also be acknowledged. The first is our use of self-reported symptomatology. Although the validity of the self-report measures used in this study is well-established (Timbremont, Braet, & Dreessen, 2004; Wood, Piacentini, Bergman, McCracken, & Barrios, 2002) and allowed us to capture the dimensional nature of internalizing symptoms in adolescents, in future studies, these results should be replicated with structured clinical interviews to establish *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994) diagnoses.

Our use of a self-report checklist of stressful life events represents an additional limitation. It is important to note that the questionnaire in our study is among the most widely used self-report measures of stressful life events in adolescence (Hammen, 2008). Additionally, the stressors measured are predominantly external, environmental changes or conditions and, consequently, are not confounded with subjective appraisals of the stressor (Grant et al., 2003). Further, stress checklists represent the most reliable methodology for assessing stress in large community samples in which stressor interviews are prohibitive in time and cost (Grant et al., 2004). Nevertheless, stressor interviews, which capture more objective indices of stressors as well as the level of threat associated with these stressors, would represent a methodological improvement and should therefore be used in future studies (Hammen, 2008). In particular, stressor interviews could establish timing of stressor onset and duration of stressors in relation to the development of anxiety sensitivity and anxiety disorders, an important avenue for further study. In addition, the stress measure we used did not allow us to examine trajectories of stressful life events over time, given that it asked about stressful life events in the preceding 6 months, and our assessment intervals were spaced at shorter intervals. Future research examining trajectories of stressful life events may help us to further elucidate the relation between stress and anxiety sensitivity to determine whether chronic versus episodic stress is more strongly related to the development of anxiety sensitivity. Finally, these results warrant replication. We identified a sample on the basis of the willingness of the school district to participate and the diversity of the student body. Replication is necessary to ensure that these findings generalize to other samples of adolescents.

In sum, the current study identified stressful life events as a factor related to the development of elevated anxiety sensitivity among adolescents. Specific types of stressors were found to differentially predict increases in specific facets of anxiety sensitivity; health-related stressors predicted increases in disease-related concerns and fear of mental incapacitation, whereas stressors related to family discord predicted increases in fear of feeling unsteady, fear of mental incapacitation, and fear of having publicly observable symptoms of anxiety. In addition, anxiety sensitivity was uniquely related to prospective increases in anxiety symptoms but not symptoms of depression, confirming previously reported cross-sectional associations in a longitudinal design. Finally, this study identified anxiety sensitivity as a mechanism linking stressful life events to increases in anxiety symptoms among adolescents, suggesting important avenues for intervention research targeting stress-related symptomatology among adolescents.

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