

## CHAPTER 1

# Conceptual Issues

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It is difficult to characterize the current state of the study of personality disorder (PD). The field is obviously vigorous and productive. Extensive empirical data are being collected about an increasingly wide range of topics. In important areas, conclusions based on empirical findings are replacing traditional ideas that were more speculative in nature. However, the field is hampered by the lack of a coherent conceptual framework to guide research and systematize findings, resulting in a mass of information that often seems to lack coherence. This makes it difficult to evaluate the extent to which progress is being made because science is organized knowledge (Medawar, 1984): It involves facts and findings that have internal coherence because they are held together by general principles and laws. Current theories of PD do not offer a solution to this problem: Most are conceptual positions rather than actual theories and are insufficiently developed to bring coherence to the field (Lenzenweger & Clarkin, 2005).

This situation reflects the early state of the field's development. All sciences begin this way, amassing vast amounts of relatively unrelated observations. This is how biology started as natural history. Viewing the situation from the perspective of Kuhn's (1962) description of the nature of scientific change, the current situation may be viewed as either characteristic of the preparadigmatic phase in the development of a science or as a period that Kuhn referred to as "extraordinary science." In the preparadigmatic phase, data collection dominates, but

there is uncertainty about the value and significance of these data. As a result, scholars practice science, but the results of their efforts do not constitute a science. Kuhn also noted that the phase is marked by multiple schools of thought and intense debates about legitimate methods, problems, and standards of evidence that serve more to define the different schools than to produce agreement. In some ways, this seems an apt commentary on contemporary study of PD. Extensive data are being collected. Multiple schools and perspectives exist, such as cognitive therapy, psychoanalysis, trait psychology, neurobiology, interpersonal theory, behavioral theory and therapy, traditional phenomenology, and so on, each with its own focus of interest, methodology, and mode of explanation. Since communication between schools is limited, knowledge tends to get stovepiped. From time to time, there is talk of integration, but it never occurs.

However, it may also be argued that the study of PD does have a paradigm and has for much of its recent history: the paradigm of the medical model than underpins contemporary psychiatry. The model has structured the field and informs most aspects of practice and research. However, recently, concerns have been raised about the model and its relevance to mental disorders, raising additional concerns about the conceptual foundations of the study of PDs.

Although the medical model is usually assumed to be a unitary framework, there are several versions (Bolton, 2008). The version

implicitly adopted by psychiatry is a somewhat simplified form of the traditional disease-as-entity model of modern medicine (Sabbarton-Leary, Bortolitti, & Broome, 2015). With this model, symptoms are organized into discrete syndromes that are explained by an underlying impairment that is generally assumed to be biological. The model's appeal to psychiatry is understandable given its success in general medicine, and its assumed relevance was undoubtedly bolstered by its success at the beginning of the 20th century with the discovery that general paresis, a relatively common form of psychosis at the time, was a form of tertiary syphilis due to the spirochete *Treponema pallidum*. This created the expectation that major causes of other mental disorders would also be identified (Pearce, 2012). Despite the fact that a century later this early success has not been repeated, the idea that "big causes" will be identified for mental disorders lingers on, with infectious agents being replaced with causes such as genes, with major effects and specific impairments in neural mechanisms.

This version of the medical model was adopted by the neo-Kraepelinian movement (Klerman, 1978), which sought to reaffirm the medical foundations of psychiatry. Since the neo-Kraepelinian perspective formed the conceptual foundation for DSM-III and subsequent editions, this version of the model underpins much of the contemporary study of PD. Recently, however, several authors have noted that the disease-as-entity version of the model is not applicable to many disorders in general medicine, let alone mental disorders (Bolton, 2008; Kendler, 2012b). The model does not work for disorders with a complex, multifaceted etiology. Since most mental disorders, and certainly most PDs, have this feature, the models' relevance to the study of PD requires reconsideration.

Kuhn referred to periods in the evolution of a science when an established paradigm is no longer viable as periods of extraordinary science. Current problems with the medical model and problems arising from the neo-Kraepelinian paradigm, most notably the failure to identify discrete diagnostic categories and the extensive patterns of diagnostic co-occurrence among all forms of mental disorder, may be considered to create within psychiatry, and hence within PD, a situation resembling Kuhn's ideas of extraordinary science (Aragona, 2009). In such periods, progress is fragmented, there is widespread disagreement about appropriate methods

and procedures, extreme and speculative concepts emerge, and there is usually an increased interest in the philosophical assumptions of the field. The latter point is interesting given the recent spate of texts and articles on the philosophy of psychiatry.

Whether the current situation represents the preparadigmatic or extraordinary science periods in the emergence of a science of PD is a matter for philosophers of psychiatry to explore. However, both perspectives have similar consequences: Either way, the field needs an agreed paradigm and conceptual framework to guide the acquisition and interpretation of empirical findings. However, such developments need not involve a sudden change. The Kuhnian model of scientific progress is one of revolutionary change, with the creation of a new paradigm that leads off a period that he called normal science, in which progress is incremental until another paradigm crisis. Other views of scientific progress consider change to occur for a variety of reasons and to involve a more gradual process. This seems more appropriate to PD. This chapter explores these issues. In the first section, I begin by briefly tracing the history of the field prior to the publication of DSM-III in 1980 because current conceptions of PD have tangled roots that continue to exert an influence. The second section deals with what is referred to as the "DSM era," dating from the publication of DSM-III to the publication of DSM-5. DSM-III was a landmark event that helped establish systematic empirical research on PD and the assumptions underlying DSM-III continue to shape and dominate the contemporary study of PD. Although authors of successive revisions of DSM often emphasize the distinctiveness of their revision, continuity across editions is extensive compared to the differences between them (Aragona, 2015). The section focuses particularly on the impact and relevance of the medical model and the problem of diagnostic validity. The third section examines principles that may contribute to a new conceptual framework for a science of PDs, including an alternative version of the medical model. In the final section I briefly consider how these principles might contribute to a more coherent nosology.

### Early Conceptions of PD

Although interest in personality patterns that are similar to modern PD diagnoses date to

antiquity, Berrios (1993) argued that the contemporary concept of PD only truly emerged with the work of Schneider (1923/1950). Nevertheless, several developments during the 19th century helped to structure current ideas. The term “character” was widely used during that time to describe the stable and unchangeable features of a person’s behavior. Writings on the topic also used the concept of “type,” and Berrios noted that “character” became the preferred term to refer to psychological types. Although the term “type” was used in the contemporary sense to describe discrete patterns of behavior, the term “personality” was used largely to refer to the mode of appearance of the person (Berrios, 1993), a usage derived from the Greek term for “mask.” Gradually, the term took on a more psychological meaning when used to refer to the subjective aspects of the self. Hence, 19th-century writings about the disorders of personality referred to mechanisms of self-awareness and disorders of consciousness, and not to the behavior patterns that we now recognize as PD. It was only in the early 20th century that the term “personality” began to be used in its present sense. However, it is interesting to note the recent resurgence of interest in self-awareness as a core impairment of PD.

The evolution of the concept of PD during the 19th century was influenced by studies of moral insanity by Pritchard (1835) and others. Although “moral insanity” is often considered the predecessor of psychopathy, Pritchard’s description shows little resemblance to Cleckley’s (1941/1976) concept of psychopathy or DSM antisocial personality disorder (ASPD; Whitlock, 1967, 1982). Rather, Pritchard used the term to describe forms of insanity that did not include delusions. The predominant understanding of the time was that delusions were an inherent component of insanity, an idea developed by John Locke. The term “moral insanity” described diverse conditions, including mood disorders that had in common the absence of delusions. Berrios (1993) suggested that Pritchard encouraged the development of a descriptive psychopathology of mood disorders that promoted the differentiation of these disorders from related conditions and the differentiation of personality from other disorders by distinguishing more transient symptomatic states from more enduring characteristics. This important development promoted the emergence of PDs as a separate diagnostic group. Interest in moral insanity continued throughout the 19th

century. Maudsley (1874) extended Pritchard’s concept with the observation that some individuals seemed to lack a moral sense, thereby differentiating what was to become the concept of psychopathy in the more modern sense. Toward the end of the 19th century, German psychiatrist Julius Koch proposed the term “psychopathic” as an alternative to moral insanity. At about the same time, the concept of degeneration, taken from French psychiatry, was introduced to explain this behavior.

The significance of these developments was that the idea of psychopathy as distinct from other mental disorders gained acceptance, which set the stage for Schneider’s concept of psychopathic personalities as a distinct nosological group. Before this occurred, however, Kraepelin (1907) introduced a different perspective by suggesting that personality disturbances were attenuated forms (*formes frustes*) of the major psychoses. Kraepelin’s seminal contributions to nosology with the distinction between dementia praecox and manic-depressive illness are generally considered to firmly establish the medical model as the basis for conceptualizing and classifying mental disorders. Subsequently, Kretschmer (1925) took the idea of PDs as attenuated forms of mental state disorders further by positing a continuum from schizothyme through schizoid to schizophrenia—an idea that anticipated current thinking about schizophrenia spectrum disorders. The notion that PDs such as borderline personality disorder (BPD) are on a continuum with some major mental state disorders rather than distinct nosological entities, and hence that PDs are not a distinct nosological grouping, continues to be raised intermittently despite extensive conceptual and empirical evidence to the contrary.

Nonetheless, the overriding assumption of psychiatric classification for much of the last century has been that mental state disorders and PDs are distinct, although the nature of this distinction has differed across conceptual frameworks. Jaspers (1923/1963) offered a cogent theoretical rationale for the distinction by differentiating personality developments from disease processes. The idea had little impact on American psychiatry, although it is probably worth revisiting. Personality developments are assumed to result in changes that are understandable in terms of the individual’s previous personality, whereas the changes associated with disease processes are not predictable from the individual’s premorbid status. Jaspers sug-

gested that these different forms of psychopathology require different methods of classification, with conditions arising from disease processes being conceptualized as either present or absent and hence classified as discrete categories, whereas PDs (and neuroses) should be classified as ideal types. This issue is still unresolved and contributed to much of the confusion associated with the DSM-5 classification of PD.

Schneider's volume *Psychopathic Personalities* published in 1923 was a landmark event that largely established the contemporary approach to PDs. Berrios (1993) suggested that by adopting the term "personality," Schneider made concepts such as temperament and character redundant. There is much to be said for this position, although, unfortunately, this clarity has not been widely accepted (for further discussion, see Chanen, Tackett, & Thompson, Chapter 12, this volume). Schneider also made the important conceptual distinction between abnormal and disordered personality, an issue of current significance given the demonstrated continuity between PDs and normal personality. Schneider defined abnormal personality as "deviating from the average." Thus, abnormal personality merely represents the extremes of normal personality variation. However, Schneider also recognized that this was not an adequate definition of pathology because extreme variation does not necessarily imply dysfunction or disability. He referred to the subgroup of abnormal personalities that are dysfunctional in a clinical sense as psychopathic personalities, which were defined as "abnormal personalities who either suffer personally because of their abnormality or make a community suffer because of it" (p. 3). Schneider did not discuss abnormal personality in detail but concentrated instead on describing 10 varieties of psychopathic personality: hyperthymic, depressive, insecure (sensitive and anankasts), fanatical, attention-seeking, labile, explosive, affectionless, weak-willed, and asthenic. Here the term "psychopathic personality" was used to cover all forms of PD and neurosis. In the preface to the ninth edition, written in 1950, Schneider noted that the term "psychopath" was not well understood and that his work was not the study of asocial or delinquent personality. He added that "some psychopathic personalities may act in an antisocial manner but . . . this is secondary to the psychopathy" (p. x). Thus, he avoided the tautology inherent in conceptions of ASPD that

are defined in term of social deviance, whereupon the diagnosis is then used to explain deviant behavior.

Although psychopathic personalities were portrayed as types, it is important to note that Jaspers's (1963) and Schneider's (1923/1950) concept of ideal type was not that of a simple diagnostic category, as is the case with DSM-III to DSM-5. Ideal types are patterns of being rather than diagnoses. According to Jaspers, an ideal typology consists of polar opposites such as dependency and independence or introversion and extraversion. Diagnosis does not involve ascribing a typical diagnosis. Instead, individuals are compared to contrasting poles of the type to illuminate clinically important aspects of their behavior and personality. Thus, the typology is essentially a framework for conducting clinical assessment and formulating individual cases. Moreover, ideal types are not stable in the sense that DSM diagnoses were originally assumed to be stable. Instead, some are episodic and reactive. Thus, Schneider's (1923/1950) system represents a more complex understanding of types and the relationship between normal and disordered personality than that of DSM-III to DSM-5. Although he used the term "type," his conceptualization implicitly acknowledges continuity with normal personality. In addition, Schneider's "types" are not discrete categories; rather, they refer to individuals at the extremes of a continuum, much as Eysenck used the term later to refer to those as the poles of the continuum introversion–extraversion. In this sense, Schneider anticipated current ideas derived from trait models that PDs represent extremes of normal variation, although he added criteria to differentiate pathological from nonpathological variation. Schneider also disagreed with Kraepelin's idea that PDs are systematically related to the major psychoses, although he assumed that personality affected the form that a psychosis takes. Schneider's position is not without problems, particularly in regard to the definition of suffering. Nevertheless, he introduced into the classification of PD a conceptual clarity that has rarely been matched.

Within British and American psychiatry, the concepts of psychopathy and psychopathic personality were defined more narrowly to describe what we now call ASPD, although the two are not synonymous. Descriptions of psychopathy and, later, descriptions of PDs, were largely based on clinical observation. Theoretical factors that influenced Jaspers (1963) and

Schneider (1923/1950) played little part in nosological development, and various definitions emerged as individual clinicians emphasized different facets of these disorders and different aspects of the overall class.

Parallel to these developments, psychoanalytic concepts also contributed to classification and enriched ideas about personality pathology, but in the process they increased diagnostic and descriptive confusion. Although Freud was not primarily interested in PD, his theory of psychosexual development led to descriptions of character types associated with each stage (Abraham, 1921/1927) that became the basis for dependent, obsessive–compulsive, and hysterical (changed to histrionic in DSM-III) PDs. This development shifted assumptions about etiology away from the biological mechanisms stressed by the medical model toward psychosocial factors. Subsequently, the concept of character was formulated more clearly by Reich (1933/1949), who proposed that psychosexual conflicts lead to relatively fixed patterns that he referred to as “character armor.” Reich also influenced diagnostic concepts of PD because his interest in treating characterological conditions with psychoanalysis led to the description of individuals who were neither psychotic nor neurotic, which ultimately led to concept of BPD, also considered largely psychosocial in nature. The phenomenological tradition was also interested in borderline conditions, although these were understood differently. The “border” in which these phenomenologists were interested was between normality and psychosis stemming from observations that patient’s family members often showed unusual features, a conception that was more rooted in the medical model. Hence prior to DSM-III, the term “borderline” referred to a variety of syndromes derived from diverse positions (Stone, 1980) and hence conceptualized and described differently: Those derived from phenomenological psychiatry were largely descriptive concepts, whereas those based on psychoanalysis were described in terms of inner mental structures and processes. Later, psychoanalytic concepts of PD were further extended with the formulation of narcissistic conditions by Kohut (1971) and others. This period from approximately the 1930s to the 1970s was associated with strong reactions against the medical model by many psychoanalysts and to a substantial decrease in interest in classification, although much more so in America than in Europe.

The 1960s and 1970s saw the first empirical investigations with pioneering work of Grinker, Werble, and Drye (1968), followed quickly in the United Kingdom with studies by Presly and Walton (1973) and Tyrer and Alexander (1979). However, the pre-DSM-III era was dominated by clinical description by the classical European phenomenologists and clinical constructs formulated by psychoanalytic thinkers.

Thus, DSM-III was developed in the context of a rich but confusing array of conceptions of PD (see Rutter, 1987). These included PD as (1) a *forme fruste* of major mental state disorders as proposed by Kraepelin (1907) and Kretschmer (1925); (2) the failure to develop important components of personality, as illustrated by Cleckley’s (1941/1976) concept of psychopathy as the failure to learn from experience and to show remorse; (3) a particular form of personality structure or organization as illustrated by Kernberg’s (1984) concept of borderline personality organization defined in terms of identity diffusion, primitive defenses, and reality testing; and (4) social deviance as illustrated by Robins’s (1966) concept of sociopathic personality as the failure of socialization. In the background there also lurked the idea of abnormal personality in the statistical sense, as represented by conceptions of PD derived from normal personality structure. These different conceptions also placed different emphases on the medical model as the basis for conceptualizing PDs.

## The DSM Era

The DSM-III classification and the relatively minor revisions in DSM-III-R, DSM-IV, and DSM-5 (except for parts of the alternative models listed in Section III) have dominated research and treatment. Despite frequent revisions, continuities across editions far outweigh specific changes (Aragona, 2015), and these continuities have profoundly influenced all aspects of the field. The DSM-III decisions to place PDs on a separate axis, and to diagnose them using the diagnostic criteria approach used with other disorders, stimulated clinical interest and empirical research. It is perhaps ironic that these innovations have had such a lasting impact because neither has stood the test of time. Multiaxial classification was abandoned for DSM-5, and the assumption of discrete categories is inconsistent with empirical findings. Nevertheless, the development of di-

agnostic criteria for PDs was an important step: It encouraged construction of semistructured interviews during the 1980s that in turn facilitated empirical research. Although these measures are unlikely to make a strong contribution to future research, they established the importance of psychometrically sound measures.

To appreciate the impact of DSM-III, it is useful to recall the context in which it was developed. In the decades preceding its publication, psychiatry was under attack from many directions (Blashfield, 1984). First, psychiatry's credibility was challenged by concern about diagnostic reliability and marked international differences in diagnostic practices. Second, concerns were voiced from multiple sources, including humanistic psychology, psychoanalysis, and the antipsychiatry movement, about the emphasis placed on the medical model and its relevance to psychiatry. Third, criticism also arose from sociology and labeling theory that the diagnostic labels psychiatrists used became self-fulfilling prophecies that strongly affected the person being labeled. This criticism was reinforced by Rosenhan's (1973) study showing that mental health professionals could not differentiate severely mentally ill from healthy individuals. The study involved eight healthy individuals seeking admission to 12 different inpatient units. They reported accurate information about themselves except their names (to preserve their privacy) and having heard a voice saying a single word such as "thud" or "hollow." All were admitted for an average of about 22 days, and in 11 instances, participants were diagnosed as having schizophrenia; the other participant was diagnosed as having mania. In all cases, the discharge diagnosis was schizophrenia in remission.

These criticisms led to the formation of the neo-Kraepelinian movement (Blashfield, 1984) that reaffirmed psychiatry as a branch of medicine and the medical model as the foundation for conceptualizing and treating mental disorders. The neo-Kraepelinian credo, as summarized by Klerman (1978), consisted of nine propositions that strongly influenced DSM-III. The propositions with most impact on the classification of PD included the following: psychiatry is a branch of medicine; there is a boundary between the normal and the sick; there are discrete mental illnesses; diagnostic criteria should be codified; and research should be directed at improving the diagnostic reliability and validity. In the rest of this section I critically examine the

DSM classification in terms the medical model and the problem of validity. The intent is not to provide an in-depth review of DSM-III–DSM-5 but rather to highlight issues that are critical to improving the conceptualization and diagnostic classification of PD. A more detailed review of official classifications is provided by Thomas Widiger (Chapter 3, this volume).

### **The Medical Model**

The medical model was the foundation for understanding mental disorders and hence for classification for much of the early 20th century. Subsequently, its role was diluted by the impact of psychoanalysis, and its relevance was challenged by the various critiques of psychiatry discussed earlier. The neo-Kraepelinians sought to change this situation. As a result of their influence on DSM-III, their version of the medical model exerted an enormous impact both directly through an emphasis on discrete syndromes and the search for a major cause and specific pathologies for given diagnoses, and indirectly through the neglect of possible contributions of other perspectives, most notably normal personality research. The neo-Kraepelinian understanding of the medical model more than anything else accounts for the way the study of PD has evolved over the last 30 years and for the failure of the DSM to show evidence of consistent improvement across revisions. This section explores the relevance of this model to PD and its impact on the field.

### **Relevance to PD**

The medical model adopted by psychiatry works best for disorders with a specific etiology and pathogenesis. It does not work well when disorders have complex etiology involving multiple interacting mechanisms (see Kendler, 2012a, 2012b). This circumstance clearly applies to PDs: A wide range of psychosocial and biological risk factors has been identified in the last two decades. Psychosocial factors are extremely variable, ranging from attachment problems to cultural influences (see Paris, Chapter 17, this volume). Each factor seems to exert a small effect, and none is necessary or sufficient to cause disorder. Biological influences have a similar structure. Although PDs are heritable, multiple genes contribute to the predisposition toward PDs, each having a small

effect, so that the absence of a given gene probably has little effect. More importantly, PD does not appear to be explained by a specific genetic mechanism (Turkheimer, 2015). This situation also appears to apply to other biological risk factors. Although there is in PDs an underlying biology in the general sense that any psychological process must be accompanied by some kind of neural event, major biological cause has not been identified. Here, the term “major biological cause” is used in Meehl’s (1972) sense of a biological factor that is found in all individuals with the disorder but not in individuals without the disorder. The failure to find major biological cause is not specific to PDs but has proved elusive for most mental disorders (Turkheimer, 2015). This does not mean that the effort to unravel the biological mechanisms associated with PDs is unimportant. To the contrary, such research can only add to our understanding of these conditions and enhance treatment options. It does, however, mean that these mechanisms need to be understood as part of a complex etiology, and that they are unlikely to be very helpful in resolving taxonomic problems.

The etiology of PD also incorporates a complexity not observed with most medical conditions: The diverse etiological factors contributing to a given clinical picture often influence different components of psychopathology. For example, with the DSM diagnostic construct of BPD, trauma and abuse may primarily affect emotional reactivity and stress responsivity, whereas consistent invalidation may primarily affect self pathology through the development of self-invalidating thinking. This is a very different circumstance from that occurring with many medical conditions in which the primary causal factor is implicated in most symptoms.

Recently, other concerns about the relevance of the medical model to psychiatry have emerged that go beyond matters of etiology by raising questions about the very nature of mental disorders that have prompted the suggestion that psychiatry has a unique status among medical specialties (see Gadamar, 1996). One such conceptual challenge relates to the fact that psychiatry addresses a far wider range of “symptoms” than other medical disciplines (Varga, 2015). Whereas most general medical disorders are diagnosed through relatively straightforward symptoms consisting primarily of sensations, perceptions, and motility anomalies, mental disorders are diagnosed on the basis of more complex, less readily observed features,

including actions, emotions, beliefs, meaning systems, interpretations, motivations, thoughts, and cognitive processes. With PDs, the situation is even more complex. Other mental disorders bear some similarity to general medical disorders in that they may also be represented by symptoms and signs, as are the disorders of general medicine, albeit with more complex symptoms. However, PDs are also diagnosed on the basis of attitudes and traits (Foulds, 1965, 1976), and current diagnostic conceptions also include identity problems, self pathology, relationship issues, and narratives. This introduces a different order of complexity, one that is difficult to capture fully using the disease-as-entity version of the medical model espoused by psychiatric nosology.

A second problem is that features used to diagnose PDs are not necessarily indicative of disorder, a circumstance that applies to other mental disorders. This contrasts with the symptoms of general medicine. Pain, for example, always indicates a change for the normal state, even if the pain is transient and without lasting diagnostic significance. However, it is hard to find a feature of PD that invariably indicates disorder. In fact, it is hard to find any feature that does not occur in healthy individuals. Thus, the significance of a diagnostic item cannot be determined in isolation: It always needs to be evaluated within the context of the person’s total personality and life experience.

The problems created for the medical model approach to classification and diagnosis are compounded by the diverse psychopathology of PD and by the way pathology extends to all parts of the personality system. As a result, many psychopathological features are common to multiple putatively distinct diagnoses, and few features are specific to a given condition. Discrete and nonoverlapping clusters of symptoms so characteristic of general medical disorders do not occur with PD. This fact that this has often been downplayed and even ignored by DSM in order to create distinct types has sometimes been distorted the way PD is represented. A good example is the decision to exclude quasi-psychotic features and transient psychotic states from BPD criteria in DSM-III in an attempt to ensure a clear distinction from schizotypal personality disorder, a decision later reversed in DSM-IV.

The rich and diverse pathology observed in all cases creates the additional problem of how to decide what features to focus on for diag-

nostic purposes. With most disorders in general medicine, symptoms are obvious, few in number, easily identified, and closely related to tissue pathology. PDs are palpably different in this respect in that they represent differences in kind. As a result, rules or guidelines are needed to establish what is and what is not pertinent to diagnosis. Currently such guidelines are poorly developed. With DSM, diagnostic features were selected through a committee process presumably guided by traditional clinical opinion. As a result, most sets of criteria are a mixture of items that include general behaviors, specific behaviors, traits, interpersonal matters, self-problems, and self-attitudes, and the constructs used vary widely across diagnoses. The case could be made that some medical conditions are symptomatically more diverse than has been suggested. However, this merely strengthens the case against applying the diseases-as-entity model to PDs. Such disorders tend to have a complex etiology, and these are the disorders that have prompted the observation that the medical model is not even applicable to some disorders of general medicine (Bolton, 2008; Kendler, 2012b).

The contemporary study of PDs has either largely neglected these problems or reframed them in terms of the medical model. Thus, diagnostic criteria are commonly referred to as “symptoms” of PD even though they are highly inferential in nature and radically different in content and form from the symptoms of general medicine. The traditional medical practice of defining symptoms as features of illness that patients complain about is neglected in what often seems to be an attempt to medicalize PDs. Similarly, diagnostic overlap due to the absence of discrete boundaries between putatively distinct disorders and the failure to conceptualize distinct entities is referred to as “comorbidity,” although the term was originally developed to refer to the co-occurrence of distinct conditions. This casual use of “medical” creates that impression of continuity between psychiatry and general medicine when there are important differences and imply the relevance of the medical model when this is not the case. The rigid application of such a narrow version of the medical model to PDs has led to the continued use of a mode of diagnostic assessment ill-suited to either understanding and treating the heterogeneity and individuality of clinical presentations or providing the foundation for a science of PD.

### ***Consequences of the Medical Model***

The version the medical model applied to psychiatry and PD has hindered progress by focusing attention on the identification of discrete types, decreasing interest in alternative models, and inadvertently leading to a neglect of psychopathology.

### ***Assumption of Discrete Categorical Diagnoses***

A brief examination of recent articles in key journals or conference presentations reveals the extent to which research and treatment are dominated by the assumption that disorders distinct from each other and from normal personality variation exist. We only need to look at how DSM performs in practice to see that the system is fatally flawed. The rampant patterns of diagnostic co-occurrence refute the neo-Kraepelinian assumption of discrete disorders on which DSM-III to DSM-5 rest, and the problem is compounded by the prevalence of *personality disorder not otherwise specified* (Verheul & Widiger, 2004). There is no need to look beyond DSM to realize that it fails to meet its design criteria. However, if we turn to research designed to evaluate the system, the magnitude of the problem is even more apparent. We have known for nearly a quarter of a century that the features of PD are continuously distributed (see early reviews by Livesley, Schroeder, Jackson, & Jang, 1994; Widiger, 1993), conclusions confirmed by the failure of more recent studies to identify replicable personality types (Eaton, Krueger, South, Simms, & Clark, 2011; Leising & Zimmermann, 2011; Widiger, Livesley, & Clark, 2009). However, the dominance of the medical model is such that the field is impervious to empirical evidence on this point. Perhaps the most blatant example of disregard for evidence is provided by DSM-5: Although the Personality and Personality Disorders Work Group concluded that “personality features and psychopathological tendencies do not tend to delineate categories of persons in nature” (Krueger et al., 2011, pp. 170–171), categorical diagnoses were retained and the work group even opted to retain typical diagnoses in the alternative model presented in Section III of DSM-5.

The consequences of the persistence reliance on categorical diagnoses are not trivial. Considerable research effort is devoted to studying problems such as diagnostic overlap, which are largely artifacts of the assumption of discrete

disorders, and to identifying the most effective way to diagnose each type. However, the effects of pursuing pseudoproblems are modest compared to the extent to which the category assumption distorts research by influencing the problems studied, the research questions asked, and the methods used. It also promotes the assumption that there is a limited array of PDs as opposed to multiple ways in which personality can be disordered, an alternative clinical conception that I explore later.

### *Inattention to Psychopathology*

An unintended consequence of the DSM's adherence to medical model and attendant emphasis on reliability is the comparative neglect of the broader psychopathology of PDs. There is a tendency to assume that DSM is the ultimate authority on a disorder and its psychopathology leading to a preoccupation with whether patients "meet criteria" for a given condition (Andreasen, 2006). There is also a tendency to equate diagnostic criteria with the diagnostic construct rather than to recognize criteria as a few of many possible indicators of an underlying condition. The authority placed in sets of DSM criteria also had an ossifying effect that has discouraged exploration of alternative conceptual frameworks. Some authors also see this stance as contributing to a growing disconnect between advances in the neurosciences and psychiatry (Hyman, 2010). However, more concerning in the case of PDs is how heightened concern with reliability has led to an impoverished understanding of psychopathology. Diagnostic criteria are essentially lists of relatively superficial features selected from a wide range of possibilities rather than definitive definitions as is so often assumed. Each criterion also tends to be seen as a distinct "self-contained" entity that can be assessed independently of the personality and the individual's other qualities and life experiences. The result, as Andreasen (2006) noted, is that DSM inadvertently led to a neglect of descriptive psychopathology and to a dehumanizing effect on clinical practice. Although Andreasen was referring to the general impact of DSM, her comments seem especially pertinent to PDs. The syndrome-based descriptive categories of DSM seem remarkably crude when viewed against the rich psychopathology of individual cases. They are simply lists of common features divorced from any coherent understanding of the disorder and the complex

processes critical to understanding the psychopathology involved.

### *Neglect of Normal Personality Science*

Another indirect consequence of the medical model is the failure to draw on normal personality research in the search for better conceptual and taxonomic models. This neglect is curiously inconsistent with the medical model that the field seeks to emulate. Disorder is a normative concept that can only be understood with reference to some kind of norm. Within medicine, the norm is the normal structure and function of a given system (Bolton, 2008). This suggests that the norm for understanding PD is normal personality. However, conceptions of normal personality were largely neglected in formulating classifications including DSM-5. This neglect is somewhat understandable, since normal personality research is at an early stage compared to the biological sciences underlying general medicine. Nevertheless, personality science is substantially more advanced than the study of PD and it has the potential to enrich ideas about classification and treatment.

### **The Problem of Validity**

DSM-III was primarily concerned with improving diagnostic reliability to address attacks on psychiatry's credibility, with the assumption that once this problem was solved, attention would subsequently focus on validity (Klerman, 1986). This progression has not occurred. As a result, it is difficult to find evidence that DSM-IV/5 is more valid than DSM-III, or indeed that it is more valid than the taxonomy Schneider proposed nearly a century ago. Nevertheless, proponents of DSM commonly proclaim the validity of both the system and specific diagnoses. Such claims often reflect different understandings of the meaning of diagnostic validity. As Kendell and Jablensky (2003) noted, validity is often confused with clinical utility—the issue of whether a diagnosis is clinically informative. One could argue that DSM PDs have clinical utility because clinicians find them useful, but evidence of validity is lacking.

Confusion also occurs because validity is often approached from the different perspectives of clinical medicine and academic psychology. Although these perspectives are sometimes intertwined, they tend to be pursued

independently and they originated from different concerns. Within medicine, the issue of validity is less prominent, probably because most syndromes are relatively clear cut. Instead, the primary concern is to validate disease status (Zachar & Jablensky, 2015), which is largely a matter of establishing that a person had a given disease. Psychology, from the outset of psychological assessment and test construction, was concerned with reliability (i.e., with whether an attribute is being measured in a consistent way) and validity (i.e., with whether an attribute is being measured accurately). The major development in validity was the elaboration of construct validity by Cronbach and Meehl (1955). Psychological tests are primarily concerned with assessing attributes that are hypothetical constructs, such as intelligence or neuroticism. Construct validation is concerned with demonstrating that a measure actually assesses the construct in question. This is largely a matter of providing evidence to support inferences drawn from the measurement of the construct (Cronbach, 1971). Prior to Cronbach and Meehl, there had been dissatisfaction with the way validity was conceptualized and evaluated. Subsequently, the psychological literature referred to content, criterion, and construct validity.

The differences between medical and psychological approaches to validity became somewhat blurred in psychiatry. The classical contribution to validity in psychiatry was Robins and Guze's (1970) article on establishing the diagnostic validity of specific diagnoses such as schizophrenia. They proposed five phases of validation—clinical description, laboratory studies, differentiation from other disorders, studies of outcome, and family studies—that provide a standard that a psychiatric diagnosis should meet (Zachar & Jablensky, 2015). Application of this approach to PDs reveals major deficiencies. Clinical description is inadequate: Many patients, in some studies, the majority, do not meet criteria for any specific diagnosis and hence the prevalence of the PD not otherwise specified category. Differentiation from other disorders is also poor, with most patients meeting criteria for multiple disorders. However, the important issue for validating PDs is that Robins and Guze's approach shows the same concern with validating indicators of a diagnostic construct focus as the construct validation approach. Also, the proposed phases of validation incorporated elements of content, criterion, and construct validity (Cloninger, 1989), although

construct validity is represented only by delimitation from other disorders. Despite these similarities, Robins and Guze's framework differs substantially from the construct validation model because it strongly reflects the medical model espoused by the neo-Kaepelinian movement that they helped to found by emphasizing the kinds of external validators that are appropriate for confirming a diagnosis in general medicine. The problem is that such validators are not readily available for many mental disorders including PDs. This suggests the need to consider alternative strategies such as those used to validate psychological instruments, most notably Loevinger's (1957) seminal integration of different forms of validity within an overarching framework for conceptualizing and establishing the construct validity of assessment instruments. Although Loevinger was primarily concerned with improving test structure, her approach is relevant to developing and evaluating psychiatric classifications (Skinner, 1981) and offers a model for constructing and validating classifications of PD (Blashfield & Livesley, 1991; Livesley & Jackson, 1991; see also Jacobs & Krueger, 2015).

As conceptualized by Loevinger (1957), construct validity has three components: substantive, structural, and external components. With PDs, "substantive validity" is largely a matter of developing precise definitions of diagnostic constructs based on theoretical considerations and selecting diagnostic items that conform to this definition. This important step establishes a theoretical taxonomy that is then evaluated empirically. DSM-III may be said to represent such a theoretical classification except that it was not constructed to meet the requirements of substantive validity as outlined by Loevinger. Internal or "structural validity" refers to the extent to which the relationships among components of the theoretical classification are supported by empirical evidence. This step establishes an iterative process in which evaluation leads directly to changes in the theoretical classification that are subsequently reevaluated. Over time, the process progressively enhances validity. "External validity" refers to the extent to which the classification and specific diagnoses predict clinical outcomes, have descriptive validity (i.e., differentiate among postulated disorders), and whether the classification is generalizable across different populations. Loevinger argued that construct validity "is the whole of validity from a scientific point of

view” (p. 636) and that the three components “are mutually exclusive, exhaustive of the possible lines of evidence for construct validity, and mandatory” (p. 636).

This framework profoundly influenced test construction. Subsequently, Skinner (1981) showed how it could usefully be applied to psychiatric classification. In contrast to Loevinger’s profound impact on psychological assessment, Skinner’s innovative proposal was largely ignored because psychiatric nosologists have not been interested in the detailed steps needed to establish a classification that possesses construct validity (Blashfield & Livesley, 1991). Nevertheless, the construct validation framework is especially pertinent to classifying PD due to the lack of external validators that could serve as a “gold standard” against which to validate diagnostic constructs (Jacobs & Krueger, 2015) and the need for an iterative process that systematically enhances the system (Livesley & Jackson, 1991, 1992). It is also relevant because the complexity of personality pathology and the diverse ways personality pathology may be organized for diagnostic purposes mean that greater attention needs to be given to structural validity.

### ***Substantive Validity***

Construction of a carefully defined theoretical classification resembles Robins and Guze’s (1970) phase of clinical description. This is crucial step because definitions have a pivotal role in concept formation in science (Hempel, 1961). For this reason, the theoretical classification should include a comprehensive set of diagnostic constructs that encompass all aspects of personality pathology, and each construct should be specified by a set of exemplars (diagnostic criteria or items) that systematically samples all facets of the construct. Failure to meet these requirements incurs the risk of limited coverage of the overall domain of PD and inadequate or biased representation of a constructs.

The attention given to substantive validity in personality assessment contrasts markedly with the almost casual way classifications of PD are constructed. Whereas test construction pays careful attention to construct definition and systematic item development, successive editions of DSM have been produced without systematic definitions of diagnoses or concern with ensuring criteria sets that comprehensively assess all facets of the diagnosis. This has led to

serious and repeated concerns about whether all manifestations of PD are adequately represented and to a tendency to equate criteria sets with the construct, as noted earlier. It has also led to the failure to incorporate important features in the criteria for some diagnoses and excessive diagnostic co-occurrence. For example, with BPD, the conflict between neediness and desire for closeness and fear of abandonment and rejection that is generally considered a core feature of the disorder, is poorly represented. Similarly, the impulsivity criterion fails to recognize the multidimensional nature of impulsivity. As a result, the tendency to experience a sense of urgency observed in these patients is not represented. More problematically, this behavior is assumed to be identical to the impulsivity associated with ASPD, leading to inappropriate overlap with this condition. With careful attention to definition and explicit criteria for establishing and validating diagnostic constructs, issues about what diagnoses should be included or excluded from a classification become little more than political jousts between different factions, as occurred with DSM-5.

### ***Structural Validity***

Structural validity is a necessary feature of classifications (Jacobs & Krueger, 2015). It refers to the extent that diagnostic criteria for a given disorder observed in samples of individuals with the disorder converge with the organization proposed by the theoretical classification. In DSM terms, structural validity requires evidence that diagnostic criteria for a given diagnosis are internally consistent and sort into the diagnostic entities proposed. With DSM PDs, problems with substantive validity pale in comparison to fundamental problems with structural validity. The failure to find evidence that PDs form discrete categories is a challenge to the classification’s basic tenet. Also, structural analyses of DSM personality criteria and PD traits have consistently failed to find structures resembling DSM diagnoses (see early reviews by Livesley et al., 1994; Widiger, 1993). Instead, multiple studies show that four broad factors or dimensions underlie PDs (for a review, see Widiger & Simonsen, 2005). These factors represent emotional dysregulation and associated interpersonal problems centered on attachment insecurity and dependency, dissocial behavior, social avoidance, and compulsivity. The four-factor structure, one of the more robust findings

in the field, is stable across measures, samples (clinical and nonclinical), and cultures. Since these factors cut across DSM-IV disorders, they explain much of the overlap among diagnoses. Unfortunately, these dimensions show limited resemblance to traditional diagnoses: None match DSM diagnoses closely, although similarities exist between these factors and clinical concepts of borderline, schizoid/avoidant, anti-social/psychopathic, and obsessive–compulsive personalities.

### **External Validity**

External validity is based on evidence that the classification shows meaningful relationships with external variables, especially etiological factors and clinical outcomes such as prognosis and response to treatment. As noted previously, external validators are difficult to identify for PD. Even the use of clinical outcomes is a problem because most treatments are relatively nonspecific, with similar effects across different disorders. Nevertheless, concerns have arisen about the external validity of DSM diagnoses. Thus, doubts have been voiced about the value of DSM diagnoses for treatment planning (Sanderson & Clarkin, 2002), and studies suggest that severity of personality pathology is a better predictor of outcome than specific diagnoses (Crawford, Koldobsky, Mulder, & Tyrer, 2011). Also, for reasons discussed earlier, etiology, which was so useful in establishing diagnoses of general medicine, is not helpful with PD. Nevertheless, as Jacobs and Krueger (2015) noted, psychiatric nosology has placed considerable emphasis on external validation, which is often tautological, because common validators such as external variables linked to impairment, disability, and dysfunction are not necessary independent of the diagnoses but rather are incorporated in it. The challenges of external validation are strong reasons for concentrating substantive and structural validity. Systematic application of Loevinger's construct validation framework to PD would eliminate many of the problems with current classifications (Jacobs & Krueger, 2015).

### **The Persistent Influence of Clinical Tradition and the Medical Model**

As the previous discussion documents, we have known for more than a quarter of a century

that although the DSM PD classification lacks structural validity, the field largely functions as if this were not a problem. To move beyond this situation, we need to understand why evidence is neglected and why the field clings to a version of the medical model that does not even apply to some areas of general medicine. Two issues stand out. First, psychiatry is strongly influenced by the philosophical notion of essentialism: the idea that disorders have an underlying nature or pathology (Zachar & Kendler, 2007). Second, cognitive heuristics have a considerable impact on clinical thinking.

### **Essentialism and the Medical Model**

Psychiatry's identification with essentialism is understandable. Psychiatrists' formative experiences with medical disorders seem to have an "essence" in the form of a defined pathophysiology and specific etiology. Not surprisingly, these assumptions were transferred to mental disorders leading to the reification of diagnostic constructs. Although Robins and Guze's (1970) discussion of diagnostic validity had a considerable impact, the field has never really adopted the broader concept of construct validity or shed the primary concern of medicine with confirming diagnoses. Consequently, psychiatric nosology has primarily been concerned with establishing the best way to diagnose conditions whose validity is never seriously questioned. This was clearly illustrated by the way the DSM-5 PDs work group functioned. The validity of diagnoses that have received the most empirical attention was taken for granted; hence, the focus was on how best to diagnose them.

The impact of essentialism is illustrated by Richard Dawkins (2009), the evolutionist, who suggested that essentialism is the reason why it took until the mid-19th century and Darwin to formulate the idea of evolution through natural selection, when the fossil record had been understood for centuries. Essentialism, which led to the idea that each species has an immutable essence or basic nature that cannot be changed, made it difficult to accept the idea that a species can gradually change, until it eventually becomes a new species. Although Dawkins's views have been challenged, his argument illustrates how rigid adherence of essentialism can seriously hinder scientific progress. Essentialism pervades ideas about PD and the field functions as if there is an essence to conditions such as BPD and psychopathy. However, as Kendler

(2012b) subsequently noted, an “approach which assumes that (mental) diseases have single clear essences, is probably inappropriate for psychiatry (and for much of chronic disease medicine). Rather, our disorders can be more realistically defined in terms of complex, mutually reinforcing networks of causal mechanisms” (p. 17). Nevertheless, nosological endeavors continue to assume that the task is to capture the essence of current diagnostic constructs with appropriate diagnostic criteria. Thus, if one set of criteria does not work well, the assumption is that it should be modified, not that the concept should be questioned.

### ***The Impact of Cognitive Heuristics***

The assumption of discrete diagnoses also persists because it is consistent with everyday cognitive strategies and heuristics used to organize information into categories. Our cognitive system seems to have evolved to organize information into categories and force exemplars that straggle several categories into a single specific category. As several authors have noted, PD diagnoses are essentially prototypical categories organized around classical cases that function as heuristics for organizing clinical information (Hyman, 2010). Despite the emphasis placed on diagnostic criteria, most clinicians make a diagnosis by matching patients to their conception of a given disorder. Prototypes seem “real” and intuitively convincing because they are organized around classical cases that are easily recalled; hence, they seem to validate the prototype. In contrast, less prototypical cases are less accessible and more difficult to remember despite that fact that they constitute the majority of cases. It is interesting to note as an aside that DSM-5 seriously considered using prototypes as the basis for classification and diagnosis, and even developed a draft proposal to this effect. Such is the impact of cognitive heuristics on clinical decisions.

Confusion about the value of prototype diagnosis seems to arise because prototypical thinking, like other cognitive heuristics, is effective under some circumstances. These mechanisms permit rapid decisions in situations in which it is better to make a wrong decision if it leads to cautious behavior than to make a slow decision. For example, in the environment in which these mechanisms evolved, it was better to identify a possible predator quickly when there was not a predator than to fail to identify and respond

quickly to an actual predator. However, this does not mean that the conclusion or product of prototypical categorization is invariably correct or that it is useful when making considered decisions. Heuristics are useful because they introduce considerable economy into cognitive functions by organizing information so as to make it readily accessible. However, this economy is achieved at a cost—the process is subject to biases that introduce error into decision making (Kahneman, Slovic, & Tversky, 1982). These biases affect not only thinking in everyday situations but also decision making in professional situations ranging from finance and investing (Ferguson, 2008) to medical practice. These biases consolidate the clinician’s conviction that there are discrete categories of disorder. Given this conviction and its consistent reinforcement in clinical practice, empirical findings are unconvincing. Also, the considerable discrepancy between empirical findings and traditional clinical concepts seems to foster both heuristic thinking and the philosophical assumptions of essentialism that become mutually reinforcing.

### ***Summary and Concluding Comments***

Three broad arguments advanced in this section challenge some of the fundamental assumptions underlying the contemporary current study of PD. First, the applicability of the disorder-identity syndromic version of the medical model espoused by psychiatric nosology has been questioned given the etiological complexity of these conditions and their diverse and wide-ranging psychopathology, including the absence of pathognomonic features. Second, the DSM categorical classification has been shown to lack structural validity. Third, it has been argued that the construct validation framework is the most appropriate methodology for constructing and evaluating a classification of PDs given the psychopathology of PDs and the limited opportunities for external validation.

### ***Charting a New Course***

The conclusions drawn in the previous section point to the need to chart a new course. There seems little point in repeatedly doing the same thing, as occurs with the regular revisions to our main classifications, in the hope of a different outcome. A new conceptual framework is needed to guide research and treatment that

also captures the complexity of PD. In a sense, the conclusions drawn in the previous section are alarming: They appear to challenge the very identity of psychiatry and create uncertainty about how to proceed. However, they may also be liberating conclusions that free the study of PD from the procrustean bed of the syndromic version of the medical model with its discrete syndromes based on concepts derived from diverse and often incompatible sources. Also, there are obvious and potentially fruitful ways to proceed.

Alternative versions of the medical model might be examined for their relevance to understanding mental disorders, including PDs, and the study of PD could do what medicine has always done: Turn to its basic sciences and fundamental disciplines to form a new and more broadly based conceptual framework. However, it seems important to not only turn to the biological sciences but also look further afield and add psychology and philosophy to this list. Drawing on these disciplines, three principles are proposed to establish the metatheoretical underpinnings that could contribute to an alternative conceptual foundation of a science of PD: (1) The normative framework for conceptualizing PD is normal personality; (2) the most appropriate metastructure for describing and explaining normal and disordered personality is evolution; and (3) a comprehensive account of PD requires multiple levels of description and explanation, and a plurality of perspectives. I discuss these principles in the following section and revisit the medical model before briefly considering how such conceptual framework would impact classification.

### ***Normal Personality***

The first strut of a conceptual framework was introduced earlier when I noted that the exclusive focus on the medical model leads to neglect of normal personality science as a source of concepts that might contribute to a valid classification. The principle establishes normal personality as the normative framework for diagnosing PDs and conceptualizes these disorders as pervasive impairments to the structure and functions of normal personality. Although this principle seems obvious—what other frame of reference is possible?—its adoption would lead to a different understanding of PD.

First, implementation of this principle requires that concepts and classifications of PD

be consistent with the findings of normal personality research. This requires the field to relinquish typical concepts of PD, since these were abandoned by normal personality study nearly a century ago and to instead adopt a taxonomy that recognizes that personality pathology is continuous with normal personality variation. Second, the principle implies a more comprehensive view of personality pathology. Normal personality is broadly conceived to be a loosely organized system with multiple structures and processes forming a complex dynamic system. Disorder in such a system invariably encompasses all aspects of the system, leading to multiple forms of impairment rather than to circumscribed patterns. Such a perspective would be clinically useful because it focuses on all aspects of personality, not merely those included in criteria sets including strengths and assets. It also draws attention to assessing broad domains of personality dysfunction such as symptoms, impaired regulatory and modulatory mechanisms, interpersonal impairments, and self pathology (Livesley & Clarkin, 2015; see Clarkin, Livesley, & Meehan, Chapter 21, this volume).

Third, the study of normal personality has traditionally been concerned with not only the contents of personality (traits, motives, expectations, etc.) but also the organization and coherence of personality functioning. Although this aspect of personality has not been a prominent feature of DSM criteria, it provides the basis for developing a systematic definition of PD. Since some form of personality dysfunction is common—most people have some kind of personality quirk—it is important to differentiate dysfunction from disorder. Disorder is more pervasive and involves extensive disorganization of the personality system. Potential markers of such disorganization are the failure to develop a coherent self-structure and chronic interpersonal dysfunction (Livesley et al., 1994). This proposal creates a distinction between the core or defining features of PD and characteristics, such as traits, that delineate individual differences in the way disorder is manifested.

Finally, normal personality study would be useful in defining the scope of a conceptual model of PD. Over half a century ago, Kluckhohn and Murray (1953) made the often cited proposal that personality needs to account for how every person is like all other persons, like some other persons, and like no other person. The idea suggests that an integrative framework for PD needs to account for (1) features common

to all individuals with PDs; (2) features common to some individuals with PDs, that is, individual differences in PD; and (3) features unique to the individual. Kluckhohn and Murray's statement captures a dilemma that has troubled personality science since its inception—the quandary between the nomothetic approach, with its search for broad and preferably universal laws, and the idiographic concern with uniqueness. Researchers are rightly concerned with the nomothetic nature of science, but clinicians cannot afford to ignore the substantial impact of the individuality and uniqueness of individual cases on treatment. For the last 40 years, common and unique features have been neglected in the search for discrete categories of individual differences. However, if a conceptual model is to have clinical utility, it needs to explain both clinically important individual differences and the universal and idiographic features of PD. Unfortunately, this requires an approach to diagnostic classification that is at odds with that of the DSM.

### Evolution

As McAdams and Pals (2006) noted, “Personality psychology begins with human nature, and from the standpoint of the biological sciences, human nature is best couched in terms of human evolution” (p. 206). Millon (1990) made a similar point about PD (see Davis, Samaco-Zamora, & Millon, Chapter 2, this volume). The notion that personality structures and processes evolved because they enhanced the reproductive success of our remote ancestors provides a broad conceptual framework for understanding normal and disordered personality. The idea anchors personality constructs to adaptive biological mechanisms and forces a consideration of what personality structures and processes are designed to do. Such a perspective brings structure to the complexity of personality phenotypes and focuses attention on the functions of personality mechanisms and how they are impaired in PD.

However, evolutionary psychiatry has not gained much attention, largely because most proposals have sought to offer evolutionary explanations for established diagnoses. Since many diagnoses are simply heuristics, the resulting explanations look contrived (Troisi, 2008). Some formulations also assume that disorder results from a mismatch between the ancestral and contemporary environment—

an idea that is not very tenable (Dupré, 2015). However, the framework being proposed does not adopt either approach. Instead, evolution is used as part of a metatheoretical context for conceptualizing normal personality. Since evolution works through the formation of mechanisms that evolved to solve problems occurring in the ancestral environment, an evolutionary perspective implies that *personality structures and processes are either based on, or are the products of, adaptive mechanisms*. These mechanisms form the basic architecture of personality. Since adaptive mechanisms, evolved to solve specific adaptational problems, they are relatively specific in nature (Tooby & Cosmides, 1990). PD is assumed to involve impairments to these mechanisms. Hence, evolution is proposed as a way to conceptualize and thereby clarify the structure that PD takes rather than to explain its occurrence.

Nevertheless, an evolutionary perspective would substantially influence how PD is conceptualized and studied. First, it implies that normal and disordered personality are shaped by an adaptive architecture that places constraints on personality development. However, this does not imply genetic determinism. Adaptive personality mechanisms, such as those underlying personality traits, are influenced by a large number of alleles, each having a small effect. Each allele is probably best considered as increasing the probability of the individual behaving in a given way. Also, the mechanisms linked to personality appear to be highly plastic. During development, they undergo substantial developmental elaboration that gives rise to a variety of phenotypes. The polygenic nature of genetic influences means that we are unlikely to find “genes for personality disorder” or to explain the disorder in terms of a specific genetic mechanism (Turkheimer, 2015), although we are likely to find genes with small effect linked to specific personality characteristics.

Second, the idea that personality phenotypes are based on specific genetically based adaptive mechanisms anchors personality constructs to biological mechanisms and makes the identification and elucidation of these mechanisms a primary research focus. This proposal is consistent with contemporary emphases of mechanisms, as illustrated by the Research Domain Criteria (RDoc) initiative of the National Institute of Mental Health, that seeks to base diagnosis and research on basic biological mechanisms. However, as I argue shortly, it involves

a broad conception of mechanisms as neuropsychological structures that need to be studied using an array neurobiological and psychological methodologies.

Third, since adaptive mechanisms evolved to solve a specific problems (Tooby & Cosmides, 1990), the mechanisms underlying personality are context specific—evolution leads to mechanisms designed to have a specific function not general purpose mechanisms. Hence, the adaptive architecture of personality and PD is complex and highly specific, a proposal that is consistent with the specificity of genetic influences on personality (Livesley, Jang, & Vernon, 2003). This means that the basic constructs used to conceptualize PD need to be relatively narrow in their conception rather than broad like constructs used by current models and classifications.

Fourth, an evolutionary perspective also draws attention to the significance of the interpersonal aspects of personality. In a discussion of the origins of the human mental apparatus, Alexander (1989) argued that a powerful factor in the rapid elaboration of the mental apparatus, including personality, is the need to adapt to the pressures of living in social groups. The threats facing our remote ancestors during the period when the human genome evolved did not emanate from competition from other species because these threats had been largely overcome, but primarily from competition with other individuals from the same species. Successful competition under these circumstances required the evolution of mechanisms that enabled individuals to cooperate effectively with members of their own group. This required a host of adaptations involving language, cognition, and interpersonal behavior. Thus significant aspects of personality have social origins. Finally, it should be noted that an evolutionary perspective means that any account of the etiology of PD should include an understanding of both the distal factors that shaped personality mechanisms in our remote ancestors and proximal factors that constitute risk factors for PD.

### ***Levels of Explanation and Pluralism***

A full account for the different aspects of disordered personality requires an understanding of biological, psychological, and cultural factors. These factors break down into multiple levels of description and explanation: (1) genetic (molecular and aggregated); (2) neurobiological

(molecular and systems); (3) neuropsychological mechanisms; (4) psychological mechanisms; (5) personality constructs and dispositions; (6) personal narratives; and (7) sociocultural processes. Note that the term “level” is used in this context in a general way to refer to differences in abstractness and generalization. Hence, it is being used in the way that the term “strata” is used in geology. Although some levels or strata were laid down before others, none is intrinsically more important than the rest.

The different levels of explanation fall into roughly into two groups. The first two, and possibly three, levels are concerned with publically observable phenomena in the form of neurobiology and overt behavior. The remaining levels deal with epistemologically private activities of mind—cognitions, intentions, meaning systems, and so on, that are largely inferred (Kendler, 2012b). This dichotomy causes a tendency to assume that the more observable levels are in some way more important or essential than more inferential levels. However, PD is primarily a psychological disorder in the sense that its manifestations and treatment are primarily psychological. Also, in the sense that the matter of whether a given phenomenon is indicative of disorder or not is a normative question that can only be decided by reference to psychological functioning. This does not mean that the biological levels are unimportant or less essential, just that they are simply facets of a comprehensive understanding of the disorder.

Since the subject matter of each level is distinct and not reducible to that of other levels, the study of PD needs to avoid what Daniel Dennett (1995) called “greedy reductionism” and Panksepp and Northoff (2009) called “ruthless reductionism.” Within a multilevel explanatory framework, all levels are necessary for a comprehensive account of PD, and no single level is more important or fundamental than the rest. This is an important point because a significant feature of the current *zeitgeist*, especially in American psychiatry, is to view psychiatry as clinical neuroscience. However, this is too limited a perspective for conceptualizing PD, and probably many other mental disorders.

Each level of explanation needs its own language, constructs, and modes of explanation, and the conceptions that emerge at each level are not explicable using the constructs and modes of explanation of the level below. We cannot explain fully higher-level psychological structures such as self and identity even in

terms of lower-level psychological mechanisms such as attention and memory, let alone in terms of biological mechanisms, despite the progress being made in understanding the neural mechanisms associated with these phenomena. The distinctiveness of each level and the importance of all levels for a comprehensive account of PD requires the conceptual framework for PD to be based on what has been called “empirically based conceptual pluralism” (Kendler, 2012a; Longino, 2006). “Pluralism” is the general idea that some natural phenomena cannot be fully explained by a single theory or single mode of investigation (Kellert, Longino, & Waters, 2006). Since a comprehensive understanding of the different levels of personality pathology cannot be provided by a single approach but rather requires contributions from multiple disciplines and different fields within a given discipline, pluralism seems to be the most relevant philosophical approach.

With this approach, different models would be constructed to account for the phenomena at each level. For most levels, multiple models are likely to be needed to provide a full account of the phenomena involved. Although each level is in a sense self-contained, the models need to be consistent with empirical findings about other levels, although one-to-one correspondence across levels is unlikely and unnecessary. The models developed for any level are a facet of a comprehensive account of PD. Consequently, the combination of models developed for all levels form a loosely organized descriptive and explanatory structure rather than a defined theory of PD. To use a term coined by Cartwright (1999), this structure forms a “dappled world.” And, within this dappled world, any account of PD would be incomplete if any facet or level of explanation were missing.

An implication of a pluralistic perspective is that the constructs used to account for PD, including diagnostic constructs, are constructions, and as such they may be useful for some purposes but not others. This idea is at odds with the disease-as-entity syndromic model and with the essentialist assumptions that Zachar and Kendler (2007) suggested underlie contemporary psychiatry. Both assume that diagnoses are fixed entities and that the task is to find the optimal way to represent them. Pluralism is also at odds with most contemporary theories of PD. Earlier, I suggested that current theories are in an early stage of development; however, from the perspective of pluralism, some also appear

to be misconceived since they attempt to offer a comprehensive, unified, and often one-dimensional explanation of PD, although components of some of these theories may undoubtedly contribute to explanation at specific levels.

The syndromic model and the assumptions of some theories also differ from the way philosophy of science is conceptualizing scientific explanation, especially in the biological and psychological sciences, which largely involve models of various kinds (Dupré, 2015). Models are representations of a phenomenon that make the phenomenon more accessible (Bailor-Jones, 2009). They do not provide a total representation of the phenomenon but rather highlight some features and downplay others to facilitate understanding of the critical features of the phenomenon. With PD, models make a phenomenon at a given level of explanation understandable in terms of its important features and functions.

### *The Medical Model Reconsidered*

The medical model adopted by the neo-Kraepelinians and DSM postulates the occurrence of disease entities with distinct and clearly defined boundaries. This is more stringent than the models used by contemporary medicine, which accept that conditions such as some forms of hypertension are extremes of normal variation, a direct equivalent to the idea of PDs as extremes of normal personality variation. Models used by medicine also recognize that some symptoms of a disease such as fever and cough are not due to the disease entity itself but rather are adaptive ways for the body to respond to disease (Bolton, 2008). Again, such responses do not seem so different from the defense mechanisms proposed by psychoanalysis or the coping mechanisms proposed by cognitive therapy to deal with psychological adversity.

It is also clear that the models actually used by medicine accept that some syndromes are defined by a coherent cluster of symptoms that does not arise from a common etiology because they represent the failure of a functional system that may occur for diverse etiological reasons (Nesse & Stein, 2012). Congestive cardiac failure and renal failure are examples. Again, the model is far removed from that of modern psychiatry. However, it does not seem a far stretch to see this formulation as being akin to the idea that some forms of PD may similarly represent dysfunction in a specific personality system

due to different combinations of risk factors. This approach seems to work for medicine because it is based on a detailed understanding of the normal anatomy and physiology of these systems that can be used to explain symptoms. Psychiatry and PD cannot draw on a similarly rich understanding of the functioning personality systems. Nevertheless, they provide a more appropriate conceptual framework than the neo-Kraepelinian credo.

Given the complexity and diversity of models in general medicine, it is puzzling why contemporary psychiatry has so uncritically embraced a version of the model so ill-suited to mental disorders. It is as if psychiatry, which in many ways is considered to occupy the bottom of the totem pole of medical specialties, responded to criticisms from the antipsychiatry movement by seeking to be more medical than general medicine by adopting a more extreme version of the medical model than that of medicine itself. Nevertheless, it is clear that the study of PD need not totally discard the medical model; rather, it should adopt a more liberal version that is compatible with the other principles needed to form a coherent conceptual basis for the field.

### Diagnostic Classification

Problems with the neo-Kraepelinian conception of the medical model and the poor structural validity of current classifications point to the need for a new approach that does not require the occurrence of discrete types. The proposal that conceptions of PD be compatible with those of normal personality, along with the notion that PD represents a pervasive disorganization of the personality system, suggests that it may be more productive to think of PD as single diagnostic entity that is expressed in multiple ways rather than a set of discrete types. In previous publications, I have suggested that these ideas imply a two-component structure to a classification of PD: (i) a representation of general PD and (ii) a system for describing clinically important individual differences in the manifestations of PD (Livesley, 1998; Livesley et al., 1994, 2003).

To flesh out this framework requires attention to both the purposes for which classification is used and accommodation of the complex psychopathology of PD. Blashfield and Draguns (1976) noted that psychiatric classifications serve the multiple purpose of providing

the nomenclature needed for communication, facilitating information retrieval, providing a set of descriptive constructs, predicting outcomes, and forming the foundation for concept development and theory construction. Besides these functions, classifications also serve a variety of administrative functions. Given these diverse usages, it probably unrealistic to expect a single system to cover all contingencies. If we factor into this mix the complex psychopathology of this disorder that leads to individual cases showing features common to all with the disorder, features shared with some with the disorder, and features unique to the individual, the idea of a single classification looks even less feasible. Classification is one area in which a plurality of concepts and models seems especially useful.

To make the task of exploring an alternative approach more manageable, let us consider only the clinical and research functions of a classification. Clinicians are primarily concerned with establishing a diagnosis in order to predict outcome and plan treatment. Most diagnostic evaluations are conducted either as part of a general clinical evaluation or specifically to plan treatment. Although the traditional assumption is that the same diagnostic scheme may be applied to both situations, the needs of these situations are different. With a general evaluation, the intent is to establish whether the patient has a PD or not. This does not require a detailed evaluation of the nature of the disorder or domains of impairment. What matters is whether the patient has a PD that co-occurs with another mental disorder because it is the presence of general PD, not the nature of the disorder, that has implications for clinical management. Assessment prior to initiating treatment specifically for PD is a different matter. Here, assessment of severity is also important because prognosis is more a function of severity than any specific diagnosis (Crawford et al., 2011). Also, severity is useful in determining treatment intensity and the relative balance of supportive or generic treatment methods versus more specific change-focused interventions (see Clarkin et al., Chapter 21, this volume). Treatment planning also requires information on the major constellations of traits present. The four-factor model of personality traits described earlier is sufficient for this purpose. It is assess the broad constellations of emotional dysregulation (emotional and interpersonal traits), dis-social traits, social avoidance, and compulsivity

because these four patterns of disorder are managed somewhat differently (Livesley & Clarkin, 2015). However, a different kind of information is needed to select interventions or modules and tailor treatment to the needs of the individual—information about the specific impairments of individual cases (see Clarkin et al., Chapter 21, this volume). Earlier four domains were described: symptoms, regulation and modulation impairments, maladaptive interpersonal behavior, and self pathology. Assessment of functional impairments associated with these domains makes it possible to tailor treatment to the individual. Since these domains cover a wide range of impairments, a variety of constructs and methods are often required to cover the different levels of explanation involved, and the depth of the assessment of each domain would depend on the nature of the treatment being planned. An additional benefit of incorporating domain assessment into the overall scheme for diagnostic assessment is that it focuses attention on the functional aspects of personality pathology and on specific mechanisms, an important step toward the development of mechanism-based treatment (Livesley, 2017; Schnell & Herpertz, in press).

Diagnosis for research purposes is different. Again, specific DSM diagnoses are not generally helpful because it is difficult to see how data collection organized around diagnoses that lack structural validity can make a substantial contribution to a science of PD. As with clinical practice, different kinds of research have different assessment requirements. For example, with some epidemiological research, a diagnosis of PD and possibly severity may be sufficient because these predict some outcomes better than do specific diagnoses. If more detailed information is required, this assessment could be supplemented with information on the four constellations mentioned earlier.

Many kinds of research, both biological and psychological, however, are not concerned with diagnosing PD but rather with investigating specific mechanisms or constructs. In these cases, a relatively narrow assessment of the construct of interest is needed because adaptive mechanisms have relatively specific functions. This requires the classificatory scheme to include a comprehensive set of specific constructs. Identification of this component of the classification is more challenging, since our understanding of the mechanisms underlying personality pathology is limited. One initial solution would to

focus on the specific or facet-level traits identified through structural analyses of PD traits such as emotional intensity, emotional reactivity, attachment insecurity, lack of empathy, low affiliation, and so. A possible refinement of the approach would be to use a combination of methods to define specific constructs as a step toward delineating specific mechanisms. This would involve identifying a specific or facet-level trait based on factor-analytic studies, then refining the construct using behavioral genetic methods, an evolutionary analysis of the adaptive functions of the phenomenon, and any relevant neurobiological information. For example, research on emotional dysregulation may focus on specific components such as anxiousness, emotional intensity, and emotional reactivity. Anxiousness could initially be defined on the basis of factor analyses of normal and disordered personality and behavioral genetic analyses, showing that anxiousness is a homogeneous construct. Evolutionary analyses could then be used to light on the adaptive functions of anxiousness and a possible underlying adaptive mechanism. For example, Gray (1987) suggested that anxiousness is based in the behavioral inhibition system, a mechanism for managing threat. Together these approaches suggest an initial descriptive formulation of anxiousness and associated mechanism that would be used to construct an assessment instrument. The results of subsequent neurobiological and psychological research on the structure and functioning of the mechanism could then be used to revise the construct. Thus, this kind of research requires a diagnostic assessment system that is far more detailed and specific than is currently needed for most clinical purposes. The specific traits listed in Section III of DSM-5 provide a possible source of some primary traits that might be used for this purpose. However, the value of the overall list is seriously compromised by the fact that the original version was heavily influenced by a committee process that potentially introduced bias into the final list.

In summary, I have argued in this chapter that there are serious conceptual problems with contemporary conceptualizations and classifications of PD. The current mishmash of diagnoses compiled from diverse sources and based on inappropriate assumptions derived from the neo-Krepaelinian position is not conducive to building a coherent body of scientific knowledge about PD. For this purpose, we need a more broadly based conceptual framework and

a different approach to diagnostic classification that would replace the current focus on specific diagnoses with a multifaceted scheme that combines diagnosis and assessment, and makes it possible to tailor assessment to the purposes for which it will be used.

Diagnostic assessment would begin by establishing a single diagnosis—whether a patient has a PD. This would be sufficient for many general clinical purposes and some research endeavors. The nature and depth of any subsequent assessment would depend on its intended purpose. For many research purposes, subsequent assessment would focus on specific mechanisms. However, diagnostic assessment for treatment requires a different kind of evaluation. In view of what we know about the nature and functions of personality and the complex interrelationships among components of personality pathology, assessment prior to therapy requires a broader and more nuanced understanding of the individual's personality system, problems, and assets. This is needed to construct the kinds of narrative case formulations required to plan a structured treatment strategy and to help patients in turn to construct more meaningful narratives and scripts for managing their problems and organizing their lives. Viewed in this way, it becomes clear why a multiple-component diagnostic assessment is needed and why the study of PD needs to be open to the idea of incorporating a plurality of perspectives.

## REFERENCES

- Abraham, K. (1927). *Selected papers on psychoanalysis*. London: Hogarth Press. (Original work published 1921)
- Alexander, R. (1989). Evolution of the human psyche. In P. Mellars & C. Stringer (Eds.), *The human revolution: Behavioral and biological perspectives on the origins of modern humans* (pp. 455–513). Edinburgh, UK: Edinburgh University Press.
- Andreasen, N. C. (2006). DSM and the death of phenomenology in America: An example of unintended consequences. *Schizophrenia Bulletin*, 33(1), 108–112.
- Aragona, M. (2009). The role of comorbidity in the crisis of the current psychiatric classification system. *Philosophy, Psychology, Psychiatry*, 16, 1–11.
- Aragona, M. (2015). Rethinking received views on the history of psychiatric nosology: Minor shifts, major continuities. In P. Zachar, D. St. Stoyanov, M. Aragona, & A. Jablensky (Eds.), *Alternative perspectives on psychiatric validation* (pp. 27–46). Oxford, UK: Oxford University Press.
- Bailor-Jones, D. M. (2009). *Scientific models in the philosophy of science*. Pittsburgh, PA: University of Pittsburgh Press.
- Berrios, G. E. (1993). European views on personality disorders: A conceptual history. *Comprehensive Psychiatry*, 34, 14–30.
- Blashfield, R. K. (1984). *The classification of psychopathology*. New York: Plenum Press.
- Blashfield, R. K., & Draguns, J. G. (1976). Evaluative criteria for psychiatric classification. *Journal of Abnormal Psychology*, 85, 140–150.
- Blashfield, R. K., & Livesley, W. J. (1991). A metaphorical analysis of psychiatric classification as a psychological test. *Journal of Abnormal Psychology*, 100, 262–270.
- Bolton, D. (2008). *What is mental disorder?* Oxford, UK: Oxford University Press.
- Cartwright, N. (1999). *The dappled world: A study of the boundaries of science*. Cambridge, UK: Cambridge University Press.
- Cleckley, H. (1976). *The mask of sanity* (5th ed.). St. Louis, MO: Mosby. (Original work published 1941)
- Cloninger, C. R. (1989). Establishment of diagnostic validity in psychiatric illness: Robins and Guze's method revisited. In L. V. Robins & J. E. Barrett (Eds.), *The validity of psychiatric diagnosis* (pp. 9–18). New York: Raven Press.
- Crawford, M. J., Koldobsky, N., Mulder, R., & Tyrer, P. (2011). Classifying personality disorder according to severity. *Journal of Personality Disorders*, 25, 321–330.
- Cronbach, L. J. (1971). Test validation. In R. L. Thorndike (Ed.), *Educational measurement* (2nd ed., pp. 443–507). Washington, DC: American Council of Education.
- Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological testing. *Psychological Bulletin*, 52, 281–302.
- Dawkins, R. (2009). *The greatest show on Earth*. New York: Free Press.
- Dennett, D. C. (1995). *Darwin's dangerous idea*. New York: Simon & Schuster.
- Dupré, J. (2015). What can evolution tell us about the healthy mind? In K. S. Kendler & J. Parnas (Eds.), *Philosophical issues in psychiatry III: The nature and sources of historical change* (pp. 259–271). Oxford, UK: Oxford University Press.
- Eaton, N. R., Krueger, R. F., South, S. C., Simms, L. J., & Clark, L. A. (2011). Contrasting prototypes and dimension in the classification of personality pathology: Evidence that dimensions, but not prototypes, are robust. *Psychological Medicine*, 41, 1151–1163.
- Ferguson, N. (2008). *The ascent of money: A financial history of the world*. New York: Penguin.
- Foulds, G. A. (1965). *Personality and personal illness*. London: Tavistock.
- Foulds, G. A. (1976). *The hierarchical nature of personal illness*. London: Academic Press.
- Gadamar, H.-G. (1996). *Philosophical hermeneutics* (D. E. Linge, Ed., & Trans). Berkeley and Los Angeles: University of California Press.

- Gray, J. A. (1987). *The psychology of fear and stress*. Cambridge, UK: Cambridge University Press.
- Grinker, R. R., Werble, B., & Drye, R. C. (1968). *The borderline syndrome*. New York: Basic Books.
- Hempel, C. G. (1961). Introduction to the problems of taxonomy. In J. Zubin (Ed.), *Field studies in the mental disorders* (pp. 3–22). New York: Grune & Stratton.
- Hyman, S. E. (2010). The diagnosis of mental disorders: The problem of reification. *Annual Review of Clinical Psychology*, 6, 155–179.
- Jacobs, K. L., & Krueger, R. F. (2015). The importance of structural validity. In P. Zachar, D. Stoyanov, M. Aragona, & A. Jablensky (Eds.), *Alternative perspectives on psychiatric validation* (pp. 189–200). Oxford, UK: Oxford University Press.
- Jaspers, K. (1963). *General psychopathology* (J. Hoenig & M. W. Hamilton, Trans.). Baltimore: Johns Hopkins University Press. (Original work published 1923)
- Kahneman, D., Slovic, P., & Tversky, A. (1982). *Judgment under uncertainty: Heuristics and biases*. Cambridge, UK: Cambridge University Press.
- Kellert, S. H., Longino, H. E., & Waters, C. K. (Eds.). (2006). *Scientific pluralism*. Minneapolis: University of Minnesota Press.
- Kendell, R. E., & Jablensky, A. (2003). Distinguishing between validity and utility of psychiatric diagnoses. *American Journal of Psychiatry*, 160, 4–12.
- Kendler, K. S. (2012a). The dappled nature of causes of psychiatric illness: Replacing the organic–functional/hardware–software dichotomy with empirically based pluralism. *Molecular Psychiatry*, 17, 377–388.
- Kendler, K. S. (2012b). Levels of explanation in psychiatric and substance disorders: Implications for the development of an etiologically based nosology. *Molecular Psychiatry*, 17, 11–21.
- Kernberg, O. F. (1984). *Severe personality disorders*. New Haven, CT: Yale University Press.
- Klerman, G. L. (1978). The evolution of a scientific nosology. In J. C. Shershow (Ed.), *Schizophrenia: Science and practice* (pp. 99–121). Cambridge, MA: Harvard University Press.
- Klerman, G. L. (1986). Historical perspective on psychopathology. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward DSM-IV* (pp. 3–28). New York: Guilford Press.
- Kluckhohn, C., & Murray, H. A. (1953). Personality formation: The determinants. In C. Kluckhohn, H. A. Murray, & D. M. Schneider (Eds.), *Personality in nature, society, and culture* (pp. 53–67). New York: Knopf.
- Kohut, H. (1971). *The analysis of the self*. New York: International Universities Press.
- Kraepelin, E. (1907). *Clinical psychiatry* (A. R. Diefendorf, Trans.). New York: Macmillan.
- Kretschmer, E. (1925). *Physique and character*. New York: Harcourt Brace.
- Krueger, R. F., Eaton, N. R., Clark, L. A., Watson, D., Markon, K. E., Derringer, J., et al. (2011). Deriving an empirical structure of personality pathology for DSM-5. *Journal of Personality Disorders*, 25, 170–191.
- Kuhn, T. S. (1962). *The structure of scientific revolutions*. Chicago: University of Chicago Press.
- Leising, D., & Zimmermann, J. (2011). An integrative conceptual framework for assessing personality and personality pathology. *Review of General Psychology*, 15, 317–330.
- Lenzenweger, M. F., & Clarkin, J. F. (Eds.). (2005). *Major theories of personality disorder* (2nd ed.). New York: Guilford Press.
- Livesley, W. J. (1998). Suggestions for a framework for an empirically based classification of personality disorder. *Canadian Journal of Psychiatry*, 43, 137–147.
- Livesley, W. J. (2001). Commentary on reconceptualising personality disorder categories using trait dimensions. *Journal of Personality*, 69, 277–286.
- Livesley, W. J. (2010). Confusion and incoherence in the classification of personality disorder: Commentary on the preliminary proposals for DSM-5. *Psychological Injury and Law*, 3, 304–313.
- Livesley, W. J. (2017). Psychotherapy for personality disorder: Where are we? Where should we go from here? Where do we need to end up? In J. L. Ireland, C. A. Ireland, M. Fisher, & N. Gredecki (Eds.), *International handbook on forensic psychology in secure settings* (pp. 194–216). London: Taylor & Francis.
- Livesley, W. J., & Clarkin, J. F. (2015). Diagnosis and assessment. In W. J. Livesley, G. Dimaggio, & J. F. Clarkin (Eds.), *Integrated treatment for personality disorder: A modular approach* (pp. 51–79). New York: Guilford Press.
- Livesley, W. J., & Jackson, D. N. (1991). Construct validity and the classification of personality disorders. In J. Oldham (Ed.), *Personality disorders: New perspectives on diagnostic validity* (pp. 3–22). Washington, DC: American Psychiatric Press.
- Livesley, W. J., & Jackson, D. N. (1992). Guidelines for developing, evaluating, and revising the classification of personality disorders. *Journal of Nervous and Mental Disease*, 180, 609–618.
- Livesley, W. J., Jang, K. L., & Vernon, P. A. (2003). The genetic basis of personality structure. In T. Millon & M. J. Lerner (Eds.), *Handbook of psychology* (Vol. 5, pp. 59–83). New York: Wiley.
- Livesley, W. J., Schroeder, M. L., Jackson, D. N., & Jang, K. L. (1994). Categorical distinctions in the study of personality disorder: Implications for classification. *Journal of Abnormal Psychology*, 103, 6–17.
- Loevinger, J. (1957). Objective tests as instruments of psychological theory. *Psychological Reports*, 3, 635–694.
- Longino, H. E. (2006). Theoretical pluralism and the scientific study of behavior. In S. H. Kellert, H. E. Longino, & C. K. Waters (Eds.), *Scientific pluralism* (pp. 102–131). Minneapolis: University of Minnesota Press.
- Maudsley, H. (1874). *Responsibility in mental disease*. London: King.

- McAdams, D. P., & Pals, J. L. (2006). A new Big Five: Fundamental principles for an integrative science of personality. *American Psychologist*, *61*, 204–217.
- Medawar, P. B. (1984). *The limits of science*. New York: Harper & Row.
- Meehl, P. E. (1972). A critical afterword. In I. I. Gottesman & J. Shil (Eds.), *Schizophrenia and genetics* (pp. 367–416). New York: Academic Press.
- Millon, T. (1990). *Toward a new personology*. New York: Wiley-Interscience.
- Nesse, R. M., & Stein, D. J. (2012). Towards a genuinely medical model for psychiatric nosology. *BMC Medicine*, *10*, 5.
- Panksepp, J., & Northoff, G. (2009). The trans-species core SELF: The emergence of active cultural and neuro-ecological agents through self-related processing with subcortical-cortical midline networks. *Consciousness and Cognition*, *18*, 193–215.
- Pearce, J. M. S. (2012). Brain disease leading to mental illness: A concept initiated by the discovery of general paralysis of the insane. *European Neurology*, *67*, 272–278.
- Presly, A. J., & Walton, H. J. (1973). Dimensions of abnormal personality. *British Journal of Psychiatry*, *122*, 269–276.
- Pritchard, J. C. (1835). *Treatise on insanity*. London: Sherwood, Gilbert & Piper.
- Reich, W. (1949). *Character analysis* (3rd ed.) New York: Farrar, Straus, & Giroux. (Original work published 1933)
- Robins, E., & Guze, S. B. (1970). Establishment of psychiatric validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry*, *126*, 983–986.
- Robins, L. (1966). *Deviant children grow up*. Baltimore: Williams & Wilkins.
- Rosenhan, D. L. (1973). On being sane in insane places. *Science*, *179*, 250–258.
- Rutter, M. (1987). Temperament, personality, and personality disorder. *British Journal of Psychiatry*, *150*, 443–458.
- Sabbarton-Leary, N., Bortolitti, L., & Broome, M. R. (2015). Natural and para-natural kinds in psychiatry. In P. Zachar, D. St. Stoyanov, M. Aragona, & A. Jablensky (Eds.), *Alternative perspectives on psychiatric validation* (pp. 76–83). Oxford, UK: Oxford University Press.
- Sanderson, C., & Clarkin, J. F. (2002). Further use of the NEO-PI-R personality dimensions in differential treatment planning. In P. T. Costa, Jr., & T. A. Widiger (Eds.), *Personality disorders and the five-factor model of personality* (2nd ed., pp. 351–375). Washington, DC: American Psychological Association.
- Schneider, K. (1950). *Psychopathic personalities* (9th ed., English trans.). London: Cassell. (Original work published 1923)
- Schnell, K., & Herpertz, S. C. (in press). Emotion regulation and social cognition as functional targets of mechanism-based psychotherapy in major depression with comorbid personality pathology. *Journal of Personality Disorders*.
- Skinner, H. A. (1981). Toward the integration of classification theory and methods. *Journal of Abnormal Psychology*, *90*, 68–87.
- Stone, M. (1980). *The borderline syndromes*. New York: McGraw-Hill.
- Tooby, J., & Cosmides, L. (1990). On the universality of human nature and the uniqueness of the individual: The role of genetic and adaptation. *Journal of Personality*, *58*, 17–67.
- Troisi, A. (2008). Psychopathology and mental illness. In C. Crawford & D. Krebs (Eds.), *Foundations of evolutionary psychology* (pp. 453–475). New York: Erlbaum.
- Turkheimer, E. (2015). The nature of nature. In K. S. Kendler & J. Parnas (Eds.), *Philosophical issues in psychiatry III: The nature and sources of historical change* (pp. 227–244). Oxford, UK: Oxford University Press.
- Tyrer, P., & Alexander, M. S. (1979). Classification of personality disorder. *British Journal of Psychiatry*, *135*, 163–167.
- Varga, S. (2015). *Naturalism, interpretation, and mental disorder*. Oxford, UK: Oxford University Press.
- Verheul, R., & Widiger, T. A. (2004). A meta-analysis of the prevalence and usage of personality disorder not otherwise specified (PDNOS). *Journal of Personality Disorders*, *18*, 309–319.
- Whitlock, F. A. (1967). Pritchard and the concept of moral insanity. *Australian and New Zealand Journal of Psychiatry*, *1*, 72–79.
- Whitlock, F. A. (1982). A note on moral insanity and the psychopathic disorders. *Bulletin of the Royal College of Psychiatrists*, *6*, 57–59.
- Widiger, T. A. (1993). The DSM-III-R categorical personality disorder diagnoses: A critique and alternative. *Psychological Inquiry*, *4*, 75–90.
- Widiger, T. A., Livesley, W. J., & Clark, L. A. (2009). An integrative dimensional classification of personality disorder. *Psychological Assessment*, *21*, 243–255.
- Widiger, T., & Simonsen, E. (2005). Alternative dimensional models of personality disorder: Finding a common ground. *Journal of Personality Disorders*, *19*, 110–130.
- Zachar, P., & Kendler, K. S. (2007). Psychiatric disorders: A conceptual taxonomy. *American Journal of Psychiatry*, *164*, 557–565.
- Zachar, P., & Jablensky, A. (2015). Introduction: The concept of validation in psychiatry and psychology. In P. Zachar, A. D. S. Stoyanov, M. Aragona, & A. Jablensky (Eds.), *Alternative perspectives on psychiatric validation: DSM, ICD, RDoC, and beyond* (pp. 3–24). Oxford, UK: Oxford University Press.