Editorial

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Clinical Psychological Science: Then and Now

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In case you were wondering (and in case you weren't), the title of this article possesses a double meaning: It refers to both the Association for Psychological Science (APS) journal *Clinical Psychological Science* (*CPS*), of which I am the new Editor, and to the field of clinical psychological science at large. In this editorial, I examine where our journal has been and where it is headed over the next several years, using past and ongoing developments in our field as context. Along the way, I will share my, at times, heterodox views of the field and explain where I see *CPS* fitting into the broader domain of clinical psychological science. In particular, I highlight *CPS*'s long-standing emphases as well as a few novel ones.

Scope and Content

Fortunately, founding and outgoing Editor Alan Kazdin, who has left me with huge shoes to fill, has made my job much easier by delineating the scope of *CPS* in an inaugural editorial published several years ago (Kazdin, 2014). Alan has already been remarkably generous in imparting his wisdom and insights to me, and I can only hope to maintain the extremely high standards to which *CPS* readers have become accustomed. I have little to add to Alan's superb list of the kinds of articles that *CPS* seeks, so I will instead merely refer readers and potential manuscript submitters to it (Kazdin, 2014). I urge authors to consult Alan's list prior to submitting manuscripts to *CPS*, as well as to the detailed submission guidelines on the APS website at www.psychologicalscience.org/cps.

For prospective authors who wish to cut to the chase and obtain the journal's elevator pitch, here is my take: *CPS* seeks to publish articles that bring to bear the best available basic science from any discipline to inform our understanding of psychopathology. As I will discuss later, what makes *CPS* distinctive from other psychopathology journals in psychology and psychiatry is its explicit focus on multiple dimensions of analysis, stretching from "neurons to neighborhoods" (see Shonkoff & Phillips, 2000) and well beyond. Hence, *CPS*'s purview is vast, as it is open to work drawn from a variety of subdisciplines within basic psychological science, including physiological, evolutionary, comparative, cognitive, developmental, social, vocational, personality, cross-cultural, and mathematical psychology, as well as from scientific disciplines that fall outside the traditional borders of psychological science, including genetics, neuroscience, economics, business, sociology, anthropology, microbiology, medicine, nursing, computer science, linguistics, and public health. Secondarily, *CPS*'s mission is translational, as we aim to bridge the often yawning gap between basic and applied science relevant to clinical problems.

In at least one key respect, however, *CPS* is more circumscribed in scope than several other excellent psychological journals dedicated to psychopathology research. In general, *CPS* shies away from manuscripts on descriptive psychopathology, not because such manuscripts are unimportant but because they can usually be comfortably accommodated within many extant journals. Hence, studies relying on case-control designs comparing patients with Disorder X and normal individuals on Variables Y and Z are unlikely to be considered at *CPS* unless the authors can make an especially persuasive case that their investigation provides distinctive insights into the correlates or etiology of the condition of interest. In one respect, *CPS* is also narrower in scope than several other superb journals devoted to psychotherapy research. With rare exceptions, *CPS* does not publish "horse race" treatment studies that compare one intervention against another or one intervention against a wait-list control condition; the primary exceptions are manuscripts that (a) examine especially innovative and promising treatments; (b) examine extant treatments that are scaled up to comprise extremely large samples, or that are delivered in novel formats or using novel modes of administration,

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thereby bearing important implications for public health; or (c) afford strong tests of potential mechanisms (and not merely mediators; see Kazdin & Nock, 2003) of treatment-based change.

Loyal *CPS* readers will recognize *CPS* 2.0 in almost all respects, although they may notice a few minor differences. Specifically, I have introduced two new features: Letters to the Editor and Book Reviews; please see the submission guidelines for instructions on submitting such manuscripts. I am also open to commentaries on and critiques of articles previously published in *CPS*, although submitting authors need to make a particularly compelling argument that their responses will advance the field in significant ways, either substantively or methodologically. In contrast, critiques of or comments on specific issues or analyses in published articles should almost always be submitted as Letters to the Editor.

New Directions

CPS will continue to focus on how basic science emanating from numerous domains informs our understanding of clinical problems. At the same time, I have decided to slightly expand the scope of *CPS* while retaining its overall mission. Here, I highlight two new emphases in particular: (a) conceptual, historical, and meta-scientific perspectives on clinical psychological science and (b) open science. In the following section, I highlight a third emphasis: (c) differing lenses of analysis. This third emphasis is by no means new to *CPS* (see Kazdin, 2014), but I feel compelled to say more about it. I address each of these emphases in turn, illustrating them with the aid of examples.

Philosophy of science, history of psychology, and meta-science

I am especially open to conceptual and historical manuscripts that provide thoughtful reflections on the past and present state of clinical psychological science, as well as thought-provoking inquiries of our disciplines' standard ways of doing business. I have long believed that as a field we do not spend nearly enough time engaged in self-reflection or self-scrutiny, and I hope to play a modest role in reversing this trend. I also welcome accessible and user-friendly articles on novel methodological and statistical approaches to enhancing the quality of research in clinical psychological science. In addition, I am seeking articles that propose novel approaches to dealing with issues of replicability, questionable research practices, and other challenges to the robustness of clinical psychological science. Such meta-scientific articles—namely, those that rely on the methodological and conceptual tools of science to improve science itself—should in the long run assist us with the goal of reducing error in our inferences, which is the engine that ultimately drives scientific progress (McFall & Treat, 1999).

By encouraging conceptual, historical, and meta-scientific articles in *CPS*, I also hope to combat the growing trend toward hyperspecialization and fragmentation in our field (see also Bevan, 1991; Sternberg, 2005). Many of us, myself included, read and publish in our specialty journals, attend our specialty conferences, and collaborate primarily with specialists in our principal research areas. Although a certain degree of specialization is necessary and to some extent healthy, it must be balanced against the often unappreciated need to maintain a bird's-eye view on past and ongoing themes in the field (that is one reason among many that I strongly encourage my graduate students to attend the annual APS convention). Excessive specialization can predispose us not only to intellectual narrowness but also to at least some "symptoms" of groupthink (Janis, 1962), whereby scholars in circumscribed subdomains tend to construe problems in similar ways and presume that their approaches are optimal. As Benjamin and Baker (2009) observed in an insightful essay, a historical perspective on psychology is often essential for forging connections across diverse theoretical viewpoints. A healthy appreciation of the history of clinical psychological science can also help us to avoid repeating the errors of earlier generations of scholars.

In all of these respects, my reasoning harkens back to a letter penned by none other than Albert Einstein (see Lewens, 2016). In 1944, a young American professor, Robert Thornton, wrote to Einstein to ask if he thought he should incorporate philosophy of science into his physics course at the University of Puerto Rico. Here is how Einstein responded:

> I fully agree with you about the significance and educational value of methodology as well as history and philosophy of science. So many people today—and even professional scientists—seem to me like somebody who has seen thousands of trees but has never seen a forest. A knowledge of the historic and philosophical background gives that kind of independence from prejudices of his generation from which most scientists are suffering. This independence created by philosophical insight is—in my opinion—the mark of distinction between a mere artisan or specialist and a real seeker after truth. (Einstein to Thornton, December 7, 1944, EA 61-574)

Scholars in clinical psychological science would do well to attend to Einstein's words of wisdom, which strike me as applying with at least equal force to psychology, including clinical psychology and allied domains, as to

physics. Indeed, I have increasingly come to suspect that many of the problems ailing our field stem from our all-too-frequent intellectual myopia and our neglect of the lessons imparted by a broad historical and philosophical perspective on basic psychological research relevant to clinical psychology.

For example, at the risk of engaging in hindsight bias, I have to wonder how much the current debates regarding replicability in psychology could have been foreseen had contemporary researchers heeded the prescient warnings of such scholars as Mischel (1968), Block (1977), and Epstein and O'Brien (1985), who pointed out the often erratic nature and questionable reliability of laboratory indicators and one-shot behavioral observations nearly half a century ago (see also Kenrick & Funder, 1988). At the very least, the classic writings of these authors, which seem to have been largely overlooked in recent debates concerning replicability, underscore the point that even minor and seemingly trivial changes in laboratory protocols and experimental contexts can yield markedly disparate results.

As a second example, perhaps one closer to home for most of us in clinical psychology, I suspect that clinical psychological science would be more appropriately skeptical of the perils of mono-etiological theories of mental disorders were it more cognizant of the unimpressive track record of such theories in clinical psychology and psychiatry (Kendler, 2005). The pages of our journals are replete with articles positing "the etiological model of disorder X"; I plead guilty to having contributed more than my share of such articles to the literature, too (e.g., Lilienfeld, 1992). To take merely one example, a knowledge of the decades-long history of failed attempts to discover necessary (and, in some cases, necessary and sufficient) causes of schizophrenia, such as parental double-bind communications, parental communication deviance, dopamine overactivity, glutamate overactivity, hypofrontality, attentional abnormalities, a single dominant gene, and scores of other putative etiological agents, should imbue us with a certain humility when it comes to positing models of specific etiology (Meehl, 1977) for other complex psychopathological phenotypes, such as psychopathic and borderline personality disorders (Lilienfeld, Smith, & Watts, in press). The lesson here is not that we should abandon the search for models of specific etiology, but that should we elect to pursue them, we should do so with a keen appreciation of their *a priori* low likelihood.

Open science

Following the lead of the APS journal *Psychological Science, CPS* is now formally embracing the core tenets of the movement toward open science, which underscore the importance of transparency and self-scrutiny in science. The last decade has taught us that many established psychological conclusions may be considerably less robust than initially believed and that a host of questionable research practices—many of which may often reflect largely unconscious biases (e.g., persuading oneself of the existence of consistent patterns in one's data despite their absence)—can substantially boost the risk of false-positive findings (Ioannidis, Munafo, Fusar-Poli, Nosek, & David, 2014; Lilienfeld & Waldman, in press). Nevertheless, clinical psychological science has been largely absent from the table with respect to conversations concerning replicability and questionable research practices, perhaps in part because our often difficult-to-recruit samples have made a culture of replicability less normative in our laboratories. We need to change that state of largely benign—and perhaps, in some cases, not so benign—neglect, and I again hope that *CPS* can play at least a modest role in doing so.

Specifically, authors of *CPS* manuscripts submitted on or after July 1, 2016, will be eligible for up to three badges for (a) open data, (b) open materials/measures, and (c) preregistration of hypotheses and analyses (see www.psychologicalscience.org/badges for a description of these badges and how to earn them). Because most or all of these practices remain controversial among some of our psychological science colleagues (e.g., Goldin-Meadow, 2016) and because clinical psychological science may pose special challenges for the implementation of some of these practices (e.g., the sensitive nature of the information we collect may make open-data requirements difficult to fulfill in some cases), *CPS* will not currently require any of these practices for manuscript acceptance. My hope, however, is that by encouraging the tenets of open science, we can gradually shift the norms of research in clinical psychological science toward greater transparency. Recent data strongly suggest that badge systems have been effective in nudging researchers toward providing open data and open materials in *Psychological Science* (Kidwell et al., 2016), and my hope is that we will eventually begin to witness similar changes in *CPS*.

But before continuing, a few words about preregistration are in order, especially given that this concept has spawned a host of misconceptions. To be clear, the movement to encourage preregistration does not require that all analyses be confirmatory. To the contrary, preregistration requires only that investigators be explicit at the outset about which analyses are confirmatory and which are exploratory. In this way, preregistration reduces the risk of hypothesizing after results are known, or HARKing (Kerr, 1998), a practice that I suspect is far more often unintentional than intentional (see Conway & Ross, 1984, for evidence that many of us tendentiously "rewrite" our

memories in light of present information). In the spirit of the writings of Nobel Prize–winning physicist Richard Feynman (1974), who underscored the role of science as a means of protecting ourselves from self-deception, preregistration diminishes the risk that scientists will fool themselves. As *Psychological Science* Editor Stephen Lindsay (2016) aptly observed with respect to the distinction between confirmatory and exploratory research,

> the young Jane Goodall might have preregistered something like "I plan to go to Gombe Stream National Park and unobtrusively observe chimpanzees, making notes of my subjective impressions of their behavior; I have no planned measures and no a priori predictions; I just hope to learn about chimps by observing them in their natural habitat." You might ask "What's the use of such a vague preregistration?" but it is valuable precisely because it helps the researcher to remember (and the reviewers and editors to know) that the researcher did not go in with specified measures and hypotheses.

In principle, *CPS* is open to articles that report largely or entirely exploratory analyses and findings, provided that (a) a strong case can be made that the results are scientifically important and (b) these exploratory results are accurately presented as unpredicted rather than as predicted.

CPS is also more than open to replications, both successful and unsuccessful, of articles previously published in our journal, provided that they are methodologically rigorous. Under my tenure as Editor, we will do our best to adhere to the "Pottery Barn rule" (Srivastava, 2012), whereby journals that publish an article will be obliged to publish methodologically rigorous direct replications of that study, regardless of whether the results are positive or negative. Admittedly, judging whether a replication is "direct" is even more challenging in clinical psychological science than in most other domains of psychology, including social and cognitive psychology, but I will leave that discussion for another day. I recognize that replication studies are rarely as "sexy" or as widely cited as are initial studies, but they are essential to the self-correcting nature of science (Lykken, 1968) as well as for clarifying the boundary conditions under which effects do and do not hold (Nosek & Lakens, 2014).

Multiple Lenses of Analysis: A Renewed Emphasis

As Kendler (2005) observed in a thoughtful disquisition that should be—forgive the hackneyed phrase, which is actually apropos in this case—required reading for all present and would-be clinical psychological scientists, an adequate understanding of psychopathology demands an appreciation of multiple levels of analysis (see also Schwartz, Lilienfeld, Meca, & Sauvigne, 2016). As he noted, if a person were to detect a bug in a statistical computer program (e.g., SPSS), such as an error in its factor analysis procedure, opening up the hard drive to tinker with the computer's circuitry would be worse than useless, because the problem would lie with its software, not its hardware.

Similarly, many clinical problems may in large measure be disorders of the brain's software (functioning) rather than of its hardware (structure; see McNally, 2011). For example, some specific phobias probably arise from an aversive classical conditioning experience followed by consistent avoidance of the feared event, although even here genetic predispositions almost surely provide diatheses to phobia acquisition in many cases (van Houtem et al., 2013). Hence, the increasingly fashionable position that psychological disorders are best conceptualized as neurological disorders (e.g., Insel & Quirion, 2005) is conceptually problematic. Although the distinction between brain "software" and "hardware" is admittedly oversimplified (Tryon, 2014), it is nonetheless a helpful metaphor for conceptualizing differing levels of analysis at which clinical problems are instantiated.

A key implication of this distinction is not that certain levels of analysis, such as the neuroscientific, are irrelevant to understanding clinical problems (cf. Tryon, 2014), but instead that certain levels may be more informative and pragmatically helpful for some clinical problems than for others. A further crucial point is that different levels of analysis may not be strictly reducible to one another (Lilienfeld, 2007). Even if they ultimately prove to be, a proposition that I am inclined to doubt, we are a long way from even a partial explanatory reduction across differing levels. Note, however, that a multilevel approach does not imply mind-body dualism, as it acknowledges that everything that is "mind" is ultimately the brain and the remainder of the central nervous system in action. At the same time, this approach holds that psychological levels of analysis cannot be fully explained by biological levels, or vice versa, as each level affords distinctive information in its own right (Kendler, 2005).

In the past, I have referred to these differing perspectives on clinical problems as "levels" of analysis (e.g., Lilienfeld, 2007; Satel & Lilienfeld, 2013), as I have done in the preceding three paragraphs. Nevertheless, critics have persuaded me that this terminology is potentially problematic, as it may imply a hierarchy, à la Comte (1830-1842), with ostensibly "harder" sciences, such as neuroscience, toward the bottom and ostensibly "softer" sciences, such as psychology, sociology, and anthropology, toward the top (see also Murphy, 2013). The implication of

this hierarchy is that the functioning of lower levels in some way constrains the functioning of higher levels, but not vice versa. As Gregory Miller (1996) noted, however, the implication that certain levels of analysis, such as the biological, necessarily "underlie" or "underpin" others, such as the psychological, is typically misleading, as it implies a causal primacy that is unwarranted. Indeed, basic research in psychological science, including epigenetics, reminds us that the linkages among these levels are typically bidirectional (Nigg, 2016). Hence, hereafter I adopt the phrase *differing lenses of analyses* to make explicit the point that each perspective affords a different glimpse of psychological reality but that no perspective is inherently more causally basic, let alone superior or more scientific, than any other.

In the section to follow, I provide examples of how clinical psychological science has erred in the past by simultaneously neglecting or insufficiently emphasizing one or more of three lenses of analyses (the biological, the psychological, and the cultural), at the expense of other lenses for certain clinical problems. Just as important, I impart the story of how psychological science engaged in healthy self-correction by according greater emphasis to a lens of analysis that was more appropriate to the question at hand. These narratives, which will surely be familiar to at least some readers, remind us that each lens of analysis can be optimally informative for certain clinical problems but less so for others.

The importance of recognizing the biological lens of analysis

Example 1: *Autism spectrum disorder.* I trust that most readers of *CPS* take for granted the assertion that autism spectrum disorder (once more succinctly termed *autism*) is a highly heritable condition with clear-cut neurological correlates (Abrahams & Geschwind, 2008). Nevertheless, it is all too easy to forget that as recently as a few decades ago, psychogenic theories of autism reigned supreme. The now notorious notion of "refrigerator mothers," which emerged in the early 1950s, most likely derived from Johns Hopkins University pediatrician Leo Kanner's (1949) observation that children with autism "were left neatly in refrigerators which did not defrost. Their withdrawal seems to be an act of turning away from such a situation to seek comfort in solitude" (p. 425). Although Kanner elsewhere acknowledged that the origins of autism are partly genetic, the refrigerator-mother concept took hold—and stuck for decades. As recently as 1980, I recall learning in an undergraduate course that autism was the product of distant and neglectful parenting. The instructor assigned with nary a hint of criticism Bruno Bettelheim's (1967) poignant but profoundly misleading book *The Empty Fortress: Infantile Autism and the Birth of the Self*, which argued unequivocally that autism stems entirely from severe early environmental deprivation akin to that experienced by concentration camp survivors (Bettelheim was himself such a survivor). In a sobering reminder that citation impact does not necessarily equate to scientific quality, this book has been cited 2,080 times as of this writing, according to *Google Scholar*.

The early prominence of psychogenic models of autism led scores of researchers down blind alleys and impeded scientific progress regarding the condition's biological etiology. Even worse, such models also had inestimable personal consequences. A generation of parents, especially mothers, were blamed by legions of therapists for causing autism in their children, leading to unwarranted guilt (Dolnick, 1998; Wing, 1997).

Fortunately, the bad old days of blaming parents for autism are now largely behind us, although perhaps not entirely. Even today, the legacy of psychogenic models of autism may live on, albeit indirectly, in the scientifically discredited but still widely used (Lilienfeld, Marshall, Todd, & Shane, 2014) technique of facilitated communication (now sometimes termed "supported typing"), which is premised on the erroneous idea that individuals with autism are cognitively normal people trapped inside of malfunctioning bodies. Proponents claim that by stabilizing previously uncommunicative individuals' hand and arm movements, the technique allows them to communicate with great eloquence via a keyboard or letter pad. Although facilitated communication is not based explicitly on the assumption of an early traumatic etiology for autism, numerous parents of individuals with autism have been the victims of unsubstantiated facilitated accusations of sexual abuse and other serious forms of maltreatment. Given that the technique is wholly ineffective (Schlosser et al., 2014), we now know that these accusations originated from the minds of facilitators, not those of individuals with autism. One suspects that many facilitators continue to cling to the outmoded idea that autism is in part caused by early psychological trauma.

Example 2: *Narcolepsy.* In 2013, with the publication of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association, 2013), the sleep-wake disorder of narcolepsy became the first (and still only) condition in the *DSM* to include an explicit biochemical marker—low hypocretin (orexin) levels, as verified by an assay of cerebrospinal fluid—among its diagnostic criteria. Psychologists and psychiatrists now recognize that narcolepsy is a substantially heritable condition that is largely of biological origin; they have also demonstrated that dogs with a specific hypocretin receptor mutation display narcoleptic features extremely similar to those of humans (Sakurai, 2013).

Yet it was not all that long ago that psychodynamic models of narcolepsy were widespread in the mental health community. Such models, which originated in part from the observation that the hallmark narcoleptic feature of cataplexy is commonly triggered by potent emotions, frequently conceptualized narcolepsy as the product of deeply repressed impulses springing to life. One author conceptualized the cataplectic episodes of a patient with narcolepsy as "regressive phenomena, the result possibly of the unconscious wish to return to an incestuous relationship with his sister during a somnolent state" (Coodley, 1948, p. 696). Another conjectured that the sleep paralysis episodes seen in narcolepsy reflect defenses against unconscious homosexual impulses (Schneck, 1948). Such views gradually receded in the wake of growing evidence of electroencephalographic abnormalities in patients with narcolepsy, but they continued to appear in the published literature as late as the mid-1970s. Needless to say, these unsubstantiated and needlessly stigmatizing theories only added to the immense emotional burdens already faced by patients with narcolepsy (Bladin, 2000).

The importance of recognizing the psychological lens of analysis

Example 1: *Panic disorder.* According to many, if not most, researchers, panic disorder is a condition marked by repeated "false alarms"—that is, dramatic fight-or-flight reactions triggered in the absence of objective threat (Barlow, 2004). Because panic disorder is a relatively "tight" (cohesive) syndrome, it may be one of the few *DSM* conditions for which a model of specific etiology is tenable (Lilienfeld, 2014). Over the years, several theorists have advanced etiological explanations for panic disorder that rely substantially on the biological lens of analysis. In the 1970s and 1980s, for example, some researchers suggested that mitral valve prolapse syndrome (MVPS), a largely benign heart condition commonly associated with palpitations and chest discomfort, was a promising biological marker, and perhaps even a specific causal factor, for panic disorder (see Dager, Cowley, & Dunner, 1987, for a discussion). Alternatively, the influential *suffocation alarm theory* of panic disorder (Klein, 1993) posited that the brains of patients with panic disorder are characterized by an overactive brain-based suffocation-signal detector, leading to erroneous alarms of imminent asphyxiation.

One difficulty with these and other etiological models is that panic attacks can be triggered by a panoply of substantially different and at times even opposing sensations, including abnormally slow or rapid heart rate, abnormally slow or rapid breathing, dizziness, and faintness, not to mention diverse cognitive symptoms, such as depersonalization, derealization, fears of dying, fears of losing control, and fears of becoming psychotic. I once treated a patient with panic disorder whose panic attacks were exclusive to reading. He would begin reading a passage, find himself distracted and having to reread it, discover that he was still not understanding it fully, try rereading it again, and eventually become convinced that he was losing his mind, a thought that then triggered his panic attacks. The enormous diversity of proximal triggers of attacks among patients with panic disorder is not readily accommodated within an explanation at the purely biological lens of analysis. Indeed, data suggest that, contrary to suffocation alarm theory, respiratory symptoms do not adequately distinguish patients with panic disorder from those with isolated panic attacks, whereas cognitive symptoms, such as fears of dying, do (e.g., Vickers & McNally, 2005).

Clark's (1986) cognitive model of panic disorder, which posits that the panic attacks of panic disorder stem from the catastrophic misinterpretation of unexpected bodily sensations and thoughts, instead affords a more compelling and parsimonious account of the causes of this condition. According to this model, virtually any unanticipated change in physiological or cognitive functioning can be misinterpreted by susceptible individuals as a harbinger of impending disaster, whether it be a heart attack, stroke, psychotic episode, or loss of control over one's impulses. This model helps to explain not only why patients with MVPS are susceptible to unexpected panic attacks, but also why patients with very different physiological symptoms—such as those arising from vestibular dysfunction (Jacob, Lilienfeld, Furman, Durrant, & Turner, 1989)—are prone to such attacks. In all of these cases, individuals experience largely or entirely unexpected changes in their physiological functioning that they misinterpret as signals of imminent calamity. In contrast to explanations posed at the purely biological lens of explanation, the cognitive model of panic disorder implies that individuals' construal of their symptoms is critical to unlocking the mystery of their pathology.

Example 2: *Alcohol use disorder.* The notion that alcohol use disorder (colloquially called *alcoholism*) and other addictive conditions are "brain diseases" (Volkow, Koob, & McClellan, 2016) has become so deeply entrenched in popular lingo and everyday culture that it has gone essentially unquestioned in many quarters (Satel & Lilienfeld, 2014). One challenge to evaluating the brain-disease model of alcoholism is that its core tenets are rarely clear. To be sure, the assertion that alcoholism is instantiated somewhere in neural tissue is a biological truism, as all behaviors, both pathological and nonpathological, are necessarily mediated by the central nervous system. Moreover, there is little question that there is a genetic predisposition to at least

some forms of alcoholism (Verhulst, Neale, & Kendler, 2015) and that chronic and severe alcohol use can damage brain tissue (e.g., Jacobus, & Tapert, 2013). Hence, through the prism of *one* lens of analysis, alcoholism is unquestionably a brain disease.

At the same time, it is not at all evident that this lens is usually the most profitable for understanding the causes of heavy drinking, and it is certainly not a sufficient one. Some classic variants of the brain-disease model (e.g., Jellinek, 1960) posit that endemic to alcoholism is the phenomenon of "loss of control." Exposure to alcohol, the theory goes, purportedly triggers a physiological cascade that results ineluctably in continued drinking ("one drink, one drunk"). Yet laboratory data provide scant support for this view. Seminal research on the balanced placebo design (Marlatt, Demming, & Reid, 1973; Rohsenow & Marlatt, 1981), which is rarely cited by advocates of the brain-disease model, has demonstrated that expectancies (which may in part reflect classical conditioning experiences) play critical roles in individuals' subjective and behavioral responses to alcohol. In the balanced placebo design, participants are assigned randomly to one of four cells; specifically, they are (a) administered alcohol and told that it is alcohol, (b) administered alcohol and told that it is a placebo, (c) administered a placebo and told that it is a placebo, or (d) administered a placebo and told that it is alcohol. Studies using this design demonstrate that expectancies typically carry the day: When individuals with alcoholism believe that the drink contained alcohol (regardless of whether it did), they tend to drink considerably more, as well as become more aggressive and less anxious, than when they do not believe that the drink contained alcohol (again, regardless of whether it did; see George, Gilmore, & Stappenbeck, 2012).

Although balanced placebo effects in turn appear to be moderated by other variables, such as the nature of the laboratory setting (McKay & Schare, 1999), these findings suggest that an adequate understanding of heavy drinking will not be possible without a consideration of the psychological lens of analysis. Yes, alcoholism is in part a brain disease, but it is every bit as much a motivational disease, an expectancy-based disease, a learning disease, a social disease, a cultural disease, and so on. Reducing it exclusively to the brain lens of explanation overlooks its enormous, multilayered complexity.

The importance of recognizing the cultural lens of analysis

Example 1: *Kayak angst.* A number of culture-bound syndromes almost certainly reflect variants of psychological conditions that we can readily recognize in Western culture (Lynn, Hallquist, Williams, Matthews, & Lilienfeld, 2007). A *pathogenic-pathoplastic* view of culture-bound conditions implies that the predispositions toward mental illnesses are similar or identical across cultures but that the manifestations of these predispositions are shaped by cultural variables (Kleinman, 1987; McNally, 1994). This model may not hold for all culture-bound conditions, but it is likely to be adequate to a first approximation for most of them (see also Keel & Klump, 2003, for an examination of cultural influences on eating disorders).

A striking example of this principle is the culture-bound condition of *kayak angst*, which has been extensively documented among Eskimo seal hunters in certain regions of Greenland (Amering & Katsching, 1990; Gussow, 1993). Sufferers commonly describe feeling frightened and dizzy while out at sea alone and are often seized out of the blue with overwhelming fears of drowning or of losing consciousness. Along with terror, they frequently report dizziness, confusion, sweating, trembling, hot and cold flashes, heart rate acceleration, and other symptoms. Upon returning to land, most kayak-angst sufferers are reluctant to return to hunting. In some cases, they say that their attacks are less frequent when they are accompanied by fellow kayakers. The description of the features of kayak angst leaves little doubt that it is a cross-culturally specific variant of agoraphobia, in many or most cases preceded by panic attacks.

A number of other culture-bound anxiety disorders probably follow a similar pathogenic-pathoplastic "recipe." Take *taijin kyofusho*, an anxiety disorder commonly reported in Japan in which individuals experience pronounced fears of offending others, often by their comments, appearance, or body odor (Kirmayer, 1991). The symptoms of taijin kyofusho correlate moderately to highly with those of social anxiety disorder (social phobia; Kleinknecht, Dinnel, Kleinknecht, Hiruma, & Harada, 1997). It is likely that the two conditions reflect a differential expression of social anxiety in Eastern as opposed to Western cultures (see also Zhu, Yao, Dere, Zhou, Yang, & Ryder, 2014) as a function of cultural differences in the premium placed on not displeasing others. It would be difficult, if not impossible, to comprehend these differences without the benefit of the cultural lens.

Example 2: *Dissociative identity disorder.* Many of us in Western societies probably think of culture-bound syndromes as restricted to non-Western societies. Perhaps because of our Eurocentrism, it rarely occurs to us that we may be inadvertently fostering certain pathologies in our own backyards—and perhaps exporting them to non-Western cultures along the way (Watters, 2010).

One potential example of this phenomenon is dissociative identity disorder (DID), formerly called multiple personality disorder. Although the etiology of DID is controversial, there is little dispute that prior to the early 1970s, diagnoses of the condition were exceedingly rare.

Back then, the number of reported worldwide cases was less than 100. Nevertheless, not long after the appearance of the best-selling book *Sybil* (Schreiber, 1973), which described a woman who purportedly developed 16 discrete personalities in the aftermath of horrific early sexual trauma, and the television release of the Emmy Award–winning film *Sybil* the following year, the number of DID cases skyrocketed.[1] Some scholars now estimate that DID is more prevalent than schizophrenia (see Lilienfeld & Lynn, 2014, a review).

Proponents of the DID diagnosis (Gleaves, 1996; Putnam & Lowenstein, 2000) maintain that the "new" DID cases that emerged post-Sybil were merely lying dormant for decades and had been overlooked by previous generations of clinicians. To them, modern-day clinicians are *discovering* new cases that previous generations had missed. In contrast, skeptics (Lilienfeld et al., 1999; Spanos, 1996) contend that many or most cases of DID are being inadvertently mass-produced by iatrogenic practices, such as repeated therapeutic prompting and cuing of "alter" personalities, as well as broader sociocultural and media expectations regarding the presumed features of DID. To them, contemporary clinicians are *creating* new cases that had not previously existed.

The debate here is exceedingly complex, and I do not intend to try to resolve it here. Nevertheless, it is worth noting that DID, whatever its etiology, appears to be shaped substantially by cultural variables. Until only a few decades ago, DID diagnoses were limited largely to North America, where the condition has received widespread media and cultural attention (Spanos, 1996). Even today, patients in the United States and Canada account for approximately 50% of published cases of DID (Boysen & VanBergen, 2013). Nevertheless, DID is now diagnosed with considerable frequency in some countries, such as the Netherlands, in which it has recently become more widely publicized (Lilienfeld & Lynn, 2014), as well as in countries that are becoming increasingly westernized, such as Japan, China, Taiwan, and Turkey (Dorahy et al., 2014). The behavioral manifestations of DID also appear to vary across cultures. For example, in India, the transition period as individuals shift between alter personalities is typically preceded by sleep, a distinctive clinical presentation that reflects widespread media portrayals of DID in this country (North, Ryall, Ricci, & Wetzel, 1993).

All of these intriguing findings raise the possibility, although they do not demonstrate, that DID is a largely culture-bound syndrome that originated in Western cultures but that has spread to many non-Western countries. Even if this hypothesis is incorrect, it is becoming increasingly evident that a full understanding of DID and its manifestations will necessitate a careful consideration of cultural norms and expectations regarding the enactment of multiple identities (Spanos, 1996).

Closing Thoughts

Different lenses of analyses afford distinctive glimpses into psychopathology, with some lenses being more valuable for certain clinical problems, and certain aspects of these problems, than others. I view one of the major goals of *CPS* as fostering this multi-lens (or what I once termed "multi-level") approach to psychological disorders, as a complete understanding of both mental illness and mental health is likely to come only with an adequate appreciation of approaches derived from a variety of perspectives, both inside and outside of the traditional borders of psychology.

Tempting as it may be in this era of increasing intellectual hyperspecialization, we must take pains to avoid the error of "fervent monism," the tendency to regard one approach to human behavior as inherently superior to all others for most questions (Kendler, 2014). Because tribalism is deeply ingrained in human nature (Wilson, 2012), we are all vulnerable to it, especially when it comes to defending our preferred theoretical turfs. But we must remain vigilant against it, as the propensity toward intellectual insularity hinders scientific progress. With the help of the terrific team of Associate Editors I have been fortunate enough to assemble—John Curtin (University of Wisconsin-Madison), Stefan Hofmann (Boston University), Kelly Klump (Michigan State University), Michael Pogue-Geile (University of Pittsburgh), Kenneth Sher (University of Missouri-Columbia), and Erin Tone (Georgia State University)—I very much hope that *CPS* can provide a partial antidote in this regard.

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Note

1. There is now clear evidence that many important details of the Sybil case, including Sybil's reported history of brutal sexual abuse at her mother's hands, were fabricated, and that her psychiatrist (who authored the best-selling book about her) coached her to display multiple personalities, which she often elicited with the aid of heavily sedating drugs (Nathan, 2011).

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