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Article

The Brain and Causality: How the Brain Becomes an Individual-Level Cause of Illness

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ABSTRACT

How do individual-level explanations become applied to social issues? Neurobiology – the study of the connections between behavior and the cells and structures of the brain – receives substantial public funding and influences social institutions, policy debates, and core aspects of human experience. With respect to mental health, neurobiology has ramifications for the way disorders are defined, diagnosed, and treated, along with how public funding for mental illness is allocated. This article addresses how neurobiologists establish the brain as a cause of mental illness. I analyze 17 months of ethnographic observation at a well-regarded neurobiological research laboratory, as well as observations at professional meetings, to detail three strategies: *Linking the Brain to Mental Illness*, *Explaining Mental Illness with the Brain*, and *Asserting the Causal Importance of the Brain*. These strategies first connect the brain to mental illness, and subsequently establish the causal primacy of the brain relative to alternative explanations (e.g., poverty). I connect findings to medical sociological theories, biological reduction, and emerging national health policies.

KEYWORDS: neuroscience; psychiatry; individual differences; science studies; mental illness.

How are individual-level explanations applied to social issues? In the social sciences, numerous scholars have interrogated individualistic explanations of economic, racial, and gender disparities (e.g., Anderson 2011; Bourdieu 1993; Hyde, Fennema and Lamon 1990). Health researchers also assert the relevance of social accounts of illness in contrast to individualistic explanations (e.g., Freeman Anderson 2017; Link and Phelan 1995). Here, I demonstrate how laboratory scientists produce individual-level neurobiological explanations of mental illness.

Neurobiology has considerable influence on practice and policy. First, neurobiologists receive substantial public funding to support their research initiatives (e.g., Insel 2013; Whitehouse.gov 2013). Second, neurobiological methods are shaping many fields, including law, medicine, and economics

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(Littlefield and Johnson 2012; Rose and Abi-Rached 2013). Third, numerous social scientists argue that neurobiology has implications for redefining dimensions of human experience, such as free will, consciousness, and subjectivity (e.g., Abend 2017; Pickersgill 2013). Fourth, neurobiological approaches are increasingly applied to the study of social problems, connecting issues such as gender disparities and crime to the brain (e.g., Eliot and Richardson 2016; Larregue and Rollins 2019). Last, neurobiology is influencing both public debates and social policy (Broer and Pickersgill 2015; Whooley 2014).

Neurobiology is similarly consequential for mental health institutions, concepts, and practices. Responding to mental health professionals' inability to improve the lives of those living with mental illnesses, National Institute of Mental Health (NIMH) Director Tom Insel (2013) announced the Research and Domain Criteria Initiative (RDoC). RDoC aims to "transform" diagnosis by abandoning the categorical and descriptive schema of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) in favor of a new taxonomy grounded in neurobiological and genetic biomarkers. Neurobiological research is similarly informing brain-based interventions for mental disorders (Fagan et al. 2019; Pickersgill 2011).

To investigate how neurobiological research is shaping mental health, I conducted 17 months of ethnographic observations at a well-regarded neurobiological laboratory (the "Becking Laboratory"), as well as observations of major psychiatric conferences. While the majority of neurobiological studies use cross-sectional data, the Becking Laboratory uses longitudinal imagining data to explain the emergence of mental disorders. In contrast to cross-sectional studies, longitudinal data capture information on time sequence, documenting brain changes that occur prior to the development of mental illness. Becking researchers use these data to speak directly to the causes of mental illness.

The Becking Laboratory also applies a neurophysiological "individual differences" framework, which aims to use biology to explain 1) why some individuals become ill while similar individuals remain healthy, and 2) why one person develops a condition such as depression, while another comparable person develops a different disorder, such as addiction. In this individual differences framework, social factors (e.g., poverty) have limited explanatory utility, as individual-level neurobiological processes are thought to best explain mental illness (see *Explaining* and *Asserting* below). Some scientists using an individual differences approach also advocate investigating individuals instead of groups, as they perceive psychological phenomena (e.g., mental disorders) as organized within the individual, with group-level analyses seen as potentially obscuring individual, physiological processes (e.g., Fisher, Medaglia, and Jeronimus 2018). By backgrounding group-level analysis and non-biological approaches, the individual differences framework provides challenges for sociological analysis, non-biological approaches in psychiatry (e.g., psychosocial, cultural, behavioral, and psychodynamic), and biosocial approaches in sociology and psychology (e.g., Engel 1977; McEwen and McEwen 2017). With individual differences listed as central to NIMH's strategic research objectives (NIMH N.D.a) and operating as one of "seven pillars" of RDoC (Cuthbert and Insel 2013), this framework is also becoming increasingly influential. Given their use of longitudinal imaging data and individual differences analyses, NIMH Director Insel personally lauded Becking Lab research as the "future" of psychiatry. Accordingly, this paper describes a rising mental health paradigm and exemplifies mental health research in the RDoC era.

Building on prior investigations of the linkages between the life sciences and medical practices (e.g., Clarke et al., 2010; Fujimura 1996; Halpin 2011; 2018; 2020; Keating and Cambrosio 2011; Lakhani and Timmermans 2014; Maynard and Turowetz 2019; Nelson 2018; Panofsky 2014; Turowetz and Maynard 2015), as well as the social implications of neurobiology (e.g., Abend 2017; Joyce 2008; Pickersgill 2013; Pitts-Taylor 2016), this paper details how laboratory science is used to explain social issues. In contrast to prior investigations of neuroscience that examine the content of published papers, discrete medical conditions, or professionals' use of specific imaging technologies, this study provides an ethnographic analysis of a longitudinal neuroimaging study to demonstrate how neuroscientists make causal claims about the brain and mental illness. Put simply, this paper

details how scientists use "brain facts" (Star 1983) to explain mental illness. More specifically, I argue that scientists use three interconnected strategies to make the brain a cause of mental illness. First, researchers' strategy of *Linking the Brain to Mental Illness* connects brain data to psychiatric data, such that fMRI images and scores on psychiatric assessments become associated. Second, researchers' strategy of *Explaining Mental Illness with the Brain* establishes the brain as the centerpiece of causal models, with neurobiology determining an individual's psychiatric destiny. Third, the strategy of *Asserting the Causal Importance of the Brain* emphasizes the causal primacy of the brain vis-à-vis alternate causes of mental illness (e.g., poverty) or modes of explanation (e.g., non-neurobiological psychiatry). In addition to detailing these processes, I discuss reduction, medical sociological theories, and emerging NIMH policies.

THE BRAIN, BIOLOGY, AND ILLNESS

Biological accounts of social phenomena are a reliably contentious project and reduction is similarly a central tension in studies addressing brain science. Numerous scholars (Abend 2017; Choudhury and Slaby 2011; Fallin, Whooley and Barker 2018; Pitts-Taylor 2016; Uttal 2001; Vidal 2009) suggest that neurobiology forwards thin, inadequate, or incorrect descriptions omitting the texture of human experiences and complexity of social phenomena. Aligning with these and previous studies noting the absence of environmental and social factors from psychological and psychiatric studies (Danziger 1997; Dewey 1896; Horwitz 2002), I demonstrate how neuroscientists put together explanatory frameworks that elevate brain-based, individual-level explanations of mental illness.

Reductionism is commonly associated with neurobiological research (e.g., Choudhury and Slaby 2011); Kirkmayer and Gold (2016) suggest that neurobiologists reduce complex phenomena to physiology and assert that biology is sufficient for understanding complex experiences. Other scholars suggest popular media, funders, and policy makers often encourage biological reduction (Campbell 2007; Nelson 2018; Panofsky 2014; Wynne 2005). Furthermore, while scientists emphasize the complexity of their own work, they also critique overly simplified uses of biological data among their peers and popular media (Beaulieu 2002; Nelson 2018).

In contrast to analyses of reduction, other social scientists caution against critiques of neurobiology (Rose 2013), use neurobiological data to advance sociological analyses (e.g., Zerubavel et al. 2015), or advocate for the unification of sociology and neuroscience (e.g., Franks' "neurosociology," 2010). Accordingly, there is a range of social scientific perspectives on neuroscience, ranging from critiques of reduction to calls for disciplinary integration. This article enters this debate not by investigating *if* neurobiology research is reductive but rather demonstrating *how* laboratory scientists use longitudinal data to make individual-level arguments about the brain causing mental illness.

While many qualitative studies of neuroscience analyze the content of scholarly articles, researchers have also investigated the *in situ* practices of neuroscientists. This work details the practices scientists and health professionals use to "make facts" about the brain (Alac 2008; Dumit 2004; Joyce 2008; Lynch 1985; Maynard and Turowetz 2019; Star 1983; Turowetz and Maynard 2015). Additional studies demonstrate how neuroscientists orient to central tensions in their field or construct core concepts, such as the potential brain-basis for psychopathy (Pickersgill 2011), or whether the data produced by imaging machines are statistical or numerical in nature (Beaulieu 2002). While these previous studies consider how neuroscientists "make facts," this paper demonstrates how scientists connect "brain facts" to mental illness, and, in so doing, make mental illness a neurobiological problem that is tethered to neurobiological solutions.

In addition to investigating neurobiology, I analyze the definition and conceptualization of mental illnesses. While these illnesses have considerable social and personal ramifications (e.g., Sugie and Turney 2017; Thoits 2011; Wright et al. 2007), the validity of psychiatric categories has been extensively interrogated. Scholars have been particularly critical of the rapid proliferation of diagnostic labels and the expanding list of behaviors that are perceived as pathological. For example, diagnostic

categories have been used to both pathologize normal experiences (e.g., Conrad 2005; Horwitz 2002; Lane 2008) and allocate resources (Barker and Galardi 2015; Halpin 2020); numerous researchers have asserted that psychiatric categories are created, in part, to align with the corporate interests of large pharmaceutical companies (e.g., Conrad 2005; Halpin 2016; Kutchins and Kirk 2003). Here I show how these categories of mental illness are reconfigured in the RDoC era, with mental illnesses regarded as brain disorders resulting from neurodevelopmental problems.

Neuroscientific explanations of mental illness demonstrate the increasing influence of the life sciences upon medical practices, a central claim in medical sociological theories. For instance, Conrad's (2005) revision of medicalization theory details three new "engines" of medicalization, with biotechnology (i.e., pharmaceutical companies and genetics) operating as one such engine driving diagnostic expansion. This paper brings laboratory scientists into medicalization theory, arguing that treating mental illness as a brain disorder legitimates monetizable brain-based treatments and creates therapeutic entry points across the life course.

Adapting medicalization research, Clarke and colleagues (2010) detail a societal transition from medicalization to biomedicalization. They argue that science is central to this transition, creating an "epistemic shift" and reorganization of medicine, with the legitimacy of biomedicalization resting upon science and technology. Biomedicalization is characterized by five processes: bioecomomics, an increasing focus risk and surveillance, increasing prominence of technoscience, transformation of knowledge, and transformation of identities. While Clarke and colleagues (2010) argue the core processes of biomedicalization "overlap" and are "interactive," less is said on how these processes might tangibly interact in specific sites. Through my analysis on a longitudinal imaging study, I demonstrate one possible mode of interaction, a feedback loop among three identified processes: increasing reliance on technology, transformations of biomedical knowledge production, and the elaboration of risk and surveillance. Both medicalization (Conrad 2005) and biomedicalization (Clarke et al. 2010) theory omit or background neurobiology, causal analyses, and longitudinal studies. The present study demonstrates how these topics might inform these theories.

METHODS

I spent 17 months observing the Becking Laboratory. Following previous ethnographic studies of scientific activity (e.g., Collins 1985; Fujimura 1996), and responding to Pickersgill's (2013) call for increased empirical investigation of the sites, interactions, and practices associated with brain science, I documented the everyday activities of scientists, detailing how they collect their data, how they discuss phenomena, and how they advance analyses. I observed laboratory-wide meetings, data collection activities, data analysis meetings, research presentations, and informal conversations. My laboratory fieldnotes were supplemented by observations of the 2014 and 2016 annual meeting of the American Psychiatric Association (APA) and the 2014 annual meeting of the Society for Biological Psychiatry (SOBP). At these conferences, I attended 81 individual presentations, taking fieldnotes on presentations, interactions, and question and answer sessions. As I was a known observer operating in academic settings, I was able to write my fieldnotes directly onto a laptop computer or tablet. Nevertheless, following ethnographic conventions (Emerson, Fretz, and Shaw 1995), I would relocate to a nearby library or office and develop my initial notes into more extensive fieldnotes after completing an observation session.

The Becking Laboratory occupies one-and-a-half floors of a university building devoted to neurobiological research. The lab is run by Dr. Normand Becking,[1] a psychologist who has authored more than 300 articles and a dozen books. In addition to accolades in popular media, Dr. Becking and the lab members routinely publish in *Science*, *Nature*, *Neuroimage*, and other high impact journals. Not counting the dozens of undergraduate researchers, 90 people (31 male, 59 female) are affiliated with the lab, with weekly laboratory-wide meetings which regularly host 50 staff from numerous fields

1 All the names are pseudonyms.

(e.g., computer science, psychiatry, economics). Dr. Becking presides over these meetings, and two senior researchers (Michelle and Lisa) run them if he is absent. He also arranges regular project meetings, but he delegates day-to-day operations to project leaders.

The lab is extremely well-funded, securing numerous operating grants from federal agencies (e.g., NIMH, NIH), as well as millions of dollars in private donations and "non-trivial" remuneration from Dr. Becking's talks. The Becking Lab has established a non-profit to manage these funds, which are used to secure facility space, support tenure-track faculty, and pay research assistants. Dr. Becking also arranges teleconferences with NIMH staff to gather knowledge about how to best target papers and grants to align with emerging NIMH initiatives.

I selected the Becking Lab as they are a well-regarded and influential neurobiology laboratory. I gained access by emailing Dr. Becking and requesting his permission to observe the lab. Access was granted after I introduced myself at a laboratory-wide meeting, during which lab members also expressed support for my data collection. Lab members were exceptionally collegial and eager to discuss their neurobiological findings with me. Although I am included on a laboratory ethics protocol and participated in some research activities (e.g., a training session for fMRI sensor application), my primary aim was observation, as I sat beside researchers documenting their activities. I spent 8 to 30 hours per week at the laboratory, often observing from the morning until the late evening while members were imaging participants.

During my tenure at the Becking Lab, I focused observations on the **RE**search into the **N**eurobiology of **E**motions (RENE) study. The RENE study is a longitudinal fMRI-based project examining the neurodevelopment of emotions and psychiatric symptoms. The RENE study is not investigating a discrete condition (e.g., depression) but analyzing the development of all the symptoms and disorders of psychiatry. With longitudinal imaging data on 180 participants, the RENE study has exemplary data for questions relating to brain development and psychopathology. Participants have been enrolled in the study since they were *in utero* and the centerpieces of the RENE study are three waves of imaging data, completed when the participants were children, teens, and young adults. Including myself, there are 28 researchers (11 male, 17 female) on the RENE protocol, although most work is completed by Sara (PhD Research Scientist), Louis (Professor), Jake (Professor), Audrey (Professor), Michelle (PhD Data Scientist), Irene (MD/PhD doctoral researcher), Katy (doctoral researcher), Bonnie (MSc. researcher) Tiffany, Diane, and Kent (all undergraduate researchers), and Dr. Becking.

I coded study data using the Nvivo[tm] software for qualitative analysis. Data were inductively coded and analyzed following the conventions of grounded theory (e.g., Charmaz 2006). Analysis began with line-by-line coding, with each line of fieldnotes read, analyzed, and labeled with an initial code. After line-by-line coding was complete, I re-coded the fieldnotes, developing and operationalizing broader thematic codes. The codes used in this analysis are labeled "measurement/methods/procedures," "design and analysis," "telling stories," "individual differences," "brain data," and "brain as psychology." These codes emerged from my analysis of lab members' interactions and analytic practices (e.g., how they used various surveys and scales), with initial codes capturing explicit actions (e.g., the use of correlation) before being developed to address analytic processes, which form the basis of the thematic analysis below.

FINDINGS

I argue that scientists use three interconnected strategies to characterize the brain as causing illness. First, using the strategy of *Linking the Brain and Mental Illness*, brain data become fused to psychiatric data, such that fMRI images and scores on psychiatric assessments become analogous. Second, using the strategy of *Explaining Mental Illness with the Brain*, researchers situate the brain as the centerpiece in both causal models and "stories" on the emergence of mental illness, with variations in neurophysiology determining an individual's psychiatric destiny. Third, in their everyday talk and professional

rhetoric, scientists use the strategy of *Asserting the Causal Importance of the Brain* to place the brain above all other causes, such as social or non-brain-based psychiatric approaches to mental illness. While I present these strategies sequentially, laboratory meetings and analytic interactions can and do display all three strategies.

Linking the Brain and Mental Illness

Before the brain can be said to cause mental illness (see *Explaining* and *Asserting*), neurobiological data (e.g., fMRI images) must be connected to psychiatry, while psychiatric assessments must be associated with neurophysiology. Scientists use the strategy of *Linking* to fuse brain and psychiatric data.

Researchers link the brain to illness by correlating participants' results on psychiatric assessments with neurophysiological data. Indeed, while policy makers argue neurobiology will revolutionize psychiatry (Insel 2013), longstanding "traditional" pen-and-paper assessments are central to neurobiological research. RENE study participants complete extensive psychiatric surveys, including the Beck Depression Inventory (BDI), Beck Anxiety Inventory (BAI), the Rumination Response Scale, Addiction Severity Index, and a post-traumatic stress disorder scale. These surveys provide a quantitative assessment of psychiatric symptom severity. For instance, the BDI asks participants to rate their agreement with 21 sets of face-valid statements, such as "I feel like a complete failure as a person" and "I don't have any thoughts of killing myself," each receiving a numeric value from 0 to 3. Based on their total score, participants are placed into one of four categories: minimal, mild, moderate, or severe depression. Researchers similarly use the SCID, a structured interview tool that assesses participants against every DSM category (Halpin 2016).

The RENE team correlates questionnaire results (e.g., a BDI score) with brain data, such as differences in brain structures (e.g., volume of white matter) and brain activity patterns documented during imaging tasks. For researchers, associations between psychiatric surveys and neurophysiological data demonstrate how the brain is connected to illness. For example, Louis discusses his analysis of "internalization," an umbrella category for inward or self-directed disorders (e.g., depression, anxiety, and PTSD) and symptoms (e.g., rumination, anhedonia), while further detailing how the brains of "internalizers" might operate:

> Louis states that [in a previous analysis] he used data from teenage participants to analyze childhood adversity and internalizing at the "brain level." [Summarizing his results], he tells the RENE team that high internalizing was associated with variation in neural connectivity. He suggests that the RENE group could build on his analysis by drawing on their longitudinal data to further investigate the association between internalizing and neural connectivity over the life course, with Louis hypothesizing that high internalizers lack some form of "neural mechanism."

Louis uses scores on the BAI and BDI to characterize those participants who experience depression and/or anxiety as internalizers. In his study, internalizers have distinctive neural connectivity patterns, which are different from those of both "healthy" participants and "externalizers" (an umbrella category for outward or other-directed symptoms or disorders, including substance use, conduct disorder, and aggression). At this stage of the conversation, Louis' analysis does not extend beyond correlation but he will subsequently suggest that brain differences explain these elevated scores (see *Explaining*).

RENE researchers also situate fMRI tasks as identifying links between the brain and psychiatry. In one task, the RENE team presents images designed to elicit emotional responses, detailing how participants' brains respond to oscillations between positive and negative emotional stimuli:

> While Katy and Bonnie are talking, I am watching the images that are being shown to the participant. The first image that catches my eye is a dog that has been severely injured by a

> porcupine. The image is a close up of the dog's face, which is almost completely obscured by hundreds of quills. A subsequent image displayed is a black man who appears to be yelling and holding a large stick as though he is about to strike the camera, while yet another depicts a white man with a knife to a woman's neck, as though he is about to slash her throat. Interspersed with these images are displays of skydivers, automobiles, sunsets or scenic coastal towns.

Here, positive images (e.g., a sunset) are thought to evoke a different neurophysiological response than negative images (e.g., an attack). RENE researchers use such tasks to suggest that some participants are neurophysiologically vulnerable to emotional disturbance and struggle to return to a "baseline," while others "bounce back," demonstrating psychological resilience.

Although psychological tasks, fMRI tasks, and their underlying stimulus-response arc logic have been extensively interrogated (e.g., Abend 2017; Danziger 1997; Dewey 1896, Fallin et al. 2018; Morrison et al. 2019; Pitts-Taylor 2016), my aim here is to detail how researchers connect brain processes to psychiatry, an essential step in their arguments that the brain causes mental disorder. Specifically, these fMRI tasks use psychological constructs (e.g., emotional regulation) to provide a bridge between the two primary concerns of the RENE team: variations between neurophysiology and psychiatric outcomes:

> The RENE team discusses how the image task measures a participant's ability to regulate their emotions and, specifically, how participants manage exposure to negative emotional content. Sara suggests the brains of resilient people will quickly return to baseline after viewing a troubling image (e.g., an injured woman). The group then describes the task as providing insight on why some individuals are resilient in the face of a traumatic event, while others develop mental health conditions like anxiety or PTSD. Here, resilient brains do not produce mental illness, while more reactive brains potentially do. Towards the end of the conversation, Irene tells the team she plans to analyze the image data in relation to "impulsivity" and "risky behavior" (two symptoms of mental disorder), while also "taking the data further" to "get at depression and anxiety and stuff."

Accordingly, in analysis meetings, the images of scenic coastal towns, cars, threatening men, and wounded women are evidence of "emotional regulation," "emotional recovery," and "resilience." As such concepts are highly salient to mental illness (e.g., emotional dysregulation is associated with depression; APA 2013), the RENE imaging tasks are establishing how variations in neurophysiology are associated with variations in emotional processes, which, in turn, are tied to the symptoms and disorders of psychiatry. Here, Irene's plan to "[take] the data further" by analyzing amygdala function, risk taking, impulsivity, anxiety, and depression not only emphasizes how tasks connect the brain, psychological processes, and psychiatry, but also demonstrates how associational data is promptly enrolled in causal explanations, which is mobilized in the strategies of *Explaining* and *Asserting* (see below).

Through *Linking*, scientists conceptually and empirically fuse brain data to psychiatric states. While scientists employ "transformative" (Insel 2013) neuroimaging data, they also rely on more traditional pen-and-paper assessments, such as the BDI and BAI. Via *Linking*, these assessments and DSM categories (e.g., major depressive disorder) adopt a neurophysiological texture as they become tied to the brain. These associational data are central to their subsequent causal arguments (see *Explaining* and *Asserting*).

Explaining Mental Illness with the Brain

Neurobiological research involves more than *Linking*, as scientists are not simply arguing that the brain is associated with mental illness, but rather that the brain causes mental illness. The strategy of

Explaining describes what scientists do with the associational data produced by *Linking*. Specifically, using the individual differences framework, researchers forward causal models and explanatory "stories" that identify how variations in neurophysiology cause variations in psychiatric outcomes. Their models might include non-neurobiological variables but it is the brain that determines an individual's psychiatric destiny.

Becking scientists' empirical work is guided by a background assumption that the brain causes mental disorders, and researchers are bemused when I ask questions about the causal role of the brain. Ted, a Becking researcher, tells me, "in neuroscience and psychology, we assume that all clinical disorders are produced by the brain. I think I've heard sociologists ask questions about that – but as a psychologist it just seems so obvious to me [laughs]." While the RENE study does collect social data (e.g., race, educational attainment, income), such data are not used to explain mental illness. Indeed, the terms "social," "biosocial," and "environmental" do not appear in the 19, 000 word RENE study protocol, despite extensive detail on research design and analytic procedures. For members, mental disorders are quite simply "brain disorders" (see also *Asserting* below), and neurophysiology is pivotal for understanding the emergence, treatment, and variability of these disorders.

While members operate with an understanding that "clinical disorders" are "brain disorders," each discrete analysis forwards a specific causal model demonstrating the explanatory properties of the brain. In the context of this explanatory work, the associational data of *Linking*, regardless of potential statistical significance, are insufficient. Instead, as Becking members often repeated, good models and analyses need to "tell a story" demonstrating how the brain causes mental disorder. An interaction between Bonnie and Michelle details the push to move from *Linking* to *Explaining*:

> Bonnie says she has "found something really cool and reportable" telling the group she discovered brain reactivity in some regions of interest during the "recovery epoch" of one of the fMRI tasks. Michelle tells Bonnie to interpret her findings, asking "what story does this tell?" Bonnie responds she "hasn't thought about interpretation." After re-summarizing the findings and suggesting some new statistical procedures to perform, Bonnie says "but I don't know what will make the most sense. I don't want to keep running analyses and I want a nice clean story to tell." Michelle encourages Bonnie to take another look at her data, adding "the findings look good and have a good reason to be there. We can maybe get a good story for that," suggesting the story appears to be about "amygdala connectivity" and the development of symptoms.

Bonnie reports a compelling physiological finding, that some participants have more active amygdalas during a "recovery" period in an fMRI task. However, as Michelle suggests, her finding does not yet tell a "story." Specifically, while Bonnie has a potential individual difference (i.e., variation in amygdala activity), she has not connected this variation to psychiatric outcomes, meaning her causal story has no ending. Michelle offers an ending for her story, suggesting that this amygdala activity pattern might explain symptom emergence. Here, even a statistically compelling physiological finding (i.e., *Linking*) is situated as incomplete until it can be integrated into a causal story about the brain and illness (i.e., *Explaining*).

To advance their explanatory models, RENE members rely on their longitudinal dataset, which includes three waves of neuroimaging. Specifically, members aim to identify the neurophysiological features that best predict the subsequent emergence of mental disorders. Sara describes a paper she has been working on with Audrey for several years. Sara asks the team for help re-working the paper, after discovering a systematic bias in her analysis software:

> Sara says that she is looking at maternal depression, the participants' childhood cortisol levels, the brain systems activated during an fMRI task, and the appearance of psychiatric symptoms. While discussing amygdala reactivity in the fMRI, she states cortisol "didn't predict anything"

> but "maternal depression predicted a lot." However, afternoon cortisol level is the "only significant predictor of [amygdala] recovery," and is also strongly correlated with maternal depression. Sara asks the group how "this [data] might work together to predict psych[iatric] symptoms." Adding that she wants to find a way to get the model "back to publishable." Irene suggests the "biggest issue is the brain stuff" and "making [that] significant." Sara responds that the "amygdala stuff is still there [i.e., statistically significant]." Michelle agrees with Sara, adding, "the story with amygdala is easy."

Participants' *in utero* "exposure" to maternal depression "predicted a lot" but without a causal story about the "brain stuff," Sara's paper is not competitive in the top-tier journals (e.g., *Science*, *Nature*, *Journal of Neuroscience*) that Becking members pursue. If she cannot use the model to "predict psych symptoms," she faces the prospect of abandoning the paper. Fortunately for Sara, Michelle sees an "easy" story in the data; specifically, Michelle observes that while maternal depression "predicts" mental illness, it is amygdala activity that is predicting "what type" of mental disorder a participant develops, with different activity patterns predicting internalizing and externalizing disorders. In this light, Sara's analysis now potentially reveals how individual differences predict illness, with Michelle optimistically concluding, "it's still a paper."

Like other neurobiologists, RENE researchers pursue the "neural substrates" (e.g., Abend 2017; Pitts-Taylor 2016) underlying behaviors. However, the individual differences framework adds additional complexity, as researchers are not trying only to connect the brain to illness (which is a settled question for members) but working to demonstrate how neurophysiology accounts for differences between the healthy and ill, as well as explaining how variations in brain activity predict the emergence of disparate sets of disorders. Expanding on his analyses introduced in the *Linking* section above, Louis details his individual differences model:

> Louis asks the group if they should touch base on other analyses or potential analyses with the RENE data. Several members murmur "yes" or nod their heads in agreement. Louis says he has been working on some analyses with Sara and they are focusing on identifying and distinguishing the neural substrates for externalizing versus internalizing disorders. Louis adds that they are also attempting to identify any neural substrates that are common for both sets of behaviors.

Here, Louis forwards an analysis that will identify neurophysiological differences between people with internalizing (e.g., depression, anxiety) and externalizing (e.g., ADHD, substance use disorder) conditions, while also attempting to explain what differentiates both "internalizers" and "externalizers" from "healthy" participants. He continues:

> "We want to be looking at how longitudinal change in the data would predict sets of symptoms. We've added that one piece would be how early life stress, in childhood and adolescence, plays into bias in the brain circuitry for one way or another [i.e., either externalizing or internalizing]." Jake asks Louis "what neural substrates are you looking at?" Louis responds, "the idea is that internalizing will be most strongly predicted by amygdala and PFC [pre-frontal cortex] connection, while externalizers map with impulsivity, reward, striatal activation and potentially striatal connectivity.

Although early life stress appears to be a factor biasing brain circuitry, the role this environmental variable plays in his model counterintuitively emphasizes the explanatory power of neurophysiology in an individual differences framework. Put simply, while participants might share exposure to childhood stress, it is connectivity variations in the amygdala, striatum, and PFC that determine what type of disorder (i.e., externalizing, internalizing) an individual develops. Moreover, in Louis' model,

participants who experience early life stress but do not have either of these brain activity patterns are predicted to remain healthy. Thus, it is specific variations in neurophysiology that best explain both health and illness.

As suggested above, it is not the case that the RENE team uses only neurobiological variables in their explanatory models (see Fallin et al. 2018 for a contrastive example), as illustrated by Louis' use of exposure to early life stress and Sara's use of exposure to maternal depression as potential environmental factors. However, when these models are fully explicated, the environmental variables are often accounted for in terms of additional physiological differences. While maternal depression "predicted a lot" in Sara's model, this model holds only for female participants, suggesting to her that there is some aspect of women's neurophysiology required for *in utero* exposure to maternal depression to result in mental disorder. Indeed, an aim of the RENE study is to "explore gender differences," but such analyses focus on articulating how brain differences between men and women might produce different types of disorders (see also Eliot and Richardson 2016). In the individual differences framework, the brain reins in other variables, solidifying and biologizing their impact, and it is ultimately the brain that explains *if* and *how* mental disorders manifest.

Asserting the Causal Importance of the Brain

While *Explaining* details how scientists use their data to make brain-based explanatory models, *Asserting* characterizes how researchers diminish competing frameworks while emphasizing the causal importance of the brain. Here, researchers use the "realness" of brain science to critique non-neurobiological modes of explanation and to inform policy.

Emblematic of Collins' (1985) observation that scientific disagreements are resolved via social processes, in both the lab and at the policy level, brain science is identified as more empirical, more factual, and "more real" (see also Dumit 2004; Joyce 2008) than alternative explanatory approaches. For example, Stephanie (a Becking researcher) critiques social survey research as we watch a neuroimaging session, noting her concerns with both interviewer and question-order effects. In contrast, she suggests neuroimaging data are "more pure and less problematic" because "you can actually see what's going on in the brain." For Stephanie, social scientific tools are inadequate for research, while neuroimaging provides "real data."

Such skepticism towards social research is rare in the Becking Lab, with researchers far more concerned with critiquing non-brain-based approaches in psychiatry. For example, after a RENE meeting, Sara tells me about a university professor who is a "complete dualist" and "doesn't believe in the brain" or psychiatric medicine, as she questions how he is even permitted to supervise students. Similarly, in the extended example that follows, Irene lauds the Becking Laboratory and soundly critiques non-neurobiological psychiatry. She presents her criticism in context of advising Bonnie, who has just been accepted to medical school:

> [In contrast to other medical school rotations], Irene states the "psychiatric stuff" is amorphous and vague. Emulating a psychiatrist, she says, "like maybe [your problem] is this, but maybe it's that. Or maybe it's just because you are in a room full of people." Irene then wonders "how many of these effects [that show therapy improves patients' symptoms] are real" or if "other researchers" are "spending their time just looking for anything that has any significant effect at all." She positions her hand a 45-degree angle to the table. Slightly lowering her hand, she says, "like what does it matter if we change your curve from this to this?"

While Irene's comments resonate with sociological critiques of psychiatry (e.g., Conrad 2005; Horwitz 2002), she uses the same language as NIMH Director Insel (below), suggesting conventional psychiatric treatments are unable to "bend the curve" in mortality and morbidity caused by mental illness. She then connects the inadequacy of psychiatric treatment to flaws in research:

> Irene asks if "the methods [outside of the Becking Lab] are perhaps not as rigorous as they could be." She says the Becking Laboratory uses neurobiology to produce "real" data. She adds, "like, the amygdala is real. It's right there. You can see it. . . . I'm more comfortable to be dealing with something that is tangible like that," than other psychiatric measures, "like that crappy rumination questionnaire."

Here, *Asserting* nullifies the relevance of non-neurobiological modalities, whether they are social scientific or psychiatric. Irene argues that psychiatric treatments are ineffective because they are based on unscientific research. Moreover, given their inability to provide neurobiological explanations, the methods of non-neurobiological mental health researchers are not "rigorous," "real," or empirical, in comparison to the work conducted at the Becking Lab.

The RENE team uses the DSM and these "crappy" questionnaires (including the Rumination Response Scale) to measure participants' symptoms severity (see *Linking*), and these tools become valid only via their connection to neurobiology. As with Irene's critique of traditional psychiatric approaches, RDoC similarly permits conventional standards, measures, and tools as long as they can be connected to cells, neurons, and others aspects of physiology (NIMH N.D.b). This both demonstrates the legitimating role technoscience plays in biomedicalized reconfigurations of health (Clarke et al., 2010) and shows that *Asserting* not only privileges neurobiological data, but that the empiricism of brain data elevates and transmutes traditional tools and measures. What is regarded as unscientific becomes defined by the absence of brain data.

At the policy level, *Asserting the Causal Importance of the Brain* adopts an additional tenor, legitimating the neurobiological turn in mental health (Littlefield and Johnson 2012; Rose and Abi-Rached 2013), and the associated transfer of public resources towards biological approaches (NIMH N.D.b). For instance, at the 2014 Annual Meeting of the American Psychiatric Association (APA), NIMH Director Insel introduces RDoC, NIMH's attempt to "transform" psychiatry via biological research. While the DSM is designed for clinicians and researchers "from many different orientations (e.g., biological, psychodynamic, cognitive, behavioral, interpersonal, and family/systems)" (APA 2013: xli), such conciliatory moves towards non-biological approaches are absent from RDoC Initiative, which labels the DSM a "failure" and illnesses as "brain disorders" (Insel 2013; Whooley 2014).

Insel's APA talk generates substantial interest. Although I arrive half an hour prior to the presentation, I am relegated to sitting on the floor against the back wall, as a security guard prevents those in the hallway from entering the at-capacity room. Insel begins by soundly critiquing psychiatry:

> Insel says it's finally time to start viewing psychiatric disorders as "brain disorders," emphasizing that focusing on the brain "is not reductionist." Insel tells the crowd that even though we know schizophrenia is a neurodevelopmental disorder, we still diagnose it based on the presence of psychosis. Insel quips that this is like defining heart disease by the presence of a myocardial infarction [i.e., heart attack]. Somewhat exasperated, Insel tells the audience that in cardiology, "we don't do that anymore!"

Insel implores the audience to recognize that mental illnesses are brain disorders, situating current diagnostic practices as painfully backward in comparison to other medical fields.

> Insel says that, unlike every other medical field, psychiatry has been unable to "bend the curve in mortality and morbidity" caused by mental illnesses. While speaking, he shows slide after slide documenting how other medical fields have made substantial improvements to treatment, recovery, or life expectancy, and then shows how the outcomes for psychiatric patients have remained stagnant over decades. Insel tells the audience that only through "clinical neuroscience" will we finally "bend the curve in mortality and morbidity." He says adopting clinical

> neuroscience will require talking about psychopathologies as "brain circuit dysfunctions," adding, "We've got to approach the mind through the brain" and acknowledge that the "path to better health care goes through science."

After driving home the therapeutic inadequacy of psychiatry, Insel argues that adopting a brain-based approach, or "clinical neuroscience," is necessary for the field to finally improve the lives of patients. The *Asserting* work of Irene and Director Insel share the same rhetorical strategy of first identifying the failures of psychiatry before establishing neurobiology as the only tenable solution.

While Insel's speech levies critiques against non-neurobiological psychiatry, he also elevates brain science above social/environmental approaches. Nearing the end of the question and answer period, an audience member asks Insel about the major role poverty plays in mental illness and how he reconciles that with his vision of mental health:

> Insel responds, agreeing that socioeconomic status is important but that "ending poverty won't bend the curve [in mortality and morbidity]." Insel then proudly contrasts himself with previous NIMH Directors, stating that they all thought ending poverty was the primary way to improve outcomes for the mentally ill, but he is redirecting NIMH towards innovative, cutting-edge brain science. Insel adds, "poverty is important" but "we need neuroscience."

Director Insel critiques the social focus of previous NIMH leadership, arguing that "ending poverty" won't decrease the incidence or consequences of mental disorders (i.e., "bend the curve"). Insel's claims are contrastive to the incidence of major mental illness among homeless individuals and research linking poverty to mental health (e.g., Turney, Kissane and Edin 2012). However, his statements align with the individual differences perspective endorsed by NIMH (N.D.) and applied in the Becking Lab (see *Explaining* above), as poverty might be associated with mental disorder, but cannot explain why individuals develop these conditions, as many people who encounter poverty (or some other social factor) will not become mentally ill.

As both Irene and Director Insel highlight, in combination, *Linking*, *Explaining*, and *Asserting* legitimate a movement towards "real" brain-based diagnostics and interventions. Through *Linking*, neurophysiology and mental illness become analogous, with mental disorders characterized as "brain disorders" or "brain circuit dysfunctions." *Explaining* articulates the physiological causal processes that produce these brain disorders, outlining avenues for intervention. Resting on these neurophysiological causal arguments, Becking researchers investigate the therapeutic potential of emerging brain-based treatments, such as transcranial magnetic stimulation (TMS), and TMS companies at the APA "product theatre" demonstrate for clinicians how these machines treat illness. Through *Asserting*, the utility of non-neurobiological diagnostics is dissolved, with Katy declaring neurobiologists will be able to "look at [patients'] brain[s] post-treatment and see if they changed and, if not, they might relapse, because they only changed their behavior [and not] their physiology." As a result, "clinical neuroscience" is left as the only valid approach for understanding and treating these brain disorders.

DISCUSSION

This study provides an *in situ* investigation of scientists' practices and interactions (see also Alac 2008; Beaulieu 2002; Dumit 2004; Joyce 2008; Lynch 1985; Pickersgill 2011; Star 1983; Turowetz and Maynard 2015) as they work to explain mental illness with the brain. This study describes three strategies scientists use to make the brain an individual-level cause of illness. First, through *Linking*, scientists imbue existing tools, surveys, and inventories, such as the BDI, SCID, or DSM, with neurobiological meaning. Here, researchers employ fMRI tasks based on reflex-arc logic (Dewey 1896; see also Abend 2017; Danziger 1997; Fallin et al. 2018) to connect brain data to the symptoms (e.g., rumination) and disorders (e.g., major depressive disorder) of psychiatry. Second, through *Explaining*,

scientists make causal models and stories that demonstrate how the brain determines psychiatric outcomes. Third, by *Asserting*, researchers draw on the self-evident realness of neurobiology (see also Alac 2008; Dumit 2004; Joyce 2008) to establish the causal importance of the brain in relation to alternative explanations. Although presented sequentially, these strategies can and do co-occur, as has been detailed above.

These three processes are both distinct and interconnected. *Linking*, without *Explaining* or *Asserting*, relegates neurobiology to the domain of associational data, with the brain unattached to broader arguments. The causal models in *Explaining* cannot operate without the data produced in *Linking*, and these causal arguments would remain bound to the laboratory without *Asserting*. Similarly, *Asserting* is speculative neuro-rhetoric without the empirical arguments generated through *Linking* and *Explaining*. That is, *Linking* and *Explaining* provide a scientific narrative for an emerging rational myth (Meyer and Rowan 1977) in psychiatry (i.e., *Asserting*), which situates neurobiologists as both redeeming and "transforming" psychiatry. I argue that these three strategies provide a framework for examining how scientific practices provide individualistic explanations of social issues.

Some social scientists caution against criticizing the implications of neurobiological research (Rose 2013) or call for the integration of social and neurobiological approaches (e.g., "neurosociology," see Franks 2010). Others critique the reductive, acontextual, and thin descriptions of human experience forwarded by neurobiologists (e.g., Abend 2017; Fallin et al. 2018; Pitts-Taylor 2016; Vidal 2009). While this analysis demonstrates how scientists make and elevate such thin descriptions through *Linking*, *Explaining*, and *Asserting*, it also aligns with arguments identifying policy as a driver of reductive approaches (Campbell 2007; Halpin 2016; Whooley 2014; Wynne 2005). Specifically, in his RDoC announcement at APA, Director Insel encourages professionals to join the neurobiological bandwagon (Fujimura 1996), identifies brain science as the future of psychiatry, and outlines his intention to direct funding to physiological research. In this context, laboratories characterizing mental illness as a product of neurophysiology, such as the Becking Lab, are liable to be the "winners" in relation to funding, media coverage, and institutional support.

There is also a reductive symbiosis between neurobiological laboratories and NIMH. The Becking Lab situates mental illnesses as the product of individual differences, a framework that is endorsed by NIMH (N.D.) and central to RDoC (Cuthbert and Insel 2013). NIMH tailors funding to support neurobiological research, while labs like the Becking Lab produce empirical research buttressing Insel's claims that mental disorders are "brain circuit dysfunctions" that require "clinical neuroscience." In this context, it is not surprising that Irene's statements on the inferiority of non-neurobiological methods and language about "bending the curve" echo NIMH positions. An additional byproduct of this relationship is that social/environmental factors are of diminished explanatory relevance in an individual differences framework, and such explanatory insufficiencies in research underscore the necessity for programmatic support of neurophysiological research. With the individual differences framework emphasizing the explanatory primacy of individual-level neurobiological variations, RDoC's physiological truncation of "social processes" (NIMH N.D.b), and NIMH's decreased interest in the social determinants of health, the social disadvantages tied to mental illness are liable to be biologized (see also Conrad 2005; Clarke et al. 2010; Halpin 2016; Horwitz 2002; Kempner 2014; Pickersgill 2011; Pitts-Taylor 2016; Whooley 2014), as *Asserting* ensures there will be no alternative framework for evaluating their impact.

The conversion of mental illness into the domain of "brain facts" (Star 1983) likewise challenges non-neurobiological psychiatric modalities (e.g., psychodynamic, cognitive, behavioral, interpersonal, and family/systems approaches; APA 2013), research on the social determinants of health (e.g., Link and Phelan 1995), and biosocial approaches in both sociology and psychology (e.g., Engel 1977; McEwen and McEwen 2017). Indeed, the RENE study has ample data on social factors (e.g., income, race, educational attainment) to pursue comparable investigations, but these analyses are of dubious utility when mental illnesses are perceived as brain disorders that result from variations in individual neurophysiology.

Science has considerable social implications (e.g., Fujimura 1996; Keating and Cambrosio 2011; Halpin 2020; Nelson 2018; Pitts-Taylor 2016), and when laboratory work meets public policy, the brain becomes not just a cause but also a solution. The brain is not just the centerpiece of a causal model on the white board of a prestigious laboratory, but, for Becking members, NIMH, and Director Insel, it also becomes a site of intervention. This formulation of mental illness as a problem of neural circuits and brain structures is emblematic of larger shifts towards biomedicalization (Bell and Figert 2012; Clarke et al. 2010) and evidence of the new directions of medicalization (Conrad 2005). Although brain science is omitted from Conrad's (2005) reformulation of medicalization theory, this article demonstrates that neurobiology also drives medicalization. While Conrad (2005) highlights the commodification potential of genetics (e.g., genetic manufacture of hormones), neurobiological research likewise legitimates brain-based products, such as TMS, electroconvulsive therapy, and deep brain stimulation. Although these neurotechnologies are currently used to treat conditions like depression and obsessive-compulsive disorder, meta-analyses suggest most patients do not respond, and many who do respond do not experience lasting remission of their symptoms (see Fagan et al. 2019, for example). As the first psychiatrist to clinically use TMS, Dr. Mark George, further observes, these treatments cost $5,000-10,000 dollars, while comparable medications cost "a couple hundred bucks" (Raeburn 2016). Psychiatry might require "clinical neuroscience" to "bend the curve" in mortality and morbidity, but a clear upside of these treatments is increasingly the profitability of mental health care.

Although medicalization theory (Conrad 2005) focuses most precisely on diagnostic expansion, the present study shows that the scientific revision of categories is a salient dimension of medicalization. Specifically, the RENE study does not produce new psychiatric disorders, but instead reformulates existing disorders in neurobiological terms. Here, neurodevelopmental problems slowly escalate over the course of years or decades before resulting in mental illness. Via neurodevelopmental models, everything after and including the *in utero* phase of participants' lives is relevant to pathological development, legitimating numerous therapeutic entry points across the life course. For example, individuals defined as "at risk" for schizophrenia receive pharmaceutical and therapeutic intervention before they meet official diagnostic criteria (Liu and Demjaha 2013) based on the perception that "schizophrenia is a neurodevelopmental disorder" (see *Asserting* above). This technoscientific reformulation of illness, as well as the considerable expansion of risk surveillance is likewise emblematic of biomedicalization processes (Clarke et al. 2010). As therapeutic entry points increase, the contestation of clinical assessments might conversely be reduced, with brain data positioned as more accurate than a patient's own self-report (see *Asserting*). Even when diagnostic categories are revised rather than created, laboratory work "drives" medicalization, redefining mental illness from a transient or episodic experience to a lifelong neurodevelopmental problem.

This paper also suggests how core biomedicalization processes might interact (Clarke et al. 2010). Specifically, my analysis demonstrates a feedback loop between the processes of increasing reliance on technology, transformations of biomedical knowledge production, and the elaboration of risk and surveillance. First, RENE researchers rely on neuroimaging technologies to produce knowledge about the brain and mental illness. Second, knowledge about the brain is promptly coupled with risk and surveillance (see also Eliot and Richardson 2016; Pitts-Taylor 2016; Vidal 2009), such as the RENE researchers' suggestion that female brains are most "vulnerable" to in utero exposure to maternal depression. Third, while these steps demonstrate a technology-knowledge-surveillance interaction, the interaction continues as perceptions of risk and surveillance guide subsequent knowledge production, such as Sara and Louis' attempts to find the neural substrates that produce externalizers and internalizers, as well as Insel's steering of NIMH in order to "bend the curve" of psychiatric mortality and morbidity. Finally, the feedback loop closes as knowledge production on neurophysiological risks elevates neuroimaging technologies. That is, once mental disorders are situated as neurodevelopmental conditions, neurotechnologies become the only adequate means of studying these "brain diseases," while traditional psychiatric measurement tools are no longer "real," and, without an association to

brain data, are relegated to the methodological dustbin. Accordingly, this paper has articulated one possible way by which three biomedicalization processes might interact.

While my study details how neurobiologists establish the brain as a cause of mental illness, the study has limitations. First, ethnographic fieldnotes were collected at a single, extremely well-resourced laboratory, with members unfavorably comparing their previous labs to the Becking Lab. Future research could attend to these less-resourced laboratories that nonetheless contribute to neurobiological scholarship. Second, the RENE study is longitudinal and many neurobiological investigations of mental illness are cross-sectional. Although *Linking* and *Asserting* might not change in a cross-sectional study, such projects would display a different orientation to *Explaining*, as they lack the longitudinal data RENE members use to form their causal models. Lastly, the Becking Lab heavily supports interdisciplinary research and were supportive of, and interested in, my research. These qualities of the lab are perhaps reflective of case selection bias and a laboratory amenable to ethnographic observation might differ in important ways from a lab less enthusiastic about hosting an outside observer.

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