

the Behavior Therapist

special issue

The Biomedical Model of Psychological Problems

Co-Editors: Brett Deacon, University of Wollongong
Dean McKay, Fordham University

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PRESIDENT'S MESSAGE

The Biomedical Model: Caveat Emptor

Jonathan S. Abramowitz, University of North Carolina-Chapel Hill



THIS SPECIAL ISSUE of *tBT* on the biomedical model and treatment of mental illness covers thought-provoking topics for clinicians, researchers, and students alike. The biomedical model proposes that mental disorders are medical diseases caused by abnormalities in brain structure or function, or in genetics; and that treatment must therefore address such biological mechanisms. A heady atmosphere of excitement and anticipation presently surrounds the biomedical model, bolstered by the widespread belief that we are on the verge of discoveries in neuroscience that will transform our understanding and treatment of mental illness. On the one hand, there is a certain allure in the idea that mental illnesses are medical diseases like meningitis or diabetes. Yet the truth is that there is no credible scientific evidence for this assumption. No studies have convincingly shown that mental illnesses are caused by broken brain parts or bad genes. Yet there is evidence that promoting the biomedical model can be harmful to our patients. I'll get back to all of this; but first let me congratulate *tBT* editor Brett Deacon, who has amassed an unparalleled collection of writings from a diverse range of scholars to address this important and wide-ranging topic from various angles. I am pleased to have the opportunity to provide some opening remarks.

President George H. W. Bush proclaimed the 1990s as the "Decade of the Brain," and the

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INSTRUCTIONS for AUTHORS

The Association for Behavioral and Cognitive Therapies publishes *the Behavior Therapist* as a service to its membership. Eight issues are published annually. The purpose is to provide a vehicle for the rapid dissemination of news, recent advances, and innovative applications in behavior therapy.

- Feature articles that are approximately 16 double-spaced manuscript pages may be submitted.
- Brief articles, approximately 6 to 12 double-spaced manuscript pages, are preferred.
- Feature articles and brief articles should be accompanied by a 75- to 100- word abstract.
- Letters to the Editor may be used to respond to articles published in *the Behavior Therapist* or to voice a professional opinion. Letters should be limited to approximately 3 double-spaced manuscript pages.

Submissions must be accompanied by a Copyright Transfer Form (a form is printed on p. 35 of the February 2011 issue of *tBT*, or download a form from our website): *submissions will not be reviewed without a copyright transfer form*. Prior to publication authors will be asked to submit a final electronic version of their manuscript. Authors submitting materials to *tBT* do so with the understanding that the copyright of the published materials shall be assigned exclusively to ABCT. Electronic submissions are preferred and should be directed to the editor, Brett Deacon, Ph.D., at bdeacon@uow.edu.au. Please include the phrase *tBT submission* and the author's last name (e.g., *tBT Submission - Smith et al.*) in the subject line of your e-mail. Include the corresponding author's e-mail address on the cover page of the manuscript attachment. Please also include, as an attachment, the completed copyright transfer document.

years since then have seen numerous initiatives and advances in studying this most complex of human organs. To be sure, neuroscience aids in our knowledge of many areas of mental and behavioral health. Yet some have heralded this work as placing us on the cusp of confirming that the “underlying basis” or “fundamental etiology” of psychopathology is in the brain and our genes. Leading the charge is the National Institute of Mental Health (NIMH), whose Strategic Plan for Research states that “Fundamental to our mission is the proposition that mental illnesses are brain disorders expressed as complex cognitive, emotional, and social behavioral syndromes” (<http://www.nimh.nih.gov/about/strategic-planning-reports/introduction.shtml>). This statement goes well beyond the available scientific data (Satel & Lilienfeld, 2013). It assumes what remains to be proven. Yet this type of rhetoric is rampant in our field. Researchers refer to the “fundamental biological substrates of behavior” when they speak and write. Clinicians explain to patients how psychological disorders are caused by “underlying brain or neurotransmitter abnormalities.” Advertisements for psychotropic medications strongly reinforce this sentiment.

But even leading neuroscientists take issue with this type of biological reductionism—the idea that psychological concepts and experiences can be reduced to and explained by biology (e.g., Miller, 2010). Indeed, biological data may provide interesting information about psychological phenomena that cannot be obtained with self-report or behavioral measures; but these biological indices are not inherently more fundamental, accurate, or even more objective than psychological data. There is no shortage of causal arrow ambiguity. And it is furthermore a logical error to assume that the best way to understand and communicate about mental illness is by reducing it to abnormal biological functions.

Take anxiety. We mean so much more than neurotransmitters, brain structures, and neuron firings when we consider this uniquely psychological experience. We cannot directly observe anxiety in the brain—it must be inferred from verbal and overt behavior (cognitive biases, avoidance), and from diverse central and peripheral biological phenomena (e.g., Barlow, 2004). And whatever we know about biological phenomena in anxiety, we must still communicate about its psychological (i.e., cognitive and behavioral) aspects. Even if the amygdala is involved in anxiety and

fear, a description of activity in the amygdala cannot capture the experience of anxiety—although it might help us understand what is unfolding in the brain when one experiences anxiety.

Gregory A. Miller's (2010) analogy aptly elucidates this point: the best way to understand architecture is not to reduce it to the raw materials used to build the structures. Although the nature of the building materials puts constraints on the types of structures that can be built, it does not characterize the structure's design or function. Similarly, human emotion, cognition, and behavior (pathological or not) do not require a biological explanation. They are not reducible to neural firings, gene expression, neurotransmitter levels, or other biological processes—though they are certainly constrained by these factors.

These logical arguments aside, there is still no convincing scientific evidence for a mechanism by which abnormal brain functioning leads to psychopathology. In fact, very little is known more generally about how events in the brain drive psychological experience at all (or the converse). Moreover, unlike in medicine, there are no sensitive or specific biological or genetic markers or tests for any DSM disorder or psychological state (e.g., Deacon, 2013).

You might ask, “What about the fancy neuroimaging studies showing that the brains of people with disorders ‘light up’ more than the healthy peoples’ brains?” It is important to remember that such studies are correlational and one cannot draw causal inferences from correlations. While the differences in brain images might reveal a cause of psychopathology, it has long been known that the environment and our behavior also alter the brain (as well as alter gene expression; e.g., Baxter et al., 1992). Thus, it is equally plausible (and cannot be ruled out) that imaging studies merely reveal what is happening in the brain when one has a psychological condition. Moreover, one cannot reasonably infer the presence of brain abnormalities from correlational studies. The logical error is clear if you apply it to other areas of research; for example, can one infer that studies showing a correlation between anorexia nervosa and being female indicate that being female is an abnormality? Although some believe that studies exist wherein changes in biological variables, such as neurotransmitter levels, are related to changes in psychological experiences, the evidence for these being causal relationships is circumstantial (e.g., Kirsch, 2011). Furthermore, the precise mecha-

nisms of how such putative causation might work have yet to be convincingly explained (Miller, 2010).

Finally, many clinicians and mental health advocacy groups appeal to biomedical models as a rationale for negating blame and stigma. National antistigma campaigns have promoted the “disease like any other medical disease” meme in efforts to convince the public that mental disorders are medical illnesses for which sufferers do not deserve blame or discrimination. Yet the effects of this approach are neither what one might expect nor hope for. Research on public attitudes toward people with schizophrenia, for example, reveals that as acceptance of the biomedical model has increased in recent decades, so too has the desire for social distance from people with this condition (Schomerus et al., 2012). Other studies show that attempts to reduce blame by invoking biomedical explanations only reinforce (unfounded) concerns about the chronic and untreatable nature of mental disorders (Deacon & Baird, 2009; Read, Haslam, Sayce, & Davies, 2006) and the unpredictability and dangerousness of sufferers (Read et al., 2006). So, promoting the biomedical model to reduce stigma seems “at best ineffective and at worst potentially stigmatizing” (Pescosolido et al., 2010, p. 1327).

In conclusion, it is my view that while the focus on biological mechanisms and neuroscience has added to our understanding of human functioning, a dose of scientific humility is needed. Logically, biomedical explanations do not replace empirically supported psychological (e.g., cognitive-behavioral) models, nor their implications for psychological treatments. In fact, the existing data do not even support reductionist biomedical models. And although it might seem intuitive that casting mental illness as rooted in biology absolves the individual from blame and therefore reduces stigma, there has been no such decline in stigma after decades of promoting the biomedical model. The articles in this special issue are sure to be thought provoking and I hope your perspective on this matter is enhanced (whether you agree or disagree) by the material that appears on these pages.

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The Biomedical Model of Psychological Problems

Co-Editors: Brett Deacon, University of Wollongong
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Introduction

Brett Deacon, *University of Wollongong*

Dean McKay, *Fordham University*

THE BIOMEDICAL MODEL assumes psychological problems are brain diseases. Within this approach, research aims to identify the genetic and neurobiological causes and correlates of mental health problems, and treatment emphasizes biological interventions that target presumed neurobiological abnormalities. The biomedical approach has dominated science, practice, and policy in the United States for more than three decades and has profoundly affected mental health professionals, individuals with mental health problems, and society in general. Yet despite its popularity, the biomedical model is controversial. Critics contend this approach is based on flawed assumptions and that the available scientific evidence does not support its validity and utility. Recent events, particularly debate surrounding the DSM-5 revision process, have prompted widespread critical analysis of the biomedical paradigm. The purpose of this special issue of *the Behavior Therapist* is to contribute to this analysis.

This special issue features 11 articles that present critical analyses of different aspects of the biomedical model. Contributors to this special issue include award-winning scientists and journalists, three ABCT presidents, the president-elect of the British Psychological Society, and individuals from clinical psychology, counseling psychology, journalism, neuroscience, psychiatry, and social work. These authors share a commitment to scholarly rigor and scientific evidence as the foundation for critical analysis of the biomedical approach. The exceptional articles featured in this special issue deserve a careful reading, and their provocative conclusions warrant serious consideration and ongoing professional dialogue.

We hoped this special issue would include articles written by proponents of the biomedical model. Unfortunately, National Institute of Mental Health director Thomas Insel and National Institute on Drug Abuse director Nora Volkow declined our invitation to contribute an article describing their support for this approach. Jeffrey Lieberman, former president of the American Psychiatric Association, also declined to contribute an article to this special issue. Lieberman's invitation was prompted by his remarks on a Canadian Broadcasting Corporation radio show on April 26, 2015. Lieberman characterized one of our contributors, journalist and *Anatomy of an Epidemic* author Robert Whitaker, as a “menace to society.” Unfortunately, Lieberman declined to elaborate on why he believes Whitaker is “fomenting misinformation and misunderstanding about mental illness and the nature of treatment.”

On behalf of *the Behavior Therapist* and ABCT, we extend our sincere gratitude to the authors who contributed their time and expertise to this special issue. We are proud to present this outstanding collection of articles to the ABCT community and hope readers will find them useful in contributing to a more informed opinion on this important topic. We also hope this special issue will encourage open dialogue and critical analysis of the biomedical approach to psychological problems.

...

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Neurocentrism: Implications for Psychotherapy Practice and Research

Scott O. Lilienfeld, *Emory University*

Seth J. Schwartz and Alan Meca, *University of Miami*

Katheryn C. Sauvigné, *Georgia State University*

Sally Satel, *American Enterprise Institute*

IN 1989, SAMUEL GUZE, then one of the doyens of American psychiatry, laid down the gauntlet to his academic colleagues in a provocative article, entitled “Biological Psychiatry: Is There Any Other Kind?”, published in a prestigious medical journal. On the opening page, Guze answered his own question with a resounding “no”: “There can be no such thing as a psychiatry which is too biological” (Guze, 1989, p. 316). For Guze, the study of mental illness must focus squarely on the brain as the principle, if not the exclusive, level of explanation. Because all psychiatric conditions are ultimately instantiated in neural tissue, he insisted, they are all physiological disorders once one drills down to the most fun-

damental level of analysis—the brain. Hence, it is only at this level, Guze maintained, that research will ultimately bear fruit in understanding, treating, and preventing mental afflictions.

Over a quarter of a century later, we find ourselves confronting the same question raised by Guze, but with respect to psychology. We also find ourselves in an era of creeping neurocentrism. By neurocentrism, we mean the propensity of scholars to embrace the brain and remainder of the central nervous system (CNS) as inherently the most appropriate level of analysis for conceptualizing and treating psychological phenomena, including mental disorders (Satel & Lilienfeld, 2013; Schwartz, Lilien-

feld, Meca, & Sauvigné, in press). In its most extreme form, neurocentrism regards the CNS as essentially the only adequate level of analysis for conceptualizing and treating psychological phenomena.

The early 21st century is also awash in talk of psychological conditions as “brain disorders.” For example, in a 2013 TEDx talk, Thomas Insel, director of the National Institute of Mental Health (NIMH), argued that “what we need conceptually to make progress here is to rethink these disorders [mental disorders] as brain disorders” (Insel, 2013; see also Insel & Cuthbert, 2015).

But is neurocentrism helpful in clarifying our thinking about the causes and treatment of mental disorders? What are its implications for psychotherapy practice and research?

The Long Swing of the Pendulum

While an undergraduate at Cornell University during the late 1970s, the first author enrolled in a course on psychopathology. The professor, a clinical psychologist by training, confidently informed the class that infantile autism (today known as autism spectrum disorder; American Psychiatric Association, 2013)

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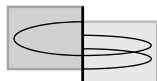
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was a disorder of purely environmental etiology. Autism, he assured us, is a consequence of inadequate or neglectful parenting. To buttress his point, he assigned Bruno Bettelheim's (1967) *The Empty Fortress*, an impassioned tome that identified "refrigerator mothers" as responsible for autism (this theory, originated by child psychiatrist Leo Kanner, 1943, has since been debunked).

This kind of thinking was hardly unusual at the time. As a number of commentators have observed, much of clinical psychology and psychiatry in that era could best be described as largely "brainless" (Eisenberg, 1986). Many mainstream authors conceptualized human nature as something akin to a "blank slate," often according scant consideration to the genetic or neurobiological context of behavior (see Lykken, 1991, Pinker, 2003, for discussions). A provocative book entitled *Not in Our Genes* (Lewontin, Rose, & Kamin, 1984), which argued forcefully against genetic and other biological influences on intelligence, schizophrenia, and behavioral phenotypes more generally, was widely read and taken seriously by scores of academic psychologists of a radical environmentalist bent. How times have changed.

As the pendulum has—thankfully—swung away from the often "brainless" psychology and psychiatry that were widespread only a few decades ago, a growing cadre of scholars, ourselves included, have expressed concerns that these disciplines now risk becoming "mindless" (Eisenberg, 2000; Lipowski, 1989; Satel & Lilienfeld, 2013). Because mental phenomena carry negative connotations in some domains of psychology, such as radical behaviorism (e.g., McDowell, 1991), we should be explicit about what we do and do not mean in this regard. First, by "mind," we do not imply a spooky, metaphysical essence that is either immaterial or materially independent from the brain. Instead, as we later delineate in more detail, we refer to a psychological level of analysis that differs from, but complements, the neural level. Second, by "mindless," we do not mean foolish or vacuous. Rather, we mean an undue neglect of what William James (1890, p. 1) regarded as the essence of psychology, namely, the "science of mental life" (see also Cacioppo & Tassinary, 1990). A mindless psychology, in our view, focuses so substantially on the neural level of analysis that it excludes or at least minimizes a host of other important levels of

analysis, such as the traditionally personological, social, and cultural levels.

Psychiatrist Kenneth Kendler (2014) similarly warned of "fervent monism" or the undue reliance on only one explanatory level, whether neural or psychological, for understanding human nature (see also Craddock, 2014, for a discussion of the need to accommodate both neuroscientific and social levels of analysis in psychiatry). Concerns regarding fervent monism were also expressed by a recent past president of the Association for Psychological Science, Nancy Eisenberg (2014), who lamented the "increasing tendency to assume that studying genetic/neural/physiological processes is more important than research on behavior and psychological processes per se because biological findings will eventually explain most of human psychological functioning" (p. 1). She noted that this trend is evident in "the funding priorities at some of the National Institutes of Health ... it can also be seen in the hiring patterns of many psychology departments that place a priority on hiring people who study biological processes or aspects of cognition that can be tied to neuroscience" (p. 1).

Evidence for the Ascendance of Neurocentrism

In a recent article, we (Schwartz et al., in press; see also Kagan, 2013; Miller, 2010, for similar arguments) laid out several lines of evidence suggesting that mainstream psychology is increasingly adopting a neurocentric approach to human nature. Among other things, we pointed to a dramatic recent upturn in the proportion of academic positions calling for expertise in neuroscience, many of which even mandate functional brain imaging skills; to the growing number of elite psychology departments (e.g., Indiana University, University of Colorado at Boulder) that have modified their names to emphasize neuroscience (e.g., "Department of Psychology and Brain Science"; see also Lilienfeld, 2012); to findings that, compared with journals in other medical areas, psychiatry journals are publishing a much higher percentage of articles devoted largely or entirely to biological correlates (Stone, Whitham, & Ghaemi, 2012); and to survey data we collected indicating that 27% of research psychologists reported "often," "always," or "almost always" feeling pressured to incorporate neuroscientific measures into their grant proposals.

We also addressed recent public statements by leading administrators at NIMH

and the National Institute on Drug Abuse (NIDA) that appear to signal a marked shift toward neurocentrism. For example, the draft of the NIMH's (2014) new Strategic Plan informs readers that this agency's major objectives comprise "defining the biological basis of complex behaviors" (p. 15), "describing the molecules, cells, and neural circuits associated with complex behaviors" (p. 17), and "mapping the connectomes for mental illness" (p. 18). As of this writing, the "Director's Page" for NIDA, which highlights the work of director Dr. Nora Volkow, states that "Dr. Volkow's work has been instrumental in demonstrating that drug addiction is a disease of the human brain" (<http://www.drugabuse.gov/about-nida/directors-page/biography-dr-nora-volkow>). Conspicuously, this web page provides visitors with no mention or even hint of research-based or conceptual criticisms of this view, which demonstrate that drug addiction, although genetically influenced in many cases, is often highly responsive to external incentives, classically conditioned cues, and other nonbiological environmental influences (Lewis, 2015; Satel & Lilienfeld, 2013).

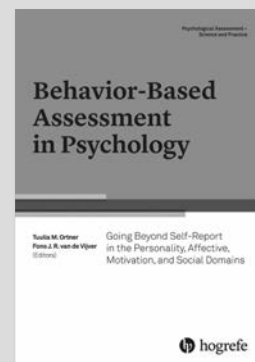
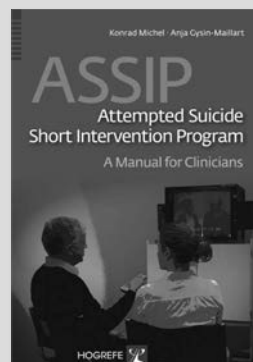
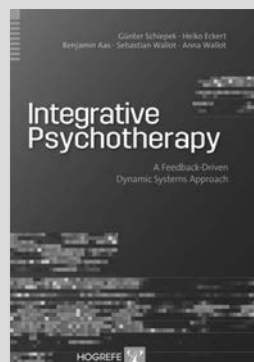
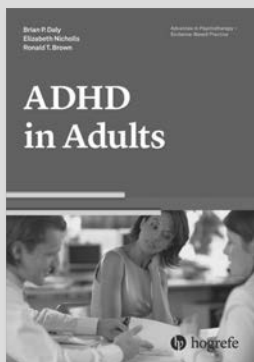
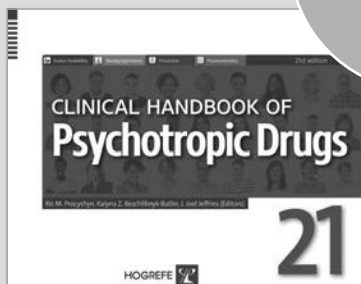
To fully appreciate the logical assumptions underpinning neurocentrism and its implications for psychotherapy practice and research, however, we first need to examine the oft-misunderstood concept of reductionism. It is to this thorny concept that we now turn.

Reductionism and Its Two Flavors

Many psychologists routinely decry "reductionism" as a scientific approach. But such criticism overlooks a key point: Reductionism is not one thing (Robinson, 1995). In particular, we must be careful to distinguish constitutive from eliminative reductionism (Ilardi & Feldman, 2001; Lilienfeld, 2007). Constitutive reductionism, which we wholeheartedly endorse, posits that the mind is what the CNS does, and that all psychological phenomena are ultimately traceable to neuronal activity. Constitutive reductionists reject "substance dualism," the dubious notion endorsed by Descartes (see Damasio, 2001) that mind and brain are composed of different material "stuff." At the same time, some constitutive reductionists, ourselves included, remain open to "property dualism," the proposition that mind and brain, although materially identical, differ in their level of analysis—much as Beethoven's 9th symphony can be conceptualized as a

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jumble of thousands of notes at one level and as a majestic subjective experience at another.

Eliminative reductionists, in contrast, go well beyond constitutive reductionists. According to them, scientists will eventually be able to dispense entirely with the psychological level of analysis, including such ostensibly “prescientific” concepts as “personality,” “thoughts,” “motives,” and “emotions” (Kihlstrom, 2010). Once the relation between brain and behavior is fully mapped out, eliminative reductionists predict that these and other psychological concepts will become superfluous, and that psychology will be reduced and relegated to a branch of biology. Philosopher Daniel Dennett (1993) termed this perspective “greedy reductionism” because it implies that the more basic levels of analyses (e.g., the neuronal), which are lower in Comte’s (1842) familiar pyramid of the sciences, will eventually “gobble up” the higher levels (e.g., the psychological).

Eliminative reductionism remains alive and well in many circles, even including some psychology departments. About a decade ago, a psychology department chair (who was a systems neuroscientist by specialization) was defending to one of us the hiring of a researcher whom many of his colleagues perceived as insufficiently interested in behavior. The chair gestured proudly to a book on his shelf by eminent neuroscientist Michael Gazzaniga (1998), entitled *The Mind’s Past*, and opened to the Preface, which proclaimed unequivocally that “psychology itself is dead . . . the odd thing is that everyone but its practitioners knows about the death of psychology” (p. 1). The department chair insisted that it only was a matter of time, and not much time at that, before psychologists would be regarded as expendable in departments of psychology. Some prominent neuroscientists are advocates of eliminative reductionism, either explicitly or implicitly. In his book *Neuronal Man*, Jean-Pierre Changeux (1997) wrote that “all forms of behavior mobilize distinct sets of nerve cells, and it is at their level that the final explanation of behavior must be sought” (p. 97). Similarly, physicist and popular writer Robert Park (2008) argued that “Psychology is becoming a ‘hard science,’ one that is last transforming the subjective study of human behavior into objective measurements of the physical entities that define us . . . we need to get inside the brain to see what’s actually happening among the billions of neurons, and simplify it to the most basic functions” (p. 198).

One pointed challenge to eliminative reductionism derives from theorizing on emergent properties: complex, higher-order phenomena that are not fully reducible to lower-order levels. Cognitive scientist Douglas Hofstadter (2007) offered a “traffic jam” as an example of an emergent property. The meaning of a traffic jam, he observed, cannot be extracted solely from its basic elements, such as cars, buses, cabs, and trucks. “You won’t locate a traffic jam,” Hofstadter observes, “if you restrict your search to the insides of a single taxi” (p. 787). To “find” a traffic jam, one must instead look to the higher-order interaction of its constituents, such as the number of cars on the road, the timing of traffic lights, the spacing between cars, the decision of drivers to change lanes at the last moment, slow driver reaction times, and so on. The whole is more than—and substantially different from—the sum of its parts (see also Marr, 1982). Although the existence of emergent properties is still actively debated among philosophers of mind, for the foreseeable future valuable information about behavior will almost always be lost when descending from higher to lower levels of analysis. Psychologist Jerome Kagan (2006) made the same point with regard to works of art. He noted that to appreciate an impressionistic painting, one must perceive more than just the sum of its parts. “As a viewer slowly approaches Claude Monet’s painting of the Seine at dawn there comes a moment when the scene dissolves into tiny patches of color.” When we adopt eliminative reductionism and focus solely on the lower-order elements of a painting, though, “the coherent psychological component vanishes” (p. 213).

Kenneth Kendler (2005) has advanced similar arguments, arguing forcefully for the importance of considering multiple levels of analysis in understanding psychopathology. Specifically, he contended that certain levels of analysis are more helpful than others for approaching different scientific questions (see also Cacioppo & Tassinary, 1990). For example, when developing and testing medications intended to target the amyloid plaques and neurofibrillary tangles of Alzheimer’s disease, the brain-based level of analysis will be the most helpful. In contrast, when attempting to understand the causes of racial prejudice and strategies to combat it, the psychological and cultural levels will be most relevant. In principle, of course, we may one day trace prejudice to the firing patterns of specific neurons in the brain. But in doing so, we would inevitably leave out crucial parts

of the story—most notably, the psychological meaning of prejudice to both its experiencer and its target.

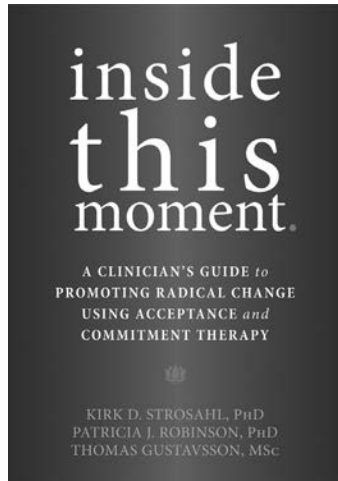
Neurocentrism: Implications for Psychological Treatment

Neurocentrism may offer us a one-dimensional view of human nature, but is it potentially harmful? We are inclined to think so. For one thing, controlled data suggest that although the framing of mental illnesses, such as schizophrenia and major depression, as brain diseases typically diminishes blame toward individuals with these illnesses, it heightens pessimism regarding prognosis and (probably) perceptions of dangerousness (Kvaale, Haslam, & Gottdiener, 2013). Although well-intentioned, the movement to reconceptualize mental disorders as brain diseases has at best mixed success in reducing stigma (Deacon, 2013). We further worry that neurocentrism has led some scholars, practitioners, and laypersons to assume that the brain is not merely the optimal level of analysis for understanding mental illness, but for treating and preventing it as well. In this way, neurocentricism may narrow the foci of potential intervention targets to the constituents of the CNS, such as neurotransmitters, neuromodulators, and receptors, often to the neglect of higher levels of analysis, such as psychological states—for example, attitudes, moods, motives, and thinking styles—that may be amenable to treatment.

This misapplication of neurocentrism may stem in part from *ex juvantibus* reasoning, a mouthful of a logical fallacy meaning “reasoning backward from what works” (Ross & Pam, 1995). It is tempting, but fallacious, to assume that if the causes of a mental disorder are in part biological, its proper treatment must also be biological, and vice-versa. But we should bear in mind the medical truism that headaches are not caused by a deficiency of aspirin in the brain. Nor do schizophrenia and vomiting share the same etiology even though both can be alleviated by means of medications, such as Compazine or Haldol, that block the binding action of the neurotransmitter dopamine in the brain. Inferring etiology from treatment, or treatment from etiology for that matter, is a tricky business.

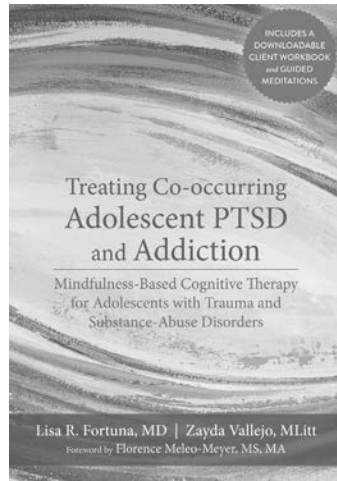
Just as important, the assumption that biomedical interventions are necessarily the optimal line of attack for psychological disorders has not stood up under empirical scrutiny. Despite the growing preeminence of neurocentrism in the public eye, psycho-

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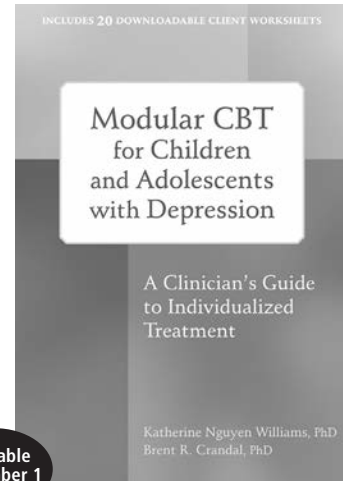
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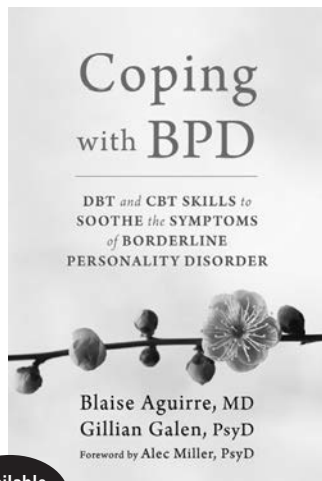
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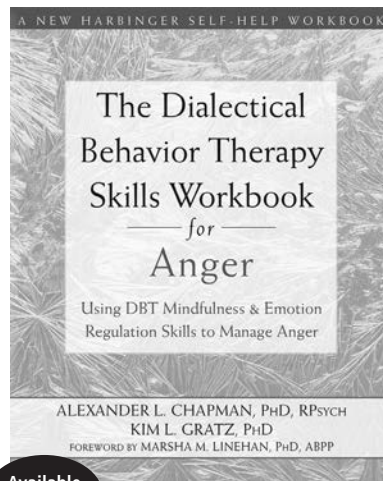
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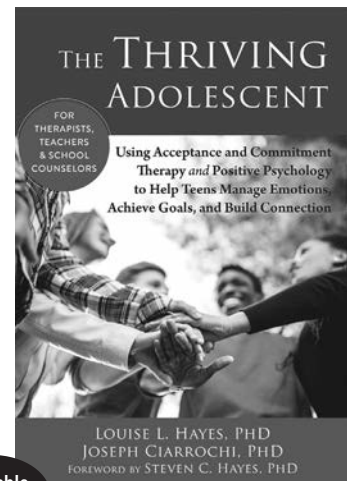
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logical and psychiatric researchers are busily working behind the scenes to develop effective psychological interventions for mental disorders, even those marked by a hefty genetic component. Although one would be hard-pressed to surmise it from the plethora of medication ads flooding our web pages and magazines, research increasingly demonstrates that cognitive-behavioral therapy (CBT), which focuses on modifying maladaptive thinking patterns and behaviors and imparting helpful skills to combat them, is at least as effective for treating major depression in the short run as is antidepressant medication. Furthermore, in several large-scale studies, CBT has emerged as more effective than medication for preventing recurrences of major depression (Butler, Chapman, Foreman, & Beck, 2006; but see Johnsen & Friberg, in press, for suggestions that the efficacy of CBT for depression is declining), probably because it provides individuals with enduring skills for warding off relapse. Similarly, the devastating signs and symptoms of schizophrenia, until recently believed to be resistant to psychosocial interventions, are now proving to be at least somewhat amenable to family and individual therapies designed to help patients manage everyday stressors (Jauhar et al., 2014).

Neurocentrism has also born witness to, and almost certainly fueled the popularity of, legions of novel—and dubious—brain-based psychotherapies of various stripes (see Cozzolino, 2002). Although these treatments differ in their specifics, all purport to draw on findings in basic neuroscience to inform psychological intervention. A selective sampling of some items from the growing menu of brain-based treatments includes the following:

- **Brain-based trauma therapy** (see Arden & Linford, 2008) is a broad approach that “synthesizes neuroscience, evidence-based treatment, psychotherapy research, and attachment theory into a hybrid therapeutic model” and accords “special attention to the neurodynamics of PTSD and the crucial role of memory” (http://www.aasw.asn.au/events/event/brain-based-trauma-therapy-integrating-neuroscience-and-psychotherapy_brisbane).

- **Neuropsychotherapy** (Grawe, 2007) advocates contend that “armed . . . with microscopic insight into the activity of a particular neural network involved with a client’s fear, as well as a macroscopic view of their interpersonal relationships and

environment, the neuropsychologist . . . [can obtain] a thorough grasp of the client’s situation” (<http://www.neuropsychologist.com/about/>).

- **Brain-spotting** (Grand, 2013) directs clients’ eye movements to specific positions that are purportedly linked to emotional trauma housed in specific brain regions, such as the amygdala and hippocampus. According to its proponents, “the maintenance of that eye position/Brainspot within the attentional focus on the body’s ‘felt sense’ of that issue or trauma stimulates a deep integrating and healing process within the brain. This processing . . . appears to take place at a reflexive or cellular level within the nervous system” (<https://brainspotting.pro/page/what-brainspotting>).

- **Brain Gym**, an educational technique in use in more than 80 countries, consists of 26 prescribed activities (most involving movement) that supposedly influence the activity of brain areas involved in learning and memory. For example, Brain Gym ostensibly claims to augment blood flow to the brain by massaging specific bodily regions (“brain buttons”), thereby boosting the acquisition of new information (Dennison & Dennison, 1989).

- **Neuropsychoanalysis**, although more of a research program than a school of therapy per se, aims to integrate Freudian therapeutic principles with cutting-edge developments in neuroscience (Panskepp & Solms, 2012; C. Schwartz, 2015), perhaps consistent with Freud’s (1895) view that psychoanalysis would ultimately be reduced to neuroscience. For example, some advocates of neuropsychoanalysis maintain that functional brain imaging data demonstrating the potency of limbic regions (e.g., amygdala, insula) in psychological processing helps to vindicate Freudian claims regarding the overriding influence of unconscious sexual and aggressive urges on behavior.

In all fairness, it is conceivable that some or all of these techniques may eventually prove to be efficacious, at least for certain clinical problems. Nevertheless, to our eyes, there are at least two serious difficulties with the marketing and dissemination of brain-based approaches. First, the claims associated with these methods go well beyond the available research evidence. Notably, none of the interventions described in the preceding bulleted list has been subjected to even a single published controlled trial, a salient caveat that one

would be hard pressed to glean from an inspection of their web sites and promotional materials. Second, these interventions are bedeviled by a vexing conceptual problem. Although it is plausible that basic neuroscience knowledge may one day inform the development and implementation of psychological treatments, not nearly enough is presently known about the linkages between such knowledge and psychopathology to effectively bridge the multiple levels of analysis that intervene between neurons and abnormal behavior (Schwartz et al., in press). As a consequence, it is not at all evident that basic brain science can tell us much about the design of psychotherapies that we do not already know. For example, although neuropsychoanalysis advocates are surely correct that emotional processing shapes our psychological make-up in powerful ways, functional brain imaging findings are not needed to achieve this age-old insight (Ramus, 2013).

Similar cautions regarding the overeager application of neuroscience are not new, and were sounded by B. F. Skinner (1955) decades ago. As described by O’Donohue (2013), “Skinner judged that [there] was too much of what he came to call ‘premature physiologizing’—that the zeitgeist of psychology of his time thought it was imperative that any discussion of perception and learning must be cased out in terms of the physiology of the nervous system” (p. 112).

The central problem with assertions regarding brain-based psychotherapies is not that they are necessarily incorrect. Instead, it is that these assertions are premature and almost always promise far more than they can currently deliver. As a consequence, mental health practitioners and consumers alike must be vigilant of “brain scams” (Beyerstein, 1990): glitzy but unsupported techniques that capitalize on the cachet of neuroscience to persuade the unwary that they are grounded in high-quality science.

Implications of Neurocentrism for Treatment Research

Neurocentrism also carries noteworthy implications for research on mental illness and its treatment. For example, neurocentrism can lead policymakers to funnel grant funding primarily or exclusively to projects that target the brain as the principal level of analysis for approaching the diagnosis, etiology, treatment, and prevention of psychological disorders. Indeed, over the past

decade, obtaining federal funding to examine the psychosocial correlates and causes of psychological maladjustment has become increasingly challenging (Schwartz et al., in press). In the case of substance addictions, the lion's share of grant funding has been channeled into the largely quixotic search for medications (e.g., vaccines, endogenous opiate antagonists) as opposed to psychosocial interventions, despite the more promising track record of efficacy of the latter (Lewis, 2015).

Another reason for caution concerning neurocentrism derives from the Research Domain and Criteria (RDoC) initiative recently launched by NIMH. RDoC aspires to develop a psychiatric classification system that can provide a viable alternative to those of both the *Diagnostic and Statistical Manual* (DSM) and the closely related International Classification of Diseases (ICD), which many scholars believe are rapidly approaching an asymptote in terms of scientific progress (Insel, 2009). Specifically, RDoC regards mental disorders as the products of dysfunctions in brain circuitry, and it delineates several promising psychobiological domains (e.g., positive valence systems, negative valence systems,

arousal systems) that may go awry in these conditions (Insel et al., 2010; Sanislow et al., 2010).

RDoC has much to recommend it, especially its loosening of the hegemony of the reigning DSM-ICD "paradigm" over psychopathology research. In this respect, it may offer a fresh transdiagnostic perspective on psychiatric classification that could eventually yield enhanced treatment utility.

At the same time, several scholars have voiced concerns that RDoC may push psychological and psychiatric research, including work on treatment and prevention, in an even more biological direction (Berenbaum, 2014; Lilienfeld, 2014). To be clear, RDoC is open to the inclusion of measures at multiple levels of analysis, including self-report, interview, and behavioral observations, and does not limit its scope to biological indices per se (Cuthbert, 2014). Nevertheless, it is worrisome that a number of prominent figures in psychiatry appear to view RDoC more narrowly than its original formulators. For example, in a comment in support of RDoC, John Scully, the American Psychiatric Association's chief officer, stated that "We want him [Thomas Insel, director of NIMH] to get biomarkers

for us" (Gever, 2013; see also Pine & Liebenluft, 2015). In addition, a recent past president of the American Psychiatric Association characterized RDoC as a blueprint for "the creation of a new diagnostic system based upon genetics, neurobiology, brain circuits, and biomarkers" (Lieberman & Ogas, 2015, p. 284). As RDoC moves forward in the coming years, NIMH must therefore ensure that the biological level of analysis is not privileged at the expense of other levels in our attempts to understand and treat mental problems. The search for biomarkers of psychopathology is valuable and should be encouraged, but it should not preclude research at alternative levels of analysis.

Parting Thoughts

Given that the history of clinical psychology and allied disciplines has long been characterized by radical pendulum swings between dogmatic sociotropy and dogmatic biotropy (see Meehl, 1990, for a discussion), some readers may justifiably wonder whether we are sounding an unjustified alarm call. After all, they might contend, it is probably only a matter of time



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before the pendulum swings away from neurocentrism, ideally equilibrating into a position in which social and biological levels of analysis are both valued.

Perhaps such readers are correct; we certainly hope so. At the same time, there are ample reasons for concern. Because faculty hiring, research, and grant funding are increasingly being directed toward neuroscientific approaches to psychopathology and away from competing approaches, there is a danger that psychosocial research on mental disorders and their treatment will not receive the attention that it deserves. As a consequence, we may be left with an impoverished picture of the causes and amelioration of psychopathology.

Furthermore, it is crucial that future generations of graduate students in clinical psychology and allied fields receive multidisciplinary training that bridges diverse levels of analysis, including the cellular, physiological, psychological, social, and cultural (Shoham et al., 2014). If anything has become clear in psychopathology research over the past decade, it is that the causes of most or all mental disorders are exceedingly multifactorial (Kendler, 2005). To make substantial inroads into the etiology and treatment of mental disorders, we will therefore need to draw upon and integrate insights from disparate disciplines, and to avoid the errors of simplistic sociotropy and biotropy that have so often impeded our field's scientific progress.

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Can Biomedical Models of Psychopathology Interfere With Cognitive-Behavioral Treatment Processes?

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THE DOMINANCE OF BIOMEDICAL models of psychiatric disorders is undoubtedly due to multiple factors. Modern research has, without question, revealed a wealth of biological factors implicated in the pathophysiology and maintenance of psychiatric problems. Grant funding has increasingly been directed at investigations of biological mechanisms and treatments, some from pharmaceutical companies and some through agencies such as the National

Institute of Mental Health (Deacon, 2013) or foundations such as the Brain and Behavior Foundation. Additionally, advocacy groups like the National Alliance on Mental Illness have emphasized to consumers and the general public alike that psychiatric conditions are akin to any other medical condition or disease. The present manuscript is not, however, concerned with the validity of biomedical models of psychiatric disorders. We instead wish to

focus on the individual, professional, and societal consequences of such conceptualizations.

Research has found that public adoption of the biomedical model has been on the increase (Pescosolido et al., 2010; Schnittker, 2006). Many might hope or believe this would lead to reduced stigmatization of psychiatric disorders (Pescosolido et al.), but evidence suggests the opposite has occurred (Kvaale, Gottdiener, & Haslam, 2013; Kvaale, Haslam, & Gottdiener, 2013; Read, 2007). Biomedical explanations of psychiatric disorders may cause laypeople to believe that those with psychiatric disorders are fundamentally different (Corrigan & Watson, 2004) and that these differences are nonmalleable (Lebowitz & Ahn, 2014). These associations may stem from “genetic essentialism” and “neuroessentialism,” which refer to views in which DNA or neurobiology, respectively, are seen as the immutable

“essences” of psychopathology and as fundamentally distinguishing people with and without mental disorders (Dar-Nimrod & Heine, 2011; Haslam, 2011).

Biomedical explanations are associated with decreases in levels of blame ascribed to those with psychiatric disorders, as well as reductions in the self-blame of those affected (Lebowitz, 2014). Importantly, though, biomedical explanations of psychiatric disorders may also create perspectives that reduce the affected person down to an automaton not in control of their actions (Haslam, 2006). This, in turn, may exacerbate the perception that affected individuals are dangerous (Kvaale, Gottdiener, et al., 2013; Kvaale, Haslam, et al., 2013), lead to a desire for social separation from people with mental disorders (Pescosolido, 2013), and reduce empathy (Lebowitz & Ahn, 2014). While these effects have been demonstrated on general perceptions of psychiatric populations, little is known about what effects biomedical explanations may have within psychiatric populations, particularly on treatment processes and outcomes. The extant literature does, however, indicate some potential effects, which we will now explore.

Potential Effects of Biomedical Models on Treatment Processes

As we have discussed, levels of stigma appear to be influenced by the manner in which psychiatric disorders are conceptualized. Beyond the effects biomedical models may have on general perceptions of people with psychiatric disorders, there may be effects within client populations as well. While the available evidence is sparse, some research has indicated that adoption of biomedical views results in reduced self-efficacy, reduced belief in the potential of psychotherapy to induce lasting change, and increased prognostic pessimism (Deacon & Lickel, 2009; Lebowitz, 2014). Essentially, biomedical views may exert effects through altering expectancies, which may have subsequent effects on treatment adherence and outcome.

Expectancies are important in that they greatly influence what people experience in response to a stimulus (Kirsch & Low, 2013). Expectancies may shape experience by influencing future emotional and mood states, as well as responses to both psychopharmacological and psychotherapeutic treatments (Greenberg, Constantino, & Bruce, 2006; Kirsch, 2005; Kirsch & Low, 2013; Westra, Dozois, & Marcus, 2007). Individuals entering treatment are likely to

have expectancies about the process of therapy, their own response to it, and ultimate outcome. Moreover, expectancies may influence behavior important within the context of psychotherapy. Experimental research has indicated, for instance, that common biomedical “chemical imbalance” conceptualizations not only generate beliefs that psychotherapy will be less effective, but also lower expectations of recovery in general (Deacon & Baird, 2009). This is significant, as lower expectations of success are predictive of poorer outcome; psychotherapy is less likely to work if the client does not think it will work (Newman & Stiles, 2006). Indeed, changes in expectancy may be a mechanism of change in cognitive-behavioral therapy (CBT; Kirsch, 2005; Newman & Fisher, 2010).

Reduced expectancy for change in psychotherapy engendered by biomedical views (Deacon & Baird, 2009; Iselin & Addis, 2003), as well as a reduced locus of control (Wiens & Walker, 2014), might reduce engagement in CBT. Diminished expectancies might negatively influence treatment-relevant decision-making (e.g., attendance, homework compliance, etc.) and ultimately limit symptom reductions. It is possible, however, that a person's expectancies may shift or somehow become less influential as CBT progresses (Wheaton, Pascucci, Foa, & Simpson, 2015), which could thus mitigate the potential effect of biomedical views on treatment engagement. On the other hand, expectancies tend to be self-fulfilling (Kirsch, 2005), and so lower baseline expectancy for change may have significant repercussions. Indeed, baseline expectancy for change has been shown to be positively associated with CBT outcome (Westra et al., 2007). As this effect was mediated by adherence to behavioral tasks, Westra and colleagues noted that higher baseline expectancy for change seems to facilitate treatment engagement—a notion supported by other research (Meyer et al., 2002). This is highly important, as client engagement within CBT robustly predicts outcome (Glenn et al., 2013; Meyer et al., 2002). Substantial gains can be made early in treatment, and early adherence to therapeutic tasks appears particularly important to CBT outcomes (De Araujo, Ito, & Marks, 1996). As such, biomedical views may exert the strongest effect on expectancy and outcome early in CBT by decreasing treatment adherence and interfering with early treatment gains. Additionally, a substantial proportion of dropouts often occur early in psychother-

apy (Simon & Ludman, 2010). Among some patients, such dropouts may be related to reduced expectancy for change fostered by clients' biomedical view of their condition. Similarly, Sullivan and colleagues (2003) suggested that biomedical views among psychiatric clients may be related to passive or fatalistic attitudes toward their condition. Such attitudes could be detrimental to CBT, given that CBT demands significant effort and engagement from clients in order to achieve optimal outcome (Glenn et al.).

Interestingly, the potential influence biomedical views may have on treatment processes has been suggested by research on obesity, another problem increasingly labeled a biological disease (American Medical Association, 2013). Similar to the case of psychiatric disorders, overweight and obese individuals who attribute their own weight status to biological causes tend to believe that their weight is unlikely to change (Pearl & Lebowitz, 2014). Research has also found that strongly emphasizing biological perspectives of obesity, with less emphasis on behavior and environment, can result in consumers engaging in counterproductive behaviors (such as consumption of higher-calorie foods), which worsen the presenting problem (Hoyt, Burnette, & Auster-Gussman, 2014). In a similar fashion, biomedical views of psychiatric disorders, with less emphasis on behavior and environment, may contribute to clients failing to make active efforts at behavioral change, or even contribute to them by engaging in behaviors that are directly counterproductive to clinical change.

In addition to the potential influences on clients' expectancies that we have explored above, the reductions in self-blame that occur among clients who subscribe to a biomedical model (Lebowitz, 2014) may also affect treatment-related behavior. While high levels of self-blame may result in more negative than positive effects, the reductions in self-blame associated with biomedical models may come at the cost of reducing some clients' perceived responsibility for behavioral change and improvement. This may have detrimental effects on a person's engagement with CBT, as perceived responsibility can help motivate a person to change their behavior (Delsignore, Carraro, Mathier, Znoj, & Schnyder, 2008). A sense of responsibility may be distressing for a person, but distress can also motivate beneficial behavioral change (Heinberg, Thompson, & Matzon, 2001). Importantly, a client's perceived responsibility entails a sense of agency over



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their condition, which may help engender positive expectancies, and which CBT relies upon. As such, clinicians might seek a balance in which clients feel a sense of responsibility—and attendant self-efficacy—to combat their symptoms, without feelings of blameworthiness and stigmatization that could interfere with motivation (Schvey, Puhl, & Brownell, 2011).

As we have explored, biomedical models may influence clients' expectancies for change and their engagement with treatment. An important clarification must be highlighted here. That is, the conceptualizations clients have of their symptoms may not be binary (i.e., biologically based or not), but may be more nuanced. In other words, to the extent that clients may see their condition as biologically rooted, they may see such biological factors as either amenable to change or as fixed and immutable. This distinction may be particularly important in shaping expectancies. Clients who hold a biomedical conceptualization of their psychiatric disorder yet view their biological makeup as malleable may have greater expectancies for change than those who perceive their biology as being largely nonmalleable (Lebowitz, Ahn, & Nolen-Hoeksema, 2013). The belief that a psychiatric condition is rooted in nonmalleable biological factors may explain research finding that biomedical views among clients were related to poorer response to psychopharmacological intervention (Sullivan et al., 2003). While research has not examined whether views related to the malleability of biology can affect treatment in the context of CBT, it is plausible there would be similar findings. Some research has found that general beliefs about the nonmalleability of symptoms predict attenuated CBT outcome (Valentiner, Jencius, Jarek, Gier-Lonsway, & McGrath, 2013), though the potential influence of biomedical views were not assessed. It remains an open question as to whether biomedical views, including more nuanced perspectives regarding the malleability of biology, affect CBT outcome.

Potential Effects of Biomedical Models on Therapeutic Alliance

The influence of biomedical models may extend beyond clients' expectancies and behaviors and into the therapeutic alliance itself. Biomedical views may influence the therapeutic alliance through the clinician or the client. For instance, research has found that biomedical views can influence clinicians by reducing empa-

thy toward clients and lowering clinicians' expectations that psychotherapy will be effective (Lebowitz & Ahn, 2014). This experimental study did find, however, that clinicians viewed a psychosocial perspective as more clinically useful than a biomedical one; the extent to which the results of this research apply to CBT clinicians in actual practice is unknown. Clinicians—especially cognitive-behaviorally oriented clinicians—may employ a biopsychosocial perspective in real-world practice despite widespread exposure to biomedical models. Additionally, when clients are considering whom to choose as their treatment provider, they appear to view biomedically oriented clinicians as less warm than clinicians who hold a psychosocial conceptualization of psychopathology (Lebowitz, Ahn, & Oltman, 2015). This, in turn, could negatively affect therapeutic alliances, especially among biomedically oriented providers.

Addressing Biomedical Views Within Clinical Practice

As we have explored, evidence suggests that biomedical models of psychiatric disorders may erode clients' expectancies for change, reduce efforts directed toward change, and influence the therapeutic alliance. While these possibilities remain to be empirically examined, in the interim, we believe some basic steps within clinical practice of CBT are reasonable and warranted in light of the evidence we have considered.

In clinical practice, it is routine for clinicians to present a treatment rationale to the client. For example, a cognitive-behavioral therapist may explain extinction learning, and the reciprocal relations between cognitions, emotions, and behaviors. A psychiatrist or other medical professional providing medication may explain the role of neurotransmission in brain functioning and psychological processes. In either case, the mental health professional may be influencing the client's expectancies. Aside from what a treatment provider may tell clients about their condition, clients likely enter treatment with pre-existing beliefs and attitudes. Clients are likely to carry at least some conceptualization of their condition, ranging from purely biological to purely psychological, with most clients' conceptualizations lying somewhere along this continuum. That being said, given the large and increasing number of Americans who subscribe to biomedical views (Schnittker, 2006), and

with causal ideas such as "chemical imbalance" permeating the common vernacular, it is probable that many clients enter treatment with a view biased toward the biomedical.

To a certain extent, clients may self-select which type of treatment they receive. Those who view psychotherapy as appropriate for their condition may seek psychotherapy, and those who view medication as appropriate may seek medication (Marcks et al., 2009). Thus, those who pursue psychotherapy may be less biomedically oriented in their views than others. However, there are many variables that determine the type of treatment a person pursues or receives. Furthermore, many CBT clients also take medication, and it is possible they view CBT as more of an adjunctive type of treatment that is merely aimed at providing coping skills as opposed to producing enduring change. Whatever the case, providing an understandable and comprehensive treatment rationale may help redress preexisting biases about the purposes of CBT.

A properly delivered treatment rationale can demonstrably enhance a person's expectancies (Ahmed & Westra, 2009). As mentioned above, conceptualizations of mental health conditions should not be binary (biologically based or not), but should be more nuanced. In other words, for improved outcome expectancy, research indicates that biological factors should be viewed as malleable to change rather than as immutable (Lebowitz & Ahn, 2015; Lebowitz et al., 2013). While this research found biomedical perspectives related to lower expectancy for change, an audiovisual presentation about the malleability of one's biology was found to improve expectancies; prognostic pessimism and hopelessness were reduced while self-efficacy was increased. Moreover, these effects were found to be both immediate and durable (Lebowitz & Ahn). Other research has found that psychoeducation emphasizing cognitive-behavioral factors and the malleability of biology resulted in greater expectancies for recovery (Farrell, Lee, & Deacon, in press). Overall, while the evidence is limited, there are indications that psychoeducation modules emphasizing the malleability of biology, together with conventional CBT principles, may foster greater expectancy for change; this may translate to improved outcomes.

Future Directions

Research examining whether clients' biomedical views about psychopathology affect the choice, process, and outcome of treatment is extremely limited (Lebowitz, 2014). At a minimum, research should explore how theoretical conceptualizations of psychopathology affect treatment choices. Future trials of CBT should also assess whether pretreatment biomedical symptom conceptualizations are related to differences in treatment processes and clinical outcomes. Another approach would be to use a pretest-posttest design and assess whether biomedical views shift within treatment and whether any shifts are related to treatment processes or clinical outcomes. A more sophisticated design could involve continually assessing biomedical and biopsychosocial views throughout the course of treatment, as well as various types of expectancies (e.g., self-efficacy, symptom malleability, perceived efficacy of treatment, and outcome expectancy), and assessing associations with treatment engagement, alliance, and primary symptom improvement(s). Assessments could also be administered to therapists, given the potential for biomedical views to affect a clinician's behavior (Lebowitz & Ahn, 2014).

Providing clear and concise biopsychosocial conceptualizations of psychopathology may provide a simple means for challenging absolute beliefs about biomedical bases of psychopathology and can shift treatment expectancies (Farrell et al., in press). It is yet to be determined if other methods can be used to change potential therapy-interfering cognitions, emotions, or behaviors that can stem from absolute beliefs about biomedical bases of psychopathology. Investigators should begin exploring whether or not traditional cognitive-behavioral strategies—such as behavioral experiments, cognitive restructuring, or even problem-solving skills—can be used to effectively challenge assumptions that psychopathology is biologically determined and immutable, which may thereby mitigate the negative consequences of such beliefs.

Conclusion

Evidence indicates that biomedical conceptualizations of psychiatric conditions may change clients' expectancies regarding their condition and treatment. It remains an open question as to whether this has demonstrable effects on behavior or on treatment processes and outcomes, partic-

ularly within the realm of CBT. We have argued that the biomedical framing of psychiatric disorders may significantly affect CBT processes and outcomes and have asserted the necessity of further empirical investigations focused on these possibilities. Given the increasing prominence of biomedical conceptualizations, further investigation is needed to improve understanding of potential consequences of said conceptualizations. With the complexity of the issues at hand, this area of research will require a large number of studies, across multiple labs and clinics, with a diversity of clinical populations. Biomedical conceptualizations of psychopathology are highly nuanced, with a multitude of treatment-relevant consequences cutting across multiple treatment modalities, and a dizzying number of candidate mediators and moderators of these outcomes of interest.

Until research determines whether biomedical views affect the course of CBT, given the extant evidence, it is reasonable for clinicians to take some simple steps within treatment. Some evidence indicates that clinicians can mitigate negative psychological effects of biomedical views by emphasizing the malleability of biological factors and the efficacy of CBT in shaping the brain. Such emphasis by clinicians may have significant influences on a client's expectancies and efforts at making positive and enduring change.

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ABCT

Misuse of Cognitive Neuropsychology in Psychiatry Research: The Intoxicating Appeal of Neo-Reductionism

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NEUROPSYCHOLOGICAL DIAGNOSTIC instruments were developed expressly for the detection of and assessment of neurological disorders (e.g., Lezak, Howieson, Bigler, & Tranel, 2012). The historic role of these instruments in locating damage in the central nervous system has diminished with the advent of various imaging technologies. Thus, the prominent role of neuropsychological assessment has been circumscribed predominantly to determining the extent of cognitive, emotional, and behavioral dysfunctions subsequent to brain damage. Nowadays, cognitive neuropsychology, the study of cognitive functioning, is being clinically utilized to demonstrate disability in educational, medical, and forensic settings, and increasingly utilized in psychiatry research. For example, a recent systematic review of neuropsychological investigations into obsessive-compulsive disorder (OCD) noted a fourfold increase in the number of peer-reviewed publications between the years 1990–2000, compared with 2000–2010 (Abramovitch, Dar, Mittelman, & Schweiger, 2013)

The age of the brain invigorated the biomedical model of psychological problems, and virtually transformed psychiatry to a new science that would be more appropriately titled “biological psychiatry” (Guze, 1989). To a considerable extent, this transformation essentially medicalized psychology as well, both in terms of its explanatory models and the search for remediation of psychopathology. Cerebral pathology has begun to take center stage as the primary focus in research on etiology of psychopathology, which is being now conceptualized as the expression of aberrations in brain functions, or “brain disease” (Deacon, 2013). The utilization of the best standardized objective behavioral approach to measure brain pathology, namely, neuropsychological testing, was the logical next step in the arsenal of identifying such diseases.

Although neuropsychological tests are sensitive to behavioral dysfunction, they

are inherently nonspecific. For example, a test sensitive to a decline in visual spatial skills cannot distinguish whether this decline is due to brain pathology, peripheral perceptual deficit, or motivational- and effort-related factors on the part of the examinee. As such, neuropsychological tests can speak to the relative difference from normative functioning, thus providing a very useful tool, but not one that speaks directly to the etiology of the deficit. Consequently, the potential for neuropsychological test results of one kind or another to serve as an endophenotypic factor or “cognitive marker” in psychopathology is extremely limited (Caspi et al., 2013). It’s no wonder, therefore, that despite years of well-funded research, not a single biological or cognitive marker, or a cluster of markers, have been identified that would predict a specific psychopathology. It is not even clear that a search for such etiological factors could yield theoretically useful fruits.

The ongoing discontent with the DSM classification system, together with the increasing appeal of neuroscience, and the trend toward a more reductionist, biologically based (preferably brain-related) approach to psychiatric disorders, engendered the NIMH RDoC initiative (Lilienfeld, 2014). The RDoC vision emerges from the assumption that “psychiatric disorders” are brain disorders and, as such, could be one day fully explained and treated by unraveling their underlying brain abnormalities. Indeed, the RDoC envisions a time when a patient would come into a clinic, have his/her brain scanned (and may undergo a saliva test or take a few brief cognitive tests), the results of which would indicate whether or not this person presents with the biomarkers that fit into one or another category of pathology (e.g., negative affect). Once identified, the subsequent treatment for such a disorder would be the appropriate biologically based agents, or neurotherapy such as deep brain stimulation (DBS). The work of the RDoC “Unit Work Groups”

produced a detailed matrix to be used as a guideline for researchers. This matrix includes specific domains for future research (e.g., positive Valence System, Cognitive Systems), specific constructs (e.g., Reward Learning, and Frustrative Non reward) to be examined using specific units of analysis (e.g., genes, neuronal circuits, molecules, or behavior).

Recent criticism regarding this vision has been leveled concerning different aspects of this approach, including the problems underlying the assumption that psychological conditions are brain disorders associated with a state of chemical imbalance (Lacasse & Leo, 2015), and the difficulties of a narrow, reductionist explanation of psychological entities (Satel & Lilienfeld, 2015) analogous to the attempt to explain “wetness” by referring exclusively to H₂O molecules. Criticism was also directed at the return to a modern version of phrenology (e.g., the relentless attempt to circumscribe psychological phenomena to highly specific brain regions or neuronal networks), sometimes referred to as neophrenology (Satel & Lilienfeld). However, in this article we focus on a specific domain within the RDoC initiative, namely, the domain of cognitive neuropsychology and neuropsychological tests in the context of psychiatry research. This domain, with its allure of objectivity, has been utilized in psychiatry research for a few decades. Recently it becomes increasingly evident that cognitive neuropsychology has been recruited to serve the premises of biological psychiatry, in a similar way to brain imaging.

Specificity

Research into cognitive function in the context of psychopathology aims primarily at identifying diagnostic markers, or to understand the involvement of cognitive functions in the etiology and presentation of psychiatric disorders. The reasoning behind this approach is that neuropsychological test performance reflects brain abnormalities. As such, this view fits nicely with the biomedical model of psychiatric disorders and its premise that psychiatric disorders reflect underlying brain pathologies. Consequently, neurocognitive assessment may be an objective and reliable tool to identify specific abnormalities and thus aid in the diagnosis of specific disorders and increase understanding of specific psychopathological processes. For example, attention-deficit/hyperactivity disorder (ADHD), a disorder characterized by

inattention, impulsivity, and hyperactivity, is associated with deficient response inhibition as measured by continuous performance tests (CPT), and Go-No/Go tests (GNG; Crosbie, Perusse, Barr, & Schachar, 2008). In particular, research indicates that individuals diagnosed with ADHD make more commission errors on these tests when compared with nonpsychiatric controls. This makes intuitive sense, given that these tests of response inhibition assess the ability to inhibit inappropriate responses, which has been traditionally linked to behavioral impulsivity (Logan, Schachar, & Tannock, 1997). Moreover, these results appear to make biomedical sense, and are in line with findings of reduced neuronal activity in the prefrontal cortex, a region associated with higher-order executive functions (Morein-Zamir et al., 2014). Indeed, the prevailing model of ADHD highlights response inhibition as the primary factor accounting for ADHD symptomatology (Barkley, 1997). In fact, this model has been widely accepted, to the extent that CPT and GNG tests are regularly employed around the world in the assessment of ADHD, particularly in educational settings.

This ideal picture, in which response inhibition has been considered a robust cognitive marker of ADHD, led some clinicians, and in particular for-profit clinics, to rely heavily upon the results of CPT and GNG tests to reaffirm, if not establish, a diagnosis of ADHD. However, examination of the data reported in research assessing response inhibition across psychiatric disorders reveals a very different picture. For example, in a comprehensive meta-analysis of GNG test performance across 11 DSM disorders, Wright and colleagues (Wright, Lipszyc, Dupuis, Thayaparajah, & Schachar, 2014) analyzed data from 318 studies and found moderate effect sizes reflecting underperformance on GNG tests in most disorders. The authors reported effect sizes for commission errors (the primary outcome measure for response inhibition) to range between $g = 0.2$ to 0.5 across ADHD, addiction, autism, bipolar disorder, depression, OCD, personality disorders, schizophrenia, and Tourette's syndrome. These findings are not limited to GNG tests, as the same research group reported very similar results in a meta-analysis assessing performance on another common response inhibition test, the Stop Signal Task (SST; Lipszyc & Schachar, 2010). Accordingly, deficit in response inhibition (as measured by neuropsychological tests) has been suggested as a cogni-

tive diagnostic marker and an endophenotype for different disorders such as OCD (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005), bipolar disorder (Bora, Yucel, & Pantelis, 2009), borderline personality disorder (McCloskey et al., 2009), schizophrenia (Turetsky et al., 2007), and ADHD (Slaats-Willems, Swaab-Barneveld, de Sonneville, van der Meulen, & Buitelaar, 2003).

Importantly, some of the foregoing disorders are associated with quite different clinical presentation and neurobiological models. As an illustration, consider the case of OCD, a disorder associated with inhibited temperament, hyper-control, and harm/risk avoidance, as well as with resting state *hyperactive* frontostriatal network (Pauls, Abramovitch, Rauch, & Geller, 2014). In contrast, ADHD is a disorder associated with prominent impulsive behavior, risk taking, hypo-control, and resting state frontostriatal *hypoactivation* (Castellanos & Tannock, 2002). Remarkably, response inhibition has been suggested as a diagnostic cognitive marker and endophenotype for both disorders. In other words, research suggests that underperformance on tests of response inhibition can predict the presence of OCD, but could also predict the presence of ADHD, as well as several other disorders. Taken together, research shows that underperformance on response inhibition tests may predict to some extent the presence of virtually any psychiatric disorder (and a number of neurological and other medical conditions), and thus has no value as a unique marker for any one of them (Caspi et al., 2013; Snyder, Miyake, & Hankin, 2015).

More broadly, it appears that the vast majority of DSM disorders are associated with underperformance on a plethora of cognitive tests, identifiable in most of the primary neuropsychological domains (i.e., executive functions, memory, attention, processing speed, and working memory). These findings have been consistently reported in meta-analytic reviews of neuropsychological test performance among samples of individuals diagnosed with depression (Snyder, 2013), schizophrenia (Fusar-Poli et al., 2012), bipolar depression (Bourne et al., 2013), OCD (Abramovitch, Abramowitz, & Mittelman, 2013; Abramovitch, Abramowitz, et al., 2015), antisocial personality disorder (Morgan & Lilienfeld, 2000), borderline personality disorder (Ruocco, 2005), eating disorders (Van den Eynde et al., 2011), PTSD (Scott et al., 2015), and ADHD (Schoeclin &

Engel, 2005). This lack of specificity may indicate that underperformance on neuropsychological tests, assessing virtually any neuropsychological domain, could be associated with any psychopathology. Indeed, in their seminal work, Caspi and colleagues (2013) examined what they termed "the p factor"—a single factor signifying psychopathology—which, as a single-factor model, was found to fare better, compared with a three-factor model (i.e., internalizing, externalizing and thought disorder). In their examination of data from more than 1,000 individuals, they provide evidence that cognitive functions showed weak or no correlations with all three factors. The authors concluded that the p factor is associated with small to moderate degree of cognitive problems in the major neuropsychological domains, such as attention, mental control, working memory, visuospatial functions and visuo-motor coordination. The authors concluded, "researchers should not expect to routinely find single-disorder loyalty in biomarkers (e.g., neuroimaging findings, cognitive task performance, and hypothalamic-pituitary-adrenal axis hormones), consequences (e.g., suicide attempts and impaired relationships), treatments (e.g., psychotherapy and pharmacotherapy), or causes (e.g., maltreatment and genes)" (Caspi et al., 2013, p. 134).

Impairment

The findings described heretofore challenge the utility of objective neuropsychological tests as disorder-specific markers. Moreover, they lead to a series of equally important questions concerning the definition of the magnitude of underperformance on these tests, namely, what is a cognitive/neuropsychological impairment? What is the operationalization and statistical definition of a neuropsychological deficit or impairment? Do these definitions require the presence of functional impairments outside the realm of neuropsychological tests? Finally, what are the clinical correlates of such impairments?

It is a common practice for neuropsychological studies of psychiatric disorders to conceptualize statistically significant lower test scores as a deficit or impairment. This common practice usually disregards the magnitude of the difference (i.e., effect size), or the clinical sample's standardized score compared with tests' norms. Reviewing the classic neuropsychological literature, a neuropsychological impairment is usually defined as a difference of 2 or 3

standard deviations from the population norm (equivalent to Cohen's *d* effect size of 2.0-3.0; Lezak et al., 2012; Zakzanis, Leach, & Kaplan, 1998). For example, research findings indicate that the cutoff for detecting cognitive decline predicting the conversion from mild cognitive impairment to Alzheimer's disease is at least 1.5 standard deviation difference (García-Herranz, Díaz-Mardomingo, & Peraita, 2015). In fact, one of the more liberal definitions of cognitive impairment on the Wechsler memory and intelligence scales has been suggested to be 1.0 *SD* (Taylor & Heaton, 2001). As noted above, review of neuropsychological meta-analyses across major DSM disorders reveals that most disorders are associated with widespread underperformance on neuropsychological tests that is usually less than 0.5 *SD* below the normative average/control group, and rarely reaches an effect size equivalent to 1.0 *SD*.

Perhaps a more important question would be: What are the clinical, behavioral, and phenomenological correlates of "neuropsychological impairments"? With a vast literature repeatedly concluding that disorders are associated with impairments in verbal memory functions, for example, or deficits in executive function, such as response inhibition—what would be the clinical expression of such a deficit in real life? Finally, assuming an association between functional impairments and neuropsychological impairment exists, is it disorder-specific?

Indeed, the association between neuropsychological test performance and functional indices across some disorders has been subject to empirical investigations. Naturally, the majority of these studies focus on disorders where cognitive deficits are a prominent part of the clinical presentation of these disorders (e.g., schizophrenia, ADHD, Alzheimer's disease, depression), neglecting other conditions such as OCD and anxiety disorders. Despite the prominence of neuropsychological symptoms, these studies are characterized by inconsistent results and most often by a low range of explained variance. Moreover, some studies find that the association between neuropsychological test performance and functional indices may be more clearly observed among control samples or large representative cohorts from the general populations (Miller & Hinshaw, 2010; Sayal, Washbrook, & Propper, 2015).

Importantly, some studies have found that participants' self-report concerning cognitive problems hold a much stronger

predictive power for functional indices than actual cognitive tests; the latter, although considered objective, were insignificant as predictors (Barkley & Fischer, 2011; Barkley & Murphy, 2010). Research suggests that cognitive functions in disorders such as schizophrenia predict social functioning, activities of daily living, and general real-life problem solving (Revheim et al., 2006). It has been further suggested that such findings may help in identifying individuals with more severe cognitive impairments in order to tailor more intensive rehabilitation programs for these disorders (for a review see Green, Kern, Braff, & Mintz, 2000). This type of (prevalent) logical inference may be appealing, perhaps even intuitive. However, this type of inference assumes a causal relationship that has yet to be proven: namely, that neuropsychological dysfunction causes such functional impairments. Such an assumption ignores the alternative converse inference, that the symptoms of schizophrenia (or any other disorder, for that matter) may produce neuropsychological problems. Thus, it remains to be ascertained whether inattention predicts social functioning, or that individuals diagnosed with schizophrenia tend to be very associative in conversations, for example, or may use blunt language, both of the latter resulting in impaired social functioning.

Causality

First-year students in psychology learn that correlation does not imply causation. Presently, the question can be stated as follows: Is an underlying brain dysfunction, expressed as underperformance on a neuropsychological test, the cause of a particular psychopathology, or its correlate? It seems quite clear that if a neuropsychological symptom appears in a variety of disorders, it cannot be a specific sign of any one of them. That is, it may be a necessary but not sufficient sign of the disorder. For example, response inhibition cannot uniquely signify the presence of OCD, since it is just as likely to be present in ADHD, the latter presenting behaviorally with quite the opposite symptoms as the former. From our review of the relevant literature, it is quite obvious that most, if not all, neuropsychological signs appear in various combinations in different psychiatric disorders. As such, they may constitute signs of psychopathology in general (and possibly also of neurologic disease, brain injury, endocrine dysfunction, and a host of medical problems), but they lack

the necessary specificity to serve as the direct cause of any. That is not to say, of course, that neuropsychological signs may, theoretically at least, reflect a network of symptoms indicative of some underlying brain pathology. However, serving as a cognitive marker requires that a sign should possess the specificity which neuropsychological indicators are lacking.

One consequence of the foregoing systematic and pervasive inferential error is that the recently expressed hope of treating neuropsychological deficits as a means of treating specific psychiatric disorders (e.g., Vandborg, Hartmann, Bennedsen, Pedersen, & Thomsen, 2015) is bound to crash onto the rock of reality. In other words, given that experiencing symptoms of post-traumatic stress disorder, ADHD, depression, schizophrenia, OCD, and so forth, result in underperformance on neuropsychological tests, does not necessarily imply that practicing cognitive skills would alleviate these different symptoms. In fact, research indicates that cognitive training hardly improves the corresponding cognitive functions, let alone generalizes improvement outside the specific targeted cognitive function (Melby-Lervag & Hulme, 2013). Similarly, the search for the underlying sign of psychiatric pathology using brain imaging may be ill conceived. Whereas the clinical and diagnostic utility of imaging research in the context of psychopathology has been extensively criticized (Satel & Lilienfeld, 2015), the use (or, in some cases, even abuse) of cognitive neuropsychology of psychiatric disorders received very little critical scrutiny. One possible reason could be the traditional role of neuropsychological assessment in providing objective information regarding deficient cognitive functions that was used to inform physicians as to the localization of damaged brain regions. A second reason is the appeal of objective tangible data such as response speed, number of errors, number of words remembered correctly, number of categories achieved, etc.

The State of the Field

Hundreds of papers and dozens of meta-analytic reviews indicate repeatedly that various psychiatric disorders are associated with underperformance on neuropsychological tests (Abramovitch & Cooperman, 2015; Abramovitch, Mittelman, Tankersley, Abramowitz, & Schweiger, 2015; Ahmari, Eich, Cebenoian, Smith, & Blair Simpson, 2014; Caspi et al., 2013; Shin et al., 2010; Snyder

et al., 2015). This is irrespective of the known differences among these conditions in terms of their psychological mechanisms, neurobiological/neurochemical models, and pharmacological and psychological interventions. Only recently did some investigators note that this variability and lack of specificity poses a major problem in the context of the search for cognitive diagnostic markers. In fact, it has recently been articulated that cross-sectional studies comparing clinical and control samples on traditional neuropsychological batteries are no longer required as these do not provide new insight (Abramovitch & Cooperman; Snyder et al.).

The aforementioned difficulties did not hinder the recent development of a freely available neuropsychological battery by the NIMH (i.e., the NIH Toolbox) that comprises the same traditional neuropsychological tests. Moreover, in contrast to the known lack of specificity, it appears that the RDoC initiative in effect brought about prioritization of funding for studies that are set to identify cognitive markers using traditional neuropsychological tests. Proponents of the RDoC vision may justify this prioritization, arguing that the lack of specificity in the context of cognitive diagnostic markers is only associated with research of DSM-defined diagnostic entities. However, review of the literature reveals that underperformance on neuropsychological tests is characteristic of psychopathological mechanisms (outside traditional DSM disorders), which are associated with RDoC domains as well. These include negative valence, behavior disinhibition, acute and potential threat, and habit formation. In a perfect world, the findings discussed in this and other papers ought to inform us that it is not the classification of psychopathology that results in a lack of specific diagnostic cognitive markers. It is the lack of specificity of neuropsychological assessment. Nevertheless, the state of the field is such that labor, resources, and funding continue to be invested in cognitive markers research. This may, in turn, obfuscate reconsideration of the role of classic neuropsychology in psychiatry research and thus hinder progress and innovation in the field.

The dubious belief, reinforced with increasingly more sophisticated technologies used in neuroscience, that the root cause of psychopathology will be found in the assessment arsenal of the neuropsychologist (or the microbiologist or the neuro-radiologist), is unlikely to deliver the

desired answers. Psychopathology and brain diseases are of different logical categories that may complement and overlap with each other, but cannot form a reductionist explanatory basis of each other, in the same way, for example, that molecular motion can explain heat. By implication, neuropsychological assessment lacks the necessary specificity to identify and individuate psychopathology. It is better left to its important role of providing increasingly more sensitive and specific information on the cognitive status of various conditions, be they of developmental, medical, or psychiatric etiology.

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Anatomy of an Epidemic: The History and Science of a Failed Paradigm of Care

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IN CONVENTIONAL HISTORIES of psychiatry, the arrival of chlorpromazine in asylum medicine is said to have kicked off a “psychopharmacological revolution.” Chlorpromazine was christened an “antipsychotic,” and soon researchers had also discovered *antidepressants* and *anti-anxiety* agents, names that told of drugs that were specific antidotes to these major mental disorders. Fluoxetine, which was brought to market in 1988, was the first of a second generation of psychiatric drugs, said to be safer and more effective than the first, and this marked another step up the ladder of progress. The public was informed that these drugs fixed chemical imbalances in the brain, and thus were like “insulin for diabetes,” and with that metaphor in mind, societal usage of the drugs soared.

That is a compelling narrative. And given that it tells of great progress in treating psychiatric disorders, it might be expected that the burden of mental illness in American society would have declined during the past 60 years. Instead, the opposite has occurred. As this revolution has unfolded, the burden of mental illness in American society, as measured by the percentage of the population on disability due to psychiatric disorders, has dramatically increased. That is also true for other societies that have adopted a drug-based paradigm of care.

In 1955, there were 355,000 people in state and county mental hospitals with a psychiatric diagnosis (another 210,000 patients in the hospitals suffered from alcoholism, syphilis-related dementia, and other neurological conditions; Silverman, 1968). That is a disability rate of 1 in every 468 Americans. The United States then emptied its mental hospitals, and today the “disabled” mentally ill receive either a monthly Supplemental Security Income (SSI) or Social Security Disability Insurance (SSDI) payment (or both). In 1987, a year that could be said to mark the end of the first-generation era of psychiatric drugs, there were 1.25 million people receiving an SSI or SSDI payment because they were disabled by mental illness, a disability rate of 1 in every 184 Americans (Social Security Administration, 1987).

It can be argued that this is an apples-to-oranges comparison. It may be that one had to be much sicker to be admitted to a mental hospital in 1955 than to receive an SSI or SSDI payment in 1987. Fortunately, since 1987, it's possible to make an apples-to-apples comparison, involving only SSI and SSDI numbers. In 2013, there were 4.5 million adults receiving a disability payment due to a mental illness (Social Security Administration, 2013). That is a disability rate of 1 in every 70 Americans, more than twice the disability rate in 1987, the year before fluoxetine came on the market.

Other countries are reporting similar increases in disability due to mental illness. For instance:

- In Australia, the number of adults on government disability rose from 57,008 in 1990 to 241,355 in 2011, a four-fold increase (Australian government, 2011).
- In New Zealand, the disability numbers rose from 21,972 in 1998 to 50,979 in 2011, a doubling of the disabled mentally ill in 13 years (New Zealand Ministry of Social Development, 2004-2011).
- In Iceland, the number of new cases of disability due to a psychiatric disorder increased from 84 per 100,000 adults in 1992 to 217 per 100,000 adults in 2007 (Thorlacius, Stef ánnsson, Ólafsson, & Tómasson, 2010).
- In Denmark there were 3,550 new disability awards due to psychiatric disorders in 1999; eleven years later, this number had jumped to 8,812 (Danish government).
- In Sweden, about 25% of all new disability claims in 1999 were due to psychiatric disorders; by 2011, this percentage had risen to nearly 60% (OECD, 2013).

There may be many factors contributing to this rise in the burden of mental illness in the United States and other societies. But the rise also necessarily begs a question: Is it possible that the widespread use of psychiatric drugs is helping to fuel it? To answer that question, two other questions

need to be investigated. First, what are the long-term effects of psychiatric medications? Do they reduce symptoms over the long term and help people function better? Or not? Second, is it possible that a psychiatric drug, because of its side effects, may transform a temporary problem into a chronic one? For example, is it possible that antidepressants induce a mood instability in some patients, which leads to a diagnosis of bipolar illness?

How Psychiatric Drugs Act on the Brain

The conventional narrative tells of drugs that are *antidotes* to a biological problem. If this is true, it could be expected that the drugs would be effective over both the short term and long term. However, a review of the relevant science reveals that psychiatric drugs are better understood as agents that *create* chemical imbalances in the brain.

The chemical imbalance theory of mental disorders rose in the 1960s after researchers discovered how antipsychotics and antidepressants acted on the brain. Antipsychotics were found to block dopamine receptors, and so researchers hypothesized that schizophrenia might be the result of too much dopamine activity. In a similar vein, tricyclics and monoamine oxidase inhibitors were found to thwart the removal of norepinephrine and serotonin from the synaptic cleft between neurons, which increased the activity of those two neurotransmitters (known as monoamines.) Thus, researchers hypothesized that depression might be due to a monoamine deficiency.

In the 1970s, researchers began testing these hypotheses. As early as 1984, NIMH-funded researchers concluded that it didn't appear that depression was associated with a deficiency in serotonin (Maas et al., 1984). Subsequent studies also failed to support the low-serotonin theory of depression. In 1999, the American Psychiatric Association's *Textbook of Psychiatry* reviewed this history, and noted that the theory had been based on faulty logic from the start (Dubovsky, 1999).

Inferring neurotransmitter pathophysiology from an observed action of a class of medications on neurotransmitter availability is similar to concluding that because aspirin causes gastrointestinal bleeding, headaches are caused by too much blood and the therapeutic action of aspirin in headaches involves blood loss. Additional

experience has not confirmed the monoamine depletion hypothesis. (p. 516)

While scientific investigations into the dopamine hypothesis of schizophrenia produced a more nuanced record, researchers—as former NIMH director Steven Hyman wrote in a 2001 book titled *Molecular Psychopharmacology*—failed to find that a “lesion in the dopamine system is a primary cause of schizophrenia” (Nestler, Hyman, & Malenka, 2001). There are at least a few scientists still studying dopamine function in psychotic patients, but, as Swedish investigators wrote in 2012, after summarizing this ongoing research, “any simple, exclusive pathology of the dopamine system (in schizophrenia) was and is doubtful” (Jucaite & Nyberg, 2012).

Many leading figures in psychiatry have now written about how the chemical imbalance theory of mental disorders failed to pan out. In 2005, Kenneth Kendler, co-editor in chief of *Psychological Medicine*, wrote a particularly succinct epitaph: “We have hunted for big simple neurochemical explanations for psychiatric disorders and have not found them” (Lacasse & Leo, 2005).

However, while researchers didn’t find that people diagnosed with schizophrenia or depression suffered from known chemical imbalances prior to being medicated, they did come to understand that the drugs created the very imbalances hypothesized to cause the disorders in the first place. For example, antipsychotics—which are also known as neuroleptics—block a particular subtype of dopamine receptor in the brain, known as the D2 receptor. The antipsychotic is acting as a brake on dopaminergic transmission. In an effort to maintain the normal functioning of its dopaminergic pathways, the brain in response tries to accelerate such activity. The presynaptic neurons put out more dopamine than normal, and the postsynaptic neurons increase the density of their dopamine receptors. While the first compensatory response may burn out after a while, the postsynaptic response—an “upregulation” of dopamine receptors—remains. The brain, researchers have explained, is trying to maintain a “homeostatic equilibrium” through this compensatory response. As a result, the person’s brain is now “supersensitive” to dopamine.

In a similar vein, an SSRI antidepressant, by blocking the normal reuptake of serotonin from the synaptic cleft, ups serotonergic activity. In response, the brain dials down its serotonergic system. Presy-

naptic neurons put out less serotonin than normal (at least for a time), and postsynaptic neurons decrease the density of their receptors for serotonin. The presence of the drug has driven the brain into a “low serotonin” state.

In 1996, Hyman and Nestler wrote a paper titled “Initiation and Adaptation: A Paradigm for Understanding Psychotropic Drug Action” that summed up this compensatory process. Psychiatric drugs, they wrote, “create perturbations in neurotransmitter function.” In response, the brain goes through a series of compensatory adaptations that “are rooted in homeostatic mechanisms that exist, presumably, to permit cells to maintain their equilibrium in the face of alterations in the environment or changes in the internal milieu.” At the end of this compensatory process, according to the authors, the brain is functioning in a manner that is “qualitatively as well as quantitatively different from the normal state” (Hyman & Nestler, 1996).

This is a very different conception of the drugs than is promoted in the conventional narrative. In this scientific narrative, the biology of mental disorders remains unknown, and psychiatric drugs, rather than fix known abnormalities, create them. And with that understanding in mind, the scientific question can now be properly framed: Do these drugs, when widely used, lessen the burden of mental illness in a society? Or increase it?

The Long-Term Effects of Antipsychotics

If there is any class of psychiatric drugs that could be expected to provide a long-term benefit, it is the antipsychotics. These drugs are said to have enabled deinstitutionalization, and in many corners of psychiatry today, it would be considered heresy to question their long-term use. Antipsychotics are also the best-studied class of psychiatric drugs, and thus it might be expected that there would be abundant evidence of their long-term effectiveness.

The evidence cited for the long-term use of antipsychotics comes from “relapse” studies, dating back to the 1960s. The studies were typically conducted with this design: Patients who had stabilized well on an antipsychotic would either be maintained on the drug or abruptly withdrawn from it. With great regularity, the relapse rate was significantly higher for the withdrawn patients. That is seen as evidence

that continual use of antipsychotics lowers the risk of relapse.

However, it is easy to see that this evidence base is flawed. Abrupt withdrawal of antipsychotics is known to increase the risk of relapse. In the few studies involving gradual withdrawal of an antipsychotic, relapse rates have been much lower. Even more important, the withdrawal studies don’t provide information about how people are functioning over the long term. Are they working? What sort of social lives do they have? The relapse studies are silent on those vital questions.

In a 2002 editorial in *European Psychiatry*, Emmanuel Stip, a professor of psychiatry at the Université de Montreal, reviewed this literature, and concluded that there was “no compelling evidence” that antipsychotics were effective over the long term. As such, he wrote, “if we wish to base psychiatry on evidence-based medicine, we run a genuine risk in taking a closer look at what has long been considered fact” (Stip, 2002).

Here is what a closer look reveals.

Although schizophrenia is often said to be a chronic, deteriorating disorder, with few patients recovering, a review of outcomes for schizophrenia patients from 1945 to 1955—the decade before the introduction of chlorpromazine—presents a different picture. Studies of first-episode schizophrenia patients conducted during that 10-year period found that 60% to 70% would be discharged within 12 months, and that two-thirds of the first-episode patients would be living within the community 3 to 5 years after initial hospitalization (Cole, 1959; Lehrman, 1961; Warner, 1985). This meant that one-third of first-episode patients became chronically ill, while the remaining two-thirds recovered to some degree and were able to live outside the hospital, even though, at that time, there was no government support for community care.

In order for a medical treatment to be effective, it needs to improve on the “natural” course of the disorder. Otherwise, the therapeutic intervention is doing harm. The 1945–1955 data provides a summary of outcomes for people diagnosed with schizophrenia when chlorpromazine was introduced, and the expectation, if the conventional narrative is correct, is that antipsychotics would improve these outcomes going forward.

Although psychiatrists at that time observed that chlorpromazine and other new antipsychotics helped their psychotic patients stabilize more quickly, they also

noticed that their drug-treated patients were returning to the hospital in droves, a new clinical course they dubbed the “revolving door syndrome.” In addition, there were clinicians who worried that “relapse is greater in severity during drug administration than when no drug is given” (Gardos & Cole, 1977).

In the early 1960s, the NIMH mounted the first well-controlled study of antipsychotics. At the end of 6 weeks, the drug-treated patients were doing better. However, many of the placebo patients improved as well during this period and were discharged from the hospital, and at the end of 1 year, the NIMH researchers reported a surprising finding: “Patients who received placebo treatment were *less* likely to be rehospitalized than those who received any of the three active phenothiazines” (Schooler et al., 1967).

This was the first longer-term study of antipsychotics, and it hinted at a possible paradox. Could drugs that were effective over the short term increase the chronicity of the disorder over the long term?

During the next 15 years, four studies indicated that the drugs had that harmful long-term effect. In a retrospective study, Bockoven reported that rehospitalization rates at the end of 5 years for patients treated in 1967 at Boston Psychopathic Hospital were higher than they had been for similar patients treated in 1947, and that the 1967 group was also much more socially dependent (Bockoven & Solomon, 1975). Next, in three NIMH-funded studies that assessed outcomes of medicated and unmedicated schizophrenia patients at the end of 1 to 3 years, regular antipsychotic use was associated with a higher relapse rate and worse social functioning (Bola & Mosher, 2003; Carpenter et al., 1977; Mathews et al., 1979; Rappaport et al., 1978). These findings led William Carpenter, the lead investigator in one of the studies, to raise a haunting question:

There is no question that, once patients are placed on medication, they are less vulnerable to relapse if maintained on neuroleptics. But what if these patients had never been treated with drugs to begin with? . . . We raise the possibility that antipsychotic medication may make some schizophrenic patients more vulnerable to future relapse that would be the case in the normal course of the illness. (Carpenter et al., 1977, p. 19)

At that point, two investigators at McGill University, Guy Chouinard and Barry

Jones, offered a biological explanation for why this might be so. Antipsychotics blocked dopamine receptors, and in compensatory response, the brain increased the density of those receptors. The brain was now “supersensitive” to dopamine, and this led to a long-term trap. Patients who stopped taking an antipsychotic could be expected to suffer severe relapses, making it difficult for them to get off the medications, and yet if patients stayed on the drugs indefinitely, this dopamine supersensitivity could produce persistent psychotic symptoms, with the illness appearing “worse” than ever before. According to Chouinard et al., “New schizophrenic symptoms or original symptoms of greater severity will appear” (Chouinard, Jones, & Annable, 1978; Chouinard & Jones, 1980, 1982).

What was so troubling about this finding was that it meant antipsychotics could worsen the very symptom they were supposed to treat. In addition, the drugs were known to cause many adverse effects, and if the drugs also worsened psychotic symptoms for some patients, there was nothing left on the benefit side of the risk-benefit equation for those patients. There were only negative effects to be tallied up.

This was clearly a threat to the conventional narrative, which perhaps explains why, in the following years, the field mostly turned its eyes away from the dopamine supersensitivity worry, and instead focused on the relapse studies as evidence that maintaining patients on antipsychotics was a helpful treatment. In this way, the profession could assure itself that its long-term use of antipsychotics was “evidence based,” and, as a result, the dopamine supersensitivity threat faded into the background. But that worry arose in the early 1980s, and so it is possible now to review the relevant research conducted during the past three decades to see whether it was a false alarm.

Here is a summary of such research:

- In two studies by the World Health Organization, schizophrenia patients in three developing countries, India, Nigeria, and Columbia, had superior outcomes at the end of 2 and 5 years than patients in the United States and five other developed countries. In the developing countries, only 16% of patients were regularly maintained on antipsychotics, versus 61% in the rich countries (Jablensky et al., 1992). Thus, in these cross-cultural studies, outcomes were better in countries that minimized long-term use of antipsychotics.

- In studies using MRI technology to measure brain volumes, antipsychotics have been found to shrink gray matter and white matter volumes. This brain shrinkage is associated with a worsening of negative symptoms and functional impairment, and after 5 years, a significant worsening of cognitive abilities (Aderhold, 2014; Andreasen, 2005; Ho et al., 2003; Radua et al., 2012). This research provides a model of an iatrogenic process: A drug causes a morphological change in the brain, and this change is associated with harmful long-term effects.
- In animal models of psychosis, Philip Seeman, a researcher at the University of Toronto, found that amphetamines, angel dust, and lesions to the hippocampus all cause an increase in D2 receptors in the brain. Antipsychotics also increase D2 receptors, and thus they cause the very abnormality that psychosis-inducing agents do (Seeman et al., 2005). He and his colleagues reported that this is why antipsychotics “fail” over the long term (Samaha et al., 2007).
- In a naturalistic study of schizophrenia patients, Martin Harrow found that those off antipsychotic medication had markedly superior long-term outcomes. At the end of 15 and 20 years, those off antipsychotics were eight times more likely to be recovered, and had superior outcomes in every domain measured: they were much less likely to still be experiencing psychotic symptoms, much less anxious, had better cognitive function, and were much more likely to be employed. Harrow and his collaborator, Thomas Jobe, wrote that drug-induced dopamine supersensitivity was likely the reason that the medicated patients were so much more likely to be psychotic over the long term than the unmedicated patients (Harrow & Jobe, 2007; Harrow, Jobe, & Faull, 2012; Harrow & Jobe, 2013; Harrow, Jobe, & Faull, 2014).
- In a randomized study conducted in the Netherlands, in which patients stabilized on neuroleptics were either maintained on usual doses or tapered down to a low dose (or discontinued altogether), 40% of the minimal dose group was in recovery at the end of 7 years, versus 18% on treatment at usual doses (Wunderink et al., 2013).

As can be seen, this history of research into the long-term effects of antipsychotics tells

a consistent story. Evidence that the drugs were increasing the chronicity of schizophrenia shows up in the observation by clinicians that their medicated patients were returning to the hospital in droves; in the 1-year results of the first NIMH study; in Bockoven's retrospective study; and in the three longer-term studies funded by the NIMH in the 1970s. Researchers then stepped forward with a biological explanation for why this is so. Since then, WHO cross-cultural studies, MRI studies, an animal model of psychosis, a 20-year naturalistic study, and a 7-year randomized study all support the same conclusion: Antipsychotics increase the chronicity of psychotic disorders and impair functioning over the long term.

There are some patients who may benefit from antipsychotics over the long term. This research record simply reveals the drugs' effects on outcomes in the aggregate, and that record shows that a paradigm of care that emphasizes maintenance antipsychotic use for all patients increases the percentage of patients who end up chronically ill and disabled.

An Episodic Illness Turns Chronic

A historical review of the antidepressant literature produces a similar story. The drugs increase the chronicity of depressive disorders over the long term, and increase the risk that a person who suffers a bout of depression will become disabled.

Prior to the introduction of antidepressants, depression was understood to be an episodic disorder. Patients hospitalized for a first episode of depression could be expected to recover after a period of time. Over the long-term, about 50% of first-episode patients would not experience a second episode; 30% or so would suffer two to three episodes in a 15-year period; and perhaps 20% would become chronically ill. As one expert in mood disorders concluded in a 1969 textbook, "assurance can be given to a patient and to his family that . . . a first depression will not tend toward a more chronic course" (Winokur, 1969).

However, once antidepressants began to be regularly prescribed during the 1960s, several clinicians observed that their depressed patients, after they were treated with antidepressants, appeared to be relapsing more frequently. The tricyclics, wrote one psychiatrist, were inducing a "change to a more chronic course" (Van Scheyen, 1973). In 1973, Van Scheyen examined the case histories of 94 patients, and found that this was indeed the case:

those treated with medication had more relapses. "Long-term antidepressant medication . . . exerts a paradoxical effect on the recurrent nature of the vital depression," he concluded.

Over the next three decades, numerous studies found that depression in the antidepressant era ran a chronic course. But rather than attribute this poor outcome to the drugs, leaders in American psychiatry concluded that the true course of depression was now being discovered. The old epidemiological studies, which told of an episodic disorder, were flawed. As one panel of experts wrote, "Improved approaches to the description and classification of [mood] disorders and new epidemiologic studies [have] demonstrated the recurrent and chronic nature of these illnesses, and the extent to which they represent a continual source of distress and dysfunction for affected individuals" (Consensus Development Panel, 1985).

However, in 1994, Giovanni Fava, editor of *Psychotherapy and Psychosomatics*, wrote that perhaps the change in course was real, and that antidepressants were inducing a change in the brain that made patients more biologically vulnerable to depression (Fava, 1994). This was a déjà vu moment for psychiatry, as it recalled the work of Chouinard and Jones, and since then, Fava has repeatedly sounded that worry, and detailed the evidence to support it (Fava, 1999, 2003; Fava & Offidani, 2011).

Although there are no long-term RCTs of antidepressants, naturalistic studies conducted in the past 25 years have regularly found that the off-medication patients have better outcomes. They are less likely to be symptomatic years later, and more likely to be functioning well (Goldberg et al., 1998; Patten, 2004; Ronalds et al., 1997; Weel-Baumgarten et al., 2000). At least two studies have examined the risk of becoming disabled by the disorder. In a 6-year study by the NIMH, the patients who were treated were three times more likely than the untreated group to suffer a "cessation" of their "principal social role," and nearly seven times more likely to become "incapacitated" (Coryell et al., 1995). In a Canadian study, the long-term disability rate was twice as high for those who took an antidepressant compared to those who did not (Dewa, 2001).

With such results piling up, researchers in Canada, Netherlands, and elsewhere have now written about why antidepressants may worsen long-term outcomes,

and they have focused on the compensatory adaptations induced by the drugs as a likely explanation (Andrews et al., 2012; Bockting et al., 2008). Finally, in 2011, American psychiatrist Rif El-Mallakh proposed a name for this long-term adverse effect of antidepressants: tardive dysphoria (El-Mallakh, Gao, & Robert, 2011):

A chronic and treatment-resistant depressive state is proposed to occur in individuals who are exposed to potent antagonists of serotonin reuptake pumps (i.e., SSRIs) for prolonged time periods. Due to the delay in the onset of this chronic depressive state, it is labeled tardive dysphoria. Tardive dysphoria manifests as a chronic dysphoric state that is initially transiently relieved by—but ultimately becomes unresponsive to—antidepressant medication. Serotonergic antidepressants may be of particular importance in the development of tardive dysphoria. (p. 771)

Once again, even in this cursory review of the literature, it can be seen that history and science are telling a consistent story. When antidepressants were introduced, there were clinicians who worried that their depressed patients were relapsing more frequently than before. A Dutch study found that to be so. Epidemiological studies and other research revealed that depression in the antidepressant era runs a more chronic course than it did before. Modern naturalistic studies routinely found that unmedicated patients have better long-term outcomes. Investigators then put together a biological explanation for why this would be so. There is a consistency to this narrative of science that stretches over four decades.

A Universal Problem?

There are similar histories that can be dug out from the literature for other classes of psychiatric drugs. There is evidence that long-term users of benzodiazepines may become chronically anxious and functionally impaired (Rickels et al., 1991; Rickels et al., 1999). Long-term studies of stimulants for ADHD have failed to find that they produce a benefit on any domain. Meanwhile, in the modern era of lithium and mood stabilizers, the leading experts in manic-depressive illness have noted that it runs a more chronic course than it did in the pre-drug era (Huxley & Baldessarini, 2007; Zarate et al., 2000).

As investigators confront these dismal long-term outcomes, they are focusing on

the possibility that the drugs fail over time because they induce compensatory adaptations “the opposite of what the medication originally produced.” This, El-Mallakh wrote, may “cause a worsening of the illness, continue for a period of time after the discontinuation of the medicine, and may not be reversible” (El-Mallakh et al., 2011). However, the biology of this drug failure clearly needs to be further investigated, for there is also evidence that the neurotransmitter pathways affected by a drug simply become less functional. The increase in D2 receptors in patients treated with antipsychotics is also associated with tardive dyskinesia, a disabling condition caused by dysfunction in the basal ganglia. Antidepressant-induced tardive dysphoria also seems to differ from feelings of depression; people suffering from it tell of an inability to mount an emotional response. There is a deadening of emotion, as opposed to the acute pangs of depression.

As El-Mallakh also warned, it may be that the drug-induced compensatory adaptations are not reversible, even if the drug is withdrawn. The changes in receptor densities may not renormalize. That is regularly the case with tardive dyskinesia; the motor dysfunction remains even after the offending antipsychotic is withdrawn. Meanwhile, many people struggling with tardive dysphoria tell of feeling dead to the world years after they have stopped taking an antidepressant.

Creating Bipolar Illness

It is easy to understand why treatments that increased the chronicity of a disorder over the long term, and also increased the risk of functional impairment, would stir an increase in the burden of that illness in society, and stir an increase in the number of people disabled by that disorder. There is also a second cause of the disability epidemic: the propensity of antidepressants to stir mood instability that leads to a diagnosis of bipolar disorder.

In the 1970s, clinicians realized that antidepressants could induce manic episodes, and thus increased the risk that a person diagnosed with unipolar depression would turn “bipolar.” Swiss investigators tracking changes in the patient mix at a mental hospital in Zurich reported that following the introduction of antidepressants, “bipolar disorders increased; more patients were admitted with frequent episodes” (Angst, 1985). Researchers at Yale University have quantified this increased risk. They reviewed the records of 87,290

patients diagnosed with depression or anxiety between 1997 and 2001 and determined that those treated with antidepressants converted to bipolar at the rate of 7.7% per year, which was three times greater than for those not exposed to the drugs (Martin, 2004). Finally, a survey of members of the Depressive and Manic-Depressive Association found that 60% of those with a bipolar diagnosis reported that they had initially fallen ill with major depression and had turned bipolar after exposure to an antidepressant (El-Mallakh, 2002).

This is an “evidence base” that tells of a paradigm of care that routinely manufactures bipolar patients. In a 2005 interview, Fred Goodwin, a former director of the NIMH, acknowledged that this was so. “If you create iatrogenically a bipolar patient, that patient is likely to have recurrences of bipolar illness even if the offending antidepressant is discontinued. The evidence shows that once a patient has had a manic episode, he or she is more likely to have another one, even without the antidepressant stimulation” (see *Primary Psychiatry*, 2009).

Given that antidepressants, which are so frequently prescribed, may induce a chronic dysphoria and bipolar disorder, it could be expected that an increase in disability due to mood disorders would be driving the disability epidemic in the United States (and elsewhere). In 1955, there were only 50,937 people in state and county mental hospitals in the United States with a diagnosis of major depression or manic-depressive illness (Silverman, 1968). The prevalence of those affective disorders, in a severe form that “disabled” people, was quite low. In 2013, there were 2.1 million people receiving a disability payment due to a mood disorder (Social Security Administration, 2013.) This was 47% of the total number of people on SSI or SSDI due to a mental illness, and more than twice the number of people receiving such payments due to psychotic disorders. In the age of Prozac, mood disorders are the leading cause of disability.

A Failed Paradigm of Care

The rising disability numbers due to mental illness, in the United States and other developed countries, provide reason to investigate the long-term effects of psychiatric medications. A thorough review of the history of such research reveals that, on the whole, psychiatric medications increase the chronicity of mental disorders and

impair functioning over the long term. At the same time, the widespread use of antidepressants is stirring a dramatic increase in the number of people disabled by bipolar disorder.

This is evidence of a failed paradigm of care. Peter Göttsche, a co-founder of the Cochrane Collaboration, has investigated the broader effects of psychiatric drugs on society, and he summed up his thoughts in this way:

I know some excellent psychiatrists who help their patients a lot . . . I also know that some drugs can be helpful sometimes for some patients. And I am not “antipsychiatry” in any way. But my studies in this area lead me to a very uncomfortable conclusion: Our citizens would be far better off if we removed all the psychotropic drugs from the market, as doctors are unable to handle them. It is inescapable that their availability creates more harm than good. (Göttsche, 2013, p. 233)

In the United States and other developed countries, psychiatric care is organized around a conventional narrative that tells of how the arrival of chlorpromazine kicked off a “psychopharmacological revolution.” But there is another narrative of science to be dug out from the research literature, and it tells of a pressing need for societies to fundamentally rethink that drug-based paradigm of care.

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When Marketing Met Science: Evidence Regarding Modern Antidepressants and Antipsychotic Medications

Glen Spielmans, *Metropolitan State University
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PRACTITIONERS ARE WIDELY ENCOURAGED to engage in evidence-based medicine (EBM). Put simply, EBM encourages the preferential use of treatments that have been deemed efficacious and safe in randomized controlled trials (RCTs). Such trials are the foundation for treatment guidelines endorsed by prestigious organizations such as the American Psychiatric Association in the United States and the National Institute for Health and Care Excellence in the United Kingdom.

The efficacy and safety of antidepressant and antipsychotic medications have been examined in an impressive volume of RCTs. The drug industry and much of academic psychiatry uses findings from these trials to buttress the idea that psychiatric medicines are among the most studied in all of medicine. Published clinical trials are used to market drugs as having scientific support for various uses. For instance, a memo from Pfizer reads, "What is the purpose of publications?" and responded with, "High quality and timely publications optimize our ability to sell Zolofit [the antidepressant sertraline] most effectively" (Clary, 2000). Internal documents from various drug firms, statements from executives and employees of firms that contract with the drug industry to help write clinical trial manuscripts, and materials on such firms' websites all emphasize that clinical trials are of the utmost importance for drug marketing (Sismondo, 2011). Physicians want to practice EBM and the drug industry is happy to let physicians know that their products are, of course, based on solid evidence. Data from clinical trials are typically presented in a main journal article, followed by slicing and dicing of data into secondary analyses, post-hoc analyses, pooled analyses, narrative reviews, and meta-analyses. A clinical trial often serves as a key element in many—sometimes dozens—of subsequent papers that help sell the idea that a product is safe and effective (Melander, Ahlqvist-Rastad, Meijer, & Beermann, 2003; Spielmans, Biehn, & Sawrey, 2010). With so many papers to produce, a large industry of medical educa-

tion and medical writing companies has emerged to help drug firms produce papers that best match desired brand images.

Usage

Among adults, the usage of antidepressants in the United States increased over 450% from 1988–1994 to 2007–2010 (National Center for Health Statistics, 2011, 2010). Antidepressant prescriptions in children have also increased, with one estimate suggesting an increase of over 300% from 1987–1996 (Olfson, Marcus, Weissman, & Jensen, 2002) followed by an increase of close to 200% from 1995–1998 to 2007–2010 (Olfson, Blanco, Wang, Laje, & Correll, 2014). Antipsychotic usage has similarly skyrocketed. Among children, the use of antipsychotics increased 1,236% between 1995–1998 and 2007–2010, whereas the increase was 501% for adolescents (Olfson et al., 2014). For adults, antipsychotic prescriptions doubled from 1993–1998 to 2005–2009 (Olfson, Blanco, Liu, Wang, & Correll, 2012). Meanwhile, the percentage of Americans who participate in psychotherapy has remained essentially flat (Olfson & Marcus, 2010).

Efficacy of Antidepressants and Antipsychotics

Given the wide swath of the American population taking antidepressants and antipsychotics, one would hope for impressive drug efficacy. A large meta-analysis of second-generation antidepressants (selective serotonin reuptake inhibitors [SSRIs] and other newer drugs) versus placebo found a standardized mean difference effect size of $d = 0.31$ (Turner, Matthews, Linardatos, Tell, & Rosenthal, 2008), which two of the researchers interpreted as "measurable and significant" (Turner & Rosenthal, 2008). A similar meta-analysis performed by a different research team arrived at an effect size of $d = 0.32$ (Kirsch et al., 2008), which the authors interpreted as clinically insubstantial. Cohen's (1988) rough guidelines for interpreting effect size ($.2 =$ small, $.5 =$ moderate, $.8 =$ large) are

often utilized but are themselves arbitrary. That being said, it seems unlikely that effect sizes in the realm of .30 (often about equal to about two or three points on the Hamilton Depression Rating Scale or Montgomery-Asberg Rating Scale, the most common measures in antidepressant trials) reflect impressive treatment benefit. The extent to which these modest benefits on depressive symptoms carry over into important life domains (employment, interpersonal relationships, daily functioning, quality of life) is largely unknown (Hotopf, Lewis, & Normand, 1997), though a meta-analysis of the few available trials found no benefit for antidepressants over placebo for measures of well-being among depressed children and adolescents (Spielmans & Gerwig, 2014). Thus, the clinical significance of antidepressant benefits is questionable.

While antipsychotics can yield very impressive benefits in the treatment of schizophrenia, such changes are the exception, not the rule. In trials used to support applications for Food and Drug Administration approval, second-generation or "atypical" antipsychotics (AAPs) had an average effect size of .44 versus placebo (Turner, Knoopfelmacher, & Shapley, 2012). In clinical trials, most patients who receive antipsychotics do not show a treatment response (Leucht, Arber, Engel, Kissling, & Davis, 2009). Perhaps only about 1 in 6 patients taking an antipsychotic experiences "dramatic" improvement of about 60%–80% symptom reduction (Levine, Rabinowitz, Case, & Ascher-Svanum, 2010).

When AAPs first came to market, they were hailed as major improvements over older, "typical" antipsychotic drugs in terms of efficacy. In particular, AAPs were described as providing superior relief of negative symptoms (e.g., apathy, inability to experience pleasure from normally enjoyable activities, flat affect) than first-generation antipsychotic medications (FGAs). Janssen, manufacturer of risperidone, wanted to make marketing claims regarding the purportedly superior efficacy of risperidone versus older drugs; however, FDA disallowed such marketing because of a lack of supportive evidence (Miller, 1994). Nonetheless, such claims were often made in the scientific literature. A comprehensive meta-analysis comparing AAPs to FGAs found that some AAPs offered a small, statistically significant advantage over conventional medications in terms of overall symptom reduction (Leucht, Corves, et al., 2009). However, among

drugs approved for use in the United States, only clozapine, olanzapine, and risperidone generated such benefits—and the benefits for risperidone were clinically negligible ($g = .13$, equating to a very small effect). Of drugs approved in the U.S., only clozapine exhibited a benefit over an FGA on quality-of-life measures—and this was a small benefit found in only one trial. The same three drugs also demonstrated small advantages over older medications on negative symptoms. Yet even these small advantages observed for these drugs on negative symptoms may be largely due to biased study design.

Study Design Problems

Several design flaws in RCTs of antipsychotics and antidepressants are described in this section. Many RCTs suffer from more than one of the following problems, substantially lowering their value in determining which treatments are “evidence-based.”

Biased Inclusion Criteria

Trials comparing AAPs to FGAs often only included participants who had experienced a poor response to one (sometimes more) trials of FGA medication (Rosenheck, 2005). However, participants were not required to have had a poor response to an AAP, and in some trials, participants who had taken the AAP under investigation were excluded from the study. A study including those with a history of poor response to FGAs but excluding those with such poor response to AAPs is clearly biased. While this rather obvious methodological flaw occurred in many trials, it was often not acknowledged as a limitation by study authors (Leucht, Heres, Hamann, & Kissling, 2007; Volavka et al., 2002).

Prophylactic Antiparkinsonian Medication

Extrapyramidal side effects (EPS) are mainly movement-related disorders often caused by antipsychotic medications, more commonly occurring during treatment with many FGAs relative to most AAPs. Akinesia is often categorized under EPS, but it is not characterized by abnormal movements; rather, it manifests in such ways as apathy, lack of spontaneity, drowsiness, or depression (Rosenheck, 2005). Thus, akinesia is not assessed on popularly used measures of EPS, and such symptoms are also difficult to tease apart from apathy and emotional blunting, which are categorized as negative symptoms of schizophrenia. While such symptoms may occur at

any dose, they appear more common among patients taking higher dosages (Putten & Marder, 1987). Given that comparative AAP vs. FGA trials tended to use high doses of FGAs, this suggests that some of the purported advantage for AAPs is due to side effects caused by high dosages of FGAs.

In some cases, akinesia and other EPS can be controlled through the use of anticholinergic medications such as benztropine (Rosenheck, 2005). Thus, in clinical practice, anticholinergic medications are often co-prescribed with haloperidol on a prophylactic basis (i.e., starting when the antipsychotic is prescribed) as opposed to after EPS have manifested. However, in clinical trials comparing AAPs to FGAs, the vast majority of trials did not allow prophylactic anticholinergic medications. Indeed, of the 150 studies comparing AAPs with FGAs in one meta-analysis, only 11 allowed prophylactic anticholinergic treatment. Authors of some trials stated clearly that such prophylactic treatment was needed to maintain the double-blind—leaving one to wonder why this was not implemented as standard practice in clinical trials. Among the 11 trials allowing prophylactic anticholinergic treatment, six used high doses of haloperidol (≥ 20 mg/day) or chlorpromazine (≥ 600 mg/day). Akinesia can occur even when prophylactic antiparkinsonian medications are used; this appears most likely at high doses of antipsychotic medication (Van Putten & Marder, 1987). Thus, most of the few studies that used prophylactic antiparkinsonian medications still suffered from potential bias due to a high FGA dose.

Concomitant Medication

In some of the trials that led to regulatory approval of the antidepressant fluoxetine, participants receiving fluoxetine were allowed to take sedative medications. The confound of allowing a second psychoactive medication casts doubt on the validity of the findings—which were not particularly impressive anyway, with fluoxetine eking out a quite modest $g = .26$ effect size (Turner et al., 2008). In the two large trials underlying aripiprazole’s approval as an adjunctive treatment for depression, the FDA’s review reported that the sponsor’s analyses included patients who took prohibited medications. In at least one of the trials, many of these patients were taking opioids or barbiturates, which certainly could have impacted the psychological state of participants. When ineligible participants were excluded from analysis, the

drug effect on depression dipped somewhat while the small benefit on overall functioning vanished entirely (Spielmans et al., 2013). Yet the published versions of the trials included these patients in analyses without even a passing mention of such problems.

Compromised Blinding

Many purportedly double-blind trials are unlikely to actually achieve blindness of raters or participants. In addition to inducing akinesia, high doses of FGAs are likely to severely compromise the double-blind, given the distinctly different side effect profiles of commonly studied drugs (particularly haloperidol) in comparison to AAPs. Unblinding due to side effects is even more problematic in trials comparing any antipsychotic to placebo. Use of active rather than inert placebos (drugs that provide similar side effects without putatively antipsychotic pharmacological activity) and using different raters to assess adverse events and efficacy would reduce these problems (Perlis, 2010). Double-blinding is one of the most important elements in elevating the results of placebo-controlled trials to the supposed “gold standard” in evidence-based medicine. Thus, the almost certain lack of acceptable blinding in antipsychotic trials introduces a troubling element of potential bias.

The integrity of blinding is rarely assessed in antidepressant trials (Even, Siobud-Dorocant, & Dardennes, 2000). Patients who are randomized to taking an antidepressant and who experience side effects about which they were warned during informed consent may logically conclude they are taking the active medication. This conclusion amplifies the expectation of improvement in a placebo-controlled trial and potentially produces an “enhanced placebo effect” (Kirsch, Moore, Scoboria, & Nicholls, 2002). Conversely, patients who do not experience expected side effects may have lowered expectations for improvement due to the belief that they are taking inert placebo. A meta-analysis of several fluoxetine clinical trials found strong correlations between the percentage of fluoxetine participants reporting adverse events and the advantage for fluoxetine over placebo ($r = .85$ for a clinician-rated measure of depression and $r = .96$ for a depression self-report; see Greenberg, Bornstein, Fisher, Zborowski, & Greenberg, 1994). Thus, unblinding due to adverse events may impact ratings of symptom severity, though this phenomenon needs more study.

“Wish bias” refers to an assumption that a new drug under investigation is more effective than an older drug. A meta-analysis found that fluoxetine was significantly more effective, relative to placebo, when it was (a) the experimental drug under investigation rather than (b) a reference drug to which a new drug was being compared (Barbui, Cipriani, Brambilla, & Hotopf, 2004). This finding was not due to different dosages being used in the trials and may reflect unblinding occurring during the course of the trials.

Lack of Meaningful Outcome Measures

Psychopharmacological RCTs often use dichotomous outcomes, determining which participants meet criteria for “response” or “remission” based on a cutoff score on a rating scale. In antipsychotic trials, treatment response is often defined by a 20% decrease in symptoms. This criterion is unimpressive, equating to “minimally better” improvement on another widely used outcome measure (Levine, Rabinowitz, Engel, Etschel, & Leucht, 2008). Knowing that more participants on one drug show minimal improvement relative to those taking another drug or placebo is not particularly useful. Further, the cut points used to determine response or remission are largely arbitrary. For instance, the typical practice in antidepressant trials of labeling someone who improves by 50% as a “responder” versus someone who improves by 49% as a “non-responder” seems hardly logical. There is some intuitive appeal in categorizing outcomes, but such outcomes should be only one piece of the outcome puzzle.

Total scores on symptom rating scales are most commonly used as the primary outcome measures in psychopharmacology RCTs. Scores on such scales typically represent an average of items which measure a broad constellation of individual symptoms. In two large trials of quetiapine as an add-on treatment for depression, items on the Montgomery-Asberg Depression Rating Scale (MADRS) assessing “apparent sadness” and “reported sadness” showed a very small effect, outperforming placebo by only about 10%—a much smaller effect than seen on an item assessing “reduced sleep” (Bauer, El-Khalili, Datto, Szamosi, & Eriksson, 2010). Quetiapine’s sedative effect on sleep accounted for its greatest impact on any individual MADRS item. Many people would be surprised that an approved antidepressant barely beats placebo in terms of two items assessing sadness. Examining only the total

rating scale score overlooks the drug’s minimal effect on sadness.

Further, a narrow focus on symptomatic measures largely misses the point of treatment. If a patient has experienced a positive treatment response according to a rating scale yet still has a low quality of life and functions poorly in the community, this can hardly be considered a truly successful outcome. Thus, trials should include assessments of quality of life and functioning. Self-reports are particularly useful in this regard (Hunter, Cameron, & Norrie, 2009). It is at best paternalistic that psychiatric trials often relegate patient-rated outcomes to secondary status if they are included at all. Without some form of self-report, it is difficult or impossible to get a broad outcome assessment. Many researchers, government agencies, and consumers of mental health services endorse a more broad-based idea of assessing mental health recovery (Sklar, Groessl, O’Connell, Davidson, & Aarons, 2013; Substance Abuse and Mental Health Services Administration, 2012). Further, some treatments, including add-on antipsychotic treatment for depression in adults or antidepressant treatment for depressed youth have demonstrated vanishingly small to zero benefits on symptomatic self-reports or quality-of-life measures—yet are widely prescribed (Spielmans et al., 2013; Spielmans & Gerwig, 2014).

In summarizing the results of a Cochrane systematic review of trials comparing aripiprazole with placebo for schizophrenia, the authors noted, “We found it disappointing that, despite considerable investment in clinical trials, no outcome data were available on death, service outcomes, general functioning, behavior, engagement with services, economic outcomes and cognitive functioning” (Belgamwar & El-Sayeh, 2011, p. 17). Similar critiques could be levied at trials of nearly every psychiatric medication.

Studies on cost-effectiveness may shed light on the relative value of various treatments. These analyses typically found that AAPs were more cost-effective than FGAs. This was despite rather dubious clinical trial evidence attesting to the superior efficacy of AAPs and much higher acquisition costs for AAPs. A thorough methodological review of such cost-effectiveness studies found they were actually too poorly designed to reach firm conclusions (Polsky, Doshi, Bauer, & Glick, 2006). The cost-effectiveness literature on SSRIs in treating depression has been similarly critiqued (Barbui, Percudani, & Hotopf, 2003).

High Dropout Rate

In antipsychotic trials, dropout rates are high—typically over 25% and sometimes over 50% in trials lasting up to 12 weeks (Hutton et al., 2012). A survey of psychiatrists, researchers, and those who care for people with schizophrenia found that trials should have a completion rate of 70%–75% at 12 weeks to be considered credible (Xia et al., 2009). The rates of short-term trial dropouts reported in influential Cochrane systematic reviews of clinical trials were 53% in olanzapine studies, 31% in risperidone studies, 56% in quetiapine studies, and 36% in aripiprazole studies (Hutton et al.). These short-term trials may thus lack credibility. There are several statistical methods of handling missing data in clinical trials, but all such methods become less valid as the rate of dropout increases. No amount of statistical wizardry is likely to provide dependable results when few participants complete a trial.

Data Reporting Problems

Publication and Reporting Bias

The antidepressant efficacy literature provides a compelling view of discrepancies between the published literature and the underlying clinical trial evidence. An ambitious meta-analysis contrasted data contained in FDA reviews of 12 antidepressants to published journal articles based on the same underlying data. Data from all antidepressants approved by the FDA from 1987 to 2004 were included. Among trials yielding positive results according to the FDA, 97% were published in a medical journal. Some trials generated a “questionable” outcome, which found negative results on the primary outcome but found some positive results on secondary outcomes. Studies in which both the antidepressant under investigation and an older, established antidepressant failed to outperform placebo were also counted as “questionable.” Half of these “questionable outcome” trials were not published in medical journals, whereas half were published but written as if the results were positive. Even worse, only one-third of studies finding a negative outcome on the primary measure were published, and five of eight such articles were written as if the study had a positive outcome. The effect size for antidepressants over placebos was inflated by 32% (from $d = 0.31$ to $d = 0.41$) when comparing the published results to the results of all trials lodged for these drugs at the FDA (Turner et al. 2008).

The overestimation of antipsychotic efficacy for schizophrenia appears less than what has been observed for antidepressants in treating depression. A meta-analysis compared (a) data lodged at FDA from trials submitted for marketing approval for AAPs and (b) publications in the medical literature based upon the same trials. The medical literature overestimated efficacy versus placebo by only 8% relative to data lodged at FDA (Turner et al., 2012). However, several published journal articles presented efficacy data in a misleading and overly positive fashion. Four unpublished trials of AAPs were uncovered, three of which showed no efficacy relative to placebo and one of which found an antipsychotic inferior to a competing drug. In addition, three trials of iloperidone included both a placebo and competing antipsychotic. In all three trials, iloperidone had poorer efficacy than the competing drug. Yet the journal articles that reported iloperidone clinical trial results did not report these inconvenient outcomes (Turner et al.).

A recent meta-analysis using similar methods found publication bias regarding antidepressants used to treat anxiety disorders. Again, positive studies were more likely to be published than negative studies and data were spun to make statistically nonsignificant benefits appear positive (Roest et al., 2015).

Questionable Veracity of Published Data

During the course of legal action, AstraZeneca (manufacturer of the antipsychotic drug quetiapine) was compelled to release many internal documents. Several documents discuss the relative efficacy of quetiapine to its generic competitor haloperidol as well as the firm's strategy of handling negative research results.

At the American Psychiatric Association's annual convention in 2000, a favorable meta-analysis was presented regarding quetiapine. This study concluded that quetiapine was superior at inducing treatment response among patients with schizophrenia relative to its generic competitor haloperidol (Schulz, 2000). In an accompanying press release, the author of the presentation stated: "I hope that our findings help physicians better understand the dramatic benefits of newer medications like Seroquel [quetiapine], because, if they do, we may be able to help ensure patients receive these medications first" (Olson, 2009). An internal document described the results of research comparing the two com-

pounds, and it found that quetiapine was actually inferior to haloperidol in reducing symptoms (AstraZeneca, 2000). The company document was produced in March 2000, two months before the aforementioned conference presentation. A publications manager at AstraZeneca sent an email regarding the internal data analysis, reading in part: "The data don't look good. In fact, I don't know how we can get a paper out of this" (Tumas, 2000). In response to a journalist's inquiry several years later, the lead researcher on the 2000 presentation conceded that the claim regarding quetiapine being "significantly superior" was an exaggeration, yet maintained that the data analysis was accurate (Olson, 2009).

An AstraZeneca employee was lead author on a 2000 paper claiming that quetiapine had a neutral effect on weight (Brecher, Rak, Melvin, & Jones, 2000). Yet an internal analysis of data from schizophrenia trials conducted from 1993–1999 found that "the incidence rate in adult patients with weight gain $\geq 7\%$ in all trials was 18.2%" and that in placebo-controlled trials, "the relative risk of clinically significant weight gain was 2.5" (Alam & Jeffries, 2008). The internal analysis further noted that "the results of the analysis show that long-term treatment with quetiapine monotherapy was associated with moderate weight gain in patients with schizophrenia." Misleading information on the drug's purported weight-neutral profile appears to have also been disseminated in marketing presentations (Spielmans & Parry, 2010).

The availability of several internal documents regarding studies of quetiapine leads to some concern regarding both the veracity of published data and related claims regarding quetiapine's efficacy and safety. The extent to which such concerns may apply to other medications is unknown at this point. However, a recent analysis found that when FDA inspections revealed likely or definite problems with the reliability of data in clinical trials, published versions of the clinical trials almost always included these questionable data in their analyses and rarely mentioned any violations found by FDA inspectors (Seife, 2015).

Serious adverse events (SAEs) are deemed as such when they are life-threatening; result in death, hospitalization, birth defect or significant disability; require hospitalization; or require intervention to prevent one of the aforementioned outcomes. Truly practicing evidence-based medicine

requires a good understanding of SAE risk associated with various interventions. One investigation of antipsychotic and antidepressant medications compared the reports of SAEs in journal articles to the reporting of SAEs among the same trials published in summary form in an online registry. Just over 43% of SAEs listed in online trial summaries were not listed in the associated journal article. Shockingly, 62% of deaths and 53% of suicides reported in trial summaries did not appear in associated journal articles. The online clinical trial registry used for this analysis is now defunct (clinicaltrialsresults.org), with many study summaries both not listed in another registry and not published in journals (Hughes, Cohen, & Jaggi, 2014).

Other Methodological and Data Reporting Problems

In clinical trials that compared (a) adding an atypical antipsychotic to an antidepressant to (b) adding placebo to an antidepressant, several methodological and data reporting problems were noted. The study protocol for one trial of risperidone stated that the trial's primary endpoint was at 4 weeks; yet, the journal article reporting the study results indicated that 6 weeks was the primary endpoint. The effect size on the primary outcome measure was 30% smaller at 4 weeks compared to 6 weeks, raising the possibility that the changing endpoint was related to poor results at the originally designated endpoint.

In studies of aripiprazole as an add-on antidepressant, all participants took an antidepressant with adjunctive placebo for 8 weeks. Those who improved were removed at the end of this phase, at which point remaining participants (who had all showed inadequate response to adjunctive placebo) were either continued on adjunctive placebo or switched to aripiprazole. In other words, before aripiprazole was compared to placebo, only participants who had already demonstrated poor response to placebo for 8 weeks were allowed to participate. This clearly stacks the deck against the adjunctive placebo group.

Data on adverse events are sometimes reported in a manner that minimizes their true frequency or severity. For instance, conceptually similar events such as sedation, fatigue, and somnolence are sometimes reported separately, without an attempt to pool them together (Spielmans et al., 2013). This directly contradicts FDA guidance (Food, Evaluation, & Research, 2006). Further, suicidal ideation and self-harm were obfuscated under the vague

umbrella term “emotional lability” in a trial of paroxetine for depressed adolescents. In the same trial, three additional cases of treatment-emergent suicidal ideation and self-harm were reported in the sponsor’s internal final report of the trial, which were not reported in the published manuscript of the trial’s results (Jureidini, McHenry, & Mansfield, 2008). Several incidents of suicidal behavior and an actual suicide occurred across several studies of sertraline but were unreported in the published manuscripts that reported the results of said studies (Healy & Cattell, 2003).

In nearly all psychotropic drug clinical trials, most or all potential adverse events are assessed using only vague, open-ended questioning. This has been shown to elicit far fewer reported adverse events than using adverse events checklists (Montejo, Llorca, Izquierdo, & Rico-Villademoros, 2001; Zimmerman et al., 2010). If researchers are not specifically looking for potential adverse events, then they often do not see them. As currently performed, clinical trials fare quite poorly in assessing the incidence of most adverse events. Despite this major limitation, most clinical trials contain statements indicating that a drug is generally safe or well-tolerated.

Biased Clinical Guidelines

Clinical practice guidelines rate findings from RCTs as the highest form of evidence. Thus, unless developers closely control for methodological and data reporting flaws in the underlying RCTs, guidelines will reflect whatever biases are present in these trials. The 2004 American Psychiatric Association practice guideline recommended atypical antipsychotics over first-generation antipsychotics—based on biased underlying studies. Further, multiple authors of these guidelines had financial ties to manufacturers of atypical antipsychotics. In 2009, these guidelines were updated to reflect independently funded studies which found no advantage for atypical antipsychotics over first-generation drugs. This change in guidance reflects that when new drugs enter the market, the supporting research is nearly always controlled by the drug sponsor—with accompanying biased study design, data suppression, selective reporting, and spinning of results that quite often occurs. It often takes many years for researchers to uncover problems in the sponsored trials and for independent studies to find differing results. If a new treatment quickly moves to the top of a practice guideline in psychiatry, skepticism is warranted given

the unimpressive advances in terms of safety and efficacy that have emerged in drug treatments over the past several decades (Deacon, 2013).

Valproate is widely prescribed as a maintenance treatment in bipolar disorder. This widespread prescription is reflected in, and may have been partially caused by, its inclusion on treatment guidelines. Indeed, the 2002 American Psychiatric Association treatment guideline for bipolar disorder recommended valproate as one of the two options “with the best empirical evidence,” alongside lithium (p. 18). Yet only two clinical trials, one of which was very small, met inclusion criteria for a rigorous systematic review of valproate as a maintenance treatment for bipolar. The maintenance effect was barely statistically significant and was no longer significant when a more conservative random effects analysis analytical model was used. The review found the evidence scant enough to state that compared to placebo, valproate’s efficacy “cannot be made with any degree of confidence” (Cipriani, Reid, Young, & Macritchie, 2013). The APA bipolar treatment guideline appears online with a 2010 copyright date, though it is acknowledged as out of date on its title page. The guideline was scheduled to be updated in 2009, yet only the outdated guideline currently appears online. It is puzzling that a leading medical organization would leave an outdated, inaccurate treatment guideline online alongside a statement indicating that the guidelines “provide evidence-based recommendations for the assessment and treatment of psychiatric disorders” (American Psychiatric Association, 2015). Alongside the bipolar treatment guideline sit a variety of other outdated treatment guidelines.

Controlled clinical trial evidence regarding aripiprazole maintenance therapy in bipolar disorder is limited to a single trial in which participants who responded positively to an open-label trial of aripiprazole were randomly assigned, in double-blind fashion, to either continue taking aripiprazole or abruptly switch to placebo. After 26 weeks of double-blind treatment, remaining participants entered a 74-week extension phase. There was no benefit shown for the drug during the 74-week extension phase in terms of relapse into mania or depression. All benefit occurred during the first 26 weeks, when any drug withdrawal effects would have been prone to occur (Franks, Macritchie, Mahmood, & Young, 2008). Indeed, it is rather interesting that 23% of placebo-assigned partici-

pants relapsed during the first 26 weeks, yet only 5% did so during the remaining 74 weeks. Rendering the study yet more problematic, the completion rate at the end of 100 weeks was 1.3%. Yet this study has been cited as solid evidence of the drug’s efficacy in many publications, including review articles and treatment guidelines (Tsai et al., 2011).

Conflicts of interest are common among committee members who design psychiatric treatment guidelines. It is possible that committees consisting of members who do not depend on pharmaceutical industry money for research funding, or who do not provide paid consulting or paid promotional speeches for drug firms, may develop treatment guidelines that more accurately reflect the treatment literature and its shortcomings.

Conclusions

Those who control the evidence and its dissemination control EBM. Drug marketing messages painting an overly optimistic picture of efficacy and safety have often ridden roughshod over solid scientific practices in the conduct and reporting of clinical trials. Given the various problems described in this paper (and in more detail elsewhere), it is wise to consider the clinical trials literature as presenting an upper limit for efficacy and as substantially underreporting adverse events. Problems with clinical trials have carried over into clinical practice guidelines. Trials need to utilize higher standards, avoiding the wide variety of biases often built into the current literature, as well as assess more meaningful outcomes and make a more serious attempt to assess adverse events. Public access to trial data is also sorely needed.

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"The nervous system, however, is only one specialized part of the organism, and it is open to influences of various kinds from other cells of the body."

—Clifford T. Morgan

Physiological Psychology

McGraw-Hill (1943)

Antidepressants and the Chemical Imbalance Theory of Depression: A Reflection and Update on the Discourse

Jeffrey R. Lacasse, *Florida State University*

Jonathan Leo, *Lincoln Memorial University*

A DECADE AGO, WE PUBLISHED an article in *PLoS Medicine* about the serotonin deficiency theory of depression (Lacasse & Leo, 2005). We transposed the psychiatric literature on serotonin and depression with what pharmaceutical companies had been claiming in their consumer advertisements for years—that a chemical imbalance (serotonin deficiency) caused depression and this imbalance was corrected by selective serotonin reuptake inhibitor (SSRI) drugs. For instance, advertisements for fluoxetine (Prozac) had stated:

When you're clinically depressed, one thing that can happen is the level of serotonin (a chemical in your body) may drop. So you may have trouble sleeping. Feel unusually sad or irritable. Find it hard to concentrate. Lose your appetite. Lack energy. Or have trouble feeling pleasure...to help bring serotonin levels closer to normal, the medicine doctors now prescribe most often is Prozac® (Eli Lilly, 1998)

We knew that such advertisements did not accurately reflect the scientific status of the serotonin theory in the psychiatric research community (see Table 1; we have modified the original table to integrate new material that came to our attention since 2005). Some advertisements were more tentative or clever in their wording than others, but it seemed obvious that the drug companies were at least pushing the boundaries. We thought several of them were going over the line, in plain sight of the Food and Drug Administration (FDA), which ostensibly regulates direct-to-consumer advertising. Our goal was to illustrate the clear disconnect between the existing psychiatric science and what the public was being told in these advertisements, and we argued that the FDA should issue warning letters to pharmaceutical companies (Lacasse, 2005; Lacasse & Leo, 2005). Of course, there were ramifications for clinicians—if it was illegal to claim this in advertisements, wasn't it also an unac-

ceptable thing to be telling vulnerable clients?

After the publication of the paper, we were interviewed by numerous journalists. Several of them thought our work was provocative and that we were “attacking” a well-accepted theory. If it was an attack, it was an inside job, as our sources included NIMH-funded scientists, an award-winning biological psychiatrist, and a popular psychiatric textbook. Anyone familiar with the history of serotonin research would find our argument unremarkable (e.g., Healy, 1997, 2004; Moncrieff, 2008; Valenstein, 1998). In the United Kingdom, psychiatrist David Healy has been making this point for decades (e.g., Healy, 1987, 1997, 2004, 2012, 2015). But the questions from journalists reminded us that the enormous marketing campaigns promoting SSRI drugs (and surely many of the physicians prescribing them) had convinced the U.S. public that the serotonin theory of depression was firmly grounded in science. This wasn't just an issue of misleading advertising. Instead, the incredulity seemed fueled by the significant number of mental health clients who had heard the chemical imbalance explanation from their prescribers.

We urged these reporters to query the FDA, American Psychiatric Association (APA), NIMH, and other official organizations about the science behind the advertisements. *New Scientist* interviewed Wayne Goodman, at the time a University of Florida psychiatrist and Chair of the FDA Psychopharmacological Committee. Dr. Goodman called the serotonin theory “a useful metaphor”—but one he never used when informing his own patients, stating, “I can't get myself to say that” (Lacasse & Leo, 2006; *New Scientist*, 2005). One has to expect that patients whose doctors *had said that* found this news upsetting.

Serotonin imbalance as metaphor is obviously a deep problem for many of the patients who have heard their physicians explain that their depression is caused by a chemical imbalance. These patients must

have assumed that they were hearing real science, and not metaphor. Goodman's public statement raised the question: How are patients with both diabetes and depression who listen to their doctor's explanation of their two conditions supposed to know that one explanation is based on scientific measurement, and one is just a metaphor?

The Problematic Advertisements Disappear

In the early 2000s, the serotonin metaphor of depression was widely advertised by the makers of antidepressants, including advertisements for citalopram, escitalopram, fluoxetine, paroxetine, and sertraline (Lacasse & Leo, 2005). In particular, Zoloft (sertraline) advertisements featuring the miserable ovoid creature were unavoidable in U.S. television and magazines. An on-line repository of direct-to-consumer advertisements for psychiatric drugs lists many from 1997–2007 referring to a chemical imbalance, across many drugs and diagnostic categories (Hansen, 2015a, 2015b).

A 2010 study compared on-line drug advertising of antidepressants regarding the issue of chemical imbalance in both 2004 and 2009 (Lacasse & Hughes, 2010). The number of websites making such claims dropped, with some websites going dark or minimalist as the drug patents ran out. Interestingly, some on-patent drug websites had simply removed the chemical imbalance claims. Newer medications were promoted as “adjusting” or “affecting” neurotransmitter levels, in contrast to “correcting a chemical imbalance.”

From 2014–2015, we collected further data, finding that the simplistic narrative of chemical imbalance that was so common in direct-to-consumer advertising in the 2000s is not widespread any longer. Consumers are no longer informed that antidepressants will normalize their neurotransmitter levels. The Abilify thermostat is gone (Lacasse & Leo, 2006) and drugs are now advertised as “affecting” neurotransmitters. This is mostly true for other classes of medications as well, as advertisements for psychostimulants (Leo & Lacasse, 2009) have also moderated their language substantially. While we still see problematic advertisements, the overall situation has obviously improved.

There is no public explanation for why this happened. To our knowledge, FDA has never sent a warning letter to a pharmaceutical company over claims that antidepress-

sants correct a chemical imbalance. In our assessment, the promotion of chemical imbalance theory in advertisements for SSRI drugs was wildly successful for the drug companies and the psychiatric profession alike. While it's difficult to imagine that they pulled them arbitrarily, we don't know why they largely disappeared.

By roughly 2007, anyone who Googled "serotonin and depression" could easily find articles explaining the mythical nature of serotonin imbalance, or at least the argument. We don't claim that our one little article was responsible, or even original (see Breggin, 1998; Glenmullen, 2000; Healy, 1997, 2004). But given that the public had accepted the serotonin theory as fact (Pescosolido et al., 2010), the widespread public criticism of it and emerging transparency of information on the Internet would obviously create problems, or at least a dilemma. Below, we highlight a few examples of the recent discourse on these issues (see also Levine, 2014; Lynch, 2015; Whitaker, 2010, 2015; Whitaker & Cosgrove, 2015).

I Don't Really Believe It, but I Say It to Patients Anyway

Psychiatrist Daniel Carlat is a practicing psychiatrist, a clinical instructor at Tufts University, and editor of *The Carlat Psychiatry Report*, which we have read for years. On July 13, 2010, he appeared on National Public Radio (NPR; Davies, 2010) to promote his book, *Unhinged* (2010), in which he describes psychiatry as a profession in crisis. Carlat had received some attention in *The New York Times*, candidly reporting his experience pitching venlafaxine (Effexor) to other doctors as a paid consultant for Wyeth. He found himself "tweaking and pruning the truth to stay positive about the product" and eventually resigned (Carlat, 2007). We find that Carlat is unusually transparent, providing interesting insights into uncomfortable issues.

Carlat was asked what we know about psychiatric medication. He responded:

What we don't know, is we don't know how the medications actually work in the brain. . . . I'll often say something like the way Zoloft works, is, it increases the level of serotonin in your brain (or synapses, neurons), and, presumably, the reason you're depressed or anxious is that you have some sort of a deficiency. And I say that [chuckles] not because I really believe it, because I know the evidence really isn't there for us to understand the mecha-

nism—I think I say that because patients want to know something. And they want to know that we as physicians have some basic understanding of what we're doing when we're prescribing medications. They certainly don't want to know that a psychiatrist essentially has no idea how these medications work. (Davies, 2010)

This is surely a remarkable public admission. Carlat continues:

We're in a paradoxical situation, I think, where we prescribe medications that do work according to the trials. And yet as opposed to essentially all other branches of medicine, we don't understand the pathophysiology of what generates mental illness and we don't understand exactly how our medications work. (Davies, 2010)

A practicing psychiatrist could understandably report that they see the medications working in their practice and find them useful. Invoking the clinical trials is perhaps a strange direction to go here, because the consistent lack of difference between SSRI and placebo in the clinical trial literature is one of the most compelling arguments against the serotonin deficiency theory. So Carlat is aware of the clinical trials, which essentially refute the serotonin theory, yet still tells patients that they have a serotonin imbalance. And while some prescribers of psychiatric medication object to misleading SSRI advertisements (Rickels, 2006), Carlat sees widespread pharmaceutical propaganda as an opportunity:

One thing that has happened is that because there's been such a vacuum in our knowledge about mechanism, the drug companies have been happy to sort of fill that vacuum with their own version of knowledge, that usually if you see a commercial for Zoloft on TV, you'll be hearing the line about serotonin deficiencies and chemical imbalances, even though we don't really have the data to back it up. It becomes a very useful marketing line for drug companies, and then it becomes a reasonable thing for us to say to patients to give them more confidence in the treatment they're getting from us—but it may not be true. (Davies, 2010)

Carlat's straightforward admissions are likely to cause reactions, and we think they mostly speak for themselves. It's worth noting that he sometimes frames the serotonin issue as one of scientific uncertainty:

we "essentially" or "exactly" don't know how SSRI medications work, and the serotonin theory "may not be true." Such statements need to be evaluated in light of the existing literature (see Table 1). In fact, scientists have known for a long time that the serotonin theory presented by the drug companies and Carlat is not true (see Healy, 2004, 2012, 2015; Lynch, 2015). Claiming scientific uncertainty about the issue could reflect a lack of familiarity with the scientific literature, or a need to justify the use of such statements. In our opinion, neither option is flattering or desirable in an era of shared clinical decision-making. The simple alternative would be to tell patients the truth—that the pathophysiology of depression is unknown and that we have no idea how SSRIs work.

The Positive Aspects of Misinformed Thinking

On January 23, 2012, NPR Morning Edition aired "When it Comes to Depression, Serotonin Isn't the Whole Story" (Spiegel, 2012). While Carlat states that the serotonin theory "may not be true," psychiatrist Joseph Coyle makes a much clearer statement: "I don't think there's any convincing body of data that anybody has ever found that depression is associated to a significant extent with a loss of serotonin." Yet part of the segment focuses on the positive aspects of telling patients that a serotonin imbalance causes depression (see Levine, 2014). For instance, Alan Frazer, Professor of Pharmacology and Psychiatry, stated that the serotonin theory allowed patients to:

Feel better about themselves if there was this biological reason for them being depressed, some deficiency, and the drug was correcting it. They had a chemical imbalance and the drug was correcting that imbalance . . . yeah it's like, I have depression but I have a chemical imbalance, and you have hypothyroidism and you have a chemical imbalance, and my chemical imbalance just happens to affect my brain. (Spiegel, 2012)

Psychiatrist Pedro Delgado added, "When you feel that you understand it, a lot of the stress levels dramatically are reduced. So stress hormones and a lot of biological factors change."

Not surprisingly, there were many angry comments on the NPR website. Apparently, many psychiatric patients never realized they were hearing a

metaphor and not science. They didn't know that the chemical imbalance metaphor was used in an attempt to reduce stigma, or stress hormones, rather than being accurate information presented by their trusted health-care provider. Since chemical imbalance is often presented as a rationale for taking SSRIs, some such patients now understandably feel lied to by their clinicians. Levine (2014) calls this "Psychiatry's Manufacture of Consent."

The claim that presenting the chemical imbalance metaphor is in the best interests of patients needs to be considered in light of the existing empirical research. This in fact is not what the literature shows (e.g., Deacon & Baird, 2009). For instance, in a rare controlled experiment on this topic, one group of depressed students were told they had a confirmed serotonin imbalance underlying their depression, while a control group was not (Kemp, Lickel, & Deacon, 2014). The group who was told they had abnormal serotonin levels found medication more credible than psychotherapy and expected it to be more effective. They also had more pessimism about their prognosis and a lower perceived ability to regulate negative mood states, yet experienced no reduction in self-blame. These results suggest that the chemical imbalance explanation may indeed be helpful in persuading patients to take medication but that this is likely accompanied by undesirable effects. Data such as this should be a major part of the conversation regarding informed consent in psychiatry.

The Role of Journalism

Perhaps the most interesting part about both of these NPR pieces is that neither reporter questioned the experts about the ethics of telling a falsehood to patients because you think it is good for them. In contrast to how, say, a foreign-policy expert might be grilled on NPR, the tone was deferential and accepting. We would have liked both reporters to have asked the following questions: (a) Do you believe it is ethical to present a falsified scientific theory as a fact to a patient? (b) What are the possible negative effects of doing so? (c) Should the information you tell your patients be consistent with the psychiatric textbooks on your shelf? (d) How does it affect the psychiatrist-patient relationship when your patients look up serotonin imbalance on the Internet and conclude that they have been misled?

It Wasn't Us, It Was the Drug Companies

Ronald Pies is a psychiatrist at Tufts University and served as editor of the prominent trade journal *Psychiatric Times* from 2007–2010. From 2011 on, he authored several pieces on the chemical imbalance issue, which we recommend (Pies 2011a, 2011b, 2014). These are available on the web, cited frequently, and Pies is the most prominent figure in U.S. psychiatry to take up this issue publicly. Pies doesn't believe that the chemical imbalance metaphor should be attributed to psychiatry:

... opponents of psychiatry . . . mendaciously attribute the phrase ["chemical imbalance"] to psychiatrists themselves . . . And yes [it has] been vigorously promoted by some pharmaceutical companies, often to the detriment of our patient's understanding. . . . In truth, the "chemical imbalance" notion was always a kind of urban legend—never a theory seriously propounded by well informed psychiatrists. (Pies, 2011a)

We suspect that Pies had no idea how many of his fellow psychiatrists he was throwing under the metaphorical bus by making this claim. While we don't know exactly how many clinicians have told their patients they were suffering from a chemical imbalance over the last 25 years, we believe that the number is significant and consequential. Among 237 psychology students, Frances, Lysaker, and Robinson (2007) found that 46% had heard the chemical imbalance explanation from a physician. Empirical studies report use of the chemical imbalance theory by prescribers, including psychiatrists (e.g., Cohen & Hughes, 2011; Schreiber & Hartwick, 2002; see also Acker, 2013). Also, over the years, we've been in touch with many people who reported hearing "it's a chemical imbalance" from psychiatrists: people in our social circles; "consumers" at conferences; our students who work in community mental health settings; subjects in our research (Lacasse, Lietz, Hayes, Rider & Hess, in press); and people who emailed us in response to our work. And, one of the authors once worked with a talented psychiatrist and heard this explanation given weekly. If Pies is correct, that's an awful lot of uninformed clinicians.

A Bumper-Sticker Slogan to Educate Patients

In subsequent articles Pies moderates his tone and concedes that practicing psychiatrists may have used the chemical imbalance explanation at times (Pies, 2011b). He claims that it is the result of overbooked clinicians looking for quick explanations to accompany medication, perhaps to reduce self-blame on the part of patients (he acknowledges that this may backfire). He states:

My impression is that most psychiatrists who use this expression feel uncomfortable and a little embarrassed when they do so. It's kind of a bumper-sticker phase that saves time, and allows the physician to write out that prescription while feeling that the patient has been "educated." (Pies, 2011b)

To us, this sounds similar to what Carlat was reporting. Pies also notes that academic psychiatry hasn't done a great job of communicating with Primary Care Physicians (PCPs), who write most of the prescriptions for SSRIs. This might be seen as a question of priorities, because academic psychiatry in general has done a highly effective job of convincing PCPs to diagnose and treat depression with antidepressants.

Academic Psychiatry as Silent Partner in the Promotion of Chemical Imbalance

Pies admits that both he and official psychiatric associations should have done more to dispel the chemical imbalance myth (Pies, 2014). He adds that there "were sincere attempts to do just that, by several prominent psychiatrists." Unfortunately, he doesn't provide any recent examples (he does cite Shildkrauddt & Kety, 1967). It is easy to imagine that a single prominent academic psychiatrist, authoring an Op-Ed in *The New York Times*, could have set the record straight on serotonin imbalance decades ago. Yet, to our knowledge, no one did so.

We have long been concerned about how conflicts-of-interest with the pharmaceutical industry might shape the behavior (unconsciously or not) of academic psychiatrists, including the promotion of the chemical imbalance metaphor. In 2009, we wrote about misleading direct-to-consumer advertising of psychostimulants such as Adderall, where the claims were at

least as misleading as SSRI advertisements (Lacasse & Leo, 2009). Noting the lack of objections to these advertisements from within academic psychiatry, we asked, “Is it possible that the flow of money from the pharmaceutical companies to influential academic psychiatrists . . . has brought with it a certain willingness to remain silent?” We doubt Ronald Pies would find this irrationally conspiratorial, or a crazy question to ask—because we published this in *Psychiatric Times* (Editor: Ronald Pies, M.D.).

Thus, while we don’t know why Ronald Pies himself didn’t speak out on the chemical imbalance issue decades ago, readers should be aware of his past financial relationship with pharmaceutical companies. He sounds vaguely critical of the drug industry in his recent articles and never discloses any history of financial conflicts-of-interest. However, Pies has received funding from GlaxoSmithKline, Abbot Laboratories, and Janssen Pharmaceuticals—the makers of Paxil, Wellbutrin, Lamictal, Depakote, and Risperdal (Chaudron & Pies, 2003; Pies & Rogers, 2005). For years, Paxil and Wellbutrin were advertised as correcting a chemical imbalance in the brain. These three companies have recently been fined a combined \$6.7 billion for illegal marketing of their products.¹ Pies has also consulted for ApotheCom, a “Medical Communications Agency” that “provides services to support the commercialization of new products . . . [including] . . . publications planning, [and] promotional communications . . .” (Pharma Voice Marketplace, 2015). While useful context, this isn’t uncommon among academic psychiatrists, and some would say it was par for the course in the 2000s. However, in a public forum, more transparency is preferable. Pies blames the drug companies for running misleading advertisements about

chemical imbalance, belatedly admits he should have said something sooner, but fails to mention that he was paid to help them promote their products at the time the advertisements were running.

It’s important to realize that organized psychiatry doesn’t always remain silent, such as when the interests of psychiatric prescribers and pharmaceutical companies converge. In the mid-2000s, press releases endorsed by some of the most prominent psychiatrists in the United States were issued objecting to the FDA black box warning on SSRIs (e.g., American College of Neuropsychopharmacology, 2006; Healy, 2012). The APA also issued a press release defending antidepressants (APA, 2004; Healy, 2006). This was at a time when the chemical imbalance metaphor was omnipresent in direct-to-consumer advertising. While that was seen as a pressing issue to present to the public, misleading messages on chemical imbalance were not.

But We Never Promoted the Theory

Remaining silent is one thing, promoting chemical imbalance theory is another. Pies has also stated, “I am not aware of any concerted effort by academic psychiatrists, psychiatric textbooks, or official psychiatric organizations to promote a simplistic chemical imbalance hypothesis of mental illness” (2014). In the age of the Internet, it didn’t take long for MadinAmerica.com blogger Philip Hickey (2014) to make him aware of some. We added to the list by consulting Lynch (2015, Chapter 5) and searching the Internet. The resulting list (Table 2) is admittedly incomplete but sufficient to address Pies’ point.

Clearly, mainstream psychiatry (including academic psychiatry and professional organizations) has promoted the chemical imbalance theory. Comparing Table 1 and Table 2, it is apparent that there are often

two different conversations occurring (Whitaker, 2010; Whitaker & Cosgrove, 2015). One is the actual scientific discourse, as exemplified in the APA’s *Textbook of Psychiatry* (Hales, Yudofsky, & Talbott, 1999), which accurately describes the empirical status of serotonin imbalance theory 16 years ago. The other conversation is between influential psychiatrists and the public, or between psychiatrists and primary care physicians. In this second conversation, the drug company advertising line about SSRIs correcting chemical imbalances is repeated as fact by psychiatric authorities, including the APA.

The Chemical Imbalance Theory as a Little White Lie

Pies started out enthusiastically critiquing the chemical imbalance theory. We obviously believe he tried to rewrite some history along the way. But, by 2014, Pies refers to the use of the chemical imbalance metaphor as “a little white lie”² (Pies, 2014; see also Hickey, 2014). While previously psychiatrists who used this language were not well-trained, or knowledgeable, or well-informed, now they are just telling white lies—little ones.

We found this disappointing. When our physicians are educating us, we prefer they not tell us any lies, white or otherwise. Unfortunately, characterizing the chemical imbalance metaphor as a “little white lie” communicates a paternalistic, hierarchical approach that sounds suspiciously like the days of medicine that we thought we had left behind. It’s a “little white lie” if you’re a psychiatrist; if you’re a confused, vulnerable depressed person who agrees to take an SSRI after hearing it, you might not consider it so little. After all, if your trusted physician tells you that you have a chemical imbalance in your brain that can be corrected with medication, not doing so sounds foolish, if not scary (Lacasse, 2005). How many patients with reservations about SSRIs have agreed to take medication after being told this “little white lie”?

Discussion

In the last decade, widespread claims of chemical imbalance in depression have essentially been withdrawn by both the profession of psychiatry and the pharmaceutical industry. We believe the profession of psychiatry should be strongly critiqued for withdrawing the serotonin theory belatedly, long after the science was in, and for not speaking up while drug advertisements

¹We want to be clear that we are not accusing Ronald Pies of anything. Conflicts-of-interest are routine in academic psychiatry and many of the major pharmaceutical companies have been fined in the recent past. We do believe that readers deserve to know of his past financial relationships with the drug companies that promoted their products as correcting a chemical imbalance. The details of these financial relationships are not publicly available.

²Pies’ (2014) original quote reads as follows: “In the narrative of the antipsychiatry movement, a monolithic entity called ‘Psychiatry’ has deliberately misled the public as to the causes of mental illness, by failing to debunk the chemical imbalance hypothesis. Indeed, this narrative insists that by promoting this little white lie, psychiatry betrayed the public trust and made it seem as if psychiatrists had magic bullets for psychiatric disorders.” It’s important to realize that “little white lies” is Pie’s characterization of chemical imbalance, not how it is presented in the critical narrative. Writers like Whitaker (2010) vigorously critique the idea of chemical imbalance exactly because they do not see it as a “little white lie.”

Table 1. Evidence the Chemical Imbalance Theory of Depression Is Not Valid: Selected Quotations

Quote	Citation
“By 1970...[biochemist and Nobel Prize Winner Julius] Axelrod had concluded that, whatever was wrong in depression, it was not lowered serotonin.”	Healy, 2004, p. 12
“I spent the first several years of my career doing full-time research on brain serotonin metabolism, but I never saw any convincing research that any psychiatric disorder, including depression, results from a deficiency of brain serotonin” (Psychiatrist David Burns, who conducted award-winning serotonin research in the 1970s).	Lacasse & Gomory, 2003, p. 393
“Tianeptine is an interesting compound with antidepressant activity thought to be related to increased rather than decreased 5HT [serotonin] uptake” [meaning, in 1989 it was known to be an antidepressant that depletes, not increases, serotonin].	Ives & Heym, 1989, p. 22
“The simplistic idea of the ‘5-HT [serotonin]’ neurone does not bear any relation to reality” (John Evenden, Astra pharmaceutical company research scientist, 1990).	Shorter, 2009, p. 204
“In the 1990s...No one knew if SSRIs raised or lowered serotonin levels; they still don’t know...There was no evidence that treatment corrected anything.”	Healy, 2015
“...Patients have been diagnosed with ‘chemical imbalances’ despite the fact that no test exists to support such a claim, and there is no real conception of what a correct chemical imbalance would look like...Yet conclusions such as ‘depression is a biochemical imbalance’ are created out of nothing more than semantics and the wishful thinking of scientists/psychiatrists and a public that will believe anything now that has the stamp of approval of medical science” (Psychiatrist David Kaiser of Northwestern University Hospital, 1996).	Kaiser, 1996; Lynch, 2015, pp. 31-32.
“Although it is often stated with great confidence that depressed people have a serotonin or norepinephrine deficiency, the evidence actually contradicts these claims” (Neuroscientist Elliot Valenstein).	Valenstein, 1998, p. 100
“The monoamine hypothesis...holds that monoamines...such as... [serotonin]...are deficient in depression and that the action of antidepressants depends on increasing the synaptic availability of these monoamines...However, inferring neurotransmitter pathophysiology from...[SSRIs]...is similar to concluding that because aspirin causes gastrointestinal bleeding, headaches are caused by too much blood...Additional experience has not confirmed the monoamine depletion hypothesis.” (American Psychiatric Association <i>Textbook of Psychiatry</i> , 1999).	Dubvosky & Buzan, 1999, p. 516
“A serotonin deficiency for depression has not been found” (Psychiatrist Joseph Glenmullen, Clinical Instructor of Psychiatry at Harvard Medical School).	Glenmullen, 2000, p. 197
“...I wrote that Prozac was no more, and perhaps less, effective in treating major depression than prior medications...I argued that the theories of brain functioning that led to the development of Prozac must be wrong or incomplete” (Brown University Psychiatrist Peter Kramer, author of <i>Listening to Prozac</i>).	Kramer, 2002
“[We must] abandon the simplistic hypotheses of there being either an abnormally high or abnormally low function of a given neurotransmitter” (Avid Carlson, Nobel Prize winner for his work on the neurotransmitter dopamine, 2002).	CINP Meeting with the Nobels (2003); Shorter, 2009, p. 204
“Indeed, no abnormality of serotonin in depression has ever been demonstrated” (Psychiatrist and historian David Healy in 2004).	Healy, 2004, p. 12

Table 2. Promotion of the Chemical Imbalance Theory of Depression as Valid: Selected Quotations

Quote	Source	Citation
“Celexa helps to restore the brain’s chemical balance by increasing the supply of a chemical messenger in the brain called serotonin.”	Celexa website, 2005	Lacasse & Leo, 2005
“Antidepressants may be prescribed to correct imbalances in the levels of chemicals in the brain.”	<i>Let’s Talk Facts About Depression</i> , a patient information leaflet distributed by APA	American Psychiatric Association, 2005, p. 2
“Antidepressants...have no effect on normal mood. They restore brain chemistry to normal.”	Nada Stotland, president of the American Psychiatric Association, 2007-2008	Stotland, 2001, p. 65
“[antidepressants work] only if there was a chemical imbalance in the brain that needed fixing”	Donald Klein, psychiatrist and psychopharmacologist	Talan, 1997
“While the patient may require a somatic therapy to correct the underlying chemical imbalance, he may also need psychotherapy...”	Nancy Andreason, psychiatrist and author of <i>The Broken Brain</i>	Andreason, 1985, p. 258
“...some depressed patients who have abnormally low levels of serotonin respond to SSRIs...”	Psychiatrist Richard Friedman in <i>The New York Times</i>	Friedman, 2007
“There is truly a real deficiency of serotonin in depressed patients.”	Psychiatrist Charles Nemeroff	Nemeroff, 2007
“The physician should stress that depression is a highly treatable medical illness caused by a chemical imbalance.”	MacArthur Foundation Depression Education Program for Primary Care Physicians	Cole, Raju, Barrett, Gerrity, & Dietrich, 2000, p. 340
“Patients with neurotransmitter dysregulation may have an imbalance of serotonin and norepinephrine...duloxetine [Cymbalta] may aid in correcting the imbalance of serotonin and norepinephrine neurotransmission in the brain.”	Madkur Trivedi, psychiatrist at University of Texas Southwest Medical School, in <i>The Primary Care Companion of the Journal of Clinical Psychiatry</i>	Trivedi, 2004, p. 13
“Restoring serotonin’s imbalances not only helps brighten mood and restore normal sleeping and eating patterns, but it also seems to promote a sense of well-being.”	Michael Thase, psychiatrist and psychopharmacology researcher at the University of Pennsylvania, and science writer Susan Lang	Thase & Lang, 2004, p. 106
“We now know that mental illnesses—such as depression or schizophrenia—are not ‘moral weaknesses’ or imagined but real diseases caused by abnormalities of brain structure and imbalances in chemicals of the brain...medications and other treatments can correct these imbalances. Talk therapy can directly improve brain functioning.”	Richard Harding, president of the American Psychiatric Association, 2000-2001	Harding, 2001, p. 66
“At some time in the course of their illness, most patients and families need some explanation of what has happened and why. Sometimes the explanation is as simplistic as ‘a chemical imbalance’...”	Robert Freedman, psychiatrist at the University of Colorado	Freedman, 2003, as cited by Hickey, 2014

(and many clinicians) were convincing the American public that the chemical imbalance theory was legitimate. We previously argued that the propagation of misleading advertising “is only possible in the absence of vigorous government regulation . . . or outcry from professional associations” (Lacasse & Leo, 2006). That outcry never came, and these issues weren’t addressed publicly until the patents for most blockbuster SSRIs had expired, and Big Pharma moved onto mood stabilizers and atypical antipsychotics. While we are hesitant to overemphasize conflicts-of-interest as an explanation for what has occurred, we can’t help but notice that the silence of psychiatry regarding chemical imbalance only ended when the profits had been extracted from the SSRI marketplace.

The new narrative will apparently be that psychiatrists recently discovered that the chemical imbalance theory was incorrect. Psychiatric researchers are changing their mind based on data, so the story goes, and it just took a while to let the public know. We believe this is empirically incorrect (Table 1; see Healy, 2015; Shorter, 2015). The idea that the withdrawal of the chemical imbalance theory was caused by recent data should be rejected.

As the theory has been withdrawn and a dialogue has taken place, many mental health clients have reacted negatively to the news that there was never any reason to believe that depression was caused by a serotonin imbalance (Healy, 2015). Many mental health clients find it unacceptable, and perhaps a violation of ethical informed consent, for clinicians to give patients metaphorical explanations for their mental health problems and promote them as scientific truth. Patients who start an SSRI because they have been told it will correct their chemical imbalance, that it is like thyroid medication for hypothyroidism, are likely to eventually conclude that they have simply not been told the truth. This obviously creates awkward dynamics in patient-prescriber relationships and also represents a potential public relations problem for the profession of psychiatry.

Previously, we argued that misleading consumer advertisements for SSRIs should end (Lacasse & Leo, 2005). A decade later, the serotonin theory of depression is acknowledged to be dead, and most SSRI advertising campaigns are now part of history. We look forward to a day when telling depressed patients they have a serotonin imbalance is as anachronistic as the miserable ovoid creature from the Zoloft advertisements of the past, and we believe that

day will come sooner than some might suppose. We encourage our colleagues in organized psychiatry to work towards this end by improving medical education and ongoing training, by endorsing shared decision-making, and by ensuring that informed consent is based on the scientific literature.

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The Myths and Realities of Drug Treatment for Mental Disorders

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PSYCHIATRIC DRUGS ARE CURRENTLY assumed to exert their beneficial effects by acting on the underlying basis of an abnormal brain process, or “disease.” Thus, antipsychotics are believed to help reverse the pathology that produces psychotic symptoms, or schizophrenia; antidepressants are believed to act on the biological processes that produce symptoms of depression; mood stabilizers are thought to help normalize the processes that produce abnormal mood swings; and so on. Although the general assumptions involved in understanding drug action are rarely articulated, modern psychiatric thought is premised on the idea that psychiatric drugs work by helping to normalize an underlying brain disorder. Drugs work by acting on specific abnormal brain processes or disease states. In popular language, they are thought to help rectify a “chemical imbalance.”

This view is promoted most obviously by the pharmaceutical industry, whose websites frequently refer to the idea that psychiatric drugs work by “balancing the chemicals naturally found in the brain” (Eli Lilly, 2006). Literature produced by psychiatric organizations like the Royal College of Psychiatrists convey the same message, albeit more tentatively. The College leaflet on antidepressants, for example, acknowledges that “we don’t know for certain” but goes on to suggest, “we think that antidepressants work by increasing the activity of certain chemicals that work in our brains called neurotransmitters . . . the chemicals

most involved in depression are thought to be serotonin and noradrenalin” (Royal College of Psychiatrists, 2009).

Over the last few years I have been challenging these assumptions about how psychiatric drugs work (Moncrieff, 2008). I have suggested that there is an alternative explanation for the effects of drugs on psychiatric disorders. Psychiatric drugs are psychoactive substances, which induce characteristic artificial mental and physical states in anyone who ingests them. These drug-induced effects can influence people’s behavior and it is inevitable that they will have an impact on the problems that are classified as psychiatric symptoms.

In order to clarify and contrast these two different ways of thinking about what psychiatric drugs do, I have formulated two “models” of drug action (see Table 1).

I have called the conventional model the “disease-centered” model of drug action, in reference to the core idea that drugs act on disease processes, although in psychiatry these processes are hypothetical, since none have been clearly demonstrated. The disease-centered model assumes that drugs exert their relevant effects only in people with an abnormal nervous system. The effects of drugs can therefore be meaningfully divided into the therapeutic effects, which are the effects on the disease process, and other effects, which are referred to as “side effects.” The therapeutic effects will only be apparent in people who have the underlying pathology.

The drug-centered model, by contrast, suggests that rather than correcting underlying abnormal brain states or diseases, psychiatric drugs produce abnormal or artificial drug-induced states. Drugs do not restore normal bodily function; drugs disturb normal bodily function. There is no essential distinction, according to this view, between drugs used for psychiatric treatment and recreational psychoactive drugs like alcohol and cocaine. All psychoactive drugs produce altered physical and mental states that can influence the way people think, feel, and act, although different sorts of substances have different sorts of effects. The drug-centered model suggests that it is these psychoactive effects that explain the changes that are sometimes seen when drugs are given for psychiatric conditions. Drugs like benzodiazepines and alcohol, for example, reduce arousal and induce a usually pleasant state of calmness and relaxation. This state may be experienced as a pleasant relief for someone who is intensely anxious or agitated. But taking a drug like this does not return the individual to normal. It is simply that the drug-induced state may be preferable to a state of intense anxiety.

According to a drug-centered model, therefore, psychiatric drugs produce global neurophysiological states characterized by a range of physiological effects, which are experienced by everyone who takes these drugs, not just by people with underlying psychiatric or neurological disorders. Although some of these effects may be useful, while others may not, dividing the components of these states into therapeutic effects and side effects obscures the full nature of the effects drugs produce. Understanding these global effects is necessary to assess when drug treatment might be useful, and to properly weigh up the costs and benefits involved.

The disease-centered model has been imported from general medicine, where most modern drugs are correctly understood as working to reverse the biological processes that produce a disease, or the symptoms of a disease. Most medical treatments do not in fact reverse the original disease process, but act on the physiological processes that produce the symptoms of a disease. Thus, beta-blockers help reverse airways obstruction in asthma and chemotherapeutic agents counteract the abnormal cell division that occurs in cancer. Even most analgaesics work in a disease-centered manner by acting on the physiological processes that produce pain. Opiates, however, may have dual actions.

Table 1. Disease-Centered vs. Drug-Centered Models of Drug Action

Disease-centered model	Drug-centered model
Drugs correct an abnormal brain state	Drugs create an altered physiological state
Drugs as medical treatments	Drugs as psychoactive substances
Beneficial effects arise from the drugs’ action on the underlying disease process	Beneficial effects are a consequence of being in an altered, drug-induced state
Therapeutic effects can be distinguished from side effects	Some effects may be more useful than others, but there is no categorical distinction between therapeutic effects and side effects

They reduce pain directly by inhibiting the conduction of pain stimuli, but they also induce an artificial, drug-induced state of emotional indifference and detachment, which may lessen the impact of pain.

Evolution of the Disease-Centered Model

Prior to the 1950s, there was little interest in drug treatment for mental disorders, and available drugs were understood to act as either sedatives or stimulants, according to a crude drug-centered model of drug action. From the 1950s new drugs, such as chlorpromazine (now classed as an antipsychotic) and iproniazid (now thought of as an early antidepressant), inspired great interest and enthusiasm. However, they too were initially understood according to a drug-centered model (Moncrieff, 2008). Pierre Deniker, for example, one of the early pioneers of modern psychiatric drug treatment, felt that the useful effects of chlorpromazine and drugs like it were attributable to their ability to induce an abnormal neurological state that resembled Parkinson's disease. This state was characterized by what he referred to as "psychomotor indifference," a state that suppressed or replaced psychotic symptoms and other preoccupations (Deniker, 1960).

During the course of the 1950s and 1960s, thinking about the nature of drug action changed significantly, although without any explicit discussion or debate. The principal drugs used in psychiatry came to be seen as specific, disease-centered treatments for particular psychiatric disorders. This transformation was not based on an emerging evidence base for the specificity of psychiatric drugs. There was little evidence available that could distinguish between the drug-centered and disease-centered models of drug action at that time. It appears that the change came about because of the desire of the psychiatric profession to have treatments that mimicked physical medicine. This desire was supported by the pharmaceutical industry, in an attempt to mark out certain products as having disease-specific properties, and by the state, in its endorsement of the medical management of psychiatric disorders (Moncrieff, 2008).

Evidence for the Disease-Centered Model

Placebo-controlled trials do not distinguish whether drugs have a disease-cen-

tered or drug-centered action—they only indicate that drugs have different effects from an inert substance: the placebo. The following areas, which might provide evidence of disease-specificity, also fail to provide support.

Underlying Pathology

The best evidence for the disease-centered model would be if the action of a drug could be deduced from knowledge of the underlying pathology of a particular disorder. There is no known biological mechanism underlying any psychiatric disorder, however. There are theories that propose possible biochemical mechanisms for some psychiatric disorders, but the evidence for these theories is inconclusive. For many decades now, for example, it has been suggested that schizophrenia and psychosis may be caused by abnormal levels of the neurotransmitter dopamine. The "dopamine hypothesis of schizophrenia" was formulated after researchers observed that the actions of haloperidol correlated closely with its potency in blocking the dopamine D2 receptor. Therefore, the theory was based from the beginning on the assumption that the drugs act in a disease-centered way, and that the origins of a disorder can therefore be deduced to be the opposite to the effects of the drugs used to treat it.

Although many antipsychotics strongly block dopamine receptors, they all affect a range of other neurochemical substances, and some, including the highly effective drug clozapine, appear to have weaker dopamine blocking action, which does not correlate with their therapeutic potency (Yilmaz et al., 2012). Research on dopamine activity in people with schizophrenia or psychosis is also inconsistent. Studies of dopamine content of post-mortem brains and dopamine metabolites are negative, for example. The increased concentration of dopamine D2 receptors, which was identified in brains of people with schizophrenia, transpired to be due to the effects of drug treatment. Some recent studies report that indirect measures of dopamine activity are abnormal in people with acute psychosis. However, we know that dopamine is implicated in a range of functions including arousal, movement, and stress that will confound its relations with any specific psychiatric disorder (Moncrieff, 2009). Moreover, the number of people who had not been exposed to antipsychotic drugs, which affect dopamine function, was small.

The evidence on whether depression is caused by abnormalities of brain chemi-

cals, including serotonin and noradrenalin, is even more contradictory. It is now acknowledged that chemical hypotheses of depression lack evidential support (Lacasse & Leo, 2005; Moncrieff & Cohen, 2006).

Comparison With Nonspecific Drugs

Although drugs may exert useful effects through a drug-centered mechanism, a drug that is believed to have disease-specific effects should, by definition, be more effective than a drug that produces only nonspecific, drug-induced effects. Therefore, a drug considered to be an "antidepressant" should be superior to drugs that are not thought to act on the biological basis of depression, and drugs that exert effects on the presumed basis of psychotic symptoms should be superior to drugs that do not act on these processes. A drug centered model would also predict that some drugs may be superior to others, but it would seem to be a minimum requirement of a disease-centered view that specific drugs are superior to nonspecific drugs.

The comparative studies that exist, however, do not strongly support the idea of specificity. Numerous drugs that are not normally considered to be antidepressants, for example, have been found to be superior to placebo in randomized trials, or equivalent to standard antidepressants. The list includes substances with such diverse actions as antipsychotics, benzodiazepines, and stimulants (Moncrieff, 2008). Moreover, antidepressants themselves come from a wide variety of chemical classes, and cause a huge array of physiological effects, such that it is difficult to believe that there could be any common underlying pathway for their action.

There is also little evidence that so-called antipsychotic drugs are superior to other sorts of sedatives, even despite the distinctive quality of their sedative effects. Comparisons between antipsychotics and benzodiazepine drugs (e.g., Valium) have given mixed results, for example, with many finding benzodiazepines to be equal or superior (Wolkowitz & Pickar, 1991). Anecdotally, opiates are said to have antipsychotic properties, possibly due to their ability to induce a state of emotional detachment. A randomized controlled trial published in 1960 found no difference between opium and chlorpromazine for the treatment of acute psychosis (Abse, Dahlstrom, & Tolley, 1960). So overall, evidence that antipsychotics are more effective than other sedatives is inconclusive, and their superiority to sedatives with similar emotion-dampening effects has not

been demonstrated. Comparative studies have also not found lithium to be superior to other sedative drugs such as antipsychotics and benzodiazepines for the treatment of acute mania or other acute mood states (Braden, et al., 1982; Chouinard, 1988).

Psychiatric Treatment Based on a Drug-Centered Model

Since there is little evidence to support a disease-centered model of drug action, I suggest that the drug-centered model of drug action should be taken more seriously. However, a drug-centered approach to the use of psychiatric drugs fundamentally challenges much current psychiatric knowledge and practice. Instead of prescribing treatments for particular conditions, drug treatment would be offered in order to produce an altered mental state, which people may or may not find helpful, depending on the nature of their situation or problem. In order to use drugs carefully and wisely in this way, we need to have comprehensive information about the sort of state that different drugs induce, and the full consequences of taking them over short and longer periods. Only then do individuals have any chance of discerning whether taking a particular drug for a particular problem will do more good than harm.

Unfortunately, the disease-centered model has meant that psychopharmacology research has focused on effects on presumed disease mechanisms, like dopamine or serotonin receptor levels, and ignored the many other effects that drugs have. There is a particular paucity of research on the subjective effects of psychiatric drugs and on their long-term consequences. Using data from the few existing published volunteer studies, coupled with patient accounts, we can deduce something about the states produced by ingesting psychiatric drugs of different sorts, although much still needs to be explored and clarified.

“Antipsychotics”

Early investigators described how the first antipsychotic drugs induced a state of restriction of movement and thought, similar to Parkinson’s disease. Subsequent accounts by patients and volunteers describe a state of the mental and physical slowness, in which there is a loss of interest in everyday activities, a blunting of emotional responses and a loss of initiative. According to a drug-centered model, it is these general suppressant effects that

reduce psychotic experiences, as well as other states of physical and mental agitation and overarousal.

Some of the “atypical” antipsychotics appear to produce a slightly different sort of state, characterized by strong sedation associated with metabolic disruption resulting in substantial weight gain. People describe feelings of indifference and apathy, coupled with an irresistible desire to eat (Moncrieff, Cohen, & Mason, 2009). Thus it appears the neurological effects induced by the typical antipsychotics, and the metabolic effects produced by olanzapine and clozapine, appear to be integral to the mental effects they produce.

Antidepressants

As previously described, antidepressants come from a wide variety of chemical classes, and the drug-induced state they produce depends on their chemical properties. Tricyclic antidepressants are chemically related to early antipsychotic drugs like chlorpromazine and at the doses usually prescribed, the main mental effects appear to be extreme sedation and cognitive impairment and slowing (Dumont et al., 2005).

The psychoactive effects of SSRIs are more subtle. They also induce lethargy and drowsiness and chronic apathy is reported with long-term use. They produce a state of emotional indifference or detachment, with individuals reporting that they cannot cry and feel emotionally numb. This is associated with loss of libido and sexual impairment. Some people experience a mild but unpleasant arousal state characterized by agitation and tension, which may be coupled with emotional instability and suicidal impulses (Goldsmith & Moncrieff, 2011).

Other Psychiatric Drugs

The psychoactive effects of stimulants, like amphetamine, and benzodiazepines are well-known. Drugs prescribed for bipolar disorder consist of a variety of substances, including lithium, some antipsychotics and some anticonvulsant drugs used for epilepsy. All have highly sedative properties. Slow thinking, reduced creativity, and unpleasant feelings are described by volunteers on lithium (Judd et al., 1977), but the characteristic subjective effects of anticonvulsants are not well-described.

A New Psychiatric Practice

The drug-centered model fundamentally changes assumptions about the purpose and benefits of psychiatric medica-

tion. The disease-centered model assumes that taking drugs is likely to be beneficial, because they help correct a (hypothetical) underlying biological abnormality or disease. There is therefore a presumption of benefit built into the disease-centered model, although this may be outweighed by adverse effects. The drug-centered model, by contrast, demands that we start by presuming that drugs may be harmful. Psychiatric drugs are drugs—that is, chemical substances foreign to the human body, which produce an artificial or altered bodily state. Drugs may be directly toxic to the body, and the body may react to the presence of a drug in ways that are unpredictable and can be harmful and irreversible. Tardive dyskinesia, for example, the involuntary movements that occur during and after long-term treatment with antipsychotics, is thought to be caused by overcompensation of the dopamine system reacting to the presence of dopamine-blocking drugs.

Taking drug treatment is always a question of weighing up potential risks and benefits, but the drug-centered model makes it clear that the benefits have to be clear and well defined to balance out the intrinsic risks of taking something that disrupts rather than restores the normal functioning of the body. It is also important to emphasize that the effects of a drug may change over time in ways that have not been well researched, and are not always predictable.

A Drug-Centered Approach to the Treatment of Depression

A drug-centered approach suggests that drugs have a limited role in the treatment of depression. We know that many drugs, including alcohol, amphetamine, and Ecstasy, can induce a temporary state of euphoria, but there is no evidence that any drug helps return depressed people to “normal.” Drugs with sedative effects, such as benzodiazepines or “Z” drugs (e.g., zopiclone), may be useful for people with agitation or insomnia in the short-term, although they have to be prescribed with care to avoid dependence.

It is likely that some drugs, such as antipsychotics and SSRIs, will suppress or blunt feelings of depression, along with other emotions. There may be times when people are so distressed they desire this effect. However, most people take antidepressants because they believe the drugs are helping to reverse an underlying biochemical imbalance that is producing their symptoms, and thereby helping to return

them to a normal state. It is difficult to know how many people would want to take antidepressants if they were told that the drugs would merely suppress their emotions, along with their libido. Although modern antidepressants are not seriously toxic drugs, they do have adverse effects, including occasionally irreversible sexual impairment and protracted withdrawal effects after long-term use (Farnsworth & Dinsmore, 2009). Moreover, the physiological and psychological effects of taking antidepressants may prove a hindrance to the resolution of the personal and social difficulties that have, in most cases, led to depression in the first place.

A Drug-Centered Approach to the Treatment of Psychosis and Bipolar Disorder

The term *psychosis* covers a myriad of situations, but most people with acute psychosis are preoccupied with abnormal or exaggerated ideas and many experience strange mental phenomena such as hallucinations. People with acute psychosis, especially mania, are often physically or mentally overaroused. In such situations, antipsychotic drugs may reduce abnormal mental experiences and the emotional response to these experiences by suppressing mental and emotional activity in general. Indeed, research has shown that antipsychotic treatment does not generally remove abnormal thoughts altogether, but it makes people less concerned by them (Mizrahi et al., 2005). The sedative effects of antipsychotics or other drugs may be also employed in order to calm someone who is overaroused or disturbed. Whether it ultimately makes sense to use drugs to suppress the effects of a psychotic episode, however, depends on many further considerations. Existing studies show that antipsychotics are superior to placebo in reducing psychotic symptoms in people with schizophrenia, although the difference is only 18% (Leucht et al., 2008). Moreover, we know that some people recover spontaneously, or with social support, and hence drug treatment is not necessary in everyone (Bola & Mosher, 2003).

Antipsychotic drugs have serious and life-threatening "side effects," especially when used over long periods (including the neurological disorder tardive dyskinesia, brain shrinkage, diabetes and heart disease) and may impair functioning as they do in volunteers. Although long-term treatment is typically recommended for most people with psychotic disorders, studies are flawed and follow-up generally too short. A recent

7-year evaluation of a randomized trial found that people randomized to maintenance antipsychotic therapy were less likely to make a full social recovery than those randomized to have medication gradually withdrawn. Relapses, which were more common among those in the discontinuation group initially, evened out over time (Wunderink et al., 2013). Antipsychotics may effectively suppress psychotic symptoms, but are unlikely to benefit everyone in the long term.

Alongside traditional psychotic conditions, increasing numbers of people are identifying themselves as having bipolar disorder, and seeking pharmacological treatment (Moncrieff, 2014). What is on offer is an array of sedative substances, including some antipsychotics, anticonvulsants, and lithium, all of which reduce arousal and are likely to suppress emotions. It is likely that some people are seeking just such effects, although we have no evidence on whether or not these effects are actually helpful. None of the commonly used agents have been tested for the sort of people they are now being prescribed to, however, and given the serious adverse effects they induce, it seems unlikely they will do more good than harm.

Implications

It seems intuitive that if patients were presented with a drug-centered understanding of psychiatric drugs, and presented with information about the drug-induced effects, long-term consequences and limitations of these drugs, they would be less willing to take them. The drug-centered model could therefore result in a reduction of unnecessary and harmful use of psychiatric medications. However, we live in a society that encourages people to believe there is a quick fix to all of life's problems, hence the appeal of the medical model of mental distress and of seemingly medical solutions like drugs. Abandoning the disease-centered model of drug action raises profound questions about the idea that mental illness is a "disease, just like any other," however. Those defending the medical model of mental illness usually start by citing the evidence that drug treatment "works." However, this only constitutes evidence that mental illness is a bodily condition if it is assumed that drugs can only "work" by acting on an underlying disease process. If the alternative drug-centered model of drug action is accepted, then the effects of drugs in people with emotional and behavioral problems is entirely pre-

dictable, without having to conjure up a phantom underlying disease. It seems likely that the success of the disease-centered model owes more to the wishful thinking of the psychiatric profession, and its desire to consider its subject matter as exactly the same as other medical problems. Understanding the use of drugs according to a drug-centered model would free people from the need to view themselves as biologically flawed in order to receive drug treatment, while simultaneously highlighting the limitations of such treatment.

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Why Neurobiological Models Can't Contain Mental Disorder and Addiction

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The Contemporary Rush to Reductionism in Mental Disorders

In 1981, I wrote "Reductionism in the Psychology of the Eighties" for *American Psychologist*. Here are two quotes from that text worth considering 35 years later. First, from neuropsychiatrist Richard Restak (1977):

[I]t's hard to leave out the exclamation points when you are talking about a veritable philosopher's stone—a group of substances [the endorphins] that hold out the promise of *alleviating, or even eliminating*, such age-old medical bugaboos as pain, drug addiction, and, among other mental illnesses, schizophrenia. (emphasis added)

Restak is a physician. But one of the leading figures in psychology of that era, Norman Garmezy, wrote for the 1975 American Psychological Association Master Lecture series four decades ago:

We stand on the threshold of advances in the biological sciences so relevant to psychopathology that one can look forward *in the decades ahead to an ultimate resolution of the major psychotic disorders that have plagued mankind for centuries*. (emphasis added)

(Need I note that neither of these professionals has/had ever done neuroscientific research? Garmezy's specialty, resilience, would seemingly resist reductionism.)

We might fairly ask at this point, "How are we doing?" In fact, as former *New England Journal of Medicine* editor Marcia Angell (2011) asserted (based primarily on Whitaker, 2010) in the *New York Review of Books*, we are experiencing an epidemic of mental disorder, one tracing back to exactly the period in which I was writing (when DSM-III [APA, 1980] was published). Not only have entirely new categories of mental disorders been identified (e.g., ADD) and then proliferated, but formerly rare diagnoses (e.g., bipolar disorder) have exploded in their incidence, along with virtually every other type of mental disorder (e.g., depression). And,

per Restak, there is addiction, which I will discuss separately.

More than being misguided, Restak and Garmezy were completely wrongheaded; their predictions were diametrically opposite of what has actually occurred. But what's most interesting is that they and others who have taken these positions—and continue to do so—feel no need to apologize for such miscalculations. Indeed, the same predictions have been made continuously in the period since then and are frequently today. They are welcomed as wholeheartedly by scientists, the public, the media—and, seemingly, psychology—as they were in the 1970s. As Deacon (2013b) observes in "The United States of the Biomedical Model," "It is difficult to overstate the ubiquity and influence of the biomedical model that provides the foundation for psychiatric diagnosis and treatment in the United States."

Objections and Alternatives to the Biopsychiatric Revolution

But this isn't working. In fact, the only potential result of this headlong rush to reductionism is to exacerbate our emotional and addictive vulnerability, both culturally and individually. Here are six progressively more fundamental relationships between our reductive views and approaches and the epidemic of mental disorder (DSM, of course, refers to "mental disorders," including substance use disorders, formerly addiction).

1. The view that we have erroneously departed from the true biological bases for mental disorders

In this view, we are casting the mental disorder net too broadly, and redefining normal behavior as diseased. Allen Frances (2013), who was the Chair of the DSM-IV Task Force, is perhaps the main proponent of this viewpoint and of the idea that Big Pharma is behind this expansion. But Frances is a true believer in biological causation, and simply feels we have departed from this gold standard in defining mental disorder. This is a variation on the view of the head of the National Institute of Mental Health, Thomas Insel, that DSM-5 fails

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because it is too behavioral, whereas we need “to reshape the direction of psychiatric research to focus on biology, genetics and neuroscience” (Belluck & Carey, 2013; cf. Insel, 2010).

This view pervades contemporary thinking, with the result that “the biomedical model era has been characterized by a broad lack of clinical innovation and poor mental health outcomes” (Deacon, 2013a). As Belluck and Carey note: “Basic research into the biology of mental disorders and treatment has stalled, . . . confounded by the labyrinth of the brain. Decades of spending on neuroscience have taught scientists mostly what they do not know, undermining some of their most elemental assumptions.” This confusion follows the disintegration of optimism about finding the genetics of mental disorder through the massive, decade-long Human Genome Project, completed in 2003, which has led to no diagnoses, treatments, or certainly answers for mental disorders (Peele, 2013a). A coordinator for a multisite, big-data analysis of the genetic basis of the principal mental disorders declared, “these [individual] genetic associations individually can account for only a small amount of risk for mental illness, making them insufficient for predictive or diagnostic usefulness by themselves” (NIMH, 2013).

So, we see, the failures to find reductive causes and cures for mental disorders lead to continuing speculation, further and further removed from human experience, about their biological sources. The focus is now on the brain impulses underlying behavior, thought, and mental disorder, as represented by the new massive successor to the Human Genome Project—the BRAIN Initiative (Peele, 2013c).

2. Drug treatments hurt our brains

The most severe critics of the bio-diagnosis and treatment of mental disorder—i.e., Angell and the principal book she reviews for the *New York Review of Books*, and from which the title of her article is derived, Robert Whitaker’s *Anatomy of an Epidemic*—argue persuasively that psychiatric medications damage the brain. Whitaker (2010) shows that the introduction of new psychiatric medications never reduces the incidence of the disorders being treated, but rather does the opposite, and that the benefits from these medications for individuals taper over time, and wash out entirely, even reverse, when the individual chooses or is forced to quit the medication. One reason for this, according to Angell, is that “the use of antipsychotic

drugs is associated with shrinkage of the brain, and that effect is directly related to the dose and duration of treatment.” The drugs themselves, Angell and Whitaker argue, exacerbate mental disorder through their impact on the brain. While data support their position, this argument reinforces the idea that mental disorder can be described solely through physical manifestations of the brain (although both Angell and Whitaker would undoubtedly reject this idea).

3. Reductive thinking is stigmatizing and antitherapeutic

A less reductive, more experiential view of how biological psychiatry hurts mentally ill people is that this view reduces therapeutic empathy and increases stigma for the mentally ill. Lebowitz and Ahn (2014; p. 17786) empirically investigated this dynamic, finding that:

biological explanations evoked significantly less empathy. These results are consistent with other research and theory that has suggested that biological accounts of psychopathology can exacerbate perceptions of patients as abnormal, distinct from the rest of the population, meriting social exclusion, and even less than fully human. . . . This is alarming because clinicians’ empathy is important for the therapeutic alliance between mental health providers and patients and significantly predicts positive clinical outcomes.

4. How we think as a culture causes mental disorder and addiction

Arthur Kleinman’s (1991) work is the missing ingredient in global criticisms of biopsychiatry. Humans think about themselves in the categories provided by their cultures. We are incapable, for the most part, of questioning our social and cultural assumptions. These categories are for us simply realities, virtually as real as gravity and the daily appearance of the sun and the moon. As a result, people internalize cultural memes: we conform our behavior, even our emotions, to them (Kirsch, 1999). That is, we examine our behavior and feelings and put ourselves into the cognitive categories available to us. This is a subtle process that operates in addition to how our agency, or resilience, or hope for recovery, or ability to control our behavior exacerbates mental disorders (cf. Deacon & Baird, 2009). For instance, simply believing in the disease theory of addiction makes people more likely to relapse (Heather,

Winton, & Rollnick, 1982; Miller et al., 1996).

5. Our cultural investment in reductive treatments lessens our ability to address the actual cultural, social, and human sources of mental disorder and addiction

Keith Humphreys, an influential community psychologist in the addiction field, strongly advocates for the curative impact of broad environmental factors (White, 2011): “what all three of those great thinkers (Rudolph Moos, et al.) make clear is that in the long-term, most people are made better by the broader world and not by short-term treatments.” In response, his interviewer, Bill White, notes: “In Philadelphia, we have introduced the concept of community recovery—the idea that whole communities can be wounded by a critical mass of alcohol and other drug problems and that a community-level healing and recovery process may be required to restore the health of individuals, families, neighborhoods, and the community as a whole” (see White & Evans, 2014).

Humphreys and White both correctly indicate that addictive (and mental health) problems are best addressed, societally and individually, by enhancing societal and community resources. And, yet, they are among the most forceful defenders of Alcoholics Anonymous, 12-step, and disease-based folk treatments. In order to maintain these contradictory positions, they must ignore that *infusing our culture and individuals with the “addiction-as-disease” meme depletes the attention and money devoted to the very community resources (e.g., housing, education, individual case management) that support recovery from both mental disorder and addiction*. Instead, the disease concept fuels investment in expensive but ultimately ineffectual medical treatments. Imagine, for instance, removing someone from a deprived environment to a residential treatment site for some period and then repositing him or her in that same environment without assisting the person to find a way of making a living or a stable home. Humphreys and Rappaport (1993) illuminated how this process began during the Reagan and Bush administrations as the federal government rebudgeted basic funding from direct social services to funding for treatment. *This is the American version of anti-community psychiatry*; it is a path to mental health disaster from which we seemingly cannot depart.

6. Belief that one is infested with a disease depletes the resilience and self-efficacy that are the best guards against addiction and mental disorder, and the most effective therapeutic process for overcoming them

CBT is built on the concept of self-efficacy, that psychological amelioration is the result of belief in one's control over oneself and one's environment. Not only does inculcating the belief that mental disorder and addiction are diseases undercut the self-efficacy essential to recovery, the disease model saddles the person with the belief that they have a lifetime deficiency—their addiction becomes a core part of their self-concept, of their being. Nothing is more self-defeating than this idea, as Ilse Thompson and I argue in *Recover! Stop Thinking Like an Addict* (Peele & Thompson, 2014). Yet more societal energy and greater societal effort are put to this way of thinking all the time.

Addictive Brain Disease

Perhaps even more than in the case of mental disorder, the chronic brain disease model of addiction has been embraced by both scientists and—fed nothing else—the public. Following on the endorphin revolution Restak announced, beginning in the 1980s, schematic views of the brain with regions specified for causing addiction have regularly graced the covers and pages of *Time*, *Newsweek*, and *Scientific American*. This public relations effort in the area of addiction has been cheer-led by Nora Volkow, Director of the National Institute on Drug Abuse, who has achieved a unique international public status. NIH Medline Plus (2007), for instance, presented “The Science of Addiction: Drugs, Brains, and Behavior”:

Two NIH institutes that are already on the forefront of research into drug and alcohol addiction recently joined with cable TV network HBO to present an unprecedented multi-platform film, TV, and print campaign aimed at helping Americans understand addiction as a chronic but treatable brain disease. . . .

Many Americans today do not yet understand why people become addicted to drugs or how remarkable scientific advances are literally redefining the arena of addiction, notes Nora D. Volkow. (From NIH Webpage.)

The prestigious international journal *Nature* (2014) declared addiction a brain disease with nothing more than a generic reference to Volkow's claims.

Drug addiction is a disease. Images of the brains of addicts show alterations in regions crucial to learning and memory, judgment and decision-making, and behavioural control. . . . The brain's central reward system is overstimulated and flooded with dopamine. The brain adapts to this flood by turning down its ability to respond to dopamine — so addicts take more and more of the drug to push dopamine levels higher. . . .

None of that is particularly controversial, at least among scientists.

Here are five objections to this model (Peele & Thompson, 2014):

1. Brain images represent the effects of drugs, and have never been related to compulsive drug-taking (or any other behavior).

There is no brain scan according to which a person can be said to be addicted, as opposed to showing the acute or chronic effects of cocaine or another drug or powerful experience. No one is diagnosed as “addicted” based on a brain scan. And no one ever will be. (Peele & Thompson, 2014, p. 21)

2. Rather than addiction comprising a chronic brain disease, recovery without treatment (i.e., natural recovery) is the typical course for addiction (Heyman, 2013; Lopez-Quintero et al., 2011; Peele, 2014a).

3. No brain images distinguish between compulsive and episodic users, and particularly those who cut back or quit (common paths taken by long-time users), versus those who do not.

4. Stimulation of these same areas of the brain and dopamine flooding occur with a wide variety of appetitive behaviors and emotional reactions (seeing a baby smile, sex, food, romantic love, gambling, ad infinitum), all of which can be measured in brain images.

5. Higher-SES addicts show far better remission rates than impoverished or socially deprived people who are addicted.

The Volkow dopamine model of addiction outlined by *Nature* is itself over a decade old (cf. Volkow, Fowler, & Wang, 2004). Yet, while *Nature* (2014) glorifies Volkow and her mission—“Europe should look to the United States and to inspirational fig-

ures such as Nora Volkow”—Volkow and her colleagues have not generated a single diagnostic or prognostic tool, nor any treatment for addiction. *Nature* remains optimistic: “Given the technical tools now available for looking deep inside the brain, there is realistic hope that such treatments will emerge from research in the coming decades.”

The note *Nature* sounds—“hope that such treatments will emerge from research in the coming decades”—is more restrained than Restak's vision of “alleviating, or even eliminating, such age-old medical bugaboos as pain, drug addiction, and, among other mental disorders, schizophrenia” or Garmezy's “ultimate resolution of the major psychotic disorders” made 40 years ago. This caution is necessary because *no one believes* that mental disorders, substance abuse and addiction, as well as DSM-5's newly recognized category of behavioral addictions (cf. Peele, 2014b), have been declining and are likely to decline in some foreseeable time frame.

Worse, biomedical models in the form of drug treatments for addiction have demonstrated their counter-efficacy. A study conducted by investigators whose own Center for Global Tobacco Control had spent millions on nicotine replacement therapy (NRT) tracked over several years smokers who either relied on NRT or did not in order to quit. The odds of relapse for a heavily dependent NRT quitter who had quit less than 6 months were 3.5 times that for a heavily dependent quitter who quit without NRT or professional help (Alpert, Connolly, & Blener, 2012). Apparently, those who quit on their own were more confident in their ability to control their own destinies—that is, they had higher self-efficacy, which is a self-fulfilling belief (Peele & Thompson, 2014).

In another remarkable demonstration of the failure of the neuropsychiatric and pharmacogenetic approach to addiction, a group central to the promotion of these views, Oslin et al. (2015) found no differences between double-blinded alcohol-dependent subjects receiving either naltrexone or placebo on percentage of days of heavy drinking, number of heavy drinking days per week, number of days until first heavy drinking, and weekly cravings. The authors' conclusion—“Despite the results of this trial, pharmacogenetics continues to hold promise as a way to improve the targeting of medications to improve treatment response” (p. E6)—is yet another demonstration that reductionism cannot

be refuted by mere facts, research, and data. Note: these results do not disprove that alcoholic subjects reacted to naltrexone, only that they were subjectively able to reproduce entirely the drug's effects in therapy.

Conclusion—Addicted to Failure

In his tour-de-force satire referencing NIMH directives, Brett Deacon (2014) captures their vision, which Deacon characterizes as “we’re about to solve all of mental disorder momentarily, but unfortunately we haven’t gotten to first base, and we don’t know in which direction first base is.” On this basis, I argued in favor of a nonreductive view of human behavior:

There is an idea—no longer very popular in America—that personality traits, human behavior, and psychopathology just don’t exist at the level of biochemistry, that the effort to “reduce” them to this level falls prey to the philosophical fallacy of “reductionism.” Instead, these human manifestations entail all of our lived experience, our physical settings, and our social relationships. After all, even the most committed biological determinists recognize the impact on children of deprivation and abuse. (Peele, 2013b)

Meanwhile, our brain revolution has resulted in no diagnostic tools or effective treatment while the prevalence of the maladies of concern grows, in many cases exponentially. And, yet, the dominant public health and political forces in the U.S. and internationally push the brain disease approach ever harder. There is nothing—except an unlikely return to reason and empiricism—to stop these forces from continuing to expand their influence as a paradoxical reward for their failures. As one answer to the question of how we are doing, the latest version of the National Epidemiologic Survey on Alcohol and Related Conditions found a 50% increase in past-year alcohol-use disorders between 2001–2002 and 2012–2013 (Grant et al. 2015).

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Using the Functional and Contextual Approach of Modern Evolution Science to Direct Thinking About Psychopathology

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WHEN DEALING WITH THE HUMAN BODY, there is a tendency to think of health as a natural state and ill health as a disturbance. When disturbances appear, a reasonable place to begin to understand ill health is to note how signs and symptoms gather in empirical collections.

Biomedical thinking of this kind has been useful and successful in many areas of physical health, in part because it contains within it convenient fictions that simplify the actual situation. Most especially, the idea that health is a baseline sidesteps current ignorance: treatment can proceed with a symptomatic target, despite ignorance about the mechanisms or systems involved. Within the lifetime of people who are reading this article, no consideration was given anywhere to DNA, or epigenes, or the microbiome in physical health treatment, because those important areas of knowledge about the body simply did not exist. Even now they are generally ignored.

A focus on the body as a physical entity seemingly also allowed complexities of situational context and history to be put aside. A physician could look at an EKG or at a scan of the arteries in the heart and generate treatment targets without knowing anything about what a person ate, how much they exercised, the stressors in the workplace, or the history of heart disease in the family. You do not need to know “why” to treat, even if everyone agrees that knowing why would be nice.

Psychopathology has been dominated as a field by these same biomedical assumptions. This was always an uncomfortable fit for psychology, however, in which concepts of function, development, and learning, among others, demanded a more contextual focus.

In the early days of behavior therapy “diagnosis” meant a functional analysis using behavioral principles, but with the arrival of the third revision of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association, 1980) that changed, with the help of behavioral and cognitive therapists, who

were willing to look past their philosophical concerns with syndromal diagnosis in order to speed the progress of scientifically validated protocols that targeted measurable problems. The DSM became an atheoretical nosological system with expanding categories—in many ways a devil's bargain (Follette & Houts, 1996). It helped produce enormous growth in the scientific database underlying CBT, but also supported the medicalization of human psychological problems and the spectacular rise in the long-term use of psychoactive medications (e.g., Mojtabai & Olfson, 2014).

It is hard to argue that the symptom-based syndromal model assumed by the DSM has been a scientific success. The comorbidity levels are overwhelming (Lenzenweger, Lane, Loranger, & Kessler, 2007), specificity is overwhelming (Galatzer-Levy & Bryant, 2013), and after 50 years of effort none of the psychiatric syndromes have led to the discovery of those more functional entities called diseases. In frustration, the National Institute of Mental Health (NIMH) has declared its intent to avoid funding studies based simply on the DSM, preferring studies linked to its Research Domain Criteria (RDoC) project that seeks a dimensional diagnostic system based on attempts to find biological and behavioral underpinnings of mental illness (<http://www.nimh.nih.gov/research-funding/rdoc/index.shtml>). RDoC will foster that search inside a matrix of five major domains (Negative Valence Systems, Positive Valence Systems, Cognitive Systems, Systems for Social Process, and Arousal & Regulatory Systems) that are nested within seven levels of analysis: gene, molecules, cells, circuits, physiology, behavior, and self-report.

RDoC appears to be a step toward a more functional approach, but the structure of biomedical thinking is still dominant. The first five levels of analysis (gene, molecules, cells, circuits, physiology) are properties of organisms. They need include nothing about history and context. The last two (behavior and self-report) can be

thought of as actions of whole organisms, but they can also be thought of as disembodied elements or mere areas of study. The domains are atheoretical systems.

The weak link to theory, history, and context presents a huge problem for psychotherapists and others who want to use scientific knowledge about psychopathology. Theory is needed to give the field guidance. History and context help combine knowledge across levels of analysis, and provide manipulable targets. The properties of organisms often cannot be manipulated directly, or they can but only outside of the set of interacting systemic elements that help give that element a function in the first place. For example, we cannot currently splice in a gene to a human being but we can change the contextual features that are known to control some forms of gene expression (Kaliman et al., 2014).

Authors of RDoC materials have made it explicitly clear that psychology is more an area (especially the area of subjective experience) than an approach, stating in the “FAQ” section that “Mental disorders involve both psychological and biological components. That is why subjective experience and the questionnaires that measure them are a significant part of the RDoC matrix.” (<http://www.nimh.nih.gov/research-priorities/rdoc/rdoc-frequently-asked-questions-faq.shtml>; accessed July 1, 2015).

At the same time, this domain is to be approached “biologically”: “RDoC is an attempt to explore the biological systems relating to these psychological constructs” (same citation as above). Indeed, in an editorial in the *American Journal of Psychiatry*, Insel made quite explicit that RDoC was inspired by the fundamental assumption that mental disorders are brain diseases:

RDoC classification rests on three assumptions. First, the RDoC framework conceptualizes mental illnesses as brain disorders. In contrast to neurological disorders with identifiable lesions, mental disorders 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, functional neuroimaging, and new methods for quantifying connections in vivo. Third, the RDoC framework assumes that data from genetics and clinical neuroscience will yield biosignatures that will augment clinical symptoms and signs for clinical management. Examples where clinically rele-

vant models of circuitry-behavior relationships augur future clinical use include fear/extinction, reward, executive function, and impulse control. For example, the practitioner of the future could supplement a clinical evaluation of what we now call an “anxiety disorder” with data from functional or structural imaging, genomic sequencing, and laboratory-based evaluations of fear conditioning and extinction to determine prognosis and appropriate treatment, analogous to what is done routinely today in many other areas of medicine. (Insel et al., 2010, p. 749)

The intention to tilt toward the study of neurological circuits that underlie behavior has been criticized as advocating a reductionistic view of psychopathology (Cuthbert & Kozak, 2013; Miller, 2010). In this short paper, we raise a similar but more positively worded concern. By diminishing the role of theory, history, and context, a growing area of cooperation between contextual approaches to psychology and biology is needlessly being set aside that could link psychopathology to treatment more directly.

Evolution: A Functional and Contextual Approach in Biology and Psychology Alike

Select any biological feature whatsoever and ask, “Why does this exist and why does it have a function?” The answer will *always* be the same provided only that the time frame encompassed by the answer is allowed to be large: It evolved that way. The title of Dobzhansky’s (1973) famous article made the point on which virtually all biologists would agree: “Nothing in biology makes sense except in the light of evolution.”

At the time of Darwin, nothing was known of genes or DNA, and the grand synthesis that put these topics tightly together gradually redefined evolution in a way that narrowed its relevance of behavioral scientists. Even in evolutionary psychology books, evolution was sometimes *defined* simply as “change in gene frequencies in a species due to selective survival” (Bridgeman, 2003, p. 325). Such a restrictive perspective provides little room for many areas in applied psychology, including psychotherapy. “How do genes impact psychotherapy” is a legitimate question—but it is not an obviously important one and is not one that would realign the field itself.

A more modern view of evolution science can do so. Evolution science is emerging from the tight grip of gene-centric analysis into a more general science that can guide intentional behavior change (Wilson, Hayes, Biglan, & Embry, 2014) including psychotherapy (Hayes & Sanford, 2015) based on a functional, contextual, and systemic perspective. Understanding the reasons why evolution science is changing will help situate our brief example of a new way of thinking about psychopathology that better integrates biology and psychology. Although there are others, we will mention seven major reasons.

First, the progress of the human genome project has shown conclusively that the old idea that genes code for specific phenotypic attributes is largely false (Jablonka & Lamb, 2014). Genes are obviously involved in mental health issues, but the dream of a simple formulation of that kind is largely dead. In the area of psychopathology we now have very large studies with tens of thousands of control and mentally ill subjects with full genomic mapping finding only a handful of general genetic risk factors that apply to a wide variety of disorders (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013).

Second, the rise of multilevel selection (Nowak, Tarnita, & Wilson, 2010; Wilson & Wilson, 2008) has altered the theoretical landscape in evolution science. There is a renewing interest in thinking of multicellular organisms as themselves being composed of competing and cooperating groups of cells; or of thinking of the social context in which behavior is embedded as the subject of evolutionary analysis. The “good of the group” can drive evolution under special circumstance and with that knowledge comes a new flexibility in applying evolution science concepts to topics such as altruism (Wilson, 2015), language and cognition (Hayes & Sanford, 2014), and human groups, not just genes.

Third, the data on genetic promoters and epigenetic processes such as chromatin markers have shown that organisms are systems for turning environment and behavior into biology (Slavich & Cole, 2013). To say it in a more colorful way, Lamarck is back (Jablonka & Lamb, 2014). Indeed, there is a growing body of work showing that experiences that are protective or risk promoting in mental health work in part through epigenetic processes (e.g., Uddin, & Sipahi, 2013), and that these epigenetic processes in turn impact the

organization of the brain (Mitchell, Jiang, Peter, Goosens, & Akbarian, 2013).

Fourth, the role of learning as the very ladder of evolution and speciation is now better appreciated (Bateson, 2013). With the arrival of contingency learning the relation between organisms and their environment fundamentally changed (Schneider, 2012). Behavior now could select niches, altering selective pressures. Flamingoes did not evolve beaks to begin eating river bottom crustaceans: the reinforcing effectiveness of eating such crustaceans maintained contact with the environments in which beak adaptations could be selected (Schneider). Learning constructs functional sequences of behavior that then can be made more efficient by evolutionary adaptation (Bateson).

Fifth, niche construction—the impact of learned behavior on long-lasting changes in environmental conditions—is now widely understood to alter the selective effects of the environment (Odling-Smee, Laland, & Feldman, 2003). With only a few exceptions (e.g., the Ice Age), the major events that have impacted humans over the last 50,000 years (agriculture, animal domestication, population density, exposure to animal pathogens, lactose tolerance, pollution, deforestation) are all the result of human behavior (Laland, Odling-Smee, & Myles, 2010)

Sixth, learning and the environment are known to be primary sources of input to epigenetic processes such as DNA methylation and histone acetylation, which in turn accounts for the neurobiological processes that regulate the long-term effects of learning (Miller, Campbell, & Sweatt, 2008). This is forcing a more multidimensional view of evolutionary processes, in which learning, culture, and cognition stand on a more equal footing with genes and epigenes. For example, animals repeatedly exposed to unpredictable aversive events show methylation of gene promoters in the germline across generations (Franklin et al., 2010); animals with genes removed as to create a learning disability can be successfully taught to learn using an enriched environment—and their offspring retained that ability even when the enriched environment is withdrawn (Arai, Li, Hartley, & Feig, 2009). Perhaps most surprisingly, at least some operant or classical conditioning can be inherited across generations due to epigenetic changes that this learning induces (Dias & Ressler, 2014).

Finally, cultural evolution, social learning, and the social processes involved in

language and cognition are understood to be inheritance systems in their own right. The words you are reading right now may be retained for many centuries in data archives. Through electronic means, someone may accidentally wander across these words generations from now and be impacted in some small way (at least to smirk at their ignorance, or smile at their innocence). Language and cognition is an inheritance system in its own right.

The impact of all of these developments is to refocus psychology and biology alike on the essence of evolution: variation and selective retention, in historical, situational, and systemic context, of a given phenomena occurring across multiple dimensions and at multiple levels. B. F. Skinner was arguably one of the first evolutionary psychologists of that kind, and had the recent developments been known while he was still alive, it is unlikely that the historical breach between behavioral psychology and evolutionary biology would ever have occurred:

In summary, then, human behavior is the joint product of (i) the contingencies of survival responsible for the natural selection of the species and (ii) the contingencies of reinforcement responsible for the repertoires acquired by its members, including (iii) the special contingencies maintained by an evolved social environment. (Ultimately, of course, it is all a matter of natural selection, since operant conditioning is an evolved process, of which cultural practices are special applications.) (Skinner, 1981, p. 502)

Evolutionists largely rejected this comparison 35 years ago because contingencies of survival and cultural evolution were viewed as matters of life and death (of organisms or of practices), while operant learning was not that. But now we know (among other things) that evolution is not restricted to genes; there are multiple interacting inheritance streams; and some of what evolves within the lifetime of individuals can live on through culture, social learning, changed environments, and in some cases through separate inheritance processes such as epigenetic changes. This means there is no easy dividing line between the contextual approach called evolution and contextual approaches to learning, behavior, and cognition.

Implications for Psychopathology

Consider where this puts the topic of psychopathology as it applies to, say, genes or the brain: the kind of easy targets that RDoC envisions. Evolution is the best-established general theory in the life sciences—it helped organize exploration to look at genetic and the neuropsychological inputs to behavior in the context of functional, contextual, multidimensional, and multilevel evolutionary processes. Cast as a physical object, a gene is just a sequence of nucleotides; the brain is just a neurobiological organ. Cast as part of an evolving system, a gene (or the brain) alters the functioning of other biological processes and evolved because it does so; a gene (or the brain) impacts learning, cognition, and culture and it is in turn impacted by these same processes; a given gene may be up and down regulated by myriad biological processes such as methylation, or the folding of DNA into proximal loops, and these regulatory processes are themselves regulated by environment and behavior. The brain or genes become dependent variables just as much as independent ones. This means it is impossible to separate out biological elements from the systems in which they are embedded over time—they need to be understood historically and in context.

Any statement that genes or the brain cause psychopathology misses the ongoing evolutionary and systemic complexity of the obtained relationships once even a short time frame is added to the picture. As we add other biological or psychological issues beyond the brain or genes, what is missing only becomes more obvious.

A promising alternative is to view psychopathology as a way of speaking about a set of multidimensional and multilevel evolutionary processes within and across lifetimes. In such an approach, the word “psychopathology” would be used when these interacting processes produced self-amplifying loops or self-sustaining processes that restrict needed processes of variation, selection, or retention, and/or sensitivity to context, dimensions, and levels. In other words, psychopathology is an evolutionary process gone awry in a specific way: it prevents further positive development via normal evolutionary processes. Psychopathology is a kind of adaptive peak, with no way forward via variation and selective retention. This is useful because the key processes specified by evolutionary theory can then guide the empirical and practical search for a solution.

An Example: Experiential Avoidance

Experiential avoidance (EA) has been defined as an individual's attempts to change or reduce the form or frequency of private experiences such as bodily sensations, emotions, thoughts, or urges, even when doing so causes psychological difficulty (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). In a very brief example we will mention a few of the RDoC domains as they apply to EA and then will note how reconsidering EA from the point of view of evolution science recasts key biological and psychological issues.

EA is one of the most toxic processes known to behavioral science. Higher levels of reported EA are positively correlated with depressive and anxious symptomatology, psychological distress, a number of specific phobias, general physical health, and quality of life (Bond et al., 2011; Hayes et al., 2004). Furthermore, EA has been shown to be a strong predictor of daily anxiety-related pathology and emotional distress while also being inversely related to daily positive emotions, life appraisals, and events (Fledderus, Bohlmeijer & Pieterse, 2010; Kashdan et al., 2006). When escape and avoidance is applied to private events such as memories or thoughts, it often has the paradoxical effect of increasing their frequency or intensity (Abramowitz, Tolin, Street, 2001; Magee, Harden & Teachman, 2012)—but this in effect creates a self-amplifying loop in which the output of a process is the input to that same process. Self-sustaining loops of that kind tend to produce context insensitivity because they are reflective and self-focused. Said in another way, EA is a result of variation and selection (i.e., variations in attempts to reduce or avoid private experiences and selection based on their suppressive or avoidance effects) but it diminishes further variation and selection (i.e., reducing the likelihood of engaging in more useful actions if they include targeted emotional or cognitive experiences). In other words, EA is the result of natural variation, and becomes problematic when it is not fitted to a context (i.e., overgeneralized), or is selected by its short-term consequences (i.e., immediate relief) at the expense of competing behavioral repertoires.

We can see some of these processes in several of the RDoC levels of analysis: an evolutionary perspective helps weave them into a more coherent whole. EA is clearly present in subjective experience, but it also has an impact on overt behavior. For example, those who score high on a measure of

experiential avoidance are less likely to persist in a variety of challenging tasks such as perceptually difficult tasks (Zettle, Petersen, Hocker, & Provines, 2007; Zettle et al., 2012), exposure to pain (Branstetter-Rost, Cushing & Duleh, 2009), or breathing CO₂-enriched air (Eifert & Heffner, 2003).

EA has an impact on physiology and brain circuitry. During avoidance people high in EA are more highly lateralized (Cochrane, Barnes-Holmes, Barnes-Holmes, Stewart, & Luciano, 2007), which makes sense if it is a deliberate, cognitively guided process, and they show higher physiological arousal and lower heart rate in response to stimuli as compared to a low EA sample (Sloan, 2004), which makes sense of the intended function of EA, namely avoidance of aversive events. Similarly experiential avoidance is known to be associated with reduced frontal and limbic reactivity during avoidance trials (Schlund, Magee, & Hudgins, 2011). There is even some evidence that EA shows the characteristic of an endophenotype as related to the 5HTT gene (Gloster et al., 2015).

Our point is EA is known to be important across range of levels of analysis being targeted in RDoC. It changes the meaning of these data a bit to look at EA as a process that restricts variation, selection, retention, and sensitivity to context. EA is more likely with early aversive experiences (Fiorillo, Papa, & Follette, 2013; Shenk, Putnam, & Noll, 2012) and can be thought of as a verbally governed attempt to reduce aversive stimulation and threat by psychological and behavioral means that are effective in the short term and counterproductive in the long term. This set of circumstances produces a kind of self-amplifying process that is an evolutionary black hole. EA restricts needed variation by reducing emotional, cognitive, and behavioral flexibility. EA itself signals threat, leads to a state of chronic stress, and in turn alters the regulation of stress-related genes, further cementing the pathological process and producing a range of physical and mental health problems. On these grounds it may make more biological and psychological sense to name and describe this set of conditions as an experiential avoidance disorder than it would to use most of the pathology labels in the DSM that are symptom based.

A great advantage of doing so would be to link experiential avoidance disorder to treatment. From an evolutionary perspective any method of intervention that increases emotional, cognitive, and behavioral flexibility in the presence of repertoire

narrowing stimuli that occasion EA, should be helpful. This would include exposure, mindfulness, cognitive defusion, or similar methods—and indeed all of these are helpful (e.g., Hayes, Villatte, Levin, & Hildebrandt, 2011). They are helpful in part because they reduce EA (e.g., Bond et al., 2006), and are helpful in part because they alter the biological associates associated with EA (e.g., epigenetic regulation of stress-related genes; see Kaliman et al., 2014). From an evolutionary perspective, retention of these changes could be helped by the right selection criteria and by behavioral practice. The work on values (Branstetter-Rost et al., 2009; Paez-Blarrina et al., 2008) and psychological treatments that heavily rely on homework (Cuijpers, van Straten, & Warmerdam, 2007) suggest that this is the case as well.

Summary

Psychology overlaps with biology and vice versa. The attempts to integrate these fields through neuroscience or other organismic features alone leave too many key features out of the equation, most especially the role of context and history. The atheoretical mistake made by the DSM could easily be repeated with RDoC. Evolution science puts this right, and does so using the best-established and most generally applicable theory in all of the life sciences.

As a gene-centric perspective on evolution science gradually retreats into history, and a multidimensional, multilevel, and systemic perspective steps forward, psychology is seen to have a central seat at the table of the evolution sciences, and applied psychology is understood to be one of the best areas in which evolutionary ideas can be tested. By thinking of psychopathology as an evolutionary problem, the domains and levels of RDoC can be focused on the key conceptual issues known to be most important in the development of successful systems. Evolutionary thinking can turn the dimensional complexity of factorial visions such as RDoC into the systemic clarity of the search for evolutionary dead ends, and ways they can be broken down. Psychopathology describes and unpacks those dead ends, while psychotherapy constructs pathways out of biopsychosocial systems that lack healthy variation, proper selective retention, context sensitivity, or a proper focus on the right dimension or level of selection. Seen from such a perspective, empirical psychopathologists and psychotherapists are more likely to make

progress by thinking of their fields as part of applied evolution science.

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A Psychological Model of Mental Health and Well-Being: Rational but Radical

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WE HAVE SLIPPED INTO the pervasive and seductive idea that distressing emotions can best be understood as symptoms of physical illnesses. But in my view this is a myth, and a harmful one. These old-fashioned, inhumane, and fundamentally unscientific ideas about the nature and origins of mental health problems severely hamper our attempts to help people in acute emotional distress. We need radical change in how we design and commission mental health services, and even, more fundamentally, in how we understand mental health problems.

The Fundamental Nature of Psychological Distress

Our current (often tacit) models of mental health problems—especially severe problems that attract diagnoses like bipolar

disorder or schizophrenia—tend to assume that these experiences emerge from mystery biological illnesses, unrelated to a person's life and experiences. When we start asking questions about this traditional "disease-model" way of thinking, however, those assumptions start to crumble (Kinderman, Read, Moncrieff & Bentall, 2013).

Much of the literature available to the general public reinforces an assumption that serious problems as hallucinations and delusional beliefs are fundamentally biological in origin, despite considerable evidence that traumatic childhood experiences (poverty, abuse, etc.) are associated with later psychotic experiences. When media commentators report on tragic news stories, and particularly when prominent individuals take their own lives, there is an almost knee-jerk assumption that suicide is a consequence of an underlying illness,

explicable only in biological terms and often described as coming "out-of-the-blue"—in other words, inexplicable in human terms. Of course, in all mainstream, physical, health care, the links between social circumstances (especially inequalities) and health outcomes are clear. It's particularly clear in the case of mental health—and particularly understandable. Across Europe, the recent economic recession has had a direct impact on suicide rates—a rather dramatic (and sad) example of how social factors have an impact on our mental health. It's understandable that people who find themselves in desperate economic and social circumstances may believe their future is hopeless—and equally obvious that they are tragically responding to events.

None of this denies the physical, biological, reality of our embodied lives. All our experiences in some sense relate to, and depend upon, the functioning of our brains. But this is true of all human behavior and every human emotion—falling in love, declaring war, solving Fermat's last theorem. It is unscientific to make a distinction between "normal" emotions and

distress—which we then classify as illnesses and explain as a product of chemical “imbalances.” The neurological processes of the brain enable us to think, to behave, to feel emotions. They don’t “cause” them.

The hugely complex biological structures of the human brain support an impressive learning machine. Even severe mental health problems are not merely the result of faulty genes or chemical abnormalities. They are also a result of learning: a natural and normal response to the things that can happen to us, and that shape our view of the world.

Diagnostic Fog

The diagnostic systems used in psychiatry have been widely criticized for their poor reliability, validity, utility, epistemology, and humanity (*British Medical Journal*, 2013; Division of Clinical Psychology, 2013; Kinderman et al., 2013; Kinderman, 2015; Lancet, 2012). Using standardized approaches, we can generate statistically reliable diagnoses. But the standardized interview schedules used to achieve this are rarely used in routine clinical practice; perhaps more important, it is entirely possible to make reliable diagnoses of invalid concepts—agreement between diagnosticians is no guarantee that their diagnoses correspond to meaningful clusters of symptoms, with distinct pathophysiology and etiology, and which predict the effectiveness of particular interventions.

It is a perhaps unfortunate fact that psychiatric diagnoses are invalid in the sense that they fail to map onto any entity discernible in the real world, fail to predict course or outcome or indicate which treatment options are beneficial. Psychiatric diagnoses do not map neatly onto biological findings, which are often nonspecific and cross diagnostic boundaries. For example, depression and anxiety co-occur so commonly that the term “common mental disorder” is often used instead (although, clearly, not being a recognized diagnosis). Unsurprisingly, people with psychotic experiences and seeking help commonly also describe problems that in other circumstances would attract a diagnosis of some form of “affective disorder” and it remains somewhat unclear as to whether “bipolar disorder” can easily be separated from “schizophrenia” or indeed “borderline personality disorder.” At the same time, such biological findings as do emerge from the scientific literature are equally nonspecific—with genetic associations being both weak (explaining little of

the variance in human experience of mental health problems) and conveying very general risks to a very wide range of problems (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013).

Traditional psychiatric diagnoses also have epistemological or philosophical problems. Despite overt denials, the diagnoses tend to be used as pseudo-explanations—people are tacitly believed to be behaving as they are, or experiencing unusual perceptions, because of the illness (Morgan, 2015). This might have some validity if the putative illness represented pathologies, but it is difficult to see how a label for something can also explain something. The unobtrusive use of a diagnostic approach also tends to reduce our capacity for empathy (Lebowitz & Ahn, 2014). Since medicalized diagnoses convey the idea that mental health problems can be understood as illnesses or diseases, as pathologies of the body, we are in danger of ignoring any psychological meaning in people’s “disordered” responses and experiences. This tends to diminish our appreciation of the social context of these discussions. The American Psychiatric Association classified homosexuality as a mental disorder until 1973, and the World Health Organization did not declassify homosexuality as a “disorder” until 1990. This seems extraordinary in 2015, and I strongly welcome these changes and the celebration of human freedom that this represents. But it does illustrate how context-bound these diagnoses are, and how alien to psychological science and humanism. And, of course, in other areas of human life, such assumptions remain. There may be a profound and long-lasting grieving process after the death of a loved one. But in what sense is it a “disorder” if we remain distressed by bereavement after 3 months (Lancet, 2012)? In what sense is a person “disordered” if she is traumatized by the experience of industrialized military conflict (Kinderman et al., 2013)? Children need to regulate their emotions and grow up with a sense of moral and social responsibility. But is it appropriate to say they have a “disorder” when they need extra help?

Clarity Without Diagnosis

As I have argued elsewhere (Kinderman et al., 2013; Kinderman, 2014a; Kinderman 2015), we need a wholesale revision of the way we think about psychological distress. The manifesto for reform is far-reaching and comprehensive, but it starts with diagnosis and assessment. And here we should

remember that we already have available alternative approaches to diagnosis.

When we identify, describe, and respond to distress, we should use language and processes based on the recognition that such distress is a normal, not abnormal, part of human life. In particular, we should appreciate the clear evidence that psychosocial factors such as poverty, unemployment, and trauma are major causal factors for psychological distress (Read & Bentall, 2012). We must also reflect the overwhelming evidence that psychiatric symptoms lie on continua with less unusual and distressing mental states. There is no easy cutoff between “normal” experience and “disorder” (Bentall, 2003). And we should reflect the understanding that people’s behavior, thoughts, and emotions are best understood in terms of the ways in which people makes sense of the things that are happening to them, a framework of understanding that is, itself, learned through experience.

A clear improvement upon diagnostic labels for putative “disorders,” therefore, would be simply to identify specific problems. We are applied scientists, and the scientific method—a method or procedure that has characterized natural science since the 17th century, consisting in systematic observation, measurement, and experiment, and the formulation, testing, and modification of hypotheses” (Oxford English Dictionary, 2013)—means that we can develop valid operational definitions of relevant concepts, hypothesize, collect data and thereby refute and develop better models (Kinderman, 2015). We do need an alternative to diagnosis and the “disease model.” But we’ve had that alternative since the 17th century.

A New Ethos

In short, then, we need a new basis for understanding mental health and well-being (Kinderman, 2014a). Moving away from a diagnostic approach to assessment and planning is only a small part of the reforms needed in mental health care. We should move away from the disease model, which assumes that emotional distress is merely symptomatic of biological illness, and instead appreciate that our role is to help and support people who are distressed as a result of their life circumstances, and how they have made sense of and reacted to them.

The manifesto for reform has several elements:

1. Services should be based on the premise that the origins of distress are largely social

The guiding ethos underpinning mental health services needs to change from assuming that our role is to treat “disease” to appreciating that our role is to help and support people who are distressed as a result of their life circumstances, and how they have made sense of and reacted to those challenges. Biological factors, social factors, circumstantial factors—our learning as human beings—all affect us; those external factors impact the key psychological processes that help us build up our sense of who we are and the way the world works (Kinderman, 2014b). This is, of course, a key element of the underpinning ethos of clinical psychology, but is entirely compatible with wider health and social care systems.

The World Health Organization describes health as “... a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity” (World Health Organization, 1948). The European Commission takes a step further, describing mental health as “a resource which enables them to realize their intellectual and emotional potential and to find and fulfill their roles in social, school and working life. For societies, good mental health of citizens contributes to prosperity, solidarity and social justice” (European Commission, 2005). The European Commission, interestingly, also suggests that “the mental condition of people is determined by a multiplicity of factors including biological, individual, family, social, economic and environmental.” They cite the role of “genetics, but also gender, personal experiences, social support, social status and living conditions” in our mental well-being.

This strongly suggests that this biopsychosocial (or, perhaps, even psychobiosocial [Kinderman, 2014a]) approach echoes across health and social care. While psychologists might claim that their specific role is better described as helping people fulfill their potential as human beings, rather than treating illnesses (Kinderman, 2013), doctors—medical practitioners, psychiatrists—have always prized an element of their profession that goes beyond merely treating disorders.

2. Services should replace “diagnoses” with straightforward descriptions of problems

As I have outlined above, we must stop regarding people’s very real emotional distress as merely the symptom of diagnosable “illnesses.” Scientific objectivity in the definition and measurement of people’s problems is perfectly within reach and more than sufficient as a basis for individual care planning and for the design and planning of services (Kinderman, 2015).

3. Services should radically reduce use of medication, and use it pragmatically rather than presenting it as “treatment”

We should listen to the expressed views of people using mental health services (McHugh, Whitton, Peckham, Welge & Otto, 2013) and sharply reduce our reliance on medication to address emotional distress. Medication should be used sparingly and on the basis of what is needed in a particular situation—for example, to help someone to sleep or to feel calmer. We should not look to medication to “cure” or even “manage” nonexistent underlying “illnesses” (Moncrieff, 2008).

In practical terms, we should aim for a massive reduction in the level of psychiatric prescription. There should be many fewer prescriptions, at lower doses, for much shorter periods. In essence, this means adopting a “drug-based” approach in contrast to the more common “disease-based” approach (Moncrieff, 2013). We should respond to people’s specific problems, rather than make the mistake that we’re treating illnesses that can be identified by diagnosis. We need to listen to the person’s own experiences of which drugs have helped in the past and how they are finding those currently prescribed, and use what they say to guide our prescribing. Most important, perhaps we should only use psychiatric medication in the very short term (i.e., for a matter of days) in the vast majority of cases.

4. Services should be tailored to each person’s unique and complex needs

Just as a psychosocial alternative to diagnosis should be firmly scientific, and in no sense a vague free-for-all, so we need to equip both services (in the abstract) and individual practitioners with the tools to enable them to help with the full range of people’s social, personal, and psychological needs, and to address both prevention and recovery. We must offer services that help people to help themselves and each other rather than disempowering them: services

that facilitate personal “agency,” in psychological jargon. That means involving a wide range of community workers and psychologists in multidisciplinary teams, and promoting psychosocial rather than medical solutions.

Again, however, we have these tools. We know that psychological therapies can be effective in a wide range of problems (see, for example, the recommendations of the U.K. National Institute for Health and Clinical Excellence; www.nice.org.uk). All such therapies should be evidence-based and delivered by qualified, competent professionals, and decisions about what therapy or therapies should be offered to whom should be based on a person’s specific problems and on the best evidence for the effectiveness of the intervention, not on the diagnosis. Individual formulations (Division of Clinical Psychology, 2011; Johnstone & Dallos, 2013) should be used to put together an individualized package of care for each person’s unique set of problems. Using psychological therapies as part of a psychosocial, rather than biomedical, approach would significantly change the way clinical psychologists and others work. Our psychiatric colleagues would play an important medical role within this overall psychosocial ethos. Their role would be analogous to those of general practitioners, public health physicians, and doctors who offer their expertise to athletes.

5. Services should offer care rather than coercion

Even when people are in crisis, and when residential care may be needed, this need not be seen as a medical issue. Since a disease model is inappropriate, it is also inappropriate to care for people in hospital wards; a different model of care is needed. As with other services, residential units should be based on a psychosocial rather than a medical model. There are many examples of this approach (see, for example, the Dayton Park Women’s Crisis Centre in London, U.K.; <http://www.candi.nhs.uk/services/services/dayton-park-womens-crisis-service/>).

Residential social workers or nurses who have retrained in a psychosocial approach (and possibly with a more appropriate professional title) are likely to be best placed to lead such units. The nature of extreme distress means medical colleagues may well be valuable members of the team but again their role should be as consultants to the team, rather than automatically as leaders of the team.

Adopting a psychosocial, as opposed to disease-based, approach would also change our stance towards compulsion. In those instances where compulsory detention was necessary, decisions would be based on the risks that individuals are thought to pose to themselves and others, together with their capacity to make decisions about their own care. This approach is already the basis for the law in Scotland.

6. *Mental health teams need to be radically different*

Teams should be multidisciplinary, democratic, and based on a psychosocial model. A psychosocial approach to service delivery would mean increased investment in the full range of professionals able to deliver these therapeutic services. Peer professionals, namely people with lived experience of mental health problems, will be particularly valuable, as will those skilled in practical issues such as finding employment or training. In the multidisciplinary teams delivering these services, psychiatric colleagues will remain valuable colleagues. An ideal model for interdisciplinary working would see leadership of such teams determined by the skills and personal qualities of the individual members of the team, rather than by their profession. It would not be assumed that medical colleagues should have “clinical primacy” or unquestioned authority. Such an approach is common in many European, especially Scandinavian, countries.

7. *A social and community focus*

Mental health services should be organized alongside other social, community-based, services. The psychological, emotional, and behavioral problems that are commonly referred to as mental health problems are fundamentally social and psychological issues. Psychologists, therapists, and social workers must work closely alongside GPs, public health physicians, nurses, and psychiatrists. But mental health is fundamentally a psychological and social phenomenon, with medical aspects. It is not, fundamentally, a medical phenomenon with additional psychological and social elements. It follows that the correct place for mental health care is within the social care system.

8. *We must establish the social prerequisites for genuine mental health and well-being*

Our mental health and well-being are largely dependent on our social circumstances. To promote genuine mental

health, therefore, we need to protect and promote universal human rights, as enshrined in the United Nations’ Universal Declaration of Human Rights. Because experiences of neglect, rejection and abuse are hugely important in the genesis of many problems, we need to redouble our efforts to protect children from emotional, physical, or sexual abuse and neglect. Equally, we must protect both adults and children from bullying and discrimination: whether that is racism, homophobia, or discrimination based on sexuality, gender, disability or “mental health” or any other characteristic. We can all do more to combat discrimination and promote a more tolerant and accepting society. More generally, if we are serious about preventing mental health problems from developing, and about promoting genuine psychological well-being, we must work collectively to create a more humane society: to reduce or eliminate poverty, especially childhood poverty, and to reduce financial and social inequality. We need to work harder to promote peace, social justice and equity, and ensure that citizens are properly fed, housed, and educated, and living in a sustainable natural ecosystem. We need to promote social mobility and social inclusion, encourage actions aimed at the common or collective good (for instance, through practical support of local charitable activities), and reduce both corruption and materialistic greed (Kinderman, 2013). In a fair society, in a society that protects our mental health and well-being, we would ensure that everyone had a meaningful job or role in society and we would eliminate unhealthy organizational cultures at work.

Conclusions

We should turn from the diagnosis of illness and the pursuit of biological etiology and instead identify and understand the causal mechanisms of operationally defined psychological phenomena. Our psychiatrists and health services should sharply reduce our reliance on medication to address emotional distress. We should not look to medication to “cure” or even “manage” nonexistent underlying “illnesses.” We must offer services that help people to help themselves and each other rather than disempowering them: services that facilitate personal agency. That means involving a wide range of community workers and psychologists in multidisciplinary teams, and promoting psychosocial rather than medical solutions. When

people are in acute crisis, residential care may be needed, but this should not be seen as a medical issue. Since a disease model is inappropriate, it is also inappropriate to care for people in hospital wards; a different model of care is needed.

Adopting this approach would result in a fundamental shift from a medical to a psychosocial focus. This is a call for a revolution in the way we conceptualize mental health and in how we provide services for people in distress. But this revolution merely reflects the applied science, praxis, and values of many existing professions in mental health . . . and it’s probably already under way.

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The Biomedical Model of Psychological Problems: A Call for Critical Dialogue

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THE CENTRAL TENET OF THE BIOMEDICAL model is that psychological problems are literal diseases of the brain. This model has dominated mental health research, policy, and practice in the United States for more than three decades. During this time, federal agencies like the National Institute of Mental Health (NIMH) and the National Institute on Drug Abuse (NIDA) have focused their grant funding initiatives on biomedical research, medications have replaced psychosocial interventions as the modal treatment for psychological problems, "brain disease" and "chemical imbalance" explanations for mental disorders have been heavily prompted by the pharmaceutical industry and academic psychiatry, and the general public has come to regard mental disorders as diseases of the brain caused by biogenetic abnormalities (Deacon, 2013). There is a consensus among biomedical proponents that we are on the verge of a new era of personalized medicine characterized by biological diagnostic tests and disease-specific curative treatments.

Although the biomedical approach has enjoyed longstanding popular support, recent developments have promoted a reconsideration of its validity and utility. These include: (a) books and media reports by respected scientists and investigative journalists who present evidence disputing key tenets of the biomedical model (e.g., Angell, 2011; Kirsch, 2010; Satel & Lilienfeld, 2013; Stahl; 2012; Whitaker, 2010); (b) public controversy surrounding the DSM-5 (APA, 2013) revision process, which included a concerted effort by DSM-IV (APA, 2000) task force chair Allen Frances (2014) to discredit the new diagnostic manual and the validity of psychiatric diagnoses; and (c) public statements by NIMH director Thomas Insel (2013) and DSM-5 task force chair David Kupfer (APA, 2013) that DSM diagnoses are not valid and that biomarkers for mental disorders (i.e., disorder-specific biological correlates) have not been found. These are important developments. A growing critical analysis of the biomedical model is now under way, and

this special issue of *the Behavior Therapist* is intended to contribute to this analysis.

Although critical analysis of psychological theories and practices has a rich tradition in academic psychology (e.g., Lilienfeld, Lynn, & Lohr, 2015), the biomedical model has rarely been subjected to open critical analysis within the professional community. There are numerous reasons for this, including desire for harmony between various mental health professions (e.g., between psychology and psychiatry), protection of guild interests, and fear of retaliation from biomedically oriented grant funding agencies. However, given the concerns about the validity and utility of the biomedical model described by contributors to this special issue, such a critical analysis is urgently needed. In our view, this analysis is also necessary to combat the current level of arguably uncritical and disingenuous discourse on the biomedical model at the highest level in the United States, which we illustrate below with recent essays written by the directors of the NIDA and NIMH.

"Addiction Is a Disease of Free Will"

On June 12, 2015, NIDA director Nora Volkow published a Huffington Post article titled, "Addiction Is a Disease of Free Will." Volkow tells the story of how she learned her grandfather committed suicide in his distress at not being able to control his urges to drink alcohol. To Volkow, this family tragedy illustrates two lessons. The first is that the biomedical model explains why addicted individuals seem unable to control their drug use. Specifically, "because of drug use, a person's brain is no longer able to produce something needed for our functioning and that healthy people take for granted, free will." Second, "embrac[ing] the concept of addiction as a chronic disease" is necessary to reduce stigma and facilitate access to effective treatments.

Beyond simply describing addiction as a brain disease, Volkow explains the "underlying pathology" that renders

addicts apparently unable to control their behavior. She claims:

We can do much to reduce the shame and stigma of drug addiction, once medical professionals, and we as a society, understand that addiction is not just “a disease of the brain,” but one in which the circuits that enable us to exert free will no longer function as they should. Drugs disrupt these circuits. The person who is addicted does not choose to be addicted; it’s no longer a choice to take the drug. (Volkow, 2015)

Volkow argues that drugs disrupt “the most fundamental brain circuits” that enable us to “make a decision and follow through with it.” To Volkow, the notion that addicted individuals do not have free will is heartening. Addicted individuals who accept this notion will “simply, non-judgmentally receive the help they need” (she cites two medications as examples) “like a child with diabetes or a person with heart disease or cancer.”

Volkow’s blog post describes a remarkable scientific story in which biomedical research has revealed addiction to be a literal disease of the brain with a known pathophysiology in the form of faulty neural circuitry. Neuroscientists have discovered the brain circuit that produces free will and have shown that drug-related disruption in this circuit robs addicts of their ability to make decisions and control their behavior. Disseminating this message will reduce stigma and pave the way for effective treatment.

The scientific narrative described by the NIDA director would indeed be remarkable if it were true. However, it bears little resemblance to the scientific evidence described by contributors to this special issue (Kichuk, Lebowitz, & Adams, 2015, this issue; Lilienfeld, Schwartz, Meca, Sauvigné, & Satel, 2015, this issue; Peele, 2015, this issue). Although drug use (like all rewarding experiences) affects the brain in predictable ways, this observation is insufficient to classify addiction as a “brain disease.” Genomic and neuroimaging studies have not identified abnormalities that distinguish addicted from nonaddicted individuals with a clinically meaningful degree of sensitivity and specificity (Hall, Carter, & Forlini, 2015). In other words, there are no biomarkers for addiction. Addicted individuals are often capable of controlling their behavior in certain contexts, and psychological research using the balanced placebo design demonstrates that their

excessive substance use is more a product of expectancies than physiological dysfunction (George, Gilmore, & Stappenbeck, 2012). Most individuals who develop a substance addiction eventually overcome it without receiving treatment (Hasin, Stinson, Ogburn, & Grant, 2007). Psychological treatments that emphasize self-efficacy in controlling substance use are recommended first-line interventions in clinical guidelines (National Institute for Clinical Excellence, 2011). Acceptance of a neurobiological view of addiction does not improve stigma (Pescosolido et al., 2010) and reduces self-efficacy in controlling substance use (Wiens & Walker, 2014).

The discordance between Volkow’s narrative and the scientific evidence is concerning. Some of her assertions lack evidentiary support (e.g., addiction is a literal brain disease with a known pathophysiology), some are scientifically implausible (e.g., drug use eliminates free will by disrupting the brain circuits that produces it), and some are contradicted by reliable evidence (e.g., endorsement of the brain disease model reduces stigma). Moreover, the claim that drug-addicted individuals lack free will raises a host of troubling questions about personality responsibility, legal culpability, and the credibility of psychological treatments that emphasize the directed application of free will (Peele, 1989; Satel & Lilienfeld, 2013).

Cognitive-behavioral therapy (CBT) is cited as an evidence-based treatment for substance use disorders in the NIDA publication “Principles of Drug Addiction Treatment: A Research-Based Guide” (2012; preface by Nora Volkow). According to the guide, a central element of CBT is “enhancing patients’ self-control by helping them develop effective coping strategies,” including “strategies for coping with cravings” (p. 49). Another evidence-based treatment listed in the guide, motivational interviewing, “helps individuals resolve their ambivalence about engaging in treatment and stopping their drug use” and “aims to evoke rapid and internally motivated change” (p. 55). Our attempt to integrate the information in NIDA’s treatment guide with Volkow’s scientific narrative raises numerous questions, including the following:

- If addicted individuals suffer from a disease-induced lack of free will, how could they possibly benefit from psychosocial treatments that emphasize motivation, self-efficacy, and skills for controlling urges to use? How can the

efficacy of such treatments be explained?

- Does acceptance of the message that “addiction is a disease of free will” rob addicts of the very sense of agency that is a prerequisite for success in evidence-based psychological approaches like CBT?
- Is it fundamentally disempowering to inform addicts they are biologically incapable of making decisions, controlling their behavior, and directing the course of their lives in accordance with their values?

In an interview published in *Newsweek* (Interlandi, 2008), Volkow predicted, “The future is clear. In 10 years we will be treating addiction as a disease, and that means with medicine.” At present, 3 years shy of this predicted future, the biomedical approach has yet to produce clinically meaningful treatment innovations. The few novel addiction treatments that have been developed in recent decades (e.g., naltrexone) are not particularly effective, and the most commonly used pharmacological treatments (e.g., methadone) preceded the modern biomedical era by decades (Hall et al., 2015). We can’t help but wonder, if 2018 arrives without Volkow’s predicted disease-based medicine for addiction having come to fruition, if the media, scientific community, and other stakeholder groups will take notice. We hope this article and special issue of *the Behavior Therapist* will encourage increased accountability, public scrutiny, and rigorous scientific analysis of the biomedical approach.

“Psychiatry Is Reinventing Itself Thanks to Advances in Biology”

On August 19, 2015, NIMH director Thomas Insel authored an editorial in *New Scientist* titled, “Psychiatry Is Reinventing Itself Thanks to Advances in Biology.” Insel stated the following:

The problem is that even though there have been thousands of studies looking for biological markers of mental health problems such as depression or schizophrenia, none has proven clinically actionable. And, in truth, little has been replicable even in a research setting. So some psychiatrists understandably reason that this approach offers no advantage, but large costs. (Insel, 2015)

He also observed: “Objective diagnostic categories that are reliable and biologically valid are long overdue in this field.”

These are extraordinary admissions. In his editorial, the NIMH director acknowledges the following realities: (a) DSM-defined mental disorders are neither adequately reliable nor biologically valid, (b) biomedical research conducted in the modern DSM era (1980-present) has failed to identify a single biological variable that is useful in the diagnosis or treatment of any mental disorder, (c) findings from biomarker studies have not been consistently replicated, and (d) it is understandable to conclude the biomedical approach has not worked, at great cost. We are pleased that Insel has conceded these principal objections to the biomedical model noted by contributors to this special issue (Abramovitch & Schweiger, 2015, this issue; Kinderman, 2015, this issue; Lacasse & Leo, 2015, this issue; Moncrieff, 2015, this issue; Peele, 2015, this issue; Whitaker, 2015, this issue).

Yet surprisingly, Insel views the failure of biomedical research to date as evidence that we need more biomedical research. His editorial tells of a “revolution under way in psychiatry.” Built on genomics and neuroscience, the revolution is founded on the assumption that mental health problems are “brain disorders related to physiological changes rather than simply behavioral ones.” Specifically, the thoughts and behaviors associated with mental disorders are “symptoms of an underlying disorder in a brain circuit.” To Insel, the future lies in biomarkers, which the NIMH’s Research Domain Criteria (RDoC) project aims to identify. He views the success of “precision medicine” in cancer as a model for the diagnosis and treatment of mental health problems. In approximately a decade, Insel predicts a “tectonic shift” in which “genomic, cellular, imaging, social and behavioural information” will be used to “develop tests to identify precise diagnostic groups within what we now call mental disorders.”

In our view, the scientific narrative described by Insel in this editorial is problematic. Like Volkow, Insel claims mental health problems are caused by faulty brain circuitry. However, Insel makes this claim in the same essay in which he acknowledges “thousands of studies” have failed to reveal a clinically useful biomarker for any mental disorder. In other words, Insel simultaneously advances the seemingly incompatible claims that (a) mental health problems are caused by disordered brain

circuitry, and (b) there is no mental health problem-specific brain abnormality known to science. Our critical analysis of Insel’s editorial raises numerous questions, including the following:

- If “thousands of studies looking for biological markers of mental health problems” have not found them, why should we believe problem-specific biomarkers will be discovered and lead to “precision medicine” in approximately 10 years? Given Insel’s previous, highly similar prediction that “biodiagnostics” and “treatment of core pathology” would arrive by 2015 (Insel & Quirion, 2005), why should we trust this new prediction?
- Is there a point in time when the failure of biomedical research to identify disorder-specific biomarkers and personalized biological treatments should dampen enthusiasm for this approach and prompt an honest reconsideration of its validity and utility? If so, when?
- Is it premature to announce a scientific revolution in the absence of revolutionary data? Don’t scientific revolutions follow revolutionary data?
- Is cancer a valid model for psychological problems? On what basis should we believe advances in the clinical management of cancer due to biomedical research are applicable to problems of thinking, feeling, and behaving?
- Why is the NIMH director writing editorials about psychiatry’s image?

A Call for Critical Dialogue

The opinion pieces by Volkow and Insel, both of whom direct a federal mental health agency and its influential grant funding agenda, provide a microcosm for contemporary dialogue surrounding the biomedical model. Proponents describe a scientific revolution of transformative power built upon advances in genomics and neuroscience. According to this narrative, mental health problems are diseases of the brain caused by faulty neural circuitry. Promoting this message reduces stigma and facilitates effective treatment. Advances in biomedical research will soon produce disease-specific diagnostic tests and highly effective personalized treatments, much as they have for diseases like cancer.

As described by contributors to this special issue, there are compelling data-based reasons to question this narrative.

Although the biomedical era has witnessed groundbreaking methods (e.g., neuroimaging), genomics and neuroscience have not produced the findings necessary to deliver the disorder-specific biological tests and treatments whose imminent arrival has been predicted since the 1970’s (Peele, 2015, this issue). Biomedical research has not identified biomarkers for mental disorders, and as Abramovitch and Schweiger (2015, this issue) argue, disorder-specific markers are unlikely to be found in the assessment arsenal of the neuropsychologist. Widespread acceptance of the biomedical model has not reduced public stigma and appears to elicit prognostic pessimism and reduced self-efficacy among individuals with psychological problems (Kichuk et al., 2015, this issue). We now understand that the conventional wisdom about psychotropic medications, such as newer generation “antidepressants” and “antipsychotics,” is mistaken. Much of the clinical trials data on the safety and efficacy of these blockbuster medications have been manipulated or hidden, financial conflicts of interest have compromised the integrity of the published literature and clinical guidelines based on it, and industry-funded drug trials are perhaps better viewed as marketing than science (Spielmann, 2015, this issue). The chemical imbalance theory of depression, and the notion that “antidepressants” work by correcting a chemical imbalance, is not and never has been scientifically credible (Lacasse & Leo, 2015, this issue). Given the lack of evidence that psychotropic medications correct a disease process, a fundamental reconsideration of their nature and effects is warranted (Moncrieff, 2015, this issue). Lastly, a dramatic increase in the use of psychotropic medications during the biomedical era has not improved societal mental health outcomes. Indeed, mental health disability rates have markedly increased in recent decades, and there is troubling evidence to suggest the long-term use of psychotropic medications may be to blame (Whitaker, 2015, this issue).

In addition to evidentiary warrant, the biomedical model may be criticized on conceptual grounds. Insel and Volkow’s biomedical approach is founded on the philosophy of eliminative reductionism (Lilienfeld et al., 2015, this issue), which posits that psychological experiences (e.g., obsessions and compulsions) can be fully reduced to their biological causes (e.g., faulty brain circuitry). From this perspective, the biological level of analysis is inherently fundamental to the psychological

level, and psychology will become superfluous once biomedical researchers have fully mapped the brain bases of behavior. There are three principal objections to this philosophy. First, whether psychology can be successfully reduced to biology is an empirical question; simply assuming such reduction on metaphysical grounds “achieves the goal (in Bertrand Russell’s famous phrase) by theft rather than honest toil” (Gold, 2009; p. 509). Second, psychological experience likely possesses complex emergent properties that cannot be reduced to genes, molecules, cells, circuits, or physiology without losing valuable information (Lilienfeld et al., 2015, this issue). Third, attempts to reduce psychological experience to biology violate established tenets of modern evolution science. A compelling defense of this objection is offered by Hayes, Sanford, and Feeny (2015, this issue), who review contemporary research suggesting that attempts to integrate psychology with neuroscience or genetics lose valuable information by ignoring the influence of context and history. They argue, “any statement that genes or the brain cause psychopathology misses the ongoing evolutionary and systemic complexity of the obtained relationships once even a short time frame is added to the picture” (p. 224).

In the United States, the lively debate about the biomedical approach that occurred during the DSM-5 (APA, 2013) revision process (e.g., *Open Letter to the DSM-5*, n.d.) has gradually faded since the publication of the new diagnostic manual. However, critical analysis of the biomedical model and the development of alternative approaches for understanding and treating mental health problems have gained momentum in the United Kingdom (e.g., Boseley, 2015; British Psychological Society, 2014). In this issue, British Psychological Society president-elect Peter Kinderman outlines a manifesto for reform that disentangles mental health services from the biomedical approach. His manifesto views the origins of distress as a product of life circumstances rather than biological disease, replaces invalid diagnoses with straightforward descriptions of problems, and emphasizes tailoring therapy to each person’s unique circumstances while promoting their agency in behavior change. Although they radically depart from the dominant biomedical approach, Kinderman’s (2015, this issue) recommendations are quite consistent with the standard practice of behavior therapy prior to the modern DSM era.

In closing, this article and special issue of *the Behavior Therapist* are intended to encourage open critical dialogue regarding the validity and utility of the biomedical paradigm that dominates the American mental health system, psychiatry, and increasingly, academic psychology (Schwartz, Lilienfeld, Meca, & Sauvigné, in press). Given their established commitment to scientific principles, members of the Association for Behavioral and Cognitive Therapies (ABCT) are positioned to make important contributions to this dialogue. We hope the articles in this special issue will facilitate an informed, productive, and ongoing discussion in keeping with ABCT’s mission of the “advancement of scientific approaches to the understanding and improving of human functioning” in order to improve the “assessment, prevention, treatment of human problems, and the enhancement of health and well-being.”

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AT ABCT

ABCT 2015: My Kind of Town, Chicago!

Patrick B. McGrath, *Local Arrangements Co-Chair*

Andrea Kass, *Local Arrangements Committee*

Pooja Dave, *Local Arrangements Committee*

Shona Vas, *Local Arrangements Co-Chair*

HELLO, AND WE WARMLY WELCOME you to Chicago (in November!). The home of Second City, great food, architecture, blues and jazz, and the most movable bridges in the world. The Local Arrangements Committee is happy to have you all join us in the Windy City this year. While the original meaning of Chicago, a one-time fur trading post on the shores of the Chicago River and Lake Michigan, meant either skunk or wild onion, the odors that you will smell now come from thousands of world-class restaurants, from Michelin rated to local favorites.

Chicago has an interesting place in the history of the U.S. We are not referred to as the Windy City because of weather, but rather because of the hot air that our politicians were blowing all around in 1890 as Chicago tried, and succeeded, in getting to host the World's Fair of 1893, the grandest fair in the history of all fairs. If you want to see a building from the fair, go to the Museum of Science and Industry—it was the only building not made of plaster as it served as the house of fine art, and no country would send their art to a building that could go up in flames so easily!

Chicago is the home of modern architecture, mostly due to the majority of the city burning down in the great fire of 1871. From the ashes rose amazing structures that influenced a home-town hero—Frank Lloyd Wright, the greatest architect of all. Chicago is the home of the Bulls basketball dynasty of the 1990s and the current Stanley Cup champions, the Blackhawks!

We expect that you will be busy with the conference, but be sure to make some time to enjoy our city. We have provided information on things you can do, including sightseeing, shopping, and theater. The Go Card Chicago offers discounts to most major Chicago attractions, and Twilight tours of the Chicago River Walk and the Loop will run each evening with Lawrence Byrne (312-282-7327). Mr. Byrne has been a tour guide for Chicago for over a decade.

He will personalize tours for groups and take you to see the highlights of the city. Tours leave from the lobby of the hotel at 7:00 P.M. on Friday and Saturday, and on Sunday afternoon. There is no charge for the tours—Mr. Byrne works solely on gratuities, which are highly appreciated. Call him directly and he will show you a Chicago that you won't want to miss.

Finally, join us Saturday night for the party and dance—we will have an improv comedy troupe, the Therapy Players (all local therapists), to entertain us, a DJ playing a great collection of hits, and a photo booth to capture memories of the evening!

Hotel and Immediate Surroundings

As in 2006, the conference is being held at the Hilton Downtown Chicago (locally known as the Conrad Hilton), located in the heart of the city. The hotel is on South Michigan Avenue, a street that runs parallel to the eastern shore of Lake Michigan and overlooks Grant Park, the lake, and the Museum Campus. Millennium Park, Chicago's most popular gathering spot, is just slightly north of the hotel. Further, the hotel is surrounded by museums, restaurants, blues music, and other bars, theaters, and clubs!

We are looking forward to using the Hilton's meeting facilities for the wonderful programming arranged by Brett Deacon and his committee. The hotel has a fully-equipped business center and free Wi-Fi available in the lobby and other public areas, in addition to the Internet packages obtained as part of your room reservation. If you bring your family, the hotel offers family packages and is centrally located for child-friendly entertainment (e.g., Maggie Daley Park and a free activity room at the Art Institute). For those who want to maintain their daily exercise regimen, there is a pool, fitness center, and walking/running track on site, although we recommend braving Chicago's brisk fall weather and running outside! Stay tuned

for information on a group run on either Friday or Saturday morning.

There are several dining options within the hotel. The 720 South Bar & Grill offers a "seasonally inspired menu with a focus on local ingredients." Have a pint and fish & chips at Kitty O'Sheas, the hotel's Irish Pub! For a quick bite, a cup of Starbucks coffee, snacks or meals to go, you can count on Herb 'n' Kitchen, the hotel's gourmet market. If you step outside the hotel, there are countless restaurants within a few blocks that offer a range of cuisines and options to fit your budget for both lunch and dinner. **Check the ABCT website for a list of nearby restaurants compiled by the Local Arrangements Committee.** Stop by the Local Arrangements Table if you would like a copy. For the foodies among us, we recommend making reservations (often weeks ahead of time!) at some of Chicago's most popular restaurants. Open Table (www.opentable.com) is a centralized system that will allow you to check availability at restaurants. However, if you have your heart set on eating at a particular hotspot, don't despair—most restaurants offer a full menu at the bar and do not take reservations for this area. Grab a seat and prepare yourself for a couple of hours of people-watching.

If you are interested in some early holiday shopping, the State Street shopping district is only a few blocks away from the Hilton. Head north on Michigan Avenue and walk two blocks west to State Street for popular department store flagships, boutique stores, and national chains. Block 37, bordered by State, Dearborn, Washington and Randolph Streets, is one of Chicago's newest shopping destinations—a 5-story atrium consisting of stores, restaurants, cafés, and direct access to public transportation.

Getting to Chicago and the Downtown Hilton

By Plane: Chicago has two major airports—O'Hare International Airport (ORD) is located approximately 20 miles northwest of downtown (a \$50 cab ride) and Midway International Airport (MDW) is 12 miles southwest of downtown (a \$35 cab ride). All major airlines fly into Chicago. O'Hare serves as a hub for United and Midway is a Southwest Airlines destination. Driving directions from the airport to the hotel may be found on the hotel's website: <http://www3.hilton.com/en/hotels/illinois/hilton-chicago-CHICHHH/>

maps-directions/index.html. Super Shuttle (www.supershuttle.com) is a cheaper option to get downtown. We recommend experimenting with Chicago's amazing public transportation system. Read on for details about getting around Chicago, including to/from the airport!

By Bus and Train: The Greyhound Bus Terminal is at 630 W. Harrison (www.greyhound.com) and the Megabus Stop (<http://us.megabus.com/>) is at 300 S. Canal. Chicago has two major downtown train stations (Union and Ogilvie) that service the Chicago suburbs (www.metrarail.com) as well as other parts of the country (www.amtrak.com). Bus and train stops are at most a mile away from the hotel (6–8 blocks walking distance). If you prefer to take a taxi from the bus/train stop to the hotel, plan on a \$5 cab ride. Check the websites for additional information.

By Car: All roads lead to Chicago! Not really, but I-55 (from the southwest), I-57 (from the south), I-80 (from the east/west), I-90 (from the east/west) and I-94 (from the north/east) sure do! The hotel is conveniently situated to access all the major highways. However, parking around the hotel is expensive—\$55 for the garage and \$69 for valet. More economical options will be available in the garages to the immediate north and west of the hotel. We recommend using the app SpotHero (www.spothero.com) to secure parking at very competitive rates.

Getting Around in Chicago

Chicago is a large city, but you will find it quite accessible via walking, public transportation, or car. Weather permitting, walking is perhaps the best way to enjoy the city as well as the architectural and artistic gems Chicago's Loop (downtown) has to offer. Chicago has an easily navigable downtown grid that makes it very walkable. It is helpful to think of Chicago as a vertical city, with Lake Michigan as a natural border to the east. Every address is relative to the 0/0 mark at the intersection of State Street and Madison Avenue, where Madison Avenue divides the city north and south, and State Street divides it east and west. Addresses are relative to the distance from that mark, with eight blocks to every mile. So, an address of 3600 North Clark means that it is 36 blocks north of Madison Avenue, and 4.5 miles north of the center of the grid.

You can use Chicago's vast public transportation system to access all areas of Chicago and the surrounding suburbs. The

Chicago Transit Authority (CTA) consists of trains, buses, and regional rail trains. All routes and times can be accessed via the CTA website (www.transitchicago.com), which includes a trip planner that can help you determine the best route and mode of transportation to and from your destination. The "city mapper" application is available for both Android and iOS devices and is also an excellent resource for trip planning on-the-go.

Our subway system is known as the "El," and is perhaps the most efficient and cost-effective way to travel around Chicago's downtown. "El" is short for "elevated" and is so named in reference to the elevated beginnings of the system (though today many parts of the system are at or below ground level). The El is a particularly fun way to get around and see the city since most of it is above ground and above the streets, allowing a view while travelling. The famed El is centered around the Loop—a loop of several train lines that circumnavigate the downtown core with each line extending off into different directions in the city. The Blue and Red lines operate 24 hours a day; the Blue line connects O'Hare airport to downtown and the Red line travels north and south via downtown, running parallel to the lake. All other routes run daily through the late evening with trains every 10 to 20 minutes. The closest El stop to the convention hotel is the Harrison stop located on the Red line. The Blue line's Jackson stop and the Orange line's Harold Washington Library stop are also within 5 blocks.

The El is arguably the fastest and easiest way to get to and from the main Chicago area airports, and is generally recommended over taxi for airport travel due to Chicago's unpredictable traffic patterns (estimated travel via taxi to/from O'Hare ranges from 20 to 90 minutes). The Blue line train travels directly between O'Hare and downtown and offers free transfers to connecting CTA train lines. If traveling from Midway airport, the Orange line can take you directly from the airport into the Loop and also offers free transfers. If you take the El, we recommend budgeting approximately 45 minutes to get to/from O'Hare and downtown, and 25 minutes if traveling in/out of Midway.

The CTA bus system is a great resource for destinations that are not directly accessed by the El. The hours and frequency of the bus routes vary, but all generally run from 4 a.m. to the late evening hours. Many buses run along Michigan Avenue and are cheap transportation

options to visit a restaurant in the South Loop or head north for some shopping on the Magnificent Mile. Finally, Metra is the regional rail system that runs 12 suburban commuter train lines from several downtown Chicago terminals. If traveling to any of the Chicagoland suburbs, Metra is your best bet. For Metra fare and schedule information, visit www.metrarail.com.

Travelers have a variety of fare options when using public transportation in Chicago. Fares for CTA trains and buses are listed at transitchicago.com/fares. Tickets are sold as reloadable "Ventra" cards and can be purchased using card or cash at a number of stations. The initial cost of a Ventra card is \$5.00, which can be used on both the El and buses. Fares are deducted from this initial amount and can be replenished at any station. The base fare for the El is \$2.25 and \$2.00 for CTA buses. However, if boarding the Blue line train from O'Hare, there is a fixed fare of \$5.00. One, three-day (\$20 and may be purchased at the airport), and seven-day unlimited passes (loadable onto your Ventra Card) are also available at any station, and offer an affordable option if planning to use public transportation frequently during your visit.

Cabs can be a very convenient way to access all parts of the city. The base fare is \$3.25 and \$1.80 for each additional mile. There is a \$2.00 airport departure/arrival tax if you choose to take a taxi for airport travel, and there is no extra charge for baggage, baggage handling, or payment by credit/debit card. No cash? No worries! All Chicago taxis are required to accept credit/debit cards. Alternatively, you may choose to use the Uber app to hail a taxi from your phone. UberX is a popular option among Uber users because it offers a more affordable alternative to the standard taxi fares, and it is staffed by local drivers who are available to pick you up and take you anywhere you want to go in the city. However, Uber drivers are only available for travel to airports, and are not allowed to pick up passengers at any airports.

Things to Do in Chicago

Nearby Attractions

As we continue to boast, Chicago is an incredible city with lots to do, much to see, and plenty to eat. The proximity of the hotel to Millennium and Grant Parks will make for a conference treat! Step outside and "discover a state-of-the-art collection of architecture, landscape design and art" in the beautiful Millennium Park

(www.millenniumpark.org). Stroll through the 5-acre urban Lurie garden, take a selfie at the famous Cloud Gate “Bean” sculpture, play in the interactive Crown Fountain, or go ice skating in the outdoor street-level McCormick Tribune Ice Rink (scheduled to open November 13, weather permitting).

Art and Architecture

From Millennium Park, take the Nicholas Bridgeway (a 625-foot pedestrian bridge) to the Art Institute of Chicago and see why it is ranked as TripAdvisor’s #1 of things to do in Chicago. Located a half-mile from the Hilton, we recommend a visit here (<http://www.artic.edu/>) to enjoy art from different eras. Prefer a more modern art style? In addition to the Art Institute’s amazing new modern wing, the Museum of Contemporary Art is also downtown and just 1.8 miles north of the hotel (<http://www2.mcchicago.org/>).

Deemed “a laboratory for architectural innovation and experimentation,” the Chicago skyline is a major part of this city’s charm. Learn about the history and innovation of the city from the Chicago Architectural Foundation, located four blocks from the Hilton. They offer tours of the city on foot, train, bus, or boat with experienced docents. The river cruise is a local and visitor favorite! Alternate “boat tour” companies include Shoreline Sightseeing and Wendella Boats. CONVENTION 2015

Downtown Delights

Chicago’s Magnificent Mile is “one of the great avenues of the world,” located on North Michigan Avenue (<http://www.themagnificentmile.com/>). Shopping, dining, and entertainment adorn this 13-block stretch just north of the Chicago River. For a breathtaking view of the city, visit 360 Chicago, an observation deck on the 94th floor atop the John Hancock Center and at the northern end of the Magnificent Mile (<http://www.360chicago.com/>). While there, hold onto the railings of TILT, a “moving experience” that tilts you forward to a 30-degree angle as you overlook the Magnificent Mile 1,000 feet below. Want the skyscraping views but prefer to sip cocktails instead? The Signature Room has delicious food and drinks on the 95th floor of the John Hancock Center, one of Chicago’s tallest buildings (<http://www.signatureroom.com/>). For a sweet treat or souvenir to bring back home, stop at one of the Garrett Popcorn Shop locations and pick up a bag of the Garrett Chicago Mix: a delicious combination of caramel and

cheese popcorn. Also to the hotel’s north is Navy Pier, a Chicago landmark that features local attractions like the Ferris Wheel, the Children’s Museum, the Chicago Shakespeare Theater, and the IMAX theater (<https://navypier.com/>).

Museums

Less than one mile south of the Hilton is the Museum Campus, home to the Field Museum (<http://www.fieldmuseum.org/>), Shedd Aquarium (<http://www.sheddaquarium.org/>), and Adler Planetarium (<http://www.adlerplanetarium.org/>). Animal lovers can visit the Lincoln Park Zoo and experience a “wild” outing with family or friends. Open year round and free to the public, the Zoo is a short cab or El ride from the convention hotel (<http://www.lpzoo.org/>).

Want to soak in more history, art, and science? Venture 6.7 miles south to Hyde Park and visit Chicago’s Museum Campus South (<http://www.visitmuseumcampus-south.com/>), which comprises seven fantastic museums that surround the historic site of the Chicago World’s Columbian Exposition. You may also know Hyde Park as the home of the Obama Family and The University of Chicago!

Theater

Broadway theater and the arts are just steps from the hotel, with many excellent shows over the duration of the convention. Theater includes Blue Man Group, Million Dollar Quartet, *Kinky Boots*, Chicago Jazz Philharmonic, Chicago Symphony Orchestra, and you can all join Alec Pollard in seeing his favorite band ABBA in *Mamma Mia!* Alec secretly coordinated ABCT in Chicago just for this!!! Look here for local listings and tickets: <http://www.chicago-theater.com/>; <http://broadwayinchicago.com/>.

For a different kind of theater experience, check out Chicago’s well-known improvisation comedy theaters. The Second City offers a multitude of comedy shows and improv sketches across its many stages, located in Old Town (<http://www.secondcity.com/>). We also recommend the improv shows at the iO Theater, located in Lincoln Park (<http://ioimprov.com/chicago/>).

Music

Sweet Home Chicago is definitely a blues town! Buddy Guy’s Legends is across the street from the back entrance of the hotel (700 S. Wabash). The House of Blues (329 N. Dearborn), where “music feeds the

soul,” is a quick cab ride away. For the jazz fans, Jazz Showcase (806 S. Plymouth) is only 2 blocks away and Andy’s (11 E. Hubbard) is one of the best jazz clubs in the city.

Outdoor Activities

Although Chicago’s weather can be unpredictable, we encourage those who are willing to brave the elements to take advantage of Chicago’s constantly expanding array of outdoor activities. Join walkers, joggers, and bikers along the Chicago Lakefront to experience views of beautiful Lake Michigan and our amazing city skyline. You need only walk two blocks east from the hotel (crossing over Grant Park) to hit the water’s edge. Walk a little north to experience the newly opened Chicago Riverwalk, which has relaxing seating areas, bars and dining, and a Vietnam Veteran’s memorial. For a little more adventure, take a cab or the El’s Blue line to The 606, Chicago’s new 2.7 mile above-ground trail and park (<http://www.the606.org/>). Jump on or off this highline at one of several entry points at your convenience: the 606 is open daily from 6 A.M. until 11 P.M.

Weather

November tends to be rather cool in the Windy City, with an average high of 48 and low of 32 degrees Fahrenheit. Chicago’s first snowfall of the winter usually arrives in November (average total of 1.2 inches in November), so it is possible that you will be able to enjoy the beauty of a white Chicago during your visit! Any Chicago native will tell you that the best way to brave a Chicago winter is layers, layers, layers! So, be sure to pack your sweaters, jackets, warm socks, and boots!

See You Soon, ABCT

We are at your service! If you have any questions about Chicago, please feel free to email us and we will be glad to assist you (Patrick: patrick.mcgrath@alexian.net; Andrea: akass@bsd.uchicago.edu; Pooja: pdave@bsd.uchicago.edu and Shona: svas@bsd.uchicago.edu). Keep checking the website and ABCT listserv for information on Dining with a Chicagoan (dinners have been arranged for Friday and Saturday nights), a fun run, and other tidbits about the Windy City. We will have a table at the conference near the Registration booths, so stop by and let us assist you with where to go and what to do. We look forward to seeing you all in Chicago!!

call

for

Continuing Education Sessions

Please send a 250-word abstract and a CV for each presenter.

For submission requirements and information on the CE session selection process, please see the Frequently Asked Questions section of the ABCT Convention page at www.abct.org.

50th Annual Convention | October 27–30, 2016 | New York

Workshops and Mini Workshops

Workshops cover concerns of the practitioner/educator/researcher. Workshops are 3 hours long, are generally limited to 60 attendees, and are scheduled for Friday and Saturday. Please limit to no more than FOUR presenters.

Mini Workshops address direct clinical care or training at a broad introductory level. They are 90 minutes long and are scheduled throughout the convention.

Please limit to no more than FOUR presenters.

When submitting for Workshops or Mini Workshop, please indicate whether you would like to be considered for the other format as well.

Barbara Kamholz, Workshop Committee Chair
workshops@abct.org

Institutes

Institutes, designed for clinical practitioners, are 5 hours or 7 hours long, are generally limited to 40 attendees, and are scheduled for Thursday.

Please limit to no more than FOUR presenters.

Lauren Weinstock, Institute Committee Chair
institutes@abct.org

Master Clinician Seminars

Master Clinician Seminars are opportunities to hear the most skilled clinicians explain their methods and show taped demonstrations of client sessions. They are 2 hours long, are limited to 40 attendees, and are scheduled Friday through Sunday.

Sarah Kertz, Master Clinician Seminar Committee Chair
masterclinicianseminars@abct.org

DEADLINE

for Submission : February 1, 2016

Nominations for ABCT Officers: The Quality of Leadership

Christopher Martell, *Chair, Leadership and Elections Committee*

The American Society for Association Executives (ASAE) has posted six key attributes of board members. The first, “the ability to think strategically and analytically and to effectively communicate thoughts and the reasons for them” (ASAE CEO Symposium, © 2014 Tecker International, LLC; www.asaecenter.org/) describes the membership of ABCT perfectly. Our association is filled with bright, talented people that already serve in leadership positions in a variety of contexts, or who are well on their way to leadership because they possess the ability to “think strategically and analytically.” It is time once again to nominate colleagues, or to self-nominate for positions in ABCT.

Historically, well below half of our members make decisions that affect the entire membership. Even fewer members participate in the nominations process. We all can contribute to ABCT as our professional home, and help in guiding the future of ABCT by participating in the election of the association’s leadership. Also, please consider running for an office! Self-nominations are accepted. If you don’t wish to hold an office, nominate someone you know who would be willing and capable of holding an office. We honor our colleagues by nominating them.

There are many reasons why people either do not run for offices, or do not participate in the nominations process. Some believe they do not have a sound understanding of how ABCT operates, what the various positions in governance do or how decisions are made. Many people wonder

about the kind of time commitment ABCT is asking of its volunteer leadership. Allow us to make the process transparent by attending “Getting Involved and Running for ABCT Office,” Sunday, November 15, 9 to 10:00 A.M., Conference Room 4A. Members of the Leadership and Elections Committee, Christopher Martell (Chair), Debra Kaysen, and David Pantalone, along with Linda C. Sobell (Past President) and Mary Jane Eimer (ABCT’s Executive Director), will provide an overview and advice on how you can build your vitae by getting involved in ABCT.

This coming year we need nominations for two elected positions: President-Elect and Representative-at-Large. Those members who receive the most nominations for the slates available will go forward to appear on the ballot. In April, members in good standing vote for the candidates of their choice to serve for 3 years. The President-Elect serves in that function from 2016–2017, then serves as President from 2017–2018, and then serves as Past President from 2018–2019.

Each of the Representatives serves as a liaison to one of the branches of the association. The representative position up for 2016 election will serve as the liaison to the Academic and Professional Issues Coordinator and Committees. Their term of office will be from November 2016–November 2019.

All full members in good standing are eligible to be nominated, and there is no limit to the number of members you can nominate for any of the positions. Accord-

ing to ABCT’s bylaws, we require two candidates for President-Elect and three candidates for Representative-at-Large. As you consider colleagues you would like to nominate, or if you consider a self-nomination, keep in mind some of the ASAE’s suggestions regarding key attributes of board members. For example, members of the board should have earned respect of other key stakeholder group members, have the ability to work well with others and be faithful in their duties, have an earned reputation for emotional maturity, personal integrity, and honesty, and, finally, demonstrate familiarity with the body of knowledge related to the process and the subject area within which decisions and choices will be made. Electioneering starts at the Annual Convention. So, if you have a candidate in mind, or wish to nominate yourself, start the campaign now with the nominations and go to the Annual Convention and start making your case to the electorate. Remember, the candidates with the most nomination will ultimately be the only official names on the ballot: two for President-elect and three for Representative at Large.

How to Nominate: Three Ways

- ➔ Mail the form to the ABCT office (address below)
- ➔ Fill out the nomination form by hand and fax it to the office at 212-647-1865
- ➔ Fill out the nomination form by hand and email the PDF as an attachment to membership@abct.org.

ABCT needs your participation to insure good governance to continue to thrive as one of the world’s leading associations representing behavioral and cognitive therapies. Let’s make this an exemplary year for numbers of nominations and ultimately for percentage of members casting votes!

I nominate the following individuals:

PRESIDENT-ELECT (2016–2017)

REPRESENTATIVE-AT-LARGE (2016–2019)

NAME (printed) _____

SIGNATURE (required) _____

Every nomination counts! Encourage colleagues to run for office or consider running yourself. Nominate as many full members as you like for each office. The results will be tallied and the names of those individuals who receive the most nominations will appear on the election ballot next April. **Only those nomination forms bearing a signature and postmark on or before February 1, 2016, will be counted.**

Nomination acknowledges an individual’s leadership abilities and dedication to behavior therapy and/or cognitive therapy, empirically supported science, and to ABCT. When completing the nomination form, please take into consideration that these individuals will be entrusted to represent the interests of ABCT members in important policy decisions in the coming years. Contact the Leadership and Elections Chair for more information about serving ABCT or to get more information on the positions.

Please complete, sign, and send form to: Christopher Martell, Ph.D., Leadership & Elections Chair, ABCT, 305 Seventh Ave., New York, NY 10001.



ABCT | Welcome, Newest Members!

Associate

Brjánn Ljótsson
Kristin Kicki Martinsen
Joshua Patras
Jennifer Plotnek

Full Members

Regina Abramoff
Kaltham Jabor Al-Kuwari
Cecilia Arlinger Karlsson
Simon Beaulieu-Bonneau
Steven Behling
Mitchell Berman
Lisa Bolden
Elizabeth Brokamp
Michelle Nicole Burns
Andrew James Carini
Anthony Chambers
Brian Andrew Chapman
Jennifer Y. Chen
Jeffrey Danforth
Sapna Doshi
Claudia Drossel
Karyn Erkfritz-Gay
Shawn Christopher Ewbank
Natalia Ferrero
Dalia Gefen
Dorothy Grice
Ryma Talaat Hady
Christopher Hansard
Rebecca Hanson Richardson
Amy M. Jacobsen
Sheila Josephs
Sarah Keedy
Ernst Koster
Martin Lamm
Steven Lawyer
Tara Levinson
Elizabeth McMahon
Patrick David McMahon
Aja Meyer
Thomas Daniel Meyer
M. Ellen Mitchell
Katherine Niemela
Velizar T. Nikiforov
Sarah Victoria Revels
Thomas Lee Robertson
Frederick Rotgers
Valerie Saltz
Jessica Samson

Josefin Särnholm
Jacquelyn Norry Smith
Jacqueline Blair Sperling
Tyrel John Starks
Emel Stroup
Jennifer Taub
Christina J. Taylor
Jan Tyson Roberts
Patrick Anthony Vogel
Lyndsay Kate Volpe Bertram
Mona Williams
Lawrence David Willison
Fred Waltzer

New Professionals

Stephanie Ann Beck
Katrina Marie Bell
Sarah Bellovin-Weiss
Stacey R. Belmont
Rebecca Rialon Berry
Rebecca Blais
Katrina Cook
Danielle Doucette
Lisa Dreger
Daniella Ganger
Ivan Gonzalez
Mariana Hoar
Heather M. Jones
Corina Evelyn Klein
Catherine Maliszewski
Ashley Marzullo
Amy M. Williams

Postbaccalaureates

Leigh Alexander Andrews
Peter Peek Ehlinger
Leah Kate Feinberg
Rachel Hannah Grasfield
Natalie Hong
Lindsay Hylek
Yuri Kim Kim
Nicholas Klinkefus
Alison Legrand
Rebekah Mennies
Damian Morden-Snipper
Amanda Kate Nixon
Jessica Samantha Stinnette
Stephanie Violante
Elizabeth Mary Waldron

Katherine A. Walukevich

Students

Paula Aduen
Suzanne Chinyere Amadi
Brenda Arellano
Nicholas A. Armenti
Hallie Avizad
Danielle Elyse Baker
Mary Becker
Rachelle Bernadel
Johan Bjureberg
Bjureberg
Sarah R. Black
Marianne Bonnert
Colin Maxwell Bosma
Summer Bottini
Ashley Braun-Gabelman
Morgan Brazille
Teah-Marie Bynion
Bridget Cauley Cauley
Kaitlyn Diane Chamberlain
Dominique Cheung
Annie Chinneck
Elisabeth Cordell
Amanda H. Costello
Alexandra D'Agostino
Issar Daryanani
Alan K. Davis
Kimberlye Dean
Lee Del Priore
Allison Detloff
Justine Diener
Cody G. Dodd
Sarah Dreyer-Oren
Emily R. Dworkin
Cierra Brooks Edwards
Rebecca Emery
Kamran Eshtehardi
Reina Factor
Emily Fanguy
Jason Feinberg
Haley Ford
Lauren Friedman
Alyssa Gatto
Ansley Geno Geno
Tiffany Rose Glynn
Marie-Helene Gosselin
Christopher Grandits
Fiona Grubin
Grace Gu
Shaina Gulin

Laura Katherine Hansen
Jenny Hardy
Gregory Harms
Alexa Hays
Sean Hollis
Melanie Hom
Shabnam Hossein
Claire Houtsma
Mortie Hvenegaard
Melissa Ivins-Lukse
Jennifer Dianne Jackson
Maryanne Jaconis
Anjali Tanya Jain
Alecia Jarvis
Carly Johnco
Gilly Kahn
Faria Kamal
Andrea Kaniuka
Anna Michelle Karam
Jonathan Katibian
Benjamin William Katz
Michelle Kaufman
Nathan T. Kearns
Mun Yee Kwan
Julian Christopher Lagoy
Shari Lagrange-Aulich
Maria Lalouni
Alisson Lass
Matthew Lebowitz
Cara Levitch
Anne Limowski
Nicholas A. Livingston
Ira Lowinger
Shannon Marie Manley
Gabrielle Marcotte-Beaumier
Jaime Marrus
Ana Martinez de Andino
Kristen G. Merkitch
Berhane Messay
Isha Walwina Metzger
Alexandria Meyer
Michelle L. Miller
Raymond Lee Moody
Kelly Elizabeth Moore
Ursula Susan Myers
Kara Lynn Nayfa
Rachel Nelson
Christine M. Newman
Danielle N. Newman
Binh-an Nguyen
Kimberly O'Leary
Marykate T. Oakley
Clarissa Ong

Joseph Orsini
Janelle Painter
Tyler Pendleton
Natalie Marie Perkins
Christina Pimble
Caitlin Mary
McNamara Pinciotti
Kelly L. Polnaszek
Natalee N Price
Sahar Rabizadeh
Jonathan Rabner
Sharon Radomski
Lene-Mari Rasmussen
Nathalie Ricard
Julia Rickard
Caroline Mason Roberts
Rachael Rogers
Leah Rothschild
Kim Aisling Rottier
Caryn Kseniya
Rubanovich
Joshua Rutherford
Hanna Sahlin
Rachel Hannah Salk
Laurel Sarfan
Jenna Lillian Schleien
Gabrielle Schreyer
Andrew Joel Schwehm
Susan R. Schwendener
Rakhel Shapiro
Karen Shebuski
Isaac Smith
Donya J. Sorensen
Ian H. Stanley
Elizabeth Stein
Hara Stephanou
Melissa Marie Stewart
Philip Stoner
Eivind Rauø Strand
Alexandra Sullivan
Kristen Thompson
Matthew A. Timmins
Annabelle Torsein
Elise Trim
Sophie Louise van Uijen
Stephanie Vettorazzi
Martin Viola
Lori A. Wagner
Samantha Walsh
Lindsey Whitcomb
Bonnie Yap
Wanni Zhou

Anne Marie Albano Early Career Award for Excellence in the Integration of Science and Practice

Dr. Anne Marie Albano is recognized as an outstanding clinician, scientist, and teacher dedicated to ABCT's mission. She is known for her contagious enthusiasm for the advancement of cognitive and behavioral science and practice. The purpose of this award is to recognize early career professionals who share Dr. Albano's core commitments. This award includes a cash prize to support travel to the ABCT Annual Meeting and to sponsor participation in a clinical treatment workshop.

Eligibility requirements are as follows: 1) Candidates must be active members of ABCT, 2) New/Early Career Professionals within the first 5 years of receiving his or her doctoral degree (PhD, PsyD, EdD). Preference will be given to applicants with a demonstrated interest in and commitment to child and adolescent mental health care.

Applicants should submit: Nominating Cover Letter, CV, Personal Statement up to three pages (statements exceeding 3 pages will not be reviewed), and 2 to 3 supporting letters. Application materials should be emailed as one pdf document to Awards.ABCT@gmail.com. Include candidate's last name and "Albano Award" in the subject line. Also, mail a hard copy of your submission to ABCT, Anne Marie Albano Early Career Award, 305 Seventh Ave., New York, NY 10001.

This award is made possible by a generous donation to ABCT. A family who benefitted from CBT and knows of Dr. Albano's work expressed wanting to see others benefit from CBT and CBT-trained therapists.

Nomination Deadline: March 1, 2016

Student Dissertation Awards

- Virginia A. Roswell Student Dissertation Award
- Leonard Krasner Student Dissertation Award
- John R. Z. Abela Student Dissertation Award

Each award will be given to one student based on his/her doctoral dissertation proposal. Accompanying this honor will be a monetary award to be used in support of research (e.g., to pay participants, to purchase testing equipment) and/or to facilitate travel to the ABCT convention. Eligibility requirements for these awards are as follows: 1) Candidates must be student members of ABCT, 2) Topic area of dissertation research must be of direct relevance to cognitive-behavioral therapy, broadly defined, 3) The dissertation must have been successfully proposed, and 4) The dissertation must not have been defended prior to November 2015. Proposals with preliminary results included are preferred. To be considered for the Abela Award, research should be relevant to the development, maintenance, and/or treatment of depression in children and/or adolescents. Self-nominations are accepted or a student's dissertation mentor may complete the nomination. The nomination must include a letter of recommendation from the dissertation advisor. Please complete the nomination form found online at www.abct.org/awards/. Then e-mail the nomination materials (including letter of recommendation) as one pdf document to awards.abct@gmail.com. Include candidate's last name and "Student Dissertation Award" in the subject line. Also, mail a hard copy of your submission to ABCT, Student Dissertation Award, 305 Seventh Ave., New York, NY 10001. **Nomination deadline:** March 1, 2016

Nominations for the following award are solicited from members of the ABCT governance:

Outstanding Service to ABCT

Please complete the nomination form found online at www.abct.org/awards/. Then e-mail the completed form and associated materials as one pdf document to awards.abct@gmail.com. Include "Outstanding Service" in the subject line. Also, mail a hard copy of your submission to ABCT, Outstanding Service to ABCT, 305 Seventh Ave., New York, NY 10001.

Nomination deadline: March 1, 2016

the Behavior Therapist
Association for Behavioral
and Cognitive Therapies
305 Seventh Avenue, 16th floor
New York, NY 10001-6008
212-647-1890 | www.abct.org

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