ORIGINAL RESEARCH—WOMEN'S SEXUAL HEALTH

The Etiological Relationship Between Anxiety Sensitivity, Sexual Distress, and Female Sexual Dysfunction Is Partly Genetically Moderated

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ABSTRACT

Introduction. Presence of sexual distress is diagnostic requirement for female sexual dysfunction (FSD). However, previous correlational research indicates that sexual distress in women may be related to general anxiety per se rather than being an outcome of FSD.

Aim. In this exploratory study, we test, for the first time, whether the correlation between anxiety sensitivity, sexual distress, and FSD can be explained by shared genetic and nongenetic factors using multivariate twin modeling.

Methods. Questionnaire data were available on a representative final sample of 930 Caucasian British female twin individuals (119 monozygotic twin pairs, 67 dizygotic twin pairs, and 558 single twins; aged 18–85 years). Validated scales assessed anxiety sensitivity, sexual distress, and FSD and included the Female Sexual Function Index, the Female Sexual Distress Scale, and the Anxiety Sensitivity Index.

Main Outcome Measures. Questionnaire responses were subject to trivariate heritability analyses to assess common genetic and environmental influences underlying specific trait variance and the covariance between the phenotypes.

Results. Heritability for FSD was 28%, 48% for anxiety sensitivity, and 44% for sexual distress. The phenotypic associations among anxiety sensitivity, sexual distress, and FSD were all significant. Trivariate analysis indicated that additive genetic factors accounted for approximately 75% of the covariance between anxiety sensitivity and FSD 35% of the covariance between anxiety sensitivity and sexual distress, and 11% between sexual distress and FSD.

Conclusions. The association between anxiety sensitivity and FSD has a common genetic component. There is a weaker genetic link between anxiety sensitivity and sexual distress and between sexual distress and FSD. These data, while silent on direction of causality, suggest a role for pleiotropic genetic factors influencing anxiety sensitivity and FSD. They also highlight a need to refine the inclusion of distress in classifications of disorders of female sexual functioning. **Burri A, Spector T, and Rahman R. The etiological relationship between anxiety sensitivity, sexual distress and female sexual dysfunction is partly genetically moderated. J Sex Med 2012;9:1887–1896.**

Key Words. Sexual Distress; Female Sexual Dysfunction; FSD; Anxiety; Genetics; Female Sexual Function Index

Introduction

According to the Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV, female sexual dysfunction (FSD) falls into the four categories of desire, arousal, orgasm, and pain disorders. In 1994, "marked distress or interpersonal difficulty" was added to the criteria sets for all the sexual dysfunctions in the DSM-IV in order to separate them from a normal variant of functioning [1]. In addition, the International Consensus Development Conference held in 1998 suggested that a woman should show evidence of significant negative feelings and anxiety in relation to her presenting sexual problem, in order to receive an FSD diagnosis [2,3]. Sexual dysfunctions *not* experienced as distressing, or which do not cause considerable interpersonal problems (in terms of

intimate relations), are not considered disorders under these definitions. Indeed, does the extant empirical evidence show that sexual problems do not robustly correlate with distress, even when controlling for severity [4–10]. A landmark study in a nationally representative household sample of over 30,000 U.S. women found that self-reported prevalence of low sexual arousal decreased from 25.3% to 3.3–6% (depending on age) when including distress as assessed with the Female Sexual Distress Scale (FSDS) [10,11]. A community sample study involving 500 U.S. women reported that 16% of women aged 20–49 had low sexual desire compared with only 7% when personal distress was included in the diagnosis [12], while a study of women attending British general practices (local community doctors) reported that of 38% of women with International Classification of Diseases (ICD-10) diagnoses of sexual dysfunction, only 18% of women perceived that they had a problem [7] (see Graham, 2010, for a comprehensive review; [8]). We have recently shown a robust association between self-reported anxiety, obsessive behavior symptoms, and sexual distress independent of FSD [13]. High prevalence of FSD have also been found in women with anxiety disorders [14–16]. In a large community-based epidemiological survey, Dunn et al. reported that women with moderate to high scores on a self-report measure of anxiety were at a significantly higher risk for arousal difficulties in particular [16]. A comparable British study (N = 1,489) did not replicate this association but reported that high levels of general anxiety were associated with lifelong lubrication problems and poor orgasm experiences [17]. The association between anxiety and FSD symptoms can also be seen in objective measures of genital arousal. Bradford and Meston reported that moderate state anxiety scores in healthy women were related to greater vaginal pulse amplitude (VPA), responses whereas low and high anxiety scores were associated with lower VPA (this curvilinear association may explain why some studies report a facilitative effect of state anxiety on female genital arousal whereas others do not, e.g., [18,19]).

The extant literature suggests a robust overlap between anxiety and FSD-type symptomatology. Therefore, the main aim of this study was explore the biological and nonbiological sources of this covariation. Few studies have attempted to resolve genetic and nongenetic factors in FSD itself. A series of questionnaire studies in twins have established heritability in FSD using quantitative genetic methods [13,20–22]. Univariate and multivariate genetic analyses have shown that individual differences in female sexual desire, arousal, lubrication, orgasm, satisfaction, and pain posses a modest genetic component, with heritability estimates ranging from 19% to 34% (heritability estimates of up to 34%, meaning that more than a third of the total variance of the trait is accounted for by genetic effects) [13,20]. We recently showed [13] that genetic factors also explained approximately 46% of the variance in sexual distress, with no role for shared environmental factors (i.e., those that tend to make siblings the same, such as upbringing). Anxiety sensitivity levels are also modestly heritable with genetic effects explaining 45% of the phenotypic variation [23]. In view of growing evidence indicating a relationship not only between anxiety and sexual problems but also between anxiety and sexual distress, we used multivariate twin modeling techniques to test the relative influence of *specific* and *shared* genetic and nongenetic influences on the phenotypic c-variation between anxiety, sexual distress, and FSD.

Methods and Materials

Study Group

In this exploratory study, we used data from a subsample of 930 (50% response rate) monozygotic (MZ) and dizygotic (DZ) female twin individuals from the UK Adult Twin Registry [24]. The UK Adult Twin Registry is a cohort of unselected volunteer Caucasian twins who have been recruited through successive national media campaigns in the United Kingdom and Ireland and from other twin registers. In extensive clinical and nonclinical investigations, the twins have shown to be comparable with age-matched singletons in terms of disease prevalence, lifestyle characteristics, and sexual behavior and functioning [17,25]. The study was approved by the St. Thomas' Hospital Research Ethics Committee and all twins provided informed consent. The subjects were not selected on the basis of variables being studied (such as the presence or absence of sexual dysfunctions) and were unaware of the specific research aims.

Of the 1,589 responders, nine females who reported never having been sexually active were excluded from further analysis. In order for the sample to be more homogenous, women reporting being homosexual were omitted (N = 19). Females with missing data for more than five of the 19 items in the Female Sexual Function Index (FSFI) and/or more than two items of the FSDS were also excluded (N = 72) [11,20]. To maximize the