

THE CLINICAL NATURE
OF PERSONALITY
DISORDERS:
*Answering the Neo-
Szaszian Critique*

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ABSTRACT: This article describes five models that could be used to demonstrate that personality disorders are valid clinical kinds: the vulnerability model, the pathoplasticity model, the spectrum model, the decline in functioning model, and the defect model (of which there are three versions). It is argued that the empirically based vulnerability and pathoplasticity models make the best case, given the current evidence, for the clinical relevance of personality disorders. It remains a possibility that a version of the defect model may in due course be developed that provides an acceptable theory of the pathological processes underlying personality disorders and thus more directly validate their clinical status.

WHEN I WAS in graduate school, I inadvertently walked in on a fellow student taking his comprehensive exams. He was extremely frustrated because two of the questions asked about conceptual issues in personality and personality disorders. This student was not expecting such questions and considered them to be unfair. I knew other students in that same program who would have considered it a gift to get such “interesting” questions. Those clinical and counseling psychologists with theoretical–philosophical interests are often attracted to the topics

of personality and personality disorders. It has, therefore, puzzled me why these same topics are largely ignored by those philosophers who study the conceptual issues of psychiatry.

One notable exception is Louis Charland’s (2004, 2006, 2010) neo-Szaszian critique of the Cluster B personality disorders. The Cluster B disorders include the borderline, antisocial, histrionic, and narcissistic personality styles. According to Charland, these diagnoses are primarily identified by morally laden criteria. Their medical or clinical natures, he claims, are not specified. Thomas Szasz (1961) famously suggested that psychiatric disorders such as schizophrenia are better seen as *problems in living* and not as legitimate medical illnesses. Charland’s critique is neo-Szaszian because it does not question the legitimacy of all psychiatric disorders, only the Cluster B personality disorders.

Charland also argues that if a clinician was to successfully cure a case of borderline or antisocial personality disorder, one would likely judge that this patient has become a more moral person. Successful treatment, he says, is therefore “moral treatment,” and quite likely similar in nature to a conversion experience. In short, Charland claims

that the Cluster B personality disorders are really moral, not clinical, kinds.

Although the problem of the overlap between the moral and the clinical could be said to reach back to antiquity, Charland has revitalized philosophical interest in it. Zachar and Potter (2010a, 2010b) explore the moral versus clinical issue from the standpoint of virtue ethics. In doing so, they attempt to articulate a *compatibilist* perspective on the problem. Compatibilism claims that personality disorders can be validly conceptualized using both a moral and a clinical framework. Arguing for a compatibilist position, however, requires answering Charland's charge about the unspecified clinical nature of Cluster B personality disorders. The purpose of this article is, therefore, not to dispute Charland's emphasis on the moral features of Cluster B disorders, but rather to better justify the clinical nature of personality disorders (including Cluster B disorders).

I do not focus on the narrow concept of the medical. If medical means disease entity such as tuberculosis, then many conditions treated by physicians, such as hypertension, are not medical in nature. If hypertension has a "clinical nature," then it is to be found in the reasons for believing that it is a legitimate target for clinical intervention. As Hanna Pickard (2009) suggests, this is a scientific, a social, and a cultural question.

The very concepts of "personality" and "psychiatric disorder" are deserving of philosophical scrutiny, and the combination of the two into "personality disorder" even more so. For example, personality *disorder* might be a term primarily applied to people who are unlikeable (Potter 2009; Zachar and Potter 2010a). Despite these problems and complications, I contend that personality disorders remain clinically relevant kinds, and are more than myths promulgated on an unsuspecting society by the mental health professions.

My list of models for justifying the clinical nature of personality disorders is neither comprehensive, nor original to me. Some models are historically based, others are taken from work on comorbidity and on the relationship between personality and psychopathology, and others draw on previous work (Clark 2005; Eaton, South, and Krueger 2010; Livesley 2003a; Millon and Simon-

sen 2010; Zachar and Kendler 2010).

The models are arranged from the least to the most controversial. In order, they are the vulnerability model, the pathoplasticity model, the spectrum model, the decline in functioning model, and (three variations of) the defect model. It is impossible to keep these models completely separate, but the distinctions are still informative. In the early models, the clinical nature of Cluster B personality disorders is derivative, based largely on considerations of clinical relevance. As we progress through the models, the claims for the clinical nature of personality disorders become bolder, and the inferences required to justify these claims become more abstract and less tied to the evidence base.

THE VULNERABILITY MODEL

Some psychiatrists distinguish between Axis II personality disorders and Axis I syndrome disorders (such as depression and schizophrenia). According to the vulnerability model, personality "pathology" is clinically relevant because it is a risk factor for the development of Axis I syndrome disorders (Millon 1981; Zachar and Potter 2010a). It should be noted that risk factors are not unique to psychiatry—they are accepted as important issues deserving of clinical attention in general medicine as well; for example, hypertension and high cholesterol are risk factors for cardiovascular and cerebrovascular disease. Nor are risk factors in psychiatry limited to personality disorders. For instance, many cases of hypomania are treated primarily because they are risk factors for either mania or clinical depression. Treating risk factors as clinical entities in their own right can prevent more serious problems from developing in the future.

An important justification for the vulnerability model comes from epidemiological research, which has shown that between sixty-six percent and ninety-seven percent of psychiatric patients with personality disorders also meet criteria for at least one Axis I syndrome disorder (Dolan-Sewell, Krueger, and Shea 2001). These include psychotic, mood, anxiety, substance abuse, somatoform, attention deficit, and eating disorders (Dolan-Sewell,

Krueger, and Shea 2001; McGlashan et al. 2000; Oldham et al. 1995).

In addition to categorical personality “styles” and “types,” personality traits such as neuroticism (a “dimension” on which everyone has a value similar to “height”) are also risk factors. High neuroticism is a risk factor for the “internalizing” disorders such as major depression and generalized anxiety disorder (Kahn et al. 2005; Kendler et al. 2003; Krueger 1999). In psychological testing, scales measuring neuroticism are also correlated with somatization, paranoia, and schizophrenia (Morey 1991; Harkness, McNulty, and Ben-Porath 1995). A construct that is a close cousin of neuroticism, termed *general distress*, seems to be present in most psychometric measures of psychopathology.

One compelling theory of the nature of psychiatric vulnerability has been proposed by the psychologist Lee Anna Clark (2005), who emphasizes the role of temperament as a unifying basis for both personality and psychopathology. By “temperament” psychologists mean biologically based emotional/behavioral dispositions that appear at a young age (Goldsmith et al. 1987). A child’s temperament is evident in the first years and tends to be fairly stable throughout life. Psychiatric disorders, Clark says, are more likely to develop in individuals who are more extreme on specific temperament dimensions (particularly those of negative emotionality and disinhibition).

There is a difference between a temperamental disposition such as negative emotionality and a personality trait such as neuroticism. Temperaments are similar to what, inspired by James Russell (2003, 2009), might be called core affect traits. The temperament of negative emotionality is a prelinguistic and noncognitive state of displeasure and high arousal. The personality trait of neuroticism, in contrast, is an elaborate cognitive-level construct that encompasses patterns of thinking about self and others, preferred coping styles, and dispositions to experience more complex emotions such as guilt and shame.

Knowing that temperament developmentally precedes both personality traits and psychiatric disorders, however, still leaves unanswered the nature of the relationship between personality

traits, personality disorders, and syndrome disorders such as major depression. Does something like borderline personality name only a collection of distinct personality traits (especially high neuroticism, impulsivity, and antagonism) or does it name a coherent organization of traits, cognitive–emotional schemas, and coping styles that is more than the sum of its parts?

If the personality traits are the only “real entities,” then the vulnerability relationship between personality disorders and psychiatric symptom clusters can be explained by appeal to the overlapping component traits. If so, then the diagnosis of personality styles can be eliminated in favor of the component traits. Only the traits would be clinically relevant. If the borderline personality style is emergent with respect to component traits, then the whole style should be a distinct risk factor in addition to the risk factors associated with component traits.

In sum, the vulnerability model answers the neo-Szaszian critique by saying that Cluster B personality disorders (or their component traits) are clinically relevant in the same way that hypertension is clinically relevant. They gain clinical legitimacy by being risk factors for high consensus disorders, rather than being clinical kinds in their own right.

THE PATHOPLASTICITY MODEL

According to the pathoplasticity model, personality traits and personality disorders are more than vulnerabilities; instead, they influence and even mold the expression of psychiatric disorders. For example, having a co-occurring personality disorder is associated with earlier onset, increased severity, and worse outcome for Axis I syndrome disorders (Clark 2007).

Consider what differences might be expected were a depressive disorder to be had by a person who is motivated, successful at work, and has supportive relationships, versus a person who is lacking in confidence, isolated, and easily quits. One would expect that the first person’s depressive disorder would have a better outcome. She would likely be more committed to therapy, more compliant with medication if it is prescribed, more

optimistic about what her efforts can achieve, and when she gets discouraged, less likely to stay that way.

Along the same lines, a depressive disorder might develop and be maintained differently in the context of a borderline, dependent, or paranoid personality disorder. The person with borderline personality might experience a dark and brooding depression where he hates everything and everyone, attempts suicide, and seeks to modify his mood with sex, drugs, or other impulsive behaviors. One might expect to see someone with dependent personality have depressive episodes that involve weeping, clinging, and feeling helpless. Clinical depression in a patient with a paranoid personality disorder might involve more isolation, irritability, and complaining.

An important question regarding clinical relevance is whether these variations in expression make a difference in treatment. If they do not make a difference, then there is no reason to make the distinction. Historically, the notion that they make a difference is one of the inspirations for initially recognizing certain personality disorders. For example, the construct of borderline personality disorder was introduced to account for a type of patient who did not improve with traditional psychoanalytic psychotherapy—and often got worse (Gabbard 1994; Linehan 1993). The initial explanation for why they got worse was that they were on the border between psychosis and neurosis. It soon became apparent that although they did not do well with the kind of exploratory therapy used for neurotic (depressed and anxious) patients, they functioned better than psychotic patients (Hoch and Catell 1962; Kernberg 1975; Millon 1981; Zetzel 1968). This diagnosis of borderline personality remains useful because, even if the focus of treatment is depression or anxiety, working successfully with such patients requires a distinct set of management techniques.

In sum, the pathoplasticity model responds to the neo-Szaszian critique by saying that Cluster B personality disorders are clinically relevant because they affect the course of other disorders whose status is not in dispute. It should be noted that this model conforms to Alvan Feinstein's (1970) original reason for introducing the concept

of *comorbidity*—where the course of a primary disorder is complicated because of the presence of another condition.

THE SPECTRUM MODEL

In the spectrum model, personality disorders gain legitimacy by being genetically linked to less controversial disorders. This model can be traced to the seminal work of Kraepelin (1907), who proposed that some personality types were attenuated or frustrated forms (*formes frustres*) of more severe mental illnesses. These included hypomanic, depressive, irascible, and emotionally unstable types. Kraepelin's perspective was developed further by Kretschmer (1925) and in a different form much later by Meehl (1962).

For Meehl, as opposed to Kraepelin, personality disorders on the spectrum are milder expressions of the latent vulnerability, not attenuated forms of the serious mental illness. To illustrate, in DSM-IV terms, a schizotypal personality can be considered a milder (and common) expression of the genetic liability that Meehl called *schizotaxia*, whereas the corresponding severe expression would be disorganized schizophrenia. Schizotypy is not a mild case of schizophrenia—it is a different, more adaptive expression of the underlying genetic liability.

Conceptualizing personality disorders within the spectrum model framework is also diagnostically helpful. For example, the construct of schizoid personality disorder includes lack of interest in close relationships, a preference for solitary activities, and an inability to experience pleasure. From the spectrum perspective, it is a mild manifestation of the same factors that predispose one to simple schizophrenia. People who are robustly independent and occasionally depressed could be diagnosed as schizoid according to the diagnostic criteria—but, unless there is a family history of schizophrenia or some other reason to believe that the patient belongs on the schizophrenic spectrum, a schizoid personality disorder diagnosis might be inappropriate. (Or at the very least, we can be more confident of the diagnosis when there is a family history because the base rate of the condition is higher in families with a history of schizophrenia, and therefore the chances of making a false positive diagnosis are lower.)

Another example of a possible spectrum disorder is the cyclothymic personality—a construct suggested by both Kraepelin and Schneider, and more recently by Hagop Akiskal (Akiskal and Akiskal 2007; van Valkenburg et al. 2006). Kay Redfield Jamison's (1995) compelling description of her own bipolar illness also includes a good account of the cyclothymic personality style. Jamison reports that it was difficult for her to stay on lithium, not only because it required forgoing the enjoyment of hypomania, but also because her own preferred labile personality was blunted by the drug.

Two other examples of potential spectrum disorder constructs are depressive personality disorder and anxious personality disorder. The first is listed in the appendix of the DSM-IV and the second is a personality disorder in the ICD-10. As might be expected, proponents of these diagnoses are inclined to think of them as genetically influenced temperamental vulnerabilities that have been elaborated into dysfunctional personality styles (Akiskal 1983; Huprich 2009; McLean and Woody 1995; Phillips, Hirschfeld, and Shea 1995), whereas opponents see them as early onset and mild forms of syndrome disorders, for example, depressive personality disorder is chronic dysthymia (McLean and Woody 1995).

In sum, the spectrum model responds to the neo-Szaszian critique by saying that some personalities are milder expressions of the same genetic predispositions associated with more serious mental illnesses. Even if they typically do not progress to more severe conditions, the impairments associated with them are deserving of clinical attention.

THE DECLINE IN FUNCTIONING MODEL

This model emphasizes the importance of a developmentally unexpected decline in functioning, or what in the nineteenth century was called morbid change. Such declines provide strong evidence of a disorder (Zachar and Kendler 2010). Broken bones, deliriums, comas, and dementias all involve declines from a previous level of adaptation. Another example is childhood disintegrative disorder, a tragic condition that involves at least two years

of normal growth and development, followed by loss of language, motor, and social skills, and the onset of life-long mental retardation. Declines in functioning are also evident in the least controversial psychiatric disorders, including major depressive disorder, disorganized schizophrenia, and bipolar I disorder.

Personality disorders, however, supposedly do not involve a decline in functioning; instead, a common way of describing them is to say they are *something a person is*, not *something a person has*. Personality disorders are expected to be stable, and to begin in adolescence or early childhood. In the terms of the vulnerability model, they are considered to be “premorbid.”

The exception to this rule is organic personality disorder, which in the DSM-IV is called “personality change due to a general medical condition.” It is diagnosed on Axis I with other syndrome disorders. Here we have a case of personality pathology involving an unambiguous decline in functioning. The decline typically follows a traumatic brain injury or some other neurological insult, and can include impulsivity, emotional lability, poor judgment, rage, paranoia, apathy, flat affect, withdrawal, and lack of empathy. Many of these symptoms are clearly moral criteria in Charland's sense.

The ICD-10 also allows psychiatrists to similarly classify enduring personality changes after a psychiatric illness (such as schizophrenia) or after a catastrophic experience. These later two versions of decline in functioning have been labeled the *scar* model, that is., negative experience leaves the personality scarred (Dolan-Sewell, Krueger, and Shea 2001; Klein, Wonderlich, and Shea 1993).

Responding to the neo-Szaszian critique by saying that Cluster B personality disorders are clinically relevant because they represent a decline from a previous level of functioning would misconstrue the developmental trajectory of most personality disorders. One could, however, highlight their similarities with organic personality disorders. If the same problematic personality traits and associated impairments characterize both organic personality disorders and styles like borderline and antisocial personality, one might assert, on grounds of family resemblance, that borderline

and antisocial personality are also “disordered,” although there is no decline in functioning because they begin at such an early age.

The similarity between the two kinds of personality pathology might be further justified by the presence of aberrant causal histories. For example, a traumatic brain injury represents an aberrant causal history that explains organic personality disorder. Analogously, the high prevalence of childhood sexual and physical abuse seen in many cases of personality disorder could also be considered aberrant casual histories.

The major difficulty with this justification is that not all cases of personality disorder have abuse histories (Paris 2001; Spatz Widom, Czaja, and Paris 2009). Discovering alternative aberrant causal histories raises the problem of first diagnosing the disorder, and second claiming that a certain causal story counts as aberrant. The problem is that those “causes” may be judged “aberrant” primarily because one has already decided that the personality is aberrant. This is similar to the problem of labeling the causes of homosexuality as aberrant and the causes of heterosexuality as normal based on an a priori decision that homosexuality is pathological.

THE DEFECT MODEL

The defect model does not justify personality disorders by means of their association with or similarity to less controversial disorders; rather, it represents a straightforward claim that personality is disordered. Three version of this model are reviewed here: the descriptive approach, the dysfunctional organization approach, and the capacity failure approach. As we move from the descriptive to the capacity failure approach, increasingly more inferences and speculations are required.

Before introducing these three approaches I would like to set the stage by exploring *degeneration theory*—a historically important version of the decline in functioning model. Readers may be familiar with how Eugenics-inspired worries about the proliferation of “degenerates” (supposedly leading to national decline and moral decline) was a primary justification for the implementation of sterilization laws in both the United States and

Germany (Proctor 1888; Lifton 1986). This same concept played an important role in thinking about what was called *the morbid personality*, and especially its association with immoral behavior.

One of the first appearances of degeneration was in a pre-Darwinian depiction of evolution, articulated as a thought experiment by the Comte de Buffon in the 1760s. In Buffon’s speculative proposal, as pure species types encounter different climates, they can either improve or degenerate into different forms (Mayr 1882). This notion of degeneration was quickly adapted to what Gould (1996) terms the monogenist theory of the fall. According to this theory, the different races are degenerated forms of Adam and Eve’s perfection—with some races being more degenerated than others.

The concept of degeneration was introduced to psychiatry by Benedict Morel in 1857, who is best known for coining the term *dementia praecox*—which was later renamed schizophrenia (Berrios 1993; Pick 1989). *Degeneration* referred to a progressive and irreversible decline from a normal human type. Once this process was begun, it would continue across subsequent generations within family groups. Such physical degeneration, it was believed, inevitably included intellectual and moral decline.

By the 1870s, the theory of degeneration had gained popular currency. It had also shed its theological underpinnings and become wed to an evolutionary theory that was a somewhat incoherent mix of Darwin, Spencer, Haeckel, Galton, and Lamarck. Pick (1989) notes that one of the more influential advocates of post-Darwinian evolutionary degeneration theory was the Italian psychiatrist Cesare Lombroso, who proposed that delinquents represented a reversion to more primitive stages of human development. In England, Henry Maudsley picked up on Prichard’s idea of moral insanity and came to see degeneration as a counter force to evolution (Pick 1989). Whereas evolution pushed us forward, Maudsley believed that degeneration pulled us back into decadence. At about the same time, in Vienna, Richard Krafft-Ebbing developed the degeneration model into a theory of a tight relationship between criminality, sexual perversion, and madness (Millon and

Simonsen 2010). Near the end of the century, J. J. Koch introduced the term *psychopathic inferiority* to refer to the “morbid personalities” that resulted from a process of degeneration (Tyrer and Ferguson 2000).

The extent to which this concept penetrated the popular culture can be seen in stories such as the *Strange Case of Dr. Jekyll and Mr. Hyde* by Robert Louis Stevenson. Published in 1886, this novella is often described as an early description of multiple personality disorder, but it is more historically accurate to see it as a portrayal of degeneration (or morbid personality). Nowhere is the degeneration theory origin of Stevenson’s “strange case” better depicted than in the 1931 film adaptation in which Frederick March’s Mr. Hyde was literally made into a Neanderthal (considered incorrectly at that time to be an ancestor of modern humans).

THE DESCRIPTIVE APPROACH (IMPAIRMENT-DISTRESS MODELS)

According to German Berrios (1993), the formal study of personality disorder was initiated by Kurt Schneider in his 1923 book *Psychopathic Personalities*. Unlike the British and American use of the term “psychopathic” to refer to immoral, irresponsible, and antisocial personalities, Schneider used psychopathy as term for pathological personality in general.

In contrast with the advocates of degeneration theory, Schneider (1923/1950) said that the term “illness” is not appropriate for describing personality pathologies because they did not result from a “morbid change in the body” (p. 9). By this time, degeneration theory had been largely rejected by most psychiatrists, including both Jaspers and Freud (Shorter 1997). From its very inception, the clinical nature of personality disorders was not explained by a disease model.

According to Schneider, his ten personality types such as depressive, labile, and explosive were *statistically abnormal*. By this he meant that they represented either an excess or a deficiency of “personal qualities” (p. 3). The statistical abnormality, he suggested, is what makes personality disorder a clinical, not a moral concept. The other necessary criterion for a personality disorder was that, because of the abnormality, either the patient

suffers or he makes the community suffer.

A similar descriptive approach is advocated by proponents of the five-factor model (Widiger 2006; Widiger, Costa, and Samuel 2006; Widiger and Sanderson 1995). According to these thinkers, personality disorders can be comprehensively modeled as extreme values (very high or very low) on the normal personality traits of neuroticism, extroversion, openness to experience, agreeableness, and conscientiousness. Each of these larger trait domains is further subdivided into facets (such as anxiety, angry hostility, and self-consciousness or neuroticism). A personality disorder can be diagnosed when the expressions of the personality traits are sufficiently extreme, inflexible, and are associated with impairment in social role functioning and/or clinically significant distress.

THE DYSFUNCTIONAL ORGANIZATION APPROACH

The dysfunctional organization approach is a lack of balance and harmony model, arguably one of the oldest models for conceptualizing disorders in medical science. Its best known historical example is Hippocrates’ humoral theory of illness, where an imbalance between the four bodily humors was considered to be the cause of sickness. The humoral theory was also the basis for an early model of personality functioning. The four humors with their associate traits are phlegm (reserved/calm), blood (careless/outgoing), yellow bile (irritable/ambitious), and black bile (melancholic/thoughtful).

Psychodynamic models of personality, especially ego psychology models, are also consistent with the dysfunctional organization approach (Hartmann 1958; Reich 1949; Shapiro 1965). In ego psychology, the structure of personality involves three separate and conflicting organizations of experience—id, ego, and superego. The id refers to unconscious and socially—morally unacceptable drives that have been repressed. The ego refers to physiological mechanisms responsible for perception, memory, reasoning, and decision making. The superego refers to a mostly unconscious, strict, and punitive view of morality and also to an ego ideal (or view of an ideal self). The function of the ego is to keep us connected to external

reality, whereas the id and superego are more fantasy based. These three structures are in constant conflict, and the personality results from how an individual works out a compromise between competing demands. The most dysfunctional organizations of character manifest as inflexible, stereotyped, and maladaptive strategies for coping with stress and anxiety.

The dysfunctional organization approach is somewhat transitional between the first (descriptive) and the third (capacity failure) models in this section. Like the descriptive model, it emphasizes the importance of maladaptivity (impairment and distress) in defining a disorder. Like the capacity failure model, it says that the problematic behaviors are the expression of an underlying pathological process. That underlying pathology is what gives personality disorders a “clinical nature.”

THE CAPACITY FAILURE APPROACH (DEFICIT MODEL)

The capacity failure approach is the boldest version of the defect model. According to this approach, a personality disorder represents a failure to develop a psychological capacity that contributes to normal, healthy functioning. Something that should be there is missing. Capacity failure is very similar to what Wakefield (1992) terms a dysfunction. From the deficit perspective, one response to Charland’s critique is to claim that a disorder of personality potentially involves a failure of many psychological capacities, including moral capacities (i.e., empathy and impulse control are psychological capacities that are part of normal development).

Unlike previous models, clinical relevance is not derivative; rather, the failure of normal psychological capacities is psychopathological itself. A conceptual difficulty faced by the capacity failure model that is not a problem for the vulnerability, pathoplasticity, and spectrum models is that deficits cannot be conceptually justified apart from a background theory of normal, healthy functioning. Norms for healthy functioning vary across cultures and historical eras, and, therefore, are partly an expression of values. The concept “health” as what a personality *should* be like is also inherently evaluative.

Otto Kernberg’s (1975) object relations-inspired approach to personality disorder is among the most historically important versions of the deficit model. In general, psychodynamic approaches tend to construe even mental illnesses such as depression, mania, and schizophrenia to be manifestations of personality dynamics. A residue of this perspective can be detected in the naming conventions for psychologist tests, such as the Minnesota Multiphasic Personality Inventory. The Minnesota Multiphasic Personality Inventory, which is currently among the most important psychological tests for assessing psychiatric symptoms, was developed during the heyday of psychoanalytic theory in the United States. Each of its basic clinical scales can be conceptualized as a personality dimension.

Kernberg uses the term *borderline organization* to refer to a group of patients whose representations of self and others are unstable and highly distorted. Kernberg attributes these problems to a lack of cohesive self-structure (the underlying pathology). For such patients, their negative emotions are not tempered by memories of more positive interactions, so something like anger quickly becomes all-consuming rage. Intense negative emotions contribute to self-fragmentation rather than integration (Jacobson 1964). As a result, identity is unstable, regulation of self-esteem is dependent on external events rather than on internal resources, and relationships are conflict ridden (Mahler 1971). The corresponding healthy capacities are the ability to hold good and bad representations of self and others in mind at the same time and to be able to regulate self-esteem autonomously rather than having it completely depend on one’s current emotional environment.

One problem with psychodynamic theories is that they rely on many speculative, “metapsychological” inferences and are jargon laden. Kernberg’s model is no exception. As might be expected, a wide array of alternative deficit models have been advanced, including cognitive (Pretzer and Beck 2005), interpersonal (Benjamin 1996), evolutionary (Livesley 2003b); Millon 1990), and various biological and behaviorally based approaches (Depue and Lenzenweger 2005; Lieb et al. 2004; Linehan 1993). Although each deficit

model proposes a distinct theory about the clinical nature of personality pathology, Livesley (2001) points out that a common theme among many of them is that personality disorder in general involves (1) an impaired sense of self/identity and (2) chronic problems with interpersonal relationships. Currently, none of these models offer well-supported theories of normal self-structure, identity, or interpersonal relationships, and it doubtful that such models could ever avoid value judgments.

In sum, the models discussed in this section answer the neo-Szaszian critique by specifying the nature of the pathological process for personality disorders. The boldest of these models says that personality disorders represent deficits of normal psychological capacities. This approach directly answers Charland's critique, but it is also more vulnerable to a critical analysis because it requires considerable speculation about what counts as normal and about the underlying pathology.

CONCLUSION

Let us return to the general question of whether Cluster B personality disorders are legitimate clinical kinds. As we have seen in our brief exploration, the prehistory of the personality disorder construct—the *morbid personality* of degeneration theory—emphasized decline in functioning. This particular clinical approach was largely rejected in psychiatry by the time Schneider's seminal book was published. Unlike psychotic and mood disorders, personality disorders were formally introduced into psychiatry as neither illnesses nor diseases. They were a kind of clinically relevant problem in living, and remain so to this day. Attempting to answer the neo-Szaszian critique of the Cluster B personality disorders by re-introducing a disease oriented notion (i.e., the morbid personality) would be to misunderstand the conceptual history of personality disorder.

Several thinkers (Pickard 2009; Zachar 2008; Zachar and Kendler 2007) would contend that seeking the essence of a construct such as personality disorder in either conceptual analysis or scientific discovery is a quixotic quest. Abstract concepts such as *personality disorder*, *disease*,

and *natural kind* are conceptual tools that were developed by disciplines such as psychiatry and philosophy to help make progress on their professional problems. These concepts tend to be molded by users to address a variety of problems, and not all of them are consistent with each other.

Quite likely, the vulnerability and pathoplasticity models' arguments for the clinical relevance of Cluster B personality disorders are the most persuasive. Together, these two empirically based models indicate that people diagnosable with personality disorders represent a vulnerable population with respect to psychiatric disorders in general, and for these reasons personality disorders are clinically relevant constructs. Their "clinical nature," however, is indirect and correlational.

Advocates of Charland's neo-Szaszian position would recognize the clinical validity of mood disorders, psychotic disorders, and the Cluster A (paranoid, schizoid, and schizotypal) and Cluster C (avoidant, obsessive-compulsive, and dependent) personality disorders. Although the Cluster A disorders are the primary target of the spectrum model, the so-called clinical natures of the milder Cluster C personality disorders are not any better specified than those of the Cluster B disorders. Are Cluster B disorders being held to a higher standard of adequacy because they are associated with compromised moral capacities? I submit that if the vulnerability and pathoplasticity models can be used to legitimize the Cluster C personality disorders, the burden is on the neo-Szaszians to explain why they do not do the same for the Cluster B personality disorders.

It is unlikely that personality disorder constructs will ever be associated with developmentally unexpected declines in functioning and, therefore, will always be potentially controversial diagnoses; and rightly so. The introduction to the DSM states that the manual classifies not people, but the disorders that people have—by which it means that individual patients should always be considered to be more than "a schizophrenic" or "a depressive." They are persons, not diagnoses. The same caution applies to patients with personality disorder diagnoses, but its application is more complicated. If a personality is what someone is rather than what they have, then something more

central to the patient as a person is being labeled as disordered.

I am of the opinion that, in addition to the descriptive model's emphasis on impairment and distress, developing a credible account of the underlying pathological processes is important for a more satisfactory justification of personality disorder diagnoses. Ideally, a consensus on what counts as a failure of normal psychological capacities that is acceptable to both professionals and patients could be developed. This may even include a way of conceptualizing personality disorders as personally unwanted conditions, much like other psychiatric disorders (i.e., people who are depressed know that there is something wrong with them and do not want to be that way). If possible, over the long run these models should also be acceptable cross-culturally and trans-historically. As is true with mental illnesses such as psychosis and the mood disorders, consensus over the long term is not a proof of clinical legitimacy, but it counts for a lot.

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NOTE

1. By "medical" Charland means a biologically based disease entity and by "clinical" he means a psychological dysfunction. He considers "clinical" to be the broader term, and uses it to avoid getting caught up in turf wars between psychiatrists and psychologists. I follow him in this respect and focus on how the various models support the claim that personality disorders are clinical kinds or clinically relevant.

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