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Anxiety sensitivity: Prospective prediction of anxiety among early adolescents

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ABSTRACT

Emerging evidence suggests that anxiety sensitivity (AS) predicts subsequent development of anxiety symptoms and panic attacks as well as clinical syndromes in adult samples. The primary aim of the present study was to determine whether AS similarly acts as a vulnerability factor in the pathogenesis of anxiety symptoms among youth in early adolescence (ages 9–13). A large nonclinical community sample of youth ($n = 277$) was prospectively followed over 1 year. The Childhood Anxiety Sensitivity Index (CASI: Silverman, Fleisig, Rabian, & Peterson, 1991) served as the primary predictor. After controlling for baseline anxiety symptoms as well as depression, AS significantly predicted the future development of anxiety symptoms. Consistent with the adult literature and expectancy theory, AS appears to act as a risk factor for anxiety symptoms in youth.

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Expectancy theory (Reiss, 1991) and contemporary psychosocial accounts of anxiety psychopathology posit that fear of anxiety (i.e., anxiety sensitivity; AS) is important in the development and maintenance of these conditions (Barlow, 2002; Clark, 1986; McNally, 1990). AS is a relatively stable cognitive characteristic that predisposes individuals to the development of anxiety problems (Taylor, 1999); it encompasses fears of physical, mental, and publicly observable experiences (Zinbarg, Barlow, & Brown, 1997), all of which are believed to amplify pre-existing anxiety (Reiss, 1991). Thus, individuals with high AS are theorized to perceive bodily sensations associated with autonomic arousal as a sign of imminent personal harm and, as a result, to experience elevated levels of anxiety and be at increased risk for a panic attack.

Individual differences in AS are hypothesized to emerge from the combined influences of genetic variation along with any number of learned experiences that ultimately lead to the acquisition of beliefs about the potentially aversive consequences of arousal and anxiety-related states (Reiss & Havercamp, 1998). Research conducted across diverse populations has supported the AS model, providing strong evidence of cross-cultural and developmental specificity in terms of the latent structure and stability of the construct (Chorpita & Daleiden, 2000; Muris, Schmidt, Merckelbach, & Schouten, 2001; Zinbarg, Brown, & Barlow, 2001; Zvolensky, Kotov, Antipova, Leen-Feldner, & Schmidt, 2005; Zvolensky, Goodie, McNeil, Sperry, & Sorrell, 2001). AS is also unique from, and demonstrates incremental validity to, trait anxiety (Rapee & Medoro, 1994) as well as negative affectivity (Zvolensky et al., 2005a, 2005b).

Unlike many other cognitive conceptualizations of anxiety, AS is believed to be a dispositional characteristic that may precede the development of clinical anxiety symptoms or diagnoses. Empirical studies provide converging evidence that AS does indeed act as a risk factor for anxiety problems. First, laboratory studies indicate that baseline AS predicts fear responses to bodily sensations (Rabian, Embry, & MacIntyre, 1999; Unnewehr, Schneider, Margraf, & Jenkins, 1996; Zvolensky et al., 2001a, 2001b); these effects are observed above and beyond variance accounted for by trait anxiety (Zinbarg et al., 2001). Second, AS levels are elevated among individuals with anxiety disorders compared to those without anxiety disorders (Kearney, Albano, Eisen, Allan, & Barlow, 1997; Rabian, Peterson, Richters, & Jensen, 1993; Taylor, Koch, & McNally, 1992). Third, prospective studies with healthy adults (Schmidt, Lerew, & Jackson, 1997, 1999) and adolescents (Hayward, Killen, Kraemer, & Taylor, 2000) indicate that AS predicts the future occurrence of anxiety symptoms and panic attacks, even after controlling for trait anxiety. Additionally, AS predicts the maintenance of panic disorder among untreated patients, the prospective emergence of panic attacks among infrequent (nonclinical) panickers, and the emergence of panic among individuals free from a history of panic attacks (Ehlers, 1995), thereby indicating that AS is a predictor of (future) panic-related problems over time. Finally, AS has been found to prospectively predict the development of clinically significant anxiety syndromes (Schmidt, Zvolensky, & Maner, 2006).

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Work on AS has expanded dramatically as accumulating evidence supports its relationship to anxiety psychopathology. Although the vast majority of studies on AS have focused on college age or adult samples, AS research among children and adolescents has recently emerged and is consistent with the more extensive adult AS literature. For example, AS has been associated with general anxiety symptoms in both children and adolescents and has shown specificity to anxiety over depression (e.g., Joiner et al., 2002; Lambert, Cooley, Campbell, Benoit, & Stansbury, 2004). AS is also associated with symptoms of various anxiety disorders in youth. Specifically, cross-sectional research with children and adolescents has indicated that AS is associated with panic symptomatology (Calamari et al., 2001; Deacon, Valentiner, Gutierrez, & Blacker, 2002) and with panic disorder (Kearney et al., 1997; Lau, Calamari, & Waraczynski, 1996). Further, research with trauma exposed children and adolescents indicates that AS is associated with PTSD symptoms above and beyond trauma exposure (Hensley & Varela, 2008; Leen-Feldner, Feldner, Reardon, Babson, & Dixon, 2008). Biological challenge studies have further validated this construct among nonclinical youth by demonstrating that AS is predictive of fearful reactivity to voluntary hyperventilation paradigm among adolescents (Leen-Feldner, Feldner, Bernstein, McCormick, & Zvolensky, 2005) and to a stair stepping challenge among children (Rabian et al., 1999).

Similar to the adult AS literature, the prospective evaluation of AS among community samples of youth is limited. There are two relevant studies with youth well into adolescence. In a 6-month longitudinal investigation among African-American adolescents (M age = 15.6), Ginsburg and Drake (2002) reported that AS was associated with elevations in panic symptoms. However, baseline AS levels did not predict subsequent panic symptoms after controlling for baseline panic symptoms. Hayward et al. (2000) conducted a 4-year, longitudinal study within a large diverse sample of high school students (M age = 15.4). Results indicated that elevated AS predicted the future occurrence of 4-symptom panic attacks. AS also predicted more severe panic attacks and anxious avoidance (Wilson & Hayward, 2006). Additionally, Hayward et al. (2000) reported that when controlling for relevant factors, AS showed specificity in predicting panic, but not depression. Further exploration of this sample indicated that those with stable high or escalating AS over time were more likely to have experienced a panic attack; however, panic was not a strong predictor of future AS scores (Weems, Hayward, Killen, & Taylor, 2002).

To date, Weems, Costa, Watts, Taylor, and Cannon (2007) have conducted the only longitudinal investigation of AS including early adolescents (n = 145; M = 11.36 years). Among this ethnically diverse sample, 52 participants completed a follow-up assessment 1 year later (M = 11.15 years), where baseline AS was predictive of anxiety disorder symptomatology as measured by the Revised Child Anxiety and Depression Scales (RCADS) at the 1-year follow-up. Although this investigation meaningfully contributes to the prospective AS literature among youth, the follow-up sample was a small subset of the original sample, and due to limited details, it is difficult to determine the extent to which this subset differs systematically from the overall sample. Perhaps most importantly, the analyses did not control for baseline anxiety, which renders the predictive validity of the CASI in this case largely uninterpretable (i.e., it is unclear if AS is a predictor of change in anxiety symptoms).

The extant literature examining the effects of AS on youth is consistent with the adult literature and suggests that AS may act as a premorbid risk factor for the genesis of anxiety problems among children and adolescents. However, this has not been clearly demonstrated due to the limited prospective investigations of AS among youth, particularly among youth in early adolescence. This is particularly notable given the age of onset for many anxiety disorders tends to peak in adolescence or early adulthood (American Psychiatric Association, 2000; Kessler et al., 2005). Thus, it is crucial to prospectively investigate AS in this age group to truly evaluate AS as a risk factor in the development of these anxiety disorders.

Together, the current study provides a more definitive evaluation of whether AS acts as a premorbid risk factor for the pathogenesis of anxiety problems by including a large nonclinical sample of youth in early adolescence. The study prospectively followed 277 nonclinical participants ages 9–13 over a 1-year time frame. It was hypothesized that AS would act as a cognitive diathesis, placing individuals at risk for the development of anxiety during the follow-up period, even after controlling for baseline levels of anxiety (see Lilienfeld, Turner, & Jacob, 1993). More specifically, it was hypothesized that, independent of baseline levels of anxiety, AS would predict the changes in anxiety symptoms during follow-up. We also predicted that AS would show specificity in terms of being relatively more strongly associated with anxiety versus mood symptoms over time.

1. Method

1.1. Participants

Data were collected from a community sample of 277 early adolescents and their primary caregiver (80.5% mothers) participating in a larger prospective study of behavioral, environmental, and genetic mechanisms of risk for HIV-related risk behaviors in youth. Of the original 277 subjects, 5 subjects (1%) were deleted due to incomplete data specific to hypotheses in this manuscript. These 5 subjects did not significantly differ from the retained subjects on any demographic variable. The final sample consisted of 272 subjects. At baseline, the average age of this sample was 11.00 years (SD = .82; range, 9–13 years) and 44.1% (n = 120) of the sample was female. In terms of racial/ethnic background, 49.4% (n = 134) of the sample indicated European American, 35.1% (n = 95) indicated African American, 3.0% (n = 8) indicated Hispanic, and 12.5% (n = 35) indicated other. At follow-up, 31 subjects did not return for the study (11%). These 31 dropout subjects did not significantly differ from the retained subjects on any demographic variable or major study variable (follow-up n = 244).

Permission to conduct research was obtained from the University of Maryland Institutional Review Board (IRB). Families were recruited through media outreach in the greater Washington, DC metropolitan area, as well as through contact with area schools, libraries, and Boys and Girls Clubs. The study was advertised as an investigation of youth health-related behaviors. Interested families called to set up an appointment and were informed over the phone that we were conducting a study examining youth risk-taking behaviors. Families were eligible for participation if the child was in 5th or 6th grade and both primary caregiver and child were fluent in English. Interested families who met inclusion criteria were invited to come to the Center for Addiction, Personality, and Emotion Research (CAPER) located in the University of Maryland campus. Upon arrival at the assessment session, a more detailed description of the study procedures was provided and the primary caregivers and youth signed informed consent/assent.

1.2. Measures

1.2.1. Demographics

The parent/guardian completed a basic demographics form for personal information, as well as information about the child. The form included age, gender, race, status of biological father's presence in the home (yes/no), and annual family income. The total family income variable was positively skewed (s = 4.81, SE = .15); thus, a square root transformation was conducted (Tabachnick & Fidell, 1996). The resulting skew was less than 2 (s = .94, SE = .15).

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