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Construct Validation Theory Applied to the Study of Personality Dysfunction

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Abstract

The authors review theory validation and construct validation principles as related to the study of personality dysfunction. Historically, personality disorders have been understood to be syndromes of heterogeneous symptoms. The authors argue that the syndrome approach to description results in diagnoses of unclear meaning and constrained validity. The alternative approach of describing personality dysfunction in terms of homogeneous dimensions of functioning avoids the problems of the syndromal approach and has been shown to provide more valid description and diagnosis. The authors further argue that description based on homogeneous dimensions of personality function/dysfunction is more useful, because it provides direct connections to validated treatments.

In this article, we make the following argument: The application of theory validation and construct validation principles to the study of personality dysfunction provides clear direction on the most promising way to proceed in understanding disorders of personality. Specifically, the use of a syndromal approach for diagnosis and description necessarily results in diagnoses of unclear meaning and constrained validity. In contrast, description of personality dysfunction in terms of homogeneous dimensions of functioning avoids the problems of the syndromal approach and has been shown to provide more valid description and diagnosis. In addition, description based on homogeneous dimensions of personality function/dysfunction is more useful, because it provides direct connections to validated treatments.

To make this argument, we first review the relevant principles of theory and construct validation. With those principles in mind, we then consider the classic approach to diagnosing personality disorders in terms of categorical syndromes. Doing so highlights fundamental problems with syndromal approaches: we show that syndromal systems involving (a) the use of a single diagnosis or score to represent multiple, heterogeneous symptoms and (b) the presence of common symptoms in multiple syndromes necessarily constrain the validity of personality dysfunction description. We then consider the alternative approach of describing personality dysfunction in terms of homogeneous dimensions of personality. We show how this approach leads to more valid description of constructs and more valid theory tests, and we note that, in stark contrast to the situation with syndromal diagnosis, there is impressive evidence for the validity of this alternative approach. Next, we show how dimensional description facilitates treatment planning. We conclude by addressing the issue of diagnosing the presence or absence of dysfunction: We suggest that diagnoses should be made based on demonstrated impairment in social, emotional, and/or occupational functioning, not based on symptom counts or personality dimension scores.

Theory Validation and Construct Validation

The development of descriptions of psychopathology, and hence systems for diagnosing its presence, is based first of all on theory: One proposes that a certain form of dysfunction exists, that it has certain characteristics, and that it is associated with certain negative life outcomes. One relies on empirical research conducted both prior to and following one's proposal to determine the proposal's validity. One might examine the frequency of the occurrence of the dysfunction, whether the hypothesized characteristics of the dysfunction co-occur to a substantial degree, whether the dysfunction does lead to the hypothesized negative life outcomes, and whether clinicians can assess the dysfunction reliably. To test any of these hypotheses, one needs measures of both the proposed dysfunction, which involves measurement of each of its characteristics; and the negative life outcomes of interest. Our focus in this article is on measurement of dysfunction. A focus on measurement of dysfunction is important, because it is true that the validity of the conclusions one draws from psychopathology research depends on the validity of the measures that were used in that research.

One cannot presume that any measure one develops represents the intended target construct validly; the history of psychological assessment testifies to that reality (Smith & Zapolski, 2009; Strauss & Smith, 2009). How, then, does one develop evidence that a measure of a psychological construct is valid? A crucial challenge one faces is that most constructs in the domain of personality dysfunction are not directly observable. Concepts such as "identity disturbance," "impulsivity," "lack of empathy," and "perfectionism" do not have tangible, concrete embodiments that are isomorphic to their intended meaning. Accordingly, to provide evidence that a measure of such a construct is valid, one must demonstrate that the measure relates to measures of other constructs as predicted by theory (Cronbach & Meehl, 1955). For an unobservable, and hence hypothetical, construct, there is no way to determine whether a measure reflects the construct validly, except to test whether scores on the measure conform to a theory, of which the target construct is a part (Cronbach & Meehl, 1955; Smith, 2005).

Suppose, for example, one were to investigate the validity of a measure of identity disturbance. One would need evidence of this kind: (a) independent expert agreement that the content of the measure conforms to one's definition of identity disturbance; (b) scores on the measure covary positively with scores on measures of other constructs that theory suggests relate to identity disturbance (perhaps scores on measures of affective instability and relationship problems); and (c) scores on the measure do not covary with scores on measures of constructs thought to be unrelated to identity disturbance (one might demonstrate that one's measure of identity disturbance is unrelated to measures of intelligence or agreeableness). If the results of studies testing these hypotheses conform to one's theory, one becomes more confident that one's measure validly reflects identity disturbance. Of course, validation evidence of this kind is far from determinative; there may well be other constructs that have positive relationships with affective instability and relationship problems but not with intelligence or agreeableness. For that reason, researchers do everything they can to increase their confidence that a measure validly reflects the intended construct.

One crucial first step is to define the target construct in precise terms. The content domain that is included in a measure must represent all aspects of the construct represented in its definition, and it must exclude all content not represented in its definition. A problem that has surfaced repeatedly in psychological assessment is the inadvertent inclusion of content not prototypic of the target construct in measures, with the result that a single scale includes items representing more than one construct. For example, the Novelty Seeking scale of the

Temperament and Character Inventory (Cloninger, Przybeck, & Svrakic, 1991) includes the item (a) “I often try new things just for fun or thrills, even if most people think it is a waste of time” and also the item (b) “I am usually able to get other people to believe me, even when I know that what I am saying is exaggerated or untrue.” As noted by Smith, Fischer, and Fister (2003), these two items appear to reflect different constructs: item (a) may be prototypic of novelty seeking, but surely item (b) is not. In fact, item (b) may represent content in a domain relevant to antisociality.

This problem is very important for the construct and theory validation process. Imagine a scale in which half the items were parallel to item (a) and half were parallel to item (b). Correlations between such a scale and risky behaviors would probably be lower than they would have been if all the scale’s items were parallel to item (a). In addition, such a scale might well result in higher correlations with measures of antisociality than would have otherwise occurred. The result might be to underestimate the role of novelty seeking in risky behavior involvement and overestimate the contribution of novelty seeking to antisociality. In the end, researchers’ basic understanding of these forms of psychopathology could be distorted due to the inclusion of items representing two different constructs on a single scale.

For many years, this problem plagued research in the domain of impulsivity. Seminal work by Whiteside and Lynam (2001) indicated that many measures of impulsivity actually included items representing multiple different constructs, including a failure to plan, a failure to persevere on tasks, sensation seeking, and emotion-driven impulsivity. These constructs are only modestly related to each other and they are not alternative representations of a common impulsivity construct (Smith et al., 2007). Thus, single scores on a measure of “impulsivity” actually reflected variation in two, three, or even four different constructs. A good example of one of the many problems that resulted from this lack of clarity in the meaning of impulsivity measures was this: For many years, impulsivity was thought to have a very weak relationship to bulimia nervosa (Stice, 2002); however, meta analysis based on distinctions among the different impulsivity-related constructs indicated that emotion-driven impulsivity correlates with bulimia nervosa symptoms quite substantially ($r = .40$), and the other traits had trivially small correlations with that disorder (Fischer, Smith, & Cyders 2008). This clarified finding has informed new theories of risk for bulimia nervosa (Combs & Smith, 2009; Fischer, Smith, & Cyders, 2006).

The inaccurate findings that can result from using a single score to represent multiple constructs, or multiple dimensions of a phenomenon, can be attributed to two sources of uncertainty in such measurement. First, with a single score, one cannot know whether different dimensions play different roles in relation to external behaviors. For example, one dimension may be highly correlated with the criterion, and other dimensions may not correlated with it at all: A single score representing all those dimensions essentially averages the positive effect of the first dimension with the non-effects of the other dimensions, such that the overall score under-represents the role of the first dimension and over-represents the roles of the other dimensions. This process is what appears to have occurred in studies relating “impulsivity” to bulimia nervosa (Fischer et al., 2008). In any given case, one would not know the relative contributions of the different constructs to the effect that was observed.

The second source of uncertainty is perhaps of even greater concern; it lies in the fact that different profiles of scores on the constructs within the single measure can produce the same overall score. Consider that the correlation between emotion-driven impulsivity (called negative urgency) and sensation seeking has been found to be $r = .12$ (Cyders & Smith, 2007). The two constructs share very little variance, but if they are combined into a single score, a person high in negative urgency and low in sensation seeking could earn exactly the

same score as a person with the opposite pattern of being low in negative urgency and high in sensation seeking. The same score could reflect two very different psychological experiences. That reality would not be apparent to researchers, and there would not be a clear way to interpret correlations between this measure and measures of other constructs or measures of psychopathology (Smith, McCarthy, & Zapolski, 2009; Strauss & Smith, 2009).

For these and other reasons, validation theorists have come to conclude that single scores on measures should represent single, homogeneous constructs (Edwards, 2001; Hough & Schneider, 1995; McGrath, 2005; Paunonen, 1998; Paunonen & Ashton, 2001; Schneider, Hough, & Dunnette, 1996; Smith et al., 2003; Smith & McCarthy, 1995; Smith et al., 2009; Strauss & Smith, 2009). Only when single scores represent single constructs is it possible to conduct fully accurate validation and theory tests, because only then can one develop confidence in the meaning of variation on a test score.

Issues Concerning the Validation of Descriptions of Personality Dysfunction

Any approach to description and diagnosis represents a theoretical position concerning the nature of dysfunction to be described. The classic approach to personality dysfunction description and diagnosis has been to make use of the syndrome perspective to identify a set of separate personality disorders (PDs: Millon et al., 1996). The syndrome perspective involves the identification of a constellation of symptoms that are thought to stem from a common cause or to indicate a disease or abnormal condition (Kraepelin, 1981). Syndromal disorders can, and often do, include heterogeneous symptoms, as long as the various symptoms reflect a common cause. Individuals with the same disorder may not have the same set of symptoms as each other; not all symptoms are thought to necessarily ensue when the disease cause is present. Similarly, the symptoms within a syndrome may not correlate perfectly, because some symptoms may be caused sometimes by one disorder and other times by a different disorder. To use an example from medicine, the experience of headaches, muscle aches, sore throats, and fatigue do not covary perfectly because, although all of them may be symptoms of the flu, there are other disorders that lead to headaches but not sore throats, or fatigue but not headaches, and so on. The identification of separate, syndromal PDs is an expression of the theoretical position that personality dysfunction is best understood as sets of syndromes. There is a cause for each syndrome and the different syndromes may share some symptoms in common.

In light of the foregoing discussion of theory validation and construct validation, how might one validate the syndromal theory of PD? If a given syndrome does exist and stems from a common cause, then presumably one could develop a test that gives you a single score concerning the presence or absence of the syndrome (and one could then show that variation on the test covaries with variation in the predicted symptom expression). An analogue in medicine would be a lab test to determine the presence or absence of a bacterial infection, and the presence of the infection explains the observed symptoms of fever, muscle aches, and fatigue. To date, however, this has not occurred for a single PD. Kupfer, First, and Regier (2002: Kupfer is chair and Regier vice chair of the American Psychiatric Association's [APA] fifth edition of the Diagnostic and Statistical Manual of Mental Disorders [DSM-5] task force) noted that:

“... the goal of validating these syndromes and discovering common etiologies has remained elusive. Despite many proposed candidates, not one laboratory marker has been found to be specific in identifying any of the *DSM*-defined syndromes. Epidemiologic and clinical studies have shown extremely high rates of

comorbidities among the disorders, undermining the hypothesis that the syndromes represent distinct etiologies.” (p. xviii)

In short, despite many decades of research, the most straightforward method of validating the theory that the different PDs represent different syndromes of dysfunction has produced no evidence supporting the syndromal model. That reality has led to a serious, if not fully recognized, problem in assessing PDs. In the absence of “syndrome tests,” researchers have instead used overall symptom counts to measure the putative syndromes, whether reflecting the presence or absence of a disorder or its magnitude along a continuum.

In light of the foregoing discussion of test validation and construct validation procedures, using an overall score is most appropriate when the score refers to a location along a single, homogeneous dimension. But certainly from the syndromal theory of personality dysfunction, PD diagnoses are understood to include a heterogeneous collection of symptoms (Trull & Durrett, 2005). If the symptoms that contribute to a given PD diagnosis do reflect different dimensions of psychological functioning, then summing them to produce a single score or single diagnosis is to produce scores or diagnoses of unclear meaning: Different individuals can receive the same diagnosis despite having important differences in their personality structures and in their psychological experiences. Considering just the PD diagnostic criterion sets, two different people can share the schizoid, schizotypal, antisocial, borderline, narcissistic, or avoidant personality disorders and in each case have only one diagnostic criterion in common. From the standpoint of syndromal theory, this reality is fine, as long as one can identify the specific syndrome accurately. But there is not a single PD for which that is the case.

In the absence of evidence that current PD descriptions represent true syndromes, the validity of the strategy of combining heterogeneous symptom counts to produce a single diagnosis or single score can only rest on the premise that, in fact, the heterogeneity of the symptoms is more apparent than real; that is, that the symptoms within a given putative disorder correlate highly with each other, so that each is an alternate expression of the same psychological experience. Livesley (1998) made a similar point, by referring to the internal validity of diagnoses as defined by the extent to which diagnostic criteria form homogeneous clusters.

Heterogeneous, Modestly Correlated Symptoms within Putative Disorders

The evidence suggests quite the opposite. There appears to be limited internal validity, or limited homogeneity, for DSM-based PDs; in fact, some diagnostic criteria correlate more highly with diagnoses other than the ones to which they contribute (Morey, 1988a, 1988b). For borderline personality disorder (BPD), the intercorrelations among the nine symptoms have been found to range from $-.30$ to $.30$ within a nonclinical sample and from $.29$ to $.51$ in a clinical sample (Sanislow, et al., 2002; Taylor & Reeves, 2007). Similar findings for other PDs have been reported. Indeed, median within diagnosis intercorrelations for each of the eleven personality disorders found in the DSM-III-R (Morey, 1988a) ranged from 0.10 (obsessive-compulsive personality disorder) to 0.29 (paranoid personality disorder). The heterogeneity of symptoms within disorders has not changed with the DSM-IV-TR (APA, 2000). In short, the criteria within PD disorders are only very modestly related to each other. It is thus the case that (a) there are no identified syndrome tests, for any of the PDs, and (b) symptoms within a PD are not highly correlated with each other, i.e., they are not alternate expressions of the same psychological experience. To combine such modestly correlated symptoms into a single score is to create scores with unclear meaning: Two individuals can have the same score yet have very different symptom pictures. Again, this would not be problematic if evidence for the syndromal nature of PDs existed; however, the absence of

such evidence suggests that use of a single score for a PD is a measurement problem that can produce misleading, inaccurate results.

It may not just be that there is an absence of evidence that the symptoms within a disorder have a common cause; it may be more accurate to say that there is good evidence that symptoms within disorders have different causes from each other. Expert consensus on the personality dimensions underlying paranoid personality disorder (PPD) suggest that this PD is characterized by, among other things, high levels of angry hostility, low levels of warmth, low levels of openness to actions, low trust, and low compliance (Lynam & Widiger, 2001). Research on the heritability of these personality traits has indicated that their specific variance, i.e., that trait variance not shared with other traits, is substantially heritable (Jang, McCrae, Angleitner, Riemann, & Livesley, 1998). That is, there appear to be genetic causes of variance on each trait that does not cause variance on other traits. This analysis applies to the personality contributors to each PD. If trait contributors to PDs have, in part, different sources of heritability from each other, then they have at least partly different causes. Thus, a given PD is comprised of symptoms that vary in the psychological experiences to which they refer, that correlate with each other very modestly, and that have, at least in part, different causes.

It is worth noting that the same problem exists in the use of Axis I disorders. One person could have the symptoms of depressed mood, significant weight loss, insomnia, feelings of worthlessness, and diminished ability to think or concentrate. Another person could have no depressed mood, but have the symptoms of a loss of interest in or pleasure in most activities, significant weight gain, psychomotor retardation, fatigue/energy loss, and recurrent thoughts of death. These two individuals have virtually no symptoms in common, yet both meet the criteria for a major depressive episode. The use of the syndrome perspective, and the problems resulting from it, plague the DSM as a whole.

The Same Symptoms Across Multiple Putative Disorders

It is a well-known characteristic of the DSM-IV-TR system that similar criteria are used as indicators for many disorders, including both Axis I and Axis II disorders. As noted by Widiger and Samuel (2005), there is a great deal of common, shared pathology across disorders. Perhaps the most frequently shared pathology is negative affectivity or subjective distress. Indeed, Lynam and Widiger (2001) found that experts identified some form of negative affectivity to be present in 9 of 10 PDs (all but schizoid, which was identified as low on positive emotions). Lynam and Widiger also found that experts (a) identified expressions of abnormally low extraversion in 7 of 10 PDs and abnormally high extraversion in 2 of 10 PDs; (b) abnormally low openness to experiences in 5 of 10 PDs and abnormally high openness in 4 of 10 PDs; (c) abnormally low agreeableness in 3 of 10 PDs and abnormally high agreeableness in 3 of 10 PDs; and (d) abnormally low Conscientiousness in 4 of 10 PDs. Similarly, Shedler and Westen (2004) examined the diagnostic criteria of DSM-IV-TR personality disorders and found substantial overlap in the clinical descriptions and features of the diagnostic categories for avoidant and dependent personality disorder, borderline and histrionic personality disorder, and schizoid and schizotypal personality disorder. It is also the case that many symptoms of PDs are also identified as symptoms of Axis I disorders (e.g., negative affectivity/subjective distress and the mood disorders).

The use of the same or similar symptoms as criteria for more than one PD leads to a finding of high comorbidity among PDs, just as the use of the same symptoms for both Axis I and Axis II disorders results in a finding of high comorbidity across the axes. Numerous studies have documented high comorbidity of these kinds. Grilo, Sanislow, and McGlashan (2002) and Watson and Sinha (1998) both reported high comorbidity among PDs. Individuals

diagnosed with BPD have also been found to be more likely to receive a current diagnosis of major depressive disorder (MDD), bipolar I and II disorder, panic disorder with agoraphobia, social and specific phobia, posttraumatic stress disorder (PTSD), obsessive compulsive disorder (OCD), eating disorder NOS, and any somatoform disorder (Zimmerman & Mattia, 1999). The American Psychiatric Association (2000) reported that among clinical populations with personality disorder, thirty to sixty percent of individuals are also diagnosed with BPD. Of Axis II disorders, avoidant, dependent, and paranoid personality disorders have been identified as the most frequently diagnosed comorbid conditions (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). Widiger et al. (1991) also noted that individuals diagnosed with paranoid personality disorder are almost always diagnosed with a secondary co-occurring diagnosis. Even obsessive-compulsive personality disorder has been reported to have a comorbidity rate of approximately 70 percent (Livesley, 1998).

If the syndromal model had proved valid, then these findings of high comorbidity would perhaps be viewed as interesting, important substantive findings concerning the co-occurrence of many forms of mental dysfunction. But to the degree that the apparent comorbidity is really a function of the same form of dysfunction being counted multiple times (e.g., negative affectivity contributing to diagnoses of BPD, Paranoid PD, and narcissistic PD), it is necessary to rethink the idea of comorbidity. In the absence of evidence for the validity of the syndromal model, this appearance of comorbidity may be a simple function of having the same psychological dysfunction contribute to multiple different diagnostic labels.

Prototypal Matching Preserves the Problems with Syndromes

One of the original proposals for the DSM-5 description of personality dysfunction involved moving from symptom counts to a prototypal matching model (Skodol, et al., 2011). In this model, clinicians rate how closely a patient's personality matches a profile identified as the prototype for a given PD. One is asked to read a description of, for example, the prototypic BPD patient and then rate whether one's patient's personality is a very good match, a good match, a moderate match, a slight match, or no match with that prototype. Part of the rationale for using prototype matching is to reduce the high rate of comorbidity found using the DSM-IV-TR PD diagnostic system. Using this approach, clinicians would not be counting symptoms to meet a diagnostic threshold; in the absence of symptom counting, there will presumably be less assignment of multiple PD diagnoses. Instead, clinicians would perhaps be more likely to identify a single PD that their client most embodies.

However, the issue with comorbidity is not resolved using prototype matching. For example, Westen, Shedler, and Bradley (2006) compared empirically the extent of diagnostic co-occurrence that is obtained with prototype matching with the extent of diagnostic co-occurrence that would be obtained if the same clinicians systematically considered each DSM-IV diagnostic criterion. The authors reported considerably less diagnostic co-occurrence using prototypal matching, but when the clinicians were required to systematically assess specific features of the personality disorders the co-occurrence reappeared. Clearly, this finding does not mean that prototype matching solves the comorbidity problem; rather, it simply does not provide an adequate recognition of its existence. Thus, prototype matching appears only to provide an illusion of having solved the problem of comorbidity with respect to personality disorders.

The prototypal matching approach begins with the assumption that the PDs are valid descriptions of personality dysfunction (at least those PDs that are maintained in DSM-5: Skodol et al., in press). In the absence of evidence that the PDs represent identifiable syndromes, and in the presence of evidence that symptoms within a given PD correlate with

each other very modestly, and in the presence of evidence that symptoms within a PD may stem from different causes, it does not seem advisable to begin with this assumption. To use prototypes is to reify diagnoses that involve combinations of multiple, different psychological processes with no identified common cause. When PD research is considered from the standpoint of basic principles of theory and measure validation, there is not a sufficient basis for constructing a diagnostic system organized around discrete PDs. Accordingly, the construction of prototypes of discrete PDs is to perpetuate a fundamental problem with description of personality dysfunction.

It may be the case that the prototypal matching approach will be replaced by diagnostic criterion sets that include information concerning level of self dysfunction, level of interpersonal dysfunction, and a list of maladaptive personality traits (Siever, 2011). Of course, a decision to continue to use criterion sets preserves the problems of the syndromal approach without realizing the true benefits of dimensional trait model.

Description of Personality Dysfunction in terms of Dimensions of Personality Functioning

Fortunately, there is an alternative approach to describing personality dysfunction that is quite consistent with basic principles of theory validation and has proven useful to clinicians: It is the topic of this special section. When personality dysfunction is described in terms of basic personality traits, the problems with the current system largely disappear. A number of researchers have developed systems to describe personality dysfunction based on homogeneous dimensions of personality. The Dimensional Assessment of Personality Pathology-Basic Questionnaire (DAPP-BQ: Livesley & Jackson, in press) and the Schedule for Nonadaptive and Adaptive Personality (SNAP: Clark, Simms, Wu, & Casillas, in press) were both constructed to derive conceptually cohesive, homogeneous trait constructs that underlie the symptoms identified as relevant to personality dysfunction through the DSM system. Not surprisingly, the two systems overlap heavily (Clark, Livesley, Schroeder, & Irish, 1996). A number of other models have been developed in similar ways.

The NEO Personality Inventory-Revised (Costa & McCrae, 1995) representation of the five factor model of personality comes at the problem from a slightly different direction: the NEO PI-R has 30 traits that represent variation in normal personality functioning, and researchers have argued that personality dysfunction represents extreme and/or maladaptive variants of normal functioning, and so can be understood in terms of the five factor model (cf. Lynam, this issue; Widiger & Costa, this issue; Widiger & Mullins-Sweatt, 2009; Widiger & Trull, 2007).

This new approach represents a fundamental departure from the idea of using discrete syndromes for diagnosis. Instead, one assesses all individuals on a basic set of personality dimensions, so each patient can be described in terms of all aspects of personality relevant to his or her dysfunction. To the degree that the personality dimension measures do reflect homogeneous dimensions, i.e. meaningful, coherent psychological processes, scores on dimension measures can have clear meaning. Accordingly, the validation process becomes feasible: Researchers can study each individual dimension, with a clear sense of what psychological experience is thought to be represented by elevations on the dimension, and test hypotheses concerning the relationship between a given dimension and various indicators of life dysfunction. Similarly, descriptions of patients become clearer. This approach of departing from syndromes for diagnosis is also consistent with the DSM-5 proposal for a dimensional model of personality disorders. The dimensional approach for DSM-5 also clearly aligns with the domains presented for the five factor model of personality.

Descriptions of individuals along multiple dimensions of personality fundamentally change the relevance of the concept of comorbidity. The term comorbid is defined as “pertaining to two diseases which occur together ...” (Dictionary.com, 2011) or “existing simultaneously with and unusually independent of another medical condition ...” (Merriam-Webster Dictionary Online, 2011). In the traditional, syndromal system, a great deal of psychiatric comorbidity is artifactual, due to symptom overlap in purportedly distinct diagnostic categories (clearly disorders are not independent of each other if they have some of the same symptoms). In contrast, description of individuals along dimensions of personality will typically result in the recognition that there are multiple dimensions along which individuals experience some level of dysfunction; in that sense, this approach will result in a richer, fuller description of co-occurring problems when they are present. At the same time, though, the word “comorbid,” with its association with disease processes, may not be the most useful term for characterizing personality-based description. Many individuals will have, to varying degrees, elevations on multiple scales that reflect some dysfunction, but it does not seem accurate to describe such multiple elevations as co-occurring disease processes.

Not only is the validation process for describing personality dysfunction in terms of dimensions of personality feasible from a construct validity perspective, it is in fact well underway. In stark contrast to the lack of validity evidence for syndromal descriptions of personality dysfunction, there is considerable evidence for the validity of dimensional models of normal personality and dimensional models of personality dysfunction, as well as for the hypothesis that dysfunction represents extreme and/or maladaptive variants of normal personality functioning. Lynam (this issue) and Widiger and Costa (this issue) summarize much of this evidence. The impressive and rapidly growing body of validation evidence for dimensional models of personality dysfunction, and for the five factor model in particular, supports construct validity theorists’ argument that a focus on homogeneous dimensions of functioning facilitates the validation enterprise.

We briefly summarize examples of this validation research to illustrate the benefits of this approach. The structure of personality as represented in the five factor dimensional model has been replicated numerous times, in numerous languages, and across the major regions of the world (McCrae, 2002; Widiger & Costa, this issue; Widiger & Mullins-Sweatt, 2009). Personality disorders are defined as enduring, stable patterns of experience and behavior (American Psychiatric Association, 2000). Whereas longitudinal studies have failed to verify the stability of syndromal PD diagnoses (Livesley, 2005), dimensional personality structure has proven quite stable across the life span (Skodol, et al., 2005). There is clear evidence for the heritability of both broad personality dimensions and specific, homogeneous personality traits within the five factor model (Jang et al., 1998). Of particular importance, item response theory analyses relating the NEO PI-R and measures designed to assess personality pathology, i.e., the DAPP-BQ (Livesley, & Jackson, in press) and the SNAP (Clark, 1993) have found that the three measures share latent constructs, with the NEO PI-R providing better discriminative information in the normal range of personality and the DAPP-BQ and SNAP providing better discriminative information in the abnormal range of personality (Samuel, Simms, Clark, Livesley, & Widiger, 2010). This finding provides direct support for the theory that personality pathology falls on the same dimensions as normal personality, but represents extreme or maladaptive variants of normal traits.

Strikingly, researchers who developed homogeneous trait measures of maladaptive functioning and those who developed homogeneous measures of adaptive functioning have reached a consensus on how to describe personality dysfunction (Widiger, Livesley, & Clark, 2009). There is an impressive convergence of evidence speaking to the validity of this consensual approach (Widiger & Costa, this issue; Widiger et al., 2009). Clearly, this

alternative model of personality dysfunction that relies on homogeneous dimensions of functioning and that views dysfunction in relation to normal personality functioning has led quickly to well validated descriptions of dysfunction.

The Utility of Dimensional Description

So far in this article, we have focused on validation principles and argued that description of personality dysfunction in terms of homogeneous dimensions facilitates valid measure development and valid theory tests in ways that the classic syndromal approach does not. In light of the impressive evidence for the validity of the dimensional approach, it is important also to give consideration to its utility: are dimensional descriptions useful for clinicians? Mullins-Sweatt and Lengel (this issue) address this issue in depth. In this article, we briefly touch on utility concerns because a thorough discussion of the validity of applied measures should include consideration of such measures' usefulness in practice.

Certainly it is true that shifting to dimensional description would be disruptive in important ways: It would require retraining, cause administrative problems, complicate record keeping, change the focus of some research, and make it harder to apply past research to current clinical care (First, 2005). Of course, as First noted, if even this disruptive a change provided improved validity and applicability, it might well be worthwhile. It seems clear that the improvement in validity from this change is likely to be quite large; indeed, in order to describe dysfunction in coherent, meaningful terms, the move to dimensional description is necessary.

One interesting line of research offers a means to ease the transition from discrete syndromes to homogeneous dimensions. Miller (this issue) and Miller et al. (2010) use a prototype approach to provide evidence that one can translate dimensional description of personality dysfunction back to DSM-IV PD constructs. The capacity to do so is important: It helps clarify the relevance of past research to current dimensional description, helps clinicians familiar with traditional PD constructs make the transition to the dimensional, and incorporates explicitly the underlying traits into the description of the syndrome. However, over time, as researchers and clinicians become familiar with the dimensional approach, we believe the syndromal and prototypal matching approaches will become obsolete and professionals will prefer more precise description at the trait level. We next briefly note the value of improved validity for parsimony and comprehensiveness; we then consider the utility of a dimensional system for treatment planning.

Parsimonious Description of Dysfunction

Rather than use numerous different DSM-IV-TR (or DSM-5) disorders to reflect all aspects of a patient's symptom picture, researchers and clinicians can describe dysfunction in terms of four or five basic personality dimensions and their facets (Widiger et al., 2009; Widiger & Simonsen, 2005). Patterns of deviation across well-validated personality traits can be used to describe the full range of dysfunction for which one currently needs multiple, separate PD categories. We believe there is an advance in parsimony from identifying basic dimensions of personality dysfunction rather than by delineating the presence or absence of multiple different syndromes.

Comprehensive Coverage of Personality Dysfunction

At the same time as dimensional description provides parsimony, it also provides comprehensive descriptive capacity. Samuel and Widiger (2006) had clinicians evaluate classic clinical cases, including that of Ted Bundy (who was not identified to the clinicians), using both DSM-IV-TR criteria and the 30 traits of the NEO PI-R representation of the five factor model of personality. Not surprisingly, 96% of clinicians diagnosed Bundy with

antisocial personality disorder, and 80% considered his case prototypic of that disorder. However, 95% of the clinicians using this system also diagnosed Bundy with narcissistic personality disorder, with 44% considering him to also be a prototypic case of narcissistic PD. Dimensional diagnosis, using the NEO PI-R, appears to have produced a more comprehensive description of his personality: he was described as lacking normal anxiety, self-consciousness, vulnerability, and warmth; as being nontrustworthy, not straightforward (i.e. manipulative and deceptive), not altruistic (i.e. exploitative), not compliant (i.e. aggressive), and not modest (i.e. arrogant); and as unusually low in tender-mindedness (i.e. callous). He was rated as unusually high in angry hostility, assertiveness, activity level, and excitement seeking, competence, order, and achievement striving. In fact, being high on these last three facets, competence, order, and achievement striving, does not fit the description of a prototypal psychopath (Mullis-Sweatt et al., 2010). This finding is not surprising: When clinicians are not constrained by the need to place individuals into discrete categories, they are free to describe patients across more dimensions, and hence in more depth.

Utility for Treatment Planning

One central function of any diagnostic system is to guide clinicians in choosing appropriate, effective treatments. Recent decades have seen the development of numerous empirically supported treatments for many forms of psychological dysfunction (Chambless & Ollendick, 2001). Interestingly, many well-validated treatment protocols share components with each other (Combs, Spillane, & Smith, in press). For example, many interventions involve cognitive restructuring, or relaxation training, or emotion regulation skill development, or distress tolerance, or behavior activation, or interpersonal effectiveness, and so on (Combs et al., in press). It is of course the case that treatment protocols are validated with respect to their usefulness in treating patients, based on patients' DSM-IV-TR diagnoses. Because so many diagnoses share symptoms, it is not surprising that treatments validated for different disorders have very similar components.

Clinicians treat patients based on the symptoms they exhibit, not based on the disorder with which they have been diagnosed (Livesley, 1998). For example, one patient diagnosed with depression, who is experiencing anhedonia, is likely to benefit from behavior activation therapy (Dimidjian et al., 2006). Another patient, also diagnosed with depression but who is not anhedonic, and is instead experiencing a great deal of sadness, might well benefit from cognitive behavior therapy (DeRubeis, et al., 2005). This reality speaks directly to the value of describing patient dysfunction in terms of homogeneous dimensions of functioning, because doing so leads directly to treatment choice.

Interestingly, there is a relative absence of empirically validated treatments for personality disorders (with the partial exception of DBT for BPD; we elaborate below). This absence may, in part, be another consequence of defining disorders that are heterogeneous combinations of symptoms (Widiger, Samuel, Mullins-Sweatt, Gore, & Crego, in press). When a disorder is characterized by multiple different dimensions of dysfunction that are modestly related to each other and that have, at least in part, different causes, it does not seem possible to fashion a treatment for the disorder as a whole. Instead, one must develop treatments tailored to specific domains of functioning. A good example is provided by research on DBT, which is often described as a treatment for BPD. In reality, DBT has been shown to be an effective intervention for self-harm and angry hostility, but not for hopelessness or other BPD symptoms (Scheel, 2000).

A five-factor based description of dysfunction can have several important treatment implications (see Widiger et al, in press). For example, individuals with elevations on scales within the Agreeableness and Extraversion domains reflect dysfunction concerning

interpersonal relatedness may be best served by therapy based on interpersonal models (Horowitz & Wilson, 2005). In contrast, individuals with elevations on scales within the Neuroticism domain are experiencing dysfunction related to mood, anxiety, and emotional dyscontrol; pharmacological interventions involving antidepressants, anxiolytics, or mood stabilizers, in conjunction with cognitive behavioral therapy, are likely to be more suitable for them. Individuals with elevations on scales within the Openness domain might be experiencing cognitive-perceptual aberrations and benefit from neuroleptics, and so on. Crucially, intervention research would be tied to specific, identifiable, homogeneous domains of functioning; such a system would likely accelerate the rate of growth of empirically supported treatments.

Combs et al. (in press) conducted a review of all symptoms represented across most disorders in DSM-IV-TR and a parallel review of all components of empirically validated psychological treatments for DSM-based disorders. They found a striking convergence between the two: They identified 22 basic dimensions of dysfunction across the DSM-IV-TR, and a set of 10 categories of treatment components within all empirically validated treatments; each of the 10 treatment components could be mapped onto dimensions of dysfunction. The simplicity and usefulness of their system was apparent: For any given type of dysfunction, one can identify treatments that have proven effective with that dysfunction. Dimensional description of psychopathology, and of personality pathology, is more useful than the syndromal approach because it more effectively guides treatment planning.

Determining Whether Clinical Dysfunction Exists

One concern with the possible use of a model that describes individuals along multiple dimensions of functioning is that there are still dichotomous decisions that need to be made. For example, some individuals will be determined to merit insurance reimbursement for their treatment and others will not. In our view, the determination of whether a clinical disorder is present involves the determination of whether an individual is experiencing significant impairment in his or her social, emotional, and occupational functioning. Determination of a disorder should not be based on symptom counts, because it is the end product of symptoms, impairment in life functioning, which truly matters. Ro and Clark (2009) proposed that clinicians measure impairments in functioning with as little confound from the measurement of psychopathology as possible, in order to assess the presence of clinical impairment separately from evaluating psychological functioning. As they noted, only by measuring the two domains separately can one inquire into the ways in which impaired life functioning relates to dimensions of psychological functioning. Life impairment can be, in part, a product of psychological factors, and it is that product—disrupted ability to manage necessary life functions—that determines whether a clinical disorder is present. We agree with Ro and Clark's (2009) call for advances in assessing psychosocial impairment. Mullins-Sweatt and Widiger (2009) provide an illustration of this approach from the perspective of the five factor model.

It is certainly the case that the assessment of impairment has been an ongoing concern in diagnostic systems, so examples of how to do so already exist. As Widiger and Trull (2007) note, the Global Assessment of Functioning (GAF) scale provided on Axis V of the DSM-IV-TR (American Psychiatric Association, 2000), can be used to determine if a significant level of impairment is being experienced by an individual. The GAF is based on social, emotional, or occupational impairment. For example, a score of 71 or above indicates a normal range of functioning: Absent or minimal symptoms (e.g., difficulty concentrating after family argument), and no more than slight impairment in social, occupational, or school functioning (e.g., temporarily falling behind in schoolwork). A score of 60 or below represents a clinically significant level of impairment (moderate symptoms or difficulty in

social, occupational, or school functioning, such as few friends, conflicts with peers, flat affect, circumstantial speech) to severe impairment (such as danger to self or others, suicidal ideation, violent behavior, hallucinations, delusions: American Psychiatric Association, 2000). Ro and Clark (2009) note that the World Health Organization's (WHO) International Classification of Functioning, Disability, and Health (WHO, 2001) was also developed to assess impairment of functioning, and they note that it is less confounded with psychopathology than is the GAF. Advances in assessing impaired functioning are well underway (Mullins-Sweatt & Widiger, 2010; Ro & Clark, 2009) and are likely to facilitate determination of clinical status in increasingly valid ways.

Summary

Classic, syndrome-based diagnosis of PDs has not met standards for valid description of personality dysfunction. To date, there is no evidence for an underlying cause for any of the putative syndromes, and symptoms within the syndromes covary modestly and often do not have the same cause. Continued endorsement of a syndrome approach, whether through symptom counts or prototypes, is unlikely to bear the fruit of improved accuracy and utility of diagnosis. In contrast, description using homogeneous dimensions of functioning has clear potential for more valid assessment; indeed, it is already working quite well in the field of personality dysfunction (Widiger & Costa, this issue). Describing individuals along basic dimensions of personality functioning makes use of well-validated constructs from the personality literature, it makes concerns about artifactual comorbidity obsolete, it provides both parsimonious and comprehensive description, and it leads directly to treatment planning. Dimensional description should guide future research and clinical activity.

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