

Chapter 2

Epistemological Issues in Diagnosis and Assessment

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Abstract This book begins with a thoughtful exploration of two fundamental questions that underlie all clinical decisions. First, what exactly is a “mental disorder,” as opposed to other kinds of suffering or maladaptive behavior that we would call *non*-mental disorders? What makes a disorder specifically *mental*? And second, on what do we base these definitions and distinctions? What do we consider reliable (and unreliable) sources of knowledge, and what are some of the pitfalls in our assumptions about what we “know” and how we’ve come to “know” it? Common cognitive errors are explored, along with their consequences. These include circular reasoning, the difficulty of determining threshold or cut-off point, assumptions about causality, and the problems inherent in mental heuristics such as anchoring and availability. The chapter then explores the role of labels and labeling theory, the aims and limitations of classification systems such as the DSM, and the challenge of trying to develop a way to think about mental disorder that is useful for both general purposes (to make predictions based on shared characteristics) and specific aims (to understand and help particular individuals).

Keywords Anchoring • Availability • Continuum • Correlation • Deviance • Dysfunction • Heuristic • Nomothetic and idiographic • Reification • Sensitivity • Specificity • Syndrome • Threshold

Introduction: Why This Matters

What exactly *is* a mental disorder, and how can we know if someone has one?

The first question is ontological (what kinds of things exist) and the second is epistemological (how can we know them). These may seem like abstract inquiries, far removed from the real-world challenges that social work clients face, but they

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are deeply practical questions that lie at the heart of all clinical work. They may not be easily or entirely answerable, but that does not mean that they are not worth asking. To *not* ask is to assume that the answers are obvious and the presumed definitions sufficient—hardly the route to ethical practice.

One reason these questions seldom get asked is that mental illness categories are usually taken for granted in intervention studies, counting as part of the “given” for testing other kinds of hypotheses. This gives a false impression of what is actually known and thus capable of serving as a basis for testing what is *unknown* (Banzato et al. 2005). In order to test or compare interventions for anxiety, for example, we need to be clear about what we are calling “anxiety”; that is the only way for anxiety to serve as a stable background against which other factors can be varied and examined. But reference to a common term does not mean that everyone agrees on what the term signifies. Surprisingly, this fundamental point is often overlooked in discussions about empirically supported treatments. When we fail to attend to these basic conceptual issues, we risk arriving at—and promoting—misleading and potentially harmful conclusions about the best way to help our clients.

Thus, it is important for anyone embarking on clinical practice to investigate two fundamental questions. First, what are the underlying notions about mental disorder that serve as the basis for clinical decisions? And second, what do we consider reliable (and unreliable) sources of knowledge? That is, how did we arrive at these notions? How do we know what we know about the phenomena we call “mental disorder”? How does our “way of knowing” affect our belief about who is disordered and who is not? Are there flaws, traps, or errors that might cast doubt on what we claim to know?

Guiding Questions

1. What are some definitions of “mental disorder” and what is each based on?
2. What is a symptom? By definition, it indicates the existence of something else. How can a symptom establish the existence of a more complex entity, like an illness?
3. How do cognitive habits affect the way diagnostic decisions are made?
4. How do words, labels, and narratives affect the way that difference, distress, and disorder are understood?

What Makes a Disorder “Mental”?

Terms like “mental disorder” and “mental illness” are used to denote problems of the mind or psyche, as differentiated from problems of the body. This mind–body split is becoming increasingly problematic, however, as more is understood about

the mutuality of mental and physical experience. The benefits of physical exercise for mental health and cognitive functioning; the impact of mental conditions such as depression on weight, somatic pain, and fatigue; the way anxiety, purported to be a “mental” state, is experienced through the body as a pounding heart and knotted stomach; and the well-documented power of neuroplasticity—the way life experience shapes the brain as a physical organ, rather than the other way around (Garland and Howard 2009)—are examples of the interconnection between these two realms.

This creates a thorny definitional problem. If, as Berganza et al. (2005) point out, both mental and physical disorders are based on some kind of central nervous system anomaly or malfunction, then where is the crucial difference? Or is there one?

Some authors, like Thyer (see also Chap. 2), take a somewhat different approach in challenging the notion that there are distinctly “mental” disorders. A mental disorder would, presumably, originate and reside in the mind, yet “there are many viable approaches to understanding unusual behavior besides contending that they are manifestations of mental phenomena” (Thyer 2006, p. 65). There is no real reason, Thyer asserts, to account for what a person does or reports by reference to mental processes that cannot be directly observed. A better approach, in his view, is to re-name these phenomena *behavioral* disorders, as long as behavior is broadly defined as “actions, reactions, and interactions in response to external or internal stimuli, including objectively observable activities, introspectively observable activities, and unconscious processes” (Corsini 2002, p. 99, as cited in Thyer 2006, p. 65). A behavioral disorder could thus include disordered thought, feeling, affect, and action.

Behavior, body, brain, nervous system—what, then, is a “mental” disorder?

This is a troubling situation. There is an entire “mental health” industry—more than half a million people in professional practice (according to the US Department of Labor’s Bureau of Labor Statistics); an infrastructure composed of insurance companies, regulations, and an ever-expanding database; a growing body of research, funded by public and private dollars—yet it is not clear if this industry is based on something real. Clearly, people “really” suffer. But is this suffering distinctly mental? Can it be separated from its context, the person-within-a-body and the person-within-an-environment?

On the one hand, many researchers believe that each psychiatric diagnosis will eventually be linked to, and understood as, an underlying neurological dysfunction—that is, that variation among psyches will be explainable by variation among brains. In their view, there is no split: A brain disease is no different from a disease of the liver or kidneys, so we should be able to map psychological problems onto structural or neurochemical anomalies, just as we do for so-called “medical” problems. Yet even if we can do this—that is, find clear correlations between behavioral and neurological differences—there is a snag, since there is no way to know which is cause and which is result. The brain of a child diagnosed with ADHD may appear different from the brain of a child who does not carry this diagnosis, but we do not and cannot know which came first. Because brains are plastic, any observed difference might be the result of the chronic experience of

being criticized for not sitting still, not listening, never feeling accepted or good enough or in control of one's actions—a neurological portrait of shame and anger, real but *secondary* phenomena, distinct from the *primary* features of the disorder.

It is appealing to think we can use a scientific tool such as an MRI to identify brain differences and conclude that these differences are the source of a disorder such as ADHD. However, that only works if *all* children with ADHD display this particular brain difference, *only* children with ADHD have it, and the brain difference was clearly there prior to the appearance of the behavior. That would require a formidable research design: scanning the brains of every newborn, following up with repeated scans until adulthood, identifying clear patterns within and between individuals, and then ruling out other factors that might account for any difference in behavior.

Perhaps the most ambitious undertaking in the quest for a brain-based theory of mental disorder is the Research Domain Criteria Project (RDoC), launched in 2009 by the National Institute of Mental Health. The goal of the RDoC is “to create a framework for research on pathophysiology, especially for genomics and neuroscience, which ultimately will inform future classification schemes” (Insel et al. 2010, p. 748). The project begins from several assumptions:

First, the RDoC framework conceptualizes mental illnesses as brain disorders ... [that] can be addressed as disorders of brain circuits. Second, RDoC classification assumes that the dysfunction in neural circuits can be identified with the tools of clinical neuroscience, including electrophysiology [and] functional neuroimaging ... Third, the RDoC framework assumes that data from genetics and clinical neuroscience will yield biosignatures that will augment clinical symptoms (p. 749).

Neuroanatomy is not the first explanatory principle that is been suggested, of course, simply the most recent. Across cultures and epochs, mental disorder has been linked to punishment for sins in present or past lives, sorcery, curses, spirit possession, misaligned organs, and repressed sexual desire. “From capture by evil spirits to the wages of sin, from the loss of soul to breaches of sacred taboos, to conflicts and tensions in our internal psychological dynamics to dysfunctional families and parents” (Saleebey 2001, pp. 151–152), people have sought an explanation for human suffering. Less than a century ago, much of the emotional distress and maladaptive behavior that we now explain as neurochemical failure was seen as the consequence of moral failure—a weakness of character or a lack of will. In an epoch that idealized the self-made pioneer, this idea was culturally syntonetic, neatly tying happiness to merit and suffering to a lack of moral fiber.

By mid-century, this was supplanted by a psychodynamic explanation in which mental illness was due, instead, to poor parenting—coldness, inconsistency, inadequate discipline, and lack of maternal bonding. Kanner's theory of the “refrigerator mother” who caused schizophrenia and the evils of “permissive parenting,” leading to hyperactivity and conduct disorder, were the examples of this kind of thinking. Bad parents replaced bad character. Then, in the 1990s, the “decade of the brain” (per President Bush's 1989 declaration), bad brains replaced bad parents. Some people were simply born with unfortunate neural wiring, an organic

mechanism that was treatable medically, like any other organic flaw. The idea of a no-fault, brain-based explanation had wide appeal, offering the hope of cure and control (Saleebey 2001). Reinforced by the pharmaceutical industry and a new wave of psychotropic medications, led by *Prozac* as it hit the U.S. market in 1987, this notion revolutionized the way Americans thought about mental disorder. Neurosis and repression were out; neurochemistry was in.

As Leo and Lacasse discuss later in this book, however, research does not support the idea of mental disorder as chemical imbalance; it is simply the most recent idea to capture popular imagination, fitting into the prevailing cultural narrative just as the notion of disorder as moral weakness fit into the cultural narrative of our predecessors.

In short, different notions of causality have shaped the beliefs about mental disorder that have been embraced in different epochs and by different groups—as if knowing what *causes* something is equivalent to understanding what it *is*. This is odd, since we know that many causes can lead to the same result. Childhood hyperactivity, for example, can be affected by diet, allergy, lack of sleep, exposure to lead paint, sensory overload, undiagnosed learning disability, giftedness, trauma, and a host of other factors. So too, a particular event or anomaly can lead to a range of different consequences. Yet the search for a causal definition of mental disorder has persisted.

In general, there are three ways that people try to explain mental dysfunction. One is to seek a fundamental “first principle”; currently, this is the brain, its structure and chemistry, although there could be and have been other principles. This approach rests on what Brown (2002) calls a *mechanistic* epistemology. In a mechanistic epistemology, we look for ways to show that A causes B or depends on B; the metaphor is a chain, with links that connect one event to another. Connections are linear, and the temporal order is clear. Another approach is transactional. Here, mental disorder is seen as the inability to handle the cumulative or intersecting stressors in one’s life. In this *contextual* or *contingent* epistemology, disorder would not exist if not for the presence of these external stressors, exacerbated by a lack of adequate supports and coping strategies. In a contextual epistemology, we seek to understand the relationship between A and B; the metaphor is a tapestry or web, a net of connections. A third approach views mental disorder as a social construction as the way those in power label people who do not conform to their notions of normalcy (a *constructivist* epistemology). Its source lies in the power of the dominant group to define who is normal and who is not.

Each way of thinking about mental disorder rests on a different approach to knowledge. It is not a question of which is best, since epistemology is unstable (Banzato et al. 2005). That is, epistemology is conceptual, not empirical. There is no way to compare the “evidence base” for each theoretical framework and demonstrate that one is superior or inferior to another, just as one value system cannot be shown to be better or worse than another value system. The terms, premises, arguments, and forms of evidence of one conceptual system may seem inadequate, irrelevant, or just plain wrong to those operating under a different system (Brown 2002). Debates about clinical methods and effectiveness are, in many cases, really debates about epistemology.

Thinking About Thinking

Given the elusive nature of mental disorder, it can be tempting to escape from what may seem like an endless hall of mirrors by saying that it simply does not exist (the “denialism” discussed in the next two chapters) or by taking refuge in cognitive shortcuts that seem to make this Gordian knot more manageable. These habits of thought are so common, so invisible, and—often—so misleading that it is important to begin by taking a careful look at *how* we think, as well as *what* we think.

Circular Reasoning As noted above, there is a tendency to define mental illness by its cause—to say, for example, that mental disorder “is” a malfunctioning of the brain. This replaces one challenge with another, however. Demonstrating causality would require a controlled prospective experiment, isolating two groups of human beings who are identical except for a core feature, hypothesized to be causal, in order to see if one group develops a mental disorder and the other does not. In a curious twist, cause is often inferred *retrospectively* by “explaining” an illness through its “cure”: if medication alleviates the symptoms, the symptoms must have had a biomedical origin and hence the condition itself must be biomedical. Yet that would be just as absurd as stating that because Advil relieves a headache, the headache must have been due to insufficient Advil (Erikson and Kress 2005). In medical practice, no physician would define a skin rash as the disorder and its disappearance as the explanation (McWilliams 2011).

Unfortunately, the tail often wags the dog when “tests” are used to demonstrate the existence of the very condition the person is presumably being “tested” for. Diagnostic terms are assumed to represent internal entities, which are then assumed to explain the dysfunctional thoughts or actions—the label thus explaining the very events from which it is inferred (Brown 2002). In this kind of circular definition, conclusions and premises “prove” each other. If the child scores high on a checklist for ADHD, indicating that he or she “has” the condition, this is taken as proof that the checklist is an accurate way to establish the presence of the disorder!

If you think about it, this makes no sense. It stands to reason that a child who scores high on a checklist of ADHD symptoms will also meet DSM criteria for the disorder, since the checklist is based on those symptoms. But this does not prove that these particular items (parts) are the best way to determine the presence of ADHD (the greater whole)—no more than a person’s SAT score has proven to be the best way to determine intellectual aptitude.

Thyer (2014) makes a similar point. “The existence of most of the conditions labeled by the DSM-5 are inferences made on the basis of the very behaviors these so-called mental illnesses are said to cause,” he writes. “If the only evidence for the disorder is the behavior the disorder is said to cause, then there is no genuine explanation” (p. 165). We decide that someone has ADHD if he or she displays the behaviors that constitute the definition of ADHD, but that does not explain what ADHD *is*, other than a collection of behaviors. Calling the behaviors a syndrome and assuming that they are linked to a common etiology, presumed to lie in brain

chemistry or genetics, does not explain anything unless it can be shown how specific anomalies of brain anatomy precede, predict, or at the least correlate consistently with these behaviors. But we cannot do that. In the absence of independent biomarkers, the assumption is made that symptom clusters are (somehow) sufficient to identify the distinct forms of pathology of which they are presumed to be either a part or a result (Kirmayer 2005). But without independent evidence, the reasoning is circular.

The problem is not new. It was noted nearly a half century ago by Temerlin (1968), reflecting on the challenge of “separating *diagnoses* [italics in the original] of mental illness from mental illness itself, if it exists, because there is no operational criterion of mental illness which is independent of psychiatric diagnosis and with which psychiatric diagnosis might be correlated in a validity study” (p. 353). In other words, criteria are not independent operational markers—even if checklists give the impression that they are.

Mixing Levels and Frames of Reference Other cognitive errors occur when we try to explain one level of organization by reference to another. Reference to a higher, more abstract level is *reification*: Assuming that something like depression has an independent existence, above and beyond its manifestations, even though we cannot ever know it. Reference to a lower, more concrete level is *reductionism*: “explaining” experience by reference to one of its components, like equating depression with a decrease in serotonin levels. Low serotonin may be part of depression, but it is unclear if it is a cause, result, or correlate. While mental processes may have biological underpinnings, “they [still] require other levels of conceptualization, since biology does not directly translate into overt behavior, normal or otherwise” (Berganza et al. 2005, p. 168). Reification of notions like depression has become so commonplace that it is scarcely questioned, especially when “authority figures such as psychiatrists use the term and act as if it is unproblematic” (Gambrill 2005, p. 256). Yet even Thomas Insel, director of the National Institute of Mental Health, has said that DSM categories do not refer to real entities that actually exist “out there” in the world (Insel 2013, as quoted in Jacobs 2014).

What *do* DSM categories refer to, then? If diagnostic terms are not indicative of discrete conditions, does it mean they are useless? Not necessarily. It depends, as Brown (2002) points out, on what we require of our terms. Terms may refer to “natural kinds,” classes of objects bounded by underlying, defining properties: this is an essentialist or naturalist approach. They may also be linguistic conventions: a pragmatist approach. In this case, categories are simply useful ways of sorting phenomena into groups in order to promote a shared understanding (Haslam 2000; Markova and Berrios 2009). Here again, the point is to make sure we know which approach we are using and not to mistake a pragmatist approach for an essentialist one.

Confusion can also occur when observational and experiential phenomena are assumed to be mutually referential, each serving as a proxy for the other—that is,

when we assume that externally observed criteria map onto internal experience. They might not. A person might be *acting* depressed yet not report *feeling* depressed. In that case, which do we trust? Does *being* depressed depend on acting or feeling? There is no rule, so it is up to the clinician's judgment. As Berganza et al. (2005) point out, diagnostic assessment often requires a double interpretation: The clinician's interpretation of the patient's interpretation of his/her own experience. There are now two layers between the symptom and the entity to which it is presumed to refer.

A similar difficulty occurs when "hardware," the organic structure of the brain, is confused with "software," the psychological mechanisms through which a person experiences and participates in life. Dysfunctional behavior does not necessarily mean that something is wrong with the organic structure of the brain. "Just as computer software can malfunction even when the underlying hardware is functioning flawlessly, so, in principle, the mind's 'programming' might become dysfunctional for reasons other than that there is a malfunction of underlying brain mechanisms" (Wakefield 2005, p. 85). Since those "other reasons" may be difficult to pin down, it can be tempting to conclude that the fault must lie in the neurological "hardware"—especially since brain activity is easier to observe, measure, and document. But that does not prove the conclusion is correct.

The Problem of Reference, or "What is a Symptom?" In clinical assessment, we use manifestations (symptoms) to make inferences about conditions at a more complex level of organization (diagnoses). The inference is sound if it can be established that the symptom reliably points to a specific referent—that is, signals or emanates from an underlying disordered mechanism and not from other sources. This is tricky, as noted above, because symptoms such as difficulty sleeping can indicate many different problems ranging from anxiety to caffeine, from a disrupted circadian rhythm to a noisy apartment. Again, it is a matter of ontology and epistemology. What sort of "objects" are symptoms, how can we know them, and what kind of knowledge can they provide?

DSM nosology is based on the idea that symptoms are observable behaviors, yet there are symptoms that are not actually "observable," such as delusions or feelings of worthlessness. We can (and do) use what people say as a proxy for internal states and events, although we cannot necessarily be certain that someone is a reliable reporter of his or her inner life. Markova and Berrios (2009) address this dilemma by dividing symptoms into subjective complaints and observable signs.

Subjective complaints are reported changes in a person's internal state, typically undesirable changes. To describe the experience, the person must turn to available categories or images, determined by sociocultural constructs, personal experience, education, and imagination. Complaints are shaped by "personal participation in their construction" (p. 344): whether one calls something sadness, fatigue, pain, hollowness, or a sense of dread depends on personal inclination and the available pool of words and meanings. These are limited in number, varying by epoch, culture, region, class, and gender. As Watters (2010) and others have noted, different cultures have different "symptom pools" that rise and fade over time.

Speaking about the “hysteria” and “hysterical paralysis” common in Victorian times, Watters comments:

This was not a matter of anyone faking those symptoms, but rather that this was the unconscious mind striving to speak the language of suffering for its given moment in history. Suffering, when it comes out in a symptom, is a form of communication. The unconscious mind searches out the expression of the symptoms that would be understood as suffering in that moment in history (www.international.gc.ca/cfsi-icse/cil-cai/magazine/v06n02/1-1-eng.asp).

Emotional distress is thus expressed in the words, metaphors, and somatic manifestations available at a particular time and place. Hearing voices, “running amok,” refusing to eat, pulling out one’s hair, cutting one’s arms, and abusing alcohol may all be expressions or externalizations (that is, symptoms) of the same internal state.

In order for something to be a subjective complaint, according to Markova and Berrios, the internal experience needs to be perceived as a *change for the worse*, and for that the individual must have had sufficient awareness of *another* inner state, perceived to be more typical or positive. Without a baseline, the sense that something is wrong may be less clear. Distance from the baseline determines the perceived severity of the experience, yet subjective descriptors of severity cannot be matched against some sort of objective grid since the same experience may be felt as devastating depression by one person and as feeling a bit blue by another, depending on where they started. Thus, the relationship between an experience and an individual’s judgment and the subsequent naming of that experience can vary.

Observable signs, on the other hand, are behaviors deemed pathological by others. They require a judgment that the behavior is abnormal rather than normal, weighed against an external rule. This raises questions of values and authority: who decides if something indicates abnormality? The American Psychiatric Association, DSM committee members, researchers, therapists, popular opinion, friends, and relatives?

There are additional challenges. For example, even if symptoms *do* represent an underlying mental problem, not all symptoms “behave” in the same way. Some arise gradually, others suddenly and acutely; some fluctuate, others are constant. What if a person has two or three symptoms of a disorder, but fewer than the minimum required by the DSM? What if those two symptoms are especially intense? Do all symptoms have the same weight and significance? DSM symptoms are unweighted, so we don’t know whether five symptoms is the “right” number for admission into a category, if six or seven or four might be more or less valid (Paris 2013). They are simply a part of the diagnostic algorithm. As Paris argues, a *syndrome* is a group of symptoms while a *disease* is the result of a pathological process. Unless the symptoms on a DSM list share a clear pathogenesis or specific pathway from cause to illness, mental disorders are actually syndromes, not diseases. Without evidence that a group of symptoms inherently cohere into a pathological entity, DSM terms are simply *labels*.

The Problem of Threshold Unless it can be established that mental disorders are discrete entities with sharp borders (“natural kinds”), there will always be the question of demarcation between categories—the line between one disorder and another, and the line between disordered and non-disordered. What is the threshold, the minimum level of pathology, that marks entrance into a category?

Many authors maintain that there is no sound empirical evidence for natural boundaries between clinical syndromes (Jablensky 2005), since conditions described in the DSM are not mutually exclusive and share many features. People also tend to have partial membership in more than one category, with some features of one disorder and some features of another; others have “sub-threshold” or non-specific complaints. Overlap, partial membership, and variation within each category mean that neither *within*-group similarities nor *between*-group differences are as clear as they should be for a good taxonomy. Because of variation and heterogeneity within diagnostic categories, people receiving the same diagnosis may actually be *less* like each other overall, and *more* like people receiving other diagnoses or no diagnosis at all.

Distinguishing between disordered and non-disordered conditions is equally difficult. On what basis do we determine that someone has crossed the line dividing normal life variation or transient response to stress from true “mental disorder”? Listing symptoms does not resolve the question (Jacobs 2014). Is it a matter of severity? That only works if severity is a reliable indicator of clinical significance. Here again, the question is: significant to whom? For some people, even mild symptoms may carry profound distress and impairment.

At the same time, the idea of mental disorder as shading imperceptibly into normalcy can trivialize the experience of those who are truly suffering (First 2005). Even if the point of demarcation along a continuum is arbitrary, that does not mean that the disorder itself is illusory, invalid, or trivial. While dimensional or continuum models allow for a graded transition between normality and pathology, they do not solve the problem of understanding mental illness. At some point on the continuum, the line still has to be drawn. Regardless of the number of gradations, dimensional approaches will always end up being categorical at some point, as differences in degree become differences in kind (Paris 2013).

One solution might be to think of the mental illness–mental health spectrum as a moveable continuum along which an individual shifts from greater to lesser symptomatology, within his or her individual range, depending on what else is occurring. Thus, a graded or continuum model may be more useful for comparing people with *themselves* at various points in time (to assess if a condition is getting better or worse) than for deciding whether a particular individual is disordered.

One-dimensional Thinking When considering complex topics such as illness and well-being, it is risky to rely on just one dimension or domain of knowledge. It is not so much that psychiatric concepts are “wrong” as it is that they represent only one kind of data and one way of thinking; additional forms of data and knowledge may be needed. Clinicians who operate from psychodynamic, humanistic, behavioral, and family

systems perspectives are often troubled by the widespread reliance on descriptive psychiatry to define disorder (McWilliams 2011). Because psychiatry has acquired the status of “expert” knowledge, its language dominates; other terms, metaphors, and narratives that might be used to describe human problems are assumed to be less useful or significant. This affects how problems are perceived, as well as the solutions that are proposed, adopted, and paid for (Gambrill 2005).

Yet there are other forms of clinical knowledge based on temperament, affect, defenses, attachment style, developmental tasks, interpersonal relations, and fulfillment of social roles that can shed equal light on human distress. When the focus is simply on observable behavior, other factors that may be crucial for treatment choices can be overlooked, leading to treatment goals defined by reduction or elimination of the criteria that defined the disorder in the first place. Important personal goals may be excluded that have to do with resilience, flexibility of coping strategies, affect tolerance, sense of agency, healthy attachment, stability of self-esteem, expansion of black-and-white thinking, and other aims that are not necessarily tied to the reduction of target symptoms.

As McWilliams (2011) reminds us, most clients’ suffering cannot be adequately captured by diagnostic categories, nor can their sense of improved well-being be as easily measured as symptom reduction. Equating mental disorder with the presence of symptoms, and mental health as the lessening of those symptoms, trivializes the depth and complexity of our clients’ lives. Yet, this is the approach endorsed in many practice settings, prompted and maintained by third-party payers. Going beyond its narrow focus requires additional effort that may not be recognized by agency personnel—and may even be discouraged because it does not match the computerized treatment plans and billing systems.

One-dimensional assessment can mean looking at only one *type* of information, like behavioral symptoms, or looking at only one *direction* of influence rather than trying to understand the reciprocal transactions between individuals and their surroundings. Most elements of human experience flow in two directions, from inside to outside and from outside to inside. Attributing the locus of a problem solely to individual characteristics that “produce” dysfunctional behavior is as much of an over-simplification as attributing the locus of a problem solely to environmental factors. Even in genetics, an area previously believed to be wholly deterministic and one-directional, we now understand from the study of epigenetics that gene expression is profoundly shaped by environment and experience.

For example, a psychological difficulty like an unstable sense of self might be due to identity conflict from forces such as acculturation following immigration, rapid social mobility, or intergenerational expectations. Assuming that it represents internal pathology such as borderline personality disorder—and should be addressed as such—can lead to ineffective and potentially harmful treatment decisions. Over-simplification can also proceed in the opposite direction. Discounting a person’s claim that dysfunctional behavior is due to external factors—as if that were a mere defensive projection, a way to deflect responsibility—may be equally wrong. It would be like

treating a woman for depression without addressing the abusive domestic relationship that led to and sustains the depressive behavior. Helping someone to function or feel better in an unjust situation is not the aim of social work.

The Power of Cognitive Heuristics

While it may be comforting to imagine a therapist consulting DSM symptom lists, comparing each set of diagnostic criteria with carefully gathered notes on a client's particular troubles, and then deciding which category is the correct match—it does not often happen that way. In the busy world of clinical practice, the choice of DSM label may depend more on the associations in the therapist's mind *at the moment the diagnostic choice is made* than on diagnostic acuity or the scientific evidence behind the various categories.

If that sounds like an alarming statement, think about how we make decisions in daily life. Suppose, for instance, that you want to select a restaurant for dinner. The choices that come to mind are the ones that are most readily accessible, either because you have heard about the place or have been there before. As options, they are more vivid and easier to retrieve, and thus have an edge of credibility—or seem to. In other words, you are more likely to go to a restaurant that is *already been activated in your mind* than to one that has not.

That shows cognitive heuristics work. Cognitive heuristics is the study of the information-selecting and information-processing mechanisms through which decisions are made. Given the overwhelming amount of information available when considering a client's situation, clinicians make choices about what to focus on by using subconscious heuristics, or information-processing shortcuts. Beliefs and the cognitive structures that support them, including prior therapeutic orientation, "have a powerful influence on the information we attend to and how we interpret it. This is particularly true when there is some ambiguity in the cues, as is almost always the case in a helping situation" (Berlin and Marsh 1993, p. 56).

There is considerable research (e.g., Tversky and Kahneman 1973, whose work earned the Nobel Prize) indicating that initial conceptions tend to be formed quickly with minimal information and then act as self-fulfilling prophecies. When faced with difficult decisions, people tend to employ a limited number of cognitive strategies that reduce complex judgment tasks to simpler ones based on comparison and classification. Sometimes the initial classification is valid, but it can lead to errors when the similarity is superficial and results in dismissal of other relevant considerations.

Many diagnostic errors are due to the set of mental heuristics collectively called "cognitive dispositions to respond" (Croskerry 2003). These cognitive shortcuts rely on pattern recognition as a means for placing new information into familiar categories.

The clinician quickly forms a working hypothesis about the nature and cause of the client's dilemma and proceeds to probe for the kind of evidence that will make it seem true. Snap

judgments about what is wrong are aided both by referral information that contains previous diagnoses and by the general ease with which certain ‘preferred’ diagnostic classifications come to mind (Berlin & Marsh, 1993, p. 57).

One powerful heuristic is *availability*, the tendency to judge the likelihood of an event by the ease with which similar instances come to mind due to recent exposure (Berlin and Marsh 1993; Groopman 2007). In a series of experiments, Tversky and Kahneman (1973) found compelling evidence that judgments about the likelihood of an event taking place or someone’s membership in a particular category are affected by the ease with which related instances are recalled. Berlin and Marsh (1993) made a similar point: information that is most accessible tends to be more vivid, specific, personally relevant, and familiar. A clinician who has recently encountered a particular disorder “is more likely to classify ambiguous clusters of symptoms as representing that disorder rather than employing other equally plausible diagnostic categories. Due to recency, frequency, and familiarity ... the diagnostic category is cognitively readily accessible” (Berlin and Marsh 1993, p. 19). If you can think of it more easily, then—because of the power of *availability*—you assume it is more important, correct, and real than alternatives that would require a bit of mental searching and stretching.

The same thing happens in diagnosis. Particularly when a clinician is pressed for time, clients may be more likely to receive one of the popular diagnoses *du jour*—to be placed in a category the clinician has heard about, utilized, and discussed—than they are to be placed in a category the clinician has not encountered or thought about as recently or frequently. If the therapist has several other patients diagnosed with OCD, just read a journal article on OCD, or attended a special workshop about treatment for OCD, it is more likely that a diagnosis of OCD will be considered if it is at all relevant. Popular diagnoses can thus become self-fulfilling prophecies, capturing the interest of mental health professionals who “see” the disorder in ambiguous cases and thus unconsciously contribute to an increase in reported prevalence (Overholser 2014).

Since symptom lists have considerable overlap, flexibility, and room for interpretation, clinical judgments frequently involve a choice between plausible alternatives. It is not simply that having a hammer makes everything look like a nail. The availability heuristic might suggest something a bit more subtle: if the hammer is easier to reach or worked well last time, then that is the tool you will reach for—though you will never know what might have happened if you had picked up the pliers or wrench instead.

Pattern recognition is the heuristic used to assess the fit between new information and familiar categories in order to determine where a new instance belongs. More weight is given to how the new instance resembles the category than to how it differs: in other words, we look for matches and, once the match has been made, ignore evidence that does not support our choice. Once someone has been diagnosed with ADHD, for example, we tend to focus on ways the person resembles an ADHD prototype rather than the ways the person is unique or deviates from the stereotype. People with the same label may seem to be the *same* in some essential

way, and essentially different from everyone outside the category, even if they are actually more different than alike in other ways such as talent, sense of humor, and spirituality.

Another powerful heuristic is *anchoring*, the tendency to lock into salient features early in assessment and then fail to adjust one's thinking in light of subsequent information about other features (Croskerry 2003)—that is, to selectively notice only those features that we expect to see. Like an anchor that sets the position, the first piece of information becomes the reference point for evaluating whatever comes next. “Anchoring” on the fact that the Sunset Café has an outdoor patio can lead a prospective diner to evaluate all other restaurants by the same criterion instead of by other criteria that might be equally important such as price or location. By setting a baseline, anchoring can shape choices and predictions. In one of their early studies, Tversky and Kahneman demonstrated that when asked to guess the percentage of African countries in the United Nations, people who were asked if it was more or less than 10 %—the anchored figure—guessed lower values, on average (that is, closer to 10 %), than those who were asked if it was more or less than 65 %. The percentage that was “anchored” in their minds, just by being uttered, influenced their response.

In the same way, clinicians who have recently diagnosed several people with bipolar disorder are likely to classify new patients as having bipolar disorder if they fall within a general zone of similarity, *even if symptoms are less severe*. Thus, it takes fewer cues to make the category seem plausible because the therapist's “bipolar radar” is already activated. Through availability and anchoring, new cases are judged by less rigorous criteria, inflating the number of people who seem to belong in the category and causing diagnostic rates to soar. This is reinforced, Overholser (2014) points out, by the research process itself. As investigators seek to recruit enough eligible participants, they may soften and expand the diagnostic criteria used for inclusion in the study. This loosening then becomes the new standard. With more people eligible for the label, there is an apparent surge in incidence, creating the impression of an “epidemic” that benefits both service providers and service recipients. Using the example of autism, Overholser notes that “when diagnostic criteria are ‘softened,’ they capture a larger market for professional services, allowing parents to pursue early intervention or special educational services” (p. 55).

In this way, epistemological assumptions and cognitive heuristics contribute to “epidemics.” It is not necessarily that more people are being *born* with autism nowadays, for example; it may also be that more people are being *diagnosed* with autism. Because of public awareness, parents are more inclined to look for signs and to seek help, and clinicians are more inclined to spot the hallmarks more readily. As diagnostic thresholds become relaxed, there is an increase in cases at the milder end of the spectrum. Because of softened borders and “diagnostic creep,” it is easier to gain entrance into the category.

Several heuristics contribute to this process. One is *confirmation bias*, the tendency to dismiss alternative or disconfirming information because of how satisfying it is to re-affirm a decision already made or something already believed to be true. This is reinforced by *satisfaction of search* or *premature closure*, when one stops searching as

soon as something has been found, even though there may be more left to find (Croskerry 2003). “Once an uncertain situation has been perceived or interpreted in a particular fashion, it is quite difficult to view it in any other way” (Tversky and Kahneman 1973, p. 230). *Diagnosis momentum* then sets in, the tendency for a label to get “stickier” over time; once made, the diagnostic decision gains momentum until it becomes solidified and other possibilities are quickly excluded.

Another heuristic that can affect clinical assessment is *dispositional bias* or *fundamental attribution error*, the tendency to attribute the source of the problem to something within the person rather than to factors within the ecological situation—i.e., to overestimate the influence of individual factors and underestimate the influence of situational factors. There is substantial evidence, Berlin and Marsh (1993) maintain, that clinicians unconsciously try to correct for the sense that clients tend to blame their troubles on external factors or project blame onto other people; as a counterweight, they may minimize or simply dismiss client assertions about external causes for their difficulties. If, for example, a client reports feeling unjustly treated and rejected, dispositional bias can cause a clinician to attribute that feeling to an internal problem such as low self-esteem rather than to factors in the client’s ecological situation. This dispositional bias may be reinforced by institutional expectations and the requirement of insurance companies that an internal mental disorder must be cited if services are to be covered.

Other heuristics include *order effects*, the tendency to focus more on what was heard or experienced at the beginning or end of an encounter, and *commission bias*, the tendency to do something rather than nothing, to want to act and be “helpful” (Croskerry 2003). Decisions about how to help are also influenced by the kinds of resources that are easily available or familiar from prior use. Empirical research indicates that any intervention “tends to do better in comparison with other interventions when it is conducted by people who are expert in its use” (Chambless and Hollon 1998, p. 12) and when it corresponds to, rather than conflicts with, the clinician’s own beliefs about how change takes place.

Priming or “Readiness to Respond”

These mental shortcuts are set in motion by a phenomenon called *priming*. In the same way that priming a wall makes it ready to accept a coat of paint, cognitive priming occurs when recent exposure, leading to mental “readiness to accept,” makes certain aspects of a situation more salient and available than they would normally be.

When an individual is asked to evaluate a novel stimulus, such as a questionnaire item, he or she will generate a response by scanning whatever information is available at that moment. The information that is most salient and relevant at the time of the question will be used to formulate a response (Moss and Lawrence 1997, p. 394).

In Moss and Lawrence’s experiment, priming theory was used to show how recent information about stress led participants to report higher levels of stress than

in the absence of priming. Some participants were given pamphlets to read about stress before taking a survey, others were not; those who had read the pamphlet reported higher levels of stress on the questionnaire than those who had not been primed. In another study by Milich et al. (1992), recent information about diagnostic labels led children and adolescents to judge their “misbehaving” peers more harshly and to give them more negative labels.

The mechanisms through which priming operates are category accessibility and confirmatory bias, the unconscious selective search for information that confirms information previously received (Moss and Lawrence 1997). Ledgerwood and Chaiken’s study (2007) further demonstrates how information falling within a person’s “latitude of acceptance” is perceived as *more* similar to the original reference point than it really is, and information falling within the “latitude of rejection” is perceived as *less* similar. A person primed to expect similarity is more likely to find similarity. In the same way, being primed to expect disorder makes a person more likely to find disorder.

Another way that priming occurs is through the suggestion of someone perceived to be an expert or of higher status. Temerlin’s (1968) experimental study offers a vivid example of how this can occur. In his study, a professional actor trained to portray a “normal” mentally healthy man was interviewed as if he were a prospective patient. Five groups listened to the taped interview, each group stratified to represent the same distribution of psychiatrists, clinical psychologists, and psychology graduate students. All were instructed to diagnose the man by drawing from a list of categories in use at the time: psychoses, neuroses, and personality types including “normal” or “healthy.” In order to isolate the influence of clinical setting, two of the groups were told that the interview was either part of a new procedure for conducting judicial sanity hearings or for selecting scientists to work on a new research project. Just before listening to the interview, a “prestige confederate” remarked to the fourth group that the patient on the tape seemed neurotic but was actually psychotic. A fifth group received the “prestige suggestion” reversed. A control group was given no special instruction.

The “prestige suggestion” had the most effect upon psychiatrists—members of the same profession as the “prestige confederate”—biasing them in the direction of psychosis. While no one in the control group diagnosed psychosis, 60 % of psychiatrists in the “prestige suggestion” group did so. Temerlin further notes: “It is doubtful that prestige suggestion could bias medical diagnosis so dramatically, on the theory that the substantive reality of physical illness would counter-balance the distorting effects of prestige suggestion” (p. 353), implying that mental illness is not “substantively real” in the way that physical illness is real.

The diagnostic system of the DSM is a potent vehicle for priming. It is a set of officially sanctioned categories that can predispose its users to “see” clients according to, and limited by, the categories it offers. A number of studies have shown how this happens. Ravotas and Berkenkotter (1998) found that therapists tend to interpret what a client says in a way that supports the DSM diagnosis *they already gave* to that person. In a self-perpetuating circle, the therapist reprocesses whatever the patient says and does in a way that “justifies the therapist’s particular

diagnosis of mental disorder which, in turn, supports the therapist's treatment choice" (p. 211). Client actions and statements that do not fit into the definition of the disorder *already diagnosed* are minimized or ignored, while actions and statements that seem to fit are inquired about more often and in more detail, described in clinical notes, and accorded more importance in case discussions. This tendency is supported by the heuristic of confirmation bias and by the assumption that diagnoses are essentialist categories. As Kirmayer (2005) notes:

Unfamiliar symptoms or problems are reinterpreted to fit a specific prototype or discounted and ignored as minor and irrelevant. Insofar as the patient's experience does not fit the template, the discrepancies are viewed as irrelevant ... This stripping down of illness experience to fit the diagnostic paradigm is justified on the basis of the notion that diagnostic entities have essential characteristics and that what is crucial about the patient's condition can be typified by these core features (p. 195).

Overholser (2014) notes that a clinician's personal interest in a particular disorder can influence what is attended to and how that information is interpreted. "When intake workers have a diagnosis they find intriguing, they are more likely to notice potential symptoms, interpret symptoms as part of the disorder, and assign the label" (p. 54). Use of ready-made diagnostic constructs thus "functions to *black box* many of the dissonances, contradictions, power relations, and situational asymmetries that exist in any institutional context" (Ravotas and Berkenkotter 1998, p. 217).

Cognitive signals that tell you to stop looking can exert a powerful influence, especially when there is pressure to come to a diagnostic decision, often within the first 45-min encounter, since treatment plans and insurance claims cannot be filed without it. Berlin and Marsh (1993) sum up the interplay of these forces in their book on clinical decision-making:

The clinician quickly forms a working hypothesis about the nature and cause of the client's dilemma and proceeds to probe for the kind of evidence that will make it seem true. Snap judgments about what is wrong are aided both by referral information that contains previous diagnoses and by the general ease with which certain 'preferred' diagnostic classifications come to mind (p. 57).

In other words, *believing is seeing* and not the other way around.

Related to priming and the disposition to *respond* is "selective attention," an attentional bias that affects our disposition to *notice*. In their well-known "invisible gorilla" experiment, Harvard researchers Christopher Chabris and Daniel Simons offer a deceptively simple demonstration of how much we miss of what is going on right in front of us—without even suspecting how much we are missing. One of the reasons that we fail to register seemingly obvious elements like the gorilla is that *we're not looking for gorillas*. When we are intent on a predetermined task, whether it is counting basketball passes or counting DSM symptoms, we tend to focus only on information that seems pertinent to our aim and unconsciously filter out information that we do not believe is relevant. As the experiment shows, it is not just that we put this information to the side; we simply do not see it. In clinical assessment, this means there we may fail to perceive—and thus fail to include—information

that is “not on the list” of elements that we’ve decided in advance may be clinically significant.

The process of diagnosing medical conditions is similar to the process of diagnosing mental health conditions—leading to similar errors. Reviewing the literature on diagnostic error among medical doctors, Berner and Graber (2008) point out that there are two general ways of coming to clinical decisions. One is through *hypothetico-deductive reasoning* in which physicians gather initial data and, often within seconds, formulate diagnostic hypotheses; they then gather additional data in order to evaluate these hypotheses and arrive at a diagnostic conclusion. Errors can occur from failure to elicit complete and accurate information from the patient, failure to recognize the significance of that information or, most commonly, failure to integrate facts about a specific patient with general medical knowledge—that is, to synthesize and apply nomothetic principles to an individual case.

As physicians gain experience and expertise, they are more likely to use the more intuitive and subconscious process of *pattern-recognition* (Berner and Graber 2008). As noted above, pattern-recognition works by recalling cases similar to the one under review, attending to what seem to be prototypical features, and matching them to familiar patterns. Other elements, which might be equally or even more significant, may be overlooked or dismissed as irrelevant. Even if more information is sought, it is likely to be information that confirms the initial hypothesis rather than information that opens up new possibilities: the physician does not even realize that anything has been left out. This reflects a failure of metacognition, “the willingness and ability to reflect on one’s own thinking processes and to critically examine one’s own assumptions, beliefs, and conclusions” (Berner and Graber 2008, p. S7). One of the marks of an expert is having “the ability to recognize when one’s initial impression is wrong and having back-up strategies readily available when the initial strategy does not work” (p. S9). If that is true, then insisting that one’s initial judgment must be right is the mark of a *non-expert*.

Berner and Graber go on to explore *overconfidence* as a contributing factor for diagnostic error: underestimating the degree and likelihood of error, believing that error rates only pertain to others, minimizing and dismissing one’s own errors when they do occur, and rationalizing that errors are inevitable. Overconfidence leads to complacency, self-deception, and an inflated sense of one’s own accuracy. This is fostered by inadequate feedback from patients combined with the use of patient outcome as a proxy for diagnostic acumen. If patients state that they feel better, get better regardless of whether the diagnosis was correct, or do not return with additional complaints, the physician may assume that the diagnosis was correct. “In the absence of information that the diagnosis is wrong, it is assumed to be correct” (p. S10). When the diagnosis is incorrect, the physician may never know.

Labels as Agents of Perception

Labeling theory, also called “societal reaction theory,” focuses on the linguistic tendency of majorities to negatively label minorities or those seen as “deviant,” and asserts that society’s *reaction* to certain behavior, more than the behavior itself, is a key factor in an individual’s defining himself as disordered. The theory has two roots, one in symbolic interaction theory, developed by George Mead in the 1930s, and the other in conflict theory, derived from the writings of Marx and Foucault. Symbolic interaction theory maintains that during socialization people learn and internalize the meanings and attitudes attached to certain behaviors; conflict theory focuses on how those in power determine what and who is labeled as deviant in order to maintain a social order that promotes their interests. Both are sociological theories concerned with the human behavior in social groups, in contrast to theories that focus on individual psychology.

Although others had written about deviance and labeling as a societal response to rule breaking, it was sociologist Thomas Scheff who offered the first complete articulation of the theory in his 1966 book *Being Mentally Ill*. Scheff proposed the radical idea that the *act of being labeled* by those in power creates the “disordered” status, not the behavior itself. His ideas had a wide appeal, fitting well with the anti-diagnosis and anti-authoritarian movement of the 1960s and 1970s.

In Scheff’s model, the effects of labeling are direct. Negative concepts of mental illness are independent of a person’s actual behavior, due instead to the label imposed on that behavior. Societal conceptions of mental illness lead to labeling, which produces responses that cause the labeled person to adopt the role of a “mentally ill person,” thus internalizing a definition of himself as disordered and forming an identity around that role that acquires master status. Behavior gradually conforms to the expectations and cultural stereotypes of the deviant role (such as being dangerous or incompetent), and it becomes increasingly difficult for the labelled person to inhabit any other role. As illness behavior becomes crystallized, the mental illness persona becomes self-fulfilling and appears to justify the label’s validity. In this way, mental illness is a social role, with societal reaction the determinant of entry into that role.

By the 1980s, labeling theory had come under attack. The proposed causal chain could not be demonstrated, making it difficult to test the validity of the theory to explain and predict. In addition, there was no way to measure concepts such as “identification with a deviant label.” Research tended to rely on proxy measures such as self-esteem, yet one can adopt a “deviant” identity with either high or low self-esteem. Critics asserted that the evidence for labeling effects was so overwhelmingly negative that the theory should be dismissed, simply a fad of the radical 1960s and 1970s.

Meanwhile, however, Bruce Link and his colleagues were conducting empirical studies and developing a new model for a *modified* labeling theory (1987, 1989) that proposed a more complex and indirect route between label and disorder. The act of labeling does not produce the mental disorder, they said, but leads to negative

outcomes that exacerbate and maintain disordered behavior. Socialization leads people to develop a set of beliefs about mental illness, with expectations that the mentally ill will be devalued and discriminated against. When a person enters treatment, these beliefs become personally relevant; he or she is now subject to a response from others based on notions of mental illness that are contained in and conveyed by the label. Believing that he himself will be devalued and discriminated against, the labeled individual avoids social contact that he fears will lead to judgment and rejection. Withdrawal and concealment, in turn, lead to further negative consequences—reduction in social support, access to employment, and self-esteem—causing additional isolation and demoralization. In a self-perpetuating cycle, stigma is experienced through others' responses and reinforced through internalization of a defective identity (Corrigan 2007).

Research also suggests that a label's effects may vary by social context and by the status of both the labeled and the labeler. When symptoms are more visible, disruptive, or severe, categorization is likely regardless of social status. But when symptoms are moderate or mild, those with more social power and resources may be able to resist being labeled or to substitute less pejorative terms like "eccentric millionaire." The more negative labels a person receives (homeless, delinquent, dropout, mentally ill, etc.), the more power and status are diminished. At a certain point, however, effects may level off and become redundant for who have received so many or such serious labels that they have already been deemed "non-members" of society.

While few people believe that labeling effects provide a complete explanation for mental disorder, it is clear that labels play a powerful role. The label defines the problem—alcoholic, unemployed, on probation, bipolar—and affects those being labeled as well as those who interact with them, turning people into "clients" and "clinicians." Resistance to being labeled can affect willingness to enter treatment, transformation of identity, and trajectories following treatment (Corrigan 2007). Consideration of the labeling process is thus an important part of assessment. That does not mean all labels should be avoided, but that the client's response to a proposed label—or to receiving *any* label—needs to be taken into account. For some, the label can feel like a loss of identity and efficacy. For others, the label may bring a sense of relief, legitimacy, and connection with others who have struggled in similar ways (McQuaide 1999; Probst 2013).

Difference, Distress, and Impairment

Definitions of mental disorder tend to include three elements—distance from "normal" experience, a sense of personal distress, and impairment in daily functioning—although with varying emphases. How "different" or "deviant" from prevailing social norms does a person need to be? How much does this difference have to trouble the person, leading to pain and suffering? How much impairment does it have to cause, in the person's own ability to live a satisfying life and in potential harm to others?

Some authors, concerned about the constructed nature of illness labels and the values on which they are based, prefer to sidestep the first question about degree of deviance and to focus on the experience of distress and dysfunction. One such attempt is Wakefield's notion of "harmful dysfunction." A hybrid approach, the "harmful dysfunction" hypothesis seeks to bridge the sociological notion of mental disorder as a social construction, primarily for the enforcement of social norms, and the biomedical notion of mental disorder as the failure or malfunctioning of part of the brain. The former, Wakefield says, is value-based while the latter is factual and thus value-free. Rather than taking sides, he attempts to include both. "Dysfunction," in his definition, refers to the failure of an internal biological mechanism to carry out the function for which it was designed; "harmful" means that this failure is or leads to conditions judged as negative by sociological standards, such as social impairment and/or psychological distress. "Dysfunction" is rooted in the evolutionary biology; "harmful" is rooted in the sociocultural context (Wakefield 2007).

Wakefield is attempting to answer the question of how we can know that something is "not merely a form of normal, albeit undesirable and painful human functioning, but indicative of psychiatric disorder" (Wakefield 2007, p. 149). Yet this definition has its own pitfalls. What exactly are these alleged functions and mechanisms of the mind, and how can we know if they are working "properly" or improperly (Jacobs 2014)? What would "proper functioning" look like, and who gets to decide? What about non-pathological eccentricity, such as the unusual but not necessarily disordered way that visual-spatial thinkers and other creative individuals think in associative spirals? The same question can be asked about "harm." Harmful to whom? Harmful, as compared to what? On whose authority is the consequence or experience deemed harmful?

In fact, neither distress nor impairment is necessary for mental disorder. Personality disorders, for instance, are by definition ego-syntonic and are not experienced as personally distressing. Other individuals, who may suffer deeply from chronic anxiety or depression, may nonetheless continue to function adequately in the world, either because they have to do so in order to survive or because these actions provide some relief from their pain. To say that they fail to meet the criteria for mental disorder, simply because they have managed to cope, would be to invalidate their suffering. The disordered condition thus needs to be decoupled with the disability that it might (or might not) lead to (Berganza et al. 2005).

Even when disability is present, that *still* cannot tell us for certain whether the condition is a true disorder of the brain and/or psyche or an adaptive response to challenging life circumstances. The problem of defining symptom or disorder has simply been replaced with the problem of defining function and harm, and we are back to the same question.

Perhaps there is another way to approach this dilemma. If we cannot decide what mental disorder *is*, perhaps we can decide what it *isn't*. By determining what lies *outside* its borders, we may have a better idea of what lies *inside*. Valid exclusionary criteria should help to distinguish a true mental disorder from other kinds of

human problems. Certainly, disorders that are fundamentally physical in nature ought to be excluded, even if they lead to emotional distress. So too, we ought to eliminate forms of suffering or dysfunction that represent a transient or adaptive response to the stresses of life, as well as what we might call non-pathological quirkiness. What else is left, once we have ruled these out? Surely *that* will indicate a trustworthy definition of mental disorder.

Unfortunately, however, the same difficulties arise when attempting to remove criteria presumed to be irrelevant—under the assumption that what remains is a valid core—as arise when attempting to isolate criteria presumed to be essential. Nearly anything can be relevant, depending on the person and situation. The problem remains.

Cataloging Disorder

While some theorists have concentrated on the question of distinguishing mental disorder *from* other kinds of difficulty, others have focused on ways to distinguish *among* the various forms of disorder—that is, on developing classification systems or taxonomies. Creating a good taxonomy is no small task. First, there is the matter of determining what constitutes a “category.” What is the defining feature? Where should lines be drawn, and how to handle cases that do not fit into any of the available categories? Is it better to have *more* categories, each with precise features, or *fewer* categories that indicate essential sub-types?

The classification system for mental disorder used in the US, as noted earlier, is the DSM, a system developed (and periodically changed) by the American Psychiatric Association. Its taxonomic principle is the grouping of psychiatric conditions into various families according to common features and “known similarities” based on “new scientific understanding of their principal features” and “advancements in our understanding of the underlying vulnerabilities as well as symptom characteristics of disorders” (American Psychiatric Association 2013). The goal of the diagnostic process is to determine which psychiatric category is the best match for a client’s presenting symptoms. The client might, of course, *also* belong to other categories if viewed against templates organized around criteria such as demographic variables, blood type, and political affiliation. Membership in these categories is not considered relevant for clinical diagnosis, which is presumed to rely on stand-alone criteria that cut across demographic and other lines. Many have questioned this assumption, however, citing evidence that the choice of diagnostic label is influenced by factors external to psychiatric criteria such as race, class, age, and gender.

Mental health classification systems are not inherent in nature, but created; they are devised, *revised*, and revised again according to historically and geographically embedded notions of what constitutes normalcy, deviance, well-being, and appropriate social functioning. DSM-5, for example, is organized quite differently from its predecessors. As the next chapter will indicate, there is no basis for

assuming that recent revisions of the system are more accurate than earlier ones. In general, the trend has been toward finer and finer distinctions, but “finer” does not necessarily mean “more accurate.” Distinguishing sub-types of disorder is only helpful if the differences are clinically important; otherwise, it is just a matter of semantics. The first system for classifying mental disorders, the 1840 census, used a single category of idiocy/insanity; a report compiled in 1888 listed seven types of mental illness (dementia, alcoholism, epilepsy, mania, melancholia, monomania, and paresis). The American Psychiatric Association’s 1917 manual included 22 diagnoses. The most recent edition of the DSM, published in 2013, lists more than 300 (see [Appendix B: Historical Overview of the DSM](#)).

When there are *too few* categories, important distinctions can be overlooked that affect treatment decisions, while *too many* categories can confuse and distract the clinician by diverting attention to minor points. What makes a classification system both practical and conceptually coherent is not the number of categories it proposes but how well it addresses questions of sensitivity and specificity.

Suppose, for instance, that a friend comes up to you and says, “Look at my new pet! Isn’t he adorable?” How can you tell if the pet is a cat or a dog?

The answer may seem obvious: How could anyone mistake a cat for a dog? But if you jot down the key features you would use to *describe* each animal, you may find that many of them do not actually *differentiate* the two species. A cat’s fur, four legs, whiskers, and tail are all significant to the cat and to anyone who seeks to understand the cat, but they have no significance when it comes to distinguishing a cat from a dog. Nor can you use behavior since not all cats climb trees and not all dogs like to swim. What, then, guides you so you know for certain that you have placed the pet in the right category?

That is what diagnostic *specificity* is about. When a feature is specific to membership in a particular category, it serves as a reliable sorting mechanism and helps to place someone in one category rather than another. Unfortunately, criteria for specificity are hard to come by in the world of human behavior and experience. The same feature can be interpreted in more than one way (ambiguity) or point to more than one category (overlap). Studies have shown, for example, that two-thirds of the children diagnosed with bipolar disorder also meet criteria for ADHD (imagine if two-thirds of your pets could be classified equally well as cats or dogs). Many people have features that seem to belong to one category and features that belong to another, or meet some but not all the criteria for a particular disorder.

The other important issue is *sensitivity*, or how accurately you can spot an instance of the category. Do you miss a lot of cases (under sensitivity)? Do you put people into the category who do not really belong (over sensitivity)? Without good sensitivity, prevalence figures don’t really mean anything. Sensitivity depends on *threshold*, the point where someone enters the category. Here again, it is not so easy since many disorders exist on a continuum. There are people with milder and more severe versions of the illness, even though they may receive the same label, and people who seem to fit into the category at some moments but not at others since they move along a continuum, depending on what is going on in their lives. If every symptom is equally important and it is a matter of having a sufficient number in

order to enter the category, then these “sub-threshold” people are excluded from a classification that might actually be appropriate. DSM-5 does include a way to indicate severity for many disorders; while helpful, these severity ratings tend to remain static once entered into a chart and rarely capture the dynamic nature of most people’s experience.

An intriguing alternative was proposed by Oldham and Morris in 1995, in their theory of personality types. They suggest that each psychiatric disorder represents a type of adaptive functioning and exists along a situationally embedded continuum. At times of low stress, a trait may manifest in a mild and functional way (for example, checking everything carefully, a behavior called “conscientiousness”), while at times of high stress, the same trait may become more intense and thus dysfunctional (checking everything so much that it interferes with your life, a behavior called “compulsiveness”). In the first instance, at the low end of the continuum, the behavior helps people cope with stress or uncertainty, providing a sense of order and control that allows them to function. At the high end, it becomes maladaptive. Same person, different situations—should the person receive a DSM label? Or should the label have a time limit, subject to renewal at intervals? That creates other difficulties, of course. People could claim mental illness when it suited them, mental health when it did not—who would determine when the person entered and left the category?

A good classification system allows people to make reliable and meaningful distinctions between categories. But distinguishing between categories is not the same thing as determining clinical significance. A rough tongue helps you to know that your pet is a cat rather than a dog (taxonomic significance); whiskers do not help to do that, yet are equally important to the animal itself, especially if they are not working properly (clinical significance). It is the same with features like loss of appetite or inability to concentrate: They may be clinically significant without necessarily helping to place someone in one group rather than another. The problem, for students, is when taxonomic and clinical significance are not taught in an integrated manner and they learn taxonomic specificity in one course, clinical sensitivity in another.

The merit of a taxonomic system rests on its sorting mechanism, the criteria used to define its categories. With a few exceptions such as Post Traumatic Stress Disorder and Reactive Attachment Disorder, the DSM uses behavior; the origin of the problem does not really matter, only the way you feel and behave. That is the rationale for elimination of the bereavement exclusion from DSM-5: in the past, depression due to bereavement did not qualify as “true” depression since it could be explained by an external event without requiring the presence of internal dysfunction.

The bereavement exclusion was, perhaps, a sensible way to avoid pathologizing human grief. It also helped to differentiate treatment for grief from treatment for other kinds of depression. With most forms of depression, the goal was to reduce the depressive thoughts, feelings, and behaviors; with grief work, feelings of loss and sadness need to be fully experienced and run their course. At the same time, the bereavement exclusion created other problems. If death of a loved one meant that a

person was not “truly” depressed, then what about depression due to loss of home or job, to serious illness, infidelity, or a host of other stressful events? Cause cannot matter in one case but not in others.

One could, of course, imagine an alternative classification based *entirely* on cause—problems stemming from impaired attachment, identity diffusion, shame, lack of agency, and so on—rather than on behavior. History would matter in that case, not observed behavior. In fact, clinicians consider both. They look at patterns and events over a client’s life course, current symptoms, and the contexts in which those symptoms occur—the details of a person’s life story and the meaning of those details to that specific individual (Phillips 2005). That is, they take an *idiographic* approach. The term “*idiographic*,” from the Greek word “*idios*” meaning “own” or “private,” refers to the unique, subjective, individual phenomena that comprise a person’s self-narrative (see Chap. 8).

Theorists and researchers, on the other hand, take a *nomothetic* approach. The word “nomothetic” comes from the Greek word “*nomos*,” meaning “law.” A nomothetic orientation is concerned with broad categories and characteristics of populations; it seeks to formulate laws or generalizations. In nomothetic inquiry, between-group differences are significant; in *idiographic* inquiry, within-group differences are just as important (Corrigan 2007). A taxonomic system such as the DSM uses a nomothetic approach to describe ideal, prototypical, or essentialist types.

The clinical encounter, in contrast, utilizes a two-step process. The initial act of diagnosis serves to map a person’s individual story and clinical presentation onto a general set of categories. Once the client has been diagnosed, the clinician moves in the opposite direction to particularize, qualify, and contextualize (Kirmayer 2005). Each approach has its emphasis and purpose; both are important for clinical understanding. Difficulty arises when categorical formulations are presumed to capture the information needed for individual assessment—when what is required for nomothetic inquiry is taken to represent what is required for idiographic practice (Phillips 2005). “The simplifying assumptions build into a model (or theory) that give the model normative value also distance it from actual descriptions of clinical reality” (Henry 2006, p. 193).

In real-world clinical practice, the diagnostic lens widens. Client problems are viewed in an “expanded explanatory context of vulnerabilities, life circumstances and stresses, personality dispositions, ways of thinking, social influences, and developmental pathways” (Haslam 2000, p. 1035). To this list, one might add cultural values and forms of expression, available outlets and supports, genetics, and overall physical health. Some disorders might have a stronger genetic component, others a stronger cognitive or cultural influence. Thus, the template for understanding the meaning and trajectory of a condition will not be the same for all diagnoses or individuals. As indicated in a recent study of clinical social workers, many consider the psychiatric paradigm to be important for more severe disorders but less so for milder ones (Probst 2012, 2013).

Conclusions

We return to the epistemological question of how we “know” someone has a mental disorder. Which kinds of knowledge are used to inform clinical decisions, how are they ranked or valued, and by what means was each kind of knowledge acquired? When we decide if someone has a legitimate reason to seek (and receive) help, who defines the terms, who benefits, and who is left out?

Discussion of mental disorder must include issues of power, access, and control. Despite the fact that most clinical services are provided by social workers, control—the way mental disorder is thought about, treated, and paid for—rests with the authors of the DSM. Defenders of the DSM have stated that its categories and criteria are merely “scientific hypothesis,” open to testing and modification if evidence is not found to support them. While that may sound reasonable, it is next to impossible for a researcher to obtain funding to test alternative criteria. DSM criteria themselves are not really “tested” either, or “re-tested” once they have appeared in print. There is no feedback system whereby clinicians can report that criteria were not accurate or helpful after all and can propose other criteria that might be considered.

It is a circular and troubling situation: We adopt certain criteria, knowing they are only hypotheses, yet alternative interpretations are never studied because there is no funding to test anything that is not already legitimized by inclusion in the DSM! Nor does research attend very much to issues outside the DSM taxonomy such as relational problems, identity conflict, and existential dilemmas. Even though these issues are important for clinical practice, affecting the trajectory of a disorder as well as the way an intervention will be understood and utilized (Kirmayer 2005), they tend to receive little scientific attention.

Social workers, committed to working with the whole person, need to look beyond the limited epistemology of the DSM—at other kinds of knowledge and other ways of accessing that knowledge. This may be especially important when working with clients whose cultural or spiritual backgrounds rely on different ways of conceptualizing illness, health, personhood, and help. Clinicians unused to examining and thinking critically about their own cognitive assumptions will not have the acuity or flexibility necessary for serving their clients. That is why a candid, thoughtful exploration of epistemology is essential for ethical practice.

Application to the Case of Ray

Subsequent chapters will offer various perspectives or ways to think about the Case of Ray (see [Appendix A: The Case of Ray](#) for a complete description of the case). Before considering these interpretations, however, it may be useful to *think about the way you think* by undertaking the following set of exercises:

First, take stock of your assumptions about the kinds of evidence that will be sought, found, and considered. Which forms of evidence do you presume are most trustworthy—Ray’s self-report, hospital records, clinician observation and intuition, practice wisdom, correspondence to DSM categories? This will provide insight into your epistemology. You might want to list the pertinent facts about the case and consider how you “know” each is true. Which facts seem most important? Why do you feel that way?

Next, consider your assumptions about the various groups that Ray belongs to. What assumptions do you have about police officers, 911 responders, Irish Catholics, victims of sexual abuse, men who admit to violence against women, people who have been hospitalized? That does not mean all your assumptions are false, only that they need to be illuminated. Listing them explicitly can help to mitigate the effects of priming and other cognitive errors.

Each of these categories is a label, evoking stereotypes and judgments. How do you think Ray felt about these labels? Which made him feel proud, ashamed, or uneasy because his inner sense of himself did not correspond to others’ perceptions about members of that group? Which labels did he choose, and which were given to him by others? Are there any contradictions among these labels?

Certainly, membership in a group like the police force implies power, while other groups that Ray belongs to imply powerlessness. How do these dissonant experiences affect his sense of identity and ego integrity? Which identities has he embraced and which has he denied?

Third, take note of Ray’s salient traits—for instance, his anger at perceived “disloyalty,” self-blame, and attempts to escape or avoid. Which of these are symptoms—that is, criteria for a DSM diagnosis? Remember that a symptom can be an observable behavior or a subjective complaint; internal experiences are not observable so we tend to use external signs, including verbal descriptions, as “evidence” of internal states. Clearly, Ray appears to have both: outer behavior such as violence, including violence toward his partner, and self-medication; he also reports subjective complaints including guilt, remorse, rage, loss, and feeling overwhelmed. Which diagnostic categories, if any, might these features point to?

Ray has, in fact, received two different diagnoses. The first was major depression, the second bipolar disorder II. Depression may have been selected because of the Tylenol overdose that led to hospitalization, his reports of ongoing difficulty sleeping, and the way he presented to hospital staff. We assume that they made the best diagnosis they could, given the information they had available at the time. After the second overdose, the diagnosis was changed, perhaps because the second depressive episode was seen as part of a cycle. Since Ray had been relatively stable in between the two episodes, the depression was not continuous; in the absence of a true manic episode, the diagnosis of Bipolar II probably seemed reasonable. It is important to remember that hospital staff had no knowledge of Ray’s prior trauma; thus, a diagnosis of PTSD was unlikely to have occurred to them.

Fourth, review your overall impressions and initial judgments. What was your first hunch about what is going on? What made you think so? What if you are wrong? What else could it be? Again, there may be merit in your initial formulation,

but the best way to guard against the effects of anchoring, confirmation bias, and satisfaction of search is to *keep looking*. Brainstorm as many possibilities as you can about the nature of Ray's core struggle, no matter how unlikely they seem, as well as their sources and implications. Share the case with a colleague to get another brainstormed list.

It can also be useful to examine the cognitive heuristics that others may have used in assessing Ray. What did hospital staff "anchor onto," based on the kinds of cases they may have seen before and on Ray's most dramatic symptom? Did they stop looking once a seemingly reasonable diagnosis had been made?

Finally, consider what you do not yet know about the case. What's missing? How might you find out? To which sources of knowledge are you most likely to turn?

Different people in Ray's life are likely to have different perceptions of him. What do you think would be a major difference in how Ray's father and Leslie, his ex-wife, would describe him? Which aspects might each highlight or ignore? What questions would you like to ask each of them? What might happen if you brought them into the room at the same time? What do you imagine each might say to Ray or ask him?

Practical Exercises

1. What are Ray's symptoms? Describe each one in terms of observable behavior. Are these symptoms severe and consistent enough to add up to a diagnosis? Why or why not? Do they represent a change from a previous level of functioning? If you had to give Ray a diagnosis, which one would you select? Why? What would be a disadvantage of selecting that diagnosis?
2. Think of client you have recently met for the first time, and review the intake form or your initial notes. What are some possible "anchors" that might have influenced the diagnosis given? How might you guard against or correct for the influence of these anchors?
3. Carlo, an 8-year-old boy, has just received a diagnosis of ADHD. What are some ways that this label might negatively affect him and acquire "master status"? What are some ways that it might be helpful to him and his family? What questions might you have about the meaning of his symptoms and his diagnosis? How would you be able to know the label's effects?

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