Borderline Personality Disorder and Childhood Trauma: Evidence for a Causal Relationship

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The debate over whether childhood trauma is a causative factor in the development of borderline personality disorder continues in the literature despite decades of research. This review examines this body of literature published from 1995 through 2007 to assess the strength of evidence for such a causal relationship. A unique conceptual approach was used, as we considered the literature in the context of Hill's classic criteria for demonstrating causation. Results of this review suggest that evidence supports the causal relationship, particularly if the relationship is considered as part of a multifactorial etiologic model. Directions for future research and clinical implications are discussed.

Introduction

The etiology of borderline personality disorder (BPD) remains hotly debated despite several decades of research. Establishing the etiologic factors leading to the development of BPD is quite significant given its prevalence, morbidity, and mortality; BPD is a disabling condition affecting approximately 2% of the general population, 10% of psychiatric outpatients, and 20% of psychiatric inpatients [1]. Individuals with BPD are significant users of health services [2], and their lifetime risk of suicide ranges from 3% to 10% [3].

Childhood trauma has long been considered an etiologic factor for BPD, and this continues to influence clinical practice. However, this assumption of causality persists despite a lack of conclusive evidence. For example, Paris [4] argued that research has failed to establish a causal link between the two variables. Moreover, a meta-analysis by Fossati et al. [5] of papers published between 1985 and 1995 demonstrated only a weak association between childhood sexual abuse and BPD.

Nevertheless, childhood trauma remains at the forefront of etiologic research and debate and therefore is still worth examining. Even if it is not alone as an etiologic factor for BPD development, it may play a significant role. This paper addresses the strength of evidence for childhood trauma as an etiologic factor in the development of BPD by examining the literature published since the reviews by Paris [4] and Fossati et al. [5]. This includes peer-reviewed literature published between 1995 and 2007 that directly addressed the relationship between childhood trauma and BPD and could fit within the conceptual framework of this paper. Due to space limitations, not all articles on this topic are discussed here. However, an effort was made to include all original research published between the specified dates that could be applied to the conceptual framework of Hill's criteria. One particular exception was research on biologic aspects of BPD, because reviewing this burgeoning body of literature was beyond this paper's scope. Instead, biologic research as it relates to this paper is addressed in the context of reviews of that particular body of literature. Other excluded publications include several previously published reviews of the topic, articles that only address a specific aspect of BPD (eg, suicidality or dissociation), some articles related to specificity of the relationship, and others that focused on multifactorial models of BPD etiology. A more comprehensive reference list is available from the authors.

Contrary to other reviews on this topic, this review adopted a unique approach by evaluating the literature in terms of Hill's classic criteria for demonstrating causal relationships [6]. This involved examining how evidence for a causal relationship fared when it was evaluated in terms of the relationship's strength, temporality, dose–response, specificity, consistency, epidemiologic/biologic plausibility, and analogous views. To our knowledge, no one has published a paper comprehensively applying this method in examining the relationship between childhood trauma and BPD. Therefore, this paper provides a more specific and conceptual test of the strength of the causal hypothesis.

Hill's Criteria

Strength of association

Hill's first criterion for establishing a causal relationship is demonstrating a strong relationship between the two variables in question. Laporte and Guttman [7] reviewed 751 psychiatric records of discharged female psychiatric patients diagnosed with personality disorders and found that 93% with BPD had experienced at least one type of loss or abuse in childhood, compared with 74% of patients with other personality disorders. In a pilot study, Oldham et al. [8] found an even larger discrepancy between inpatients with BPD (75% of whom showed a history of childhood abuse) and inpatients with other personality disorders (only 33% of whom reported a history of childhood abuse).

Trull [9] screened a large, nonclinical sample for childhood abuse and BPD pathology and found that "… although not the strongest correlate of borderline features, childhood abuse does account for unique variance in borderline features that cannot be accounted for by parental psychopathology and by personality traits." He contended that the results were "consistent with the viewpoint that although childhood abuse is not the primary etiological factor in BPD, it remains an important factor to include in models of BPD."

Golier et al. [10] examined psychiatric outpatients with BPD and observed that they had significantly higher rates of childhood physical abuse (53%) than those with other personality disorders (34%). However, they found that sexual abuse did not significantly differ between groups (29% vs 19%).

Helgeland and Torgersen [11] compared BPD patients with patients with other psychiatric diagnoses (including Axis I and Axis II disorders). Five variables distinguished significantly between borderline and nonborderline patients: abuse, neglect, environmental instability, paternal psychopathology, and having few protective factors.

More recently, Bandelow et al. [12] compared 66 BPD patients with 109 healthy controls. Reports of various types of traumatic childhood experiences, such as sexual abuse, violence, separation from parents, and serious childhood illnesses, were significantly higher in the patient group.

Bradley et al. [13] had psychologists and psychiatrists describe 534 patients and found childhood sexual abuse to be a significant predictor of BPD over and above family environment, although the latter was also a significant predictor.

Lobbestael et al. [14] examined schema modes and childhood abuse in patients with complex personality disorders and found a strong association between childhood abuse and BPD when compared with controls. Although a strong relationship was also found between abuse and antisocial personality disorder, the BPD group experienced more episodes of sexual abuse compared with the antisocial personality disorder group.

Bornovalova et al. [15] conducted a cross-sectional study of an inner-city primarily African American population with BPD in an inpatient drug and alcohol abuse treatment center. A correlational analysis found that a BPD diagnosis was significantly associated with higher impulsivity, emotional instability/vulnerability, lower well-being, many interpersonal factors, and a range of types of childhood maltreatment. When regression analyses were performed, emotional abuse reliably predicted BPD diagnosis better than any other factor.

Thus, the research supports an association between childhood trauma and BPD. Because the purpose of this conceptually based review was not to conduct a meta-analysis, no single statistical quantity was calculated or is reported here. However, considering the individual results of recent studies, the association appears to be significantly associated with BPD even compared with near-neighbor disorders and not as weak as the Fossati et al. [5] review concluded. This seems to be in line with the Zanarini [16] review of the literature that described estimates of childhood sexual abuse in BPD ranging between 40% and 70%, compared with the rate of childhood sexual abuse in other Axis II disorder patients (19% to 26%) and with further research to be addressed subsequently [17,18].

Temporality

Another one of Hill's criteria is to demonstrate that the proposed causal variable occurs earlier in time than the outcome variable. Most studies of childhood trauma and BPD use retrospective methods that confound demonstrating temporal relationships due to the inherent limitations of retrospective study design [4]. Prospective longitudinal studies—which eliminate recall bias—that examine personality disorders are rare. However, a few have been published [17–20].

Johnson et al. [20] found that individuals with documented histories of childhood sexual abuse, physical abuse, or neglect were more than four times more likely to be diagnosed with a personality disorder than those who were not abused or neglected. This association held even after statistically controlling for factors such as age, parental education, parental psychiatric disorders, and other types of childhood maltreatment. Some of the child maltreatment data for this study came from records of the New York State Central Registry for Child Abuse and Neglect, which maintains reports of cases of abuse and neglect referred to childhood protective services that are confirmed by authorities. However, maltreatment was also assessed through participant retrospective reports, and this was not presented separately from the New York State Central Registry for Child Abuse and Neglect–confirmed reports. Thus, these results cannot be considered free of recall bias because of the retrospective elements in the design.

Yen et al. [18], as part of the Collaborative Longitudinal Personality Disorders Study, reported that BPD participants showed the highest rate of childhood trauma (especially sexual trauma). Also as part of the Collaborative Longitudinal Personality Disorders Study, Battle et al. [17] reported that rates of childhood abuse and neglect were much higher in BPD patients than in patients with other personality disorders. However, similar to Johnson et al. [20], the components of these studies that assessed childhood trauma were not free of recall bias because they used participants' retrospective reports of childhood trauma.

Rogosch and Cicchetti [21] compared 185 maltreated children with 175 nonmaltreated children who attended a week-long day camp research program. Childhood maltreatment was confirmed using data from the Department of Human Services. Using several measures, they determined that childhood maltreatment and abnormal attention independently predicted the presence of what they identified as BPD precursors. These precursors included developmental processes surrounding personality features, interpersonal relationships, self-representation and representations of others, and indication of self-harm and/or suicide ideation. According to the authors, "children displaying extremes of functioning across these areas could be regarded as evincing a pattern of significant vulnerability to subsequently emerging BPD." Unfortunately, using potential precursors of BPD in children as the outcome variable does not necessarily show that the trauma leads to an actual BPD diagnosis. Thus, although what we have learned is promising, furthering this research is necessary to establish the link. If this study continues to observe these children to see if they develop BPD, it may constitute the sole true longitudinal study in this area.

Dose–response

The next of Hill's criteria is to demonstrate that as the independent variable increases in severity (in duration or degree), the outcome variable also increases in severity (dose–response).

Silk et al. [22] showed that severity of sexual abuse, particularly in its ongoing nature, was predictive of certain aspects of BPD, including parasuicidal behavior, regression in therapy, and total score on the Diagnostic Interview for Borderline Patients. The more severe the abuse, the higher the Diagnostic Interview for Borderline Patients score, indicating the potential for more severe BPD.

Likewise, Zanarini et al. [23] showed that "severity of reported childhood sexual abuse was significantly related to the severity of symptoms in all four core sectors of borderline psychopathology (affect, cognition, impulsivity, and disturbed interpersonal relationships), the overall severity of BPD, and the overall severity of psychosocial impairment."

Finally, when comparing psychiatric inpatients with BPD with those without BPD, Sansone et al. [24] found that those with BPD reported significantly more types of childhood trauma, greater use of particular health care services, and a higher number of self-harm behaviors.

These results suggest that as childhood trauma worsens, the outcome BPD is potentially more severe, and thus, they provide strong evidence satisfying Hill's criteria for dose–response in a causal relationship.

Specificity

Hill's criteria also indicate the necessity of demonstrating specificity between the etiologic and outcome variables. One of the main arguments against adopting childhood trauma as an etiologic factor for BPD involves the issue that childhood trauma is not necessarily specific to BPD. Not all individuals with BPD have experienced childhood trauma, and not all individuals who have experienced childhood trauma are diagnosed with BPD [16,25,26]. For example, Sabo [27] reports that approximately 20% to 45% of individuals diagnosed with BPD do not report a history of childhood sexual abuse. Furthermore, it also has been reported that traumatized children may later present with other psychiatric illnesses [17,28]. One way of interpreting these findings is to argue that the lack of specificity points to the multifactorial nature of an etiologic pathway to BPD, which is further addressed subsequently.

Consistency

Hill's criteria also include the condition that the relationship between variables must be consistent across studies. Studies have consistently found an association between some form of childhood trauma and BPD [23]. The studies described previously under the heading of "Strength of association" and "Temporality" are excellent examples of this and also could be included under this criterion. The study by Bornovalova et al. [15] is a particularly good example of consistency because, as the authors indicate, the participants were from a unique population of inner-city primarily African American substance users, which differs considerably from the typical white, middle- to upper-class–educated participants commonly used in BPD research, yet they still showed an association between childhood trauma and BPD.

Furthermore, as illustrated previously, the association is consistently found to be higher than in other psychiatric populations. It could be argued that the consistency with which this association is found is one of the stronger arguments for childhood trauma as an etiologic factor in BPD development.

Epidemiologic and biologic plausibility

Hill's criteria for demonstrating causation also note that the causal relationship must make epidemiologic and biologic sense. Epidemiologically speaking, although there are far more incidents of childhood trauma in the general population than there are BPD diagnoses, no studies found in this review address the epidemiologic

distribution of childhood trauma as it relates to BPD. Despite this, one could argue that the higher prevalence of BPD found in females compared with males provides epidemiologic plausibility for childhood trauma as an etiologic factor in BPD given the higher prevalence of childhood sexual abuse in females versus males [29].

Biologic research in BPD is a new and quickly expanding field, and, as noted previously, it is beyond the scope of this review to evaluate all the available research in this area. Instead, we consider it in the context of recent reviews in the area [30,31,32•]. These reviews indicate that recent research suggests a biologic link between brain changes in individuals having suffered childhood trauma and similar changes in those with BPD. For example, neuroimaging studies have found reduced volumes of the hippocampus, amygdala, and prefrontal cortex in individuals with BPD, although evidence for reduced prefrontal cortex is mixed [30,31,32•]. Some researchers have hypothesized that structural differences could be due to prolonged childhood trauma [30,31,32•]. Among their functions, the prefrontal cortex helps to regulate mood and elicit responses, the amygdala interprets emotions and is involved in fear response, and the hippocampus maintains memories, especially of emotions. Given that emotional lability often characterizes BPD, it would make sense that the noted areas of dysfunction also could be related to BPD. The brain's central serotonergic system, which controls affect, impulse regulation, and autoaggressive behavior, also has been shown to be affected by prolonged trauma in childhood [31]. Thus, there is some overall evidence for the biologic plausibility in the relationship between childhood trauma and BPD.

Analogy

The last of Hill's criteria in demonstrating causality between two variables is determining whether the proposed causal relationship is analogous to any other causal relationships. We would argue that this is the case for childhood trauma and BPD, particularly if one considers childhood trauma in the context of a larger, multifactorial etiologic model. Many researchers have proposed multifactorial models for the etiology of BPD [4,9,11–13,15,16,33–38].

Other researchers have proposed specific mediators as part of the relationship. For example, Thatcher et al. [39] illustrated that adolescent alcohol disorders may act as a mediator between childhood abuse and developing adult BPD. Their analyses also suggest that further mediators may exist, including childhood diagnoses of depression, conduct disorder, and attention-deficit/hyperactivity disorder. Minzenberg et al. [40] proposed that childhood maltreatment leads to adult social attachment difficulties, including anxious and avoidant attachment styles, which in turn mediate the symptoms of BPD. Finally, Fruzzetti et al. [41] suggested that childhood sexual abuse or other early traumas could be best understood as more distal risk factors in the development of BPD that have etiologic significance by acting through mediating variables.

By analogy, many multifactorial models have been described for the development of other psychiatric disorders. Rutter et al. [42••] recently reviewed research suggesting that interactions between child maltreatment and genetics may contribute to the development of antisocial disorders. Multifactorial etiologic models that integrate childhood trauma into the model also have been proposed for other psychiatric illnesses, such as panic disorder and eating disorders [43–45]. Thus, although to our knowledge no one has yet constructed an empirically proven multifactorial model with which to compare a proposed multifactorial model of BPD etiology, a multifactorial model that incorporates childhood trauma as a factor leading to BPD development by analogy fits with proposed models of causality for other psychiatric disorders.

Conclusions

This paper intended to examine childhood trauma and the strength of evidence for its placement as an etiologic factor for BPD development. Hill's classic criteria for establishing causality were used to assess the evidence published since 1995. These criteria include strength of association, temporality, dose–response, specificity, consistency, epidemiologic and biologic plausibility, and analogy.

As noted previously, an association is consistently shown across studies. Although an individual value of the strength of association was not determined through a meta-analysis in this review, individual studies indicate that the association is stronger than previously suggested. However, the evidence for specificity is weak, but it would appear that if one were to consider childhood trauma as a variable in a multifactorial etiologic pathway, by analogy, the argument for it as an etiologic factor strengthens.

In fact, if a multifactorial model is adopted, it may explain the variance in strength of association and lack of specificity found in the relationship. Moffitt et al. [46] described this in their discussion of gene–environment interactions in the etiology of psychiatric disorder: "One feature of a good candidate environmental risk factor is obvious but bears noting: it should not perfectly predict the disorder outcome." In other words, variance in the strength of association between the etiologic factor and outcome is expected in gene–environment interaction. Paris [4] also noted the importance in gene–environment interactions. Using this and some of the other criteria for identifying environmental pathogens (eg, biologic plausibility), childhood trauma is well placed as a potential environmental factor to be investigated in the context of gene–environment interaction leading to BPD.

Thus, if a multifactorial model is ultimately delineated for BPD and involves several mediating variables that may include gene–environment interactions, heterogeneous strength of association and lack of specificity between childhood trauma and BPD would be expected. In fact, such heterogeneity would not only be consistent with the evidence but would strengthen the argument for childhood trauma as an etiologic factor in BPD.

Returning to Hill's other criteria that were used to evaluate the literature, the data involving dose–response are supportive of childhood trauma as an etiologic factor in BPD. The research addressing the temporal relationship, although promising, still requires more true longitudinal study designs. No papers included in the review of recent literature addressed the epidemiologic plausibility for the causal relationship, although in the absence of such literature, one could argue that the relationship makes epidemiologic sense if one considers the similar sex distribution of BPD and childhood sexual abuse. On the other hand, the biologic research, although in its infancy, is supportive of the idea that it makes biologic sense for a causal relationship to exist between childhood trauma and BPD.

Although applying Hill's criteria is helpful in demonstrating causal relationships, it is not without limitations. For example, regarding Hill's first criterion of relationship strength, as discussed previously, one could argue that just because a relationship is not strong does not mean there is no causal pathway between two variables. Another example is specificity: not all causal relationships are specific between two variables. An overall criticism of applying the criteria is that it is a theoretical framework that does not lead to quantification of the relationship. Nevertheless, the criteria remain useful in providing a framework for evaluating the evidence regarding a relationship.

In summary, there is no question that despite decades of research and all the literature addressing the topic, debate continues as to the etiologic factors behind BPD. It seems likely that as we pursue the study of BPD etiology, an integrated, multifactorial model ultimately will be adopted. We do not dispute that the etiology of BPD is likely multifactorial. However, we would argue that based on the evidence, particularly Hill's criteria of strength of association, dose–response, consistency, biologic plausibility, and analogy of the relationship, childhood trauma plays a role in this model as an etiologic factor.

One of the obvious implications of this paper is that research needs to evaluate complex models for the etiology of BPD. One avenue for this research could be to investigate childhood trauma in terms of gene–environment interactions as described by Moffitt et al. [46]. No matter which research direction is pursued, the evidence suggests that childhood trauma should be included in a multifactorial etiologic model of BPD.

Having concluded that childhood trauma is an important etiologic factor in BPD development, certain cautions for clinical practice regarding this inference must be remembered. First, ample evidence indicates that psychotherapy for patients with BPD need not focus on childhood abuse to be successful. For example, the efficacy for dialectical behavior therapy (DBT) for suicidal BPD patients has been established, but DBT does not address problems related to childhood abuse [47]. These issues are left for the second stage of therapy, and the efficacy of this stage of DBT has not been formally studied. Second, focusing therapy on childhood trauma is not without risks. For example, recovered memory treatment can lead to a worsening of suicidality [48], or trauma-focused therapy with BPD patients can lead to their early termination of therapy [49]. Plainly, further research on trauma-related therapies for patients with BPD and a history of childhood trauma is required.

Disclosures

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