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Phenotypic and genetic structure of anxiety sensitivity in adolescence and early adulthood

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ABSTRACT

Anxiety sensitivity is a risk factor for emotional disorders. The structure of anxiety sensitivity was examined using phenotypic and genetic analyses. Self-reported anxiety sensitivity was measured at three time points from adolescence into young adulthood by 2651 individuals from the G1219 twin study. Confirmatory factor analyses revealed comparable statistical support for anxiety sensitivity models consisting of three or four dimensions across all time points. The three-factor model depicting Physical, Social and Mental anxiety-related concerns was favoured due to greater interpretability and parsimony. Multivariate quantitative genetic analyses supported a hierarchical structure with general genetic (.09–.61) and non-shared environmental (.39–.72) influences acting via a higher-order factor as well as dimension-specific genetic (.09–.21) and non-shared environmental (.23–.68) influences. The findings provide further evidence for a hierarchical structure underlying different dimensions of anxiety sensitivity.

1. Background

Anxiety sensitivity is an enhanced sensitivity towards physical and emotional symptoms of anxiety with a belief that these are harmful. Research implicates anxiety sensitivity as a risk factor for a range of emotional disorders, especially anxiety disorders, in both adults and children (see Cox, Borger, & Enns, 1999 for a review). Associations between anxiety sensitivity and a diverse range of emotional disorders highlights the importance of examining the underlying structure of anxiety sensitivity. It may be that specific dimensions of anxiety sensitivity depict particular mechanisms which have differential roles across anxiety and emotional disorders.

2. Phenotypic structure of anxiety sensitivity

Factor analyses of both the adult Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986) and the Children's Anxiety Sensitivity Index (CASI; Silverman, Fleisig, Rabian, & Peterson, 1991) support the notion of multiple dimensions. However, several possible factor solutions are suggested. Some support a unitary construct (Reiss et al., 1986; Taylor, Koch, McNally, & Crockett, 1992; Taylor, Koch, & Crockett, 1991), whilst others reveal a multifaceted construct with two (Chorpita & Daleiden, 2000), three (Silverman, Ginsburg, & Goedhart, 1999; Zinbarg, Barlow, & Brown, 1997) or four dimensions (Silverman, Goedhart, Barrett, & Turner, 2003). Furthermore, studies identifying multiple dimensions diverge in conclusions regarding the relationships between these dimensions. Some studies suggest distinct (orthogonal) facets (Wardle, Ahmad, & Hayward, 1990) whilst others suggest a number of correlated (oblique) dimensions (Cox, Parker, & Swinson, 1996; Muris, Schmidt, Merckelbach, & Schouten, 2001).

More recently, factor analyses have implicated a hierarchical structure of anxiety sensitivity in which all anxiety sensitivity dimensions derive from a higher-order, general anxiety sensitivity factor. For example, a large exploratory factor analysis in adolescence suggested a hierarchical model consisting of three dimensions representing 'Physical', 'Social' and 'Mental' anxiety-related concerns (Walsh, Stewart, McLaughlin, & Comeau, 2004). This model of anxiety sensitivity was subsequently favoured in a large confirmatory factor analysis comparing the relative goodness of fit to the data of previous two-, three- and four-factor models (Wright et al., 2010). However, the three-factor hierarchical model had only a marginal fit improvement compared with a four-factor model and, as a result the underlying phenotypic structure of anxiety sensitivity remains unclear. A similar model has been identified in an adult sample which shows stability across a ten-month period (Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004) although, to date, there are no longitudinal studies

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regarding the structure of anxiety sensitivity across development.

3. Genotypic structure of anxiety sensitivity

Another approach to examining the structure of a phenotype is to explore the structure of genetic and environmental influences on that construct. Twin studies reveal moderate genetic influence on total anxiety sensitivity in child (37%; Eley, Gregory, Clark, & Ehlers, 2007), adolescent (46%; Zavos, Rijsdijk, Gregory, & Eley, 2010) and adult (45%; Stein, Jang, & Livesley, 1999) samples but, to date, there have been no twin studies on the dimensions of anxiety sensitivity in children or adolescents. In adults, univariate analyses of three anxiety sensitivity dimensions identified in exploratory factor analysis ($N = 337$ twin pairs; Stein et al., 1999) estimated heritability of 35% and 22% for factors representing 'Physical' and 'Social' anxiety-related concerns, respectively whilst 'Psychological Concerns' was solely accounted for by environmental influences; 11% shared environment (factors that contribute to making children in the same family similar) and 89% non-shared environment (factors making children within a family dissimilar).

Subsequent multivariate genetics analyses using an extended sample ($N = 438$ twin pairs; Taylor, Jang, Stewart, & Stein, 2008) examined the etiological relationships between dimensions of anxiety sensitivity by comparing competing genotypic models. These models mirror those from phenotypic analyses. For example, genetic correlated factor models reflect oblique phenotypic factor solutions because they assume dimension-specific etiological influences are correlated with one another. Similarly, common pathway models mimic hierarchical phenotypic models, assuming a higher-order, global "anxiety sensitivity" factor through which genetic and environmental factors influence lower-order dimensions. This study found comparable support for both independent and common pathway models of anxiety sensitivity in females whilst the common pathway model was superior for males. These models provide preliminary evidence for differential contributions of genetic and environmental influences across dimensions of anxiety sensitivity, and thus support a multifaceted view. Furthermore, evidence for a common pathway model suggests that anxiety sensitivity may be best represented as a hierarchical structure.

4. Study summary

Previous phenotypic factor analyses support a hierarchical model of anxiety sensitivity but the number of lower-order dimensions and stability across development remains unclear. Genetic methods may help elucidate the structure of anxiety sensitivity but to date these have only been used with adult samples. Analyses to date provide preliminary support for a hierarchical structure in which common genetic and environmental factors influence all anxiety sensitivity dimensions via a latent factor. The current study extended previous research by (i) investigating the structure of anxiety sensitivity at three time points spanning early adolescence to early adulthood and (ii) using both phenotypic and genetic analyses to explore the structure of anxiety sensitivity at these three time points. Phenotypic factor analyses were expected to support a three-factor model in-line with work by Walsh et al. (2004) and Wright et al. (2010). It was also hypothesised that these three factors would share common genetic and environmental influences at all time points and be best described by a common pathway model, reflecting the phenotypic hierarchical structure. Additionally, it was anticipated, although more tentatively, that phenotypic and genetic structures of anxiety sensitivity would remain stable across all time points in-line with longitudinal phenotypic (e.g. Weems, Hayward, Killen, & Taylor, 2002) and genetic (Zavos, Gregory, & Eley, 2012) studies of total anxiety sensitivity.

5. Methods

5.1. Participants

The present analyses use data from the G1219 study. This is a longitudinal study of 3640 adolescent twins and siblings aged between 12 and 19 at initial contact. Informed consent was obtained from parents of all adolescents under 16 and from adolescents themselves when over 16. Ethical approval was obtained from the Research Ethics Committee of the Institute of Psychiatry and South London and Maudsley National Health Service Trust for the first three time points and also from Goldsmiths, University of London at time four. The sample was recruited from two sources. First, adolescent offspring of adults from a large-scale population-based study (GENESiS; Sham et al., 2000) were invited to participate in this or another study (Curran et al., 2003). Of the 3600 responses, 1818 adolescents (51%) from 1294 families agreed to participate in G1219. Second, a random selection of live twin births born between 1985 and 1988 identified by the UK Office of National Statistics were recruited by Health Authorities and General Practitioners on behalf of the G1219 team. Of the 2947 families contacted, 1381 (47%) participated. Only respondents aged 12- to 19-years were included within the final sample.

Questionnaires were sent to participants at four time points. At wave one, contact invitations included questionnaires to be completed by adolescents and their parents. At wave two (approximately 8 months after initial contact), a second set of questionnaires was sent. This included a questionnaire assessing the physical similarity between pairs to establish zygosity of the twins within the sample (Cohen, Dibble, Grawe, & Pollin, 1975). If there was disagreement between zygosity ratings between wave one and two, DNA was obtained ($N = 26$ pairs) before final classifications were made. Additional data was collected three (wave 3) and five (wave 4) years after the initial recruitment wave.

Analyses reported here focus on the second, third and fourth waves of data collection; hereon referred to as times one, two and three, respectively. At time one, total data was available from 2651 individuals from 1372 families (73% of the original sample) whilst corresponding figures for times two and three were 1597 adolescents from 824 families (44% of the original sample) and 1556 adolescents from 896 families (43% of original sample), respectively. Of these 330, 182 and 201 were sibling pairs, 350, 324 and 230 were MZ twins, 313, 207 and 214 were DZ same-sex twins, 334, 232 and 232 were DZ opposite-sex twins and 45, 11 and 19 were of unknown zygosity at times one, two and three, respectively. The mean ages of the sample were 15 years (range 12–21), 17 years (range 14–23) and 20 years (range 18–27) at time one, two and three, respectively; 51.7% were female at time one, 43.9% at time two and 61.5% at time three.

5.2. Measures

Anxiety sensitivity was measured using the Children's Anxiety Sensitivity Index (CASI; Silverman et al., 1991) at times one and two and the Anxiety Sensitivity Index (ASI; Reiss et al., 1986) at time three. The CASI is an 18-item self-report questionnaire assessing fear of anxiety sensations in children and includes items such as "It scares me when I feel shaky." The CASI was adapted from the ASI, a 16-item questionnaire assessing fears of anxiety sensations in adults. The main differences are the simplicity of language and addition of two extra items ("I don't like to let my feelings show" and "Funny feelings in my body scare me") in the CASI. Additionally,