ELSEVIER

Journal of Anxiety Disorders 22 (2008) 899–914

Journal of Anxiety Disorders

Etiology of the dimensions of anxiety sensitivity: A behavioral–genetic analysis☆

Steven Taylor [a,*], Kerry L. Jang [a], Sherry H. Stewart [b], Murray B. Stein [c]

[a] Department of Psychiatry, University of British Columbia, Vancouver, BC V6T 2A1, Canada
[b] Departments of Psychiatry and Psychology, Dalhousie University, Canada
[c] Department of Psychiatry, University of California, San Diego, CA, USA

Received 19 April 2007; received in revised form 7 September 2007; accepted 12 September 2007

Abstract

Evidence suggests that anxiety sensitivity (AS) contributes to individual differences in fearfulness and to the risk of developing anxiety disorders. To investigate the origins of AS we administered the Anxiety Sensitivity Index to 245 monozygotic and 193 dizygotic twin pairs, comprising 658 women and 218 men. Scores were calculated for the most widely replicated AS dimensions; physical, cognitive, and social concerns. For women, each dimension was influenced by a combination of genetic and environmental factors. Heritability in women significantly increased with AS scores, indicating that severe forms of AS, compared to milder forms, are more strongly influenced by genetic factors. Correlations among AS dimensions for women could be explained by genetic and environmental factors influencing all three dimensions. For men, dimensions were influenced by environmental but not genetic factors. Correlations among dimensions for men could be explained by environmental factors influencing all dimensions. Overall, the findings reveal that AS has more complex etiology than previously recognized; its dimensions appear to arise from a mix of dimension-specific and non-specific etiologic factors, whose importance vary as a function of sex and severity.

Keywords: Anxiety sensitivity; Anxiety Sensitivity Index; Anxiety disorders; Behavioral–genetics; Twin study

1. Introduction

Anxiety sensitivity (AS) is the fear of arousal-related sensations, arising from beliefs that the sensations are harmful (Reiss & McNally, 1985). A growing body of evidence suggests that AS contributes to individual differences in fearfulness and to the risk of developing anxiety disorders (Maller & Reiss, 1992; Schmidt, Zvolensky, & Maner, 2006; Taylor, 1999). AS is said to have these properties because it is an ''anxiety amplifier''; when highly anxiety-sensitive people become anxious they become alarmed about their arousal-related sensations, which further intensifies their anxiety (Reiss, 1991).

The most widely replicated AS factor structure consists of three intercorrelated phenotypic dimensions labeled *physical concerns*, *cognitive concerns*, and *social concerns* (e.g., Deacon & Valentiner, 2001; Sandin, Chorot, Valiente, Santed, & Lostao, 2004; Taylor et al., 2007; Zinbarg, Barlow, & Brown, 1997). To illustrate the dimensions, high scores on physical

☆ This research was supported in part by New Emergent Team grant PTS-63186 from the Canadian Institutes of Health Research Institute of Neurosciences, Mental Health, and Addiction.
* Corresponding author. Tel.: +1 604 822 7331; fax: +1 604 822 7756.
E-mail address: taylor@unixg.ubc.ca (S. Taylor).

0887-6185/$ – see front matter
doi:10.1016/j.janxdis.2007.09.005

concerns are associated with the beliefs that palpitations or breathlessness lead to physical collapse; high scores on cognitive concerns are associated with the beliefs that difficulties in concentrating or in controlling one's thoughts lead to mental incapacitation; and high scores on social concerns are associated with beliefs that blushing or trembling will elicit social rejection.

The three dimensions can be conceptualized in two ways; either as a constellation of interrelated dimensions (non-hierarchical model) or as a group of lower order factors loading on a single higher order factor (hierarchical model). The two models are statistically equivalent in terms of their goodness-of-fit to the data (Brown, 2006). In the present study we focused primarily on the conceptually simpler, non-hierarchic model and investigated the genetic and environmental causes of the covariance among dimensions. (For the sake of completeness, however, the hierarchical model was also examined. As we will see, it yielded very similar findings to the non-hierarchic model regarding the role of genetic and environmental factors.)

In addition to its dimensional features, AS has categorical properties. Taxometric research suggests two distinct types of anxiety-sensitive people; a group characterized by high scores, comprising 10–20% of community respondents, and low AS group comprising the remainder (e.g., Bernstein, Zvolensky, Feldner, Lewis, & Leen-Feldner, 2005; Bernstein, Zvolensky, Kotov, et al., 2006; Bernstein, Zvolensky, Weems, Stickle, & Leen-Feldner, 2005). Dimensional and categorical structures are quite compatible with one another (Schmidt, Kotov, & Joiner, 2004; Waller & Meehl, 1998). In fact, dimensions of AS have been identified within each AS category (Bernstein et al., 2007).[1]

The reasons behind the dimensional/categorical nature of AS are currently unclear. The variables contributing to a person's severity on particular AS dimensions could be quite different from the variables contributing to whether a person falls in the high or low AS group. Etiologic factors contributing to the dimensions might include (a) additive genetic influences (i.e., the genes that each make a small but additive contributions to a person's level of severity on a given dimension), or (b) cumulative environmental events (i.e., learning experiences that incrementally strengthen a person's belief about the dangerousness of particular types of arousal-related sensations). Etiologic factors contributing to whether a person falls in the high or low AS group could include (a) non-additive genetic effects (i.e., dominance or epistasis), (b) additive genetic influences that have threshold characteristics (i.e., the difference between low and high AS could be determined by whether a person exceeds some critical threshold in terms of a number of particular types of genes), or (c) ''environmental molds'' (Meehl, 1992), which include learning experiences that tend to cluster together and therefore, as a group, tend to be either present or absent (e.g., the presence or absence of severe childhood illnesses that provide salient early learning experiences about the dangerousness of arousal-related sensations).

Research on the origins of AS is important because it may shed light on the etiology of individual differences in fearfulness, and may offer insights into the nature of vulnerability factors for anxiety disorders. The present study is an extension of our earlier work (Jang, Stein, Taylor, & Livesley, 1999; Stein, Jang, & Livesley, 1999), which to our knowledge are the only published studies that simultaneously examined genetic and environmental factors in AS. Those studies offered preliminary evidence that AS is heritable (Stein et al., 1999) and, more particularly, heritable only in women (Jang et al., 1999). Those studies were based on data from 337 twin pairs. In the present study we collected further data to increase the sample to 438 pairs in order to provide a more powerful and reliable evaluation of sex differences in the etiology of AS.[2] The present study also examined issues that were not studied in our previous research. More specifically, purposes of the present study were to investigate, for each sex, the role of genetic and environmental influences (a) on the variance of each AS dimension, (b) on the interrelationships among dimensions (i.e., to investigate whether the covariances among dimensions are due to genetic or environmental factors that influence all three dimensions), and (c) on the categorical structure of AS. To investigate the latter we examined the heritability of each AS dimension across a range of severity levels, up

[1] Bernstein et al. (2007) found that the three-dimensional structure was replicated in the complement group, but a one-dimensional structure was obtained in the taxon (high AS) group. The latter result was quite possibly an artifact of range restriction (i.e., the taxon group consisted of people scoring above the 80th percentile and therefore had a very narrow range of scores). Research indicates that the three-dimensional solution is the most robust (generalizable) factor structure (Taylor et al., 2007).

[2] Throughout this article we define ''sex differences'' broadly to include effects due to biological sex (i.e., sex-limited genetic factors) and effects related to gender socialization or other environmental factors.