CHAPTER I

The Personality Disorders

History, Classification, and Research Issues

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In the dialogue between theory and experience, theory always has the first word. It determines the form of the question and thus sets limits to the answer.

—FRANÇOIS JACOB (1982, p. 15)

Theory without data runs the risk of ungrounded philosophizing, but data without theory lead to confusion and incomprehension. The definition of the personality disorders in DSM-III (and its successors, DSM-III-R, DSM-IV, and DSM-IV-TR) as well as their separation from other clinical syndromes (Axis I disorders) greatly enhanced the legitimacy of this class of psychopathology as an area for research and personality disorder research has shown unprecedented and exciting expansion over the past 25 years. It was the thesis of first edition this volume (in the spirit of the quote from François Jacob) that the time had come to articulate contrasting and competing (at times, partially overlapping) theories of personality disorder in order to stimulate some intellectual clarity within the growing body of empirical data on the personality disorders. We remain convinced that personality disorder research will only move forward appreciably when guided by rich and sophisticated models. With the second edition of this volume, it remains our hope that the models and theories of personality pathology
presented here will continue to serve not only as an organizing function but, perhaps more important, as useful heuristics for continuing empirical research on the personality disorders.

A BRIEF HISTORICAL OVERVIEW OF PERSONALITY DISORDER THEORIES

One can trace the conceptualization and articulation of personality and related personality pathology in the history of psychiatry and clinical psychology, and in the development of personality theory and research in the tradition of academic psychology. Whereas there has traditionally been considerable interaction between psychiatry and clinical psychology, the writings and research generated by the field of academic psychology have been focused mainly on normal personality and had little relationship to the clinical traditions. This separation was promoted not only by the physical locale of many clinicians (i.e., hospitals and medical centers vs. university departments of psychology) as well as the reasonable aims of both groups (clinicians diagnose and treat the impaired and dysfunctional, whereas academic personality psychologists view normative functioning and normal personality organization as the object of study). Our goal here is not to review the history of personality theory and related personality disorder theory. Rather, our major focus here is to briefly summarize the conceptualizations of those personality theorists who have ventured into the area of personality disorders or the relationship of personality to pathology. Our overview is, therefore, necessarily selective and makes no claim to be exhaustive; we provide references which the interested reader can pursue.

Vaillant and Perry (1985) trace the articulation in the history of clinical psychiatry of the notion that personality itself can be disordered back to work in the 19th century on “moral insanity.” By 1907, Kraepelin had described four types of psychopathic personalities. The psychoanalytic study of character pathology began in 1908 with Freud’s *Character and Anal Erotism* (1980/1959) followed by Franz Alexander’s (1930) distinction between neurotic character and symptom neuroses and by Reich’s (1945) psychoanalytic treatment of personality disorders.

Clinical Psychology and the Assessment of Personality Pathology

The most unique contribution of clinical psychology to the history of personality and personality pathology was the development and application of psychological testing instruments for the assessment of personality pathol-
ogy in clinical settings. The flowering of the traditional “full battery” approach to personality assessment in clinical settings is exemplified in the writings of Rappaport, Gill, and Schafer (1968). According to these authors, diagnostic testing of personality and ideational content was concerned with “different types of organizations of the subject’s spontaneous thought processes, and attempts to infer from their course and characteristics the nature of his personality and maladjustment” (p. 222). The focus of this traditional approach was shaped by the environment of the day—that is, by the psychiatric diagnostic system in vogue (officially and unofficially) and the predominantly psychodynamic treatment approaches.

In contrast to the full-battery traditional approach, the Minnesota Multiphasic Personality Inventory (MMPI), the well-known self-report inventory, was first published in 1943 by Starke Hathaway, PhD, and J. Charnley McKinley, MD (Hathaway & McKinley, 1943/1983), with scales measuring salient clinical syndromes of the day such as depression, hypochondrias, schizophrenia, and others. The fact that the MMPI was called a personality test is itself a manifestation of the intertwining of concepts of clinical syndromes and personality/personality pathology. Interestingly, however, only two (Scale 4: Psychopathic deviate and Scale 5: Masculinity/Femininity) of the original nine clinical scales actually assessed constructs akin to personality traits or attributes; Scale 0, developed later, was designed to assess social introversion.

In more recent times, there has been less emphasis in clinical assessment in psychiatric settings on projective tests used to assess personality defined in a global sense (owing to concerns about validity, see Lilienfeld, Wood, & Garb, 2000), and more focus on the development of successors to the MMPI that have used advances in psychometric development and are more closely tied to a diagnostic system that makes a distinction between Axis I syndromes and Axis II personality pathology. Illustrative of these instruments are the Millon Clinical Multiaxial Inventory (MCMI; and its successors, the MCMI-II and MCMI-III) and the Personality Assessment Inventory (PAI; Morey, 1991). Given the historical role and importance attached to the clinical interview procedure in psychiatry as well as the advances achieved in the design of structured interviews for the major mood disorders and psychoses (e.g., the Present State Examination [PSE] and Schedule for Affective Disorders and Schizophrenia [SADS]) through the 1970s, it was a natural development to see the careful development of semistructured interviews (e.g., the Structured Clinical Interview for DSM-III-R [SCID-II; Spitzer, Williams, & Gibbon, 1987] and the PDE [Loranger, 1988]) that reliably assess personality disorders as described in the DSM system. Today the standard and most well-accepted approach to the diagnosis of personality disorders remains the structured interview approach with a number of excellent interviews to choose from (International Per-
sonality Disorders Examination [IPDE; Loranger, 1999]; Structured Interview for DSM-IV Personality [SIDP-IV; Pfohl, Blum, & Zimmerman, 1997]; Structured Clinical Interview for DSM-IV Axis II Personality Disorders [SCID-II; First, Spitzer, Gibbon, & Williams, 1997]). It is worth noting that unlike its peers, the IPDE is configured to assess both DSM-IV and ICD-10 diagnostic criteria for personality disorders (Loranger et al., 1994). Axis II structured interviews still remain primarily used in research settings, their integration into training program curricula has increased, and their application in clinical work is encouraged. The interested reader is referred to Zimmerman (1994) for an excellent review of the many critical issues that surround the diagnosis of personality disorders (see also Livesley, 2003; Loranger, 1991a; Loranger, 2000).

Other self-report personality questionnaires have been developed to capture the dimensions thought to be related to the diagnostic criteria on Axis II. This would include the work of Livesely and colleagues (e.g., Schroeder, Wormworth, & Livesley, 1994, 2002) and Clark (1993). Some would speculate that the personality disorders involve maladaptive and inflexible expressions of the basic dimensions of personality as captured in the popular five-factor model of personality (see Costa & McCrae, 1990; John, 1990) or the interpersonal circumplex model of personality (e.g., Wiggins & Pincus, 1989, 2002). Energetic efforts have been made to describe the personality disorders on Axis II in terms of the five-factor model from a conceptual point of view (Costa & Widiger, 2002; Morey, Gunderson, Quigley, & Lyons, 2000), with some measure of consistent empirical support (Saulsman & Page, 2004; Schroeder et al., 1994, 2002) (see below). An alternative dimensional model that is firmly rooted in underlying neurobehavioral systems conceptualizations (e.g., Depue & Lenzenweger, 2001, and Chapter 8, this volume) is also now available as an alternative to the nonbiological lexically based five-factor approach. Finally, a comprehensive self-report instrument, developed within a clinical setting, now exists that is designed to capture both the putative dimensions underlying normal personality as well as those domains relevant to the assessment of DSM-IV-defined Axis II disorders (i.e., the OMNI Personality Inventory and OMNI-IV Personality Disorder Inventory, Loranger [2002]).

Academic Psychology

The field of personality within the larger academic world of psychology has been a time-honored tradition that has suffered its ups and downs (see Hogan, Johnson, & Briggs, 1997; Pervin & John, 1999). An examination of the reviews of the field of personality in the Annual Review of Psychology provides an historical sense of the academic debates in the field and the
issues that were passionately fought over in the past (e.g., do traits exist?) (e.g., Funder, 2001). In 1990, Pervin, a senior observer of the field, enumerated the recurrent issues in personality research and theory, some of which are relevant to and may be rethought (and fought) in the field of personality disorders: (1) definition of personality; (2) relation of personality theory to psychology and other subdisciplines, including clinical psychology; (3) view of science; (4) views of the person; (5) the idiographic–nomothetic issue; (6) the internal–external issue; (7) the nature–nurture issue; (8) the developmental dimension; (9) persistence and change in personality; and (10) emphasis on conscious versus unconscious processes.

Traditionally, academic personality psychologists studied nonclinical populations, they were more interested in the “normal” personality and consequently gave little attention in their theories to abnormal personality or personality pathology. For example, Gordon Allport (1937) one of the early leaders of normative personality theory, criticized Freud for suggesting a continuum of personality pathology; instead he postulated a division in personality processes between the normal personality and the neurotic personality. The tendency on the part of academic personologists to theorize about and research normative personality most probably reflects not only their substantive area of interest (i.e., normalcy) but also their training (i.e., absence of training in clinical methods and lack of exposure to psychopathological populations) and place of work (the university psychology department as opposed to the clinic and/or psychopathology laboratory). Until relatively recently, there were few academic personologists who extended their theorizing or empirical work to the pathological personality realm, exceptions such as Henry Murray (1938) and Timothy Leary (1957) are well known. This is a theme that will reverberate through the second edition of this volume: In what setting does the theoretician of personality disorders work, and how does that affect the resulting theory?

THE NEO-KRAEPELINIAN REVOLUTION, DSM-III, AND THE BIRTH OF AXIS II

Just as the academic personologists have focused on the normative personality and its structure and development, those in the clinical area (clinical psychologists, psychopathologists, psychiatrists, psychoanalysts) focused their attention and efforts on the pathological variations seen in human personality functioning. To begin, DSM-I (American Psychiatric Association, 1952) provided four categories of psychiatric disorder: (1) disturbances of pattern; (2) disturbances of traits; (3) disturbances of drive, control, and relationships; and (4) sociopathic disturbances. These and subsequent categories of personality disorder in DSM-II (American Psychiatric
Association, 1968) were used only when the patient did not fit comfortably in other categories. The personality disorders defined on a separate axis, whether or not a symptomatic disorder was present, first appeared in DSM-III in 1980. The interested reader is referred to Millon (1995) for one of the best historical reviews of both the process of DSM-III’s construction and its formulation of personality disorders as well as a more general prior history of personality disorders.

The advent of DSM-III and its successors (DSM-III-R, DSM-IV, DSM-IV-TR), which use a multiaxial diagnostic system that makes a distinction between clinical syndromes (Axis I) and personality disorders (Axis II), both brought into sharp focus and encapsulated the controversy concerning the nature and role of personality/personality pathology in the history of psychiatry and the history of modern personality research. The introduction of a distinction between clinical syndromes and personality disorders as well as explicit description of personality pathology within DSM-III by no means brought about unanimity and intellectual peace. In many ways, the introduction of the formal Axis II classification scheme in 1980 ushered in what would begin an exceedingly active initial phase of personality disorder research—namely, clarification and validation of the personality disorder constructs and beginning efforts at illumination of the relations between personality and personality disorder (see section “Normal Personality and Personality Disorder”).

Numerous examples can be cited of active productive discussion resulting from the introduction of DSM-III and subsequent DSM nomenclatures. Some workers have argued from accumulated clinical experience that the particular disorders defined in Axis II do not adequately match clinical reality. For example, distinctions between hysterical and histrionic personality disorders have been neglected in Axis II (see Kernberg & Caligor, Chapter 3, this volume); or the very existence of pathological masochism has been only variably recognized and fraught with debate; or clinically rich concepts related to the classic psychopathy notion have been given diminished attention in favor of a behaviorally defined antisocial personality disorder concept. Others have argued at a more basic level that DSM Axis II criteria do not meet scientific standards (Clark, 1992). For example, Clark (1992) suggested that the Axis II personality criteria were not optimally grouped into “disorders” and do not accurately reflect trait dimensions.

These issues highlight some of the difficulties that coexist with the alleged benefits of an “atheoretical” approach proclaimed by the architects of DSM-III and its successors. The development of DSM-III, really the culminating event of the so-called neo-Kraepelinian revolution in psychiatry (see Blashfield, 1984), justifiably sought a diagnostic system that would provide explicit, usually behavioral, criteria that could be reliably assessed.
Such a methodological approach to the definition and operationalization of constructs was long known in psychology (see Cronbach & Meehl, 1955) and its utility was established. Therefore, many psychiatric and clinical psychology researchers welcomed the overhaul of the diagnostic system with open arms. Unfortunately, however, it is our sense that during the rush for diagnostic reliability in the 1970s, the value placed on reliability (something all could agree on) became conflated with or necessarily implied the need for an “atheoretical” approach to diagnosis. The reasons for adopting an atheoretical approach in the contemporary DSM systems were surely complex and were likely a necessity in order to have the diagnostic systems adopted despite parochial interests of the various schools of psychotherapy and clinical practice. In other words, such an atheoretical approach was necessary given that the product (i.e., the DSM) was a quasi-political one, albeit one with important scientific impact. Our point should be obvious; we firmly endorse a methodological approach to diagnosis that is rigorous and displays adequate reliability and validity; however, such an approach need not necessarily be “atheoretical.” In 1996 when we initially presented the first edition of this volume it stood in sharp relief to the atheoretical approach of the DSM system. Since that time there has been an increase in research interest in model-guided research in personality disorders, which, to our minds, represents some of the most exciting work in this area. Nonetheless, we still see the need for a compilation of current substantive models of personality disorder as the official nomenclature (DSM-IV) as well as planned revisions (DSM-V) are likely to continue with the atheoretical perspective on personality disorders. We present the second edition of this volume precisely because the theories and models herein will not only guide empirical measurement of personality pathology but also provide a continuing context in which empirical results can be examined and understood; new hypotheses may be generated; and, ideally, etiology, pathogenesis, and development of personality disorders shall be illuminated.

ISSUES OF CONCERN FOR SCIENTIFIC THEORIES OF PERSONALITY DISORDER

As we noted earlier, the advent of Axis II in the multiaxial system introduced by DSM-III and the explicit definition of personality disorders has stimulated scientific and clinical interest in personality pathology. DSM-III’s effect on both research and practice was unambiguous and rather dramatic, primarily leading to an increase in the rate at which Axis II diagnoses were made in clinical settings (e.g., Loranger, 1990) but also a marked increase in the number of research studies directed at personality pathology. A review of articles in the prominent scientific psychopathology jour-
nals (e.g., Archives of General Psychiatry and Journal of Abnormal Psychology) since 1980 will reveal a noteworthy increase in the number of research reports on Axis-II-related topics. This era of scientific growth was rapid and substantial enough, in fact, to warrant development and publication of the specialty journal Journal of Personality Disorders as well as the formation of the International Society for the Study of Personality Disorders. The Journal of Personality Disorders continues to thrive and more and more reports on personality disorders have appeared in the general psychopathology and psychiatry journals (Archives of General Psychiatry, American Journal of Psychiatry, Journal of Abnormal Psychology). There is evidence as well that research funding has begun to increase in the area of personality disorders. By almost any objective index, the rate of scholarly inquiry into personality pathology has seen dramatic growth in the 25 years since the advent of DSM-III and the decades to come are almost certain to see sustained interest in the personality disorders.

The contributors to this volume once again have articulated their respective views on the nature and organization of personality pathology, with numerous updates and revisions to their 1996 positions. In contrast to the “atheoretical” position of DSM-IV, each of our contributors has taken a stand with respect to the fundamental nature of personality disorder, transcending an approach (i.e., DSM-IV) that explicitly describes, but, unfortunately, eschews, explanation. Consistent with the contents of this volume, our hope is that future scientific work in personality disorders will continue to become increasingly theory-guided. The benefit of such a development in the scientific approach to personality pathology lies in the power achieved through formulating testable and falsifiable models that are not merely descriptive but, rather, emphasize etiology, mechanism, and lifespan developmental sequelae of personality pathology. An additional benefit of theory-guided and empirically based models of personality pathology, of course, would be the further development and refinement of rational treatments for personality disorders that are more closely tailored to the specific deficits and dysfunctional attributes presented by individual personality pathologies, an aim embodied in the clinical approach known as “differential therapeutics” or “systematic treatment selection” (Beutler & Clarkin, 1990). Those involved in interventions research have pointed out that the field has focused on a few questions, such as the impact of a few described treatments as compared to no treatment or treatment as usual, and the generalization of treatments across problem areas and settings (National Institute of Mental Health, 2002). Other questions, most relevant to practical clinical work, such as how and why specific psychotherapeutic interventions work, have been ignored. From a technical point of view, this concern about how treatments work involves the search for moderators and mediators of change, necessitating hypothesis-generating analyses. This effort to
examine the mediators and moderators of change in psychotherapy will necessitate clear articulation of the theory of personality disorders and their key elements (Clarkin & Levy, in press). Finally, with increased knowledge of etiology and mechanism, one could ultimately consider issues related to the prevention of personality disorders, though clearly a Herculean task that challenges the imagination at present. The importance of theory-guided approaches to personality pathology is only amplified when one considers the pervasiveness of personality disorders in general clinical practice, clinic populations, and the population at large. High-quality epidemiologically derived estimates of the prevalence of Axis II disorders are now available (see below) and they suggest a prevalence between 9 and 13% in the community (Lenzenweger, Loranger, Korfine, & Neff, 1997; Torgersen, Kringlen, & Cramer, 2001; Samuels et al., 2002). These figures accord well with the initial “guesstimates” of between 10 and 15% (Weissman, 1993). Clearly, assuming 1 in 10 persons is affected by personality pathology, it is safe to assert that personality pathology is ubiquitous and we are, therefore, challenged to understand the “hows” and “whys” of personality disorder development as well as to discern the most efficient and valid classification approach for such disorders. On a related note, these prevalence estimates suggest personality disorders do represent a public health concern, and funding for research on the personality disorders is deserving of a nontrivial enhancement.

We are committed to and advocate a scientific approach to the study of psychopathology and the study of personality disorders is no exception. In this framework the necessity of reliable assessments, measures, and procedures that possess suitable validity is axiomatic; however, despite good instrumentation, we anticipate future personality disorder theories and research will be characterized by “false starts,” forays down “blind alleys,” and the customary slow progress of “normal science” punctuated by periodic substantive advances and moments of genuine clarity. This section of our introductory chapter is intended to highlight issues that any scientific theory of personality disorder must consider. We intend to raise more questions than provide answers. As most research in personality disorders is probably best considered as occurring within the “context of discovery” (Reichenbach, 1938), it seems prudent to us to draw attention to a variety of substantive and methodological issues that should guide research in this rapidly developing area in psychopathology research. We highlighted these issues in the first edition of this volume and most, if not all, of them remain unresolved and in need of continued examination. Personality disorders research, we suggest, is particularly interesting as it will need draw on the lessons we have learned, methodological and otherwise, from the study of other forms of severe psychopathology (e.g., schizophrenia and affective illness). However, it is particularly ripe with challenges that are relatively
unique to this domain of pathology. For example, Where are the boundaries between “normal” personality and personality disorder? Can one define a “case” of personality disorder in the absence of marked impairment or distress? Furthermore, one could argue that some of the specific research challenges that personality disorder psychopathology affords may not be readily illuminated by the clues we have gleaned from other areas of study and reliance on previous insights may be less useful than having the wrong map for a territory. In short, in the study of personality pathology we rely on the efforts and insights many psychopathologists—including those contributing to this volume—to chart these new territories.

In citing several methodological and/or substantive issues below, it is not our intention to suggest previous personality disorders research that has addressed these issues, directly or in part, has been somehow lax (though some clearly has been!). We are mindful that personality disorders research has only just “taken off” in the last 25 years and many would regard model-guided work in this area as just beginning to emerge (much effort in the 1980s concerned development of assessment technologies). We seek, therefore, to encourage more extensive and ambitious model-guided work in this area. We remain convinced that it continues to be sensible to highlight themes that remain troubling, challenging, and unresolved in personality disorders research. Finally, as in the first edition, we would like to stress the fact that the literature we cite later is necessarily highly selected due to space constraints and our review is not intended to be exhaustive. Our examples, as one might anticipate, will hail primarily from recent research in personality disorders and will, therefore, be heavily influenced by the prevailing DSM nomenclature. Although many of the issues we shall raise derive from studies that focus on DSM defined personality pathology, our comments are not intimately linked to that taxonomy. We continue to view the following issues, not ordered in terms of importance, as largely unresolved and in need of further work as well as worthy of considerable attention by any comprehensive theory of personality disorder, including those contained in this volume.

Normal Personality and Personality Disorder: Questions of Continuity and Structure?

With the advent of specific diagnostic criteria and a polythetic approach to classification, Axis II of DSM-III represented an opportunity for rich theoretical discussion of and empirical research on the relations between personality disorders as conceptualized by psychopathologists and normal personality as studied by academic personality psychologists. Theoretical discussion to date has focused on three key conceptual issues, namely, (1) the dimensional versus categorical nature of personality disorders (Costa &
Widiger, 2002); (2) the distinction between normal and pathological personality features (e.g., social isolation as possibly representing low sociability vs. suicidal attempts as unrepresented on any “normal” dimension of personality; e.g., Wiggins, 1982); and (3) the nature of the basic processes and structure underlying both personality disorders and normal personality (cf. Cloninger, Svrakic, & Przybeck, 1993; Depue & Lenzenweger, 2001, Chapter 8, this volume; Rutter, 1987; Livesley, Jang, & Vernon, 1998). In short, the “normal” personality correlates, if any, of specific personality disorders remain but tentatively specified. Most important, it remains unclear to what extent personality disorder symptoms are continuous, albeit exaggerated, extensions of normal traits. For example, although there are now 15 studies of the associations between the “five-factor model” and personality disorder dimensions (Saulsman & Page, 2004), these studies cannot address the issue of continuity between personality and personality disorder domains due to their fundamental design (i.e., cross-sectional correlations). Furthermore, research to date has not effectively addressed, using techniques such as confirmatory factor analysis, the comparability or goodness of fit between the overall DSM personality disorder taxonomy and the empirically based dimensional structures observed in contemporary personality research such as the interpersonal circumplex (Leary, 1957; Wiggins, 1982) and established multidimensional/factorial models (e.g., the “big five” model; see Digman, 1990, or John & Srivastava, 1999, for excellent reviews; and Block, 1995, 2001; Westen, 1996; and Shedler & Westen, in press; for strident criticism; the “three superfactor” model of Tellegen, 1985, and the three-factor temperament model, e.g., Buss & Plomin, 1984). That is, it is not clear how comparably organized personality pathology is at the latent level vis-à-vis normal personality—stated differently, do three, four, or five major dimensions also continuously underlie personality disorders? Although normal personality research now suggests that somewhere between three and five factors adequately capture the variation in the primary descriptors of personality, the same cannot be said readily for personality pathology. Moreover, the correspondence between the primary factors of personality and personality disorder remains to be explored in a fine-grained manner beyond simple correlational analyses (e.g., Saulsman & Page, 2004). This is a critical issue as it is not an uncommon experience to see reviewer comments on manuscripts or hear comments at conferences and National Institute of Mental Health (NIMH) study sections suggestive of the (mis)impression that the five-factor approach is an adequate conceptualization of normal personality and the personality disorders (see Depue & Lenzenweger, Chapter 8, this volume).

Most efforts at searching for the personality correlates of personality disorder focus principally on any obtained correlations; however, the
meaning of such discovered associations looms large. A large number of studies have focused on the relations between normal personality and personality disorder in recent years, and many more will do so in the future. However, it is worth noting that it is likely that most of these studies will not address directly the issue of whether or not personality disorder symptoms are continuously versus discontinuously distributed in the population if they rely primarily on demonstrating correlations among these variables (i.e., Are personality disorder symptoms exaggerations of normal traits?). An implicit assumption of the work generating associations between normative and personality pathology measures has been that an association between such variables suggests a continuity between the phenomena (e.g., Costa & McCrae, 1990; Costa & Widiger, 2002). This implicit assumption is fraught with substantive and statistical pitfalls. It could quite conceivably be that in some instances no genuine (i.e., real and natural) connection between a dimension of personality and a personality disorder variable exists even though a statistically significant correlation may exist between them. To begin to address this issue an exceptionally large randomly ascertained general population sample of individuals would need to be assessed for personality disorder symptoms and the distributions of these symptoms should be examined for the existence of qualitative discontinuities as evidenced, possibly, by “bimodality” (see Grayson, 1987, for a provocative review of this concept) and through application of complex statistical procedures such as admixture analysis (e.g., Lenzenweger & Moldin, 1990), finite mixture modeling (Titterington, Smith, & Makov, 1985; McLachlan & Peel, 2000), or taxometric analysis (Meehl, 1992, 1995; cf. Korfine & Lenzenweger, 1995, Lenzenweger & Korfine, 1992; Waller & Meehl, 1998). Comparable work will, of course, need to be done on normative “dimensions” of personality as well before proceeding to inferences concerning the continuous relations between personality and personality disorder (Endler & Kocovski, 2002).

A question concerning the very existence of “dimensional” continuities and “categorical” (or “typological”) discontinuities in either the personality or personality disorder realms itself remains controversial (see Meehl, 1992). In short, regardless of the application of appropriate statistical procedures to such problems, there remain quasi-ideological preferences for either dimensional or categorical conceptualizations of personality-related phenomena. The “dimensional versus categorical” issue was been discussed extensively in relation to personality pathology through the 1980s, with some psychologists advocating a dimensional approach (Widiger, 1992), whereas the psychiatric community remained essentially wed to a categorical framework (American Psychiatric Association, 1994). The “dimensions versus categories” discussion with respect to personality disorders continues to this day. The reasons for such preferences are not always im-
mediately discernible, though psychiatry has long preferred a typological approach to psychopathology (consistent with traditional medicine) and this approach is therefore familiar, facilitates communication, and is consistent with clinical decision making (American Psychiatric Association, 1994; Widiger, 1992). Although much of the “categories” versus “dimensions” debate concerns professional diagnostic or assessment-style preferences, there is a deeper level of analysis to this problem that has garnered the attention of a number of psychopathologists with interest in the structure of nature in psychopathology. Normal personality research has long preferred a dimensional or continuum view of personality and other behavioral phenomena (Meehl, 1992, 1995), due perhaps in part to reliance on parametric statistics and a focus on the study of normative aspects of psychological functioning. Interestingly, as a “dimensional” approach to personality pathology has become increasingly of interest to psychiatry (cf. American Psychiatric Association, 1994), psychological research has seen a resurgence of interest in the detection of discontinuities, “types,” or “taxa” in a variety of psychological and psychopathologic realms (see Meehl, 1992, 1995; cf. Lenzenweger & Korfine, 1992). For example, taxometric data generated using the MAXCOV technique developed by Meehl suggests that schizotypy (Lenzenweger & Korfine, 1992; Korfine & Lenzenweger, 1995) and psychopathy (Harris, Rice, & Quinsey, 1994) are taxonic at the latent level; the published taxometric data for borderline personality disorder (Trull, Widiger, & Guthrie, 1990; Haslam, 2003) are also consistent with a taxonic model (but see also Rothschild, Cleland, Haslam, & Zimmerman, 2003). The proper application of taxometric techniques to the study of psychopathology requires great care and guidelines have recently been proposed for future studies to avoid some of the difficulties that appeared in some earlier efforts (Lenzenweger, 2004).

Other than the need for an appropriate methodological approach in the determination of continuity versus discontinuity between personality and personality disorder constructs, theoretical conjectures concerning the relationships between personality disorders (and personality disorder symptoms) and normal personality must take into account the divergent behavioral, affective, attitudinal, and cognitive domains covered by these two broad areas of scientific inquiry. Are there normative counterparts of accepted personality disorder symptoms? Clearly, some personality disorder symptoms will not be expected to have normative personality counterparts (e.g., suicidal behaviors and self-mutilation). The normative construct sociability, on the other hand, clearly ranges from “high” to “low” and, perhaps, an individual with schizoid personality disorder shares much in common with a person described as displaying low sociability. All things considered, though, it is somewhat unrealistic to conceive of precise one-to-one correspondences between personality disorder symptoms and norma-
tive personality traits. We readily predict that noteworthy correspondences will be observed between several of the major dimensions underlying normal personality (or temperament) and personality disorder symptomatology; however, the meaning and interpretation of such correspondences should prove a challenge to personality disorder theorists. At a minimum, we suggest that models seeking to relate personality systems with personality disorder features do so in a manner that works rationally from the underlying personality systems to possible personality disorder configurations.

Finally, although DSM-IV has presented us with a “structure” for organizing personality pathology, namely, the disorders of Axis II, any meaningful consideration of the relations between personality pathology and normal personality must be cognizant of the possibility that the Axis II arrangement may have little genuine correspondence to the true (or, natural) latent organization of personality disorder symptomatology. By this we mean, in short, that DSM-IV presents us with 10 disorders grouped into three so-called clusters, the odd–eccentric, the impulsive–erratic, and the anxious–avoidant clusters. However, there are no published data derived from a large sample (N > 1,300, assuming 10 subjects per Axis II diagnostic criterion) of carefully clinically assessed cases in which analyses, conducted at the level of individual items (i.e., criterion level), confirm the DSM-IV cluster structure, or even the disorder structures themselves. Some factor-analytic studies have obtained three-factor solutions, corresponding broadly to the three “clusters” of the DSM-III-R/DSM-IV Axis II taxonomy. However, these studies analyzed data at the level of disorders and they seemed unaware of the fact that the data that were analyzed had been structured a priori by being organized into 10 or 11 predefined disorders.

Given the relatively high degree of overlap that can be found among the currently defined Axis II personality disorders, in the form of both correlations among symptom dimensions and/or rates of co-occurrence of categorical diagnoses (Korfine & Lenzenweger, 1991; Widiger et al., 1991), it seems quite reasonable to hypothesize that item-level multivariate analyses of the domain of symptoms found on Axis II will reveal perhaps but a handful of meaningful (i.e., interpretable) factors. While the preliminary work on this problem would by definition need to be more exploratory in nature, a confirmatory approach could be adopted for assessing the fit between an emergent structure or model and new sets of data. An illustration of such an approach can be found in the schizophrenia literature wherein the latent structure of positive and negative symptoms was resolved through application of confirmatory factor analysis and the systematic comparison of multiple competing models of latent structure (Lenzenweger & Dworkin, 1996). Efforts to discern the latent structure of
personality disorder symptomatology, whether specified by DSM or an alternative model such as one of those in this volume, must bear in mind the effect of the use of cases selected solely from clinical settings on obtained results. In short, those individuals who come to hospitals and clinics for treatment tend to be more severely affected in general and this fact alone will likely increase the degree of overlap (or correlation) seen across forms of personality pathology. Moreover, the more ill a sample is on the whole, the less likely will be subthreshold cases, which are important of “filling in” the range of personality pathology as it occurs naturally. Thus the impact of sampling on efforts to illuminate the latent structure of personality pathology must be considered.

The State–Trait Issue in Relation to the Definition and Diagnosis of Personality Disorders

Implied in the DSM definition of personality disorders is the assumption that state factors such as anxiety and depression should not substantively affect the assessment of personality pathology. DSM-IV clearly acknowledges that personality disorder symptoms may be manifested during periods of acute illness (e.g., major depression); however, it is equally clear that personality disorder symptomatology should be typical of a person’s long-term functioning and shall not be limited only to periods of acute illness (American Psychiatric Association, 1994, p. 629). Although some data do suggest that certain normative personality features, assessed via self-report instruments (not necessarily personality disorder symptoms), among clinically depressed patients do vary over time as a function of changing levels of depression (Hirschfeld et al., 1983), at present neither the relationship between personality disorder symptoms and state disturbance within the context of the cross-sectional diagnostic process nor the relationship between longitudinal symptom stability and state variability is resolved unambiguously for DSM personality disorders. A well-known study that employed structured interviews administered by experienced clinicians (Loranger et al., 1991) found that changes in clinical state (i.e., anxiety and depression) did not correspond significantly with changes in the number of DSM-III-R personality disorder criteria met at two points in time; this finding has subsequently been replicated by Loranger and Lenzenweger (1995; cf. Zimmerman, 1994). Trull and Goodwin (1993) reported that changes in mental state were not associated with either self-reported or interview-assessed personality pathology, although the levels of depression and anxiety characterizing the patients in his study are unusually low (perhaps not clinically significant in intensity). Current normal personality research also acknowledges the importance of determining the influence of state factors on trait assessment (Tellegen, 1985) and normative trait-oriented lifespan
research methodologists have long advocated the inclusion of state factors as important causal factors in longitudinal developmental models and research (Nesselroade, 1988). Therefore, a major focus of future research in personality pathology should be further clarification of the effect of anxiety and depression on both cross-sectional personality disorders symptom and personality trait assessment as well as the effect of such state factors on the longitudinal stability and change of personality disorders symptoms and traits. Any major theory of personality disorder must incorporate and address the role of state disturbances in the development and manifestation of personality pathology.

On a broadly related theme, the relatively robust association between personality pathology and affective disturbance also raises an important issue specifically concerning less severe affective pathology that is frequently accompanied by personality pathology (Loranger et al., 1991; Klein, Riso, & Anderson, 1993; Klein & Shih, 1998). For example, focusing on but one possible issue, we suggest that future research on personality-disordered populations as well as theories of personality disorder needs to address more directly the precise relationship between dysthymia and personality disorder. Klein et al. (1993, p. 234) in a careful examination of the dysthymia construct outlines four plausible, though competing, conceptualizations of dysthymia in relation to personality disorder:

1. Dysthymia is a “characterological depression,” essentially an attenuated form of major affective disorder, and this depression has an adverse impact on normative developmental processes, giving rise to the frequently co-occurring features of borderline, dependent, avoidant, and other personality disorder features;
2. Dysthymia is an “extreme” form of normally occurring depressive personality traits, a view deriving largely from psychodynamic theorists;
3. Dysthymia is the result or consequence of life stressors, notably those elicited by personality pathology; and
4. Dysthymia is a “character spectrum disorder” in which the low-grade dysphoria of the illness is a complication of a primary personality disorder traits.

Simply stated, any theory of personality disorder must not only take into account the role of dysphoric emotional states in the assessment and definition of personality pathology but also make explicit its assumptions about the relations hypothesized to exist between personality pathology, affect/emotion, and affective disorder (see Klein & Shih, 1998, for an excellent discussion).
Study Populations and the Epidemiology of Personality Disorders

An essential issue of concern in both future personality disorder research and theory is the representativeness of findings from studies and substantive conceptualizations based on hospitalized and/or clinic patient populations (vis-à-vis the general population at large) for furthering our understanding of the nature, course, and development of personality disorders (cf. Drake, Adler, & Vaillant, 1988; Kohlberg, LaCrosse, & Rickey, 1972). There can be little doubt that additional studies using inpatient and/or outpatient samples represent a necessity in future personality disorder research. However, we suggest it is critical to recognize that many individuals with personality disorders exist in the community at large and these people may never present themselves for psychiatric treatment (Dohrenwend & Dohrenwend, 1982) even though they may be quite impaired (Drake & Vaillant, 1985; Drake et al., 1988). This may be especially true for certain personality disorder diagnoses. For example, two studies that used clinically experienced raters found very low rates of schizoid and paranoid personality disorder in patient samples (Loranger, 1990; Pfohl, Coryell, Zimmerman, & Stengl, 1986), although population prevalence estimates for such pathology suggest that many more people are affected by these conditions than those who seek treatment (see Lenzenweger et al., 1997; Torgersen et al., 2001; Samuels et al., 2002). Furthermore, given the polythetic nature of DSM-IV, individuals with personality disorders who are hospitalized may be defined by substantively different configurations of symptoms than those who are not hospitalized. For example, hospitalized borderline patients with personality disorders might display more life-threatening and self-mutilating phenomenology than individuals who are also diagnosed borderline but who have not been hospitalized, although both would be validly diagnosed (the reader can surmise there are many “ways” to be diagnosed with borderline personality disorder according to DSM-IV). Moreover, as was established long ago in epidemiology, those individuals who present for hospital care for one condition are frequently afflicted with other conditions as well as driven by other factors to seek such care, and, consequently, generalizations based on the study of such patient populations must always be made cautiously (i.e., “Berkson’s bias”; Berkson, 1946). Therefore, we argue that a future studies of personality disorders that employ subjects drawn from nonclinical (i.e., community) sources will likely represent useful adjuncts to the more traditional study of hospitalized patients and, moreover, may lead to insights that reflect noteworthy differences between personality pathology that is observed in clinical versus nonclinical settings (see Korfine & Hooley, 2001).
Since 1996, significant strides forward have been made in our understanding of the epidemiology of the personality disorders through the use of well-characterized community samples. In 1993 Myrna Weissman hypothesized that the base rate of personality disorder in the community would be approximately 10–13% (Weissman, 1993). Since that time, three high-quality epidemiological studies of personality disorders in nonclinical community samples have been completed. Lenzenweger et al. (1997) applied two-stage procedure for case identification (Shrout & Newman, 1989) to a large nonclinical sample and estimated the point prevalence of DSM-III-R personality pathology to be approximately 11%. The results of this study were subsequently replicated by Torgersen et al. (2001), who found a prevalence of 13% in a Norwegian community sample, and Samuels et al. (2002), who found a prevalence of 9% in an urban community sample (Baltimore, MD). Also currently under way is the National Comorbidity Study—Replication (NCS-R), which is under the direction of R. Kessler. The NCS-R seeks to estimate the prevalence of specified personality disorders in the U.S. population using a rigorous sampling strategy and two-stage procedure for case identification (following Lenzenweger et al., 1997). The effective use of the two-stage procedure for case identification has helped to allay some of the concerns expressed earlier regarding the feasibility and cost of undertaking epidemiological work on the personality disorders (see Loranger, 1992). The appearance of empirically grounded prevalence rates for specific personality disorders will not only advance knowledge but also facilitate public health planning. For example, we now know that the community prevalence of borderline personality disorder is not 2% as suggested by DSM-IV, but is in the range of .3% (Lenzenweger et al., 1997) to .7% (Torgersen et al., 2001), and this information should prove useful to many parties.

Longitudinal Course/Lifespan Perspectives on Natural History of Personality Disorders

One of the cardinal assumptions, and perhaps most important from a theoretical perspective, concerning the nature of personality disorders is that they represent enduring conditions that are trait-like and, therefore, relatively stable over time (American Psychiatric Association, 1980, 1987, 1994). In fact, DSM-IV states, “The features of Personality Disorders usually become recognizable during adolescence or early adult life. . . . Some types of Personality Disorder . . . tend to become less evident or remit with age” (American Psychiatric Association, 1994, p. 632). However, with the possible exception of antisocial personality disorder (Glueck & Glueck, 1968; Robins, 1966, 1978), it remains safe to say, as we did in 1996, that very little is known about the long-term longitudinal course, development,
or natural history of personality disorders. Although there had been a vari-
ety of studies that have used the basic test–retest study design in the exami-
nation of personality disorder stability and change over time (see Perry,
1993, for review), it is essential to note that as Rogosa (Rogosa, Brandt, &
Zimowski, 1982), the lifespan research methodologist, remarks pointedly,
“Two waves of data are better than one, but maybe not much better”
(p. 744). Due to regression toward the mean effects (Nesselroade, Stigler,
& Baltes, 1980) and other difficulties (e.g., inability to estimate individual
growth curves; inadequacy for study of individual differences in change;
Rogosa, 1988) fundamentally inherent in simple test–retest design studies,
these test–retest studies did not address the fundamental issues concerning
stability and change in personality disorders.

In contrast to the situation with personality disorders, as of the mid-
1990s, there were abundant data in support of the general longitudinal
stability of normal personality traits and features in a variety of age
groups, including college students and young adults, (Block, 1971; Costa
Moane, 1987; McCrae & Costa, 1984; Mortimer, Finch, & Kunka, 1982;
Nesselroade & Baltes, 1974; Vaillant, 1977; Roberts & DelVecchio, 2000;
Srivastava, John, Gosling, & Potter, 2003). In this context, it should be
noted that although the impression for normal personality is one of stabil-
ity, the issue is far from closed and active discussion remains on this general
issue (see Caspi & Roberts, 1999; Roberts & DelVecchio, 2000; Srivastava
et al., 2003). Moreover, lifespan methodologists had already articulated the
preferable way to conduct such research, namely the use of multiwave
panel design studies (Baltes, Reese, & Nesselroade, 1977; Kessler &
Greenberg, 1981; Nesselroade et al., 1980) with multiple indicators for all
of the disorders (constructs) of interest. What this literature implied in
practical terms would be studies in which a large number of cases are
examined at least three, and preferably more, times for personality pathology
across meaningfully lengthy time intervals, with all cases being examined
using the same measures (procedures) at each assessment point. The
scientific utility of a multiwave design lies in the fact that it provides an
opportunity for the most informative statistical analysis of empirical rela-
tionships among constructs over time (Baltes & Nesselroade, 1973; Collins
& Horn, 1991; Collins & Sayer, 2001; Nesselroade & Baltes, 1979, 1984;
Rogosa, 1979, 1988; Singer & Willett, 2003), a fact that has been well
established in the lifespan developmental research realm for many years.

As suggested in 1996, we continue to urge the reader to bear in mind
that stability, however, is not necessarily as easy to investigate as one might
initially think because changes over time in personality pathology could be
the result of aging, period, treatment, and/or retest effects, although retest
effects appear less relevant to personality assessments (Costa & McCrae,
Longitudinal stability (and, by definition, change) of personality disorder features can be evaluated from at least four different perspectives (following Kagan, 1980; Mortimer et al., 1982; Lenzenweger, 1999; cf. Collins & Horn, 1991; Collins & Sayer, 2001), namely, structural invariance, rank-order stability, level stability, and ipsative (or intra-individual) stability. Structural invariance, or the maintenance of a temporally consistent factor structure and configuration of factor loadings, can be assessed using both confirmatory factor analysis (CFA) and causal modeling techniques (Bentler, 1984; Joreskog, 1979; Nesselroade & Baltes, 1984; Rogosa, 1979). These statistical techniques are ideally suited for use in longitudinal research as they allow the investigator to use all available panel data simultaneously (Kessler & Greenberg, 1981; Rogosa, 1979) and they allow for direct comparison of alternative structural models of stability (Bentler, 1984; Nesselroade & Baltes, 1984). Rank order, or “normative stability,” concerns the extent to which individuals maintain their relative position within a group ranking on a variable of interest from time 1 to time 2. Level stability concerns the extent to which group means remain invariant over time on a variable (or disorder) of interest. Finally ipsative stability concerns intra-individual consistency in the organization of personality disorder features or personality traits over time (cf. Mortimer et al., 1982). Finally, growth and change in a psychological attribute or behavior can be studied in a fine-grained manner using the powerful methods of individual growth curve analysis (Rogsoa & Willett, 1985; Singer & Willett, 2003), which allows one to illuminate important parameters of change such as level and slope within a statistical context that can be either descriptive or explanatory.

Given the body of evidence supporting the stability of normal personality, it is not unreasonable to expect that at least some personality disorder features would display significant long-term temporal stability, on the assumption they are reflective of normal personality variation in some manner—particularly, for example, features such as schizoid social withdrawal, compulsive rigidity, and the “extraverted” or outwardly directed interpersonal style of the psychopath. Although several early studies supported the temporal stability of personality disorder features and diagnoses over relatively short time spans (e.g., 1 year or less) (Perry, 1993), evidence concerning long-term or lifespan stability of operationally defined personality disorders was conspicuously lacking in the published empirical research literature (Drake & Vaillant, 1988; Drake et al., 1988) through the 1980s and well into the 1990s. By the late 1980s, it was recognized that long-term longitudinal work was sorely needed in the area of personality disorders, but no studies were under way.

This situation changed in 1990 due to the initiation of a large-scale prospective multiwave longitudinal study of personality disorders. The
Longitudinal Study of Personality Disorders (LSPD), under the direction of M. F. Lenzenweger, was begun in 1990 as the first NIMH-funded longitudinal study of personality disorders of any type. The LSPD concerns the stability and change of personality disorders, personality, temperament and many other aspects of psychological functioning over time. An initial report from the LSPD (Lenzenweger 1999), using three waves of data, described impressive evidence of rank-order stability in personality features over time as well as some nontrivial evidence of change in the level of personality disorder features over time. Thus, stability emerged as a complex issue for the personality disorders in these data—individuals retained their position in an ordinal sense; however, there was clear evidence that change was happening. Further analysis of the LSPD data set using state of the art individual growth curve methodology (Lenzenweger, Johnson, & Willett, in press) suggests a considerable amount of change occurring in personality disorder features over time, change that was not clearly detectable using more traditional analytic techniques. Such findings raise profound and fundamental questions regarding the very basic nature of personality disorders as defined in the DSM systems.

A second longitudinal study of personality disorders was begun in 1996, also with the support of the NIMH, known as the Collaborative Longitudinal Personality Disorders Study (CLPS) under the direction of a team of investigators at several sites (Gunderson et al., 2000; Shea et al., 2002). The CLPS focuses on four personality disorders: schizotypal, borderline, avoidant, and obsessive–compulsive personality disorders. Early reports from the CLPS suggest some degree of rank-order stability over time, with evidence of relatively substantial symptom declines with time (Shea et al., 2002). Specifically, Shea et al. (2002) found that 66% of their CLPS patients dropped below diagnostic thresholds in 1 year with highly significant declines (revealing substantial effects) for continuously measured personality disorder symptoms. These CLPS findings replicate those reported earlier from the LSPD (Lenzenweger, 1999); however, the CLPS data have not been analyzed yet with more advanced techniques. Unfortunately, complicating factors in the CLPS methodology such as the absence of blinded assessors in their personality disorder assessment protocol and the fact that all study subjects have been in treatment during the study cannot be ignored. These factors make the extraction of meaning and direction from the CLPS study less than straightforward. Finally, two other studies have taken a focus on DSM personality pathology, the Johnson et al. (2000) study that focuses on a retrospective assessment of personality disorder phenomenology from case records, which include various clinical data, for a cohort of adolescents, and the study by Zanarini, Frankenburg, Hennen, and Silk (2003) of borderline personality disorder. What is remarkable about both of these latter studies is that, despite methodo-
logical limitations, they suggest evidence of considerable change in the level of personality disorder features over time, consistent with LSPD (Lenzenweger, 1999; Lenzenweger et al., in press). For example, Zanarini et al. (2003) found massive declines in symptoms such that 73.5% of previously diagnosed borderline personality disorder subjects were “remitted” at 6-year follow-up, suggesting nontrivial mean level changes in borderline personality disorder symptoms. Johnson et al. (2000) found 28–48% reductions in personality disorder symptoms (continuous format) with time.

What is particularly fascinating about the emerging corpus of data from the longitudinal study of personality disorders is that the picture appears to be one of change. This is especially interesting as DSM-IV maintains its view that the personality disorders are stable and trait-like over time and it is not uncommon to see reviewer comments on manuscripts, NIMH study section “pink sheet” comments, or psychology textbooks reflecting the assumption that personality disorders are stable, enduring, and “set like plaster.” In short, the assumption regarding the stability of personality disorders may be just that, an assumption that will erode in time with the accrual of empirical data that actually specify the true developmental and longitudinal nature of personality disorders over time. We continue to advocate the need for additional longitudinal studies of personality disorder, from both community and clinical settings. However, in advocating the careful study of the longitudinal stability of personality disorders, we do not intend to suggest that inquiry into stability represents an end in itself, one that is but merely descriptive and statistical. Rather such study it should be viewed as a necessary step in the ongoing exploration of the lifespan developmental course of personality pathology. Once established, we foresee studies moving away from simple demonstrations of stability (or lack thereof) but toward a lifespan view with an emphasis on discerning those biobehavioral and psychosocial processes and mechanisms that underlie the etiology and development of personality pathology (i.e., moving from description toward an explanatory framework) (see Lenzenweger et al., in press).

Genetic and Biological Underpinnings of Personality Pathology

The role of genetic influences in the development and stability of normal personality as well as individual differences in personality is now well established and beyond dispute (Plomin & Caspi, 1999; Plomin, DeFries, McClearn, & McGuffin, 2000; Plomin, DeFries, Craig, & McGuggin, 2003; McGue, Bacon, & Lykken, 1993), contrarian views being most likely expressed by those with sociopolitical agendas rather than rigorous scien-
tific interests. Though the heritability estimates for features or dimensions of personality tend to be lower than those observed for intelligence or other cognitive abilities, it can be safely said that genetic factors play an influential role in determining personality—they have the status of “fact” at this point (see DiLalla, 2004; Plomin & Caspi, 1999; Plomin et al., 2000; Plomin et al., 2003). The situation for personality disorders, however, remains considerably less clear with respect to the role of genetic factors in the etiology of personality pathology. This is not to say that genetic factors do not play a role in determining these disorders but, rather, that the studies bearing on the determination of both familiality and heritability of personality disorders are only just beginning to appear; twin and adoption studies remain a rarity and familial aggregation work is accumulating slowly (see Livesley et al., 1998). To date, the greatest amount of genetically relevant data can be found for schizotypal personality disorder, which by most accounts appears related genetically to schizophrenia as well as to borderline, antisocial, and obsessive–compulsive personality disorders. However, the genetic picture for even these disorders is unclear due in large part to an absence of data for the disorders themselves or putatively correlated dimensions (e.g., sociability in schizoid personality disorder) (Lang & Vernon, 2001; Nigg & Goldsmith, 1994). Finally, quite apart from research on genetic factors, it is necessary to point out that the psychobiological underpinnings of personality pathology in terms of prominent central nervous system neurotransmitters and meaningful neurobehavioral circuitry remain in infancy (see Cloninger et al., 1993; Coccaro, 1993; Coccaro et al., 1989; Coccaro, 2001; Depue & Lenzenweger, 2001, Chapter 8, this volume; Siever, Kalus, & Keefe, 1993).

The Axis I and Axis II Interface: Comorbidity, Causality, or Confusion?

As we noted in 1996, the nature of observed comorbidity among the personality disorders was both ubiquitous and poorly understood, a view echoed by others (Pfohl, 1999), and it remains an area of active discussion. In fact, an online search using PsycINFO that tracked the joint appearance of the keywords “comorbidity” and “personality disorder” yielded nearly 800 citations, many reports appearing in the 1990s.

All models of personality disorder are, by necessity, required to deal with the relationship between personality pathology and other major forms of psychopathology, such as affective illness, anxiety disorders, and even schizophrenia. Both clinical practice and available research data suggest strongly that an individual can suffer from both a major Axis I condition as well as a personality disorder simultaneously—a clinical reality typically discussed under the rubric of “comorbidity” (Widiger & Shea, 1991).
comorbidity issue is laden with a number of complex questions that speak not only to description, diagnosis, and classification but also to etiology. For example, at the level of diagnosis, is it the case that comorbidity arises out of the fact that our current multiaxial system encourages multiple diagnoses (is it an artifact of the system) or is it the case that people can actually suffer from two or more disorders simultaneously? Clearly, one could have both pneumonia and heart disease simultaneously; can one have both depression and schizotypal personality disorder at the same time? Can one have a personality disorder even in the face of a psychotic illness—if so, what limitations or qualifications must attend the diagnosis of a personality disorder in such circumstances?

Future research needs to focus on the careful dissection of putatively highly comorbid conditions such as major depression and borderline personality disorder (e.g., Loranger, 1991b) along a variety of meaningful dimensions such as phenomenology, familiality, medication response, psychobiology, and pathogenesis (see Gunderson & Phillips, 1991, for an excellent demonstration). Such careful dissection of comorbid conditions will likely enhance our understanding not only of the boundaries existing between personality pathology and other major syndromes but also of our notions regarding the development and etiology of personality disorders. What possible roles could a major Axis I disorder play in relation to personality pathology? For example, could the presence of a major psychiatric syndrome be shown to “causally” facilitate the development of a personality disorder or merely increase the statistical risk for the development of a personality disorder? Can an Axis I syndrome represent the more severe version of a broad class of psychopathology of which the related personality disorder is but a spectrum variant (cf. schizophrenia and schizotypal personality disorder)? Could it also not be that there is no etiologically relevant connection whatsoever between a major syndrome and a comorbid personality disorder?

For research into the comorbidity issue(s) to be maximally beneficial to the field, two fundamental methodological issues should be kept in mind. First, comorbidity work is badly in need of large \( n \) studies. If the natural association between conditions (e.g., borderline personality disorder and depression) is to emerge from data, then the most stable estimate of this association will come from data drawn from large samples. Second, future comorbidity research should be done on either a consecutive admissions basis at a clinical setting or in the general population from an epidemiological perspective (cf. National Comorbidity Survey [NCS]; Kessler et al., 1994). The NCS-R, under the direction of Ronald Kessler, which includes a personality disorders component as noted earlier, should provide useful information on the personality disorders and Axis I disorder comorbidity patterns. Future reports on the comorbid diagnoses of those patients who happen to be in one’s personality disorder protocol are not likely to be
informative as any inherent sampling bias will misrepresent the natural rate of comorbidity. An excellent example of the application of multivariate statistical technique in the dissection of a large data, the original NCS data, in an effort to address comorbidity can be found in Krueger (1999). Krueger’s (1999; Krueger & Tackett, 2003) speculations regarding potentially core common processes in general psychopathology should serve to inspire comparable work on the personality disorders at the item-level of analysis in an effort to resolve comorbidity issues (see above).

Validity of Personality Pathology

Last, but by no means least, is the issue of validity in relation to personality disorder constructs. Despite the 25 years since the publication of DSM-III, the validity of specific DSM personality disorder diagnoses remains a relatively open issue in psychopathology research; however, the base of validity information is growing for most disorders, particularly schizotypal, borderline, and antisocial personality disorders. We take this opportunity to remind our reader that although reliable ratings of personality disorder symptoms are now possible, this does not necessarily ensure that the validity of the diagnoses has been established (Carey & Gottesman, 1978). This statement holds true for the DSM taxonomy as well as all those models described in this volume. Furthermore, no clear and compelling criteria of validity (Cronbach & Meehl, 1955) currently exist against which personality disorder diagnoses can be compared to assess their validity—not unlike other areas of psychopathology, there is no “gold standard” for validity in the personality disorder realm. Although Spitzer (1983) has proposed that the validity of personality disorder diagnoses might ultimately best be established by longitudinal studies of personality disorders that employ well-known expert raters as well as all available data useful for psychiatric diagnosis (the so-called LEAD standard), such a definitive study conducted on a large scale has yet to be undertaken due to the logistical difficulties and formidable expense most likely involved in such a project (cf. Pilkonis, Heape, Ruddy, & Serrao, 1991; see also Zimmerman, 1994). However, we do not wish to suggest that advances have not been made in aggregating data that can ultimately ensure the validity of personality disorder diagnoses. Many have sought to use informants to help validate clinician assigned diagnoses from the reports taken from individuals; however, the use of informant reports to validate the reports of individuals assessed for personality disorders remains a highly problematic and unclear area open to alternative interpretation (Klonsky, Olman, & Turkheimer, 2003). There have been advances in other approaches to assessment that have implications for construct validity. Creative new approaches to the assessment of personality disorders, motivated in part by an interest in increasing validity,
can be found, for example, in the novel prototype matching/Q-sort approach developed by Westen and colleagues (Westen & Shedler, 1999, 2000; cf. Westen & Muderrisoglu, 2003) and in the work of Turkheimer, Oltmanns, and colleagues on peer nomination (Oltmanns, Turkheimer, & Straus, 1998; Oltmanns, Melley, & Turkheimer, 2002). Finally, we should like to suggest that the methods of the experimental psychopathology laboratory (Bornstein, 2003; Lenzenweger & Hooley, 2003) hold considerable promise for the elucidation of fundamental processes that may be impaired in personality pathology and illumination of such processes may move personality disorder research away from nearly complete reliance on clinical features (see Korfine & Hooley, 2000, for an excellent example). Not only would such laboratory work speak to construct validity, but it might also discern reliable and valid endophenotypes for personality disorders (Gottesman & Gould, 2003).

**LANDMARKS, CRITICAL JUNCTURES, AND FRONTIERS: A GUIDE TO EXPLORING THE MAJOR THEORIES OF PERSONALITY DISORDER**

In closing this introductory chapter, as we did in 1996, we look back and see that we have raised a number of specific issues that we believe can be counted among the most challenging and important in the area of personality disorders research. For this area of psychopathology research to move forward, greater clarity must be sought along each of the dimensions noted previously. Our contributors, one and all, speak to various aspects of the foregoing issues we have raised and they can be fit into broader theoretical and scientific contexts as well. At the same time we encourage, our readers, to find how the theoreticians and researchers contributing to this volume deal with the specific substantive issues noted herein, we should also like to encourage each of them to examine the following chapters using a common set of broader guidelines. By evaluating each of the following chapters along the general dimensions specified next, we believe consistencies and inconsistencies across the models as well as possibilities for new theory, research, and treatment will emerge. Readers should consider the following dimensions with respect to each of the following theoretical models of personality disorder.

**What Are the Substantive Foundations of the Model?**

Can the roots of the model be traced to major historical or research traditions in psychology and/or psychiatry (e.g., psychoanalysis and behaviorism)? Does the author identify the level from which the data are derived that constitute the basis for classification, measurement, and treatment
What Is the Formal Structure of the Model?

Have the core assumptions of the model been formally stated and are the major explanatory principles clearly articulated? While models in psychopathology, unlike comprehensive theories, can be somewhat incomplete in their effort to explain a form of psychopathology (cf. Matthysse, 1993; see also Webb, 2001), has the model nonetheless been formulated in a manner that allows for its testability and falsifiability (and, therefore, possible refutation) (cf. Meehl, 1978, 1993)?

What Taxonomy Derives from the Model?

What is the nature of the taxonomy that derives from the model? For example, does the model admit of a structure that is hierarchical, based on a circumplex or some alternative form of a multiaxial approach? Is the classification approach based on a prototypal, categorical, or dimensional methodology? Is variation personality pathology discussed in terms of degree (quantitative) or kind (qualitative)? How independent are the personality disorder syndromes in terms of etiological origins and does this independence affect the taxonomy in any fashion? How does the model relate to DSM-III-R and the more recent DSM-IV?

Etiological and Developmental Considerations?

Does the model transcend a purely descriptive stance and speak to issues of “mechanisms” and “processes” that determine the development of a personality disorder? In short, does the model attempt to answer the question “how” with respect to the emergence of personality pathology? What are the principal components of the processes and mechanisms theorized to be etiologically relevant (e.g., genetic influences, neurobehavioral factors, and temperamental dispositions; cognitive deficits; learned characteristics; and other sources of disorder such as familial conflict and trauma or abuse)?
In the theories presented in this volume one trend is quite clear—all our contributors have clearly eschewed a merely descriptive approach in favor of an explanatory effort with clear up implications for etiology. Furthermore, all our contributors have proposed theoretical models that presume interaction across multiple levels of the individual, emphasizing not only behavioral and personality characteristics and factors but also neurobiological and environmental components as well. For example, although Otto Kernberg and Eve Caligor (Chapter 3) see character pathology largely in terms of a developmental pathology of aggression, their theory incorporates temperament, affect, and trauma components in interaction. Depue and Lenzenweger (Chapter 8) present a fundamental model of personality as defined by interacting dimensions known to be rooted in neurobiological functions and, according to neurobehavioral model personality pathology, which can also be viewed as the interactive result of these dimensions. For Theodore Millon and Seth Grossman (Chapter 7), personality pathology emerges from a complex interaction of three fundamental polarities, self versus other, pleasure versus pain, and activity versus passivity. James Pretzer and Aaron Beck (Chapter 2) see personality pathology largely emerging from and being maintained by systematic biases in information processing and memory of events eliciting pathological cognitive, emotional, and behavioral responses. Meyer and Pilkonis (Chapter 5), Pincus (Chapter 6), and Benjamin (Chapter 4) all stress the role of interpersonal experiences in the emergence of personality disorders.

How Are Assessment and Diagnosis Accounted for in the Model?

Does the model have an associated assessment and diagnostic approach? If so, what are the sources of the empirical data that are used for diagnosis according to the model? Does the assessment approach rely on therapeutic contexts, self-report inventories, or structured interviews? Has the author presented adequate information concerning the reliability and validity of the assessment and diagnostic procedures associated with the model?

Millon and Grossman (Chapter 7), Pincus (Chapter 6), Benjamin (Chapter 4), Meyer and Pilkonis (Chapter 5), and, to a lesser extent, Pretzer and Beck (Chapter 2) have exerted a great deal of effort in operationalizing their personality pathology conducts in the form of assessment instruments covering the taxonomy of the personality disorders. Millon and Grossman are guided by the DSM taxonomy to a great extent, whereas Benjamin, Pincus, and Meyer and Pilkonis have shown the consistency of their approaches with the DSM system but do not attempt to map their positions with the DSM landmarks.
Does the Model Articulate Therapeutic Procedures or, at Least, Highlight Implications for Treatment?

According to the model, how does one go about treating personality pathology and what are the treatment goals (e.g., symptom relief vs. reconstructive work)? In what tradition is the therapeutic work carried out (e.g., insight oriented vs. cognitive vs. biological therapy)? Are the principles of change/improvement clearly articulated by the model? What are the limits of the therapeutic approach (i.e., Are there personality disorders for which the therapy would not be appropriate)?

While Benjamin (Chapter 4), Kernberg and Caligor (Chapter 3), and Pretzer and Beck (Chapter 2) all relate their personality pathology constructs and theories to interventions, it is Kernberg and Beck who have articulated treatment manuals for these disorders. This translation of theories of personality disorder to treatment interventions is a necessary step to the current important focus of psychotherapy research on the mechanisms of change (National Institute of Mental Health, 2002). From this prospective, Kernberg and colleagues emphasize the importance of change in identity diffusion (conception of self and others), and Beck points out the importance of faulty cognitions, especially those relating to the self. When one overlooks the somewhat esoteric jargon of each of these orientations, there is an interesting similarity in the focus on the patients’ guiding conceptions of self and interactions with others.

Prospects for the Future: Integration of Mind, Brain, and Behavior

To our minds, the task of future theorizing and empirical research in personality disorders is the effective integration of mind, brain, and behavior. Any comprehensive model of complex human behavior, particularly forms of psychopathology, will require a clear and genuine integration of ideas and research findings that cut across the levels of analysis linking mind, brain, and behavior. One thing is quite clear to us, as well as to the contributors of this volume, monolithic theories existing at but one level of analysis are sure to fail in their explanation of complex human behavior. For example, for years normative developmental psychologists have viewed personality and emotional development almost exclusively in terms of psychosocial influences, much to the exclusion of genetic and biological factors. Indeed, David Rowe (1994), the late developmental behavioral geneticist, has termed this view of personality and psychological development “socialization science,” and he has offered a pungent criticism of such a monolithic model, demonstrating effectively the relative importance of genetic factors vis-a-vis psychosocial influences for personality de-
velopment. We maintain a similar position with respect to personality disorders—for example, personality disorders are not likely to be understood or explained solely in terms of psychosocial influences. A genuine integration of genetic factors, neurotransmitter models, and other neurobiological processes with psychosocial, cognitive, and environmental factors will be required to advance our knowledge of the personality disorders. The best models in some ultimate sense will be those that integrate across these levels (e.g., Meehl, 1990; see also Meehl, 1972). The importance of genetic factors in both normative and pathological development is indisputable (DiLalla, 2004; Plomin et al., 2000; Plomin et al., 2003; Rowe, 1994; Rutter, 1991; Rutter & Silberg, 2002) and the essential role of neurobiological factors in temperament (e.g., Kagan, 1994), emotion (Ekman & Davidson, 1994), personality development (e.g., Depue & Lenzenweger, 2001, Chapter 8, this volume), and the emergence of psychopathology (e.g., Breslin & Weinberger, 1990; Cocarro & Murphy, 1990; Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Grace, 1991) is axiomatic, some would even say confirmed. The meaningful integration of brain, emotion, behavior, and environmental influences currently represents an exceptionally active research area in various areas of psychological science, especially cognition and personality—our belief is that personality disorders research will necessarily have to strive for similar integrative work for genuine advances to occur. Our contributors are clearly leading the way in this connection. For example, Depue and Lenzenweger (Chapter 8) seek to integrate personality, behavior, and neurobiology in their model, Kernberg and Caligor (Chapter 3) propose complex interactions among temperament, trauma, and early experience, and Pretzer and Beck (Chapter 2) suggest that biased cognition must be understood within a matrix that incorporates affect and emotion as well as interpersonal factors. Indeed, interesting differences have emerged among our theorists, for example, Kernberg and Caligor (Chapter 3) argue that neurobiological factors, operating through temperament, have more of a mediating role in the determination of personality pathology, whereas Depue and Lenzenweger (Chapter 8) cast neurobiological processes, especially the role of serotonin, in modulating framework. This is precisely the type of debate and discussion that will not only provide useful heuristics for future research directed at integrating mind, brain, and behavior but will ultimately allow us to better understand and care for our patients.

At this point we should like to end our discussion of points of introduction and orientation and invite readers to sample from what we believe are the leading theories of personality disorder. We encourage readers to view each of these chapters as an independent position statement by their authors as well as the building blocks for what may ideally become a more comprehensive theory of personality pathology.
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NOTE

1. The MAXCOV data reported in Trull et al. (1990) for borderline personality disorder reveal the characteristic right-end peak suggestive of a low base-rate latent taxon (see Meehl, 1992, 1995, or Korfine & Lenzenweger, 1995) for conceptual and mathematical rationale. The data reported in the Rothschild, Cleland, Haslam, and Zimmerman (2003) report are somewhat ambiguous; however, they reveal considerable evidence of taxonicity although the authors have chosen to view the data as supporting a dimensional model (see Haslam, 2003, for a taxonic interpretation of these same data).

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