Personality Traits and the Classification of Mental Disorders: Toward a More Complete Integration in DSM–5 and an Empirical Model of Psychopathology

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Personality trait dimensions are related to a wide variety of important life outcomes, such as mortality, physical and mental health, and interpersonal relationships. Nevertheless, the diagnostic system with arguably the most influence in mental health settings (Diagnostic and Statistical Manual of Mental Disorders, 4th ed. [DSM–IV]) formally includes personality primarily in the form of 10 putatively categorical personality disorders. We advocate a more complete and extensive integration of personality in future DSMs, via the explicit inclusion of an empirically based, dimensional personality trait model. To justify this position, we provide a broad review of the ways in which personality traits have proven useful in the description and conceptualization of personality disorders and other mental disorders, as well as in the prediction of key clinical phenomena. We also discuss the importance of constructing a comprehensive quantitative model of psychopathology based on data, an endeavor that is motivated and informed by the close conceptual and empirical parallels between personality and psychopathology.

Keywords: nosology, dimensions, categories, personality disorders, psychometric modeling

There is no question that personality is closely connected with many major mental disorders and other key clinical phenomena (e.g., Hettema, Neale, Myers, Prescott, & Kendler, 2006; Krueger & Tackett, 2006; South, Eaton, & Krueger, in press). The current classification system with arguably the most influence in clinical settings, the Diagnostic and Statistical Manual of Mental Disorders (4th ed.; DSM–IV; American Psychiatric Association, 1994), incorporates personality into diagnostic conceptualization in several ways. First, DSM–IV includes diagnostic criteria for 10 putatively categorical forms of personality pathology labeled “personality disorders” (PDs). In addition to being conceptualized as qualitatively different from most other mental disorders (e.g., with regard to course), the PDs were placed on a separate axis (Axis II) to ensure that clinicians would consider their potential presence, even when the symptoms of another major mental disorder were prominent in the clinical picture. Second, the DSM–IV allows maladaptive personality traits that do not meet the level of a PD to be included in diagnostic conceptualizations by allowing the clinician to record such traits on Axis II (although DSM–IV does not provide a specific model for conceptualizing these traits, beyond their appearance as features of the 10 PDs). Finally, DSM–IV allows for personality
change to be considered pathological when it is due to a general medical condition.

Through Axis II, *DSM–IV* provides some infrastructure for connecting personality and psychopathology. However, the fifth edition of the manual (*DSM–5*) can—and should—incorporate personality to a considerably greater degree. In particular, the extensive research literature on dimensional personality traits—on their nature, structure, and impact on clinical outcomes—should influence the scope and form of *DSM–5*. More extensive integration of personality traits into the *DSM* would yield myriad benefits to conceptualization, diagnosis, and treatment of mental disorder. In the current review, we justify and elaborate this position by reviewing the relevance of personality traits to the conceptualization of mental disorders and their classification, as well as the utility of personality traits in predicting and understanding other clinical phenomena (e.g., treatment-related variables). We also argue for the importance of constructing a comprehensive, empirically based quantitative model of psychopathology. Quantitative models of personality trait variation have contributed unequivocal conceptual clarity in personality research, and would have similar utility if applied to psychopathology in a comprehensive manner that reflects the close links between personality trait variation and risk for psychopathology.

**Personality Traits and the Conceptualization of Mental Disorders**

Our first goal is to highlight the relevance of a personality trait model to the conceptualization of mental disorders. We first examine *DSM–IV* PDs and then turn to consider other mental disorders.

**Personality Traits and *DSM–IV* Personality Disorders**

Personality disorder categories versus dimensions of pathological personality. The most obvious way in which personality traits connect with *DSM–IV* mental disorders is in their role as fundamental features of the PDs (for a recent introduction to modern theoretical underpinnings of personality trait psychology and the ways in which traits encompass eliciting circumstances, see Roberts & Mroczek, 2008; Tellegen, 1991). Although the relevance of personality traits to PDs is essentially axiomatic, the specific PD classification system in *DSM–IV* has been criticized on numerous grounds, as reviewed more extensively elsewhere (e.g., Clark, 2007; Krueger, Skodol, Livesley, Shrout, & Huang, 2007; Trull & Durrett, 2005). First, the PDs are currently defined as 10 categorical entities, but there is no evidence we are aware of that the latent structure of personality pathology is best characterized in terms of 10 dichotomous variables (see Widiger, Simonsen, Krueger, Livesley, & Verheul, 2005). Second, PDs are conceptualized using arbitrary cut-points (e.g., *at least five* of nine diagnostic criteria present indicate the presence of borderline PD, although there is little to no empirical justification for that particular threshold; Kampphuis & Noordhof, 2009). Third, PDs have high levels of diagnostic overlap and comorbidity (e.g., Dolan-Sewell, Krueger, & Shea, 2001), a situation that greatly complicates both clinical case conceptualization and research design. For example, if the modal patient has “more than one PD,” which should be the focus of intervention and why?

The limitations of the categorical *DSM–IV* system for classifying PDs have led many researchers to call for PDs to be reconceptualized in terms of personality dimensions (e.g., Clark, 2007; Frances, 1993; Livesley, Jang, & Vernon, 1998; Trull & Durrett, 2005; Widiger, Livesley, & Clark, 2009; Widiger & Samuel, 2005). Widiger and Simonsen (2005) note that the “dimensionalization” of PDs could be accomplished in several ways, including simply retaining the current PD diagnostic constructs but treating them as ordinal dimensions (number of diagnostic criteria met; Oldham & Skodol, 2000). Although this approach has the advantage of retaining familiar diagnostic concepts that have some traction in the literature (e.g., borderline PD), it has the disadvantage of simultaneously retaining diagnoses that have recruited little to no research or clinical attention (e.g., paranoid PD; Blashfield & Intoccia, 2000).

As a result, the means of reconceptualizing PDs that have received the most attention involve replacing the existing PDs with an empirically derived model of dimensional personality
traits. Indeed, this was the focus of an official American Psychiatric Association–National Institute of Mental Health meeting on PDs, organized in preparation for DSM–5 (Widiger et al., 2005). As discussed extensively at that meeting, four to five broad groups of traits have been identified by numerous research groups as the leading candidates for organizing the first tier of the domain-level structure of personality pathology (De Clercq, De Fruyt, Van Leeuwen, & Mervielde, 2006; Harkness & McNulty, 1994; Harkness, McNulty, & Ben-Porath, 1995; Livesley et al., 1998; Markon, Krueger, & Watson, 2005; Nestadt et al., 2006; Tackett, Silberschmidt, Krueger, & Sponheim, 2008; Watson, Clark, & Chmielewski, 2008; Widiger & Simonsen, 2005). Various investigators adopt various labels for these domains, but here we will label the domains as (I) antagonism, (II) disinhibition, (III) negative emotionality, (IV) introversion, and (V) peculiarity. As a shorthand abbreviation, we will refer to this system as the pathological five model (PFM).

The PFM and the five-factor model (FFM) of personality. The PFM designation is apt because these domains can be generally understood as maladaptive and extreme variants of the domains of the FFM of personality traits (agreeableness, conscientiousness, neuroticism, extraversion, and openness to experience; Costa & Widiger, 2001). With regard to the first four traits, antagonism is the opposite of agreeableness, disinhibition is the opposite of conscientiousness, negative emotionality encompasses extreme neuroticism, and introversion is the opposite of extraversion.

We should note that, in distinguishing the PFM from the FFM, our intent is not to diminish the importance of the FFM. The FFM is the most influential structural model of personality traits, and with good reason: It is supported by an extensive empirical literature (McCrae & Costa, 2008), and it forms the base of the higher order structure of personality traits (Markon et al., 2005). Indeed, the similarities between the FFM and PFM far outweigh the differences. Nevertheless, our intent in articulating the PFM is to emphasize how the integration of normal and abnormal personality trait structure is an ongoing enterprise. Specifically, although the PFM domains generally map well onto FFM domains, the domain-level resemblance between the two models is not isomorphic in every respect. This is likely because the FFM was not created specifically to organize pathological personality dimensions (see, e.g., Nestadt et al., 2008). This notable yet imperfect mapping between the PFM and FFM domains points to important topics for continued research.¹

Peculiarity provides the clearest example of the partial isomorphism of the PFM and the FFM and the value of continued research in this area. Peculiarity is arguably the least well-understood domain in the PFM, and its relations to the FFM are complex. A recent study by Piedmont, Sherman, Sherman, Dy-Liacco, and Williams (2009) nicely illustrates this point. Piedmont et al. described the construction of the Experiential Permeability Inventory (EPI), a questionnaire designed to assess maladaptive

¹ An additional difference between the PFM and the FFM is that the FFM domains are typically conceptualized as bipolar (e.g., extraversion–introversion is conceptualized as a single bipolar trait domain), whereas our intention in naming the PFM domains as we have is to accentuate maladaptive poles of these domains and to acknowledge that the PFM domains may not be perfectly bipolar. By contrast, Widiger and Mullins-Sweatt (2009, e.g., Figure 2) conceptualize both the positive and negative poles of all five FFM domains as maladaptive (e.g., reckless sensation seeking is conceptualized as a maladaptive aspect of extreme extraversion). However, a number of the trait placements that stem from conceptualizing the structure of maladaptive personality as necessarily bipolar and isomorphic with the FFM may be factorially questionable. For example, reckless sensation seeking tends to align more closely with the antagonism and disinhibition (externalizing) domains than with the extraversion domain (Markon et al., 2005; Krueger, Markon, et al., 2007).

Another potentially important example is provided by contemplating the location of compulsive personality traits in factor space. Widiger and Mullins-Sweatt (2009) place the traits of perfectionism and irresponsibility at the opposite poles of the conscientiousness domain. Yet, when these types of facets are factor analyzed along with other maladaptive facets, compulsive qualities such as perfectionism tend to form their own broad domain, with irresponsibility and similar traits falling in a separate domain encompassing diverse antagonistic facets (Livesley et al., 1998; Nestadt et al., 2008). The general point is that the empirical structure of maladaptive personality facets is not necessarily isomorphic with the structure of more normal-range personality domains as delineated in instruments designed to assess FFM facets in diverse settings (e.g., the NEO-PI–R; Costa & McCrae, 1992). Nor is the structure of maladaptive personality facets necessarily bipolar. As we describe more extensively in this article using the peculiarity domain as an example, there is still more to learn about the fine-grained structure of maladaptive personality facets and how this structure intersects with the structure of normal-range personality facets (cf. Clark, 2007).
and extreme qualities theorized to lie at both the high and low poles of FFM openness. The EPI scales correlated with openness as measured by the NEO Personality Inventory—Revised (NEO-PI–R; Costa & McCrae, 1992) in expected and meaningful ways, but the EPI was not redundant with openness (e.g., the Odd and Eccentric subscale of the EPI correlated .36 with the NEO-PI–R openness domain score).

One could extend the pioneering research of Piedmont and colleagues (2009) by working toward a comprehensive structural account of elements of both openness and peculiarity. This kind of endeavor could benefit from considering the relative locations of specific traits along broader domain dimensions in addition to their correlational structure. For example, are openness and peculiarity part of a somewhat diffuse and hierarchically organized trait domain? This is a reasonable possibility inasmuch as the “core features” of openness have been somewhat challenging to identify unequivocally (John, Naumann, & Soto, 2008). Or are they better understood as falling at different locations or degrees of severity along one major axis of variation? Or are these constructs sufficiently dissimilar that openness and peculiarity should not be regarded as delineating the same domain (see Watson et al., 2008)?

**Facets of pathological personality.** As the example of openness and peculiarity illustrates, more narrow and specific (facet-level) dimensions can also be identified, and these constructs are subsumed within each of the broad domains. Facets provide an even more detailed account of a patient’s personality profile. The challenge with facets is to achieve optimal fidelity of personality description by including the “right” number of facets. A system with too many facets is potentially unwieldy (few clinicians may be enthusiastic about learning or working with, say, 40 specific personality dimensions²), yet a system with too few facets lacks the detail needed for a psychologically rich conceptualization of a specific patient (cf. Widiger & Mullins-Sweatt, 2009). For example, it is arguably more useful to understand the specific manifestations of negative emotional tendencies in a specific patient and the circumstances that tend to elicit those reactions (the kind of information conveyed by a detailed facet-level personality assessment) versus simply knowing that the patient has “elevated negative emotionality” (cf. First & Westen, 2007; Westen, Gabbard, & Blagov, 2006).

Although personality trait models line up well at the domain level (cf. Widiger & Simonson, 2005), they are less similar at the facet level. As a result, there are many potential facets and lists of facets to choose among when contemplating the inclusion of a specific set of facets in a diagnostic manual. To complicate matters somewhat further, the justification for including a specific set of facets would logically include both clinical judgment and psychometric considerations. This is because many facets in personality trait models were generated for a broad range of applications, as opposed to having been generated specifically because of their relevance in clinical settings. Clinical relevance is partly a matter of judgment (although data can of course assist in making such judgments, e.g., data on the relative predictive validity of specific facets with regard to key clinical outcome variables). The general point is that delineation of facet-level structure and articulation of the optimal facets for clinical application remain an important area for research, scholarship, and methodological innovation (Clark, 2007). For example, factor analytic methods are well suited to modeling the “big picture” of personality variation (i.e., identifying trait domains), but are not necessarily optimal for delineating fine-grained details within these broader domains. This latter task benefits from taking other approaches, such as examining the detailed hierarchical structure that can be derived from cluster analyses of correlations among specific elements of personality (Krueger, Markon, Patrick, Benning, & Kramer, 2007).

**Clinical applicability of a dimensional model of pathological personality traits, compared with a system consisting of multiple dichotomous PDs.** Although there are overwhelming scientific reasons for replacing the *DSM–IV* PD system with a dimensional trait

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² This having been said, the *DSM–IV* is considerably more unwieldy in terms of containing an extensive number of diagnostic constructs and modifiers. The issue might pertain less to the sheer number of facets in a dimensional personality trait system than to clinical familiarity with the *DSM–III* constructs that have carried through, mostly unchanged in most fundamental respects, into *DSM–IV*. 
scheme, it would be helpful if a change of this magnitude were accompanied by considerations of clinical applicability. Fortunately, the clinical benefits of this change accrue directly from the conceptual advantages of dimensional over categorical models of human variation. Consider, for example, a multidimensional personality trait model in the conceptualization of a specific patient. Formal assessment of a patient on multiple dimensions in a system such as the FFM or PFM provides a comprehensive understanding of that patient’s personality. By comparison, binary diagnoses of one or more of the DSM–IV PDs signal only that the patient passed one or more arbitrary thresholds, leaving additional information about the patient’s personality out of the clinical picture.

Consider the example of DSM–IV borderline PD, which requires at least five of nine diagnostic criteria to be present for a diagnosis to be made. The “five or greater” threshold results in 256 different combinations of criteria that justify a diagnosis of borderline PD (Johansen, Karterud, Pedersen, Gude, & Falkum, 2004; Skodol et al., 2002). As a result of this diagnostic heterogeneity, a categorical diagnosis of borderline PD could indicate a great diversity of specific personality features in specific patients. Indeed, two patients diagnosed with borderline PD could theoretically share only one diagnostic feature, and otherwise have entirely non-overlapping personality features.

Conceptual conundrums of this nature are obviated by the use of a multidimensional model of pathological personality dimensions. Rather than describing a patient in terms of one or more dichotomous variables (e.g., above or below the threshold for a borderline PD diagnosis), the patient is assessed on multiple dimensions. Graphical displays of patients’ trait profiles provide an intuitive and appealing way to communicate information about patients’ personality features, as well as their similarities and dissimilarities (as discussed at greater length in Costa & Widiger, 2001).

To illustrate the within-category heterogeneity problem, as well as the additional information contained in a multidimensional trait model, Figure 1 provides the NEO-PI–R (Costa & McCrae, 1992) domain-level trait profiles of all six participants in the St. Louis Personality and Aging Network study (SPAN; Thomas Öltmanns, PD) who have participated to date and who met criteria for a borderline PD diagnosis. SPAN is an National Institute of Health-funded community-based study of individuals ages 55 to 64; the six participants we evaluated met DSM–IV borderline PD criteria after having been interviewed on the Structured Interview for DSM–IV Personality (Pfohl, Blum, & Zimmerman, 1997). The SPAN study commenced data collection only recently, so these data should not be regarded as a complete account of findings or conclusions. However, the data do represent the trait personality profiles of six specific persons who were carefully interviewed and diagnosed and are illustrative in that regard. Specifically, although these six persons met cri-

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3 Our use of the term clinical applicability is purposeful and should be distinguished from the term clinical utility, which has been a recent topic in discussions about considerations relevant in revising the DSM (e.g., First, 2005). By clinical applicability, we mean that the translation from scientifically valid concepts to ways of using those concepts in the clinic has been made explicit. By contrast, recent “clinical utility” studies have tended to focus on assessing the opinions of practitioners regarding their preferences when asked to choose among a series of options regarding various models of psychopathology (e.g., Lowe & Widiger, 2009). We do not believe that this kind of “practitioner survey” research should carry much weight in constructing a nosology. Consider, for example, a hypothetical survey of medieval physicians regarding the clinical utility of trephination as an intervention for mental illness or a similar survey regarding their ideas about the etiology of mental illness. Few contemporary investigators would argue that the results of this kind of survey would have any meaningful implications for pursuing a model of psychopathology that could ultimately lead to both accurate understanding and effective amelioration of symptoms.

In short, scientific hypotheses about psychopathology obviously originate in clinical experiences, but they must then be formally tested, and the understanding that results from this scientific process must then feed back to clinical application. Nevertheless, it is also critical to emphasize that a novel system with greater evidence of validity (e.g., a dimensional model of personality pathology vs. the 10-category DSM–IV PD system) cannot simply be dumped in clinicians’ laps without any attempt to explain the scientific basis for the change or how to use the new system effectively in the clinic. Should DSM–5 adopt novel dimensional conceptualizations (something that has been proposed by the DSM–5 task force chair and cochair as a cardinal goal of the revision effort; Regier, Narrow, Kuhl, & Kupfer, 2009; see also Helzer et al., 2008), a significant, well-organized, and compelling effort at educating front-line practitioners and researchers must be pursued. This is an essential step in ensuring that the scientifically critical move to more dimensional conceptualizations of psychopathology resonates in both clinical and research settings, and thereby ultimately serves to improve the lives of patients.
teria for a specific *DSM–IV* PD, they have different trait profiles, and the information in those profiles is of straightforward clinical relevance.

Consider, for example, Person 3. This person has an elevated level of neuroticism, as is typical of persons meeting borderline PD criteria (i.e., although there is variation in neuroticism in these six persons; even in this very small sample, the mean neuroticism value is more than 1 standard deviation above the mean for the manual-based normative sample). Indeed, neuroticism could be described as the essential personality feature of borderline PD (e.g., Livesley, 2005, 2008). However, this elevated neuroticism is in the context of high extraversion and openness, average agreeableness, and very low conscientiousness. The clinical hypotheses that derive from this personality profile suggest that this is a person who is capable of getting along well enough with others and outgoing enough to form a therapeutic alliance. The high openness score might also suggest that this person would be open to a therapeutic approach where depth and underlying motives for behavior are explored (vs. a more concrete approach). Perhaps most important, the very low conscientiousness score suggests that this person may not adhere closely to a specific treatment regimen (e.g., this person may find it difficult to complete therapeutic homework assignments). The general point is that personality trait profiles convey useful and richer clinical information beyond the information in a dichotomous *DSM* diagnosis; for compelling and more extensive discussions of the clinical applicability of personality traits, see Harkness and Lilienfeld (1997) and Harkness (2007).

In sum, the switch to a system that focuses on multiple personality *trait dimensions* has a major clinical benefit inherent in its nature: The form of personality is always specified, via the patient’s standing on the traits constituting the system (and in the PFM, one could characterize this specification as the form of personality *pathology*). Along these lines, another notable problem with the 10-category *DSM–IV* PD
system is the high prevalence of the PD—not otherwise specified (PD-NOS) diagnosis (Verheul & Widiger, 2004). This diagnosis conveys little clinically useful information (e.g., we are not aware of programs of research focused on identifying the optimal treatment for PD-NOS). By contrast, we know a great deal more about the clinically relevant correlates of major domains of human personality variation. We review some of this research in the current article as it pertains to clinical psychology and psychiatry. But we first turn to consider two additional issues inherent in moving from the DSM–IV 10-category PD system to a dimensional personality trait system for DSM–5, namely (a) how to render a PD diagnosis, given a dimensional system for personality trait description; and (b) what to do about the 10 PDs of DSM–IV if a dimensional personality trait system were to be implemented in DSM–5.

Reconciling the clinical need to render PD diagnoses with a dimensional personality trait model. In the DSM–IV PD system, the symptoms of PDs and PD diagnoses are one in the same construct. That is, crossing the symptom threshold for a specific PD justifies assigning that PD diagnosis to a patient. If the PD categories of DSM–IV were replaced with a dimensional trait system in DSM–5, how would the information in that system translate into a diagnosis?

A simple answer to this question would involve setting cutoffs on the dimensions that constitute the DSM–5 trait model. The problem with this approach is that it equates trait extremity with disorder per se. Many would argue that the concept of disorder contains elements that are distinct from trait extremity, and conceptual problems with equating trait extremity and disorder are easily envisioned. For example, is being highly introverted a “disorder” per se? Probably not, except to the extent that the person also shows hallmark features of disorder that are connected to the introversion (e.g., inability to pursue meaningful employment because of impairing shyness in spite of unequivocal evidence that the person has the skills needed for meaningful employment).

The definitions of mental disorder and PD are challenging topics that go beyond the scope of this article, but they are nevertheless important considerations for DSM–5. These topics are difficult because they cannot be adjudicated with complete objectivity. The concept of disorder involves value judgments and is therefore inherently a matter of societal and professional opinion.

Moreover, recent papers on the concept of personality disorder (as potentially distinct from the broader concept of mental disorder) tend to agree that personality disorder is a construct distinguishable from personality traits (Parker et al., 2004). For example, Widiger and Mullins-Sweatt (2009) argued that the judgment of PD corresponds well with DSM–IV Axis V, the global assessment of functioning (GAF). They suggested that cutoffs could be placed on the GAF scale to establish clinically significant problems in psychological, social, or occupational functioning, thereby justifying the clinical judgment of disorder, with the personality features of the disorder described by traits. Although their approach is similar in many respects, Widiger and Mullins-Sweatt contrasted their views with those described by Krueger, Skodol, and colleagues (2007), who emphasized three qualities to define PD: (a) interpersonal functioning, (b) occupational functioning, and (c) having coherent and adaptive working models of the self and others. As Widiger and Mullins-Sweatt noted, the major difference between their GAF conception and that of the Krueger, Skodol, et al. conception lies with the absence of subjective distress in the Krueger, Skodol, et al. tripartite definition, this element of the GAF having been replaced by self–other representation disturbance. Somewhat similar to Krueger, Skodol, et al., Livesley (2007) argued that the quintessence of PD is “adaptive failure” of the personality system. A coherently functioning personality system allows a person to adaptively construct working models of self and others, to use those models to navigate close relationships, and to pursue cooperative and mutually beneficial relationships at a broader group level. In PD, the functioning of this system is disrupted.

Some recent empirical work on the assessment of personality pathology, as distinguishable from personality traits, may be helpful to consider in this context. Verheul and colleagues (2008) constructed the Severity Indices of Personality Problems (SIPP-118) as a self-report assessment of personality problems, as opposed to personality traits or independent clinical judgments of disorder. The SIPP-118 scales en-
compass higher order domains of self-control, identity integration, relational capacity, social concordance, and responsibility. The authors present evidence that, although these scales were correlated with the scales of Livesley’s Dimensional Assessment of Personality Pathology (DAPP; a self-report assessment of pathological personality traits per se; Livesley, 2007), the two instruments were not redundant, with median SIPP–DAPP correlations in the range of .23 to .37. This suggests that personality problems can be distinguished from pathological personality traits per se, at least in the self-report domain.

The SIPP-118, the GAF, and operationalizations of the clinical constructs delineated by Krueger, Skodol, and colleagues (2007) and Livesley (2007) provide good places to start in working to use data to inform the conceptualization of PD in DSM–5. For example, one could compare these conceptualizations as predictors of unequivocal social costs, such as days missed of work owing to mental disorder symptoms. The strength and nature of the relationship between personality pathology indices and social costs are important parameters that could be used to inform the definition of PD in DSM–5. Indeed, it is theoretically possible for level of personality pathology to have a nonlinear relationship with level of social cost, such that the likelihood of significant social costs accelerates at some point along a dimension of personality pathology. If this were the form of the relation between the two constructs, the “cusp” where social costs accelerate could be viewed as a reasonable location for the cutpoint delimiting disorder. However, a true nonlinear or other complex relationship of this sort may be unlikely, given the robust predictive utility of simple linear models (Dawes, 1979). In any event, research comparing conceptions of PD (or mental disorder more generally) in terms of their links with “objective” social costs could usefully inform thinking about the disorder construct, as well as thinking about the placement of cutpoints in DSM–5.

The vexing problem of popular diagnostic constructs that do not map neatly onto constructs from dimensional personality trait models. A critical issue in contemplating a change of the magnitude we are discussing here (replacing the 10 DSM–IV PDs with a dimensional trait model—something Widiger & Trull, 2007, equated with the shifting of continental plates) concerns the fate of the 10 DSM–IV PDs. With apologies to Mr. Gore, the “inconvenient truth” is that the 10 DSM–IV PD constructs do not map perfectly onto the broad domains of dimensional personality trait models. Various stances on this issue can be articulated.

A hard-line stance on this issue would focus on the idea that the continued use of constructs that do not accurately represent the empirical structure of pathological personality is probably not helping anyone in the long run. Clinical psychology and psychiatry continue to move toward “evidence-based practice,” and in this zeitgeist, the use of diagnostic constructs that are incompatible with what is known about the empirical structure of pathological personality is obviously problematic.

A somewhat softer stance might note that—although the resemblance is not perfect—there are notable points of connection between the PFM domains and DSM–IV PD constructs, with traction in both clinical and research settings. Relative to the other PDs, there seems to be greater interest and attention focused on borderline PD, antisocial PD, and schizotypal PD (Blashfield & Incotta, 2000). Borderline PD has been conceptualized by some theorists as being essentially the extreme end of negative emotionality or neuroticism (e.g., Livesley, 2005, 2008). Similarly, antagonism (or “meanness”) is a core feature of psychopathy (which might be thought of as a more “personality-based syndrome” than the somewhat more societally defined antisocial PD; Patrick, Fowles, & Krueger, 2009). Finally, peculiarity is the quintessence of schizotypal PD, as defined in DSM–IV. As a result of these three points of overlap, a potential strategy would involve retaining borderline, antisocial (perhaps renamed psychopathic), and schizotypal PDs as DSM–5 diagnoses, but redefining them in terms of the facets that define the broad trait domains of negative emotionality, antagonism, and peculiarity, respectively.

This idea has some appeal because (a) it results in retention of the three DSM–IV PDs in which there is substantial interest, and (b) it grounds those constructs in the domains of empirically based dimensional trait models. Nevertheless, other research speaks to some potential empirical disjunctions that require further consideration. An example that has attracted
recent empirical interest is the emotion dysregulation element of DSM–IV borderline PD. A number of recent studies suggest that emotion dysregulation is not simply the equivalent of neuroticism or negative emotionality (Glenn & Klonsky, 2009; Kamen, Pryor, Gaughan, & Miller, in press; J. D. Miller & Pilkonis, 2006; Trull et al., 2008). For example, Trull et al. (2008) used ecological momentary assessment (EMA; sampling peoples experiences as they unfold in their daily lives) to illustrate that persons with borderline PD showed more affective instability in their natural environments when compared with persons with current depressive disorder (although both diagnoses are associated with trait negative emotionality or neuroticism; Bagby, Costa, Widiger, Ryder, & Marshall, 2005; Krueger, Caspi, Moffitt, Silva, & McGee, 1996). Similarly, DSM–IV borderline PD also includes “transient, stress-related dissociation” as a symptom, and dissociation falls more in the peculiarity domain than in the negative emotionality domain (Koffel & Watson, 2009).

Recognizing the imperfect map, however, might give rise to interesting ideas for research. For example, the “transient and stress-related” part of the aforementioned dissociation criterion might be critical; perhaps this variety of dissociation (e.g., traumatic intrusions) emerges at the extremely high end of negative emotionality, whereas the general propensity to dissociate (with or without accompanying negative emotional experiences) is located in the peculiarity domain (Koffel & Watson, 2009). In addition, in other recent EMA research (D. J. Miller, Vachon, & Lynam, 2009), neuroticism (trait negative emotionality) predicted the typical level of negative affect people experienced, but antagonism (low agreeableness) was linked to unstable negative affect (i.e., the extent of extreme and frequent fluctuations in negative affect). This EMA work dovetails in an interesting way with structural work on borderline PD. Borderline PD shows relatively polymorphous comorbidity (i.e., people who meet criteria for borderline PD also tend to meet criteria for numerous other diagnoses; James & Taylor, 2008). Part of the reason for this polymorphous comorbidity might be that borderline PD, as currently defined, taps into multiple personality domains (e.g., negative emotionality, disinhibition, antagonism, peculiarity; Skodol et al., 2002; Tragesser & Robinson, 2009). Each of these domains predicts somewhat different forms of psychopathology (e.g., peculiarity confers risk for psychotic experiences; negative emotionality confers risk for internalizing disorders; Krueger et al., 1996; Zinser, Chapman, & Chapman, 1997). When these domains combine in a specific person (i.e., the prototypical borderline PD case), the combination gives rise to a diversity of problems.

In sum, the general issue and questions in this area can be framed as follows: Clinically salient PD constructs are somewhat, but not perfectly, aligned with the major domains of empirically based personality trait models. Are these disjunctions structural (e.g., borderline PD as currently defined is an interstitial, or “in between,” construct combining elements of numerous pathological personality domains)? Are the disjunctions a matter of range (e.g., extremely high levels of negative emotionality lead to dissociation from that affect or to specific cognitive disturbances accompanied by negative affect, such as traumatic intrusions)? Are they a matter of measurement domain (e.g., emotional dysregulation is harder to measure with questionnaire items, relative to trait negative emotionality, and emerges more clearly in the ecology of daily life)? Are they due to personality disorder having features (e.g., deficits in self-concept and mental representations of other people) that do not overlap completely with personality traits (Krueger, Skodol, et al., 2007; Verheul et al., 2008)?

It seems unlikely that these questions would be fully answered by the time the DSM–5 is slated to be published (2013). As a result, an even softer stance might involve mapping some DSM–IV PDs—perhaps even all 10 DSM–IV PDs—onto the trait model adopted for the DSM–5 and providing these maps in the text of the DSM–5 as a means of cross-walking back to DSM–IV diagnoses. An extensive literature shows how the 10 DSM–IV PDs can be represented in terms of dimensional personality trait models, such as the FFM, as combinations of traits (J. D. Miller, Bagby, Pilkonis, Reynolds, & Lynam, 2005; J. D. Miller et al., 2008; Samuel & Widiger, 2008). The obvious disadvantage of this approach is that it undermines the point of moving to an empirically based dimensional model of pathological personality in...
DSM–5 and reifies the structurally invalid DSM–IV PD system.

Stepping back from the scientific details and viewing this area more from the perspective of the sociology of science, these are very challenging issues because (a) they intersect with the value placed on different kinds of evidence, and (b) they have serious implications for people’s lives (e.g., the careers of professionals and the ways in which patients connect with clinical services). For example, what matters more in thinking about classification: the empirical factor structure of personality traits, or the clinical salience and documented social costs of borderline PD as currently defined? What are we to make of the extensive literature on borderline PD if it ceases to be officially recognized by DSM–5, or of programs of research focused on elucidating and ameliorating borderline PD? What becomes of the sociostructural entities that have arisen around DSM–IV PD constructs (e.g., patient advocacy groups and research foundations focused on borderline PD)?

Practically speaking, our view is that DSM–5 must include some means of describing at least some DSM–IV PDs in personality trait terms, particularly those that have traction in current clinical thinking and research (cf. J. D. Miller et al., 2008). With this in mind, we favor the last option described above: focusing effort on articulating a trait model as the foundation for conceptualizing personality and personality disorders in DSM–5, but also articulating all 10 DSM–IV PDs in trait terms, probably in an appendix that describes these PDs as “legacy constructs from DSM–IV.” These PDs can be well captured as combinations of specific traits, along with requiring the patient to pass the threshold for a PD diagnosis (see Krueger, Skodol, et al., 2007, for an example of how this approach might work focused on borderline PD; see also Widiger & Mullins-Sweatt, 2009, for a similar proposal). A reasonable system for DSM–5 would consist of (a) an empirically based personality trait model; (b) a general definition of the PD diagnosis, rendered as a threshold on an ordinal dimension of overall deficits in conceptualization of self and others and accompanying problems pursuing meaningful occupational and interpersonal goals; and (c) articulation of person-centered PD prototypes from the DSM–IV as combinations of traits, accompanied by passing the threshold for the PD diagnosis (Krueger, Skodol, et al., 2007).

On the Inevitable Role of Structural Validity in Constructing an Empirically Based and Useful System for Classifying Mental Disorders

Although improving PD conceptualization and classification is an important and timely topic, the questions and issues the foregoing discussion raises have broader implications for the classification of psychopathology. The putative Axis I–Axis II distinction proposed by DSM–IV has little empirical basis (Clark, 2005; Krueger, 2005), and as a result, personality trait research has broad implications for the classification of all mental disorders.

Since the time of the neo-Kraepelinian revolution in psychiatry in the 1970s, the emphasis in classification has been on reliability (Blashfield, 1984). The narrative paragraphs of DSM–II gave way to the explicit criterion lists of DSM–III, which led to increased reliability for a number of key mental disorders (Spitzer, Forman, & Nee, 1979). However, a reasonable argument can be made that this focus on reliability in mental disorder classification was at the expense of validity (Kendell & Jablensky, 2003).

The validity of a system for classifying psychopathology is a challenging and multifaceted topic (G. T. Smith & Combs, in press; Strauss & Smith, 2009). Nevertheless, there is one key aspect of validity that we see as both critical and tractable at this point in the evolution of thinking about the classification of mental disorders. Specifically, we believe that any diagnostic system that is a serious contender for being a useful framework for pursuing research on the etiology and treatment of psychopathology, and therefore research of potential clinical relevance, must start from a position of structural validity (Loevinger, 1957).

By structural validity, we specifically mean that a classification system for psychopathology (a) should be able to be explicated as a formal quantitative model of human variation and (b) should fit data suited to estimating the parameters of said formal quantitative model. Simply put, it is extremely difficult to make sense of (and, often, even to pursue) research on psychopathology given a structurally invalid system. In
addition, we emphasize quantification in our definition simply because models need to be stated in formal quantitative terms to see whether they actually work (i.e., to see whether they fit relevant data) and to be vulnerable to falsification (Popper, 1959). Without this step, classification science devolves into efforts that generate more heat than light, that is, arguments based on training, background, or a priori conceptual preferences versus genuine insights about the nature of psychopathology. For example, one could hypothesize that borderline PD is somehow different in kind from dimensional personality traits, and comprises a nonarbitrary class of persons (a “taxon”), based on clinical experience (albeit this hypothesis has been evaluated and rejected; Edens, Marcus, & Ruiz, 2008). But there is no way to evaluate this kind of hypothesis (“borderline PD is a category in nature”) if it cannot be stated in terms of its implications for what one would expect to observe in relevant data, and data always come in the form of numbers (Meehl, 2006). Moreover, it is helpful also to be able to state an alternative hypothesis in formal quantitative terms (e.g., “borderline PD as currently conceptualized is a configuration of extreme standing on a series of dimensions, e.g., pathological traits and overall deficits in self and other conceptualization, that vary continuously in nature”) because this facilitates direct comparison of competing models of psychopathology in relevant data (cf. Krueger, Markon, et al., 2005; Markon & Krueger, 2005).

The DSM–IV system of 10 putatively dichotomous PDs, plus a panoply of additional and putatively dichotomous mental disorders on Axis I, provides a particularly compelling example of the problems that emerge when trying to work with a system that suffers from problems related to structural validity. As an example, consider a current “hot topic”: molecular genetic research on mental disorders (Psychiatric GWAS Steering Committee, 2009). Given the rather amazing recent advances in genotyping technology, we could, for example, pursue a genomewide search for the single nucleotide polymorphisms (SNPs) that are associated with DSM–IV-defined borderline PD using a design in which cases and controls are compared on their SNP profiles across the genome (a genomewide association study, or GWAS, focused on borderline PD as the target phenotype). A key methodological consideration arises immediately in contemplating this study: Should our borderline PD cases be free of other mental disorders? Probably not, because the typical borderline PD case is a person with extensive additional psychopathology—both PDs per DSM–IV criteria as well as other DSM–IV-defined mental disorders (Skodol et al., 2002). So, perhaps we should simply work with borderline PD cases (regardless of any additional psychopathology they might have) and compare them with persons who have never met borderline PD criteria (e.g., in themselves or in their first-degree relatives). But then can we conclude that borderline PD is driving the findings of our research? This conclusion seems unwarranted because the rate of psychopathology, generally speaking, and across Axes I and II of the DSM–IV, will be significantly elevated in the borderline PD group. So, perhaps one option here might be to assess all the other psychopathology, and then include these variables as covariates in models predicting case status from SNPs. Unfortunately, this is also not a particularly compelling solution because it brings us back to our original dilemma. In the “covariate modeling approach,” we are creating (using statistical means rather than by initial screening) an artifactual “pure form” of borderline PD that we know does not exist in nature (cf. Meehl, 1971)—or at the very least an artifactual “pure form” that does not resemble the modal borderline PD patient in clinical settings.

As this discussion illustrates, thinking about structural validity leads to topics beyond the PDs and to a more fundamental consideration of the dilemmas of a diagnostic system such as DSM–IV, in which structurally awkward situations involving comorbidity, within-category heterogeneity, and NOS diagnoses are the norm (Krueger & Markon, 2006). Might a structurally valid dimensional personality trait model have implications for understanding psychopathology that go beyond its ability to resolve problems with the DSM–IV approach to classifying PDs?

### Beyond Personality Disorders and Toward a Structurally Valid Model of Psychopathology Grounded in Personality

Perhaps the most serious problem with contemporary psychodiagnosis is the lack of empirical
constraints on the form of psychodiagnostic systems. Our current scientific understanding of psychopathology is modest at best, in spite of extensive efforts aimed at identifying the underlying pathophysiology and etiology of psychopathology and significant expenditures of taxpayer dollars. Nevertheless, the hope remains that, given reliable markers of underlying pathophysiology and etiology (e.g., specific genetic polymorphisms or neurobehavioral indices) and knowledge of potent environmental elicitors, we could create a valid diagnostic system to drive appropriate intervention strategies.

The question is how to bootstrap a psychodiagnostic system with this kind of validity from our current understanding. As we began describing in the foregoing section, our view is that the only way we can accomplish this is through a first step aimed at using numerical taxonomy to articulate a structurally valid model of psychopathology. By numerical taxonomy, we are referring to the formal quantitative modeling that underlies the pursuit of structural validity. A discussion of quantitative modeling per se would go well beyond the scope of this article, but the basic idea is that data on manifest psychopathological signs and symptoms are quantified, and formal quantitative models are compared in terms of their fit to those data. As just one example, one could compare and contrast models positing that psychopathology is more continuous (or dimensional) versus more discrete (or categorical; see, e.g., Krueger, Markon, Patrick, & Iacono, 2005; Waller & Meehl, 1998). Indeed, a key finding from this kind of research is that psychopathology tends to be more continuous than discrete (Krueger, Watson, & Barlow, 2005).

Pursuing structural validity via numerical taxonomy involves conducting research designed to place nonarbitrary, empirically derived constraints on the form of a psychodiagnostic system. It is important to note that these constraints may be misleading at some other level of analysis if their conceptual strengths and limitations are not properly understood. For example, there is extensive structural evidence for the existence of a pervasive neuroticism or negative emotionality dimension in psychopathology data underlying essentially all forms of psychological maladjustment (Lahey, 2009). Is this dimension akin to fever in internal medicine—a highly general indicator of distress (cf. Ormel, Rosmalen, & Farmer, 2004)? And what should we conclude if this were indeed an apt analogy?

From the “fever analogy,” one could argue that neuroticism is clinically useless because “all patients in mental health settings have elevated neuroticism.” But few physicians would argue that fever is a useless construct, particularly if one thinks about fever on a nonarbitrary continuous scale, as opposed to thinking in terms of “high fever versus normal temperature.” Rather, a modern understanding is that distinguishable underlying etiologies can give rise to final common clinical signs and symptoms (e.g., high fever) that are important to assess, and a high fever is cause for greater alarm than a mild fever. Knowing someone suddenly developed a high fever initiates a process of differential diagnosis in search of the etiology of the high fever and brings into play diagnostic constructs that are less likely to be considered in the context of a mild elevation in body temperature (e.g., meningitis). The point is that structurally valid constructs do not necessarily have a 1:1 map with underlying etiologies or pathophysiologies (e.g., the quantitative dimension of fever can result from a variety of underlying causes, some of which are discrete, and we may very well discover that neuroticism is similarly multidetermined; Zuckerman, 2005). Nevertheless, the search for the biological and environmental factors that contribute to psychopathology is importantly constrained and assisted by knowing about the manifest empirical structure of psychopathology. The conceptualization of psychopathology in DSM–IV—that mental disorders are discrete categories with well-defined boundaries—is untenable and incompatible with the data (Kendell & Jablensky, 2003; Krueger & Markon, 2006).

Consider the following specific neurobehavioral example. The monoamine neurotransmitter serotonin is involved in many diverse behavioral functions. As a result, “the functioning of the serotonin system” probably relates to multiple “individual differences in behavioral propensities.” For example, DeYoung (2006) argues that neuroticism, disagreeableness, and unconscientiousness are sufficiently correlated to give rise to a higher order dimension of “(in) stability,” and that the confluence of these characteristics maps reasonably well onto serotonin-
ergic functioning. In a somewhat different account, Carver, Johnson, and Joormann (2009) argue that these diverse phenotypic individual differences are associated with serotonin because low serotonin reduces the organism’s capacity to bring deliberate effortful control to bear on behavior. As a result, whatever cues are salient at a more basic motivational level (e.g., signals for reward vs. punishment) are more likely to drive behavior in the context of low serotonin, and those cues could drive the organism toward ruminative neuroticism (which might eventuate in a depressive episode) or impulsive aggression (disagreeable unconscientious behavior), depending on the nature of the immediate circumstances.

The point here is that this discussion about the role of serotonin in human individual differences is framed and constrained by our understanding of the structure of human personality. The phenotypic constructs that constrain this discussion are known to map the structure of individual differences in human personality traits (McCrae & Costa, 2008), thereby usefully constraining theory and research on the role of serotonin in human individual differences. DeYoung (2006) and Carver et al. (2009) are offering somewhat different theories, but they are also talking about the same empirically derived phenotypic domains.

Contrast this with a theoretical proposition such as “the serotonin system is ‘disrupted’ in DSM–IV unipolar mood disorders.” Taken at face value, this proposition is not unreasonable (cf. Carver et al., 2009). However, it is very hard to test this proposition as stated because the DSM–IV categories are not well constrained by data on the empirical structure of psychopathological signs and symptoms. For example, Brown, Campbell, Lehman, Grisham, and Mancill (2001) found that 95% of individuals who met criteria for lifetime major depression or dysthymia in a clinical sample also met criteria for a current or past anxiety disorder. As a result, if we sought evidence of serotonergic abnormalities in depression, that evidence would probably have to be derived from cases of depression with at least some history of anxiety, and we would be unable to state with much clarity that the abnormalities we observed could be attributed to a specific diagnostic construct as opposed to a host of other putatively distinct DSM–IV diagnoses that flow naturally along with each other.

In sum, we advocate using numerical taxonomy to articulate a structurally valid model of descriptive psychopathology as a key next step in the evolution of research on the classification of psychopathology. This is a tractable goal that builds on the advances that began with DSM–III, that is, the ability to assess reliably the presence versus absence of specific psychopathological signs and symptoms. We have the statistical modeling technology needed to converge on a structurally valid model of psychopathology, given the right data (e.g., large-scale symptom-level data derived from reliable assessment instruments, but unconstrained by existing DSM conventions). A structurally valid model of psychopathology is vital because it provides a data-based framework for working toward an understanding of the underpinnings of psychopathology, and therefore, a framework with enhanced clinical applicability. Because of phenomena such as comorbidity, within-category heterogeneity, and NOS diagnoses, research framed by DSM–IV constructs is difficult to interpret at best. As a direct result, such research has limited applicability in clinical settings. For example, if the modal patient meeting criteria for a commonly encountered diagnosis such as major depressive disorder also meets criteria for other disorders, research on persons with “pure major depressive disorder” (a common strategy) has questionable relevance to everyday clinical practice. By contrast, research framed by structurally valid constructs (such as constructs from empirically derived models of personality traits) is considerably more interpretable. Such research parses people as they actually occur in nature, and is therefore more readily translated to application in clinical settings. We turn now to discuss how personality trait research per se could be drawn on to help frame DSM–5 in a broad sense and thereby propel a move to a structurally valid system of psychodiagnosis.

Linking Personality Traits With Axis I Psychopathology in DSM–5

It is probably unrealistic to think that a complete and structurally valid model of psychopathology derived from numerical taxonomic research could be prepared in time for DSM–5. A
major reason for this is that the field, and hence the potentially relevant data, are constrained by the constructs of DSM–IV. However, the potential exists to frame DSM–5 by structural research on DSM–IV constructs, as a bridge to a comprehensive, structurally valid model of psychopathology. In addition, DSM–5 could be designed to help encourage the collection of data that could be used to pursue numerical taxonomic research to inform DSM–6 (cf. Helzer, Kraemer, & Krueger, 2006; Watson & Clark, 2006).

As a starting point for this discussion, consider data on relations between personality traits and Axis I phenomena (keeping in mind that the multiaxial system of DSM–IV may also be discarded in DSM–5). In addition to their clear relevance to improving conceptualization of PDs, dimensional personality traits show robust, sizable, and psychologically meaningful connections with Axis I disorders. For example, a recent meta-analysis examined 33 studies linking the FFM personality domains with a wide variety of mental disorders and found compelling links with all FFM domains except openness (Malouff, Thorsteinsson, & Schutte, 2005). Indeed, the effect size for the link between neuroticism and clinical disorders was 0.92, a “large effect” by Cohen’s (1988) standards.

The link between personality and Axis I disorders extends beyond bivariate correlations to parallels in the multivariate structure of both domains. Specifically, comorbidity patterns linking common Axis I mental disorders can be understood in terms of two superordinate, organizing spectrum constructs: internalizing and externalizing (for recent reviews, see Eaton, South, & Krueger, in press; Krueger & Markon, 2006; Slade & Watson, 2006). The internalizing spectrum includes unipolar mood and anxiety disorders, and the externalizing spectrum includes antisocial behavior disorders and substance use disorders. These spectrum constructs reflect both the phenotypic structure of comorbidity and the underlying genetic structure of comorbidity (Kendler, Prescott, Myers, & Neale, 2003; Krueger et al., 2002). Recent structural modeling work by Markon (2010) focused on the symptom rather than the disorder level, and including both Axis I and II symptoms has extended this phenotypic model by indicating the existence of thought disorder and pathological introversion domains in addition to internalizing and externalizing. The parallels between Markon’s four psychopathology factors and the PFM are apparent (i.e., internalizing resembles negative emotionality, externalizing resembles disinhibition and antagonism, thought disorder resembles peculiarity, and pathological introversion resembles introversion).

The parallels in the structure of common Axis I disorders and personality traits can also be examined from a number of other vantage points. One way to address these connections is to see whether personality traits “fit” along with DSM constructs in the same spectra. Along these lines, trait neuroticism has been shown to fit into the internalizing spectrum when modeled simultaneously with mental disorders such as major depression, generalized anxiety disorder, panic disorder, agoraphobia, and social phobia (Hettema et al., 2006; South & Krueger, 2008). Similarly, disinhibitory personality traits fit in the externalizing spectrum, along with antisocial personality disorder features per se (Krueger et al., 2002). From this vantage point, personality traits and mental disorders can be conceptualized as manifestations of the same underlying spectra of human variation, with environmental circumstances having a key impact on the manifest form of behavior (e.g., the development of personality tendencies vs. diagnosable psychopathology).

Another vantage point on personality trait–Axis I relations involves seeing how much comorbidity can be attributed to personality traits. Khan, Jacobson, Gardner, Prescott, and Kendler (2005) investigated the proportion of comorbidity between disorders due to personality traits. Neuroticism was found to play a particularly substantial role in the comorbidity of common mental disorders. With regard to any two of the internalizing disorders assessed (i.e., major depression, generalized anxiety disorder, panic disorder, and any phobia), neuroticism accounted for a substantial proportion of the comorbidity between the disorders.

Drawing from this kind of evidence, we recently joined forces with colleagues to suggest a potential metastructure for DSM–5 and the International Classification of Diseases–11 (Andrews et al., 2009). The idea in this work was to take existing diagnostic constructs at face value, but to try to rearrange them in a way
that might be more parsimonious and better reflect data on shared underlying connections. Five broad clusters of disorders emerged from this exercise: (1) neurocognitive disorders (e.g., the dementias), (2) neurodevelopmental disorders (e.g., autism), (3) psychoses (e.g., schizophrenia), (4) emotional or internalizing disorders (e.g., unipolar mood and anxiety disorders), and (5) externalizing disorders (e.g., antisocial behavior and substance use disorders). Some of these classificatory rubrics are not explicitly connected with the structure of personality. However, the explicit basis for clustering emotional (internalizing) and externalizing disorders was their shared personality trait antecedents.

The model described by Andrews and colleagues (2009) represents a reasonable start on a classification better connected with data, but it is only a beginning because it is based on existing diagnostic categories. Nevertheless, it offers a reasonable way to incorporate personality traits in DSM–5, with regard to Axis I conditions, should those constructs persist in their current form. Specifically, if disorders were organized into these sorts of clusters, the personality trait basis for the organization of the relevant clusters could be made explicit in the text. This would help in breaking down the relatively artificial barrier between personality and psychopathology (cf. Clark, 2005; Krueger & Tackett, 2003). Nevertheless, ultimately, the symptoms of Axis I disorders and the affective, cognitive, and behavioral phenomena that constitute personality traits need to be merged more explicitly in the pursuit of a structurally valid model of personality and psychopathology (cf. Krueger, Markon, et al., 2007).

**Personality Traits and Other Outcomes of Clinical Relevance**

We have reviewed how personality traits are central to improving the classification of psychopathology. Yet, including an empirically based personality trait model in DSM–5 could also have benefits beyond clarifying the role of personality in working toward a structurally valid nosology. Specifically, personality traits are also relevant in predicting and understanding clinically important outcomes beyond psychopathology per se. We turn now to briefly describe two particularly compelling examples of domains in which the broad relevance of personality traits in clinical settings seems clear: physical health and interpersonal functioning.

**Physical health.** Perhaps no outcome is more important to the health care professions than mortality. In longitudinal data, personality traits have been shown to predict mortality more strongly than socioeconomic status (SES) and equivalently to IQ (Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). Personality traits are also relevant to overall physical health. Lahey’s (2009) compelling recent review of the public health implications of neuroticism notes that, even after controlling for other risk factors (e.g., social support), neuroticism is related to a diversity of physical health concerns. These problems range broadly from cardiovascular disease to asthma to eczema. Furthermore, the courses of at least some medical conditions can be predicted by neuroticism, such as cardiac disease and renal deterioration in diabetes (Lahey, 2009). Personality traits also appear to be relevant to resilience, for example, optimism conveys resilience to physical disease (T. W. Smith, 2006).

Evidence is mounting that personality is associated with and often prospectively predicts physical health outcomes, but the precise nature of the mechanisms underlying these links is less clear (Ozer & Benet-Martinez, 2006). One likely mechanism involves the impact of personality on health-related decision making. For example, the link between neuroticism and physical health and mortality has been hypothesized to result from more neurotic individuals engaging in behaviors that put their health at risk (e.g., smoking). Mroczek, Spiro, and Turiano (2009) tested this hypothesis, and found that the link between neuroticism and mortality was reduced by 40% when the effect of smoking was removed. Personality may also be part of the explanation for well-replicated socioeconomic disparities in mortality. Chapman, Fiscella, Kawachi, and Duberstein (in press) found that personality traits were able to explain 20% of the SES gradient in mortality.

**Interpersonal functioning.** Interpersonal functioning is clearly relevant in many clinical situations, and, similar to mortality, personality traits such as neuroticism, disagreeableness, and unconscientiousness predict divorce longitudinally (Roberts et al., 2007). Indeed, when compared with SES, personality traits are better
predictors of divorce. A recent review by Ozer and Benet-Martínez (2006) further supports the link between interpersonal outcomes and personality traits. Relations with peers in childhood are predicted by agreeableness and extraversion; young adults’ neuroticism, conscientiousness, and extraversion levels predict the quality of relationships with their parents; and romantic relationship outcomes, including abuse, conflict, and dissatisfaction, are predicted by levels of agreeableness and neuroticism.

**Personality Traits and Treatment Considerations**

A growing literature—including a recent special issue of the *Journal of Personality Assessment* (Hilsenroth, 2004)—indicates that personality is also an important consideration in framing interventions for both mental disorders and physical diseases. Personality predicts overall treatment effectiveness, as well as specific components of effectiveness, such as an individual’s adherence to a treatment regimen.

Depression has been a particular focus of research in this area. In one study, for example, depressed patients received both medication and psychotherapy, and treatment responders had lower levels of neuroticism and higher levels of extraversion and openness than nonresponders (Quilty et al., 2008). Further analyses indicated that conscientiousness as well as Neuroticism × Extraversion and Extraversion × Conscientiousness interactions were also predictive of treatment response. In addition, personality traits have been shown to predict differential treatment response of specific patients to specific treatment modalities. For instance, Bagby and colleagues (2008) investigated the link between personality traits and response to either cognitive–behavioral therapy or pharmacotherapy. The results of this study indicated that the higher a depressed patient’s level of neuroticism, the more likely he or she was to respond to pharmacological treatment rather than to cognitive–behavioral treatment. Results such as these underscore the ways in which personality traits may influence overall treatment outcome as well as inform the choice of treatment modality for particular patients given their personality profiles.

Part of the reason personality traits are relevant to intervention may lie in traits being in-direct targets of intervention. Neuroticism is etiologically connected with internalizing disorders (Hettema et al., 2006), and interventions targeting internalizing disorders may also be targeting neurobiological systems linking personality and psychopathology (Costa, Bagby, Herbst, & McCrae, 2005). A recent empirical example was provided by Hellerstein, Koocsis, Chapman, Stewart, and Harrison (2000). Patients with a primary diagnosis of dysthymia were randomized into treatment with sertraline, imipramine, and placebo; these patients also completed Cloninger’s Tridimenisonal Personality Questionnaire (TPQ; Cloninger, 1987) at both pre- and posttreatment. Pretreatment TPQ harm avoidance scores were 1.5 standard deviations higher than community norms, yet remission of dysthymia was associated with reduction in TPQ harm avoidance. Indeed, for the sertraline group, 36% showed a decrease of at least 1 standard deviation from baseline harm avoidance. Psychosocial interventions originally aimed at specific clinical problems may also have beneficial effects on personality. Blonigen, Timko, Moos, and Moos (2009) showed that a longer duration of involvement in Alcoholics Anonymous predicted a reduction in impulsivity, which in turn predicted a reduction in legal problems over a 16-year follow-up period.

Another means by which personality could affect treatment outcome is through adherence. One recent study examined cholesterol-lowering treatment adherence by objectively assessing the regularity with which patients took prescribed medication (Stilley, Sereika, Muldoon, Ryan, & Dunbar-Jacob, 2004). Among the FFM personality traits, only conscientiousness showed a significant association with medication compliance. At 1 week into the study, conscientiousness showed a small and nonsignificant link to adherence ($r = .06, ns$). Over time, however, this association increased, and, by Week 24, the link between conscientiousness and adherence was notable ($r = .24, p < .01$).

In addition, specific treatments appear to work better than others for patients with different personalities. Personality trait assessment could be used by health care professionals to select the interventions to which the client is most likely to adhere (e.g., using long-lasting medication injections every few weeks vs. requiring an unconscientious patient to take med-
ications daily, or allotting time in session for an unconscious patient to complete homework; cf. Harkness & Lilienfeld, 1997; J. D. Miller, Pilkonis, & Mulvey, 2006). Personality assessment could also be used as a prescreening for vulnerability to future disorder and as a tool for identifying individuals at high risk who may benefit from preemptive intervention (e.g., stress inoculation; Lahey, 2009).

Finally, personality traits are also worth considering on the other side of the equation, that is, the traits of the practitioners. Concerned about burnout among primary care physicians, Krasner and colleagues (2009) evaluated the effects of a continuing medical education course focused on mindfulness in clinical encounters. They found that their course resulted in significantly increased conscientiousness and decreased neuroticism among the physician participants (as well as other clinically relevant improvements, such as reduced burnout) at the end of the course. The conclusion is that the traits of practitioners can affect the care they are able to provide, and these dispositions are amenable to intervention that could enhance the nature of clinical care.

Toward DSM–5: An “Open Source” Perspective

Our aim in this review was to illustrate the diverse ways in which the inclusion of an empirically based personality trait model could constitute a critical innovation in the transition from DSM–IV to DSM–5. Our initial focus was on personality disorder classification and conceptualization because this is the area in which dimensional models of personality traits have been discussed most extensively. We also briefly touched on the role of personality in organizing Axis I disorders and the relevance of personality in predicting other clinically important phenomena, including treatment considerations. Our hope is that the reader joins us in our enthusiasm regarding the critical role personality traits can play in both research and in the clinic. Importantly, personality traits are far from “untreatable” and may, in fact, be the true targets of psychopharmacologic interventions originally intended for Axis I conditions (Knutson et al., 1998; Tang et al., 2009). Moreover, personality traits are not static entities or permanent and unchanging characteristics. Rather, traits change over time in normative ways (Roberts & Mroczek, 2008). Erroneous conceptions of personality traits as static, unchanging, and untreatable have given way to a more nuanced understanding that traits can and do change given the right circumstances.

More broadly, our hope is that the reader joins us in our enthusiasm for the construction of a structurally valid model of psychopathology that draws on parallels in the structure of personality. DSM–III and its offspring constituted a critical innovation in the history of the field because they offered concrete, observable criteria for psychopathology, many of which could be reliably assessed. Nevertheless, we have learned a lot since DSM–III was published in 1980, particularly regarding the limitations of a categorical and polythetic conceptualization of mental disorder. The time therefore has come to evolve the conceptualization of mental disorders described in DSMs III to IV. Numerical taxonomy has an important role to play in this evolutionary period in the history of thinking about mental disorder classification. Formal quantitative models are indispensable tools in the development of a structurally valid model of psychopathology, a model that could be very helpful in framing both clinical thinking and research.

Because it is slated to be published very soon (2013), creating a quantitative model of psychopathology is not a realistic goal for DSM–5. However, it is a tractable and desirable goal in general. The key to pursuing this goal is to encourage—rather than constrain through fiat—creative and novel research on the classification of psychopathology. DSM–5 will be a success story to the extent that it becomes an “open source” document, fostering novel and creative thinking about classification, as opposed to reifying necessarily provisional and imperfect definitions of mental disorders.

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