

Temperament as a Unifying Basis for Personality and Psychopathology

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Personality and psychopathology long have been viewed as related domains, but the precise nature of their relations remains unclear. Through most of the 20th century, they were studied as separate fields; within psychopathology, clinical syndromes were separated from personality disorders in 1980. This division led to the revelation of substantial overlap among disorders both within and across axes and to the joint study of normal and abnormal personality. The author reviews these literatures and proposes an integrative framework to explain personality–psychopathology relations: Three broad, innate temperament dimensions—negative affectivity, positive affectivity, and disinhibition—differentiate through both biologically and environmentally based developmental processes into a hierarchical personality trait structure and, at their extremes, are risk factors (diatheses) for psychopathology, especially given adverse life experiences (stress).

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The first model of personality–psychopathology relations may have been the doctrine of the four humors, attributed to Hippocrates and Galen (Maher & Maher, 1994). The balance of these four humors—blood (*sanguis*), phlegm, bile (*cholera*), and black bile (*melancholia*)—determined one’s temperament: sanguine, phlegmatic, choleric, or melancholic, respectively. Temperament, in turn, determined vulnerability to illness, and a humoral imbalance led to physical or mental illness. Balance—and thus health—was restored by such techniques as bleeding and purging.

With the advent of modern science, which in psychology began in the late 19th century, theories of personality–psychopathology relations were based on Darwinian concepts (Maher & Maher, 1994). One basic theme was that all types of mental illness reflected a general character deficiency that was genetically based, and the specific forms of which reflected biological and/or personal development. Freud’s well-known theory of the stages of character development, each with its associated psychopathology, is the most elaborate and influential of these views.

In the early 20th century, Kraepelin posited that personality disorders were *formes frustes* of the major psychoses. Kretschmer went a step further, hypothesizing a single continuum from personality (schizothyme) through personality disorder (schizoid) to clinical syndrome (schizophrenia; Livesley, Schroeder, Jackson, & Jang, 1994). Interestingly, Pavlovian conditioning theorists rejuvenated the ancient four-humoral theory, substituting variation in neuronal responses for the strength and balance of humors (Maher & Maher, 1994), and Eysenck explicitly related his three-factor personality model to Pavlovian concepts. Whereas Eysenck’s dimensions still play a major role in the field, the link to conditioning theories, for the most part, does not.

This precis suggests that personality and psychopathology have been studied in relation to each other since antiquity, but that is

only half the story. The other half is their division, both between psychiatry (with its major focus on abnormal processes) and psychology (which generally emphasizes the study of normal functioning) and also within each field. One sign of the split within psychology was the separation in 1965 of the *Journal of Abnormal and Social Psychology* into the *Journal of Abnormal Psychology* and the *Journal of Personality and Social Psychology*.

The Advent of the *DSM*: An Atheoretical Model

In mid-20th century, psychiatry systematized its accumulated knowledge of psychopathology in the *Diagnostic and Statistical Manual of Mental Disorders (DSM–I, DSM–II; American Psychiatric Association, 1952, 1968)*. In these *DSMs*, personality pathology was considered alongside other disorders with little attention to their potential interrelations. However, with the advent of *DSM–III* (American Psychiatric Association, 1980), personality disorders were given their own “Axis II,” which reopened the door for the systematic study of the two domains in relation to each other.

The *DSMs* were intended as descriptive documents to aid both research and treatment planning. Whereas prevailing theories no doubt influenced each of the *DSMs*, theory per se was not a guiding principle in their development, and *DSM–III* explicitly declared its atheoretical intent. However, the creation of separate axes clearly indicated that personality disorders were considered a distinct type of psychopathology, which also invited study of their interrelation.

A notable phenomenon of the successive *DSMs* was the increase in diagnoses, as well as in subtypes and specifiers to address within-diagnosis heterogeneity (Watson, 2003). Some of the diagnostic increase was due to new disorders (e.g., cyclothymia), but a substantial proportion resulted from subdivision (e.g., bipolar disorder into Bipolar I and Bipolar II disorders). These changes also were based largely on descriptive studies rather than theory-driven research.

Virtually without offering either a theoretical rationale or empirical justification, *DSM–III* included hierarchical exclusion rules

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affecting 60% of all disorders (Boyd et al., 1984), prohibiting Diagnosis A if it was “due to” Diagnosis B. Both clinicians and researchers chafed at these strictures because (a) they prevented full diagnostic representation of patients’ symptom sets, (b) they made it difficult to study disorders lower in the hierarchy, and (c) the “due to” rule was difficult to apply in that it required a subjective and inferential judgment regarding causal primacy of the relevant disorders in the absence of empirical data or clear guidance from the *DSM*.¹ Consequently, some adopted the more straightforward rule of simply not diagnosing A if B was present, whereas others, researchers in particular, ignored the rules in order to study their effects. In response to these twin pressures, the exclusion rules were largely removed from *DSM-III-R* (American Psychiatric Association, 1987).

Diagnostic Comorbidity

The most obvious and immediate effect of removing the exclusion rules was the well-known revelation of a high degree of diagnostic co-occurrence both within and across axes, which presented a challenge for a taxonomy in which, ideally, diagnoses are distinct entities. The usual term in the literature for this phenomenon is *comorbidity*, but as Lilienfeld, Waldman, and Israel (1994) discussed, there are problems with that term, including that it does not distinguish between mere overlap and statistical covariation. Nonetheless, I will use the term *comorbidity* because of its near universal usage in the literature, begging the readers’ understanding that in some places it may be imprecise.²

Clark, Watson, and Reynolds (1995) reviewed the emerging comorbidity literature and discussed various factors that affect comorbidity rates, such as time frame (i.e., concurrent or lifetime diagnoses), range of inclusion (i.e., only clear cases or also sub-clinical varieties), diagnostic method (i.e., paper-and-pencil self-report or interview), and sample type (e.g., clinic or community sample). Further, Mineka, Watson, and Clark (1998) noted that comorbidity is increased by excessive diagnostic splitting and when disorders share criteria. Others (e.g., Caron & Rutter, 1991; Klein & Riso, 1993; Lilienfeld, 2003; Maser & Cloninger, 1990; Meehl, 2001) also have examined a wide range of issues important in comorbidity, including additional methodological factors that affect the estimation of comorbidity rates.

Although all of these issues pose challenges to diagnostic validity (and a full discussion of them falls beyond the scope of this article), nonetheless, importantly, comorbidity cannot be fully accounted for methodologically (e.g., see Angold, Costello, & Erkanli, 1999). That is, even after taking various methodological reasons into account, much—perhaps most—comorbidity remains notably higher than would be expected by chance based on diagnostic prevalence rates.

Widiger and Clark (2000) reviewed more recent literature that further explores the bases of comorbidity and challenges the assumption of a distinct boundary between normal and pathological behavior. Focusing primarily on the anxiety and depressive disorders, they found evidence supporting both genetic and environmental influences on comorbidity but noted that these phenomena were not limited to those diagnoses, pointing, for example, also to literatures exploring “the high degree of overlap among personality disorders and between the personality disorders and Axis I disorders” (p. 954). Further, they noted the emergence of studies

indicating the presence of broad latent factors underlying certain sets of comorbid disorders.

Insofar as the Widiger and Clark (2000) review indicates that these various literatures are not entirely distinct, but have overlapping elements, it points to the possibility of a more comprehensive integration. Suggesting the form that this broader integration might take is the primary purpose of this article. To that end, I first examine briefly the literatures on comorbidity among Axis I disorders, among Axis II disorders, and across the axes. I then examine the literatures concerning overlap of normal-range personality with first Axis II and then Axis I disorders. In both cases, I focus on recent trends and important issues that these literatures raise. I then propose an overarching framework to integrate the broad domains of personality and psychopathology and the various literatures they subsume. I present an initial view of the form and structure of this framework and consider the various challenges that such a framework needs to address.

Etiological Models

In considering the broad literature on diagnostic comorbidity, it is useful to keep in mind the four major etiological models of comorbidity that have been proposed. These models fall into two major types based on whether they attribute causal significance to temporal order (for discussions, see Maser & Cloninger, 1990; Lilienfeld et al., 1994; Mineka et al., 1998). The predisposition or vulnerability model postulates that a prior disorder increases the likelihood that a person will experience a later onset disorder, whereas the pathoplasty model posits that a prior disorder influences the severity, course, or treatment response of a later onset disorder. The other two models posit that comorbid disorders reflect the same underlying process or etiological factor, so that neither can be viewed as clearly causing the other. The common/shared cause, liability, or factor (hereafter, I shall use the term *shared factor*) model hypothesizes a shared genetic diathesis (e.g., Carey & DiLalla, 1994), whereas the spectrum model proposes underlying continua that extend from normality to mild, moderate, and severe psychopathology (e.g., social phobia and avoidant PD overlap because the latter is a more severe form of the former).

It is important to note that these models are not mutually exclusive and each may be partially correct or simply incomplete. For example, the predisposition and pathoplasty models together would predict that an earlier onset disorder provides an initial vulnerability to a later onset disorder and, moreover, the presence of the earlier onset disorder affects the individual’s response to the later onset disorder, thereby influencing severity, course, or treatment response. I return to a fuller consideration of these models subsequently.

Comorbidity Within Axes

Comorbidity among Axis I disorders. To examine within-Axis I comorbidity, I ran a PsycINFO search for the term *comorbidity*, limiting the focus to *depression*, *anxiety*, and *substance abuse or substance dependence*, and specifically excluding articles with

¹ I thank an anonymous reviewer for contributing to this last point.

² I thank an anonymous reviewer for raising this point.

keywords *personality* or *Axis II*; this yielded over 1,600 articles in English on human subjects from 2000 to early 2004. A scan of the articles revealed that although not all of them directly addressed within-Axis I comorbidity, a substantial proportion did. Clearly, the issue is still of major interest, and I identified three trends in this recent literature: investigations of diagnostic sequencing (bi-directionality appears to be the norm; e.g., Hettema, Prescott, & Kendler, 2003; Stice, Burton, & Shaw, 2004), of less prevalent disorders (e.g., body dysmorphic disorder; Gustad & Phillips, 2003), and, more generally, of a wider variety of pairs of disorders (e.g., alcohol abuse and bipolar disorder, obsessive-compulsive disorder and schizophrenia). It is noteworthy that the latter two topics emerged despite the limitation of the literature search.

Comorbidity among Axis II disorders. The high degree of overlap among Axis II personality disorders was well documented almost by the time *DSM-III-R* was published in 1987 and certainly before *DSM-IV* in 1994. Widiger and Rogers' (1989) literature review reported an average 85% of multiple Axis II disorders in patient samples, and subsequent research has both confirmed and elaborated this finding. Examples include (a) the examination of different populations (e.g., community-dwelling adults; Zimmerman & Coryell, 1989), (b) the use of different types of assessment instruments (e.g., Oldham et al., 1992, used two different structured interviews), and (c) the study of the phenomenon in different cultures (Marinangeli et al., 2000, studied 156 Italian inpatients). Thus, Axis II comorbidity rates are high (a) in both patient and nonpatient samples, (b) when diagnosed with different assessment instruments, and (c) across different Western cultures.

It is interesting to note that whereas the general literature on personality disorders is increasing at about the same rate as the broader scientific literature, few specific personality disorders (with borderline PD the clearest exception, perhaps because of a high-prevalence rate and social cost) have active literatures (Blashfield & Intoccia, 2000).³ The high within-Axis II comorbidity rate may contribute to this slow growth because it casts doubt on the validity of the diagnostic system. Unless the validity of specific personality disorders can be established, research into them seems fruitless. Thus, understanding within-Axis II comorbidity could have a profound influence on the future of personality disorder research.

Cross-Axis Comorbidity

As mentioned earlier, the separation of Axis II personality disorders from Axis I clinical syndromes in *DSM-III* invited research into relations between these broad types of psychopathology. In a review of issues that had emerged from the ensuing research on Axis I–Axis II relations, Widiger and Shea (2001) noted three different types of relations between pairs of comorbid disorders: (1) *spectrum* (e.g., schizotypal PD with schizophrenia; avoidant PD with social phobia), (2) *cross-over* (e.g., borderline PD has features of both a mood disorder and a personality disorder), and (3) *overlapping* (e.g., substance abuse disorders share criteria with antisocial PD). They also discussed potential solutions to the problems these issues pose (e.g., converting to a dimensional format) but noted difficulties with each proposed solution. They recommended continued investigation into whether psychopathology is fundamentally categorical or dimensional. That is, they recognized that understanding the basic nature and structure of

psychopathology is a key element in resolving the problems posed by comorbidity.

It is interesting to note that perhaps because of the fundamental questions it poses, cross-axis comorbidity appears to have captured more research attention than has within-axis comorbidity. Initially, most studies were little more than descriptions of comorbidity rates between two diagnoses. Whereas this type of research was important to establish the phenomenon, it has reached the end of its usefulness (Tyrer, Gunderson, Lyons, & Tohen, 1997), and there should be a moratorium on purely and simply descriptive studies of comorbidity between two disorders.

What are beginning to emerge instead, and what we now need more of, are investigations that (a) compare comorbidity rates across related disorders (e.g., differential comorbidity of PDs with various anxiety or substance use disorders); (b) directly test one or more causal models of comorbidity; (c) examine patterns of comorbidity through meta-analyses of multiple studies, inclusion of variables other than diagnoses, or large-scale studies of phenotypic or genotypic structure; and (d) examine the effect of measurement method, instrument, or criterion set on comorbidity rates. Many individual studies exist, but an integrative review of findings is lacking and would greatly benefit the field. I illustrate each of these themes briefly.

Brieger, Ehrhart, and Marneros (2003) provide a meta-analysis of personality disorders in unipolar and bipolar mood disorder patients. Approximately half of the patients with either type of mood disorder had a comorbid personality disorder, with highly similar patterns of relations with specific PDs. These data suggest that if Axis I–Axis II comorbidity is due to a shared liability factor, then, in the case of mood disorders, this factor likely is also shared between unipolar and bipolar disorders. By contrast, meta-analysis of eating disorders and personality disorder found a nearly twofold difference in personality disorder comorbidity with bulimia (61%) versus mixed samples of eating disorder patients (34%). Thus, although one or more general vulnerability factors for psychopathology may cross Axis I and II, general factors alone cannot explain all the findings.

Klein and Schwartz (2002) tested four models of relations between dysthymia and borderline PD over a 5-year period and found that a shared-factor model best fit the data. As part of the same study, Klein (2003) found that self and informant reports of personality disorder at baseline—despite their modest correlation

³ An anonymous reviewer questioned the validity of the Blashfield and Intoccia (2000) analysis and cited several other personality disorders whose literatures he or she thought were more developed, so I ran a series of PsycINFO searches on these disorders, including borderline, antisocial, narcissistic, dependent, and obsessive-compulsive personality disorder (PD) in both keyword and title for the periods 1985–1991, 1992–1997, and 1998–2003 and found general support for Blashfield and Intoccia's (2000) conclusions. In the keyword searches, all six disorders showed roughly linear increases across the three periods (if anything, growth was slower between the latter two), but only borderline and antisocial PD reached over 50 articles a year in any period. In the title search, the curves were flat and indicated 5 or fewer articles per year for narcissistic, dependent, and obsessive-compulsive PD (which suggests that Blashfield & Intoccia, 2000 did a title search), whereas the other three showed increases that ranged from slight (schizotypal PD from 4 to 9 articles per year) to mild (antisocial PD from 5 to 17) and marked (borderline PD from 40 to 74).

($\kappa = .24$)—equally predicted depressive symptoms 7.5 years later ($\kappa = .24$ to $.29$, controlling for the other). It is interesting to note that they also predicted future social functioning to the same degree (see Kool, Dekker, Duijsens, de Jonghe, de Jong, & Schouws, 2000, for a related finding). Thus, a shared factor may underlie not only personality disorder and depressive symptoms but also social functioning.

Comorbidity between substance abuse and personality disorder has attracted a great deal of interest. Comorbidity with antisocial PD increases about twofold (approximately 30% to 60%) from mild to severe drug abuse/dependence (Flynn, Craddock, Luckey, Hubbard, & Duntzman, 1996). Other personality disorders also are prevalent, although *DSM-III-R* and *DSM-IV* criteria yield quite different comorbidity rates for a few disorders even in the same sample (e.g., Poling et al., 1999), raising further concerns about the validity of specific Axis II disorders.

A special section on borderline PD in a 2002 issue of *Biological Psychiatry* illustrates the attention that borderline PD has garnered in recent years. Skodol, Gunderson, et al. (2002) reviewed 15 studies representing over 1,500 individuals and reported that the Axis I–Axis II overlap for “any PD” averaged 46% and for borderline PD 22%, with borderline PD accounting for almost half (47%) of those with any PD. Again, not only comorbidity but the variability in comorbidity rates are explanatory challenges.

Several studies have examined patterns of comorbidity through large-scale studies of phenotypic and/or genetic structure (Kendler, Prescott, Myers, & Neale, 2003; Krueger, 1999; Krueger, Caspi, Moffitt, & Silva, 1998; Slade, cited in Watson, 2005; Vollebergh et al., 2001), revealing a remarkably consistent structure: A hierarchical model with two broad factors (externalizing and internalizing), with the externalizing factor consisting of substance dependence and either conduct disorder or antisocial PD, and with the internalizing factor having two subfactors, “misery” (comprising generalized anxiety disorder [GAD] and depressive disorders) and “fear” (comprising phobias and panic disorder). These studies largely focused on Axis I disorders, but three of them included antisocial PD or antisocial behavior, thus providing a link to Axis II disorders.

In summary, a large number of studies document and continue to explore the existence and nature of comorbidity across a wide range of disparate disorders. The pervasiveness and extent of the phenomenon is such that explanations of the overlap between pairs of disorders in isolation of the “big picture” are unlikely to move our understanding forward significantly. Before turning to studies that begin to suggest what the big picture might look like, however, there are several aspects of the recent literature that deserve comment.

The Twin Challenges of Severity and Heterogeneity

In their review of major issues in the diagnosis and classification of psychopathology, Clark et al. (1995) noted that comorbidity rates were higher in clinical versus community samples due, in part, to the fact that those with two disorders could seek help for either condition (Berkson, 1946). Berkson’s bias cannot account fully for the association of comorbidity with severity of diagnosis, however, and therefore Clark et al. (1995) suggested that severity was “a crucial factor in course, treatment, and outcome” (p. 132). The subsequent literature provides some support for this view:

Explanatory models of comorbidity often involve the concept of severity, and empirical data reveal a parallelism between diagnostic comorbidity and level of social functioning. It seems likely that if shared factors underlie not only different types of psychopathology but also social functioning, then they must represent broad, general concepts such as severity or, more specifically, type and level of functioning.

However, little has been done to elucidate the nature of severity, most likely because its extreme nonspecificity renders it elusive. That is, whereas severity may be invoked and have utility as an abstract, theoretical, explanatory concept, its ultimate viability depends on the extent to which it can be articulated in relation to measurable variables (cf. Cronbach & Meehl, 1955). Moreover, there are exceptions that will test the rule; specifically, there is some evidence that conduct disorder is milder in children with versus children without anxiety disorder (Walker et al., 1991).

In contrast to the broad nature of severity, which generally increases overlap nonspecifically, heterogeneity within classes of disorders is associated with differential comorbidity rates. Thus, at the same time that models may need a very broad concept such as severity to explain the pervasiveness and extent of comorbidity (as well as relations with social functioning), the considerable heterogeneity not only within broad diagnostic categories but within specific disorders also needs explanation.

Hierarchical Models

To capture the empirical reality of psychopathology, therefore, explanatory models must account for both broad shared factors and extensive heterogeneity within both diagnostic categories and single diagnoses. Such models have begun to emerge. An early example is the tripartite model of anxiety and depressive disorders (Clark & Watson, 1991), which was elaborated into an integrative hierarchical model by Mineka et al. (1998). More recently, Klein and Schwartz (2002) noted that whereas a shared-factor model clearly provided the best fit to explain observed relations between dysthymia and borderline PD over time, “the presence of significant unique determinants of both depressive symptoms and BPD features is also noteworthy” (p. 532). However, their data did not permit investigation into the nature of these determinants.

Kendler et al. (2003) reported not only a shared genetic risk for internalizing and externalizing disorders but also specific genetic risk factors for alcohol and drug dependence, a shared-environment factor for conduct disorder, two unique environment factors (one for major depressive disorder, GAD, and alcohol dependence, and the other for conduct disorder and antisocial PD) and, finally, unique, disorder-specific environment factors for all disorders. This rich model documents well the existence of both shared and unique factors that have been observed in a range of Axis I and Axis II disorders. Research must turn now to replicating and, if replicable, explaining the nature of these factors.

Reemergence of Interest in Personality–Psychopathology Relations

As stated earlier, through much of the 20th century psychologists and psychopathologists pursued their research independently of each other. However, just as the creation of separate axes in *DSM-III* invited study of Axis I–Axis II relations, the *DSM-III*

definitions of personality disorders in terms of traits (American Psychiatric Association, 1980, p. 305) did the same for relations between personality and PD, and much research has been carried out in this arena from a variety of perspectives.

Some researchers have sought primarily to clarify the maladaptive trait dimensions comprising personality disorder (e.g., Clark, McEwen, Collard, & Hickok, 1993; Livesley, Jackson, & Schroeder, 1992), whereas others have focused specifically on relations between normal personality traits and PD diagnoses (e.g., Costa & Widiger, 2002). Whether abnormal personality dimensions are continuous with those of normal personality or occupy their own distinctive space also has garnered attention. Finally, and not surprisingly given increasing awareness of cross-axis comorbidity, whether and how normal and abnormal personality traits relate to Axis I disorders also became a research target. I discuss each of these subtopics in turn.

Etiological Models

Just as I introduced the review of diagnostic comorbidity with a consideration of the major etiological models proposed to account for the phenomena, it is instructive to do the same regarding personality–psychopathology relations. Tellingly, the models proposed to explain diagnostic comorbidity overlap considerably with those offered to explain personality–psychopathology relations (e.g., Klein, Wonderlich, & Shea, 1993; Watson & Clark, 1995), so I recap them here briefly, pointing out the parallels.

As mentioned earlier, the predisposition or vulnerability and the pathoplasty model both postulate a temporal relation between two conditions. In the former, the first condition is a risk factor for the second, whereas in the latter, the first condition influences severity, course, or response to treatment. What is important to note here is that the prior condition may be a temperament/personality factor (i.e., a trait) as well as a disorder. Thus, for example, high trait neuroticism, as well as anxiety disorders, may be a risk factor for depression.

The pathoplasty model has two variants that are specific to personality–psychopathology relations (*scar* and *complication*), each of which posits that experience of psychopathology leads to change in personality traits (Lewinsohn, Steinmetz, Larson, & Franklin, 1981). The difference between the models is the degree and scope of the effect. The scar model posits fundamental and lasting trait-level change, so that following a major depressive episode, for example, the persons' trait neuroticism does not return to its premorbid baseline. By contrast, the complication model posits a "state" effect of disorder, with temperament/personality reverting to baseline with remission.

Finally, recall that both the shared-factor and the spectrum models posit that two conditions reflect the same underlying process or etiological factor, so that neither can be viewed as clearly causing the other. Again, the important point here is that these may be overlapping disorder–trait pairs as well as comorbid disorders.

Personality—Axis II Personality Disorder Relations

Maladaptive Personality Traits and Personality Disorder

Some proponents of dimensional approaches to personality disorder have developed instruments to assess maladaptive personal-

ity traits underlying the domain (e.g., Dimensional Assessment of Personality Pathology [DAPP], Livesley & Jackson, in press; Schedule for Nonadaptive and Adaptive Personality [SNAP], Clark, 1993). The scales of these two inventories not only show strong, systematic correlations with each other (Clark, Livesley, Schroeder, & Irish, 1996) and with measures of the five-factor model (FFM) of personality (Larstone, Jang, Livesley, Vernon, & Wolf, 2002; Reynolds & Clark, 2001) but also with the *DSM* personality disorders (Bagge & Trull, 2003; Hurt & Oltmanns, 2002; Morey et al., 2003; Pukrop, 2002; Reynolds & Clark, 2001).

Some overlap among these domains is due, no doubt, to shared content—after all, the *DSM* personality disorders are defined as "inflexible and maladaptive" personality traits (American Psychiatric Association, 1994, p. 630), and development of both instruments began with a systematic study of *DSM* personality disorder characteristics. Thereafter, however, each measure was created independently, so the high degree of observed correlation was by no means predetermined. Moreover, Livesley and colleagues' data have indicated that normal-range personality traits and those comprising personality disorder have a parallel structure, with genetic and environmental components that are "remarkably similar to the phenotypic factors" (Livesley, Jang, & Vernon, 1998, p. 941; see also McCrae, Jang, Livesley, Riemann, & Angleitner, 2001), again suggesting that the overlap is not simply explained by methodological factors.

Normal Personality Traits and Personality Disorder

The literature is replete with correlational reports of personality trait measure *X* with personality disorder measure *Y*, and a comprehensive review of this literature is beyond the scope of this article. However, the three topics that emerge most frequently—relations between personality disorder and both the FFM of personality and Cloninger's temperament–character model, and research on the construct of impulsivity—merit brief discussion.

Five-factor model. Saulsman and Page (2004) reviewed 15 samples from 12 studies published between 1990 and 1998 representing almost 2,900 individuals assessed with measures of the FFM and the *DSM-IV* personality disorders. Weighted mean correlations were consistently patterned with the respective sets of criteria comprising the 10 *DSM* Axis II disorders. Some significant differences in correlational pattern were observed across sample types (e.g., patient vs. nonpatient) or measurement method (e.g., interview vs. self-report), but the overall pattern was robust, and subsequent studies have confirmed the existence of reliable, systematic relations between the FFM and *DSM* personality disorders (e.g., Brieger, Sommer, Bloekink, & Marneros, 2000; Huprich, 2003; Morey et al., 2002; Reynolds & Clark, 2001).

Morey et al. (2002) cautioned that whereas FFM scores clearly distinguish personality-disordered groups from community norms, they largely fail to distinguish specific personality disorders from each other. However, the lower order facets of the FFM domains may provide more specific assessments of personality pathology, similarly to the way lower order trait measures do, such as the DAPP and SNAP (Reynolds & Clark, 2001).

Cloninger's model. Personality–PD relations also have been examined in the framework of Cloninger's model, using the Tri-dimensional Personality Questionnaire (TPQ; Cloninger, 1987) or its successor, the Temperament and Character Inventory (TCI;

Cloninger, Svrakic, & Przybeck, 1993). Support for Cloninger's hypotheses regarding specific dimension-disorder mappings is mixed, with some studies finding some support (e.g., Griego, Stewart, & Coolidge, 1999; Maggini, Ampollini, Marchesi, Gariboldi, & Cloninger, 2000; Mulder, Joyce, Sullivan, Bulik, & Carter, 1999; Pukrop, 2002; Svrakic, Whitehead, Przybeck, & Cloninger, 1993) and others not so (e.g., Ball, Tennen, Poling, Kranzler, & Rounsaville, 1997; Guiterrez, Sangorin, Martin-Santos, Torres, & Torrens, 2002; Nagoshi, Walter, Muntaner, & Haertzen, 1992).

A further cautionary note is also in order. O'Connor (2002) investigated whether the structure of a broad array of widely used personality and psychopathology measures differed across clinical and nonclinical samples. The results overwhelmingly revealed structural invariance across sample type for most measures, whereas the TPQ and TCI were almost unique in having very unstable factor structures both across and within sample types. That is, the instruments have an unreliable factor structure even when administered to samples drawn from the same broad population, raising concerns about the meaning of the instruments' scores. Nevertheless, whereas specific TPQ and TCI findings may not be reliable, the data clearly document the general point that normal personality and personality disorder are interrelated.

Impulsivity. Impulsivity has long been regarded a key component of psychopathy and, later, antisocial PD. Interest in trait impulsivity, including its biological basis, has a venerable history, stemming from early work by Eysenck (1967) and Gray (1972). More recently, impulsivity has garnered research attention in part because it is considered a core trait of borderline PD which, as noted earlier, has the largest current literature among the PDs. Looper and Paris (2000) hypothesized that impulsivity was the shared dimension underlying *DSM* Cluster B PDs (antisocial, borderline, histrionic, and narcissistic) and that these disorders were differentiated on the basis of severity of impulsivity (antisocial and borderline are more severe than histrionic and narcissistic), presence of other traits, gender, and sociocultural influences.

Similarly, Krueger et al., (2002) provided a brief review of the broader literature linking "disinhibitory personality traits" (p. 413), such as impulsivity and novelty seeking, with antisocial PD, criminal behavior, and substance (ab)use disorders. They noted that longitudinal studies indicated that impulsivity and related traits precede and predict subsequent behaviors related to antisocial PD (e.g., delinquency, antisocial and criminal behavior), as well as substance (ab)use. Using data from a large twin sample, they presented a hierarchical model with both impulsivity and conduct disorder-antisocial behavior and substance dependence as manifestations of an underlying externalizing construct with substantial heritability. In addition, impulsivity had a specific genetic component and all variables had significant nonshared environmental components.

In summary, extensive research has established that there are considerable relations between normal- and abnormal-range personality, at broad higher order levels, at more specific lower order levels, and even at the level of subtrait facets. The challenge now is to clarify both the most frequent patterns and the particularities of these relations.

Personality-Axis I Disorder Relations

As noted earlier, researchers turned away from the study of personality-psychopathology relations for some time during the 20th century. Thus, the vigor with which this research has re-emerged and the swiftness with which researchers have embraced the basic notion that these domains are interrelated is all the more striking. The first thrust appears to have come from depression researchers who noticed its interrelation with neuroticism (N) or negative affectivity (e.g., Akiskal, Hirschfeld, & Yerevanian, 1983; see Klein et al.'s 1993 review). Much research into comorbidity also invoked personality as an explanatory factor (e.g., Clark & Watson's, 1991, tripartite model and Mineka et al.'s, 1998, integrative hierarchical model of the depression-anxiety disorder overlap; see also reviews by Clark, Watson, & Mineka, 1994; Clark et al., 1995).

In addition, many studies report on the relation between personality trait X with Axis I disorder Y. A comprehensive and integrative literature review would be beneficial to our understanding of this broad domain but again is beyond this article's scope. However, the FFM and Cloninger's measures also have been used in a number of studies in this domain, so a brief look at these subliterations provides an overview of their breadth.

Five-Factor Model

By the mid-1990s, that high N is associated with psychopathology was well established (Clark et al., 1995) and subsequent research has continued to confirm and broaden this finding. Also of interest is that (a) domain or facet scores for Extraversion (E), Agreeableness (A), and Openness (O) differentiated among schizophrenia, unipolar and bipolar depression patients (Bagby et al., 1997), (b) community dwellers who later developed an eating disorder had low A and high O scores premorbidly (Ghaderi & Scott, 2000); (c) symptoms of attention-deficit/hyperactivity disorder correlated negatively and differentially with Conscientiousness (C) (inattention-disorganization symptoms) and A (hyperactivity-impulsivity and oppositional behaviors (Nigg et al., 2002)). A key point here is that FFM personality variables correlate with Axis I and Axis II disorders at a similar magnitude.

Cloninger's Model

Similarly, studies that have used the TPQ or TCI to examine relations between personality and Axis I disorders in patient samples also have found relations of the same magnitude as those with Axis II for a wide range of Axis I disorders, including depression (e.g., Farmer et al., 2003), bipolar disorder (e.g., Osher, Cloninger, & Belmaker, 1996), eating disorder (e.g., Vervaeke, van Heeringen, & Audenaert, 2004), social phobia (e.g., Pelissolo et al., 2002), substance abuse (e.g., Sher, Bartholow, & Wood, 2000), schizophrenia (e.g., Szoek et al., 2002), and depersonalization disorder (e.g., Simeon, Guralnik, Knutelska, & Schmeidler, 2002).

Again, the challenge in these data is the similarity in the magnitude of correlations between personality and disorders on the two *DSM* axes of disorders, indicating that personality traits do not have a "privileged" relation with Axis II personality disorders. Thus, it seems reasonable to pursue the possibility of a general model to explain personality-psychopathology relations rather

than separate models to explain relations between personality and each of the two axes.

Reintegration of Personality and Psychopathology

Insofar as there is now considerable evidence of interrelations between and among Axis I disorders, personality disorders, and personality traits, it is time to turn to the question of how these domains are related to one another. Taken as a whole, a broad conclusion that can be drawn is that a more comprehensive model of personality–psychopathology relations is warranted. To date, explanatory models of the overlap between and among diagnoses and personality traits typically have been developed to account for specific empirical observations or have been discussed as a set of theoretical models without serious consideration as to how they might be integrated into a broad, overarching theoretical framework. Given various problems with the current *DSM–IV* structure, having such a framework would, at least, have utility as a guide to research to inform the *DSM* revision process and, at most, would serve, ultimately, to replace the current *DSM* structure. In light of the above literature review, what features must such a model have, and what other features would it have ideally?

At a minimum, a comprehensive model of personality–psychopathology relations would be both a structural and a causal model that accounts for (a) extensive comorbidity both between and among Axis I and II disorders; (b) relations between a basic set of personality traits and a broad range of both Axis I and II pathology; (c) variation in comorbidity and personality–psychopathology relations due to specificity within classes of disorder and heterogeneity within disorders; and (d) temporal sequencing of traits with disorders and comorbid disorders. In addition, such a model would ideally (e) clarify the nature of personality and psychopathology, that is, how these concepts themselves are to be understood and, in doing so, address such relevant issues as states versus traits, temperament (nature) versus character (nurture), and dimensions versus categories; and, finally, (f) be consistent with the growing literatures for both personality and psychopathology regarding genetic and environmental factors, specific biological factors, and development.

As every college sophomore knows, variables A and B can be correlated because (a) A causes B, (b) B causes A, (c) A and B form a feedback loop of mutual causation, (d) a third variable causes both A and B, or (e) some combination of the above. Equally well known is that the most parsimonious explanation for a given set of data should be considered first. Given the multiple findings reviewed above, it seems most parsimonious to posit the existence of a small set of “third variables” as underlying and accounting for the many interrelations between and among personality and psychopathology.

As mentioned earlier, the various etiological models proposed to explain diagnostic comorbidity and personality–psychopathology relations are overlapping and not mutually exclusive. I argue here that they can be integrated into a single hierarchical model. I propose the following framework as an initial oversimplification and then develop various aspects of the framework through the rest of this article.

Temperament Dimensions Are Shared Factors Underlying Personality and Psychopathology

Adult personality traits emerge through differentiation from three innate biobehavioral dimensions, two of which are affective systems—positive and negative affectivity—and the third of which ([dis]inhibition) is a regulatory system,⁴ and they share these genetic diatheses with later developing disorders (shared factors). At least some disorders are phenomenologically more extreme manifestations of personality dimensions (spectra) and most, if not all, disorders are more likely to develop in individuals who are more extreme on relevant temperament–personality dimensions (predisposition–vulnerability), particularly given adverse life experiences and/or the experience of another disorder (diathesis–stress). Comorbid disorders each may affect the presentation of the other, and the personality traits of individuals who develop psychopathology also may be affected by that experience (pathoplasty), both in the short term (complication) and long term (scar).

Key points regarding this framework including some elaborations, are the following. First, a distinguishing feature is that it does not posit, as many current models do, that adult personality traits per se underlie psychopathology, but rather that personality and psychopathology are linked primarily through their shared underlying genetic diatheses of broad temperament dimensions. In this regard, the framework is consistent with, but more explicitly developmental than, the formulations of Clark and Watson (1999) and Watson, Wiese, Vaidya, and Tellegen (1999) and represents a modification of Rothbart and Ahadi’s (1994) model, which proposed infant temperament as the basis for personality from which, in turn, psychopathology develops through interaction with environmental–learning factors. To be sure, the Rothbart and Ahadi (1994) model describes perhaps the most common developmental pathway, but positing the underlying temperaments, rather than personality per se, as the etiological agent of psychopathology, provides greater conceptual flexibility and more easily incorporates cases in which personality is, conversely, affected by psychopathology.

Second, the framework explicitly embraces a two-affect systems model of positive and negative affectivity–emotionality–activation, which are manifested as general biobehavioral systems of approach–withdrawal, respectively, and which are regulated, in large part, by a third system dimension that itself is nonaffective but that plays a fundamental “gatekeeping” role in the degree to which incoming stimuli are subjected to its inhibitory influence (see Clark & Watson, 1999; Watson et al., 1999; and see also Depue’s work [e.g., Depue, 1996; Depue & Collins, 1999; Depue & Lenzenweger, 2001]).

⁴ Insofar as affect or emotion is a core and defining feature of temperament, one might argue that only two of these system dimensions may properly be called temperaments. However, because the third dimension affects how these emotional systems are experienced and manifested, I shall use the term *temperament* to characterize all three dimensions. This is consistent with Allport’s (1937) definition of temperament as “the characteristic phenomenon of an individual’s emotional nature, including his [sic] susceptibility to emotional stimulation, his customary strength and speed of response, the quality of his prevailing mood, and all peculiarities of fluctuation and intensity of mood” (p. 54) in that the third (dis)inhibitory, regulatory system clearly plays a role in these qualities.

Whereas negative temperament is associated with a broad range of psychopathology (e.g., Krueger, Caspi, Moffitt, Silva, & McGee, 1996; Watson & Clark, 1984), positive temperament plays an important but more limited role in psychopathology, being linked primarily with depression (Clark & Watson, 1991; Durbin et al., 2005) and, perhaps to a lesser extent, schizophrenia and social phobia (see Mineka et al., 1998). Indeed, important variance in positive temperament may distinguish “ordinary normality” from the high level of adaptation that characterizes those who are particularly psychologically healthy; that is, high levels of positive affectivity may distinguish those who are average versus particularly adept at dealing with life’s slings and arrows (Shiner, 2000; Shiner, Masten, & Tellegen, 2002). The third dimension, disinhibition (vs. constraint) is associated with the range of psychopathology broadly known as *externalizing disorders* (e.g., substance abuse, antisocial PD—conduct disorder, borderline PD; Kendler et al., 2003; Krueger, 1999; Krueger et al., 1998, 2002; Lynam, Leukefeld, & Clayton, 2003; Slade, cited in Watson, 2005; Vollebergh et al., 2001).

Third, positing that personality emerges through differentiation has two implications. One is that personality should become more stable over time and, indeed, Roberts and DelVecchio (2000) reviewed 152 longitudinal studies of personality and found increasing trait stability with age. A second is that the framework embraces the view that personality is hierarchically structured: Traits and subtraits that emerge from the same temperament dimension are more closely related than those that emerge from different underlying dimensional systems and also may be expected to show parallel patterns in relation to psychopathology. This is the case from the top of the hierarchy downward. For example, FFM A and C may be considered components of the Big Three factor, Disinhibition (Markon, Krueger, & Watson, 2005). As such, A and C should—and do—relate similarly to broad types of psychopathology (e.g., substance abuse, antisocial PD—conduct disorder; Lynam et al., 2003), and also show differential relations when disorder is more finely specified (e.g., recall that subsets of attention-deficit/hyperactivity disorder symptoms related differentially to A and C; Nigg et al., 2002).

Moreover, progress has been made on further specification of the middle level of the hierarchy: The three basic dimensions differentiate through development, both biological and experiential, into approximately 15 midlevel traits⁵ (e.g., aggression, affiliativeness, impulsivity) that, accordingly, have both genetic and environmental components. These traits, singly or in combination, are linked more specifically to different types of psychopathology. For example, comparing substance use among college students, Clark and Watson (1999) found that Carefree Orientation and Antisocial Behavior (subtraits of Disinhibition) were linked differentially to lower GPA and alcohol and cigarette usage versus other drug use, promiscuous sex, reckless driving, and (negatively) religious behavior, respectively. Preliminary data suggest that below the midlevel traits there is at least one more finely differentiated level (i.e., that of personality trait facets and specific criteria of disorders; e.g., Livesley et al., 1998; Nitschke, Heller, Imig, McDonald, & Miller, 2001; Watson, Wu, & Cutshall, 2004), but clarification of this level to any degree of specificity remains a future prospect.

At the higher order level, existing research suggests that the negative affectivity and disinhibition systems may be more tightly

linked or, at least, that the interplay between these systems is more important for psychopathology than that between the positive affectivity and disinhibition systems. Specifically, Markon et al.’s (2005) meta-analysis of the structure of personality trait measures supports Digman’s (1997) finding that negative affectivity and disinhibition together form a still higher order “alpha” factor (whereas E and O combine to form a “beta” factor), and other research indicates that the combination of negative affectivity and disinhibition represents a particularly maladaptive form of psychopathology (e.g., Hicks, Markon, Patrick, Krueger, & Newman, 2004; see also Clark & Watson, 1999, for a discussion of interplay between biological systems linked with negative affectivity and disinhibition, respectively).

Fourth, because the proposed framework encompasses both static, structural issues and dynamic, developmental issues, in methodological terms it hypothesizes both between-subjects and within-subjects effects. Specifically, for example, it would predict that related personality traits and disorders would (a) load on the same factor in nomothetic cross-sectional structural analyses (shared factors, spectra); (b) show shared genetic and, in some cases, environmental factors in, for example, twin, family, and adoption designs (shared factors); and (c) be related systematically within individuals in longitudinal path analyses (shared factors, predisposition–vulnerability). Each of these predictions has support in existing data, discussed subsequently. It is important to stress that it is unlikely that a shared genetic diathesis is the only causal link between personality and psychopathology; that is, they also may share one or more environmental components but, as I also discuss subsequently, much less progress has been made on this front.

Fifth, the proposed framework offers a new perspective on various difficulties with the current Axis I–Axis II distinction. For example, it is difficult to explain why certain pairs of disorders that are linked for different reasons (e.g., avoidant PD appears to be a severe form of social phobia, Widiger, 1992b; schizophrenia and schizotypal PD most likely share one or more etiological factors; Grove et al., 1991) are placed on separate axes. Similarly, it is not clear why GAD, which typically is a chronic disorder that is very strongly associated with negative affectivity is placed on Axis I rather than on Axis II. Further, the evidence is equivocal regarding

⁵ This number is, admittedly, semiarbitrary but, importantly, only semiarbitrary. That is, in between the well-known two to five broad factors (Eysenck’s [1967] or Digman’s [1997] Big Two; Eysenck’s [Eysenck & Eysenck, 1985] or Tellegen’s [in press] Big Three; the FFM) and what Wiggins and Pincus (1992) called *fine-grained* groupings, such as the 75 of Norman (1967), the 133 of Goldberg (1990), or the 160 of Cattell (cited in Wiggins, 1984), efforts have been made to delineate sets of lower order personality traits at the middle level (called *optimal* by Briggs, 1989, p. 246) and have converged on trait sets ranging from lows of 8 (Comfrey Personality Scales; Comfrey, 1970), 10 (Guilford–Zimmerman Temperament Survey; Guilford & Zimmerman, 1956) and 11 (Multidimensional Personality Questionnaire; Tellegen, in press), to mid-range numbers of 15 (Edwards Personality Preference Schedule; Edwards, 1959) and 16 (16-Personality Factors; Cattell, Cattell, & Cattell, 1993), to highs of 20 (California Psychological Inventory; Gough, 1987) and 22 (Personality Research Form; Jackson, 1989). The aforementioned SNAP (Clark, 1993; 15 scales) and DAPP (Livesley & Jackson, in press; 18 scales) also fall on or close to this number.

whether dysthymia is a separate disorder from the depressive PD currently identified as in need of further study (e.g., Ryder, Bagby, & Schuller, 2002). Livesley (e.g., Livesley et al., 1994) was among the first to argue that there is neither a theoretical nor an empirical basis for separating personality disorder from other types of psychopathology (see also Widiger & Shea, 2001). I find their arguments compelling, and thus the proposed framework focuses instead on clarifying personality–psychopathology relations without regard for the current Axis I–Axis II distinction.

Supportive Data

Clearly, this framework is built upon a number of existing pieces and supportive evidence that already have begun to accrue. I have previously mentioned the structural models that both account for anxiety and depressive disorder comorbidity through shared dimensions of positive and negative affectivity and also explain the distinctiveness of specific anxiety disorders through more specific components, such as anxious arousal in panic disorder (Clark & Watson, 1991; Mineka et al., 1998). It is not yet clear whether these specific components are unique to psychopathology or also have personality trait counterparts. However, numerous studies have shown that various symptom dimensions are not unique to psychopathology but are continuously distributed and found in normal samples (e.g., Watson, 2005), which suggests common underlying personality–subtrait–psychopathology–symptom dimensions.

Similarly, a number of studies have shown Axis I–Axis II relations can be accounted for in large part by shared personality traits. For example, disinhibition and related personality traits, such as FFM A and C, have been shown to account for the overlap between substance abuse and Cluster B personality disorders (Casillas & Clark, 2002), antisocial personality disorder (Sher & Trull, 1994), and antisocial behavior (Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Lynam et al., 2003). In a related vein, Nestor (2002) presented evidence showing that personality variables (specifically, impulse dyscontrol and affect dysregulation) mediated psychopathology–violence relations. These three-way interrelations (personality with both Axis I–Axis II psychopathology and Axis I–Axis II comorbidity) are most parsimoniously explained by positing that they all are based in a shared underlying system. The fact that disorder comorbidity is found to be due largely to shared relations with personality traits (and not vice versa) suggests that personality is a developmentally simpler manifestation of the underlying temperament dimensions than is psychopathology.

Consistent with this suggestion, Carey and DiLalla's (1994) exploration of genetic causal models for personality–Axis I relations (specifically focused on depression and anxiety disorders) indicated that personality was causally prior to disorder and explained much of the comorbidity. Subsequent longitudinal data indicate that elevations in relevant personality traits frequently, if not always, precede emergence of the related Axis I and Axis II disorders (e.g., Mufson, Nomura, & Warner, 2002; see Krueger et al., 2002). As mentioned earlier, that elevation in personality traits typically precedes psychopathology is not inconsistent with a framework in which basic temperament dimensions underlie both.

The emerging hierarchical, spectrum model for more prevalent mental disorders discussed earlier (e.g., Kendler et al., 2003;

Krueger, 1999; Krueger et al., 1998, 2002; Vollebergh et al., 2001) also fits well within the proposed framework. The consensus model proposes a stable internalizing factor to account for certain Axis I comorbidity through the common dimension of negative affectivity, and an externalizing factor that encompasses antisocial PD–conduct disorder and the personality dimension of low constraint (i.e., disinhibition–impulsivity). Various data also indicate that negative affectivity contributes to externalizing factors (e.g., Krueger et al., 1996), which may account for the modest relation found between the internalizing and externalizing factors (Kendler et al., 2003; Krueger, 1999; Vollebergh et al., 2001).

The data incorporated to date by these particular studies stop short of a full integration of Axis I syndromes, Axis II PDs, and personality dimensions. As noted earlier, however, Klein and Schwartz's (2002) model also crossed the Axis I–Axis II boundary, linking depressive disorders and borderline PD via a shared underlying factor, which lays the groundwork for the addition of borderline and other personality disorders to the emerging consensus model.

Development

Abundant evidence of continuity from infancy through to adulthood has accumulated in recent years. In a special issue of the *Journal of Personality* on personality development, 7 of 10 substantive articles presented longitudinal data (Graziano, 2003), and, importantly, several long-term projects that began in childhood now have “come of age.” I illustrate the observed continuity with an example from each of these projects. First, a small but intensive study of prosocial behavior, in which interview and other-report data were collected every 2 years from age 4–5 to 23–24, reported stable prosocial dispositions (Eisenberg et al., 1999). The large-scale, epidemiological sample Australian “Dunedin” study classified children at age 3 into three temperament groups, now in their 20s. Those in the “undercontrolled” group are more impulsive, unreliable, and antisocial adults, whereas those in the “inhibited” group are more likely to be depressed adults and to have fewer social supports (Caspi, 2000). A 10-year, University of Minnesota study reports clear relations between child (age 8–12) and young adult (age 17–23) measures of Positive and Negative Emotionality and Disinhibition (Shiner et al., 2002). Moreover, both childhood and adult antisocial conduct are related to adult Disinhibition, whereas maladaptivity is related to Negative Emotionality. Behavioral inhibition and, to a lesser extent, quality of peer relations also were stable over time (Gest, 1997).

Data are emerging also from three Scandinavian studies, two of which began in the mid to late 1960s and the other began in 1980. One study now reports, again by way of illustration, that compliance, self-control, and low aggression in 8-year-olds related to socialization and low aggression and impulsivity 25 years later when the participants had reached their mid-30s (Laursen, Pulkkinen, & Adams, 2002). Another found continuity over 17 years in the construct of *difficult temperament*, manifested more strongly as low social adjustment in childhood and as anger in adulthood (Pesonen, Raikkonen, Keskivaara, & Keltikangas-Jarvinen, 2003). The third study examined relations between temperament factors that emerged from ratings of age 4 behavioral data with teacher ratings at age 10, a psychologist's ratings at age 25, and self-ratings at age 36. Whereas all five factors showed some consis-

tency, two of the five factors—Aggression (a marker of Digman’s alpha) and Outgoingness (a beta marker)—were the most robust. Moreover, several of the childhood temperament factors were related to adult alcohol and drug-related problems (e.g., childhood aggression and low persistence predicted, respectively, intoxication frequency at age 25 and drug-related criminality at age 30).

Specific Challenges

State versus trait variance. A major challenge in modeling personality–psychopathology relations is that trait scores change with diagnostic state (Clark et al., 1994), which has led some researchers (e.g., Goel, Terman, & Terman, 2003; Reich, Noyes, Hirschfeld, Coryell, & O’Gorman, 1987) to conclude that personality scales are state rather than trait measures. However, using a 5-week pharmacotherapy trial, Santor, Bagby, and Joffe (1997) demonstrated that personality trait changes (specifically, N and E—the two major affective dimensions) were not—or only modestly—accounted for by changes in depression scores and, therefore, concluded that the meaning of the state-dependent nature of personality scores needed reexamination.

Building on this finding, Clark, Vittengl, Kraft, and Jarrett (2003) demonstrated that personality scores (including prominently SNAP Negative and Positive Temperament) could be separated into variable (state) and stable (trait) components. It is important to note that they found that, whereas both components correlated concurrently with depression severity level, only the trait portion predicted future levels. This is consistent with the findings of many studies that treatment-responders’ scores on certain personality traits decrease but still remain higher than controls’ (e.g., Agosti & McGrath, 2002; Du, Bakish, Ravindran, & Hrdina, 2002), whereas treatment nonresponders’ profiles are virtually unchanged from pretreatment scores.

Thus, personality measures—and more specifically, those related to the higher order dimensions of negative and positive temperament—combine state and trait variance. Currently, most models of personality–psychopathology relations ignore state variance or consider it a nuisance factor, part of the error term, but a comprehensive model ultimately must account for this variance as well. Notably, state negative–positive affect and trait negative–positive affectivity (neuroticism–extraversion), respectively, are themselves related (Watson & Clark, 1984, 1997), so inclusion of both sets of variables will provide a more complete picture of psychopathology both structurally and developmentally.

Heritability and environmental variance: Temperament and character? Adult personality is acknowledged widely to result from environmental influences acting on innate tendencies. From our current vantage point, without greater specification of the various components, this appears to be little more than a banal truism. However, this was not always the case. Only relatively recently have multiple studies confirmed that genes account for roughly half of the observed variance in personality traits (e.g., Tellegen et al., 1988; Turkheimer, 2000). Moreover, whereas the developmental studies discussed earlier have established clear links between child and adult temperament–personality and thus are consistent with studies showing the strong heritability of personality, a great deal of change occurs over this time span as well, and the more difficult tasks of clarifying the role of experience in turning temperament into personality–psychopathology, as well as

understanding the processes by which temperament develops into personality–psychopathology, lie ahead of us.

Until behavior genetics studies in the late 20th century indicated otherwise, shared familial environment was believed to be the major environmental factor shaping personality. Beginning with the seminal study of Tellegen et al. (1988), however, research has shown consistently that the effect of shared environment is negligible for most traits, that the predominant etiological factors in personality are genetic and unshared environment. This finding has been replicated with a variety of personality measures including the Multidimensional Personality Questionnaire (Krueger, 2000), California Psychological Inventory (Bouchard, McGue, Hur, & Horn, 1998), the TCI (Gillespie, Cloninger, Heath, & Martin, 2003), and the DAPP (Jang, Livesley, & Vernon, 1998), as well as scales from the Minnesota Multiphasic Personality Inventory and Zuckerman’s Sensation-Seeking Scales (Mustanski, Viken, Kaprio, & Rose, 2003).

Turkheimer and Waldron’s (2000) review of the nonshared environment research is particularly sobering with regard to understanding the role of environment in the development of personality–psychopathology. They conclude that studies of measurable differences in “objective” nonshared family environment (e.g., differential parental behavior) contribute little to personality. It is interesting to note that the differential effects of peers and teachers are somewhat stronger (see also Rose et al., 2003) but, for the most part, “the environment is all interaction and little main effect” (Turkheimer & Waldron, 2000, p. 92; see also Wachtel, 1994, for a longitudinal, developmental approach, a cyclical dynamic process relating personality and psychopathology).

More important, the nonshared environmental components of the 15 or so proposed midlevel traits may reflect primarily what Turkheimer and Waldron (2000) have termed the “effective environment” (p. 78). Moreover, measuring this effective environment may prove to be more tractable than would appear at first blush because it is, at least partly and perhaps largely, dependent on individuals’ perceptions and interpretations of situations which, in turn, have been shown—at least for some perceptions (e.g., stress)—to be strongly related to personality (Monroe & Depue, 1986). Therefore, if this proves to be a more general phenomenon, a start will have been made toward specifying one set of elements in interactions involving the nonshared environment.

It is important to note that Turkheimer (2000) predicts that a similar conclusion will be drawn regarding the behavioral effects of single genes, specifically, that additive gene effects provide the predictive power largely observed to date (e.g., with regard to personality stability), whereas developmental changes occur through gene–gene (as well as gene–environment, and phenotype–environment) interactions and thus will prove more elusive (but see Dick & Rose, 2002, for a more optimistic view of the future contribution of behavior genetics research).

Furthermore, although the (nonshared) environmentally influenced components of personality may stem primarily from individuals’ specific experiences (or their perceptions of those experiences), a given set of experiences (perceptions) within individuals may contribute jointly to the development of psychopathology as well as personality. Recall, for example, the Kendler et al. (2003) finding that conduct disorder and antisocial PD shared a unique-environment factor. Assuming this finding can be extended to include personality traits associated with antisocial PD

(e.g., disinhibition), research explicating environmental factors in the development of disinhibition may elucidate environmental factors in the development of conduct disorder and antisocial PD as well.

It is noteworthy that studies have not found greater heritability for traits identified as *temperament* as compared with supposedly more environmentally shaped *character* traits (Gillespie et al., 2003; Reimann & De Raad, 1998). At first glance, this finding would appear to refute the hypothesis that there are certain core traits or temperaments that are developmentally primary, but further consideration suggests otherwise. That is, personality trait structure is rather diffuse and undifferentiated in infancy and is elaborated increasingly through childhood, adolescence, and finally adulthood (Rothbart & Ahadi, 1994). Thus, all personality traits measured in adulthood may be roughly equally heritable because they all share their temperamental origin, yet all are increasingly differentiated through development via influences that include both within-person (e.g., cognitive and self-regulatory changes) and person–environment (e.g., social–interpersonal experiences, life events) factors. Consequently, whereas temperament and character may be distinguished conceptually, with only the former present at birth, attempts to identify certain adult personality traits as temperaments and others as character is most likely a fruitless venture.

In summary, whereas it clearly is premature to attempt to delineate the specific forms that these various types of developmentally critical interactions will take, for the purposes of constructing a comprehensive model linking personality and psychopathology, the important point to glean from these considerations and existing data is that adult personality variables represent complex combinations of variance due to states as well as traits, and variance due not only to basic temperament but also to the elaboration of temperament through individuals' experiences and their reactions to those experiences. The most basic tenet of the proposed model—that three temperament dimensions are the shared factors underlying observed personality–psychopathology relations—is consistent with the findings of studies that have examined genetic and environmental contributions to psychopathology, personality, and their overlap.

To cite a specific example, Mineka et al.'s (1998) integrative hierarchical model, which was developed to explain the shared and specific factors in depression and anxiety disorders, was based, in part, on genetic studies indicating that anxiety and depressive symptoms correlated because of a genetic factor they shared with neuroticism (Kendler, Neale, Kessler, Heath, & Eaves, 1993). Similarly, the externalizing factor linking personality (disinhibition) and psychopathology (substance dependence, antisocial behavior) identified by Krueger et al. (2002) may be interpreted as representing one or more underlying temperament dimensions. Thus, key factors in the proposed framework for understanding relations among different forms of psychopathology are not themselves specifically psychopathological; rather, they are identified as major dimensions of normal temperament structure that have a substantial genetic component.

This view is consistent with the additional findings of Kendler et al. (1993) that (a) the genetic component of the overarching externalizing factor (i.e., the underlying temperament(s) in the proposed framework) was notably larger than the genetic component of any other element in the model and (b) the unique (non-

shared environment) component of every element (i.e., both personality and psychopathology elements) in the model was substantial and larger than any other component, except for the externalizing factor itself, in which it represented a nontrivial but modest component. This is precisely what would be expected if the externalizing factor represented an underlying, largely genetically based, temperament factor or, more likely, the combined effects of two factors—Negative Temperament and Disinhibition—which, as stated earlier, accounts for the moderate correlation with internalizing which is associated strongly with Negative Temperament.

Biological findings. Insofar as temperament is conceived as a dimension that, being present at birth, has a large genetic component, data on biological factors that link personality and psychopathology also may provide support for the proposed framework. This literature is growing, diverse, and not yet well integrated, although there are promising beginnings (e.g., Depue & Collins, 1999; Siever & Davis, 1991; see also Clark & Watson, 1999). Clearly, a review of this literature is outside this article's focus, but a few examples give a sense of the wide scope of such research and provide a glimpse into a future in which biological variables also are integrated into the understanding of personality and psychopathology.

Among the domains being researched are (a) sleep disturbances (e.g., Asaad, Okasha, & Okasha, 2002, found similarities between borderline PD and major depressive disorder); (b) mood and social affiliation (e.g., Knutson et al., 1998, reported that a selective serotonin reuptake inhibitor, most often used to treat depression, decreased negative affect and increased social affiliation in non-patient volunteers); (c) startle reactions (e.g., abnormal potentiation of startle indexed psychopathic traits in both a community and an incarcerated sample [Vanman, Mejia, Dawson, Schell, & Raine, 2003, and Patrick, 1994, respectively]); and (d) autonomic nervous system regulation (e.g., Thayer & Lane, 2000, found restricted cardiac variability to be associated with both normal and pathological worry [GAD]).

Earlier, I noted the research interest in the construct of impulsivity, and a number of studies have addressed the biology of impulsivity or impulsive aggression. This dimension has been identified as a key component of borderline PD (and, more broadly, the Cluster B personality disorders) and generally is regarded as a personality–temperamental dimension, not a pathological dimension per se (e.g., Siever, Torgersen, Gunderson, Livesley, & Kendler, 2002; Skodol, Siever, et al., 2002; Soloff, Kelly, Strotmeyer, Malone, & Mann, 2003). In a related vein, Iacono et al. (1999) presented evidence that trait disinhibition—which they describe as the inability or unwillingness to inhibit behavioral impulses, and which has been linked to a range of externalizing psychopathology—is a manifestation of underlying central nervous system processes associated with various psychophysiological anomalies, including electrodermal responses and event-related potentials. Fowles (2000) also noted that electrodermal hyporeactivity was a reliable correlate of psychopathy–undersocialized aggressive conduct disorder and that it appears to relate to an impulsivity dimension of psychopathy.

The links between manifest behaviors and specific physiological and neurological reactions undoubtedly are complex; yet, with continued research, gradually the biological underpinnings of temperament–personality no doubt will be elucidated. Such research will be facilitated to the extent that the dimensions and

facets of observed personality traits can be clarified. That is, the more reliably and validly target behaviors and behavioral sets, as well as the personality dimensions they represent, are defined and assessed, the more likely it will be that meaningful associations with biological variables are discovered. Conversely, it seems unlikely that specific biological correlates of broad, heterogeneous targets such as borderline PD will be identified.

Dimensions versus categories. Throughout this article, temperament–personality variables have been described as dimensions, whereas disorders—both Axis I and Axis II—generally have designated conventional, *DSM*-based categories. Yet, evidence indicates that for many disorders, the reliability and validity with which they can be assessed increases to the extent that they are treated as dimensions rather than categories (Asendorpf, 2003; Flett, Vredenberg, & Krames, 1997; Widiger, 1992a). In the face of such data, how are we to justify use of the “industry standard” categories? Researchers have begun to grapple with this problem.

Recently Waldman and Lilienfeld (2001) and Haslam (2002) have confronted the widely held notion that there is a single categorical type and have distinguished a number of different types of categories ranging from *practical* categories that represent simply the dichotomization of a dimension either by using “most valid” cutpoints (e.g., high blood pressure) or by focusing only on the dimensions’ extremities (e.g., introverts vs. extraverts), to *configural* categories that emerge from the conjunction of extremity on several underlying dimensions, to *natural* categories that have a specific causal basis and appear to be quite rare biologically with regard to mental disorder (e.g., Huntington’s disorder).

Haslam (2002) argues further that the “dimensional–categorical debate” in psychology–psychiatry actually is a debate over two kinds of categories. That is, dimensionalists are not arguing that there is no useful distinction between, for example, persons low and high on depression severity or maladaptive personality traits, but rather that the distinction between normality and psychopathology is based on extrinsic, pragmatic grounds, such as increased risk for self-harm, not on an underlying, natural discontinuity. Conversely, according to Haslam (2002), categoricalists are arguing that the distinction is intrinsic, even if imprecise (e.g., Santor & Coyne, 2001). Thus, understanding of the etiology of psychopathology is needed to resolve this debate. Should etiological explanations be consistent with those that produce continuous dimensions, the former will have “won the day,” whereas the latter will hold sway should the underlying causes prove to be such that they lead to discontinuity.

Further, categoricalists who argue that the *DSM* disorders are categories on the basis of underlying discontinuities bear the burden of proof to identify the category boundaries in a relatively precise manner. To date, however, the empirical data have been supportive of the existence of such boundaries for only a few disorders (see reviews by Haslam, 2003; Haslam & Kim, 2002).

Toward a Comprehensive Hierarchical Model for *DSM–V* (or *VI*?)

A great deal of ground has been covered in this review, so I recap here the key points of the proposed comprehensive, hierarchical framework of relations among temperament, personality and psychopathology to accompany or even substitute in *DSM–V* for the current set of disorders. First, the framework is both structural

and developmental: It is intended to explain comorbidity, encompassing both more and less prevalent disorders, in virtually all combinations, as well as the overlap of disorders with personality traits by positing that both personality and psychopathology develop from three underlying, genetically based temperament dimensions: negative affectivity, positive affectivity, and disinhibition. This dimensional framework incorporates the related concepts of severity of psychopathology and strength or extremeness of personality traits, and existing data suggest its applicability across populations—patient and nonpatient, in the United States and elsewhere—and across measurement modalities.

This framework can account for the fact that comorbidity is rampant but is neither random nor uniform. That is, general vulnerability factors are necessary, but far from sufficient, to explain the relevant phenomena. The differential comorbidity rates found even between pairs of similar disorders, indicate that some pairs of disorders share more (and/or larger) underlying factors than do others. Thus, the framework proposes that initially broad temperament dimensions are differentiated through development, and more specific personality–disorder associations are due to sharing one or more differentiated traits and, most likely, one or more etiologically relevant environmental factors as well. Moreover, the model, being hierarchical, accounts for heterogeneity within disorders as currently defined. Heterogeneous disorders can be parsed into their components, permitting delineation of more specific factors that distinguish among psychopathological variants. Watson (2005) has made strides toward identifying the facets of several disorders that are continuous into the normal range, suggesting they may have personality-subtrait counterparts.

This comprehensive framework also may be extended beyond personality and psychopathology, bringing such variables as social functioning and violent behavior within its domain. It is a causal model that provides for development and diagnostic sequencing. Finally, it is important to point out that it is a scientific model—simultaneously theoretically and empirically based. With the current knowledge of psychopathology, researchers can no longer afford the “luxury” of an atheoretical taxonomy if studies are to continue to advance the field. To those who argue that the *DSM* must serve many masters and thus ultimately must yield to important practical considerations, I respond with James Clerk Maxwell’s famous dictum, “There is nothing more practical than a good theory.”

Clearly, a fully specified model of personality–psychopathology relations is a long way off, but the current proposal provides a framework within which to develop specific working models. Whereas it might be premature to adopt such a framework for all of *DSM–V*, at a late 2004 conference on refining the research agenda for *DSM–V*, cosponsored by the American Psychiatric Association and three NIH agencies, there was a remarkable consensus that a dimensional model of personality disorder would be preferable to the current system. Moreover, we are not so far away from developing a general dimensional model that the basic direction in which we need to move is unclear either conceptually or empirically.

Conceptually, the consideration that whereas the Axis I–Axis II distinction had great utility when introduced in *DSM–III*, it may have become a barrier to progress and now should be set aside, is a major step forward, as is the general acknowledgment that dimensions are more powerful, and likely more valid, explanatory

constructs. Empirically, the demonstration that broad personality dimensions explain much of the overlap among different types of psychopathology is a giant step forward from the earlier held position that personality and psychopathology are independent domains.

Finally, the importance of the gradual breaking down of traditional disciplinary boundaries and the increase in interdisciplinary research for developing a comprehensive model cannot be overestimated. As described by Plomin and Caspi (1998), for example, “genetic approaches to complex traits such as personality have moved from single-gene models and methods to approaches that can identify genes of small effect size in multiple-gene systems called quantitative trait loci” (p. 387). The success of these approaches, however, depends heavily not only on advances in molecular biology but equally on the availability of psychometrically reliable and valid measures of personality and psychopathology. Moreover, if innate temperament dimensions underlie personality–psychopathology links, developmental psychologists will be relied on to direct the longitudinal research needed to bridge from infant temperament to adult personality.

The greatest barrier to adoption of the proposed framework for *DSM-V* may be that it presents a challenge to categorical diagnoses, some of which have strong advocacy groups that likely will oppose any perceived attempts to undermine their efforts on behalf of those diagnosed with the disorder. Thus, it is important to emphasize that dimensions and categories are not fundamentally incompatible. Indeed, dimensions can underlie even natural categories. For example, Huntington’s disorder is caused by an abnormally high number of CAG nucleotide repeats. A greater number of repeats is associated with earlier age of onset, and persons having a certain intermediate range of repeats show symptoms of the disorder but may or may not develop the full-blown syndrome (Meehl, 2001). This fact in no way detracts from its status as a serious disorder, and the same may be true for the full range of psychopathology.

If the framework proposed leads, as hoped, to more sophisticated research that accounts well for increasing amounts of data in multiple domains, ranging from molecular biology to personality trait structure to developmental psychopathology, researchers will gain the knowledge needed to clarify dimension–category relations and to determine the nature of the categories formed from underlying dimensions in various domains. It is possible, for example, that some personality disorders are indeed best diagnosed by using the prototypic categorical model that was adopted explicitly for all Axis II disorders beginning with *DSM-III-R*. In the meantime, if it can be agreed that the data do not support classical categorical models for many, if not most, types of psychopathology, but that—even so—there is great practical utility in having categories for certain decision-making purposes, then a rapprochement may be possible, perhaps even for *DSM-V*.

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