5 Neuroscience and Mental Illness

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The fast-developing field of neuroscience has given philosophy, as well as other disciplines and the public broadly, many new tools and perspectives for investigating one of our most pressing challenges: addressing the health and well-being of our mental lives. In some cases, neuroscientific innovation has led to clearer understanding of the mechanisms of mental illness and precise new modes of treatment. In other cases, features of neuroscience itself, such as the enticing nature of the data it produces compared to previous behavioral methods, together with its costliness and "coldness" have complicated understanding of mental illness and decision making about mental illness. Taking neuroscientific information into account can leave practitioners in psychiatry and law with difficult questions, stemming not only from the complexity of these fields, but also from our rapidly evolving understanding of and facility with neuroscience. In this chapter, we will review several examples of the insights and dilemmas that have unfolded as mental illness has been examined through the lens of neuroscience, covering diagnoses such as obsessive-compulsive disorder (OCD), schizophrenia, addiction, and severe mood disorders.

In the first section, we will consider issues surrounding the introduction of the tools and methodologies of neuroscience to clinical research and treatment. We illustrate first with the case of treatment-resistant OCD, where being able to move from behavioral to biological explanations has led to clinical breakthroughs. Our second illustration considers efforts to identify a unifying explanation for the symptoms of schizophrenia. In this case, the contributions of neuroscience have led to little agreement, leading some to worry whether a single cohesive disorder explains the various phenomena.

In our second section, we explore how this kind of uneven progress in the identification of biological explanations for mental illness has affected our

theoretical understanding both of what mental illnesses are and the appropriate ways of investigating them. We describe change at the level of national mental health policy and institutional guidelines stemming from the influence of neuroscience. In particular, we examine the National Institute of Mental Health's (NIMH) recent challenge of the ascendancy of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) as a guiding framework for psychiatric research with their release of the Research Domain Criteria (RDoC) framework, aimed at understanding mental illness as dysfunction in general psychological and neural systems (Kraemer, 2015). This move highlights a deeper issue about cognitive ontology or the frameworks we use to delineate the mind and brain into relevant component parts, using terms, for example, such as "functional system," "neural mechanism," or "brain area." These frameworks can shape what qualifies as causal information and what does not.

Finally, our third section looks at how neuroscientific information about mental illness has affected both the clinic and the public more generally. For one, consumption of neuroscientific evidence in the courtroom has become a hot button issue, since it appears that these kinds of explanations can increase stigmatizing judgments of those suffering with mental illness. At the same time, there are areas in which neuroscience itself is viewed with strong skepticism—neurobiological and neuropharmacological interventions being characterized as too expensive, too inhuman, or even outright harmful. Like genomics, artificial intelligence, and other recently developed scientific technologies, neuroscientific findings meant to contribute to decision making about mental illness face significant public debate and controversy.

We summarize with an overview of how neuroscience has informed the philosophical understanding of mental illness, despite and because of the complexity it has brought with it. We describe our expectations for the future direction of neuroscience on the topic of mental illness across disciplines and with the public.

5.1 Mental Illness and Neuroscientific Methodology

Neuroscientific research in a medical context evokes images of complicated and expensive machines and apparatuses from the late twentieth and early twenty-first century. You are probably thinking of lying in the center of a large white drum that can scan your head by using magnets (as in functional magnetic resonance imaging [fMRI]), emitting x-rays (commonly known as computed tomography), or detecting radioactive "tracer" substances (as in

positron emission tomography). Or maybe you are thinking of wearing a cap of electrodes that can record electrical activity, as in electroencephalography (EEG). Each of these tools is a method of neuroimaging that can tell us something different about the brain and about mental illness. But before diving into *what* they can tell us, it is useful to think about *why* clinical scientists have come to rely on them, rather than on earlier tools involving behavioral methods.

Briefly, the 1970s ushered in a paradigm shift in thinking about mental illness. From a theoretical standpoint, this meant a change in focus from behavioral to biological explanations, as viewing the brain as a biological organ composed of cells and organized in a particular structure allowed mental illnesses to be understood as biologically grounded. Institutionally, this meant a restructuring of the standard document used to describe and diagnose mental disorders, the DSM. By the time the DSM-III was published in 1980, it had been reorganized based on descriptive definitions of mental disorders, introducing what are known as "operational diagnostic criteria"—lists of symptoms that could be independently and reliably identified by clinicians. Diagnosis of a mental disorder could now be accomplished through assessment of presenting symptoms such as depressed mood, fatigue, or insomnia—symptoms with biological or "biomedical" explanations.

Whether the DSM or the broader discipline of psychiatry has landed on the "right" concept of what mental disorders are is fiercely debated (Aragona, 2015). For now, we only need the uncomplicated point that both mental disorders and the symptoms that underlie them can be usefully targeted with the tools and methods of biomedical science—more specifically, neuroscience, the science of the brain. It is widely understood that mental illness involves other more complicated factors such as genetic and socioenvironmental influences.[1] Nevertheless, when trying to understand what has gone wrong with the mind, looking at its major organ is an appropriate place to start. In the next section, we look at an example where this starting orientation has provided us with major breakthroughs: tools for intervention in treatment-resistant OCD.

5.1.1 Neurostimulation and OCD

OCD is a mental disorder commonly characterized by repetitive intrusive thoughts or obsessions that the individual tries to suppress and/or repetitive behavioral or mental rituals performed in an effort to neutralize distress. It is thought to affect approximately 2–3 percent of the population or about

one in fifty people (Milad & Rauch, 2012). Like many familiar DSM diagnoses, OCD is highly comorbid with other mental disorders, in particular anxiety and depression, and is closely related to a few idiosyncratic behavior patterns that are understood as their own diagnoses (including hoarding, trichotillomania or hair pulling, and some forms of body dysmorphia). Perhaps unsurprisingly, studies of healthy populations have found that as many as 50–75 percent of people have experienced intrusive thoughts from time to time (Clark & Rhyno, 2005). The key difference between clinical and nonclinical presentations of such thoughts lies not in the quantity of intrusive thoughts alone, but rather in the appraisals of those thoughts and the cognitive effects of the appraisals. That is, while most individuals do not consider intrusive thoughts relevant to the self, do not feel much guilt about them, and are less concerned about suppressing them, individuals with OCD find the thoughts salient and unacceptable, experience intense guilt, and spend a good deal of time and concentration attempting to suppress or resist them[2] (Clark & Rhyno, 2005; Collardeau, Corbyn, & Abramowitz, 2019).

The neuroscientific study of OCD has provided a wide range of information about the mechanisms of the disorder. For our purposes, here are three key insights:

1. *Evidence of cortical–subcortical circuit disruption*—Malfunctioning cortical–striatal–thalamic–cortical (CSTC) circuits are known to be key in OCD, as is corticostriatal–limbic activation (Milad & Rauch, 2012; Stein, Goodman, & Rauch, 2000). For instance, Stein and colleagues (2000) theorize that striatal lesions lead to certain evolutionarily supported behaviors, such as hoarding supplies or washing one's hands, being performed in inappropriate excess. Moreover, it has long been known that the orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), and the caudate nucleus have been shown to be overactive among patients with OCD in resting state as well as being particularly distinctive after a patient has been presented with a stimulus related to one of their obsessions or fears (Rauch et al., 1998).
2. *High activity in language areas*—Using fMRI, Kuhn and colleagues (2013) found that language circuits in the brain, and Broca's area in particular, were associated with intrusive thoughts, indicating that they may be represented linguistically. Furthermore, in participants more prone to experiencing recurrent intrusive thoughts, these areas showed more activation during resting state (Kuhn et al., 2013).

3. *Memory and imagination in OCD*—Studies of the neural correlates of counterfactual imagination also offer insight into the mechanisms of OCD in the brain. In particular, the brain encodes mental imagery similarly to when one is actually seeing the scene. Thus, when a patient with OCD replays a certain scene in order to check the veracity of a memory, the brain treats what is being pictured as interchangeable with the actual memory. As such, increased checking paradoxically decreases certainty and only serves to exacerbate the memory distrust that is symptomatic of OCD (Radomsky, Rachman, & Hammond, 2001; Radomsky, Gilchrist, & Dussault, 2006; Moritz & Jaeger, 2018; Tyron & McKay, 2009).

Fortunately, when treated early, youth with OCD have up to a 40 percent chance of significantly recovering as they reach adulthood, while adults with OCD have a 20 percent chance of recovery over forty years. In order to maximize chances of recovery, the most common treatment plan includes exposure and response prevention, a type of cognitive behavioral therapy (CBT), alongside pharmaceuticals such as selective serotonin reuptake inhibitors (SSRIs), clomipramine (a tricyclic antidepressant), or antipsychotics. Exposure and response prevention targets the anxiety caused by increased checking and other mental rituals. During the intervention, patients are hierarchically exposed to aversive situations and stimuli while the clinician encourages them to resist performing compulsions and to instead wait until the anxiety passes. This allows the brain an opportunity to habituate to the stimuli and to a sense of calm.

Exposure and response prevention programs are effective for less severe cases of OCD. This has been demonstrated with neural findings, including decreased overactivity in the OFC after treatment (Shin et al., 2013). In addition, for a broad range of severity levels, patients' OFC, ACC, and caudate nucleus activation more closely resembles that of healthy controls after combined pharmacotherapy and exposure and response prevention (Rauch et al., 1998). Hauner and colleagues (2012) likewise found distinct changes in the ACC, insula, amygdala, and visual cortex when patients were confronted with previously aversive stimuli after exposure and response prevention treatment.

What about severe, treatment-refractory cases? If patients show no improvement after thirty or more hours of CBT intervention and various courses of SSRIs and other pharmaceuticals, more serious intervention may be warranted (Veale et al., 2014). Because we can use neuroimaging

techniques to observe areas of high activity directly in treatment-refractory cases, it is possible to target these areas directly. Indeed, transcranial magnetic stimulation, an intervention that painlessly transmits an electromagnetic pulse through the scalp to superficial brain targets, including the prefrontal cortex, supplementary motor area, and OFC, has shown promising results in treating OCD, as measured by both behavioral and neural outcome variables in an array of studies, including blinded randomized controlled trials (Cocchi et al., 2018; Goyal, Roy, & Ram, 2019).

Another method of directly targeting overactive areas of the brain comes in the form of deep brain stimulation (DBS). This technique, in which microelectrodes are surgically implanted in the patient's brain, has gradually become preferred over more invasive methods used in the past such as lesions of the OFC (Alonso et al., 2015). Recently, DBS devices have been used experimentally with success in areas outside the OFC, including the nucleus accumbens (NAc) and some areas of the striatum (Figee et al., 2013; Huys et al., 2019). One DBS intervention study saw at least a 50 percent reduction in symptoms in 85 percent of the otherwise treatment-refractory patients (Barcia et al., 2019).

Clearly, OCD is an excellent example of an illness where clinical insight has been directly gained from neural findings associated with individuals' symptoms, and where innovative, minimally invasive therapies have improved even the most severe cases. Unfortunately, there are other less encouraging examples where the neural mechanisms of psychiatric illness are less clear. In the next section, we will take a look at recent efforts to understand schizophrenia from a neuroscientific perspective.

5.1.2 The Heterogeneity of Schizophrenia

In the DSM-5, schizophrenia is a disorder characterized by psychotic states (periods of acute hallucinations, delusions, paranoia, and disorganized speech and behavior), as well as, in some cases, lack of affect, avolition, and catatonia. Outside of any psychotic episodes, schizophrenia is characterized by deficits in a number of cognitive abilities such as working, episodic, and declarative memory, semantic processing, attention, and verbal learning.[3] As might be imagined, a disorder with such heterogeneous symptoms makes for a difficult research target. While human research populations in psychiatry are commonly drawn from those diagnosed using DSM diagnostic criteria and rated according to a standard scale, schizophrenia researchers are often interested in particular symptoms in isolation—one lab may be focused

on disordered speech, and another on hearing voices. Indeed, schizophrenia is not just phenotypically but also genetically heterogeneous (Liang & Greenwood, 2015), as genome-wide association studies over the past decades have failed to find anything like the "genes for schizophrenia" (Adriaens, 2008). That is, rather than finding any particular alleles that account for the diagnosis, schizophrenia appears to be merely weakly related to hundreds of different mutations that shape individual neurocognitive variation. This overall picture has led some theorists to argue that what we know as one disorder is really a category that subsumes many related though distinct disorders (cf. Adriaens, 2008; Gruzelier, 2011; Liang & Greenwood, 2015).[4]

Nevertheless, individuals diagnosed with schizophrenia often receive effective neuropharmacological intervention in the form of antipsychotics. This strategy may be more like the proverbial hammer than the scalpel, but its efficacy is strong evidence that the dopaminergic system is one important factor in schizophrenia (because both first- and second-generation antipsychotics work to inhibit dopamine activity in the brain; Davis et al., 1991; Grace & Gomes, 2018; McCutcheon et al., 2019). However, the exact role that dopamine plays in neural systems in schizophrenia is unclear. This may be because of the inherent limitations on research on human subjects.

As Kesby and colleagues (2018) point out, basic research is often conducted using rodents as animal models (Jones, Watson, & Fone, 2011; Marcotte, Pearson, & Srivastava, 2001; Tomasella et al., 2018). In these studies, positive symptoms are induced in the animal subjects using psychostimulants. Unfortunately, there are key differences between these models and findings in clinical research with humans. While dopaminergic activity in subcortical regions is associated with positive symptoms both in humans and in nonhuman animal models, the precise location of the activity has been found to differ: dopaminergic activity in the striatum is an important correlate of positive symptoms in humans, whereas animals' symptoms are associated with dopaminergic activity in the limbic system (Kesby et al., 2018). Kesby and colleagues propose using more appropriate animal models in future studies so that the molecular-level causes can be better understood.[5]

Another approach to the neural understanding of schizophrenia focuses on electrical activity in the brain and what is referred to as the oscillatory connectome—the back and forth "harmonic" patterns of electrical activity in the brain during its resting and working states. Using EEG to measure event-related potentials, the electrical response of the brain to a particular

stimulus or "event," Light and Swerdlow (2015) report that a reliable biomarker of schizophrenia severity is low mismatch negativity. Mismatch negativity occurs in response to an unexpected stimulus (e.g., a red square amid a series of blue circles, a consonant amid a series of vowels, or a "no-go" signal amid a series of "go" signals). Briefly, response to an expected stimulus generally involves a rise in potential to around 1–2 mV as recorded by EEG and a return to zero within the first 400 milliseconds. An unexpected stimulus, on the other hand, involves a rise above or around 1–2 mV, generally followed by a drop *below* zero. This difference in amplitude constitutes the mismatch negativity. In short, the brain reacts more strongly when it is surprised.

In individuals with a diagnosis of schizophrenia, difference in response amplitude between expected and unexpected stimuli is much smaller than in controls. More recent studies have also associated this low mismatch negativity with particular brain regions. Among individuals with schizophrenia, expected stimuli prompted lessened thalamocortical activation compared to controls, and unexpected stimuli produced reduced cortical connectivity in comparison to controls (Lee et al., 2019). Synthesizing related research, Nagai and colleagues (2013) report that "mismatch negativity amplitude reduction reflects sensory network dysfunction in schizophrenia" (p. 2).

What are we to make of this conclusion? Is sensory network dysfunction an additional neurological "symptom" of the schizophrenia diagnosis to be added to the list of cognitive behavioral deficits that these individuals experience? Does it underlie one or more of those symptoms, or play a causal role in the development of psychosis? What can we learn from the more general pattern of decreased functional connectivity in individuals with schizophrenia? With luck, greater specificity in network-level analyses in these special populations will improve the future of computer-based cognitive therapy programs, and help patients better understand and respond to a range of sensory and affective stimuli. This line of inquiry remains ongoing.

5.2 Mental Illness and Neurocognitive Ontology

Our analysis of current research on OCD and schizophrenia in the last section highlights an important limitation of neuroscientific methodology. Tools such as fMRI and EEG can reveal areas of high and low activity in the brain, but it is left to the scientific theorist to infer from this evidence whether something is working well or poorly, and up to the clinician to determine the usefulness of this information.

In the case of OCD, we discussed overactivity in several areas but were able to pass by the question of what exact neural mechanism or mechanisms that overactivity represents. Safe to say, the brain likely has no dedicated "intrusive thought" or "ritual behavior" circuitry that can be identified across individuals. At most, we can say that the OFC, ACC, and likely the CSTC loop play a role in the production of these symptoms. This gap in our understanding is eased by the fact that we can leverage the tools at our disposal—microelectrodes for instance—to mitigate behavioral symptoms without necessarily knowing whether we have a case of "neural mechanism gone wrong" or how those neural mechanisms support associated cognitive functions. Unfortunately, we have no such elegant solutions for schizophrenia at this time. Thus, we are left asking *how* exactly the dopaminergic system, on the one hand, is causally related to, say, working memory deficits or auditory hallucinations on the other (and then, hopefully, how we can leverage that understanding in a therapeutic setting).

This inferential gap is a problem about cognitive ontology—the search for the structure or, more accurately, the structural components of the mind. An ontology of the mind would provide an answer to the question "What are the mind's parts?" Notice that this is a separate question from "What are the parts or systems of the brain?" A major goal of cognitive neuroscience, then, is to orient these frameworks with respect to each other or, as psychologists Russ Poldrack and Tal Yarkoni (2016) put it, to "delineate how brain systems give rise to mental function[s]." The introduction of psychiatric categories—such as "symptoms," "dysfunctions," "diagnoses," and "disorders"—introduces additional layers of complexity to the situation. In the remainder of this section, we will discuss the extent to which disciplinary interests in psychiatry and neuroscience overlap and whether any degree of theoretical reductionism is warranted. That is, we will take stock of whether the mechanisms of interest to neuroscientists are the same as the ones relevant to mental illness in the clinic and consider what happens if not.

5.2.1 Neurocognitive Ontology and Diagnosis

One influential answer to questions about neurocognitive ontology has recently been defended by Ken Kendler, Peter Zachar, and Carl Craver in their paper "What Kinds of Things are Psychiatric Disorders?" (2011). Drawing an analogy with Richard Boyd's work on property clusters as definitive of biological species, they argue that "the complex and multi-level causal mechanisms that produce, underlie and sustain psychiatric syndromes" are

researchers' most appropriate targets (p. 1146). In making this proposal, Kendler and colleagues are distinctly aware that "no one level is likely to capture the full complexity of the mechanisms sustaining or underlying . . . our best-codified diagnostic categories"—that is, that mental disorders will not be fully understood by excluding the causal contributions of our physical and social environments (p. 1148). Nonetheless, they wish to emphasize that "there are more or less general modes of functioning in the human mind/brain and [neurocognitive] mechanisms that sustain those different modes of functioning" (p. 1147). Thus, Kendler and colleagues, and others who adopt this view, are making a kind of methodological bet—namely, that continued research in cognitive neuroscience is the best route toward providing satisfactory explanations of mental illness. That is, when trying to understand the underpinnings of the diagnostic categories we are most familiar with (those in the DSM and so on), they insist neural mechanisms are the best place to shine the light.

This assumption has recently been challenged by several authors. Washington (2016) and Murphy (2017) note that the high variability and plasticity of human minds and brains suggest that the causal contributions of our physical and social environments may be more important than previously assumed. Tekin (2016) laments that much of current psychiatric research fails to incorporate first-person accounts of those with mental disorders and information about the role of the self in particular. But most pressing for our purposes here might be a kind of methodological circularity or what philosopher Kathryn Tabb (2015) calls the assumption of diagnostic discrimination—in her words, the assumption that "our diagnostic tests group patients together in ways that allow for relevant facts about mental disorder to be discovered" (p. 1049).

To understand the worry here, imagine that you have a large pile of shiny rocks that you presume are gold and wish to use in experiments. Some are gold, but some are pyrite or "fool's gold," and so they have different underlying chemical structures. On the assumption that everything in your pile is of the same kind, you might run your experiments assuming common properties in your specimens. Your research might consistently achieve only partial success and, in particular, never be very illuminating about what makes gold *gold*. In fact, what distinguishes it from different rocks might always seem mysterious. In the same way, neuroscientific research in psychiatry for the most part proceeds by focusing on populations

that have already been grouped by diagnostic category. Yet, as we have discussed, current diagnostic standards neither easily accommodate comorbidity nor involve precise tracking of the heterogeneity involved in mental disorders such as schizophrenia. It is an open question then, for example, whether examining the role of dopamine in working memory in individuals diagnosed with schizophrenia, or otherwise conducting diagnosis-based research, is the right way to proceed. Results might say nothing about how to help any particular patient, or why our population has been grouped together in the first place. One alternative would be to focus research on the connection between neurotransmitter function and observable behavioral dysfunction. In other words, Tabb (2015) and others see a future where current psychiatric diagnoses do not neatly reduce to dysfunction in neurocognitive mechanisms—where our current nosology either is reimagined to fit more neatly with neuroscientific findings or is validated by emphasizing causal pathways that crosscut neuroscientific ontological distinctions (pathways in our social, cultural, and environmental ecologies for instance).

How we best proceed from here remains open. Some argue that psychiatry should stay away from so-called mechanisms entirely (Hartner & Theuerer, 2018). On the other hand, the NIMH has doubled down on the search for mechanistic dysfunction in psychological systems. In the wake of the publication of the DSM-5, NIMH released the RDoC—not itself a diagnostic manual but rather a framework for structuring psychiatric research that stays away from diagnostic categories. Those seeking funding from the NIMH may now propose to investigate one or more domains or constructs (e.g., "reward learning" or "visual perception") from one or more levels of analysis (e.g., "cells" or "circuits"). If successful, these efforts may reshape psychiatric categories as we know them.

5.2.2 Neurocognitive Ontology and the Roots of Addiction and Substance Abuse

To cap off this discussion, let us examine a concrete case. Addiction and substance abuse are serious contemporary mental health concerns. To what extent has neuroscientific evidence advanced our understanding of these conditions or given us new tools for intervention? To begin with, we know that substance use causes immediate chemical changes in the brain. Dopamine, for example, which is associated with changes in the NAc and amygdala, plays a significant role in the reinforcement of addiction and may

drive drug reward (Koob, Sanna, & Bloom, 1998). Nutt and McLellan (2013) also note that dopamine receptor density plays a role in determining the extent to which an individual will enjoy stimulants, which might explain why certain individuals develop dependence. However, they also emphasize that because the specifics of different drugs' actions in the brain are different—opioids mimic endogenous opioid transmitters, while alcohol blocks glutamate and enhances GABA—the notion that a single neurotransmitter mechanism explains drug dependence is no longer viable. We also know that chronic drug use can adapt brains in ways that are enduring and complex. Using network analysis, McHugh and colleagues (2013) found that individuals addicted to cocaine had reduced connectivity between the bilateral putamen and posterior insula and right postcentral gyrus and scored higher in impulsivity than control subjects. Drug abuse has also been linked to impaired glutamate homeostasis, which impairs prefrontal regulation of striatal circuitry, potentially explaining why drug users are unable to control their drug seeking (Kalivas, 2009).

In sum, it seems unlikely that any one neural mechanism can explain all of the symptoms of addiction. The neurochemical drivers of cravings, relapse, and withdrawal and long-term damage are different among both different substances and different individuals. Indeed, approximately 40–60 percent of variation in levels of addiction can be attributed to either genetic factors alone or gene–environment interactions (Volkow & Ting-Kai, 2005). Thus, while effective treatment may be available in a narrow set of situations—the immediate use of an opioid overdose reversal intervention such as naloxone, for example—treatment for long-term care and recovery from substance abuse is a difficult prospect. Standard care, for those who have access, generally takes the form of psychosocial support such as family care, residential treatment ("rehab clinics"), expert counseling, and support group activity.

Perhaps this is not surprising. Addiction, like other mental disorders, is grounded in the brain, but it is also uniquely influenced by features of the social environment. Indeed, the current opioid crisis is the prime case study for the link between substance abuse and social capital. Deaths by prescription opioid overdose have tripled between 2001 and 2016 in people aged fifteen to thirty-four years (Samet & Kertesz, 2018), and there has been a pronounced increase in the prevalence of overdose and other "diseases of despair"—for instance, alcohol abuse and suicide—among middle-aged white people without a college degree (Dasgupta, Beletsky, & Ciccarone,

2018). Furthermore, Heilig and colleagues (2016) found there is a strong link between addiction and social integration, with social exclusion and addiction linked to activity in the insula.

In effect, then, the diagnostic category "addiction" groups individuals together who share important social features but who vary widely with respect the neurocognitive mechanisms that sustain their behavior. One important ontology—that which identifies neurocognitive dysfunctions in the brain—appears to crosscut another—that which groups clinically relevant populations. Is addiction, then, best understood through the lens of neurocognitive dysfunction? More pressingly, where can we best exert leverage in order to help those struggling with substance abuse? These questions are left open by current neuroscientific theory and methodology.

5.3 Neuroscience, Science Communication, and Impacts on Treatment

In our final section, we would like to return to applied issues. Mental health is a concern for all. Thus, it is worth asking how neuroscientific information is communicated to the public more generally and how it is received. To begin, we will look at a domain where neuroscientific information about mental illness is thought to be relevant to our practices of blame and punishment.

5.3.1 The Perceived Credibility of Neuroscience in the Courtroom

In 2005, Grady Nelson brutally murdered his wife and her eleven-year-old daughter in Miami-Dade County, Florida.[6] His sentence of life in prison rather than the death penalty stirred controversy in 2010, when several jurors indicated that the presentation of EEG "scans" (i.e., images of recorded waveform amplitude readings) influenced their decision making. As one juror put it, "The technology really swayed me . . . After seing [sic] the brain scans, I was convinced this guy had some sort of brain problem" (Ovalle, 2010, p. 2). As this case highlights, neural evidence can be of weighty importance in judgments of responsibility. Briefly, this is because our folk notions of responsibility typically require that actors have some degree of control or agency over their behavior, and neural evidence of a mental disorder suggests threats to a person's agency (for more, see King & May, 2018; Murphy & Washington, forthcoming). If, as Grady's defense attorney argued, "the moment this crime occurred, Grady had a broken brain," this can sometimes be seen as a reason to mitigate punishment (Ovalle, 2010, p. 1).

One may reasonably wonder what EEG readings recorded much later than the events of 2005 can tell us. Even if they are evidence of neuroatypicality in Grady, they might not be evidence of any particular diagnosis, supposing we understood how to draw such inferences.[7] They might not be evidence of whether Grady suffered from mental illness at the earlier time when he committed his crimes. And even if this is known, there is ongoing debate about whether a diagnosis itself is sufficient to mitigate blame in these kinds of cases or whether it must be demonstrated that mental illness was causally relevant to a subject's degree of control in the particular behavior (Sifferd, Hirstein, & Fagan, 2016). For example, it may be known that I suffer from compulsions, but unknown whether compulsive behavior was implicated in a particular act of theft (e.g., maybe I intended to get back at a rival). In Grady's case, prosecuting attorneys insisted that testimony from neuroscientists "was a lot of hocus pocus and bells and whistles" and that "When you look[ed] at the facts of the case, there was nothing impulsive about this murder" (Ovalle, 2010, p. 2).

The big worry here is that these questions reveal subtleties that can be overlooked by jurors and other non-experts. This is becoming a pressing problem, as the number of court cases that involve the presentation of neuroscientific evidence is growing rapidly (Farahany, 2015). Worse, recent analyses from psychologists Nick Haslam and Erlend Kvaale (2015) reveal a link between "biogenetic" forms of explanation (e.g., recourse to neural dysfunction to explain mental disorder) and increased stigma surrounding mental illness. That is, at the same time that a neurally based assessment can diminish attributions of blame for an individual, it increases perceived dangerousness and desire for social distance as well as imputing a kind of prognostic pessimism (Haslam & Kvaale, 2015). With such high stakes, there is increasing pressure on neuroscientists concerning the misinterpretation of their research.

5.3.2 The Perceived Efficacy of ECT

When aimed at ameliorating mental illness, neuroscience and neuroscientific tools themselves also face their share of criticism. In most stories in movies and television, things have taken a rotten turn for the protagonist when doctors wheel out the electrodes and restraints. It may therefore surprise you to hear that since the 1940s, electroconvulsive therapy[8] (ECT) has been considered a safe and effective treatment for severe mood disorders, with widespread clinical support. During an ECT treatment, a subject first undergoes general anesthesia. Then, a small electric current is used

to induce a brief seizure, triggering chemical changes that reverse severe symptoms. Slade and colleagues (2017) found that among patients with major depressive disorder, bipolar disorder, or schizoaffective disorder, ECT is associated with a 6.6 percent thirty-day readmission risk compared to 12.3 percent for individuals who did not receive ECT. Moreover, Bjølseth and colleagues (2015) found that ECT can be effective for elderly patients with major depression, and that both bifrontal and unilateral ECT are associated with statistically significant decreases in symptom severity. ECT can also be used to treat agitation in dementia patients. Tang and colleagues (2014) found that dementia patients who received ECT experienced a significant reduction in agitation based on Pittsburgh Agitation Scale scores (Glass, Forester, & Hermida, 2017).

In movies and on TV, unfortunately, ECT is continually portrayed as violent and frightening. In a 2016 study of TV programs and movies, Sienaert found that the patient had not given consent in 57.3 percent of scenes portraying ECT, the patient was not given an anesthetic in 72 percent, and the apparatus was used for torture in 13.4 percent. ECT has also been portrayed as a way to erase memories. This frightening picture is many people's first introduction to ECT. Ithman and colleagues (2018) found that 94 percent of medical students prior to training had learned about ECT from either a film or word of mouth, and 24.05 percent reported they were frightened by the procedure. ECT's historically low use may therefore be driven by patient and practitioner stigma or by beliefs of its negative cognitive effects[9] (Sackeim, 2017).

Better information access is associated with a reduction in negative assessments of ECT. In the study by Ithman and colleagues (2018), only 2.53 percent of medical students surveyed reported that they continued to fear ECT after clerkship. Exposure to ECT, for both the individual and their family, has been shown to be effective in reducing patient fears. For example, Elias, Ang, Schneider, and George (2019) similarly found that when family members watched ECT procedures, a majority (76 percent) of family members reported that the experience was reassuring and rewarding, and 71 percent of families reported that it lessened their fears and improved their knowledge of ECT.

5.3.3 Conclusions and Future Directions

Each development in clinical neuroscience affects the lives of people in mental distress, whether through our public institutions or in private life as

people try to make sense of the latest findings conveyed by the news, entertainment media, and word of mouth. Scientists, philosophers, and the public can neither keep up with the pace of novel findings in clinical neuroscience nor curate the findings consistently. Some diagnoses—or behavioral, cognitive, or emotional disruptions—will tend to receive more focus than others and will be better investigated with more valid results. We have reviewed several areas where neuroscientific findings have contributed directly to human welfare, including the treatment of severe OCD, ECT for severe depression, and in the understanding of addictive processes. We have described ways that neuroscience at multiple levels of analyses has complicated the understanding of mental disorders, including schizophrenia and addiction, and how this might also lead to a fundamental restructuring of how we conceptualize mental illnesses and create guidelines for diagnosis, treatment, and research.

The tools of neuroscience are enticing, and this is not a sin that needs to be punished. These tools have, in some cases, shown us new paths toward recovery and symptom alleviation when we thought none were available. Managing this powerful feature while conducting ethical research with those in mental distress is the responsibility of clinical scientists. We note that accuracy in news reporting and the presentation of findings to the public in an unbiased manner remains a problematic issue if baseline neuroscience knowledge in the public is low, as it is. Justice in the courtroom is questionable when fates are determined by scientific information that is understandable for a minority of a jury, for example.

There is currently broad support for fuller integration of neuroscience in research on mental health, and clinical interventions are rapidly developing. Ultimately, neuroscience has the potential to reveal better ways of understanding the causes of mental distress and to transform how we categorize mental illness and health.

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Notes

1. One influential model of mental illness, known as the biopsychosocial model, understands psychiatric symptoms as the products of biological, psychological, and social causes.

2. An important note is that in the case of OCD, such thoughts are ego-dystonic, meaning they align neither with who the individual is nor with their intentions/wishes. In other disorders wherein these thoughts might be ego-syntonic, a different approach to treatment is warranted.

3. Though these are not reflected in the DSM criteria.

4. Many thanks to Niklas Andersson (Washington University, St. Louis, MO) for discussions on this point.

5. Kanyuch & Anderson (2017), for instance, propose using the marmoset monkey, given that marmosets' prefrontal cortices more closely resemble those of humans, as well as the fact that marmosets have already been used as animal models in studies of working memory, anhedonia, fear generalization, and cognitive flexibility.

6. State v. Nelson, No. F05–846 (11th Fla. Cir. Ct. Dec. 2, 2010), archived at https://perma.cc/7XA5-2JXG?type=pdf; Judge Okays QEEG Evidence, supra note 8; Miller, supra note 6. For more information, see generally Transcript of Opening Statement, Nelson, No. F05–846, archived at https://perma.cc/6TZZ-NZHA?type=pdf

7. One reason Grady's case stands out to us is the availability of testimony from those directly involved in the hearings. A drawback of relying on it here is that we are limited to focusing on the influence of the EEG evidence rather than on the influence of any particular psychiatric diagnosis. However, other recent high-profile cases do highlight the diagnosis itself (see, e.g., Sifferd et al.'s, 2018, treatment of mass murderer Anders Brevik).

8. Commonly referred to by the misnomer, "electroshock" therapy.

9. The most common side effects of ECT include disorientation and impairments in learning and anterograde and retrograde memory. This can be mitigated by using brief pulse waveform over sine wave simulation and by conducting unilateral ECT rather than bilateral. The adverse cognitive effects are also attributable to an individual's seizure threshold—as there is no way to predict accurately how much electricity an individual can take, some patients (especially young, small women) may receive excess current (Prudic, 2008). This said, it is necessary to weigh these effects against the continuation of severe symptoms requiring ongoing hospitalization.

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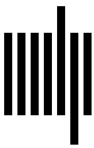

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