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# If It Looks Like a Duck and Quacks Like a Duck: Evaluating the Validity of Borderline Personality Disorder in Adolescents

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**Abstract**

**Background:** The validity of borderline personality disorder (BPD) has been a topic of much controversy in psychiatry. Over the last two decades, a wealth of empirical work has challenged long-held concerns regarding the validity of adolescent BPD. However, this research has been conducted within a traditional approach to psychiatric nosology.
**Objective:** In this article, we aim to evaluate the validity of adolescent BPD as guided by both the Robins and Guze criteria for the validity of psychiatric constructs and the new National Institute of Mental Health Research Domain Criteria (NIMH RDoC).
**Method:** We used the five principles of the Robins and Guze approach to evaluate selected research from our own and other groups regarding the validity of adolescent BPD. These principles include clinical description studies, laboratory studies, studies that delimitate the disorder from other related syndromes, follow-up studies, and family studies.
**Results:** Within the Robins and Guze criteria framework, evidence to date supports the validity of adolescent BPD to some extent. However, limitations of the research about the construct validity of adolescent BPD have also been identified, most notably regarding the delimitation of adolescent BPD from other disorders as well as a lack of longitudinal and family studies.
**Conclusions:** Given these limitations and the limitations of the Robins and Guze approach to psychiatric nosology, we recommend exploring the potential of the National Institute of Mental Health Research Domain Criteria as a complement to previous work.

**Keywords:** borderline personality disorder; adolescents; validity; developmental psychopathology

## Determining the Validity of a Psychiatric Disorder

In 2008, Chanen and McCutcheon published a paper entitled, "Personality disorder in adolescence: the diagnosis that dare not speak its name" (1). This title speaks to the concerns that were present as recently as six years ago regarding the validity of personality disorder (PD) in adolescents, specifically borderline personality disorder (BPD). Indeed, at that time, the majority of British psychiatrists (63%) considered the diagnosis of adolescent PD to be invalid (2), and many clinicians still appear to be uncomfortable diagnosing PD in adolescents (3).

Hesitations regarding diagnosing this disorder in youth are based on sound concerns. Many believe that a BPD diagnosis during adolescence engenders stigma (4); that personality is inherently unstable during adolescence (5,6); and that the clear demarcation of pathological borderline traits from typical youth behavior (e.g., impulsivity, moodiness) is elusive (7). Specifically, adolescents undergo dramatic bodily changes, significant increases in the intensity of affective and emotional drives, and deep reorganization of the self in the context of peer-directed norms and interactions in addition to pressures toward autonomy and the assumption of adult roles (8,9). Further complicating the demarcation of BPD from normal adolescence is the fact that some of the behaviors that characterize adolescent BPD (e.g., self-harm, substance abuse,

sexual risk-taking behavior) commonly occur during adolescence (10-12). These normative developmental changes resemble BPD to some extent and add to concerns regarding the validity of a BPD diagnosis in this age group.

But how do we determine the validity of a psychiatric disorder? In reaction to a lack of clarity surrounding scientific procedures to address this question, in 1970, Robins and Guze (13) proposed a set of five principles that have since guided research regarding the nosology of psychiatric disorder. In contrast with heavy reliance on a priori hypothesized diagnostic criteria, which was a common part of early psychiatric disorder classification, the Robins and Guze method ensured that only through a series of systematically conducted and empirically sound studies can the suggestion that a particular diagnostic entity exists be substantiated. It is through this framework that much of the work over the past few decades examining BPD in both adults and adolescents has been concentrated. Although they were published more than 40 years ago, the Robins and Guze criteria are still considered the gold standard approach for establishing the diagnostic validity of psychiatric disorders.

The Robins and Guze principles reflect the inclusion of five types of research studies, each of which is aimed at capturing a specific and separate component of a disorder's nosology:

1) *Clinical description studies, which aim to establish a coherent clinical picture of the disorder.* Studies in this category must demonstrate that the disorder has a particular and consistent pattern of symptoms and that these symptoms co-occur. Moreover, important non-psychopathological features that are common or prototypical of clinical presentations of the disorder must also be identified. These may include factors such as age, sex, and age of onset, among others.

2) *Laboratory studies, which focus on identifying neurobiological and physiological substrates of the disorder.* Laboratory tests provide an important complement to clinical descriptive studies, especially when they confirm clinical observations.

3) *Studies that delimitate the disorder from other related syndromes.* These studies pinpoint a disorder's uniqueness relative to other psychiatric disorders with similar phenotypic presentations by establishing rates of co-occurrence with related disorders and supporting the discriminant validity of the construct.

4) *Follow-up studies that identify a prototypical course and outcome of the symptoms.* For example, demonstrating that individuals who were first identified with the disorder in baseline assessments present with the same disorder (as opposed to a different psychiatric disorder) as identified by later assessments provides evidence for the original diagnostic criteria used at baseline.

5) *Family studies that identify a genetic basis of the biological phenomena associated with the disorder.* Demonstrating that the disorder displays heritability provides evidence for distinct psychopathological processes related to its phenomenology.

Over the past two decades, much of the work examining the validity of BPD in adolescents, including our own work, has been guided by these principles. Although these guidelines have been crucial for advancing psychiatric research in general, the system is limited in its focus on the behavioral phenotype. In other words, it relies solely on observable behavior when describing psychiatric disorder rather than understanding the disorder in terms of its underlying processes and mechanisms. Analogizing from the animal world, after observing the physical features of a hippopotamus, most would agree that the animal resembles the pig family and thus believe that the pig is the closest living relative of the hippopotamus. However, zoologists tell us that the closest living relative of the hippopotamus is the whale. Therefore, relying solely on the physical features of a phenomenon when evaluating its validity can cause obvious problems.

In the case of psychiatric diagnosis, we deal with two specific problems that are characteristic of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM)-based categorical and polythetic behavioral phenotype. First, psychiatric diagnosis is based on clinical observation and patients' phenomenological symptom self-reports. Unlike other medical disorders, for which objective diagnostic tests are used, we depend on patients' perceptions of their problems and our own (sometimes biased) clinical observations. This is particularly problematic in the case of PD, because self-reported attributes cannot capture the dynamic processes that give rise to the discrepancies between self-identified traits and the behavioral manifestations that are typically characteristic of PDs (14). Second, this implies that psychiatric conditions are described in terms of polythetic and dichotomous (categorical) diagnoses. This means that patients can present with only a portion of the criteria that define a disorder and will receive a certain diagnosis as long as they have met the predetermined symptom threshold (15).

The reliance on polythetic and dichotomous (categorical) diagnoses at the level of the behavioral phenotype alone has led to concerns about the validity of the nosological system – most notably the problem that the use of the system results in high rates of comorbidity among disorders, over-

reliance on disorder "not-otherwise-specified" or deferred diagnosis, and lack of diagnostic stability (15). Although there is much evidence in support of the DSM-based diagnostic system, some have expressed concerns that this system does not "carve nature closely at its joints" (16) and thus may have impeded the use of advances in genomics, pathophysiology, and behavioral science to aid in the diagnosis and treatment of psychiatric disorders (17). To address these limitations, the National Institute of Mental Health (NIMH) proposed the Research Domain Criteria (RDoC;18) in 2011 to implement Strategy 1.4 of the National Institute of Mental Health Strategic Plan (19). By following this initiative, psychiatry would depart from its categorical classification system of mental disorders to "develop new ways of classifying mental disorders based on *dimensions* of observable and neurobiological measures" (18). In this article, we evaluate the validity of BPD in adolescents according to both the traditional Robins and Guze criteria and the newly formulated RDoC. We will focus on work emanating from our own laboratory as well as from other laboratories. We begin by discussing research organized within the framework of the five Robins and Guze criteria, and we then evaluate the state of the science involving adolescent BPD against these criteria. We then discuss the RDoC principles and map directions for future research.

## Clinical Description

*Categorical Diagnosis*

Robins and Guze's first principle states that, for a disorder to be valid, its criterion set must constitute a coherent combination of traits and symptoms that "hang together" (13). Statistically operationalized, this principle requires that a single common factor adequately accounts for covariation among the criteria. Studies of adults (20-24) and adolescents (25) have factor analyzed the nine DSM borderline personality criteria and found evidence for a unidimensional factor structure of BPD. With this in mind, we used an item response theory (IRT) approach to examine DSM-IV BPD criteria in a large community sample of young adolescents from the United Kingdom (N = 6339; $M_{age}$ = 11.75 years; 51.55% girls; 26). The use of IRT in this regard had several important advantages. First, because IRT is a latent trait approach, it allows for the examination of the underlying factor structure of the BPD criteria. Second, it addresses important questions surrounding DSM-IV BPD criterion functioning. This process included examining the extent to which a particular BPD criterion discriminated among adolescents with regard to their standing along the continuum of BPD liability and whether certain criteria discriminate better than others. Third, it specifies the threshold of endorsement of a criterion along the latent continuum of BPD liability to determine whether certain criteria are more difficult to endorse than others. Lastly, IRT allows for the examination of differences in the performance of BPD criteria across gender to determine whether the BPD criteria are useful for both boys and girls.

In our study, BPD was assessed via the Childhood Interview for DSM-IV Borderline Personality Disorder (CI-BPD; 27). Results demonstrated that a single underlying dimension adequately accounted for covariation among the BPD criteria, which lends support to the unidimensionality of the BPD construct. These findings were consistent with studies from the adult literature (20-24) and with those of one study that used an adolescent sample (25). In addition, items demonstrated good discrimination and difficulty parameters. The most difficult items included abandonment fears and suicidal behavior; this finding converges with research in adults that has demonstrated that abandonment fears are the least commonly exhibited BPD symptom (22,28) and that suicidal behaviors are uncommon among young children (29). With regard to measurement equivalence across gender, although five criteria demonstrated differential item functioning, this balanced out at the level of the total set of the nine DSM-IV BPD criteria, which suggests that the use of the full nine criteria is psychometrically valid and advisable. Taken together, findings from this study provide evidence that the DSM criteria constitute a coherent combination of traits and symptoms, even in preadolescent and young adolescent youth, for whom the empirical base is less developed.

It is important to establish that a particular construct is valid across multiple samples so that it can be generalized to the population at large. The aforementioned study (26) demonstrated that the CI-BPD criteria were most discriminating at the highest level of underlying borderline personality pathology liability; this finding was not surprising given the community-based sample used. It cannot be assumed, however, that these criteria function similarly in a clinical setting and represent a valid approximation of the borderline personality pathology construct in clinical populations. Rather, there is a possibility that the aforementioned community sample findings capture psycho-pathology or distress in general rather than BPD psychopathology in particular. Thus, we revisited this question again with a sample of 190 inpatients ($M_{age}$= 15.39; 9% female; 33% meeting criteria for BPD; 30). We used a confirmatory factor analytic approach to examine the internal factor structure of

the nine CI-BPD items. The internal consistency (Cronbach's alpha = .80), the inter-rater reliability (kappa = .89), the convergent validity (with a clinician diagnosis kappa of .34 and a *p*-value of <.001, which indicates "fair agreement"), and the concurrent validity were examined and empirically supported as indicated by associations in the expected directions with self-report measures of BPD traits, internalizing and externalizing pathology, and rates of self-harm and emotional dysregulation. Although we did not directly compare different models of underlying factor structures with one another, confirmatory factor analytic results supported a unidimensional factor structure for CI-BPD, which indicated that the DSM-IV criteria on which the CI-BPD is based constitute a coherent combination of traits and symptoms, even among adolescents. This study was the largest inpatient study to examine the psychometric properties of this measure among inpatient adolescents.

Factor analysis of the nine criteria in the absence of other PD criteria only narrowly establishes the unidimensionality of the construct, which highlights an important limitation of the previously mentioned research. A more stringent test of unidimensionality would involve the factor analysis of the BPD criteria alongside Axis I or PD criteria. However, no work of this nature has been conducted with adolescents, and the results of adult studies are mixed. In a minority of studies, the DSM structure of PD (ten discrete disorders, including BPD) has been supported (31-34). Most studies have failed to support the DSM's putative structure for PD (35-38), which suggests that the unidimensional factor structure of BPD may disintegrate after factor analysis involving other PDs. Recently there has been a growing interest in considering models that evaluate general factors that account for common variance among PDs as well as unique sources of variance that may represent more specific forms of personality pathology. From a factor-analytic perspective, this hypothesis can be tested with what is termed a *bifactor model.* This type of model allows PD criteria to load on a large general factor that encapsulates the dysfunction shared across PDs, with additional circumscribed factors that capture the unique domains of impairment (39,40). To truly clarify whether DSM criteria hang together, factor analysis studies of this kind must be undertaken in adult and adolescent samples.

*Dimensional Self-Report Ratings*

Although support for BPD as measured by clinical interview (e.g., with the CI-BPD) is important, examining dimensional BPD traits is equally important, especially in the context of DSM-5 Section III. Taking a dimensional approach to personality pathology assumes an individual difference perspective in which PD pathology is evaluated through quantitative models developed from observable traits and symptoms. This allows for a data-driven approach that involves the data delineating the constructs. It also allows for a fine-grained description of personality pathology rather than lumping symptoms in predetermined categorical diagnoses. Finally, as demonstrated in Section III of the DSM-5, it allows for a hybrid model of personality pathology whereby general personality pathology is captured by Criterion A (dysfunction in self- and other-relatedness) and in which specific disorders and traits are captured by Criterion B. These advantages are amplified in the context of BPD in adolescents. Specifically, dimensional measures are crucial for assessing BPD in youth precisely because of the concerns expressed earlier about categorical diagnosis during youth. As compared with categorical approaches, dimensional models may better account for the developmental variability and heterogeneity common among adolescents and identify subclinical levels of BPD for early intervention (41). These models also allow for the elucidation of heterotypic or homotypic continuity of the disorder (i.e., the relationship between BPD at one point in time with continued dysfunction at a later point), either through BPD pathology or a different form of pathology. Finally, in contrast with semi-structured interviews, which often are expensive and time-consuming to administer, self-report measures are relatively quick and inexpensive to administer, and they allow the evaluator to assess a wide range of behaviors and symptomatology in a short amount of time.

With the use of the Borderline Personality Features Scale for Children (BPFSC; 6) and an IRT approach, we examined the factor structure of trait-based BPD in a community sample of 881 adolescents between the ages of 13 and 17 years (55.9% female; 42). Given the putative factor structure of the BPFSC (6), we evaluated a bifactor model in which all items were allowed to load onto one general factor and in which respective items were allowed to load onto four specific factors: negative relationships, affective instability, self-harm, and identity problems. Results did not support a bifactor model; the 24 BPFSC items did not load onto a general borderline factor or onto predicted subfactors. Importantly, strong evidence for local dependence between items (the fact that two or more items are more highly correlated with one another than with the underlying latent trait) was exhibited, which suggests item redundancy. We therefore removed 13 items to create a shortened

version of the BPFSC (BPFSC-11) and tested the factor structure of this truncated measure. With poorly functioning items removed, a unidimensional factor structure emerged. Moreover, when we tested the construct validity of the BPFSC-11 in a sample of 371 inpatient adolescents, it showed equivalence when compared with the longer version of the BPFSC, including criterion validity against the interview-based assessment of BPD (i.e., the CI-BPD).

Further support for the construct validity of trait-based conceptualizations of BPD symptomatology during adolescence was provided in a study of 171 boys between the ages of eight and 18 years who were recruited from the community (43). In this study, the cross-informant concordance between parent- and self-reported borderline personality features using the BPFSC and a newly developed parent version of the measure (BPFSP) was examined. Results demonstrated modest but significant concordance between parent- and self-report ratings, and this was consistent with findings from other psychiatric constructs in child psychiatry (44). Borderline features were robustly associated with total psychopathology problems as measured by the Youth Self-Report and Child Behavior Checklist (45) as well as by specific DSM-oriented scales. Specifically, boys with high scores on the BPFSC had up to six times the relative risk for overall psychopathology and up to five times greater risk as indicated by relative risk for specific psychopathology, including anxiety disorder (4.99), conduct disorder (4.59), attention-deficit/ hyperactivity disorder (3.88), affective disorder (2.86), somatic disorder (2.65), and oppositional defiant disorder (2.22). Similarly, boys whose parent-reports placed them in a high-BPD trait group had up to nine times greater risk for externalizing disorders: conduct disorder (8.97), oppositional defiant disorder (3.67), and mood dysregulation (affective disorder [2.75]).

In another study involving the BPFSC, we examined the performance of parent- and self-report BPFS tools for detecting the diagnosis of BPD in 51 adolescent inpatients between the ages of 12 and 18 years (46). This study made use of receiver operating characteristic analyses, which provide receiver operating characteristic curves or the plot of the true-positive rate (sensitivity) against the false-positive rate (1-specificity) on a graph. The area under the curve (AUC) provides an indicator of accuracy. Receiver operating characteristic analysis was an advantageous approach in this study, because it allowed for the direct comparison of the relative performances of the BPSFC and BPFSP for predicting a CI-BPD diagnosis. Results demonstrated that the BPFSC had high diagnostic accuracy (AUC = .93) for discriminating adolescents with a CI-BPD diagnosis, and the parent-reported version had moderate accuracy (AUC = .80; sensitivity = .73; specificity = .72). When comparing the measures directly, scores from the BPFSC discriminated significantly better than did scores from the BPFSP. Concordance between parent and child reports was also significant. This study was important in that it provided preliminary support for the relative advantage of self-report over parent-report for the assessment of borderline traits during adolescence. Given that BPD is suggested to be a confluence of internalizing and externalizing psychopathology (47,48) and given that research has shown self-report to be more valid for internalizing disorders during adolescence (whereas parent- or teacher-report may be more useful for externalizing problems [49]), we suggest that the superior performance demonstrated by the self-report BPFSC in our studies may be driven by the internalizing features of BPD.

*Screening Measures*

If BPD assessment is to be routinely included in general adolescent assessment services, valid assessment tools for this purpose must be available. Although the BPFSC-11 would perform well, the McLean Screening Instrument for Borderline Personality Disorder (MSI-BPD; 50), which was originally developed for adults, is another alternative. The MSI-BPD is a 10-item self-report yes/no measure that provides a brief screening assessment of the number of BPD criteria met. It was designed specifically with low-resource settings in mind, where a quick and economical assessment of BPD is necessary for potential inclusion in a research study or for follow-up clinical assessment. We examined the MSI-BPD's ability to accurately identify adolescents with BPD using the CI-BPD and the BPFSC in a sample of 118 inpatients recruited from a facility that serves an indigent population ($M_{age}$ = 14.64; 64.4% female) (51). At the bivariate level, results demonstrated that the MSI-BPD was significantly related to both the CI-BPD and the BPFSC, although the correlations were of moderate magnitude. Similar to results from a study conducted in a child and adolescent sample (52) as well as in a community sample of adult women (53), the MSI-BPD demonstrated moderate diagnostic efficiency when predicting the CI-BPD diagnosis, with sensitivity (Sn) and specificity (Sp) values of .71 and .65, respectively, and diagnostic accuracy of .73. This study also established an MSI-BPD adolescent-specific cutoff score of 5.5, whereas the previously established cutoff score of 7 rendered in the adult sample (53) lowered sensitivity substantially in our sample (Sn = 48 and Sp = .83).

*Summary*

The psychometric work involving at least three measures of BPD discussed here provides some evidence in support of the borderline construct in youth. Collectively, this work suggests that a coherent clinical picture of the disorder *can* be described and that a particular and consistent pattern of symptoms co-occur even when they are assessed using different measures. Moreover, our work and the work of others suggest that important non-psychopathological features (like those captured by prevalence studies) are common or prototypical of the clinical presentations of the disorder in adolescents. For example, the percentage of adolescent inpatients meeting the diagnostic criteria of the CI-BPD in our work mirrors that of other studies in adolescent inpatient settings that report BPD diagnostic prevalence rates of 33% to 53% (54-56).

However, it is also clear that further work is needed regarding the underlying factor structure of PD in adolescents in general and in BPD in particular. There is a critical need for evaluating more complex bifactor models in adolescent PD for both categorical symptom and dimensional trait approaches to establish whether a coherent syndrome is present in adolescents. In other words, to firmly support the unidimensionality of a construct, all psychopathology (including all PDs) must be considered. In the case of adolescent PD, this agenda is complicated by the fact that few DSM-based measures of other PDs have been developed for adolescents. Although the CI-BPD and BPFSC were developed for children and adolescents specifically, there are no developmentally sensitive counterparts available to assess all DSM-II–based PDs. This is necessary to move DSM-based diagnostic work forward.

For trait-based approaches to PD assessment in adolescents, the picture is less bleak. There are several adolescent trait-based instruments, including the Minnesota Multiphasic Personality Inventory – Adolescent version (57), which is a 478-item true/false self-report inventory designed to assess the social, emotional, and behavioral functioning of adolescents between the ages of 14 and 18 years that has been examined in one study of adolescent BPD (58); the Dimensional Personality Symptom Item Pool (59), which is a 172-item measure structured to assess 27 maladaptive personality facets that are hierarchically organized into four broad personality dimensions (disagreeableness, emotional instability, introversion, and compulsivity); and the adolescent version of the Personality Inventory for DSM-5 (60), which has recently been validated for use in adolescents (61) and which includes 220 items that are rated on a four-point Likert scale and structured into 25 empirically derived lower-level trait pathology facets that assess the traits associated with DSM-5 Section III.

**Laboratory Studies**

Robins and Guze's second principle calls for laboratory studies that aim to identify neurobiological and physiological substrates of the disorder. Although most laboratory studies of biological substrates of BPD have been conducted with adults, there has been a steady increase in biologically based studies involving adolescents. For example, laboratory studies of adolescent BPD have examined hypothalamic–pituitary–adrenal axis abnormalities. Kaess and colleagues (62), and Garner and colleagues (63) demonstrated that patients with BPD who were exposed to childhood trauma had smaller pituitaries (–18%) as compared with those with no history of childhood trauma. Consistent with clinical consensus on BPD, these studies suggest that the hypersecretion of cortisol may be associated with maladaptive stress responses in the development of BPD, especially in the presence of trauma history.

Structural functional neuroimaging studies in adolescents have demonstrated volume reduction in the frontolimbic network, including the orbitofrontal cortex (OFC), and the anterior cingulate cortex (ACC). For example, Whittle and colleagues (64) reported decreased left ACC volume in 15 female adolescents with BPD with a wide range of Axis I comorbidities, and Goodman and colleagues (65) found that adolescents with BPD and comorbid major depressive disorder (MDD) had reduced Brodmann Area (BA) 24 gray matter volume as compared with healthy controls, but no differences were found in the size of the prefrontal cortex. In contrast, Brunner and colleagues (66) compared 20 adolescent girls with DSM-IV diagnoses of BPD, 20 patients with DSM-IV defined current psychiatric disorders, and 20 healthy control subjects. The findings suggested that adolescent clinical control subjects displayed significantly decreased gray matter volume in the right dorsolateral prefrontal cortex as compared with healthy control subjects, but no significant gray matter differences were detected between the BPD group and the clinical control group. Similarly, no group differences were found in the limbic system or in any white matter structures. This study suggests that early morphological changes in BPD are located in the PFC but that these changes may not be specific to BPD, because changes were similar to those found in the clinical control group. Finally, Chanen and colleagues (67) compared 20 first-presentation BPD patients and 20 healthy

control participants and found a reversal of the normal (right > left) asymmetry of orbitofrontal gray matter volume in the BPD group, which reflects right-sided orbitofrontal gray matter loss in the BPD group as compared with control participants. No significant differences were found for amygdala or hippocampus volumes. The authors suggested that hippocampus and amygdala volume reductions observed in adult BPD samples may develop during the course of the disorder through early adulthood, but they acknowledged that longitudinal studies are needed to evaluate this further.

Although studies of the biological substrates of BPD are important, most etiological theories of BPD favor a stress diathesis approach, which argues for an interaction between biological and environmental vulnerabilities in the development of BPD. This approach is also consistent with a developmental psychopathology framework (68). When considering the biological underpinnings of BPD and using an attachment- and mentalization-based theoretical framework for BPD (69,70), we have begun to explore the mentalizing deficits that may underlie interpersonal dysfunction associated with BPD in adolescents. According to this theoretical approach, failures or impairments in mentalizing may account for the core features of BPD. The term *mentalizing* refers to our ability to interpret the behavior of others and ourselves in terms of mental states—that is, to treat ourselves and others as psychological agents with thoughts, feelings, desires, and so on (71). For optimal mentalizing capacity to develop, the child's attachment relationships play an important role, and disruptions of early attachment experiences can derail social-cognitive development, thereby leading to BPD (72). In this model, emergent mentalizing capacity is the result of both biological and genetic factors (inherited theory of mind capacity and sensitive temperament) and environmental factors (adverse or mismatched family environment). A recent meta-analysis of functional brain imaging studies of theory of mind (73) identified the existence of a "core network" for mentalizing, including the medial prefrontal cortex (mPFC) and the bilateral posterior temporal parietal junction (TPJ). Although work in our laboratory has yet to incorporate the biological level of explanation into study designs, we have begun examining mentalizing correlates of BPD in adolescents at the behavioral phenotypic level.

For instance, we used the Movie for Assessment of Social Cognition (MASC;74) in a sample of 132 inpatient adolescents ($M_{age}$ =15.5; 62% girls; 23% meeting criteria for BPD;75) to assess mentalizing capacity. During the administration of this tool, research subjects watch a 15-minute movie of four characters interacting around interpersonally and emotionally salient topics. The movie is paused multiple times, and research subjects are asked to reflect on the thoughts and feelings of the movie characters. Four mutually exclusive mentalizing styles are provided: no mentalizing (no reference is made to mental states to understand or explain characters' behavior), undermentalizing (some reference is made to mental states, but it falls short of fully capturing the nuance of the interpersonal situation), hypermentalizing (mental states are over-attributed, and inferences are based on little or no existing evidence), and accurate mentalizing. Results demonstrated that, of all four mentalizing styles, only hypermentalizing was associated with borderline traits; this behavior also distinguished subjects who met criteria for BPD from those who did not.

In a follow-up study, we measured hyper-mentalizing at discharge in a larger sample of adolescents from the same inpatient unit. Here, we demonstrated a significant reduction in hyper-mentalizing, which was associated with a significant reduction in borderline traits (76). Importantly, in this study we assessed other forms of mentalizing at admission and discharge as well, given the fact that mentalizing is a multifaceted construct. These forms of mentalizing included explicit mentalizing through an emotion recognition task, empathy, and pseudomentalizing. Only pseudomentalizing showed similar associations with borderline symptom reduction, although these associations were not as significant as those associated with hypermentalizing. That both hypermentalizing and pseudomentalizing were sensitive to treatment effects was not surprising, because both hypermentalizing and pseudomentalizing are considered to be indices of what Fonagy and Luyten (77) consider to be the "pretend mode" in mentalization theory. Pretend mode is a form of impaired mentalizing in which thoughts and feelings become severed from reality. Pseudomentalizing in particular involves the use of mental states that appear to be rooted in reality but that, on closer examination, are found to be suspended from it.

*Summary*

Although the above studies were important to establish the initial empirical evidence for mentalizing impairment associated with BPD in the specific form of hypermentalizing, they are far from establishing the biological basis of adolescent BPD from the perspective of mentalizing capacity. For this to occur, the biological level of explanation must be integrated into mentalization-based studies of adolescent BPD in the form of, for instance,

neuroimaging. Such studies have been conducted in adults (e.g.,78), and they demonstrate the potential of the mentalizing construct to serve as a laboratory-assessed biological correlate of BPD. Other potential targets for laboratory studies in the Robins and Guze framework include reward function (79) and emotion dysregulation (80). Of all the Robins and Guze principles, the need for laboratory studies to establish the neurobiological substrates of a disorder is perhaps most strongly compatible with the RDoC, which we will return to later in this review.

**Delimitation from Other Disorders**

A particular challenge for the validity of BPD has been its high comorbidity with other disorders. Earlier, we discussed how the BPD criteria have been factor analyzed with other PDs in an attempt to demonstrate that BPD is a discrete disorder. These studies were conducted due to the high comorbidity between BPD and other PDs, but they did not replicate the DSM-based 10-factor structure for PD. Adult BPD demonstrates equally high comorbidity with both internalizing and externalizing disorders (81-88), and adolescent studies have found similar patterns of comorbidity (89-91). These high rates of comorbidity suggest that underlying structures may account for observed comorbidity (covariance) among disorders (47). James and Taylor (48) found that BPD served as a multidimensional indicator of both the externalizing dimension and the anxious-misery (distress) subfactor of the internalizing dimension across both genders. Using more rigorous assessment tools and a larger and more diverse sample, Eaton and colleagues (47) replicated this finding, again in both genders. We recently attempted to replicate these findings in a sample of 434 adolescent inpatients. With the use of a confirmatory factor analytic approach, we found support for the notion that adolescent BPD is a confluence of internalizing and externalizing pathology (92). Broadly speaking, our findings replicated those of both Eaton and colleagues (47) and James and Taylor (48): adolescent BPD loaded onto both internalizing and externalizing dimensions, and this finding was robust across both genders while retaining unique variance in the model. In other words, complete overlap with internalizing and externalizing disorders did not occur.

*Summary*

That BPD appears to be a confluence of internalizing and externalizing disorders (47), despite retaining unique variance, challenges the notion that BPD can be successfully delimited from other disorders. These findings point to the value of taking a dimensional approach to psychopathology in general and personality pathology in particular, because this allows for the cross-loading of components of disorder onto common latent factors. Therefore, in contrast with a categorical approach that requires the demonstration of BPD as a discrete disorder, the dimensional approach can describe how features of BPD may be shared with other clinical syndromes and personality pathology. We have begun to explore such models in the field of adolescent BPD, and much work is still to be done. However, it is becoming clear that the Robins and Guze requirement of discrete categorical diagnostic entities may not reflect the reality of psychopathology.

**Follow-Up Studies**

Robins and Guze's fourth principle suggests that studies should be conducted that demonstrate a prototypical course and outcome of the symptoms. Although there is an urgent need for prospective studies of BPD, research conducted by other groups provides evidence for a prototypical course and outcome of adolescent BPD symptoms. For example, research has pinpointed the age of 13 years as a typical age of onset for BPD (93). Although it is true that adolescence is characterized by a high prevalence of all psychiatric disorders (94), adolescence appears to be a particularly vulnerable period for BPD, hence the urgent calls from research for early intervention (41). As with all psychiatric disorders, an early onset of BPD usually denotes poorer outcomes (95). With its onset during adolescence, BPD then typically peaks during early adulthood and subsequently declines (96). Moreover, research has suggested a heterotypic developmental course for BPD; this involves coherence with regard to the underlying organization or meaning of behaviors over time (as opposed to homotypic continuity, where coherence is evident at the level of the behavioral phenotype [97]). Specifically, it appears that externalizing features are most strongly manifested early during the course of BPD (98), with internalizing features becoming more prominent throughout adulthood (47). Similar to the course of adult BPD, stability appears to be moderate with adolescent BPD (99-101).

*Summary*

Although research is sparse, preliminary evidence is strong for Robins and Guze's fourth criteria for the validity of psychiatric disorder. Research has demonstrated the comparable stability of BPD among adolescents, which has also been demonstrated for adults (99), as well as a typical course, which involves symptoms appearing during

adolescence, peaking during early adulthood, and then declining (96).

**Family Studies**

Robins and Guze's fifth principle states that family studies must identify a genetic basis for the biological phenomena associated with the disorder. Several genetic studies have been conducted for BPD in adults, and these studies have demonstrated moderate heritability (96). Distel and colleagues (102) estimated a heritability of .42 in an adult twin design, and heritability rates of .69 (103) and .60 (104) were demonstrated in other studies. Recently, two studies have been conducted involving adolescents. The first study (105) made use of a longitudinal birth cohort sample of 1116 pairs of same-sex twins who were assessed from the ages of 5 to 12 years. The study aimed to demonstrate the consistency of the cause of borderline personality–related characteristics (BPRCs) in children with theoretical models of the causes of adult BPD. Results demonstrated that, at the age of 12 years, mother-reported BPRCs were correlated at 0.66 among monozygotic twins and at 0.29 among dizygotic twins, with findings pointing to genetic factors accounting for 66% of the variance in BPRCs. In addition, children categorized as belonging to an "extreme borderline group" (i.e., having more BPRCs) at the age of 12 years were the same children who, at the age of 5 years, had significantly lower levels of intelligence, self-control, and theory of mind and higher levels of impulsivity, externalizing, and internalizing problems. These children also had high rates of co-occurring conduct disorder, depression, anxiety, and psychosis. Finally, consistent with a diathesis-stress model, exposure to harsh treatment in the family environment (e.g., physical maltreatment, maternal negative expressed emotion) through the age of 10 years predicted BPRCs as well as categorization in the "extreme borderline" group at the age of 12 years. This finding suggested that environmental mediation was stronger among children with a family history of psychiatric illness. Taken together, this study provides important evidence for a genetic basis of borderline-related traits. Perhaps more importantly, it provides evidence for the interaction between genetic and environmental influences on the emergence of borderline-related traits in youth, a finding that is consistent with etiological models of adult BPD.

In the second study (106), the longitudinal course and heritability of BPD traits were examined over a 10-year period beginning at the age of 14 years and ending at the age of 24 years using a twin design. Mean-level BPD traits were found to decline significantly from adolescence to adulthood, but rank order stability remained high. BPD traits were moderately heritable at all ages, with a slight trend toward increased heritability from the age of 14 years to the age of 24 years.

Although several candidate genes have been implicated for adult BPD (e.g., tryptophan hydroxylase [107], 5HT2a [108], 5HT2c [109], monoamine oxidase [110]), thus far, only one study has examined genetic polymorphisms associated with adolescent BPD. In a replication of findings from adults with BPD, an association between 5-HTTLPR and BPD traits in children and adolescents between the ages of 9 and 15 years were demonstrated; carriers of the short allele of 5-HTTLPR exhibited the highest levels of BPD traits (111).

*Summary*

Since 1970, when Robins and Guze published their article calling for a systematic approach to establishing the validity of psychiatric disorder, research in the fields of behavioral and molecular genetics and the neurosciences have been revolutionized. At that time, one-to-one relations between genotypes and psychiatric phenotypes were thought possible, and gene–environment interactions in psychiatric disorders may have been considered but certainly not modeled. Today, we know that, for most psychiatric disorders (including BPD), evidence supports a view that constitutional factors (e.g., temperament) and environmental factors (e.g., family factors) both have etiological roles. For example, in an innovative recent study using a twin design (112), the temperamental traits of behavioral disinhibition or externalizing (EXT; impulsivity and inability to inhibit undesirable actions) and negative emotionality or internalizing (INT; predisposition to experience depression, anger, and anxiety) in interaction with child abuse (CA) to predict borderline traits over time was investigated. Three causal models were tested: a direct causal model (CA → BPD), a diathesis stress model (INT/EXT x CA → BPD), and a genetic mediation model where the CA–BPD association was better accounted for by common genetic risk factors (i.e., INT, EXT, or additive INT and EXT psychopathology could account for genetic or environmental influences common to CA and BPD). The authors found the strongest support for a genetic mediation model in which the association between exposure to traumatic events and BPD may be better accounted for by common genetic influences rather than the former causally influencing the latter.

More studies of gene/biology–environment interactions are now needed. There is also an urgent need for studies that consider more than one

biological level of explanation simultaneously. The limitations of the Robins and Guze approach are clear, and we can now turn our attention to the NIMH's answer to these limitations.

**National Institute of Mental Health Research Domain Criteria**

In the above review of the Robins and Guze criteria, we have shown that the construct of adolescent BPD meets some of these criteria. For example, there is some evidence for the unidimensional factor structure of adolescent BPD (although we acknowledge that this evidence was found at a low threshold), and there is some evidence for neurobiological correlates that are specific to adolescent BPD. We have also discussed research that demonstrates a meaningful course for the disorder during adolescence. However, the discussion of the Robins and Guze criteria has also revealed limitations of the construct validity of adolescent BPD. These limitations may be due to the fact that BPD is an invalid construct during adolescence. However, limitations may also be the result of the possibility that the Robins and Guze criteria are not an appropriate yardstick for evaluating the validity of psychiatric disorders. It is in this context that we now turn to an alternative approach to developing and evaluating the construct validity of psychiatric disorders.

The NIMH developed the RDoC in response to long-held criticisms of the DSM system of validating psychiatric disorder, which, as we discussed, was informed by the Robins and Guze criteria. In contrast with this system, the RDoC does not subscribe to a categorical system but rather propagates a dimensional system that spans the range from typical to atypical behavior. In addition, although the DSM system of diagnostic criteria was by and large determined by consensus (15), the RDoC are agnostic with regard to DSM categories. Instead, the RDoC seeks to generate classifications from basic behavioral neuroscience. Whereas the Robins and Guze system sought to find the neurobiological substrates for DSM diagnosis, the RDoC's starting point is knowledge of behavior–brain relations that can then be linked to clinical phenomena; this is typically based on dimensional ratings that cut across different psychiatric disorders (17). To this end, the RDoC provides a matrix with rows that cover five domains of function: negative valence systems, positive valence systems (approach/motivation), cognitive systems, systems for social processes, and arousal/regulatory systems. Each of the domains is associated with relevant constructs selected for the potential that a particular brain circuit or area could reasonably be specified to implement that dimension of behavior. For example (and of most relevance to the laboratory studies described previously), the RDoC systems for social processes include the following constructs: imitation, theory of mind (mentalizing), social dominance, facial expression, identification, attachment/separation fear, and self-representation. The columns of the matrix represent seven different units of analysis: genes, molecules, cells, neural circuits, physiology, behaviors, and self-report. The idea is that researchers would study these constructs across psychiatric disorders and make use of latent trait analyses to "carve nature more closely at its joints."This is done in the hope that we would develop over time a more valid nosology of psychiatric disorder.

To our knowledge, the RDoC has not yet been applied to either adult or adolescent BPD. We believe, however, that the RDoC system holds promise for further evaluating and establishing the validity of BPD during adolescence.

**Future Research Directions**

In this article, we have presented some of the research that we have conducted in our own laboratory as well as the research conducted by others to evaluate the validity of BPD in adolescents. Like most research in psychiatry, our work has been guided by the influential Robins and Guze criteria for the validity of psychiatric disorder. As discussed, the limitations of this approach are now well acknowledged, and the question arises as to how we can advance knowledge regarding the validity of adolescent BPD within the new NIMH RDoC framework. A natural first step is to abandon studies that recruit subjects based on the inclusion criterion of a BPD diagnosis. Instead, large cohorts of patients and non-patients who represent a broad spectrum of psychiatric diagnoses should be recruited. Like recent studies that make use of latent trait approaches to locate BPD within the internalizing/externalizing spectrum (47,48,92) or to evaluate BPD in the context of other PDs (113), studies of adolescent BPD must recruit across the full spectrum of typical/atypical behavior. Measures that assess the behavioral phenotype must then be used alongside biological measures and preferably across multiple levels (e.g., genes, molecules, cells, neural circuits, physiology). Multivariate statistics can then be employed to find patterns of covariation among variables. Latent class analyses can also be employed if a more person-centered approach is preferred. This enterprise is risky not only for BPD but also for other psychiatric disorders, because findings may challenge our long-held implicit belief system, which guides everyday clinical decision making. Alternatively, to better understand and effectively treat psychiatric illness,

we must be willing to flexibly expand on the foundational work that has been conducted during the past 40 years. Making the decision to embark on this endeavor may in time leave us pleasantly surprised.

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