Current Trends in BPD Research as Indicative of a Broader Sea-Change in Psychiatric Nosology

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The aim of the Special Section that this paper contributes to is to review current trends in borderline personality disorder (BPD) research. Three major trends were identified. First, there has been a marked increase in studies that attempt to locate BPD in the dimensional latent structure of psychopathology. Second, identifying the endophenotypic markers associated with BPD has become a focus of interest. Here, we focus on one endophenotype in the form of impaired self-other processing. Third, there has been an explosion of research into the developmental aspects of BPD specifically focused on uncovering complex Biology × Environment interactions in the development of BPD. This paper discusses how these trends (Dimensions, Biology, and Development) are challenging the nature and form of BPD as we know it, and may be indicative of a broader sea-change in psychiatric nosology.

Keywords: borderline personality disorder, trends, dimensions, biology, developmental

In Shakespeare's *The Tempest*, the supernatural spirit, Ariel, sings to Ferninand, a prince of Naples, after Ferdinand's father's apparent death by drowning that "Nothing of him that doth fade, but doth suffer a sea-change, into something rich and strange." At any point in the history of a scientific discipline, we can pause to assess current trends. Sometimes, however, current trends represent a Shakespearean sea-change, or what intellectual historians refer to as a paradigm shift or a change in epistemic constellations. Of course, whether a true paradigm shift is occurring can only be determined in hindsight. However, I will argue in this article that three major trends in BPD research (simply referred to here as Dimensions, Biology, and Development) are representative of a broader sea-change or even a paradigm shift in psychiatric nosology. I will begin by providing a fuller understanding of what constitutes a paradigm shift or change in epistemic constellation. I will then discuss three major trends in BPD research (by no means the only ones!). The paper will conclude with an assessment of these trends as indicative of a broader sea-change in psychiatric nosology.

Paradigm Shifts and Epistemic Constellations

In Kuhn's (1962) *The Structure of Scientific Revolutions,* he describes radical transformations within specific scientific disciplines and fields of research. Central to Kuhn's argument is the notion of a "paradigm," generally described as a "scientific achievement" that provides "model problems and model solutions" within a given scientific discipline. Such paradigms undergo a "crisis" as soon as an increasing number of either new phenomena, or specific aspects of already known phenomena, begin to resist well-established model solutions. These crises lead to the revolution of an existing body of knowledge and, ultimately, albeit slowly, begin to introduce a new paradigm that provides a *normative standard* for the scientific discipline. Although it might seem at first sight that such transformations are intrinsic to the scientific discipline or field of research in question, governed by a logic that is internal to the discipline, the transformations are shifts in "the entire constellation of beliefs, values, techniques, and so on" that are shared by a larger community of scientists, or what Fleck (1935) described as a "thought collective" and Emden (2004) described as "epistemic constellations." Such a thought collective is characterized by a particular "thought style" as it comes to the fore not only in the language and concepts used to describe scientific problems, but also in the concrete practices relevant to specific experiments and to the presentation of research results. As a consequence, paradigm shifts are deeply connected to practical forms of reasoning about scientific problems as well as to our practical engagement with the material of any given scientific discipline. This practical engagement naturally places certain constraints and at the same time facilitates new developments. In this regard, Latour (1987) talks about "technoscience" and Bachelard about "phenomenotechniques" (Rheinberger, 2005) to describe the way in which experimental phenomena are never pure, or raw, or even innocent, but always bound up with the technologies that render such phenomena visible or statistically relevant. These technological constraints—from bio assays and genetic fingerprinting to functional magnetic neuroimaging (fMRI) scanners—are "boundary objects" (Star & Griesemer, 1989) that is, the same objects or models are used by different groups and communities, and within different material contexts, in different ways, thus crossing seemingly well-established boundaries between different fields of research. As a consequence, paradigms are messy business, on the one hand constrained by both the context within which

research takes place and the historical past of the scientific discipline in question, but also necessarily open toward unexpected events and phenomena—Shakespeare's sea-change into something rich and strange.

Trend #1, Dimensions: Locating BPD in the Dimensional Structure of Psychopathology

If BPD exists as a categorical diagnosis, its criterion set must constitute a coherent combination of traits and symptoms that "hang together" (Robins & Guze, 1970), as would be evidenced by a single common factor adequately accounting for covariation among the criteria. Moreover, BPD should demonstrate qualitative distinctiveness from other disorders. However, BPD has been found to be a highly comorbid disorder with typical comorbidity rates of 50% or more within traditional Axis II and/or with Axis I disorders in clinical samples (Clark, 2007). Moreover, factor analytic studies over the last 20 years across different measures and informants have failed to support the DSM's putative PD structure suggesting limited evidence in support of a categorically defined BPD diagnosis (see Sheets & Craighead, 2007 for a review). Instead, it appears that several latent variables (other than DSM-based diagnoses) underlie PD symptoms and/or criteria. For instance, in the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) study of 35,000 adults, lifetime PD criteria were best accounted for by seven factors: paranoid, avoidant/dependent, antisocial, schizoid, obsessive–compulsive, emotional/cognitive dysregulation, and narcissism (Trull, Verges, Wood, & Sher, 2013), closely mirroring the seven factors identified by Thomas, Turkheimer, and Oltmanns (2003), (with the exception of the emotional/cognitive dysregulation factor).

Influenced by a growing trend in psychopathology research to consider models that evaluate general factors that account for both common variance shared across diagnoses and unique sources of variance that may represent more specific forms of psychopathology, we recently evaluated a bifactor model of personality pathology using the six sets of *DSM–5*-II PD criteria that account for the vast majority of diagnosed PD (i.e., borderline, avoidant, obsessive–compulsive, narcissistic, antisocial, and schizotypal; American Psychiatric Association [APA], 2013). Specifically, we hypothesized that PD criteria were best accounted for by a common general factor of personality pathology (PD "g") and six completely or partially distinct specific factors of personality pathology (PD "s" factors; Sharp, Wright, et al., 2015). Consistent with prior research (Aggen, Neale, Roysamb, Reichborn-Kjennerud, & Kendler, 2009; Clifton & Pilkonis, 2007; Conway, Hammen, & Brennan, 2012; Fossati et al., 1999), we found strong support in our data for a single latent factor underlying the nine BPD criteria when examined in isolation. Results supported our hypotheses with five of the six specific factors consistent with traditional PD constructs, whereas the sixth was residual. However, consistent with our hypothesis, there simply was no specific BPD factor after including a general factor; rather, BPD items loaded most strongly, and virtually entirely, on the general "g" factor. BPD symptoms therefore "hung together" when examined in isolation, but "disappeared" into a general trait when modeled alongside other PDs.

Combined, the work discussed above represent a first major trend: Dimensions. Central to this major trend is the principle that

there are *dimensionally defined observable behaviors and symptoms* that have unobserved underlying causes that may explain the patterns of variance and covariance among the variables (e.g., symptoms, diagnoses, etc.; Wright & Zimmermann, 2015). In this sense, seemingly distinct mental disorders may be manifestations of common underlying dimensions (or categories). Although early work on the structure of psychopathology had been conducted (e.g., Achenbach & Edelbrock, 1978), it was not until recent developments in statistical modeling that we see momentum building toward viewing mental disorders as arbitrary distinctions along underlying (latent) dimensions. In fact, a simple Web of Science search indicates about 2–4 papers on the metastructure of psychopathology up until 2009, with an increase to 6 papers per year between 2009 and 2011 and thereafter an almost threefold increase of papers each year (see Figure 1).

Most of this research has focused on elucidating the metastructure of the most common psychopathology, *excluding* BPD. Research in adult (Forbush & Watson, 2013; Krueger, Caspi, Moffitt, & Silva, 1998) and child psychopathology (e.g., Lahey et al., 2012) indicate that prevalent mental disorders are organized into higher order Internalizing and Externalizing dimensions (see Eaton, South, & Krueger, 2015 for a recent review), with more recent work including a Psychotic/Thought Disorder higher order factor (Wright et al., 2013), and an additional higher-order general Psychopathology factor (the "p-factor") that accounts for shared variance between Internalizing, Externalizing, and Thought Disorder (Caspi et al., 2014; Patalay et al., 2015). The Internalizing–Externalizing-thought disorder structure has demonstrated strong empirical and statistical evidence for its validity in terms of treatment response and genetic overlap (Kendler et al., 2011). The location of BPD within this metastructure of psychopathology has been evaluated in only seven studies thus far (Blanco et al., 2013; Eaton et al., 2011; James & Taylor, 2008; Kotov et al., 2011; Markon, 2010; Røysamb et al., 2011; Wright & Simms, 2015). In two of these studies the location of BPD (in absence of other PDs) was evaluated within the Internalizing–Externalizing spectra and was shown to be a confluence of the two underlying factors (Eaton et al., 2011; James & Taylor, 2008). The other studies included other PDs and in some cases Thought Disorder, alongside BPD. Four of these studies demonstrated two additional underlying factors beyond Internalizing, Externalizing, and Thought Disorder, namely Introversion and Antagonism, which emerged when PDs were added to traditional Axis I disorders in a latent variable model. The Internalizing, Externalizing, and Thought Disorder dimensions identified in these studies bear a striking conceptual resemblance to the pathological personality trait domains included in the *DSM–5* Section III system of PDs, such that Negative Affectivity relates to Internalizing, Disinhibition to Externalizing, and Psychoticism to Thought Disorder (Wright & Simms, 2015). Moreover, the addition of PDs at the observed level in these studies introduces dimensions of maladaptive social/interpersonal functioning into the structure of psychopathology that were previously not captured when traditional Axis I disorders were modeled alone. This conclusion is consistent with the view that PDs reflect disorders primarily in the interpersonal domain (Benjamin, 1993; Hopwood, Wright, Ansell, & Pincus, 2013; Pincus, 2005; Sharp & Fonagy, 2008; Wright & Simms, 2015). Indeed, one interpretation for the "disappearance" of BPD into a PD "g" factor in Sharp et al. (2015) is that BPD criteria may capture the impairment in person-

Published Research Articles on the Meta-structure of Psychopathology*
1978-2015

*Literature searches conducted via *PSYCInfo* and *Web of Science* with search terms of "Latent Structure of Psychopathology", or "Meta-structure of Psychopathology". Search results yielded 99 published empirical articles from 1978 to 2015.

Published Research Articles on the Endophenotype and Psychiatric Disorder*
2000-2014

*Literature searches conducted via *PSYCInfo* and *Web of Science* with search terms of "Endophenotype and Psychiatric Disorder", or "Endophenotype and Psychopathology". Search results yielded 361 published empirical articles from 2000 to 2015.

Published Research Articles on BPD in Youth*
1990-2013

*Literature searches conducted via *PSYCInfo* and *Web of Science* with search terms of Borderline Personality (Disorder, Pathology) or BPD and Adolescent(s), Child(ren), Youth(s), Juvenile,(s) Girl(s), or Boys(s). Search results yielded 196 published empirical articles from 1990 to 2013.
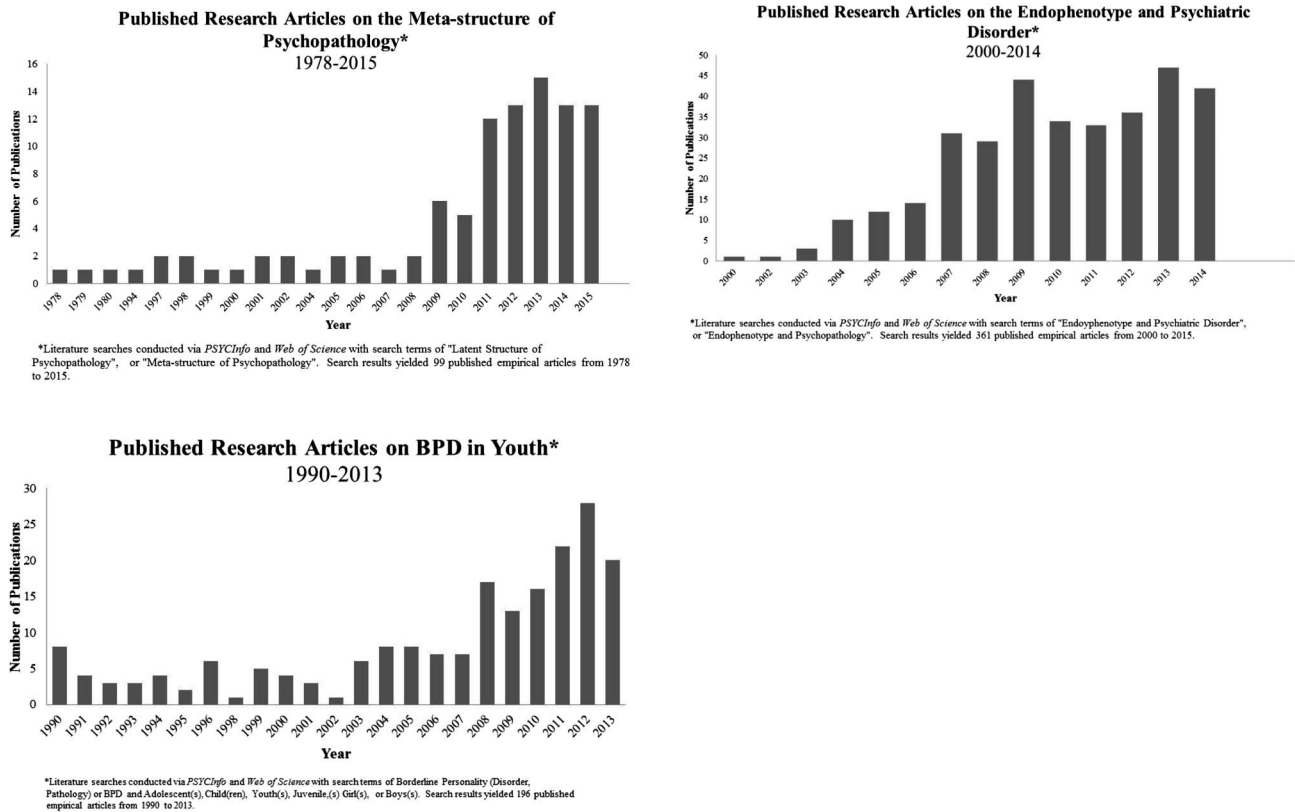
Figure 1. Trends in psychopathology and BPD research.

ality (self/interpersonal) functioning defined in Criterion A of the *DSM–5*-Section II. As described by others (Bender & Skodol, 2007; Fonagy & Luyten, 2009; Kernberg, 1984; Linehan, 1993), BPD is unique in that impairment in the ability to maintain and use benign and coherent internal images of self and others are explicitly reflected in five out of nine symptoms (abandonment, unstable relationships, identity disturbance, paranoid ideation, emptiness), with the other four symptoms manifesting themselves most often in interpersonal contexts (anger, affective instability, suicidal behaviors, impulsivity). This notion is further consistent with the only other bifactor study of DSM PD pathology, which also suggested that the general factor of PD pathology may primarily reflect lack of self–other integration (Jahng et al., 2011).

In summary, under the broader theme of *dimensions*, many current trends in BPD research may be observed. Indeed, no less than 18 proposals have been suggested for a dimensional model of PD, with the five-factor model having been shown to be particularly promising (see Widiger & Trull, 2007 for a review), alongside the *DSM–5* Section III proposal for Criterion B. The Dimensions trend in BPD research is important because it clearly utilizes new "technoscience" or "phenomenotechniques" in the form of advanced quantitative methodology. These studies also reflect consistent, palpable, and vocal resistance to the categorical DSM system and therefore challenge the very existence of BPD as we know it. For some, this potential paradigm shift causes anxiety; for others, reconceptualizing BPD in terms of dimensions (as does Section III), or viewing BPD as part of a broader covariance

structure of PD pathology (Sharp, Wright, et al., 2015) or common mental disorders (Eaton et al., 2011), gives us the tools to capture BPD more precisely and opens avenues for future research on BPD that may actually promote research on the disorder.

Trend #2, Biology: Mentalization as Endophenotype

At around the same time that consistent, palpable, and vocal resistance to the categorical system began to emerge in the lead-up to the *DSM–5*, the concept of the "endophenotype" was reintroduced in the psychopathology literature (Gottesman & Gould, 2003). This stimulated an explosion of research into endophenotypic markers of psychiatric disorders (see Figure 1). It also stimulated a convergence of the literatures on endophenotypes and dimensional concepts of psychopathology (Cuthbert, 2005; Miller & Rockstroh, 2013) in a way that allowed the integration of psychological and biological phenomena hitherto constrained by the limitations of the categorical model of psychopathology, on the one hand, and assumptions about simple gene–behavior relations, on the other hand. Endophenotypes are defined as "relatively well-specified physiological or behavioral measures that are considered to occupy the terrain between disease symptoms and risk genotypes" (Insel & Cuthbert, 2009, p. 988) that are heritable and assumed to be biologically valid. As such, endophenotypic markers are not always and exclusively biological, but bridge various levels of explanation (e.g., genes, symptoms, behavior, psychological processes, brain structure and chemistry; Gottesman &

Shields, 1973; Miller & Rockstroh, 2013). Crucially, through their variation with environmental influences, they help characterize gene–environment (G×E) interactions (Dick, 2011) and are often identified as malleable treatment targets to facilitate a bridge between basic science and practice (Sharp & Kalpakci, 2015).

Clinical dimensions of PDs that lend themselves to the study of corresponding endophenotypes include affective instability, impulsivity, aggression, emotional information processing, cognitive disorganization, social/interpersonal deficits, and psychosis (Siever, 2005). Given space limitations, and consistent with Criterion A of the new General Criteria of Personality Disorder, which states that a PD diagnosis requires moderate or greater impairment self/interpersonal functioning, we will focus here on the interpersonal domain. Readers are referred to recent reviews on the state of the science for other endophenotypes in BPD research, in particular for affective instability/emotion processing (Schmahl et al., 2014) and impulsivity (Crowell, Beauchaine, & Linehan, 2009). The interpersonal domain maps onto the National Institute of Mental Health's (NIMH's) Research Domain Criteria (RDoC) "systems for social processes," which consist of the following domains and constructs: Affiliation and attachment, Social communication, Perception and understanding of self, and Perception and understanding of others. This domain is currently studied most prominently within the conceptual framework of the mentalization-based theory of BPD (Fonagy, 1989, 1991; Fonagy & Bateman, 2008).

The mentalization-based theory of BPD posits that the core features of BPD can be explained by a vulnerability in mentalizing. Mentalizing is a multicomponent concept and defined as the metacognitive capacity to think about one's own thoughts and feelings and those of others in an attempt to predict and understand behavior (Bateman & Fonagy, 2012). It involves attributing mental states (e.g., emotions, desires, beliefs) to self and others and forms the basis for attachment relationships and the development of self (Fonagy, 1991; Fonagy, Gergely, Jurist, & Target, 2002). Mentalizing includes both interpersonal ("other") and intrapersonal ("self") processing and involves both cognitive and emotional processing. Disruptions of early attachment experiences have been suggested to derail social–cognitive development (Fonagy, Steele, & Steele, 1991), thereby leading to BPD (Sharp, Venta, et al., 2015).

Although a significant body of literature now exists in support of mentalizing deficits associated with BPD (see, e.g., Fonagy & Luyten, 2009; Jeung & Herpertz, 2014 for reviews), results have been somewhat mixed. Therefore, the mentalization-based theory of BPD was recently further delineated by suggesting that mentalizing dysfunction in BPD is present not in the form of failure or suppression, but in the form of excess mentalizing (hypermentalizing; Sharp, 2014; Sharp & Vanwoerden, 2015). Hypermentalizing is a type of mentalizing failure that involves making assumptions about other people's mental states that go beyond observable data. As such, it involves overattribution of mental states to others and their likely misinterpretation. Hypermentalizing is therefore by its very nature indicative of a metacognitive deficit because an individual engaging in hypermentalizing is failing to attain a higher-order representation from which question his or her own belief in service of generating an alternative hypothesis regarding a distressing situation (Semerari et al., 2005). More specifically, hypermentalizing reflects a lack of metacognitive differentiation (Semerari et al., 2005), because representation is conflated with reality, consistent with the notion of thinking errors of the "mindreading" type as originally suggested in cognitive-behavior therapy approaches (Beck, 1967, 1983).

In support of the above model, we showed that when presented with several mutually exclusive response options to social stimuli (no mentalizing, less mentalizing, hypermentalizing, and accurate mentalizing), hypermentalizing was the only mentalizing subscale that correlated with borderline features in adolescents (Sharp et al., 2011). We showed that across different tasks, each differentially operationalizing mentalizing, hypermentalizing was uniquely sensitive to change in inpatient treatment and correlated with reduction in borderline symptoms as a function of treatment (Sharp et al., 2013). On the basis of these findings, we put forward a hypermentalizing model of BPD (Sharp, 2014; Sharp & Vanwoerden, 2015), which describes a recursive pattern in which increased arousal, lack of integration across cognitive modalities, and errors in interpretation are precursors to the ultimate endpoint of hypermentalizing, and associated with escalating emotion dysregulation and confusion between mental states emanating from self versus the other. Additional support for hypermentalizing impairment in BPD comes from studies that demonstrate heightened sensitivity to social stimuli in various paradigms. Specifically, individuals with BPD tend to be attuned to information that may reflect a social threat, suggesting overattribution of mental states to others in the form of misattributions of malevolence or negative attributes. In a series of studies (Segal, Westen, Lohr, & Silk, 1993; Segal, Westen, Lohr, Silk, & Cohen, 1992; Westen, Lohr, Silk, Gold, & Kerber, 1990a; Westen, Ludolph, Block, Wixom, & Wiss, 1990b; Westen, Ludolph, Lerner, Ruffins, & Wiss, 1990c), individuals with BPD expressed more malevolent representations of others' actions compared to psychiatric and healthy controls, including a malevolent object world, a relative incapacity to invest in others in a non-need-gratifying way, and a tendency to attribute motivation to others in simple, illogical, and idiosyncratic ways. Consistent with these findings, other, more recent studies have shown higher rates of sensitivity to rejection associated with BPD, both when excluded and when not excluded from social interactions (Gunderson, 2007; Staebler et al., 2011). Finally, in appraising neutral social–emotional stimuli, individuals with BPD have been shown to be more likely to assign negative valence (Arntz & Veen, 2001; Daros et al., 2013).

However, for hypermentalizing to be an endophenotype it must demonstrate biological validity. Mentalizing, derived from the evolutionary-based term "theory of mind", was originally developed to capture mindreading capacity in chimpanzees (Premack & Woodruff, 1978). It has since been shown to be highly heritable during the preschool years (Hughes & Cutting, 1999) and is influenced by the dopamine-related COMT gene (Xia, Wu, & Su, 2012) as well as an oxytocin receptor gene (Wu & Su, 2015). In addition, 20 years of research on the neurobiological correlates of mentalizing has identified a "core network" across different theory of mind tasks including the medial prefrontal cortex and bilateral posterior temporal parietal junction with specific tasks probing differential brain activation in additional regions (Schurz, Radua, Aichorn, Richlan, & Perner, 2014). If the model described by Sharp (2014) is correct, then the neurobiological correlates of *hypermentalizing* are likely to also include the amygdala and insula given the model's focus on affective- and self-processing and these brain areas' role in affective and self-processing. For

instance, neurobiological studies in BPD have demonstrated enhanced amygdala response coupled with regulatory deficits of the orbital and prefrontal cortices with the presentation of social–emotional stimuli (Donegan et al., 2003; Frick et al., 2012; Minzenberg, Fan, New, Tang, & Siever, 2007), even in the absence of behavioral differences between BPD and control groups (Mier et al., 2013). Similarly, studies using interactive social paradigms (e.g., behavioral economic trust games) have demonstrated reduced insular activation (King-Casas et al., 2008) which at the level of social-information processing would include a misattribution of mental states to others (e.g., "How dare she give me a low offer when I have been giving her strong offers—she must be out to get me"; or "That's it! She is clearly not respecting my offer. I'm not putting any further effort into this exchange."; Sharp & Vanwoorden, 2015).

The overattribution of erroneous intentions to partners may of course also signal a confusion between the subject's own intentions and those projected onto the interaction partner. Consistent with the idea of confusion between self-and-other mental states, Frick et al. (2012) found that in the context of an emotion recognition paradigm, although BPD patients had superior facial emotion recognition, they also had associated increased activity in the left inferior frontal gyrus. This brain region is believed to be a part of the mirror neuron system associated with the understanding of motor events and their intentions. This suggests a greater resonance with the others' mental states in BPD, in contrast to healthy controls who showed greater activation in the insula and superior temporal gyri, areas typically associated with mental state discrimination (Baron-Cohen et al., 1999). This merging of self and other in BPD is described by Fonagy and Luyten (2009) as a lack of agency associated with BPD and a subsequent overidentification with the mental states of others.

In summary, several dimensions of BPD lend themselves to the study of corresponding endophenotypes—in particular emotion dysregulation (Schmahl et al., 2014). An additional construct with significant endophenotypic potential is mentalizing due to its translational potential (Sharp & Kalpakci, 2015) and biological validity. Although mentalizing in the context of BPD is not new, the trend identified here is in the convergence of the new DSM focus on self–other processing for all PDs (Criterion A), the addition of the "systems for social processes" in RDoC to describe it, and new tools offered by social neuroscience to capture it. Although much research is still needed to further realize its potential, mentalization provides a useful conceptual framework to theoretically organize many of the constructs in the RDoC's Systems for Social Processes, most notably attachment, perception, and understanding of self and others. It also provides a useful framework for capturing Criterion A of DSM–5-Section III's focus on impairment in self and interpersonal functioning, said to "constitute the heart of diagnostics of personality disorders in future classifications" (Jeung & Herpertz, 2014, p. 222). Although other frameworks are also available (most notably the Interpersonal Circumplex; Pincus, 2005), these are not mutually exclusive, and both frameworks are already fairly mature in their offerings of "technoscience" or "phenomenotechniques" to aid in the assessment and treatment of Criterion A-related impairment. It should also be noted that the RDoC Systems for Social Processes are currently heavily weighted to hypomentalizing, despite emerging evidence in support of a bell-shaped curve in mentalizing capacity

with hypomentalizing representing one end and hypermentalizing representing the other end of the mentalizing continuum (for reviews see Crespi & Badcock, 2008; and Gambin, Gambin, & Sharp, 2015).

Trend #3 Development: Complex Transactional Developmental Models of BPD

Borderline personality pathology has its onset around puberty, peaks in adolescence, and declines thereafter (Chanen et al., 2004). In recent years, there has been an explosion of research in adolescent BPD (that is, a nearly fivefold increase in the last 10 years; see Figure 1; Sharp & Tackett, 2014). Therefore, a third major trend in BPD research can be said to be that of a developmental focus that defines adolescence as a kind of sensitive period for the development of BPD. This research has attempted to build empirical support for long-held developmental theories of BPD, which converge to suggest that BPD is the result of complex interactions of biological and environmental risk and protective factors (Sharp & Fonagy, 2015). In Linehan's biosocial theory (1993) and Crowell et al.'s (2009) expansion thereof, as well as Paris's (2005) diathesis-stress model, a complex, heterotypic trajectory from childhood vulnerability to adult BPD begins with heritable trait vulnerabilities in the form of emotional sensitivity/reactivity (Linehan, 1993) or trait impulsivity (Crowell et al., 2009). These trait vulnerabilities result in the acquisition of poor emotion-regulation skills primarily through aberrant socialization mechanisms in the family context (i.e., an invalidating family environment), ultimately culminating in the complex disorder of BPD. In Fonagy's attachment and mentalization-based theory of BPD (Fonagy & Luyten, 2009), as well as Gunderson and Lyons-Ruth's (2008)'s gene–environment developmental model, genetic factors (inherited theory of mind capacity and hypersensitivity to interpersonal stressors) and environmental factors (adverse family or other environment) lead to an escalation of problematic transactions between primary caregiver and child and, ultimately, BPD.

Exciting new research is showing how biologically based variables (potential endophenotypes) interact with the environment in the context of adolescent BPD. Arens, Grabe, Spitzer, and Barnow (2011) showed that the biologically based temperamental traits of harm avoidance and novelty seeking interacted with an invalidating (overprotected) environment to predict BPD five years later. Gratz, Latzman, Tull, Reynolds, and Lejuez (2011) showed how affective dysfunction moderated the association between emotional abuse and borderline features in children. In a large twin study, Belsky et al. (2012) demonstrated that children who experienced maltreatment and maternal negative expressed emotion were at greater risk of developing borderline features if they also had a positive family psychiatric history (that is, inherited liability). Similarly, using a twin design, Bornovalova et al. (2013) showed that the temperamental traits of behavioral disinhibition and negative emotionality interacted with child abuse to predict borderline traits over time and that this association was best accounted for by common genetic influences. Consistent with these findings, Jovev et al. (2013) also showed that maltreatment acted as a moderator of the relationship between temperament dimensions and increases in BPD over time. In further support of the notion of complex prospective transactional processes, Hallquist, Hipwell, and Stepp (2015) recently demonstrated reciprocal influences among harsh parenting, self-control, and negative emotion-

ality between ages 5 and 14 in predicting the development of BPD symptoms in adolescent girls ages 14 to 17. This group further demonstrated a significant interaction between negative emotional reactivity and family adversity, such that exposure to adversity strengthened the association between negative emotional reactivity and BPD symptoms (Stepp, Scott, Jones, Whalen, & Hipwell, 2016). In addition, several studies have demonstrated moderating (Deborde et al., 2012; Fossati, Feeney, Maffei, & Borroni, 2011; Kim, Sharp, & Carbone, 2014) or mediating (Sharp, Venta, et al., 2015) effects for social cognition in the relation between attachment representation and borderline features in adolescents. Early attachment experiences also appear to interact with genetic variants of the oxytocin receptor genotype and the FK506 binding protein 5 gene CATT haplotype in predicting borderline features (Cicchetti, Rogosch, Hecht, Crick, & Hetzel, 2014).

In summary, the studies discussed above have necessarily made use of dimensional models of psychopathology and have included a focus on biologically tractible constructs with endophenotypic potential (e.g., affective reactivity, mentalizing, and impulsivity). The developmental focus clearly therefore builds on the traction gained with the trends of Dimensions and Biology discussed earlier by highlighting key developmental concepts, including developmental trajectories, sensitive periods, and dynamic interaction of systems across development (Casey, Oliveri, & Insel, 2014). This research not only begins to empirically support developmental theories of BPD that have remained untested until very recently, but also provides very exciting avenues for early identification, prevention, and early intervention through its potential to build biological- and context-informed algorithms of risk. For instance, Caspi et al. (2014) combined Dimensions, Biology, and Development to demonstrate that variation in a biologically rooted general psychopathology factor (p-factor) has important consequences for the developmental unfolding of more specific forms of psychopathology along the Internalizing, Externalizing, and Thought Disorder spectra. Identifying a young person's position along this latent trait continuum may therefore identify unique risk trajectories. That individuals scoring high on the p-factor were furthermore characterized by low Agreeableness, low Conscientiousness, and high Neuroticism—which maps onto the description of BPD as defined in *DSM–5* Section III as high in Negative Affectivity, Antagonism, and Disinhibition—gives a special role for personality pathology in reconsidering the structure of psychopathology.

Trends in BPD Research May Be Indicative of a Paradigm Shift in Psychiatric Nosology

So are the trends of Dimensions, Biology, and Development special in any way? In this concluding section I will argue that these trends are not accidental, but may, along with other trends, signal a potential sea-change, paradigm shift, or change in epistemic constellation in psychiatric nosology. Specifically, I will suggest that these trends may signal a transformative crisis in the field that, although a long time coming, may finally, and in a very real way, be challenging the normative standard in psychiatric nosology as represented by the DSM and by federal funding priorities.

Mental disorder can be pragmatically defined "by the useful purposes it is meant to serve" (Frances & Widiger, 2012, p. 113). In this sense, mental disorders are best understood as "useful constructs" (Borsboom, Mellenbergh, & van Heerden, 2003). Over

the last 10–15 years, criticisms centering on the "usefulness" of DSM-based diagnoses have been escalating. The DSM system, characterized by polythetic and dichotomous (categorical), criteria-based diagnoses based on patients' phenomenological symptom reports, can be traced back to the inception of modern psychiatric nosology in the 18th century, where the success of botanical taxonomists provided the impetus to physicians to categorize symptom clusters into formal diagnosis (Millon & Simonsen, 2010). Although the DSM system has served us well (Frances & Widiger, 2012), the reliance on polythetic and dichotomous (categorical) diagnoses at the level of the behavioral phenotype alone has impeded the use of recent advances in genomics, pathophysiology, psychopharmacology, and behavioral science to aid in the diagnosis and treatment of psychiatric disorders (Insel, 2014; Insel et al., 2010). For example, because syndromes like BPD are not single, homogeneous diseases, the current crisis in the development of new psychoactive agents has been attributed to inadequacies of the currently used psychiatric diagnostic system, which reifies disorders into single entities (Fibiger, 2012). These entities appear not to capture fundamental underlying mechanisms of dysfunction (Sanislow et al., 2010). We are therefore no further in understanding what causes mental disorder, nor its treatment, and "fiddling needlessly with the descriptive labels" (Frances & Widiger, 2012; p. 112) at the behavioral phenotypic level is unlikely to lead to any significant breakthroughs in research or treatment.

To improve the usefulness of our diagnostic system, the NIMH articulated in its Strategic Plan of 2011 to depart from the categorical classification system of mental disorders and to "develop new ways of classifying mental disorders based on dimensions of observable and neurobiological measures." RDoC is guided by several principles. First, in contrast to the categorical DSM system, RDoC is conceived as a *dimensional* system spanning the range from normal to abnormal. RDoC remains agnostic about dimensions versus categories (Miller & Rockstroh, 2013; Sanislow et al., 2010), but encourages the study of psychopathology across a broad spectrum of psychiatric diagnoses to study versus traditional approaches focusing on individuals with circumscribed symptom profiles (Insel et al., 2010). Second, RDoC seeks to "carve nature at its joints" by generating classifications stemming from basic behavioral neuroscience. Whereas psychiatry typically starts with a DSM diagnosis and then seeks *neurobiological underpinnings*, RDoC begins with current knowledge of behavior–brain relations across multiple levels of analyses (genes, molecules, cells, neural circuits, physiology, behaviors, and self-report) and links them to clinical phenomena (Insel et al., 2010). In this regard, the potential of RDoC therefore lies in reclassifying mental disorders from the bottom up, thereby realigning biology with behavior in a multidimensional space. Third, RDoC emphasizes the principle that mental disorders are to a large degree disorders of *development* (Sanislow et al., 2010). Therefore, by understanding the developmental aspects of mental disorders and the process of neurodevelopment itself, disorder may be redefined.

In parallel to the RDoC initiative, the *DSM–5* workgroups were instructed to produce a "paradigm shift" in psychiatric diagnosis (Frances & Widiger, 2012) with one of the main features being a shift toward a *dimensional* approach. One of the first workgroups to convene a conference was the one focusing on dimensional approaches to personality disorders (PDs) – long considered prime candidates for a dimensional or quantitative approach to assess-

ment and diagnosis (Regier, 2007). After a heated and protracted debate on whether PDs should be defined as categorical entities or combinations of extreme personality dimensions, the *DSM–5* now contains two parallel classification systems for PD. In Section II, the 10 *DSM–IV* categorical diagnoses with polythetic criteria were retained, but in Section III (Emerging Measures and Models) a dimensional trait-based approach is described with only 6 of the original 10 PDs retained. In addition to the shift toward dimensions, the *DSM–5* Preface (APA, 2013; p. xIii) heralds as two of its nine major enhancements the representation of *developmental* issues related to diagnosis and the integration of scientific findings from the latest research in *genetics* and *neuroimaging*.

Thus, despite fundamental differences in philosophical, sociopolitical, and scientific approaches to psychiatric nosology, RDoC and DSM developments converge on at least three fundamental changes in psychiatric nosology: Dimensions, Biology, and Development. Consistent with the earlier discussion on paradigm shifts, this convergence is facilitated by the lifting of technological constraints to accelerate research on these foci. Specifically, the advances in neuroimaging techniques, molecular genetics, and contemporary quantitative methodology that exemplify the trends in BPD research discussed earlier can be considered part of the "technoscience" or "phenomenotechniques" of this potential paradigm shift. In other words, although the dimensionality of mental disorders is a longstanding issue (e.g., Clark, 2007; Frances, 1993; Widiger & Clark, 2000), and the biological basis and developmental origins of psychiatric disorder have long been acknowledged, a paradigm shift in these directions was previously slowed down by the epistemic constellation of our time—not only in terms of technological constraints but also in terms of the lack of serious resistance to current practices. Previously, the kind of technology necessary to peer into the living brain simply was not available, and technology and knowledge to effectively analyze genetic data were scarce. Similarly, although mathematical models were developed to conceive of hierarchical models of psychopathology, the software was not readily available to researchers to evaluate such models. The shifts in the development of these "phenomenotechniques" have given researchers the confidence to more aggressively resist the current DSM system to ultimately accelerate change in the *practice of science* (see Figure 1)—a necessary precondition for a paradigm shift. Whether a true sea-change is afoot we cannot know, but the almost magical convergence of Dimensions, Biology, and Development is challenging the traditional way of viewing BPD. At the center of this convergence does not lie a discreet adult diagnosis caused by a refrigerator mother, but a developmental disorder of dimensionally defined traits with permeable boundaries that ebbs and flows as a result of complex nature–nurture interactions over time—hopefully, the promise of something rich and strange.

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