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CONTINUITY OF AXES I AND II: TOWARD A UNIFIED MODEL OF PERSONALITY, PERSONALITY DISORDERS, AND CLINICAL DISORDERS

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Abstract

In the current standard psychiatric nomenclature, the DSM–IV–TR (APA, 2000), mental disorders are divided into two groups: Clinical Disorders (CDs) and Personality Disorders (PDs), and CD and PD diagnoses are recorded on two separate axes (Axes I and II, respectively). This article considers evidence regarding putative bases for distinguishing between CDs and PDs, and finds that these constructs are more similar than distinct. Links between the domains may be better understood by focusing on how personality connects CDs and PDs. This perspective underlines the need to work toward a more unified model of personality, PDs, and CDs in research and in future editions of the DSM.

In the DSM–IV–TR (APA, 2000), mental disorders are divided into two groups: Clinical Disorders (CDs) and Personality Disorders (PDs). CDs are recorded on the first of the five axes used to classify patients, and PDs are described on the second of those five axes. The DSM–IV–TR describes this arrangement as linked to the importance of considering the possible presence of a PD even when a potentially more florid CD is present. In addition, the DSM–IV–TR is careful to note that the separation of mental disorders into CDs and PDs need not imply fundamental distinctions in terms of “pathogenesis or range of appropriate treatment” (p. 28) for PDs and CDs.

The purpose of the current article is to consider this arrangement in terms of both theory and data, in an attempt to inform the construction of future editions of the DSM. Shall we continue to separate mental disorders into CDs and PDs? Or has the time come to reconsider this arrangement, in light of recent research developments? In this article, I will argue that research on the link between personality and psychopathology indicates a closer connection between CDs and PDs than is implied by their separation between two axes. Although the DSM–IV–TR is careful to avoid overstating distinctions between CDs and PDs, the separation of mental disorders across Axes I and II continues to influence the way these disorders are often conceived of in both research and clinical practice. That is, CDs and PDs are often conceived of as more distinct than they actually are, at least based on current evidence.

In addition, I will argue that the closeness of the connection between CDs and PDs can be understood when viewed through the prism of general personality functioning. Basic research in personality and individual differences provides a framework that is very useful in understanding psychopathology. Integration of this basic research with research on constructs from the DSM provides an important avenue for linking heretofore somewhat distinct

literatures, as well as for helping to link science and practice. Indeed, the focus of this conference on dimensional models, many of which derive from basic research in personality and individual differences, is a strong sign that a more integrated perspective on basic personality constructs and DSM constructs is both desirable and feasible.

The current article begins by considering the state of the evidence regarding putative bases for distinguishing between CDs and PDs. I then turn to review evidence regarding connections between constructs from basic personality research and PDs (i.e., the joint structure of normal and abnormal personality). The article concludes by discussing how these constructs are also connected to CDs, and outlining how these lines of evidence could be brought together under a more comprehensive model of personality and psychopathology.

CLINICAL AND PERSONALITY DISORDERS: EVIDENCE REGARDING EMPIRICAL AND CONCEPTUAL SIMILARITIES

A variety of characteristics could form the basis for distinguishing between CDs and PDs. Here, these characteristics are divided into six broad areas: stability, age of onset, treatment response, insight, comorbidity and symptom specificity, and etiology. The evidence for each of these characteristics as a means of distinguishing between CDs and PDs will be reviewed. In addition, future research directions in each of these areas will be suggested.

STABILITY

A major putative basis for distinguishing between CDs and PDs pertains to differences in stability. PDs are conceptualized as relatively more stable over time when compared with CDs.

One way of conceptualizing stability is in terms of specific, categorical, DSM-derived diagnoses of PD. From this perspective, a lack of stability for a specific PD would be linked to the concept of remission of that PD. In turn, remission could be defined in various ways. For example: would decline below a threshold number of symptoms (e.g., the number of symptoms needed for a DSM diagnosis) be sufficient to claim remission? Is remission of a PD conceptually parallel to recovery from a CD? How might recovery/remission of CDs and PDs be compared directly?

Shea and Yen (2003) recently tackled these challenging issues in a direct head-to-head comparison of the stability of PDs, mood disorders, and anxiety disorders, using data from comparable studies of the stability of these disorders in comparable clinical samples. Shea and Yen (2003) defined remission/recovery for the three broad classes of disorder as a period of 8 weeks when no more than two criteria were present. At 2-year follow-up, remission/recovery of mood disorders was fairly typical, remission/recovery of anxiety disorders was relatively rare, and remission/recovery of PDs was in between mood and anxiety disorders. This picture was somewhat complicated by the observation that, although remission/recovery was typical for mood disorders, relapse was also common. Nevertheless, stability, in the sense of likelihood of remission/recovery using a comparable categorical conceptualization of disorder, does not appear to reliably distinguish PDs and CDs in this direct, head-to-head comparison. Particularly striking was the evidence for the relative stability of anxiety disorders, such as Social Phobia and Post-Traumatic Stress Disorder, which were unlikely to remit at 2-year follow-up.

Personality pathology can also be conceptualized dimensionally, an approach that circumvents some of the issues inherent in defining remission/recovery. From this perspective, one would ask how highly correlated PD symptom counts are across time, as opposed to asking what proportion of the sample achieved remission/recovery. One could also ask how this perspective on stability might compare with a perspective derived from a categorical conceptualization of

personality pathology within the same data. This approach was taken by Shea et al. (2002) in an analysis of data from the Collaborative Longitudinal Study of PDs (CLPS). The stability of Schizotypal, Borderline, Avoidant, and Obsessive–Compulsive PDs was examined at 6-month and 1-year follow-up of the participants, most of whom were patients recruited from clinical settings. By the time of the 6-month follow-up, many participants no longer met criteria for PD, and by 1 year, more than half the participants no longer crossed the diagnostic threshold for PD (with the exception of Avoidant, where 56% of participants continued to meet criteria). However, when the number of criteria met was correlated across the 3 waves (intake, 6-month follow-up, and 1-year follow-up), the correlations were uniformly very high for all 4 varieties of personality pathology (.84–.92). A reasonable interpretation of these findings is that people may fluctuate around a general level of personality pathology over time (such that some fall below or above a specific threshold at a specific time), yet this phenomenon masks a general, rank-order stability in personality pathology over time.

Importantly, significant levels of rank-order stability have also been reported for both self-report and semi-structured interview indices of the full range of dimensions of DSM-defined personality pathology over the college years (Lenzenweger, 1999), suggesting that the stability of PDs transcends the four disorders that were the target of the CLPS. These recent findings on the full range of DSM-defined PDs are a particularly important contribution to our understanding; a comprehensive review of the stability of PDs published as recently as 1996 noted that “when examining the degree of stability of a specific PD, previous research focuses primarily on Borderline PD. Because of a lack of information, very little can be said about other individual PD diagnoses” (McDavid & Pilkonis, 1996, pp. 7–10).

In addition to the report from Lenzenweger (1999), a variety of other recent reports using diverse samples also demonstrate the general stability of a broad range of PD symptom counts over longer periods of time and across life transitions. The full range of DSM PD dimensions were moderately stable between intake and 30-month follow-up of a sample of depressed adult outpatients (Ferro, Klein, Schwartz, Kasch, & Leader, 1998), and between intake and 2-year follow-up in a sample of gay adult men (Johnson et al., 1997). Moderate levels of rank-order stability from adolescence to adulthood, for at least some PD dimensions, have also been observed in clinical (Grilo, Becker, Edell, & McGlashan, 2001) and community (Johnson et al., 2000) samples. Moderate levels of rank-order stability of specific PDs have also been observed in spite of variation in the specific measure employed. For example, a variety of BPD measures were shown to be moderately stable over 2 years in an undergraduate sample (although self-report measures were generally more stable than the interview-based measure used in this research; Trull et al., 1998).

Rank-order stability can also be examined at the level of latent variables underlying observed, or “manifest,” indices of psychopathology. The term “latent variable” refers to the systematic variation linking a set of manifest variables; latent variables represent what a set of manifest variables have in common. In this way, latent variables capture systematic variation in common among multiple indicators of psychopathology, and are less prone to contamination with unsystematic sources of variation, such as measurement error. A recent report from the CLPS took this approach to examining the stability of Schizotypal PD, Borderline PD, Avoidant PD, and Obsessive–Compulsive PD from intake to both 1-year and 2-year follow-up (Warner et al., 2004). All four disorders were highly stable across these assessment points, with rank-order stability coefficients ranging from .60 to .90. These findings indicate that the latent propensities to experience the symptoms of the four PDs that were the targets of the CLPS study are quite stable.

However, the question of the relative stability of PDs versus CDs remains. The propensities to experience PD symptoms may be stable, but are these propensities more stable than those

that underlie CDs? Krueger, Caspi, Moffitt, and Silva (1998) examined both the structure and stability of latent propensities to experience common CDs in a longitudinal study of a birth cohort, from age 18 to age 21. At both ages, unipolar mood and anxiety disorders were found to be indicators of a latent “internalizing” propensity, and substance dependence and antisocial behavior disorders were found to be indicators of a latent “externalizing” propensity. Both propensities were highly stable, with stability coefficients in the same range as those reported for latent PD variables in the CLPS (.69 for internalizing and .86 for externalizing). These findings were replicated and extended by Vollebergh et al. (2001), who reported very high stabilities for latent CD propensities (.85–.96) across a 1-year period in the Netherlands Mental Health Survey and Incidence Study (NEMESIS), an epidemiological study of Dutch adults. Thus, latent propensities to experience both PDs and CDs appear to be highly stable.

In sum, the general picture that emerges from research on the stability of PDs is that the underlying factors that give rise to manifest PD symptomatology are generally consistent over time. Nevertheless, the underlying latent factors that give rise to common CDs also appear stable. Thus, stability is not an especially compelling differentiator of PDs and CDs, particularly when transient and unsystematic sources of variation in psychopathology are controlled for by using latent variable models.

Nevertheless, ongoing methodological and conceptual developments are certain to continue to inform our understanding of the nature and meaning of stability and change. For example, although rank-order stability can be demonstrated with data from two waves of a longitudinal study, more waves of data allow for a more nuanced understanding of stability and change in psychopathology. Along these lines, Lenzenweger, Johnson, and Willett (2004) recently reported a three-wave longitudinal study of PD features in which they found notable individual differences in patterns of stability and change in PD features. This suggests that, in the context of general stability in psychopathology over time (cf. Lenzenweger, 1999), there are, nevertheless, meaningful individual difference in patterns of stability and change that may provide new clues to understanding the natural history of psychopathological variation. Indeed, issues of stability and change are fundamental and evolving topics in the study of personality in general. For example, David Watson, the first president of the newly formed Association for Research in Personality, focused his presidential address on the study of personality stability (Watson, 2004). Watson (2004) pointed out that even apparently minor differences, such as differences in wording, instructions, and response formats can affect conclusions about stability. Incorporating these developing methodological insights into studies of the comparative stability of both CD and PD constructs in multi-wave data should substantially enhance our understanding of the natural history of personality and psychopathology.

AGE OF ONSET

The DSM-IV-TR specifies that the characteristics of a PD should appear before early adulthood. Along with stability in course, early age of onset is a fundamental basis for the putative distinction between CDs and PDs in the DSM-IV-TR, and is cited therein as a basis for the differential diagnosis of CDs versus PDs (APA, 2000, p. 688). Nevertheless, as noted in recent comprehensive reviews (First et al., 2002; Widiger & Clark, 2000), we lack a thorough understanding of the childhood antecedents of adult personality disorders. The exception to this is Antisocial PD, where the childhood onset of the disorder is definitional, in the sense that Conduct Disorder (a CD in the DSM-IV-TR) must be present before age 15 for an individual to meet full criteria for Antisocial PD.

Somewhat ironically, this requirement may be more problematic than enlightening about the nature of Antisocial PD. For example, the Conduct Disorder aspects of the Antisocial PD diagnosis show etiologic contributions from the family environment, whereas the adult aspects do not show these contributions, suggesting that the Antisocial PD diagnosis mixes entities

with distinct etiologies that might be better studied separately (e.g., Krueger et al., 2002). Along these same lines, the current DSM-IV-TR nomenclature lacks a diagnosis for persons who meet the adult criteria for Antisocial PD but do not meet the Conduct Disorder criteria. The only related entity is the code “V71.01 Adult Antisocial Behavior,” but this is conceptualized as “behavior that is not due to a mental disorder” (APA, 2000, p. 740). This conceptualization seems incompatible with literature showing that people who meet criteria for the adult aspects of Antisocial PD, but not the Conduct Disorder aspects, are not uncommon, and that such persons have personality profiles that resemble those of persons who meet full Antisocial PD criteria (Elkins, Iacono, Doyle, & McGue, 1997). In sum, in the single DSM-IV-TR PD that officially mixes childhood antecedents with adult symptomatology (Antisocial PD), the resulting mixture is problematic when evaluated in light of the relevant literature. That is, the emphasis on age of onset in the definition of PDs may be well-intended conceptually, but may actually inhibit progress by turning attention away from understanding how a specific domain (e.g., antisocial behavior) changes and develops over time.

Another implication of the DSM-IV-TR conceptualization of PDs as having an early age of onset is that CDs have a different typical age of onset (presumably later), such that knowing the age of onset of symptoms would help the clinician differentiate between CDs and PDs. However, data on the prevalence of common CDs in the population at large suggest that these disorders are actually most prevalent in younger people. For example, the National Comorbidity Survey (NCS) assessment concentrated primarily on common CDs in the broad categories of affective, anxiety, and substance use disorders. A wide age range was sampled (15–54), and analyses of the NCS data showed that past-year mental disorders were most prevalent in the youngest age group (ages 15–24; Kessler et al., 1994). Moreover, generally speaking, the prevalence of past-year mental disorders declined with age in the NCS (Kessler et al., 1994). This indicates that common CDs are most likely to be encountered in adolescents and younger adults, compared with older persons. Interestingly, existing epidemiological data on PDs also indicate that “PDs tend to favor youth and rates of PDs may decline with age” (Mattia & Zimmerman, 1999, p. 118; see also Ekselius, Tillfors, Furmark, & Fredrikson, 2001; Jackson & Burgess, 2000).

Thus, age of onset appears inadequate to differentiate CDs from PDs, in the sense that both sorts of disorders appear to be prevalent in younger persons. Moreover, PDs have been studied in conjunction with CDs in adolescence as predictors of young adult PD, and the likelihood of a PD in young adulthood is enhanced in the presence of comorbidity between CDs and PDs in adolescence (Kasen, Cohen, Skodol, Johnson, & Brook, 1999). This suggests an intertwining of CDs and PDs throughout the course of pre-adult development that is incompatible with the idea that CDs and PDs are developmentally distinctive, with markedly different ages of onset.

The most compelling focus for future research will likely stem from taking a life-course developmental perspective on the origins, course, and prognosis of CDs, PDs, and their interrelations. This perspective has been recently outlined by Paris (2003) in his recent book on the course of personality disorders over time. As noted in reviews, the perspective in this book represents an important change for the field, from a cross-sectional focus on manifest symptomatology, to a longitudinal focus on how PDs develop over time (see, e.g., Livesley, 2003). In the context of this perspective, Paris (2003) describes aspects of an “ideal prospective strategy” for studying the development of PDs. Such a strategy would involve a large community sample, an ability to distinguish between genetic and environmental factors and to study their interactions and correlations (e.g., through studying twins and measured genotypes), beginning as early as feasible in development, measuring environmental factors both within and outside the family directly, and studying outcome across multiple domains and across a wide range (e.g., studying both personality traits and diagnoses *per se*). Such an undertaking is obviously not trivial, and requires an unusual level of commitment from both the relevant

investigators and funding agencies. Nevertheless, this undertaking is crucial to filling the gaps in our knowledge of the development of PDs identified in recent reviews (First et al., 2002; Widiger & Clark, 2000). This endeavor could be rendered more feasible by adding PD assessments to the follow-up waves of existing longitudinal, community-based studies.

TREATMENT RESPONSE

PDs and CDs may also be distinguished by the efficacy of treatment for these conditions. There has been a historical presumption that PDs are less amenable to treatment when compared with CDs. There has also been a related historical presumption that PDs call for psychotherapeutic intervention, whereas CDs call for psychopharmacologic intervention. These presumptions are insufficiently nuanced in the face of current evidence. Few would argue that PDs are straightforward to treat or respond readily to easily implemented interventions. After all, PDs are defined in such a way as to be ingrained aspects of a person's behavior, affect, and cognition that are fundamentally problematic for the person and/or society, as well as for the essential ingredients of intervention, such as the ability to establish a good working relationship. For example, being uncooperative is a significant predictor of the presence and extent of PD symptomatology (Mulder, Joyce, Sullivan, Bulik, & Carter, 1999), and effective intervention obviously requires the cooperation of the patient.

Nevertheless, psychotherapy can be effective in PDs (e.g., Perry & Bond, 2000; Piper & Joyce, 1999), as can pharmacotherapy (e.g., Markovitz, 1999, 2004). Progress in treatments for PDs was the focus of a recent special feature of the *Journal of Personality Disorders* (Livesley, 2004). The topics addressed in this special feature move beyond the basic issue of whether PDs can be treated, to topics such as the comparative efficacy of theoretically distinctive approaches to the psychotherapy of Borderline PD (Clarkin, Levy, Lenzenweger, & Kernberg, 2004) and the encouraging findings from recent psychopharmacological trials focused on PDs (albeit primarily Borderline PD; Markovitz, 2004).

In spite of these advances, it is also notable that the articles in this special feature focused primarily (almost solely) on Borderline PD, rather than the full range of DSM-defined personality pathology. In some ways, Borderline PD is a fundamental target for PD treatment research, given the high social impact of the disorder. However, in other ways, Borderline PD, as currently defined, is a complex target for intervention efforts, as it mixes more enduring personality features and more potentially circumscribed behaviors such as self-harming behaviors (cf. Tyrer, 1999). Given the fact that Borderline PD is a heterogeneous condition that also shows high comorbidity, it might be useful to reframe research on the treatment of PDs in terms of the treatment of specific symptom clusters and associated psychopathological processes that transcend putatively distinct PDs, to determine if the efficacy of such treatments extends beyond Borderline PD.

Careful examination of presumptions regarding the treatment implications of PDs is also essential in the context of CD-PD comorbidity. It has often been assumed that the presence of a PD will diminish the efficacy of an intervention for a commonly encountered focal CD. This issue has recently been reviewed for both mood and anxiety disorders. Mulder (2002) reviewed studies of the impact of personality pathology on outcome in the treatment of depression. In spite of the general belief that personality pathology is a negative prognostic factor in depression, Mulder (2002) found mixed support for this idea at best and noted that "the best designed studies reported the least effect of personality pathology on depression treatment outcome" (p. 359). Dreessen and Arntz (1998) examined the impact of comorbid PD on treatment outcome in anxiety disorders, and concluded that efficacy of intervention for anxiety disorder was not convincingly diminished by the presence of a comorbid PD. Thus, the idea that PD comorbidity in mood and anxiety disorders has negative prognostic implications, relative to "pure" CD presentations, is not well supported by these recent reviews.

Some putative CD treatments may also simultaneously be effective in reducing PD symptomatology. Fava et al. (2002) studied 384 outpatients selected for a fluoxetine trial, who met criteria for Major Depressive Disorder. At entry into the study, PDs were found to be present in the typical participant, as 64% of the participants met criteria for at least one comorbid PD. This finding is striking in itself, as it underlines the prevalence of PDs in clinical samples not selected based on the likelihood of showing personality pathology per se. In addition, the study demonstrated significant declines in PD symptomatology from the baseline to endpoint for 9 of the 12 DSM-defined PDs studied.

Thus, a distinction between CDs and PDs may not accurately reflect the way that pharmacologic intervention actually works, or the most optimal way to conceptualize such interventions in clinical practice. Pharmacologic interventions may affect core psychopathological processes that transcend the putative CD–PD distinction. Indeed, many psychopharmacologists conceptualize their interventions as targeted on specific psychopathological processes, regardless of whether these disturbances are coded as CDs or as PDs. A good example of this is the approach to intervention described by Soloff (1998). Rather than focusing on PD or CD constructs, Soloff (1998) outlined an algorithmic approach to psychopharmacology focused on three fundamental domains of psychopathology that transcend CDs and PDs: cognitive–perceptual aberration, affective dysregulation, and impulse–behavioral problems. This systemic approach (as opposed to a disease–focused approach) makes sense because psychopharmacologic agents appear to have effects that transcend not only the putative CD–PD distinction, but also the putative distinction between “normal–range” personality traits and mental disorder. Knutson et al. (1998) studied the effects of the SSRI paroxetine on the personality and behavior of medically and psychiatrically healthy volunteers across a 4–week period. Compared with controls, participants in the paroxetine condition exhibited a decrease in negative affect and an increase in affiliative behavior at the end of the 4 weeks that was linked to their plasma SSRI levels at the end of the study.

In sum, treatment response is not a compelling differentiator of CDs and PDs. DSM-defined mental disorders, including PDs, respond to various interventions, the presence of PD comorbidity in treating a CD may not result in diminished treatment efficacy, interventions aimed at CDs also affect PDs, and pharmacologic intervention can be conceptualized as influencing systems that transcend CDs, PDs, and “normal-range” personality. Thus, the continued division of mental disorders into CDs and PDs is more likely to reinforce misleading notions of differential treatment efficacy than to promote novel thinking about interventions for mental disorders in general. A more promising approach in future research might be to focus on how interventions impact core psychopathological processes that transcend CDs, PDs, and personality traits.

INSIGHT

PDs are often conceptualized as mental disorders in which insight is poor, whereas CDs are often presumed to be accompanied by enhanced insight compared with PDs. Put somewhat differently, PDs are typically assumed to be “ego–syntonic,” such that persons with a PD see the PD as part of their “natural selves,” and hence, have little insight into the fact that their PD may be causing distress for themselves and others. In comparison, the “ego–dystonic” symptoms of CDs are often presumed to cause obvious distress for persons, such that they would be likely to have the insight that something is psychologically amiss. Nevertheless, empirical studies of the role of insight in PDs are lacking. Interestingly, this does not seem to be due to a lack of the capacity to measure insight (see, e.g., Beck, Baruch, Balter, Steer, & Warman, 2004), nor does it seem due to a lack of interest in understanding the role of insight in mental disorders in general. For example, there is a nontrivial literature on insight in schizophrenia, which is conceptualized as a CD in DSM–IV–TR. Indeed, a recent meta–

analysis of 40 studies from this literature indicated that the extent of positive and negative symptoms was associated with less insight, whereas the extent of depressive symptoms in schizophrenia was associated with more insight (Mintz, Dobson, & Romney, 2003). This suggests a complex relationship between insight and psychopathological symptomatology that may not be fully captured by the patient's overarching DSM diagnosis.

The lack of literature on insight in PDs may be due to the presumption that PDs are accompanied by a lack of insight by definition, such that documenting the role of poor insight in PDs would be tautological. However, without data that can speak directly to this issue, the putative tautology remains little more than an assumption. Moreover, the empirical literature on insight in other mental disorders suggests that inclusion of both CD and PD symptomatology in studies of insight could be revealing about the nature of PDs. For example, Obsessive–Compulsive Disorder (OCD), a CD in the DSM–IV–TR, can be diagnosed with the additional specification of “poor insight.” A few studies have appeared in the literature that link this specification with PDs. In a study of persons meeting criteria for OCD, Avoidant PD was more common in the OCD persons with better insight, whereas Borderline and Narcissistic PDs were more common in the group with worse insight (Tuerksoy, Tuekel, Oezdemir, & Karalin, 2002). In addition, in a post-treatment follow-up study, OCD patients who continued to show poor insight even after treatment with a combination of clomipramine and cognitive–behavioral therapy were more likely to meet criteria for Schizotypal PD (Matsunaga et al., 2002).

To summarize, the literature is too sparse to draw firm conclusions regarding the role of insight in differentiating PDs and CDs, as a comprehensive study comparing the role of insight in a range of PDs and CDs is lacking. Nevertheless, there is a literature on the role of insight in CDs, most notably schizophrenia, and some studies of insight in OCD have connected variation in insight with variation in PD status. This suggests that instrumentation and ideas regarding the role of insight in CDs could be brought to bear on our understanding of PDs. Given evidence for variations in insight within DSM-defined CDs, insight may be best conceptualized as a separable dimension of variation that can intersect with psychopathology per se in potentially complex and revealing ways. It would be interesting, for example, to determine the extent of variation in insight within persons with statistically abnormal personality profiles. Indeed, the study of insight might provide some keys to differentiating personality traits and personality disorders. This distinction may simply be a matter of degree, or PDs may also be linked to the confluence of extreme traits and poor insight. Perhaps extreme traits by themselves are insufficient to capture the social and occupational dysfunction linked to the notion of “disorder,” at least as conceptualized in the DSM–IV–TR (see Spitzer & Wakefield, 1999 for a discussion of this issue). It may be that extreme traits are more likely to lead to social and occupational dysfunction when accompanied by poor insight. This hypothesis might be usefully compared with the hypothesis that PDs are simply the extremes of nomothetic personality traits. The point here is not about the comparative merits of these hypotheses; rather, the point is that such hypotheses can be generated and evaluated by contemplating the role of insight in PDs, rather than simply assuming that PDs are accompanied by poor insight by definition.

COMORBIDITY AND SYMPTOM SPECIFICITY

CDs and PDs might be distinguished by their relative independence and the distinctiveness of their symptoms. That is, it could be the case that CDs and PDs tend to occur separately and to have very different symptoms, as opposed to tending to occur together and sharing similar symptoms.

The phenomenon of disorders occurring together is typically subsumed under the rubric of comorbidity. Unfortunately, the term comorbidity is often used in a relatively imprecise manner. Often, the term is used to refer to the percentage of patients within a sample who meet

criteria for two disorders. For example, hypothetically speaking, an investigator might report that, “56% of patients in our sample met criteria for both Major Depression and Avoidant PD.” The problem with this usage is that such statistics are relatively uninformative about the relationship between the two disorders. In the hypothetical example, 56% might be regarded as excessive overlap, signaling a lack of distinction between Major Depression and Avoidant PD. However, this interpretation could easily be incorrect. It could be the case that the 56% figure resulted from high base rates of both Major Depression and Avoidant PD, as one might expect in clinical samples. If Major Depression and Avoidant PD co-occur simply due to chance, and both are present in 75% of the patients in the sample, then, by chance, one would expect 56% of the patients in the sample to meet criteria for both disorders (i.e., $.75 \times .75 = .56$).

For this reason, the independence of PDs and CDs needs to be evaluated using statistics that index the tendency for these disorders to co-occur at greater than chance levels, something the “% overlap” statistic cannot accomplish. A commonly used statistic for this purpose is the odds ratio. Using an odds ratio, one can ask: are the odds of a PD enhanced in the presence of a CD (or vice versa)? The answer is clear: PDs and CDs tend to co-occur at greater than chance levels (for reviews, see Bank & Silk, 2001; Dolan-Sewell, Krueger, & Shea, 2001; Tyrer, Gunderson, Lyons, Tohen, 1997). Oldham et al. (1995) examined this issue in a clinical sample across both CDs and PDs. This study was notable for the wide range of both CDs and PDs studied, and the most striking aspect of the findings was the overall extent of greater than chance co-occurrence across the full range of CDs and PDs, a finding that emphasizes the lack of distinctiveness between CDs and PDs. Nevertheless, some have expressed the concern that comorbidity might be artifactually higher in clinical samples because of a tendency for persons with more than one disorder to be more likely to seek treatment (a phenomenon called “Berkson’s bias”). Thus, it is important to also examine PD-CD co-occurrence in epidemiological samples. A recent study by Grant et al. (2004) addressed this issue in a very large sample carefully constructed to be representative of the civilian U.S. population, and focused on drug and alcohol comorbidity with PDs. Both alcohol and drug use disorders, and drug use disorders in particular, were associated with notably increased odds of meeting criteria for a DSM-defined PD. Other studies in community- and clinic-based samples also demonstrate significantly increased odds of CDs in persons with PDs (e.g., McGlashan et al., 2000; Samuels, Nestadt, Romanoski, Folstein, & McHugh, 1994). In sum, in both clinical and epidemiological samples, CDs and PDs appear to co-occur at greater than chance rates, a finding that challenges the idea that CDs and PDs are highly distinctive types of mental disorders.

CDs and PDs are also indistinct in the sense that they share similar symptoms. There are a number of classic nosological conundrums associated with this general issue. A perennial conundrum pertains to the (non)distinction between generalized social phobia (conceptualized as a CD in the DSM-IV-TR) and Avoidant PD. Indeed, the overlap with generalized social phobia is probably the primary topic in the study of Avoidant PD, according to a recent comprehensive review of the Avoidant PD literature (Alden, Laposa, Taylor, & Ryder, 2002). Alden et al. (2002) noted that the overlap question has been approached in a number of ways. For example, some studies have examined the extent to which patients receive both diagnoses, some have examined whether the diagnoses are associated with the same correlates (e.g., cognitive and behavioral features), and others have focused on the treatment implications of having one versus both of the diagnoses. The basic conclusions seem to be that the disorders may represent the same basic construct (social anxiety) at different levels of severity, a viewpoint that seems to point toward a more dimensional conceptualization of CD-PD connections in this domain.

Another interesting conundrum exists with respect to Depressive PD, which is listed as a criterion set in need of further study in the DSM–IV–TR. A literature has emerged dealing with questions of the construct validity of Depressive PD, and whether its symptoms are better classified as aspects of mood disorders (CDs) or a PD. Widiger (2003) described the discussion surrounding Depressive PD in the context of the development of the DSM–IV. According to his account, the basic hurdle to including Depressive PD in the DSM–IV was articulating its distinctiveness from early onset dysthymia (a CD in the mood disorders section of DSM–IV). Thus, the basic strategy of the DSM–IV PD work group was to emphasize symptoms that were more cognitive and behavioral in nature, and less somatic in nature (i.e., less like dysthymia symptoms). However, once these cognitive and behavioral symptoms were articulated, the mood disorders work group felt these symptoms were likely relevant to dysthymia, and proposed a revision to dysthymia that incorporated these more cognitive and behavioral symptoms. In the end, both Depressive PD and the alternative conceptualization of dysthymia were placed in Appendix B of DSM–IV–TR (criteria sets and axes provided for further study). The description of Depressive PD in Appendix B also notes that Depressive PD may overlap substantially not only with the revised dysthymia criterion set, but also with other proposed categories requiring further study, such as Minor Depressive Disorder, Recurrent Brief Depressive Disorder, and Mixed Anxiety–Depressive Disorder. A solution to these nosological conundrums might involve acknowledging that mood disorders and personality disturbances that involve mood (as well as the closely related syndromes currently classified as anxiety disorders) have both shared and distinctive features, such that conceptualizing this domain in terms of a set of distinct categories spread across two axes of the DSM, as well as an appendix, may not be the most fruitful strategy. A more fruitful strategy might focus on linking empirically based accounts of the nexus of mood–anxiety–personality with the existing DSM categories to determine points of overlap and divergence, in working toward a more integrated account of this domain in future editions of the DSM (Krueger & Finger, 2001; Mineka, Watson, & Clark, 1998; Watson, in press).

In sum, CDs and PDs tend to co–occur and they encompass related symptomatology. Thus, statistical independence and distinctiveness of symptoms are not realistic bases for distinguishing CDs and PDs. The examples described above (Avoidant PD–generalized social phobia and Depressive PD–dysthymia) are perhaps not surprising examples of CD–PD connections because they represent areas of shared phenomenology. Other examples along these lines could also be cited. For example, Schizotypal PD entered the DSM nosology because of its connections with the related CD of Schizophrenia (Siever, Bernstein, & Silverman, 1996). The question, then, is: should further effort be devoted to splitting up CDs and PDs by, for example, carefully drawing fine–grained distinctions, or would effort be better spent seeking the underlying factors that lead to the continuity of CD and PDs? It seems clear that it would be more useful to focus on articulating models of how and why these domains are so interconnected, as opposed to focusing on ways of further splitting apart CDs and related PDs.

ETIOLOGY

In theory, PDs and CDs could be distinguished by distinct etiologies. Historically speaking, there has been a tendency for clinicians to regard CDs as more genetic in etiology, and hence presumably more amenable to pharmacologic intervention, whereas PDs were regarded as more environmental in etiology, and hence presumably more amenable to psychotherapy (Gunderson & Pollack, 1985; Widiger, 2003). There are a number of ways in which this putative distinction makes little sense in the context of the current literature.

A compelling strategy for disambiguating genetic and environmental contributions to psychopathology is provided by behavior genetic methods. These methods rely on the known

genetic and environmental relationships among family members to parse genetic and environmental contributions to observed characteristics (“phenotypes”). Twins are especially informative relatives for these kinds of investigations because they come in two varieties that differ systematically in their degree of genetic relationship. Monozygotic (MZ) or identical twins are genetically identical, whereas dizygotic (DZ) or fraternal twins are as genetically related as typical siblings (i.e., they share on average 50% of their segregating genes). Unlike siblings, however, DZ twins are the same age, making them a better comparison group when contrasted against MZ twins, who are also the same age. Contrasting the similarity of MZ and DZ twins provides a means of estimating the influence of genetic contributions on phenotypes; if genetic factors influence a phenotype, then MZ twins would be more similar than DZ twins. The degree of genetic influence can be expressed as a percentage of the total variation in the phenotype, a statistic known as heritability.

The twin method has been applied to the study of personality pathology, and the results show clearly that personality pathology, like other forms of psychopathology, is significantly heritable (see Plomin & McGuffin, 2003 for a recent general review of the genetics of psychopathology, and Kendler, 2001 for a review focused on twin studies of psychiatric disorders). Livesley, Jang, and Vernon (1998) reported a twin study that established a number of fundamental conclusions regarding the etiology of abnormal personality. Twins completed the Dimensional Assessment of Personality Disorder – Basic Questionnaire (DAPP–BQ), an index of 18 primary personality disorder traits. Previous analysis of twin data on the DAPP–BQ demonstrated the importance of heritability in understanding variation in the DAPP–BQ scales (e.g., Jang, Livesley, & Vernon, 1998). Livesley et al. (1998) extended this observation in a number of directions. They first demonstrated that the genetic structure of the 18 traits closely resembled the phenotypic, observed structure of the 18 traits. Four broad genetic factors underlying the 18 traits were delineated: emotional dysregulation, dissocial, inhibition, and compulsivity. Importantly, these structures also map onto four of the broad domains within the Five-Factor Model of Personality (neuroticism, disagreeableness, introversion, and conscientiousness, respectively; Widiger, 1998). Thus, the genetic structure of abnormal personality closely resembles the phenotypic structure of abnormal personality (as delineated by the DAPP–BQ), as well as major features of the phenotypic structure of personality in general (as delineated in the Five-Factor Model).

In addition, Livesley et al. (1998) demonstrated that, controlling for the influence of the four higher–order factors on the variation in the 18 scales, significant heritable variation remained for the majority of the scales. That is, the four higher–order factors do not account entirely for the genetic aspects of the etiology of personality pathology. This speaks to the importance of hierarchy in understanding personality pathology. The higher–order genetic factors that undergird abnormal personality variation are important organizing influences, but they are not sufficient to account fully for the richness of the etiologic factors that impact on observed personality variation. Personality variation results from the interplay of both broad and specific etiologic factors, and both need to be taken into account in an empirically based approach to organizing abnormal personality.

In sum, CDs and PDs are not well distinguished in terms of the role of genetic factors in their etiology (see also Torgersen et al., 2000, for evidence of the heritability of the PD constructs described in the DSM nosology). Contrary to earlier speculation that personality pathology might be primarily environmental in nature, personality pathology, like other psychopathology, is significantly influenced by genetic factors. Nevertheless, genetic influences do not provide the entire story with respect to the etiology of psychopathology. The future of behavior genetic inquiry in this area lies in better understanding the interplay of genetic and environmental influences. The primary approach used in research to date separates genetic and environmental contributions to psychopathology in an additive fashion, such that genes and environments are

modeled as acting separately, with their influences “adding up” to predict psychopathology. More integrative models, in which genetic and environmental factors are correlated and interactive, are starting to be articulated and applied. For example, Johnson and Krueger (2005) demonstrated that the genetic influence on physical health was not a constant, but rather, was dependent on a person’s sense of control over their life, such that higher levels of control were associated with suppression of genetic variation in physical health. More integrative models may also be important in identifying the specific genes that underlie the heritability of psychopathology (e.g., Caspi et al., 2002, 2003). Accordingly, approaches that integrate genetic and environmental factors are likely to be fundamentally important in better understanding the etiology of PDs. For example, the environmental insult of abuse is commonly observed in the histories of persons with PDs (Paris, 2003; Trull, 2001), yet abnormal personality variation is also clearly heritable. Hence, the task for the next generation of behavior genetic studies of PDs involves putting these observations together and articulating models that integrate genetic and environmental contributions to the etiology of PDs (cf. Posner et al., 2003).

THE JOINT STRUCTURE OF NORMAL AND ABNORMAL PERSONALITY

PDs and CDs are not well distinguished in any of the ways reviewed above (stability, age of onset, treatment response, insight, comorbidity and symptom specificity, and etiology). The most promising general direction for research would therefore focus on understanding how and why the PD and CD domains are so interconnected. A better understanding of connections between PDs and CDs may be provided by a focus on how PDs and CDs are both connected to the structure of personality. A first issue, then, is how personality is connected with PDs. That is: What is the joint structure of normal and abnormal personality?

A bewildering array of personality constructs—as well as models for organizing those constructs—have been proposed to account for human personality variation. In addition, literatures on both normal and abnormal personality variation have evolved somewhat independently (Strack & Lorr, 1994). In spite of this, increasing evidence points to the feasibility of developing an empirically based model of personality that simultaneously incorporates both normal and abnormal variation (Trull & Durrett, 2005).

One approach to delineating such an integrative model focuses on points of intersection between various normal and abnormal personality models that have been articulated in the literature. Markon, Krueger, and Watson (2005) report two studies that take this approach. The first study focused on joint structural modeling of constructs measured by the DAPP (Livesley & Jackson, in press), the Eysenck Personality Questionnaire (EPQ and EPQ-R; Eysenck & Eysenck, 1975; Eysenck, Eysenck, & Barrett, 1985), the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1985), variants of the NEO-PI broad domain scales (NEO-PI, NEO-PI-R, and NEO-FFI; Costa & McCrae, 1985, 1992), and the Temperament and Character Inventory and its predecessors (TCI and TPQ; Cloninger, 1987; Cloninger, Surakic, & Przybeck, 1993). Markon et al. (2005) took a meta-analytic approach to assembling a matrix of correlations among the 44 scales derived from all of these inventories, combining data from 52 different studies. Structural modeling of the meta-analytically derived matrix yielded the following conclusions. First, the data indicated no more than five major factors underlying variation in the 44 scales. Second, these 5 factors (neuroticism, agreeableness, conscientiousness, extraversion, and openness) strongly resembled the factors of the Five-Factor Model (FFM) that has been suggested as a useful framework for understanding PDs (see Costa & Widiger, 2002). Nevertheless, additional analyses also supported the existence of meaningful factors above the level of the five factors. Specifically, the four-factor level resembled four-factor models often articulated in the personality and psychopathology literature (e.g., Livesley, Jang, & Vernon, 1998; O’Connor & Dyce, 1998; Watson, Clark, & Harkness, 1994), in that openness did not emerge as a separate factor, but combined with

extraversion in a broader dimension of positive emotionality. The three-factor level resembled the three-factor models of theorists such as Clark and Watson (1999), Eysenck (1994) and Tellegen (1985), with dimensions of negative emotionality (neuroticism), disinhibition (a combination of disagreeableness and unconscientiousness), and positive emotionality. Finally, the two-factor level resembled the two-factor model described by Digman (1997), with one factor (alpha) combining neuroticism, agreeableness, and conscientiousness, and the other factor (beta) combining extraversion and openness.

The second study presented in Markon et al. (2005) replicated this hierarchical model using a specific sample of participants (as opposed to a meta-analytic dataset) who completed a somewhat different set of instruments: the NEO-PI-R facet scales (Costa & McCrae, 1992), the EPQ-R (Eysenck & Eysenck, 1975; Eysenck et al., 1985), the Schedule for Nonadaptive and Adaptive Personality (SNAP; Clark, 1993), and the Big Five Inventory (BFI; John, Donahue, & Kentle, 1991). This suggests that the hierarchical model identified in the meta-analytic portion of the research can also be identified in a specific sample, and that the model is robust to variation in the instruments and scales used to measure personality.

The Markon et al. (2005) findings both reinforce and extend some basic conclusions about the structure of normal and abnormal personality. The findings indicate that normal and abnormal personality constructs can be integrated within the same structural model (see also O'Connor & Dyce, 2001). Rather than representing highly separate domains of human individual differences, normal and abnormal personality measures can be located in the same factor space. In addition, the findings speak to the importance of the FFM in understanding the joint structure of normal and abnormal personality. The FFM appears to represent the “base” of the higher-order structure that links normal and abnormal personality, as there was no compelling evidence for factors beyond the five. However, there was evidence for structures above the five, and these structures resemble major conceptions of personality that complement the FFM conception. This helps in integrating various models and addressing questions of which dimensional model might be most empirically compelling. One concern that is sometimes raised regarding conversion from the categorical model of PDs in the DSM to a dimensional model is the question of which dimensional model should be chosen (cf. Livesley, 2001). The Markon et al. (2005) results suggest that the FFM level of the hierarchy is the basic organizing framework of choice because there was no compelling evidence for higher-order structures beyond the five, and structures above the five can be understood as combinations of the FFM domains.

Nevertheless, it would be incorrect to conclude that the FFM level of the personality hierarchy provides all of the information necessary to capture personality variation (cf. Harkness, 1992). Structures above the FFM, as well as the facet-level scales that delineate the FFM domains, are also theoretically and clinically important. For example, the domain of disinhibition, which is above the FFM level and combines disagreeableness and unconscientiousness, is closely linked to antisocial behavior (e.g., Lynam & Derefinko, in press). In addition, many of the scales in the Markon et al. (2005) analyses contained substantial amounts of residual variance that could not be accounted for by the higher-order factors. For example, the DAPP Self-Harm scale was a marker of the broad neuroticism domain of the FFM (more so than of any of the other domains), but the majority of the variance in DAPP Self Harm was unique to this scale, and not accounted for by the FFM domains. Self harm is of obvious clinical importance and, inasmuch as it cannot be captured entirely by the broader neuroticism domain, it represents an example of a specific, facet-level construct below the level of the FFM that might be important to include in a complete system of abnormal personality description.

Indeed, the issue of which facet-level constructs should be included in a comprehensive system of abnormal personality description is an important topic for continued research and discussion.

One thing that seems clear is that the ability of the FFM to capture the clinical PD constructs described in the DSM is enhanced by a focus on the facet level of the FFM, as specific facets have differential relevance to specific DSM PDs (see, e.g., Table 6.1 in Widiger, Trull, Clarkin, Sanderson, and Costa, 2002). Although the Markon et al. (2005) analyses demonstrate that the facet-level scales in diverse inventories can be organized into the FFM domains, such analyses do not provide guidance regarding which facets are most optimal for clinical description, and the facet-level scales included in the inventories linked by the FFM domains remain distinctive. Consider the earlier example of self-harm, which is a facet in the DAPP but not, for example, in the NEO-PI-R. Some might argue that self-harm is too specific a construct, and might be collapsed into a broader personality facet, or that the DAPP Self Harm scale lacks sufficient variability to constitute a major facet-level construct (e.g., the scale was omitted from the analyses presented in Livesley et al., 1998, owing to low variability). Others might argue that the overwhelming clinical importance of self-harm justifies its inclusion as a facet-level construct in an official nosology. The point, however, is that this kind of discussion is vital in moving toward a consensus set of facet-level abnormal personality constructs suitable for inclusion in future revisions of the DSM. The detailed information about personality functioning contained in facet-level constructs is important in connecting broad domains of personality functioning with the richness of the clinical phenomena encountered in PDs (see Shedler & Westen, 2004 for a related perspective).

Developments in methodology can also aid in informing these discussions. For example, the general analytic approach to delineating the broad domains that organize personality variation (factor analysis) may not be well suited to delineating fine-grained distinctions between personality constructs; continued methodological development in this area is highly pertinent to better understanding the facet-level structure of abnormal personality (e.g., Bacon, 2001). In addition, many factor analysis models in current use are formulated such that they do not contain parameters describing the location of variables along latent dimensions. Typical models are capable of identifying the strength of the relationships between the variables and the factors (so-called “factor loadings”) but they are not formulated to also identify the locations of the variables along the factors. Models that also include location parameters have the potential to enrich our conceptualizations of personality and psychopathology. For example, Krueger et al. (2004) used this type of model to illustrate how alcohol problems can be well-conceptualized in a dimensional framework, as problems ranging from those that were normative to those that were pathological were found to lie along a dominant dimension of severity. Finally, dimensions of personality such as the FFM domains may delineate the space in which personality is best described, but there can still be points of greater and lesser density in this space, a possibility that can be investigated using new developments in the modeling of multivariate data (Muthén, 2002). These regions of greater density (if they exist) might be thought of as frequently encountered personality configurations, and some of these configurations might be of unusual clinical importance. For example, Hicks, Markon, Patrick, Krueger, & Newman (2004) used this kind of approach (a model-based cluster analysis) to demonstrate that prisoners identified as psychopathic by the Psychopathy Checklist – Revised (Hare, 1991) consisted of two groups with very different personality profiles. The first group resembled the “primary” psychopath, with unusually low stress reactivity, whereas the second group resembled the “secondary” psychopath, with unusually high levels of aggression.

In sum, the FFM appears to represent a compelling model for organizing normal and abnormal personality variation, including the variation captured by the PD categories described in the DSM-IV-TR (Costa & Widiger, 2002; Lynam & Widiger, 2001; Miller, Reynolds, & Pilkonis, 2004; Trull, Widiger, Lynam, & Costa, 2003; Warner et al., 2004). Further discussion and research could profitably focus on delineating the optimal facets for inclusion in official nosologies such as the DSM (even if these facets are well organized into the FFM domains), the ability of different facets to cover the entire range of normal and abnormal personality, and

the possibility that certain personality configurations occur more frequently than others, and might be of particular clinical importance.

TOWARD NOVEL MODELS OF PERSONALITY AND PSYCHOPATHOLOGY

Reconceptualizing PDs in personality dimensional terms represents a good start on providing a solid empirical footing for future editions of the DSM. Yet certain conundrums will remain even if PDs are reconfigured in personality trait terms. These conundrums pertain to CDs, as DSM-defined PDs, as well as dimensional personality traits, are both closely connected with CDs (for recent reviews, see Clark, in press; Krueger & Tackett, 2003). What is ultimately needed is a model that can make sense of the connections linking all these domains (CDs, PDs, and the structure of normal and abnormal personality).

The beginnings of such a model are provided by work on the structure of common mental disorders. The motivation behind this work has been to provide an understanding of the reasons why the mental disorders defined in official nosological systems such as the DSM and ICD are frequently comorbid. Rather than viewing comorbidity as an artifact or a nuisance, this work approaches comorbidity as a reliable empirical observation in need of an explanatory model.

The focus of this work has been primarily on comorbidity among mental disorders commonly observed in epidemiological samples (unipolar mood, anxiety, substance use, and antisocial behavior disorders); in the DSM nosology, these disorders are mostly conceptualized as CDs (with the exception of Antisocial PD). These disorders represent key targets because of their prevalence and clear public health relevance. Our earlier work on the link between personality and these disorders in epidemiological samples (e.g., Krueger, 1999a) yielded evidence of systematic links between “normal-range” personality constructs and these disorders, both cross-sectionally and longitudinally. Specifically, the mood and anxiety disorders were associated with high levels of neuroticism/negative emotionality, whereas the substance use and antisocial behavior disorders were associated with high levels of neuroticism/negative emotionality and high levels of disinhibition.

Given this pattern of findings, comorbidity among common mental disorders makes sense when thought of in terms of the personological underpinnings of these disorders. As described earlier, Krueger et al. (1998) demonstrated that unipolar mood and anxiety disorders were indicators of a latent internalizing propensity, and substance dependence and antisocial behavior disorders were indicators of a latent externalizing propensity (see also Krueger, 1999b). Moreover, these propensities were highly stable over time, a finding replicated by Vollebergh et al. (2001). Recent evidence indicates that the internalizing-externalizing structure can also be observed in the primary care setting in numerous countries around the globe, and that the internalizing spectrum also appears to encompass somatoform syndromes (Krueger, Chentsova-Dutton, Markon, Goldberg, & Ormel, 2003).

Putting the personality findings together with the findings on the structure of mental disorders, neuroticism/negative emotionality appears to provide the personological basis for internalizing psychopathology, and negative emotionality paired with disinhibition appears to provide the personological basis for externalizing psychopathology. Thus, the connections between personality and psychopathology make psychological sense. Negative emotionality is internalized given normative levels of disinhibition and presents as unipolar mood and anxiety disorder. If negative emotionality is paired with high levels of disinhibition, the presentation tends more toward externalizing (substance use and antisocial behavior) problems.

These observations provide the outlines of a hierarchically organized spectrum model of common mental disorders that also extends to encompass the link between personality and

psychopathology. The findings suggest that unipolar mood disorders, anxiety disorders, and negative emotionality form a coherent group of constructs, and substance use disorders, antisocial behavior disorders, and disinhibitory personality traits form a related (owing to the role of negative emotionality in both internalizing and externalizing problems) but also distinguishable (owing to the unique role of disinhibition in externalizing problems) group of constructs. This conceptualization is bolstered by research on the genetic underpinnings of the connections between psychopathological syndromes and personality traits. That is, unipolar mood and anxiety problems share significant genetic variance with the personality trait of neuroticism; in addition, substance use and antisocial behavior problems share significant genetic variance with an unconstrained, impulsive personality style (see Krueger & Tackett, 2003 for a review). This conceptualization is also bolstered by research on the genetic underpinnings of the structure of psychopathology.

Kendler, Prescott, Myers, and Neale (2003) recently presented a study showing that the observed phenotypic structure of common mental disorders closely mirrors the underlying genetic architecture of these constructs. Kendler et al. (2003) studied the genetic and environmental underpinnings of the comorbidity among seven syndromes (major depression, generalized anxiety disorder, phobia, alcohol dependence, other drug abuse or dependence, adult antisocial behavior, and conduct disorder) that delineate the internalizing and externalizing spectra. Two general genetic factors were found, the first related primarily to the internalizing disorders (major depression, generalized anxiety disorder, phobia) and the second related primarily to the externalizing disorders (alcohol dependence, other drug abuse or dependence, adult antisocial behavior, and conduct disorder). As noted by Kendler et al. (2003, p. 935), “These results suggest strongly that genetic factors are largely responsible for the pattern of comorbidity that results in the 2 frequently co-occurring clusters of internalizing and externalizing disorders.”

In addition to these broad genetic factors, Kendler et al. (2003) also documented specific etiologic contributions that distinguish disorders within the broad internalizing and externalizing spectra. For example, finer-grained modeling of the internalizing syndromes of major depression, generalized anxiety disorder, panic disorder, animal phobia, and situational phobia revealed evidence for genetically distinguishable (but correlated) subfactors within the broader internalizing domain, with one subfactor loading more on depression and generalized anxiety disorder (with panic disorder as a weaker marker), and the other loading more on animal phobia, and situational phobia (cf. Krueger, 1999b; Watson, in press). Specific genetic factors also contributed to alcohol and drug abuse/dependence, above and beyond the contribution of the overarching genetic externalizing factor. In addition, environmental factors were important contributors to distinguishing between closely related syndromes. For example, conduct disorder showed a unique contribution from the shared family environment (cf. Hicks, Krueger et al., 2004; Krueger, Iacono, McGue, & Patrick, 2002).

These specific etiologic contributions in the context of broad, genetically coherent spectra are important phenomena because they speak to the hierarchical organization of the structure of common mental disorders. The idea behind this work has never been that there are only two constructs of relevance in understanding common mental disorders (internalizing and externalizing). Rather, the idea is that the internalizing and externalizing constructs provide the broad organizational schema and sources of genetic coherence for this domain, and are therefore major sources of the comorbidity among common mental disorders; in addition to this, other constructs (e.g., unique genetic and environmental events) explain how syndromes closely connected by shared genetic etiology come to be distinguishable. Put somewhat differently, both “lumping” and “splitting” perspectives on the organization of psychopathology are partially correct, and they can be reconciled by adopting a dimensional-hierarchical model of etiologic contributions within this domain that recognizes etiologic

factors at continually varying levels of specificity versus breadth (cf. Krueger & Piasecki, 2002).

Integrating this ongoing work on the structure of mental disorders into the DSM system represents a complex challenge, but it is a challenge that seems worth pursuing if the goal is to place the organization of the DSM on solid empirical footing. One way in which the organizational structure of future editions of the DSM could reflect the empirical structure of mental disorders would be to organize the syndromes that have been studied to date into internalizing and externalizing sections. The organization of internalizing syndromes has been particularly problematic in the DSM (see, e.g., the previous discussion of Depressive PD). Watson (in press) has recently articulated an approach to resolving some of these problems. Watson's (in press) approach focuses on delineating facets within the internalizing spectrum in terms of the distinction between disorders that are more distress-related (e.g., Major Depression and Generalized Anxiety Disorder), those that are more fear-related (e.g., Panic Disorder and Phobias), and those that involve bipolarity of mood (e.g., Bipolar I and II, Cyclothymia). As described earlier, this reorganization reflects current knowledge of the structure of internalizing syndromes better than the putative distinction between mood and anxiety disorders (cf. Kendler et al., 2003; Krueger, 1999b).

Organizing externalizing syndromes into a coherent section also requires rethinking some basic aspects of the current organization of the DSM. The externalizing spectrum encompasses problems that are currently spread throughout the DSM, across sections covering substance-related disorders; disorders usually first diagnosed in infancy, childhood, or adolescence (conduct disorder); and the section on PDs (Antisocial PD). Yet empirical evidence continues to speak to the coherence of the externalizing spectrum, as well as the dimensional nature of this spectrum. For example, Krueger, Markon, Patrick, & Iacono (in press) studied the comorbidity among the syndromes of conduct disorder, adult antisocial behavior, alcohol dependence, marijuana dependence, and drug dependence in a large, representative sample of adults. A series of models were fit to the data to ask if the comorbidity among these syndromes could be better accounted for in terms of a set of categories versus in terms of a coherent dimension of liability to experience multiple disorders in the externalizing realm. The data better supported a dimensional conceptualization, with an overarching dimension of externalizing liability connecting the disorders. A model specifying five separate categories of substance disorders and antisocial behavior disorders was untenable.

Another challenging issue relates to disorders described in the DSM that have not been studied in the internalizing-externalizing framework. This framework provides only the beginnings of a comprehensive model of clinical psychopathology because many important forms of psychopathology have not been studied in its context. This owes primarily to the limitations of existing data. For example, data on psychotic disorders are collected in large-scale investigations of comorbidity, but these disorders are conceptualized in such a way that they have very little variance in the population at large. This makes studying relations between these disorders and common mental disorders infeasible because there are too few cases of less common disorders—much less observations of patterns of comorbidity linking less common disorders with more common disorders—to allow for reliable conclusions to be drawn regarding less common disorders.

Two strategies are likely to be productive in overcoming this obstacle to broadening the internalizing-externalizing framework. A first strategy would focus on integrating novel domains of psychopathology into the framework in samples where prevalence is enhanced (e.g., in samples from psychiatric clinics). For example, Krueger et al. (2003) were able to broaden the internalizing spectrum to include somatoform disorders by studying the model in primary care samples. A second strategy would focus on broadening conceptualizations of less

common syndromes. For example, rather than focusing on dichotomous CDs such as Schizophrenia, research could focus on dimensions of psychosis such as positive symptoms, negative symptoms, and disorganization. Emerging work on dimensional approaches to psychosis (e.g., van Os et al., 1999) could be integrated with ongoing work on the internalizing and externalizing dimensions that appear to underlie common mental disorders.

Another issue relates to the placement of personality constructs in a system that recognizes the internalizing and externalizing spectra, as these spectra transcend personality and psychopathology. Also recall that the foregoing review of PDs and CDs found them to be more similar than different. In particular, the major bases for distinguishing between PDs and CDs in the DSM-IV-TR (stability and age of onset) are not very differentiating, as the propensities underlying both PDs and CDs appear to be relatively stable, and both PDs and CDs seem to be prevalent in younger age groups. This suggests that it might make the most sense to ultimately focus not on the putative PD-CD distinction, but rather, on the generation of key facet-level constructs that cover the range of symptomatology and traits currently spread across Axes I and II of the DSM-IV-TR. This idea was well articulated by Widiger and Clark (2000, p. 954) who wrote, "Ultimately, as one builds toward DSM-V, what may emerge is a structured set not of categorical diagnoses but of component dimensions, a set of symptom-cluster building blocks from which the panoply of diagnoses could be constructed." Based on our current understanding, personality-related facets (building blocks) appear well organized by the FFM domains (although more work is needed to identify the most optimal facets for clinical purposes), and common psychopathological symptoms appear well organized by the internalizing-externalizing domains. With regard to the latter, the internalizing and externalizing domains have emerged from analyses of DSM categories, and hence, more work is needed that breaks these diagnoses down into their component symptom dimensions. As noted by Widiger and Clark (2000), models for the fundamental dimensions that constitute the internalizing domain continue to be developed (Mineka et al., 1998; Watson, in press), and efforts to delineate fundamental dimensions of externalizing are also underway (Krueger et al., in press).

Clearly, however, much work is needed before a reorganization of the DSM along the lines adumbrated here is possible. Thus, for the time being, some intermediate steps can be suggested. First, as we build toward the DSM-V, we should consider converting the existing PD section to a system of facet-level constructs organized by the FFM domains, and pursue research and discussion on the most optimal facets, as well as the most optimal cut points on those facets to distinguish between personality and its pathological manifestations (cf. Livesley, 2001). That is, adoption of a dimensional approach to the description of personality in the DSM does not mean abandoning a distinction between normality and abnormality. To the contrary, it sharpens discussions about how dimensions of symptoms and traits are related to concepts such as mental disorder and psychopathology. It necessitates research and discussion focused on the links between these concepts. For example, even if alcohol problems are dimensional in nature, such that there are no natural cutpoints demarcating heavy drinking, abuse, and dependence (Krueger et al., 2004), there are levels of alcohol problems beyond which society and professionals would deem intervention warranted. Understanding where relevant cutpoints lie would involve research on the way in which symptomatology per se is linked to consequences (e.g., social and occupational dysfunction) and discussion with professionals and policymakers about levels of consequences that societies are unwilling to tolerate.

Second, we should consider reorganizing sections and disorders described in the DSM-IV-TR to recognize the internalizing and externalizing spectra, and pursue research and discussion on the most optimal organization of diagnoses within these spectra. Finally, we should encourage further research linking cutting-edge developments in methodology with novel ideas

about how to describe and organize personality and psychopathology constructs. For example, Widiger and Clark (2000) envisioned the creation of a “diagnostic table of the elements” for future editions of the DSM. What are the main dimensions that would organize such a table? Do these represent some meshing between the FFM and the internalizing and externalizing spectra? What are the quantitative properties that distinguish one “element of psychopathology” from another? Open-minded, creative research that asks probing and novel questions but sticks close to the data in pursuing these questions has real potential ultimately to result in a diagnostic system that is empirically supported, useful in the clinic, and inspires research that leads to better prevention and treatment of mental disorders.

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