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Borderline Personality Disorder In Twins: The Preponderance Of Environmental And Genetic Factors

COMMENTARY

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Abstract

The borderline personality disorder is a psychiatric disorder that despite being widely researched, still has limitations in addressing certain issues involving the degree of genetic and environmental influence. Although studies indicate the existence of such influences, little had research on different prevalences in twins.

Keywords
Borderline, Genetic factors, Personality

Borderline personality disorder (BPD) is one of the most studied personality disorders. [1, 2, 3] However, when compared to research on disorders such as depression or other psychiatric disorders, studies on the genetic factors that influence the development of BPD are surprisingly sparse. [3, 4]

BPD, one of the cluster B Axis II personality disorders in DSM-IV [5, 6], is a complex syndrome characterized by pervasive patterns of instability in emotion regulation, interpersonal relationships, self-image and self-control. [6, 7]

It is associated with a number of negative outcomes, including suicidal behavior, frequent emergency room admissions, substance abuse, impaired occupational functioning, and poor quality of interpersonal relationships. [8] Its etiology is not well understood. [9]

Individuals with BPD are well-represented in treatment settings, accounting for 10% of all outpatients and 15-20% of all inpatients [7, 8]. BPD is equally prevalent among men and women and more likely to be diagnosed in early adulthood. [8, 10]

The nine DSM-IV BPD diagnostic criteria specify the core cognitive, behavioral and interpersonal features that identify and differentiate

BPD from other personality and psychiatric disorders. [6] Given that only five of these criteria must be present, there are more than 250 ways to fulfill a BPD diagnosis. It is therefore not surprising that exploratory factor analyses give mixed results, often with three [11, 12, 13] or four [13, 14, 15] highly correlated factors. However, confirmatory factor analyses have generally supported a unidimensional structure. [6, 13, 16, 17, 18]

Initially, research in behavioural and psychiatric genetics focused on disentangling the genetic and environmental influences on traits or disorders. The findings of these studies were highly relevant in showing the influence of genetic factors in the etiology of almost all traits and disorders. Most of these studies assumed that the effects of genes and environment act independently, meaning that the effect of an environmental risk factor does not depend on the genotype. [19]

In twin-family studies, the different degree of genetic relatedness of monozygotic (MZ) and dizygotic (DZ) twin pairs and other first-degree relatives such as siblings is used to identify the relative contribution of genes and environment to the phenotypic variation of a trait. MZ twins share (nearly) all their genes while DZ twins and siblings share on average 50% of their segregating genes. [3, 20]

To date, genetic research on individual differences in BPD has been limited to non-twin family studies and classical twin studies. Family studies have consistently shown increased rates of BPD in family members of BPD patients [8, 21, 22, 23], and twin studies of BPD reported heritability estimates around 40% [8, 24, 25, 26]. Classical twin studies are important to detect whether there are genetic influences on BPD features. [8]

Over a decade ago, ''all human behavioral traits are heritable'' was stated as the first law of behavior genetics. [27, 28] While provocative at the time, evidence since then has accumulated to suggest heritability estimates of 30% or higher on assessments of cognitive ability, a variety of psychiatric disorders, and even for most classic personality traits. [28, 29, 30, 31]

Whereas the heritability of common personality traits has been firmly established, the results of the few published studies on personality disorders (PDs) are highly divergent, with some studies finding high heredity and others very low. [32]

Some studies showed that the prevalence of personality disorders varies across studies. [9] In contrast to symptom disorders, few epidemiological studies of personality disorders have been conducted to establish their prevalence. [33]

A problem with assessing personality disorders by means of interview is errors connected with interviewer bias. One reason may be measurement variance related to differences between interviewers. Different interviewers differ in their ability to establish rapport with the twins; they stress different aspects of behavior etc. On the other hand, using the same rater to interview both twins may make the twins too similar of the same reason. A way to overcome the problem is to use self-report questionnaires in addition to interviews. [32]

The currently ascendant theories of BPD etiology are diathesis–stress theories that posit interaction between a child's genetic vulnerability and harsh treatment in the family environment. [34, 35, 36, 37, 38, 39, 40]

Several studies demonstrated that traumatic life events such as sexual and physical abuse, parental divorce or illness or parental psychopathology are important risk factors for the development of BPD. [8, 41, 42, 43, 44] The interaction, however, between the influences of genes and environment on the development of BPD has not been studied. [9]

Recently, several twin and twin family studies provided evidence that genetic factors explain familial clustering of BPD, with heritability estimates ranging from 35% to 45%. [19, 24, 25, 26] However, although many researchers and psychiatrists acknowledge the importance of both traumatic life

events and biological vulnerabilities [8, 19, 45], the joint influence of life events and genetic vulnerability on the development of BPD has not yet been investigated. [19]

There is evidence that inherited and environmental factors each influence BPD etiology. Studies of psychiatric patients show BPD is familial [40, 46] and studies of twin samples show it is heritable. [24, 25, 26, 40, 47]

Some analyses assume that twin resemblance arises from two latent factors: i) additive genes, contributing twice as much to the MZ as to the DZ twin correlation and ii) shared or common environment, which contributes equally to the correlation in MZ and DZ twins. In addition to this common environment, the model also contains individual-specific environment that reflects measurement error and those environmental experiences that make members of a twin pair different. [13]

Only two twin studies so far have provided data on BPD diagnoses and features. Torgersen (1984) [48, 49] reported a monozygotic (MZ) concordance rate of 0.0% and a dizygotic (DZ) concordance rate of 11.1% for BPD, suggesting that shared environmental factors influence the variance in BPD. However, methodological problems of that study limit any conclusions. More recently, Torgersen et al. (2000) [44, 45] reported on the largest twin study to date (n=221 twin pairs) that examined BPD. Results suggested a genetic liability for BPD of 69%, though this heritability estimate must be considered approximate due to the small number of twins, the ascertainment method (sampling those who were treated for mental disorder) and the fact that the zygosity and diagnostic status of co-twins was not hidden from the interviewers. [44]

Has been studied a cohort of twins and our results may not generalize to singletons. However, there is no evidence to suggest that twins and singleton differ in their family psychiatric history, experiences of harsh treatment, or in behavior problems. [40, 46]

There are inherent limitations in all twin studies using phenotypic symptom-based data. The methods used can offer only a coarse approximation of the likely true underlying genetic and environmental structure. For example, the method assumes no gene × environment interaction or assortative mating, and it is also dealing with latent liability factors rather than measurable factors. [9]

There are discrepancies in studies of borderline personality disorder in twins as the prevalence of environmental and genetic factors. Future research should focus on possible sources of unique environmental effects and gene environment interaction to develop a comprehensive model of the development of BPD. [3]

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