Whose Schizophrenia Is It? An Intersectional Approach to Schizophrenia and Psychiatric Knowledge

by

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Class of 2020

A thesis submitted to the faculty of Wesleyan University in partial fulfillment of the requirements for the Degree of Bachelor of Arts with Departmental Honors in Neuroscience and Behavior Program and the Science in Society Program

Middletown, Connecticut        April, 2020
This thesis is dedicated to all of the patients at River Valley Services. This project could not have happened without you. Thank you for entrusting your stories with me. I am forever grateful to have worked with you all.
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Acknowledgements

This project could not have happened without two incredible professors who believed in me. I would like to express my gratitude for my mentors, Dr. Anthony Hatch and Dr. Matthew Kurtz, for giving me the immense privilege of pursuing a project that has become my passion over the past year. Thank you for teaching me that criticisms of science can not only work in two departments, but that such projects should also extend beyond this thesis. Professor Hatch, thank you for interpreting my ramblings to tangible ideas; thank you for being there for me during my crises, both academic and personal. Professor Kurtz, thank you for trusting me in working with you and this project. It has been an incredible privilege to work in the lab, and I cannot express my gratitude for the experiences I have gained. Thank you both for encouraging me to bridge together these two disciplines and showing me that I can be a critical neuroscientist.

Thank you to Dr. Margaret Meskill for supporting me at RVS throughout this project from my first day. I am grateful for your constant clinical supervision and support. Thank you for teaching me both the messiness of clinical research and also the solutions to that messy science.

Thank you to all the members of the Wesleyan Schizophrenia Cognition Lab. Thank you particularly to Christina for introducing me to the world of clinical research. Your mentorship means the absolute world to me and this project could not have happened without you. Thank you to each lab member who worked with me to collect this data, past and present. This thesis could not have happened without you all.

Thank you to Ronnie Hendrix and Dr. Erika Taylor at McNair for their constant mentorship and support throughout the past few years. I am forever grateful for every moment I’ve had with the McNair Program. Thank you for believing in me as a scientist.

Thank you to my parents and family, who although may not understand what I’m up to at any given point, trust that I’m doing good for the world. Thank you for supporting me through my confusing journey in academia — especially during the past few weeks of thesising-while-at-home. Sizi çok seviyorum. Her şey için çok teşekkürler. Desteğin benim için dünya demektir. Kendime inanmadığım zamanlarda bile bana inandığım için teşekkür ederim.

Thank you to my chaotic housemates at 146 Cross. Thank you for filling my final ¾ of a year with laughter, strange decorations, and even stranger interactions. Thank you for suggesting sentence structures for this thesis in both the best and worst of times. Thank you to all of my friends at Wesleyan who supported me throughout my years. Thank you to Mo’ath, Preksha, Clara, and Kati for your unwavering support and belief in me. I could not do this without you.
Introduction

It was 9:50 AM as I was quickly ascending the subway stairs towards New York Presbyterian Hospital. Tucked in my backpack were high heels, a tattered Moleskine notebook, and a folder filled with my new Primary Investigator’s (PI) recent papers. I glanced at the instructions I was given — go to the security and say you’re going to 1540 — and I walked over to the security guard who handed me a guest pass. “Don’t you need my ID?” I asked, but the security guard shook his head. I found the set of elevators that would take me to the fifteenth floor and stood next to a sea of white coats. Each checked their respective technologies, quietly noting their tasks for the day. I joined in on the quiet elevator environment.

I was just beginning my summer-long volunteer position at Columbia’s Perinatal Pathways Laboratory, which focused on the effect of maternal mental health on the fetus during pregnancy. I quickly took on the task of recruiting participants, receiving a small pile of referral papers with patient contact information. The papers were filled with post-its, dauntingly noting the many failed attempts of volunteers before me who had tried to reach potential participants. I held the script in my hand and called the first number. The call went directly to voicemail. I read the script and called again. Voicemail. Repeat. With each call, I would pronounce my name May-LEE-sah — the Turkish way — with an inflection pattern that resembled that of an educated, white woman, a juxtaposition that always elicited responses of praise in white spaces.

It was rare for someone to pick up the phone. When it did happen, I would begin the conversation by introducing myself. The women on the other line usually
had accents themselves, and I was always uncomfortable thinking about my own background as the daughter of two immigrants; my mom, a Middle Eastern immigrant who carries a strong accent, could have been a potential participant on the other line. When Hispanic women answered, I sensed a moment of confusion because my name sounds vaguely Spanish, but my years of Spanish in high school would not be sufficient for this discussion should the Hispanic women mistakenly identify me as one of them and continue the conversation in their language. I immediately recognized Middle Eastern and vaguely “Muslim” names, hoping that the person on the other line would realize that I was like them, too. Most of these attempts were unsuccessful — I assume because the label “Columbia University” overshadowed any immediate establishment of ethnic empathy.

My heart raced as I went from introducing myself to starting a conversation, moving from casual chatter to probing, “I’m now going to ask you some personal questions that have to do with your mental health. Is that okay?” Despite my lack of professional training in psychoanalysis — which might have prepared me to adequately engage with subjects — they always agreed to talk. They knew me as Melisa, a Research Assistant at Columbia University Medical Center (CUMC). The title itself gave me power that I did not rightfully earn. Here I was, a twenty-one-year-old, given the authority to decide if the woman on the phone was depressed enough to be considered for the study.1

1 The senior research assistants, of course, oversaw the recruitment process; however, I was the first person to determine if they would be further evaluated.
Conducting research at CUMC was drastically different from what I expected. Because CUMC is located in Washington Heights — and most of its partner clinics were either in Harlem or the Bronx, areas not yet chosen as the next sites of gentrification — the majority of the women I was contacting were poor women of color. The CUMC complex was not like other hospitals in New York City, with hospitals like Lenox Hill appealing to the Upper East Side wealthy, and NYU Langone for the Downtown gentrifiers. The study offered a stipend of around $700, a good sum of money for a project that spanned four months postpartum. At first glance, this study seemed to be doing the right thing by diversifying its sample size. After all, women of color are often misdiagnosed with anxiety and depression, clinicians sidestepping their trauma from structural violence — research studies have just begun to acknowledge their inability to generalize findings across populations due to majority-white sample sizes (Ali, 2004). I knew that psychiatric research has generally marginalized poor people of color, but I assumed that having a more diverse participant pool would result in better research.

In time, I began to realize that a so-called “inclusive” research paradigm cannot exist when psychiatry’s foundation is linked to oppressions related to racism, classism, and the patriarchy. Further, I realized that an inclusive and structurally competent psychiatry cannot exist unless researchers acknowledge how race, class, and gender have influenced the methods used to create psychiatric knowledge and begin a much-needed conversation about the politics of psychiatric

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2 There is an important conversation to be had about studies that often easily recruit low-income communities because of the high stipend. Needless to say, many of the women who agreed to participate in the study depended on this stipend for financial assistance.
knowledge-making (Structural Competency, n.d). Although traditional scientists may claim that acknowledging external influences like race, class, and gender decreases the objectivity of the sciences, this form of objectivity as conventionally described does not exist in the first place. Drawing on ideas from feminist science studies, I argue that research surrounding psychiatric disorders, and specifically, schizophrenia is narrowly applicable not only because of how it has historically been conducted, but also because of the embedded bias of those who create psychiatric tools to define and diagnose disorder. This is not to invalidate the contributions of clinical research, but instead, I want to offer critiques and solutions that will allow for an understanding of psychiatry beyond the limitations imposed by the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* and the litany of statistical measures that categorize symptoms. In this thesis, I use intersectionality and co-production as frameworks to understand the inequality in diagnoses of schizophrenia by destabilizing the truths of schizophrenia produced by psychiatric institutions. This thesis, as a result, moves towards seeing the contours of an inquiry that questions *how* we know *what* we know about schizophrenia.

Moreover, I argue that the binary in which mainstream psychiatric knowledge operates — the biological or the social — fails to consider the interrelatedness between these two entities. Rather than existing in two distinct and separate realms, the biological and social are simultaneously intertwined in the production of psychiatric knowledge. Although this argument complicates the way psychiatric knowledge-making is understood, I aim to adapt existing models of
knowledge-making, namely the Scientific Method, to acknowledge the intertwined nature of science and society in psychiatric knowledge-making (Roy 2004). This reframing will prompt scientists, including myself, to consider how biases in race, class, and gender play a role in and fundamentally shape psychiatric knowledge-making, encouraging a much-needed reflection of how current research reinforces inequalities in mental health and psychiatric treatment for minority groups. This framing will blur the subject-examiner dichotomy produced by the Scientific Method and place the scientist as both actor and subject in knowledge-making.

This thesis specifically examines how the intersections of race, class, and gender co-produce schizophrenia with psychiatric knowledge-making. In Chapter 1, I will interrogate the so-called objectivity of psychiatric disorders. By complicating the psychiatric literature regarding schizophrenia, I question how these sources consider race, class, and gender as risk factors and not social structures in producing schizophrenia. Such conceptions of schizophrenia are incomplete because they only consider descriptions of disorder that are bound to conventional psychiatric institutions, thus ignoring how schizophrenia intersects with individual experiences. Guided by literature on the social determinants of health, I discuss how the development of mental disorders is deeply affected by inequalities caused by racism, classism, and the patriarchy. As such, these oppressions result in poor, Black Men being disproportionately diagnosed with

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3 Roy’s use of Capital-M, Capital-S Scientific Method is deliberate as it acknowledges that the Scientific Method acts as its own institution of knowledge-making; consequently, the Scientific Method is an agent in producing a particular way that we both create and understand psychiatric research and knowledge. This will be discussed further in Chapter 1.
schizophrenia despite textbook definitions that argue that schizophrenia occurs in one percent of any given population (Barnes, 2004, 2008, 2013; Bresnahan et al., 2007).

This analysis, guided by Crenshaw’s (1991) intersectionality and Jasanoff’s (2004) co-production, blurs the binary between the biological and social in its complex interactions with science, power, and society that frame psychiatry (Jasanoff, 2004). These frameworks will allow me to problematize the way we know schizophrenia, which I see as a product of three hierarchical bodies. First, schizophrenia is produced with the Scientific Method’s knowledge production; next, the Scientific Method creates the descriptive tools used to enumerate schizophrenia; finally, the diagnoses are assigned to individuals based on these tools. Among the tools I will interrogate are the Diagnostic and Statistical Manual of Mental Disorders (DSM) and the Positive and Negative Syndrome Scale (PANSS), two constructs that are heavily implicated in a racist, sexist, and classist psychiatry. With this framework, I will examine the epidemiology of schizophrenia using both intersectionality and co-production to question how we understand diagnosis. This reframing will allow for an embedded acknowledgement of the external influences in psychiatric knowledge-making.

This thesis, however, does not position itself purely in the theoretical domain. Instead, this understanding of psychiatric knowledge-making guides me through an ongoing research study with which I am affiliated in the Schizophrenia Cognition Laboratory at Wesleyan University. In Chapter 2, I will present our research on a study that examines the efficacy of two models of cognitive
remediation (CR) on people with schizophrenia. By examining this research, I will interrogate the descriptive tools that we use to assess the symptoms of our clients and will offer an alternative conclusion on our research study according to Roy’s feminist model of science-making. This will allow for a tangible contextualization of the implications of co-production as outlined by Chapter 1.

Moreover, the co-production of schizophrenia holds implications far beyond the laboratory and the clinical setting. In Chapter 3, I examine how personal autonomy shifts with a diagnosis of schizophrenia. Oppressions linked to racism, classism, and the patriarchy bleed into the lived experiences of schizophrenia, seen through the narrative accounts of people with schizophrenia as they navigate through psychiatric institutions. By exploring different narrative accounts of schizophrenia, I argue that the lived experience of schizophrenia is as critical to psychiatric knowledge-making as the descriptive tools used to enumerate symptoms. I further argue that science cannot address disparities in research if it fails to take these critiques of psychiatric knowledge-making into account. Thus, in order to reconcile the marginalization of people with schizophrenia by psychiatric institutions, those same individuals should be placed on an equal footing with those producing the knowledge; these stories matter, and it is my aim to put the lived experience of schizophrenia at the forefront of this thesis.
Chapter One: Ways of Knowing Schizophrenia

I: Schizophrenia: A Brief Diagnostic Explanation

According to the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*), schizophrenia is a chronic and severe mental disorder that is typically diagnosed between the ages of 16 and 30 (American Psychiatric Association, 2013). Divided into positive and negative symptoms and cognitive disorganized features, schizophrenia categorizes how people think, feel, and behave.

The positive symptoms of schizophrenia are the most easily identifiable, characterized by a distorted sense of reality. Such distortions include (1) delusions and (2) hallucinations. First, although delusions in schizophrenia vary, they are understood as unwavering false ideas or beliefs that do not fall within social and cultural norms; second, hallucinations are defined as perceptions that are not present in the environment. Hallucinations can be auditory, visual, or olfactory (Arango & Carpenter, 2011). Auditory hallucinations, colloquially known as “hearing voices,” are the most common type of hallucination, experienced by over 70% of people with schizophrenia (Hugdahl et al., 2007).

While the positive symptoms of schizophrenia are the most easily identifiable, the negative symptoms are associated with abnormalities in typical emotional and social behaviors. Such abnormalities include the reduction of speech, emotional expression, and feelings of pleasure (American Psychiatric Association, 2013). Difficulty speaking (alogia), flat affect, inability to experience pleasure

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4 Flat affect is characterized by the reduced expression of emotions. This can manifest via facial expressions or vocal tone.
(anhedonia), asociality, lack of motivation (avolition), and overall lack of interest in daily activities are among the most common negative symptoms (Arango & Carpenter, 2011).

The cognitive features of schizophrenia, divided into neurocognition and social cognition, affect memory and thinking, including executive functioning, focusing, and working memory. Finally, disorganized features are characterized by an unusual or disordered way of thinking and behaving. People with schizophrenia typically exhibit disorganized features through abnormal speech patterns. For example, “conceptually disorganized” features are associated with the inability to link a series of conceptual ideas and can lead to difficulty forming coherent sentences (Green, 2001).

**Diagnosis and Treatment**

A diagnosis of schizophrenia is made by a clinician through a psychiatric evaluation. Symptoms are identified using the *DSM-5*, a manual for assessment and diagnosis of mental disorders (American Psychiatric Association, 2013). The *DSM* details the symptoms of mental disorders, but does not include treatment guidelines, and as such, the *DSM* is often viewed as an objective tool to standardize diagnosis. In order to be diagnosed with schizophrenia, a person must have two or more core symptoms — delusions, hallucinations, disorganized speech, grossly disorganized or catatonic behavior, and negative symptoms — for at least one month with at least

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5 Later in this section, I will interrogate this claim. Needless to say, the *DSM*, like any other descriptive tool, is not objective.
one symptom presenting as either delusions, hallucinations, or disorganized speech.

The *DSM-5* also specifies other criteria for schizophrenia, such as markedly low functioning in work, interpersonal relations, or self-care after the first disturbance. Further, a *DSM* diagnosis of schizophrenia requires a six-month timescale for the disturbance to create a distinction between schizophrenia and similar disorders, such as schizoaffective disorder and depressive bipolar disorder with psychotic features.

Although the *DSM* is widely used in the clinical setting, there are many descriptive tools that are used in the laboratory to describe and enumerate the symptoms of schizophrenia. These evaluative tests are used to assess the severity of the positive and negative symptoms and cognitive features. Table 1 lists the measures used our the study at the Schizophrenia Cognition Laboratory.

<table>
<thead>
<tr>
<th>Name of Assessment</th>
<th>Authors and Year Produced</th>
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<tr>
<td>Positive and Negative Syndrome Scale (PANSS)</td>
<td>Kay, Fiszbein, &amp; Opler 1987</td>
</tr>
<tr>
<td>Wechsler Test of Adult Reading (WTAR)</td>
<td>Wechsler 2001</td>
</tr>
<tr>
<td>Self-Efficacy Scale (SES)</td>
<td>McDermott 1995</td>
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<tr>
<td>The Awareness of Social Inference Test (TASIT)</td>
<td>McDonald, Flanagan, Rollins, &amp; Kinch 2003</td>
</tr>
<tr>
<td>Social Functioning Scale (SFS)</td>
<td>Birchwood, Smith, Cochrane, Wetton, &amp; Copestake 1990</td>
</tr>
<tr>
<td>UCSD Performance-Based Skills Assessment (UPSA)</td>
<td>Mausbach, Harvey, Goldman, Jeste, &amp; Patterson, 2007</td>
</tr>
<tr>
<td>The Specific Level of Functioning Scale (SLOF)</td>
<td>Schneider &amp; Struening, 1983</td>
</tr>
<tr>
<td>Wisconsin Card Sorting Test (WCST)</td>
<td>Berg, 1948</td>
</tr>
<tr>
<td>Hopkins Verbal Learning Test Revised (HVLT-R)</td>
<td>Shapiro, Benedict, Schretlen, &amp; Brandt, 1999</td>
</tr>
<tr>
<td>Controlled Oral Word Association Test (COWAT)</td>
<td>Lezak, 1995</td>
</tr>
<tr>
<td>Memory for Intentions Screening Test (MIST)</td>
<td>Raskin, 2009</td>
</tr>
<tr>
<td>Wechsler Adult Intelligence Scale (WAIS-IV)</td>
<td>Wechsler, 2008</td>
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*Table 1.* Assessments used in the Schizophrenia Cognition Laboratory.
In particular, the Positive and Negative Syndrome Scale (PANSS) is one of the most widely used scales in schizophrenia research. A 30-rating scale designed to assess different dimensions of the symptoms of schizophrenia, the test was originally grouped into three scales to enumerate positive symptoms, negative symptoms, and general psychopathology (Kay, Fiszbein, & Opler, 1987). Since its publication in year 1987, the PANSS has been adapted into multiple alternative models that, it is argued, better enumerate and fit the symptoms of schizophrenia. Among them is the 5-Factor model, which expands the scales to positive, negative, disorganized/concrete, excited, and depressed (Lançon, Auquier, Nayt, & Reine, 2000). In Chapter 2, I will examine the history of the PANSS in more detail.6

Once diagnosed, people with schizophrenia are commonly treated with antipsychotic medication that best reduces the positive symptoms, often paired with therapy. Treatments that specifically target the cognitive features are currently under investigation; in particular, cognitive remediation (CR) has been suggested to be promising, which I will also discuss in Chapter 2.

Critiques of the Construction of the Diagnostic Category

The simple, so-called “objective” criteria that the DSM and other descriptive tools offer are, at best, incomplete, raising fundamental questions about whether schizophrenia exists at all. Scholars in the “anti-psychiatry” movement in the 1970s argued that mental illness was a myth and was instead a diagnosis tied to

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6 Suffice it to say, the ongoing evolution of the PANSS raises questions to the usefulness of the PANSS in clinical psychology.
While anti-psychiatrists critiqued the influence of social, political, and cultural forces on the construction of mental illnesses, they failed to offer a viable treatment plan. In response, Jonathan Metzl (2009) acknowledges that although schizophrenia as a diagnostic category has been and is still influenced by factors external to medicine, questioning the empirical reality of schizophrenia often feels like a pointless debate. He suggests that stigmatizing encounters between doctors and patients are not limited to visual markers of difference like race, but are instead influenced by the diagnosis itself and its relationship to protest, resistance, and racism (Metzl, 2009).

To my mind, the experience of schizophrenia is a lived reality for people even if the category and its empirical parameters remain fuzzy. People who are diagnosed with schizophrenia not only experience the symptoms that are associated with what is called schizophrenia, but they also experience the stigma associated with being labeled as a “schizophrenic.” Instead of thinking of schizophrenia as a singular entity, I argue (along with Metzl) that schizophrenia “[aggregates] certain symptoms into particular psychiatric diagnoses [that] exist in an ongoing state of flux” (Metzl, 2009, p. xix). This flux can be both positive and negative, and the latter occurs when the different powers that allow for the flexibility of diagnosis are not monitored.

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7 Szasz (1979), Cooper (1967), and Laing (2010), to name a few
8 Chapter 3 further explores Metzl’s characterization of diagnosis as the “third race that functions in the examination room” (Metzl, 2009, xix).
9 Importantly, the field is attempting to move towards first-person language — from “schizophrenic” to “person with schizophrenia” — however, the stigma still exists despite this change.
This diagnostic flux of schizophrenia is apparent in early psychiatry, as Harrington (2019) suggests that in the twentieth century, clinicians split schizophrenia into different subtypes to account for symptomatic differences between patients. She states, “The diagnosis was applied both to floridly psychotic and to socially isolated patients, and to confused, belligerent, and underachieving adolescents,” and notes that this “slicing and dicing” of schizophrenia did not go unnoticed (Harrington, 2019, p. 140). Among those who were concerned about the diagnostic methods was R.G. Hoskins, who asked, “Is it an entity, or mayhap, merely a semantic conversation?” (Hoskins, 1946, p. 7). Yet, the use of the diagnostic category persisted, allowing for bias culture to affect the category. As Metzl notes, the flux of schizophrenia produced negative consequences as it created the category of the angry, Black, male schizophrenic. He states, “Racial concerns, and at times overt racism, were thereby written into diagnostic language in ways that are invisible to us now” (Metzl 2009, xix). Diagnostic language is further influenced by a sexism and classism that are still felt today, as we will later see with the contemporary epidemiological accounts of schizophrenia.

10 When Bleuler coined the term “schizophrenia” in the early 20th century, he did not think it was a singular unified condition. Instead, he spoke about “the schizophrenias” (Bleuler, 1950).
II: Race, Class, Gender, and Power: How Science and Technology Studies Can Help Explain Psychiatry

Intersectionality as a Means of Understanding Schizophrenia

In order to deconstruct the interactions between society and psychiatric knowledge-making, it is essential to understand how race, class, and gender work in tandem to produce societal realities. This understanding, however, cannot be viewed within a binary that treats them as mutually exclusive entities. Instead, race, class, and gender must be viewed as social systems that work *simultaneously* in producing our understanding of schizophrenia. Therefore, examining schizophrenia requires an intersectional framework that directly addresses these interactions (Crenshaw, 1991). According to Cho, Crenshaw, and McCall (2013), intersectionality examines how “the dynamics of difference and sameness has played a major role in facilitating consideration of gender, race, and other axes of power” (Cho, Crenshaw, & McCall, 2013, p. 787). Although intersectionality was first theorized to analyze Black women’s subjectivity, it has since been expanded to constitute a body of critical social theory (Collins, 2019). As such, intersectionality examines the relationship between different institutionalized structures of oppression and the possibilities for resistance that these systems expose.

Within the context of schizophrenia, intersectionality can help explain the contemporary epidemiological accounts that attempt to identify the risk factors for
This is imperative because, as Metzl suggests, “institutional forces supersede the best individual intentions when race and insanity are the topics of diagnostic intervention” (Metzl, 2009, p. xii). When non-intersectional scholarship treats race, class, and gender as competing and independent systems of power, they miss how these systems of power cross and interact with each other to produce structure inequality. Collins (2019) suggests that intersectionality directly challenges these binaries, stating,

Intersectionality has certainly contributed to paradigm shifts in thinking about how mutually constructed power relations shape social phenomena. Across academic disciplines, traditional paradigms approached racial inequality and gender inequality, for example, as distinct, separate, and disconnected phenomena…their interactions remained invisible because no one thought to look for them…intersectionality was not just an adjustment to business as usual. It pointed toward a fundamental paradigm shift in thinking about intersecting systems of power and their connections to intersecting social inequalities (Collins, 2019, p. 43).

Thus, intersectionality reframes these binaries by recognizing that racism, classism, and sexism work together to shape individual experiences, social groups, and social

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11 Attempt is important here. Later in this chapter, I will present several epidemiological explanations of schizophrenia that do not offer much room for an intersectional analysis. This is frustrating at best, deeply problematic at worst because there is no one source that examines the intersection of race, class, and gender in schizophrenia diagnoses. Reconciling this gap will necessitate embedding intersectionality in future epidemiological studies.
institutions. Moreover, racism, classism, sexism, and related oppressions may “mutually construct one another by drawing upon similar and distinctive practices and forms of organization that collectively shape social reality” (Collins, 2019, 46). In other words, institutionalized structures of oppression always work simultaneously instead of independently. Thus, intersectionality is crucial to problematize the conclusions of epidemiological data because it is unable to sufficiently interrogate the influence of race, class, and gender. As we will see later in this chapter, this interrogation destabilizes assumptions of epidemiological truth.

Similarly, debates regarding the biological versus environmental etiology of mental illness fall into the same binary logic applied to race, class, and gender. The commitment to producing psychiatric knowledge within a biology-environment binary begins with psychiatry’s presumption that mental illness results from a disordered brain, affecting people of all races at a similar rate. Schizophrenia is among the most biologized mental illnesses — with the search for a biological cause of schizophrenia predating modern neuroscience — with multiple theories suggesting that defects in brain structures, peptides, and neurotransmitters cause the disorder. According to Harrington (2019), biologizing mental illness began in the late nineteenth century with psychiatrist Emil Kraepelin, who, by focusing on the anatomical basis of mental disorder, called for the medicalization of psychiatry (Harrington, 2019). By the twentieth century, schizophrenia became “the great nut to be cracked” (Harrington, 2019, p. 47). Initial theories pointed towards infectious agents as the cause of schizophrenia, but with time, research began to suggest a
binary root for schizophrenia, suggesting that mental disorder either biological or environmental. Harrington problematizes this binary, stating,

[Schizophrenia] was curable or it wasn’t. If curable, it was best treated with intensive psychotherapy, or it was best treated with insulin, electric shock, or surgery. If biological, then metabolic and endocrinological research was the most promising direction, or maybe it was genetic research. If environmental, then it was probably caused by a bad mother, except no one could decide what exactly made her bad. Some said she was rejecting, others called her rigid, others thought she was domineering, and still others focused on her anxiety (Harrington, 2019, p. 139).

The debate between the biological and social prompted numerous studies that attempted to find the cause — and cure — of schizophrenia. Some studies argued that family life caused schizophrenia, with Freudians placing the blame on mothers for creating schizophrenic children. With time, these arguments dissipated, and the focus of schizophrenia research shifted to neurobiology. With the creation of lysergic acid diethylamide (LSD), chemists began to see that hallucinogenic trips were similar to experiences reported in people with schizophrenia. This led to a surge of research that attempted to explain the neurochemical foundations of schizophrenia. Excessive serotonin was the first to be suggested, but dopamine was

12 Harrington cites Louise Wilson’s (1969) memoir This Stranger My Son, who quotes her son’s psychiatrist. The psychiatrist states, “Let me put it this way. Every child born, every mind, is a tabula rasa, an empty state. What is written on it” —a stubby finger shot out, pointed at me—“you wrote there” (Harrington, 2019, p. 143; Wilson & Wilson, 1968).
ultimately to blame. With the acceptance of the dopamine hypothesis, the characterization of schizophrenia shifted: what was considered a psychological imbalance for the mad was now reframed as a mental illness derived from chemical imbalances.

The expansion of neurochemistry and the dopamine hypothesis leaves us with an incomplete picture of not only what schizophrenia is, but also to whom it affects. Studies like that of Keshavan, Nasrallah, and Tandon (2011) — which critically assessed all “facts” of schizophrenia — do not let this ambiguity go unnoticed. An analysis of seventy-seven “facts” revealed that none of the established “facts” regarding schizophrenia were fully robust, lacking criteria essential to valid science-making such as reproducibility, relevance for understanding schizophrenia, or durability over time (Keshavan, Nasrallah, & Tandon, 2011). They state that the field seemed to be operating “like the fabled six blind Indian men groping different parts of an elephant coming up with different conclusions…there may be no elephant, more than one elephant, or many different animals in the room” (Keshavan et al., 2011, p. 10).

While the metaphor may be a bit tongue-and-cheek, it highlights a central issue with any efforts to biologize schizophrenia: literally no one knows what in the world is going on. Although schizophrenia should occur in one percent of any given population, epidemiological studies consistently report that Black men are three to five times more likely to be diagnosed with schizophrenia (Barnes, 2004, 2008, 2013; Bresnahan et al., 2007). Clearly, there are other factors that lead to the disparity in diagnoses. According to Miller (2010), the DSM provides symptoms of
mental illnesses, rather than the *causes* of mental illness, stating that the National Institute for Mental Health (NIMH) has funded research that examines both the neural and genetic basis of psychiatric illness (Miller, 2010).

These research studies, however, only explore the causes of schizophrenia within the aforementioned binary. Studies that focus on the social determinants of mental health offer an alternative lens into the relationship between social factors and mental health (Cuffe, Waller, Cuccaro, Pumariega, & Garrison, 1995; Holley, Tavassoli, & Stromwall, 2016; Sparks, 2002). According to Fisher and Baum (2010),

Public health research on social determinants of health (SDH) within a particular society investigates and compares patterns of health status within and between subpopulations defined according to characteristics such as socioeconomic status (SES) assessed by levels of income or education, living or working conditions, gender or ethnicities (Fisher & Baum, 2010, p. 1057).

They argue that prevalence of many common, non-communicable diseases increases with lower socioeconomic status, with the lowest SES groups reporting to have the highest prevalence of heart disease, diabetes, obesity, and major depression linked to group-based inequalities in access to the social, political, and economic resources that are necessary for good health (Fisher & Baum, 2010). Further, Allen, Balfour, Bell, and Marmot (2014) connect social inequality to mental disorder, suggesting that those who experience inequality — especially the poor and disadvantaged — suffer from mental illness at a disproportionate rate in
comparison to those in the middle and upper classes (Allen et al., 2014). These studies further problematize the binary understandings of mental disorders, suggesting that perhaps schizophrenia can be viewed as the product of both biological and social forces.

Co-Production as a Means of Understanding Schizophrenia

These binary explanations of schizophrenia, however, fail to consider how these structures interact with each other to produce a collective understanding of schizophrenia. Although intersectionality is useful for deconstructing the interrelation of oppressive structures to define diagnosis, it does not directly address how these structures’ interrelation produces a biased schizophrenia with institutions of knowledge-making. Here, I argue that schizophrenia is not exclusively the product of race, class, and gender, but is instead produced by the intersections of race, class, and gender and psychiatric knowledge-making. This is guided by Sheila Jasanoff’s (2004) analysis of co-production, which I use as a framework for understanding the relationship between science and culture in producing knowledge.

Jasanoff suggests that the ways in which we know the world are inseparable from the ways in which we live in the world, arguing that co-production describes the “simultaneous production through which modern societies form their epistemic and normative understandings of the world” (Jasanoff, 2004, p. 2). In other words, co-production acknowledges how science and society interact with each other, reconceptualizing them as linked in knowledge production. In the common imagination, science and technology are viewed in their own distant vacuum,
producing knowledge irrespective of society. Jasanoff argues against this by suggesting that developments in science and technology reflect “the imaginative faculties, cultural preferences and economic or political resources of their makers and users” (Jasanoff, 2004, p. 16). In other words, developments in science and technology actively reflect and redirect the needs of particular societies, resulting in an intertwined relationship between the two.

Such understandings acknowledge the complicated relationship between knowledge and society, as “[scientific knowledge] both embeds and is embedded in social practices, identities, norms, conventions, discourses, instruments and institutions” (Jasanoff, 2004, p. 3). As a result, scientific knowledge is reconstructed as a reflection of the social rather than its own natural truth. Such reframing challenges established binaries between a so-called objective scientific knowledge and a so-called subjective culture, prompting a deeper examination of the interplay between science and culture in knowledge-making.

The co-production of knowledge permeates deeper than the binaries between scientific knowledge-making and society, as evident in the State’s methods of controlling the body. Examining how the State’s methods of power and knowledge-making control and modify life, Michael Foucault (1976) theorized biopower, which focuses on the “relationships through which the life and health of bodies and populations become the objects of scientific discourse and institutional regulation by governments and corporations” (Hatch, 2016, p. 33). Hatch argues that there are three central themes in biopower. First, biopower emphasizes the methods in which the life and health of populations become objects of power. These
methods are used by institutions of power to organize exploitative economic and politics relationships that “are deployed through racial categorization,” the second and third themes in biopower (Hatch, 2016, p. 33).

Transforming life and health into objects of power and knowledge is accomplished through two technologies of biopower: disciplinary power and regulatory power. Scientific truths — produced by observing, judging, and examining bodies — are tools of disciplinary power used to extract political and economic productivity from individual bodies. The body, therefore, becomes “both a ritual of power and a procedure for the establishment of truth” (May, 1993, p. 43). While disciplinary power targets the individual body, regulatory power identifies populations as “a political problem, as a biological problem, and as power’s problem” (Foucault, 1976, p. 243). Regulatory power, thus, relies on large-scale methods of population data extraction, like epidemiology, to maximize health and life. Disciplinary power and regulatory power, thus, work in tandem to “conform bodies and populations to unequal political and economic arrangements” (Hatch, 2016, p. 34). As we will see later in this chapter, epidemiological data is used to establish truths about race, thus confirming Foucault’s theory of race and racism. As Hatch states, “For Foucault, race serves as a transfer point between the production of biological knowledge about population health and the exercise of political power; race becomes a means of ‘transcribing a political discourse into biological terms’ (Hatch, 2016, p. 35). Consequently, biopower justifies racism in the name of improving population health, confirming the relationship between power and knowledge.
Similarly, Orr (2016) suggests that power and knowledge are further used to regulate the psychological life of individuals and populations through what she calls psychopower. She states, “Psychopower operates through psychological monitoring, measurement, and discipline, administering order in the unruly psychic realms of perception, emotion, and memory” (Orr, 2006, p. 11). Consequently, psychological expertise that emerges from psychopower is constantly adjusted by the subjective forces that constructed them in the first place, and co-production allows for an analysis of the interplay between power and knowledge-making. Psychopower is inextricably tied to the co-production of knowledge between oppressive institutions, and as Jasanoff states,

Co-production offers new ways of thinking about power, highlighting the often invisible role of knowledges, expertise, technical practices, and material objects in shaping, sustaining, subverting, or transforming relations of authority (Jasanoff, 2004, p. 4).

This reframing interrogates not only who is producing knowledge, but also what the implications of these particular productions of knowledge are for upholding the social order in which those in positions of power dictate knowledge production. As a result, co-production prompts an examination of power inequalities and investments in any practices proclaimed to be science.

Thus, co-production and intersectionality in tandem examine the interplay between science and power in knowledge-making. Jasanoff argues that science does not operate exclusive to society, politics, or culture, but rather, the production
of science also adjusts society. Research grants, for example, reveal how politics and science interact as research grants that are ultimately given to preferred lines of inquiry. According to Pollock et al. (2017), while rare diseases that mainly affect white people are continuously funded by national grants funds, common diseases like sickle-cell that disproportionately impact African American communities are often underfunded by national health grants (Pollock et al., 2017). Jasanoff deconstructs the interplay between politics and science by stating,

How objectivity is understood and institutionally embedded in a given political system has enormous implications for the sponsorship of science by the state; it influences the kinds of work that are deemed appropriate for public funding…as well as the organization of scientific research…In turn, concepts of objectivity and reliability affect the uptake of science and technology by state institutions: how the results of research are construed in public domains…(Jasanoff, 2004, p. 34)

Thus, institutions of power dictate the types of scientific knowledge that can be produced, particularly through grant funding. As Pollock et. al suggest, because white people have always assumed positions of power, grant funding is often given to studies that address so-called “white issues.” Consequently, the available biomedical knowledge concerns questions that are relevant to white scientists and white populations, leaving so-called “Black issues” underfunded and ignored. For science to reflect the needs of already marginalized bodies, scientists must find
alternative funding sources to produce the needed scientific knowledge for their communities.

As mentioned earlier, the interplay between science and politics\textsuperscript{13}, however, is not exclusive to biology. In psychiatric knowledge-making itself, tensions exist between those producing the science and the external factors that influence science-making. Thus, I want to argue that the tools that we use to both diagnose and understand schizophrenia are influenced by the institutional context of the people who create them. This influence, however, is not exclusive to the producers of knowledge; instead, co-production and intersectionality allows us to consider how nature, society, and culture influences scientific language; as a result, co-production and intersectionality are necessary to interrogate the tools used to describe, diagnose, and treat schizophrenia. This framework shows that these entities are not simply the product of scientific innovation. Instead, they are product of the natures and cultures that influence them (Jasanoff, 2004).

In the case of psychiatry, and specifically, schizophrenia, the majority of knowledge is produced by what feminist science studies scholar and biologist Deboleena Roy calls “the Scientific Method,” a 5-step module that serves as the medium to create an accessible format for the presentation of science.\textsuperscript{14} This process of psychiatric knowledge-making is the foundation of defining and diagnosing schizophrenia. Although regarded as the standard method of science-making, the Scientific Method is flawed like other forms of knowledge-making. As

\textsuperscript{13} And, of course, other intersectionalities.
\textsuperscript{14} Capital-S, Capital-M
Roy notes, the Scientific Method does not consider the implications of co-production in its model, requiring a reconceptualization of the Method that embeds a recognition of the external influences in science-making.
Co-Production in Research

Scholars in science and technology studies (STS) have called for a reframing of the Scientific Method, suggesting the need for an embedded acknowledgement of how bias influences science-making. Because the traditional Scientific Method fails to do so, an STS-derived Scientific Method would allow for an embedded discussion of how race, class, and gender affect science-making, allowing us to reconsider the way power and science and intricately related in research. Feminist studies has grappled with the limitations of contemporary knowledge-making, (Haraway, 1988; Spanier, 1995) with Roy (2004) conceptualizing a model based on this scholarship that attempts to reconcile these limitations. Transforming traditional models of knowledge-making to a radical, feminist model, Roy states, “In order for feminists to thrive in science and develop careers in science…they need to receive a science education that is not based on the use of sexist, classist, and racist paradigms...” and I would further add that this is not only true for feminists in science, but for both future and current scientists who are invested in creating a psychiatry that is acutely aware of its co-production with race, class, and gender (Roy, 2004, p. 256).

A reframing of psychiatric discourse necessitates not only a fundamental restructuring of psychiatric knowledge-making, but a restructuring of how it is presented. This can first be accomplished by distancing science writing from the ever simplistic, but equally powerful, Scientific Method which intentionally removes any possibilities for criticism or acknowledgement of subjectivity in
science-making. Roy’s model of feminist STS inquiry challenges certain ideological assumptions that uphold Scientific Method, including the assumption that biology, the environment, and culture are separable entities, thus allowing a scientist to claim that they can “approach a research question with ‘pure’ objectivity” (Roy, 2004, p. 270). The same can be said about schizophrenia research, as the illusion of objective measures often fails to acknowledge the external influences of psychiatric research. Completely removing the very idea of objectivity from the sciences, however, would be an irresponsible dismissal of scientific knowledge. In this context, feminist studies are not interested in invalidating scientific knowledge; instead, feminist scholars like Harding (1991) call for strong objectivity, which extends scientific research to include “a systemic examination of powerful background beliefs” (Harding, 1991, p. 149).

It is essential to understand that by addressing the inherent ideological forces that shape science making, feminist science further approximates science-making to objectivity because of its embedded reaffirmation of the external influences that contribute to bias in research. By building this strong foundation, feminist science allows for a consistent acknowledgement of how bias permeates into scientific practice, and this standard allows for a more inclusive science that recognizes how race, class, and gender often are intertwined with scientific practice.

To standardize feminist science practice, Roy adapts Spanier’s (1995) model of a feminist science, which closely follows Harding’s (1987) feminist inquiry. The components are as such: (i) Locating the Origins of Problematics, (ii)
uncovering the *Purposes of Inquiry*, (iii) interpreting the *Hypothesis and Evidence*; and (iv) establishing a *Relationship between the Inquirer and her/his Subject of Inquiry* (Spanier, 1995, p. 41). This model imagines what an intersectional scientific objectivity can be by acknowledging bias beginning with the Origin of Problematics, which considers how research is often influenced by external factors. The ultimate goal of the Origin of Problematics is to recognize that it is rare — if not impossible — to conduct research that is devoid of bias. As a result, the Origin of Problematics forces a scientist “to think not only about her hypothesis but about what factors have influenced her to arrive at this particular hypothesis” (Roy, 2004, p. 264).

Following the Origin of Problematics, the Purpose[s] of Inquiry formally makes the distinction between “purpose” and “hypothesis,” constructs that have become synonymous in normative practices of science. As Roy notes, the deliberate conflation of “purpose” with “hypothesis” removes the scientist’s responsibility to note the social forces that prompts their research. Like the Origins of Problematics, the Purpose[s] of Inquiry allows for a deeper examination of external factors that could potentially introduce further bias to the motivations of science-writing. As Roy states,

> Identifying bias in language, concepts, paradigms, applications, and personnel policies of science acknowledges the harm done to science and to society by scientific studies based on questionable assumptions about the meaning of gender, race, and sexual orientation (Roy, 2004, p. 264).
The Purposes of Inquiry, therefore, deliberately makes the scientist consider not only the impact of their research but also both the positive and negative contributions their science-making will make to both the scientific community and other communities at large. The third component of feminist inquiry model involves the Hypothesis and Evidence, which further challenges the ideological assumptions that the Scientific Method fails to explore.

The final step of the model of feminist inquiry is arguably the most critical for psychiatric knowledge-making. Adding further context to the materials and methods, the Relationship Between the Inquirer and Her/His Subject of Inquiry goes beyond the Scientific Method by examining the relationship between the subject and the examinee. Roy notes that the Scientific Method’s distancing from the subject is “one of the greatest limitations of the Scientific Method” (Roy, 2004, p. 273). To reconcile this limitation, Roy places herself in a mutual footing with the material used in her study. Similarly, Spanier argues that by placing the scientist on the same “critical plane” as the materials, it “helps make visible the researcher’s actual relationship to the information and interpretations of the research and promotes a self-reflexive search for researcher bias” (Spanier, 1995, p. 43)

In Chapter 2, I use this framework to discuss and analyze my research in the Schizophrenia Cognition Laboratory, where we explore the efficacy of two types of cognitive remediation therapy (CR) in the treatment of schizophrenia. This framework will be used to not only present the findings in our pilot study, but it will also be used to discuss the limitations of this research in an effort to suggest...
future models of research that incorporate the implications of co-production into its future scientific practice.

**Co-Production in the Classification of Disorder**

Both Roy and Jasanoff argue that neither scientific knowledge-making nor the institutions and practices that are influenced by it exist in a vacuum. Therefore, the diagnostic tools that we use to define and diagnose schizophrenia are created, and thus, influenced by the Scientific Method. This is best evident in the evolution of the *Diagnostic and Statistical Manual of Mental Disorders (DSM)*, which has been influenced by medical and popular attitudes about mentally ill people (Metzl, 2009). Metzl argues that both the accounts and diagnosis of schizophrenia have suggested that race shapes not only diagnostic categories but also the medical and popular attitudes towards the mentally ill. Metzl suggests that “race impacts medical communication because racial tensions are structured into clinical interactions long before doctors or patients enter examination rooms (Metzl, 2009, p. xi). I argue further that race is not the only factor that shapes diagnostic categories, but the interactions between race, class, and gender shape the perception of schizophrenia, which can be further investigated using co-production and intersectionality through an analysis of the *DSM*.

The co-production of the *DSM*, now in its fifth iteration, is best seen through its history that serves lens into the societal understanding of disorder within a given period. Therefore, tracking the *DSM*’s revisions exposes both the influence and evolution of institutional bias in psychiatric practice. The first attempt to create a formal, standardized nomenclature of psychiatric disorder in the US began in
1918. By the late 1800s, the leading viewpoint of psychiatric disorder was the somatic theory developed by German physicians Kraepelin and Alzheimer who argued that mental illness may have a biological basis; thus, physical impairment was thought to be the source of mental disorder (Surís, Holliday, & North, 2016). Early psychiatry adopted this framework and began to construct what became a somatic framework of disorder.15

The push towards an American standardized diagnostic criterion and adoption of a biological basis of disorder was prompted due to the marginalization of American psychiatry from the rest of medicine. American psychiatry, which had been following models of psychoanalysis at the time, ignored the somatic theory until the twentieth century (Surís et al., 2016). According to Surís et al. (2016) American psychiatry “[lacked a] unified classification system and [lacked] progress toward embracing a biological appreciation of psychiatric illness” (Surís et al., 2016, p. 2). Resulting in an attack on psychoanalysis due to both the discovery of psychiatric medications and the advent of the biological research of medical disorders, American psychiatry began to adopt the somatic theory. This led to the publication of the Statistical Manual for the Use of Institutions for the Insane, the predecessor to the DSM. The volume included 22 diagnostic categories that linked abnormal behavior to organic brain dysfunctions. The nine subsequent editions of the Statistical Manual continued to emphasize the somatic viewpoint but was

15 As we will see, the popular explanation of the basis of mental disorder is ever-changing. Kraepelin abandoned the clinicopathological approach as studies began suggesting that there was no correlation between disorder and neuroanatomy.
largely ignored by American psychiatrists (Kawa & Giordano, 2012; Surís et al., 2016).

However, with time, the somatic view, which viewed physical impairment as the source of mental disorder, began to lose popularity to the psychodynamic approach, which viewed the human psyche as a product of its unconscious. By 1946, the psychodynamic theory gained acceptance and replaced the somatic viewpoint and became one of the leading schools of thought by the American Board of Psychiatry. The *Statistical Manual* was updated to reflect this shift to the psychodynamic approach, leading to the publication of the *DSM-I* in 1952. It contained 102 broadly construed diagnostic categories divided into two main classes: (1) conditions caused by brain dysfunction and (2) conditions caused by socio-environmental stressors “on individuals’ biological constitution and patients’ inability to adapt to such pressures” (Kawa & Giordano, 2012, p. 3). The latter category was subdivided into disorders of psychosis, psychoneurosis, and personality, with schizophrenia framed as a “reaction” to stressors (Surís et al., 2016).

Although the *DSM-I* set the stage for standardized categories of mental disorders, it had little bearing on psychiatric practice because it was incompatible with international diagnostic systems and was subsequently updated to the *DSM-II* to address incompatibilities between the *DSM* and the World Health Organization’s International Classification of Diseases (ICD) (Surís et al., 2016). The *DSM-II* continued to rely on the psychodynamic theory — which was quickly becoming outdated — but had removed the term “reaction.” Despite this update,
the *DSM-II* was unsuccessful due to its imprecise language. The *DSM* was updated to its third edition in 1980 and replaced the psychodynamic formulations of the previous two iterations with criteria that were atheoretical. Unlike the *DSM-II*, *DSM-III* was successful in its compatibility with the ICD, so much so that the ICD was modified to be consistent with the *DSM-III* (Surís et al., 2016). Marking the radical redirection of psychiatry towards medicalization, the *DSM-III* increased the number of mental disorder categories — from 182 to 265 — supposedly reflecting the increase and specificity in psychiatric language. The *DSM-III* saw great success and was considered to be a “formal common language that facilitated communication between multiple mental health professionals” (Kawa & Giordano, 2012, p. 5). The success of the *DSM-III* and its empirically-based definition of disorder added legitimacy to psychiatry as a medical specialty (Surís et al., 2016).

However, the broadening of mental disorders with the *DSM-III*, and its updates with the *DSM-IV* and *DSM-5*, have significant implications for diagnosis. Although the broader aims of classification are positive — to facilitate communication between researchers and clinicians and to provide a reference system that can be used for diagnosis, prognosis, and treatment — the *DSM* has caused unforeseen implications that have led to inappropriate medical treatment of vulnerable populations (Lemperière, 1995). According to Kriegler and Bester (2014), the *DSM* has “powerful influence on psychiatry praxis, other mental health professionals and people diagnosed with mental disorders” on the global stage despite it being published for American psychiatrists (Kriegler & Bester, 2014, p. 393). Arguing that classifications in psychiatry became reductive academic
exercises, Lempérière (1995) argues that these labels can be potentially dehumanizing and can be used as a tool for social and political violence (Lempérière, 1995). Such dehumanization is seen through the revisions of the DSM to follow the medical model and keep up with science. Consequently, the focus on a somatically oriented description of disease has led to the rise of biopsychiatry and pharmaceuticalization (Kawa & Giordano, 2012). As a result, both severe and mild conditions are now treated with pharmacologic treatment instead of psychotherapeutic and behavioral approaches.

The continued biologization of mental illness has resulted in a rapid increase in psychotropic drug use since 1990; in fact, since 1988 two new classes of psychotropic drugs have been introduced, including nine new antidepressants and five new antipsychotic drugs (Frank, Conti, & Goldman, 2005). According to Friedman (2012), second-generation antipsychotic drugs have evolved from being prescribed to only a few serious psychiatric disorders to mild mood disorders, anxiety, and insomnia (Friedman, 2012). By 1997, 77 percent of mental health treatment cases were treated with psychotropic drugs, a trend accompanied by unprecedented rises in spending on prescription drugs (Frank et al., 2005). This is further exacerbated by the diagnostic inflation and medicalization of normality of the DSM-5 (Carroll, 2013).

Following Jackie Orr (2006), I argue that such implications of the DSM are not coincidental, but rather, are the product of the interrelation between medicine,

16 Of course, there are many global and local forces that shaped the emergence of biopsychiatry. Orr (2006) examines the use of technology in constructing panic.
politics, and corporate power. As such, co-production and intersectionality offer a nuanced account of the implications of the increased presence of pharmaceutical companies, seen through the evolution of contemporary psychiatric discourse that has changed to reflect such need for pharmaceuticals. As Pickersgill (2012) states, “We have now learnt to understand ourselves as individuals whose ‘desires, moods and discontents’ are mapped upon the brain” (Pickersgill, 2012, p. 337). The biomedical model easily characterizes disorder as chemical deficiencies, allowing for pharmaceuticals to take control of psychiatric therapies. This is not a coincidence, as the pharmaceutical industry and the DSM-IV are interrelated, with 56% of the DSM panel members having one or more financial associations with companies in the pharmaceutical industry (Cosgrove, Krimsky, Vijayaraghavan, & Schneider, 2006). Therefore, it is critical to consider the many institutions that influence this particular form of psychiatric knowledge-making.

Further showing the importance of interrogating the multiple intuitions implicated in psychiatric knowledge, the DSM and its evolution specifically highlights the volatility of the parameters used to define schizophrenia. According to Maj (1998), the DSM-IV has major limitations in its characterization of schizophrenia (Maj, 1998). First, Maj criticizes the very definition of schizophrenia itself, arguing that schizophrenia is oversimplified to broad categories of symptoms that do not highlight the interaction between these symptoms. Maj points out how mania, for example, contains both disorganized speech and grossly disorganized behavior, and delirium contains both disorganized speech and hallucinations (Maj, 1998). This lack of clustering of symptoms results in a mischaracterization of
symptoms that can lead to misdiagnosis. Second, the *DSM-IV* gives equal weight to the major classes of symptoms that are required for a diagnosis, which can be of issue because the boundary between mental illness and social deviance can blur. As Maj suggests, if a person were to present as markedly disheveled or dressing unusually — categorized as “disorganized behavior” in the *DSM-IV* — and poverty of speech, they would have the necessary qualifications to be diagnosed as a person with schizophrenia.

Finally, Maj critiques the baseline symptoms required for a diagnosis, stating, “[The change is base rates] is even more sensitive for disorganized speech and disorganized behavior, which are likely to be more common in the general population” (Maj, 1998, p. 458). This low barrier to diagnosis leaves the ultimate responsibility to the subjectivity of the clinician. While textbooks routinely suggest that schizophrenia occurs in one percent of any given population equally across races, doctors diagnose African Americans, particularly African American men, more often with schizophrenia (Barnes, 2004, 2008, 2013; Bresnahan et al., 2007). This, as Allen Frances (2013) writes, is not due to differences between African American and other patient populations, but because of cultural biases in the diagnostic process itself (Frances, 2013).

Burns, Tomita, and Kapadia (2014) offer an explanation for this bias by suggesting that although the *DSM* provides criteria that define mental disorders, the *labeling* done by the psychiatrist is a subjective process (Burns, Tomita, & Kapadia, 2014). Regardless of any assessment about the objective validity of DSM categories, a diagnosis is ultimately dependent on the final judgement of the
psychiatrist. These judgements are influenced by the racial and ethnic identities of clinicians. In 2013, 83.6% of the psychology workforce was white, less diverse than both the general US workforce and other medical fields (Lin, Nigrinis, Christidis, & Stamm, 2015). According to Burns, Tomita, and Kapadia, the disproportionate amount of white mental health personnel may “lead to less sensitivity to cultural differences about the behavior of minority group members” (Burns et al., 2014, p. 10). As a result, behavior deemed appropriate within a minority group may be read as a disorder by white mental health personnel, reported in the epidemiological accounts of schizophrenia.

**Co-Production in Epidemiology**

Without an intersectional analysis of the co-production of race, class, and gender with the epidemiology of schizophrenia, we are faced with an incomplete reality of how schizophrenia is deeply embedded within institutional racism, classism, and sexism. Although I am going to present studies that attempt to enumerate these biases, they are incomplete beyond measure. Scientists tend to conduct epidemiological research in binary paradigms, choosing either race, class, or gender as the focus of their study, with no study that has attempted to interrogate all three in tandem. In this section, I aim to use these epidemiological data to not only deconstruct the embeddedness of bias within the medical institution, but also to implore that future epidemiological studies use intersectionality to complicate the binaries of these data. In this moment, intersectionality can help fill the gaps where epidemiology fails, but it can only do so much. Although I cannot fully explain the intersections of race, class, and gender in schizophrenia diagnoses, the
frameworks I have established previously can complicate and add nuance to these studies. First, gender is the least intersectional area of study in the epidemiology of schizophrenia. Studies have simply suggested that men are more likely to develop schizophrenia than women. (McGrath, Saha, Chant, & Welham, 2008). Further, men develop schizophrenia earlier in life, suggested to result in lower levels of social development and socially adverse behavior (Häfner, 2003). Fortunately, studies that examine the relationship with socioeconomic status (SES) and schizophrenia are more substantial, identifying low SES as a risk factor for schizophrenia. This is because the social inequalities associated with lower socioeconomic status, including unemployment and housing, can predict schizophrenia, suggesting that environmental factors associated with poverty can trigger or exacerbate the symptoms of schizophrenia (Gallagher & Bernard, 2018; Harrison, Gunnell, Glazebrook, Page, & Kwiecinski, 2001). As Murali and Oyebode (2004) suggest, alienation and distress of poverty can indirectly affect one’s ability to develop and maintain emotional, behavioral, and psychiatric problems (Murali & Oyebode, 2004). They suggest that the poor are doubly victimized as they are not only exposed to additional life stressors, but they also have fewer resources to manage those stressors (Murali & Oyebode, 2004, p. 217).

Intersections of race and class are also important. African Americans comprise the highest percentage of individuals in poverty, which poses them at a higher risk for developing schizophrenia due to this increased exposure to life stressors (Kaiser Family Foundation, 2018). The data surrounding race and schizophrenia diagnoses, however, speaks for itself. I mentioned earlier that
although neurobiological research suggests that schizophrenia affects one percent of any given population, African Americans are three to five times more likely to be diagnosed with schizophrenia rather than a mood disorder (Barnes, 2004, 2008, 2013; Bresnahan et al., 2007). The implications of this statistic bleeds far beyond the diagnosis itself. In treatment, African American clients are at an increased risk of incorrect drug therapy, where antipsychotic medications are likely to be administered rather than antidepressants (Barnes, 2013).

Racism has been the foundation of psychiatry as we know it; insanity was racialized far before schizophrenia, with white psuedo-scientists in the 1850s who thought that African American slaves who ran away from their masters were suffering from a mental illness. And racism did not magically end there; in fact, it continued to bleed far beyond definitions of disorder. Racism has been embedded within the fabric of psychiatric practice, best seen through reconciliatory attempts to make doctors more “culturally competent” and somehow aware of their biases when diagnosing patients. Instead, the effects of cultural competency have further exasperated the racialization of medicine as doctors often oversimplify the patient’s experience, effectively allowing doctors to not only determine a patient’s medical diagnosis, but also their racial, ethnic, and cultural diagnoses that reinforces the embedded racism of medicine (Metzl 2009; Structural Competency, n.d.). Metzl notes that race impacts medical communities because racism is structured into the clinical setting long before either the doctor or patient enters the examination room, stating,
Psychiatric diagnoses can also define, circumscribe, and contain abject populations in ways that harm people in these populations under the guise of helping them. Not coincidentally, these people are, in a Farmenian sense, already vulnerable…[for example] addicted war veterans and unruly children. Or angry black men. At the level of the structural unconscious, concerns about the actions… of such people impacts the ways in which we aggregate symptoms into diagnoses, and the ways in which certain populations come under diagnostic purview as a result (Metzl, 2009, 203).

As Pickersgill (2012) notes, “Neither psychiatric institutions nor clinical knowledge itself is completely reducible to either ‘social’ or ‘natural’ forces; rather, they are material-semiotic hybrids that are, in the idiom of science and technologies studies, co-produced” (Pickersgill, 2012, p. 340). These intersections are important because they produce tangible realities outside of the DSM and diagnosis of schizophrenia. Thus, the intersections of race, class, and the patriarchy permeate into doctor-patient relationships and further into interpersonal relationships affected by fears of stigma. In this chapter, what began as interrogation of the co-production of schizophrenia had led to a recognition of how racism, classism, and the patriarchy permeates into our assumptions of objectivity and truth, leading to questions regarding the validity of the processes that produce scientific knowledge. In the next chapter, I address these questions by applying the feminist method of scientific practice to reconcile these limitations.
Chapter Two: The Scientific Method, But Make it Feminist

I: Introduction: The Case of D.

Inside the small office at River Valley Services\textsuperscript{17}, I sit across from D., a woman in her sixties. D., who was diagnosed with schizophrenia over forty years ago, is dressed in colorful layers and a knitted hat. It’s our third session together, and she doesn’t really say much. As a research associate in the Schizophrenia Cognition Laboratory at Wesleyan, I’m responsible for administering batteries of tests that assess the client’s symptoms, which will eventually be analyzed through SPSS. I take out the Positive and Negative Syndrome Scale (PANSS), a 30-item measure that assesses both the positive and negative symptoms and general psychopathology of schizophrenia. I was tasked with one thing: ask the questions. Take notes that were sufficient to make judgements about the severity of her symptoms later to be enumerated into variables and analyzed later on.

I turn to D. and recite the well-crafted script that I’ve been trained to perform, “Next, I’m going to ask some personal questions about your illness. If you ever feel uncomfortable with these questions, let me know, and we’ll move on to something else.” She nods, and we begin. “Have you been feeling worried or nervous in the past week?” She pauses and says “no.” I continue, “Then would you say that you’re usually calm and relaxed?” She pauses again, and, this time, nods. I continue. I ask. She nods. She gives a brief response. After this question-pause-nod exchange, I reach the portion of the PANSS that assesses hallucinatory

\textsuperscript{17} River Valley Services (RVS) is a public psychiatric outpatient center in Middletown, CT.
symptoms. I pause, aware that the next line of questioning is much more intimate than simply asking how she’s feeling. I reiterate that we can stop testing at any point, and ask, “Sometimes people tell me that they can hear noises or voices inside their head that others can’t hear. What about you?” She pauses and looks towards the window. She says, “No, no I don’t.” The silence is astounding and continues to grow with each subsequent question. I ask her if she has any talents — aimed to assess grandiose behavior, associated with schizophrenia — and she simply replies that she’s good at arts and crafts. She’s mentioned her crafting hobby several times over the past few sessions, so I mentally note to not consider this response as grandiose behavior. I briefly wonder if someone else would think otherwise.

After D. describes her current crafting projects, we slowly return to our original cadence. I ask. She pauses. Brief response. Next question: “Did you ever do something wrong – something you feel bad or guilty about?” D. pauses, and nods in agreement. In the PANSS, if the client responds “yes,” the interviewer is prompted to probe further into the response. I continue, “Just how much does that bother you now?” Quite a bit, evidently. She mentions her family, who abandoned her years ago. “Do you feel that you deserve punishment for that?” Pause. “Yes.” I process her response, unsure of how to respond. Her response lingers in the air, and I take a moment to let us sit with this confession. In retrospect, this moment transformed the interview from data collection to a much more intimate and human encounter, a story, a reminder that clinical research is not static and one-sided. Instead, D., the PANSS, and I are entangled in an interaction that changes all of us and, importantly, my working conception of schizophrenia.
It is moments like these where I am reminded that my role as researcher can quickly transform into something much more valuable. Working directly with clients affords me the immense honor and responsibility to take care of those who are in a vulnerable position when they describe their experience with schizophrenia. These moments cannot be — nor am I sure if they should be — quantifiable by the PANSS and tools like it, and I wonder what parts of D.’s story is lost to the PANSS and its data analysis.

Now, if this thesis were to be written with the traditional Scientific Method in mind, the above introduction would not begin like this. I would have begun with a literature review of schizophrenia, much like in Chapter 1, where I would begin with the symptomology of schizophrenia. I would describe the three main classes of symptoms and features — positive, negative, and cognitive — and describe the epidemiology of schizophrenia. I would distance myself from D. and look at her symptoms through the objectivity of the DSM. I would ignore the conversations we had in between the different tests I had administered, the small glimpses I had into her life. I knew that D. lived alone, but still managed to make it to therapy. I could see how the intervention and consistent therapy was helping her. But I could only report on her answers on the questionnaires. If she didn’t mention her life improvement at the particular time the questionnaire solicited it, it was to be ignored. Forgotten. Irrelevant to the dataset. Some testing days were successful, while others were not — the dataset would never know, nor would it have cared to in the first place. I would view myself as the objective scientist and go about my day without considering how my own biases played a role in how I conducted my
research. I had to, because the Scientific Method would render me captive within a system that doesn’t consider the humanity of psychiatric knowledge-making, but instead essentializes each experience I had with D. to its bare bones, extracting any semblance of objective “fact.” It would have provided, as Roy suggests, a so-called objective account that enumerates and specifies the biological and societal influences of schizophrenia (Roy 2004).

In this chapter, I ask how a follower of the Scientific Method would approach D’s case. I position this thought experiment alongside a science and technology studies (STS) analysis of the PANSS that aims to show how important parts of D.’s story can potentially be forgotten if we continue to create psychiatric knowledge with the medical model of objectivity. In truth, I am not concerned with the remainder of the Scientific Method, which would require me to analyze the data with the entirety of the testing battery; however, I will still conduct an analysis of data in order to consider alternative ways to discuss clinical research as suggested by the feminist model of inquiry. Because the central aim of my thesis is to deconstruct the means in which we produce psychiatric knowledge and the meanings that come from it, from this point on, I will diverge from the typical layout of the Scientific Method to focus on the history and use of the PANSS and how we have unquestioningly used the PANSS to define and redefine schizophrenia.

Thus, I aim to provide an interrogation of the PANSS to highlight how the PANSS is not only a subject of co-production, but it is also itself a tool of co-production as it defines and measures schizophrenia. I argue that although the
PANSS was created as the first standardized tool to evaluate symptoms of schizophrenia, there are several factors that question both its standardization and reliability in psychiatric knowledge-making. First, the PANSS’ foundation in the neurobiological model results in its inability to account for the personal experience of schizophrenia, resulting in only a partial understanding of the symptoms due to the scale’s limited ability to interpret the experience of schizophrenia. Second, the PANSS lacks reliability and reproducibility due to its multiple models, inaccurate scaling method, and subjective rating scales. These factors result in a PANSS that is too weak to establish objectivity in psychiatric knowledge-making, holding severe implications for clinical research in schizophrenia. Thus, this interrogation of the PANSS will allow for a reimagination of what an analysis of clinical research data can look like, guided by the feminist model of inquiry discussed in Chapter 1.
II: A Pilot Study Comparing Two Therapeutic Models of Cognitive Remediation in Psychosis-Spectrum Disorders

Preface: This section of the thesis will provide a “backstage” approach to the Scientific Method. We left off at the Introduction, and following the Scientific Method, we would continue with describing the study.

Cognitive Remediation (CR)

Like a good scientist, I outline the current therapeutic models that treat the symptoms of schizophrenia, focusing on cognitive remediation (CR), a model explored in the Schizophrenia Cognition Laboratory. In the traditional Scientific Method, this part of the text gives background information on the intervention. To do so, I first define CR and say that it is a therapeutic model designed to improve the cognitive features of schizophrenia. Then, I describe the methods of CR, saying that it is a behavioral intervention that is designed to improve cognitive abilities in those who have experienced a decline in neuropsychological functioning.

I then describe the aims of CR and suggest that the aim of CR is to reduce the difficulty of everyday tasks that would otherwise be difficult with neurocognitive and social cognitive impairment. Because our study adds to the current literature, I cite the meta-analyses that evaluate the effects of CR in individuals with schizophrenia. I would suggest that these analyses have found that CR leads to significant improvements in cognitive performance, clinical symptoms, and functional outcomes (McGurk, Twamley, Sitzer, McHugo, & Mueser, 2007; Wykes, Huddy, Cellard, McGurk, & Czobor, 2011). Then, I would cite additional studies to justify my research methodology and focus. For example, Kurtz (2016) reported that CR has positive effects when administered in low-intensity interventions over a short period of time (Kurtz, 2016). The study suggests an
intervention model of two to three times per week for a period of three months, a protocol that the Schizophrenia Cognition Laboratory follows in the present pilot study.

Present Study

The discussion of CR serves as a segue into our present study. At this point in the traditional thesis, the reader would have a comprehensive background of both schizophrenia and its treatment. As a result, this section would focus on the specific aims of this study. I begin by introducing the Schizophrenia Cognition Laboratory, where I conduct my research study. I specify that the laboratory operates in three locations, with this thesis focusing on the former two. First, at River Valley Services, where the clients of our study receive treatment and are assessed using the testing battery. Second, on the third floor of Judd Hall at Wesleyan University, where the analysis is completed. Third, at the Institute of Living (IOL), where we have partnered with the Hartford Hospital to expand our study.

From there, I go on to describe the nature of our research by stating that the present study builds on the previous literature that assess the efficacy of CR for schizophrenia. I state that the Schizophrenia Cognition Laboratory aims to directly compare the efficacy of two therapeutic models of Cognitive Remediation (CR) at River Valley Services (RVS), a public outpatient mental health center in Middletown, CT: first, a computerized drill-and-practice approach (PSSCogRehab) and second, a manualized practice strategy-based compensatory cognitive training program (CCT). I will state the aim of the study is to directly compare the
effectiveness of these two models of CR in treating and improving cognitive features and functional outcome.

**Hypothesis**

This section of the Scientific Method is seemingly simple, as the only task at hand is to state the hypothesis of the study. In this case, I state that we hypothesize that the participants in the CCT condition would show larger improvements in functional outcome when compared to the PSSCogRehab condition because the targeted strategies of CCT better represent everyday tasks that the participants will encounter in their daily lives.

It is within the simplicity of the hypothesis that we can see the distancing between the scientist and the study, a central tenant of the Scientific Method. As mentioned earlier in Chapter 1, Roy makes explicit that a feminist Scientific Method would consider the “dynamics of culture that shapes both biology and behavior” that further influence science-making (Roy, 2004, p. 272). The only part of the above hypothesis that claims any personal responsibility is the use of “we” in the statement of the hypothesis. Otherwise, the hypothesis lacks any acknowledgement of the external influences in the project. To follow the model of feminist inquiry, therefore, I am responsible for explicitly noting the underlying cultural dynamics influencing our research. An important dynamic in this study involves our participant pool. As mentioned earlier, the pilot study recruits participants from River Valley Studies, a public outpatient center. The participants we recruit to the study are often low-income, an important note because these participants often rely on clinical therapy trials to receive not only treatment, but
financial aid, an important dynamic to consider when considering the results of the study.

**Methods**

**Participants**

Participants were clinically stable adults recruited to the study by staff at River Valley Services. All participants received a *DSM-5* diagnosis of either schizophrenia, schizoaffective disorder, or a mood disorder with psychotic features. Participants were excluded if they had a neurological disorder, history of intellectual disability, were actively using illicit substances, or were enrolled in another cognitive training program. Participants were admitted to the program after providing written consent and a Release of Health information form.

<table>
<thead>
<tr>
<th>Demographic</th>
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<tbody>
<tr>
<td>Age</td>
<td>54 ± 11.4</td>
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<tr>
<td>Gender, % Male</td>
<td>64%</td>
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<tr>
<td>Race/Ethnicity, % Caucasian</td>
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<td>Housing, % Living Independently</td>
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<tr>
<td>Education (years)</td>
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<tr>
<td>Father's Education</td>
<td>11.1 (± 4.99)</td>
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<tr>
<td>Mother's Education</td>
<td>12.36 (± 3.11)</td>
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<table>
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<tr>
<td>Schizophrenia</td>
<td>42.90%</td>
</tr>
<tr>
<td>Schizoaffective</td>
<td>42.90%</td>
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<tr>
<td>MDD with psychotic features</td>
<td>14.30%</td>
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<td>Onset of Illness (year)</td>
<td>24.42 (± 9.69)</td>
</tr>
<tr>
<td>Number of Hospitalizations</td>
<td>9.08 (± 13.4)</td>
</tr>
<tr>
<td>Duration of Illness (years)</td>
<td>29.5 (± 13.5)</td>
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*Table 2.* Demographic values of participants analyzed in this study using descriptive tests. 14 total participants were analyzed in the study.

**Procedure**

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The procedure marks a transition in the Scientific Method as I now begin to outline what I call the “backstage” components of neuroscientific research. As such, this section will focus on the important processes that allow research to exist in the first place. These components specifically include approval processes, designed to ensure the protection of human subjects; consequently, it is crucial to embed a critical account of the procedure as it concerns the methods in which researchers interact with subjects.

First, I state that the study and all associated personnel, including RVS staff and research assistants, were approved by Wesleyan University and the Connecticut Department of Mental Health & Addiction Services (DMHAS) Institutional Review Board (IRB). This leads to the study design, where I state the experimental design was a single-blind randomized control trial, such that after the participants completed written, informed consent and baseline testing, subjects were randomly assigned to one of three conditions using a random number generator: PSSCogRehab, CCT, or treatment as usual (TAU). To maintain blindness, participants were given subject identification numbers. I describe the execution of the study itself, including the timeline of the study with each cohort. Both of the cognitive training interventions were administered over the course of a 12-week period. During the intervention, participants were treated with their standard doses of antipsychotic medications by their treating psychiatrist.

From here, we move onto the steps required to assess the change in symptoms and, thus, the validity of our hypothesis. In order to do so, participants were assessed by a multimodal evaluation of symptoms and functioning. This
assessment, which I will now refer to as the “testing battery,” was administered by research assistants at three times: first, at baseline; second, immediately after the intervention; lastly, three months post-intervention. Finally, I note the methods of compensation for our participants: a $30 gift cards after each evaluation.

**Experimental Conditions**

This last section simply outlines the two experimental conditions in the study. It is important to note that similar to the hypothesis, this section is rather short and fails to acknowledge the historical and cultural impact of these interventions. Because of this, the reader is left unaware of not only the previous studies that have used these interventions, but also how the interventions were developed in the first place.

**PSSCogRehab**

Psychological Software Services Cognitive Rehabilitation Therapy System (PSSCogRehab) is an online computerized cognitive training program that uses a repetitive drill-and-practice model to restore impaired cognitive areas (Bracy, 1995). The program aims to teach compensatory strategies to cope with and overcome cognitive difficulties. Using a neuropsychological approach, PSSCogRehab uses 84 exercises to improve attention, verbal and non-verbal memory, and language processing. Participants attended three sessions per week that was supervised with a trained clinician. PSSCogRehab is built on a hierarchical principle in which therapy begins at an elementary level of neurocognitive training and advances to more difficult tasks at the client’s pace.

**CCT**
Compensatory Cognitive Training (CCT) is a 12-week long manualized, group-based, computer-free treatment intervention. CCT focuses on teaching and practicing of compensatory strategies to help clients with their cognitive difficulties (Twamley, Savla, Zurhellen, Heaton, & Jeste, 2008). CCT ultimately aims to habituate these compensatory strategies so the client can become more independent and less reliant on others for everyday tasks. To do so, CCT targets four cognitive domains that are often impaired in patients with psychosis: (1) prospective memory, (2) sustained attention, (3) learning and memory, (4) executive functioning. CCT is designed to be generalizable and engaging for participants, including both paper-and-pencil practice activities and games. To address the four mentioned cognitive deficits, CCT uses internal and external supports to develop habits that allow the client to work around their cognitive impairment.

Control Condition, Treatment as Usual (TAU)

Participants assigned to TAU were not given a CR intervention but, instead, were given the opportunity to participate in either of the CR interventions after completing the study.

Study Measures

This section is used to list and briefly describe the different components of the testing battery. To follow the Scientific Method in its entirety, I begin this section by describing the cover sheet. The information for the cover sheet is collected for the following demographic data: date of birth, ethnicity, age, years of education of both the participant and their parents, employment status, duration of time at current job, maximum years of employment, age of schizophrenia diagnosis, number of hospitalizations, duration of illness, current support services, last
medication appointment, living status, other medical illness, history of loss of consciousness, and a list of current medications and daily dosage. Crucial to the study, this data allows the researcher to correlate data according to different demographic factors in the participant pool. From here, I list the different clinical assessments used in the testing battery, which can be found in Table 1 (Chapter 1). We divide the testing battery into five different categories, including: (1) clinical assessments, (2) self-efficacy measures, (3) social cognitive measures, (4) functional outcome measures, and (5) neurocognitive measures.

From here, I would complete the presentation of the procedure and present the data, results, and discussion; however, at this point I will diverge from the Scientific Method to interrogate the PANSS in order to continue following the feminist model of inquiry. As such, I will continue presenting the remainder of the study following this section.

See Tables 2 and 3 for demographic information.
III: The Positive and Negative Syndrome Scale for Schizophrenia

Thus far, I have explored the concerns regarding the descriptive tools used in psychiatric knowledge-making. In the case of the Positive and Negative Syndrome Scale, there are important considerations to be raised, including both external concerns raised by STS scholars and internal concerns about the methodological process used to standardize the PANSS. The PANSS is a 30-item rating scale designed by Kay, Fiszbein, and Opler (1987) to assess symptom reduction and is divided into three subcategories: positive symptoms, negative symptoms, and general psychopathology (Kay et al., 1987). According to Yang et. al (2018), the PANSS is still one of the most widely used symptom severity rating scale for psychosis-spectrum disorders for both the management of clinical outcomes and intervention efficacy (Yang, Lim, Lam, Keefe, & Lee, 2018). The PANSS was created to address two main concerns: first, researchers had long acknowledged the heterogeneity of schizophrenia and needed a consistent set of categories to explain schizophrenia’s complexity; second, there was a need for a well-characterized, standardized measurement technique in the positive-negative research of schizophrenia. According to Kay, Fiszbein, and Opler, the PANSS was conceived as an “operationalized, drug-sensitive instrument that provides balanced representation of positive and negative symptoms and gauges their relationship to one another and to global psychopathology” (Kay et al., 1987, p. 261). Each item is administered by a trained research assistant and is rated with a scale from one (1)

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19 There are variations of subcategories of the PANSS. I will discuss them later in this section.
20 The word *standardized* is especially relevant, to which I will discuss later in this section.
to seven (7), with one indicating the absence of symptoms, and seven indicating extreme presence of symptoms.

Kay, Fiszbein, and Opler outline the different phases of the administration of the PANSS. Within the first fifteen minutes of its administration, patients are encouraged to discuss their history and experience with schizophrenia. It is recommended that the interviewer remains as an observer and allow the patient to speak as needed in order for the interviewer and patient to establish rapport. From there, the interviewer is prompted to “probe” the patient to discuss their symptoms through “prototypic leading questions that progress from unprovocative, non-specific inquiry to more direct probe of pathological themes” (Kay et al., 1987). The final phase of the PANSS aims to secure information on mood state, anxiety, and abstract reasoning ability. As such, they suggest that the PANSS allows for an observation of physical manifestations, interpersonal behavior, cognitive-verbal processes, thought content, and response to structured questioning.

Acknowledging that research is only as reliable and valid as the measures in which they are based, Kay, Fiszbein, and Opler suggest that no scales before the PANSS has gone through the “thorough process of psychometric standardization that is necessary to address fundamental, and as yet highly contested, issues of content and construct validity,” however, in order to achieve satisfactory reliability and validity, they argue that “more rigor is needed in providing strict operational criteria for eliciting, defining, and measuring symptoms” (Kay et al., 1987). The

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The introduction of this chapter shows an example of the transition from indirect to direct probing.
creation and execution of the PANSS, however, is fundamentally flawed for several reasons, one of which is embedded within the justification for the creation of the PANSS. As they suggest, the PANSS was created in response to the emerging body of psychiatric knowledge that was beginning to chart the neurobiological factors that cause schizophrenia (Crow, 1980). Although they admit that such research was contested in the field, as I discussed in Chapter 1, they fail to consider the one-dimensional understanding of schizophrenia that the PANSS offers due to its neurobiological foundation. As such, because the neurobiological research aimed to pinpoint the brain mechanisms for the positive and negative symptoms of schizophrenia, the categories of the PANSS mirrored the same subcategories. In response, there has since been criticism of the PANSS in its limited ability to accurately describe the complete symptomology of schizophrenia.

Addressing the limitations of the three-factor model of the PANSS, Lançon et al. (2000) explored possible alternatives to the original conception of the three subcategories. They, like Kay, Fiszbein, and Opler, acknowledge Crow’s contribution to the distinction between positive and negative symptoms, but they also acknowledge the limitations of Crow’s two-dimensional model of schizophrenia. They state, “The research conducted in that direction has non the less stressed the need to take into account other symptomatic dimensions in order to describe the various clinical aspects of schizophrenia” (Lançon et al., 2000, p. 232). Therefore, in order to account for these dimensions, they examined the stability of the five-factor model of the PANSS, including three categories in addition to the positive and negative categories: excitation, depression, and
cognitive. They conclude that the five-factor model is necessary to account for the various clinical aspects of schizophrenia, allowing for an analysis beyond the biological in which PANSS was originally founded upon.

Although it is well-known that the three-factor model of the PANSS is flawed, there is no one consensus of which new model of the PANSS should be used. According to Wallwork, Fortgang, Hashimoto, Weinberger, and Dickinson (2012), several factor-analytic studies have suggested that a five-factor model better captures the structure of the PANSS, but they note that other factors “constantly emerge” such as disorganized/concrete, excited, and depressed (Wallwork et al., 2012, p. 246). As a result, although the five-factor model has been widely used from research areas concerning treatment-response, functioning, and insight, proposed alternative dimensions of the PANSS range from four, six, and seven-factor. Moreover, there are multiple five-factor models, and despite the fact that these models are similar, no single model has achieved broad consensus; not only this, but the original three-factor model is still widely used. The use of the original three subscales is extremely problematic because according to their study, it fits the data poorly and does not consider the multifaceted experience of schizophrenia. The consequence of this is very clear: although the original PANSS was created as a solution to the lack of standardized techniques to evaluate symptoms of schizophrenia, the expansion of several subscales and models have since destroyed its standardization across neuropsychological research because no two studies follow the same model.
Further, there are internal methodological flaws in the PANSS that raise further concerns about its use in descriptive statistical procedures. Among these concerns, Obermeier et al. (2010) argue that the scaling model of the PANSS is also fundamentally flawed. As mentioned earlier, the lowest score that can be assigned per item is a one, and thus, the lowest score a client can receive with the thirty subsections is a thirty. This causes serious problems because conventional statistical procedures depend on a natural zero point (Obermeier et al., 2010). Although recent publications suggest a subtraction of 30 points to every PANSS before calculating percent change to adjust towards the natural zero point, the so-called traditional approach that uses uncorrected scores is still common practice. Rescaling the PANSS from 1 to 7 to 0 to 6 would “avoid such often neglected pitfalls,” but the fundamental issue of standardization remains the same (Obermeier et al., 2010, p. 455). Because this pitfall is overlooked, different calculation methods have emerged when analyzing the PANSS: some have simply subtracted 30, some have opted to use the raw score, and others provide no information regarding their calculations.

This holds two significant limitations in research. First, because there is no accepted method to compensate for the lack of a natural zero point, the PANSS’ standardization is once again compromised. This makes comparing the findings between data sets difficult, especially if two given studies differ in both subscale model and scaling. Second, different calculation methods might generate different significance levels, which can lead to misinterpretations of treatment effects for a given intervention (Obermeier et al., 2010). They conclude that the results of their
simulation study show a rate of more than 50% inconsistent test decisions due to the 1-7 scale. These results have far-reaching implications in drug approvals that rely on such studies for drug models due to either an improper statistical method or not having stated the method at all. Such oversight is incredibly problematic for one of the most used tools in neuropsychological knowledge-making. Furthermore, Cohen, Alpert, Nienow, Dinzeo, and Docherty (2008) elaborate that although the most common measurement strategy in clinical research are symptom-rating scales like the PANSS, they do have considerable limitations. They argue that the current measurement strategies are reliant on subjective symptom rating scales that can potentially lead to psychometric and practical limitations. First, Cohen et. al suggest that the utility of the PANSS is constrained because these scales require training to establish inter-rater reliability and often require multiple sources of information to make accurate ratings. They state,

> From a psychometric perspective, symptom-ratings scales are not ideal because they often employ vague rating systems (e.g., “mild”, “moderate” and “severe” categories…) that may be insensitive to subtle changes in symptom severity that occur over time… Moreover, they yield ordinal data that are less than ideal for parametric statistical analysis (Cohen et al., 2008, p. 828).

This consensus is further elaborated by Blanchard, Kring, Horan, and Gur (2011), who argue that new assessment approaches are needed to address the limitations of these instruments. They suggest that the PANSS can reflect “several, conceptually distinct processes or domains that are not necessarily part of the negative symptom
domain (Blanchard et al., 2011, p. 292). For example, items such as “emotional withdrawal,” “poor report,” and “passive/apathetic social withdrawal” are defined as terms of internal states but fail to probe into these labels as experiential states. Consequently, the negative symptom ratings in the PANSS rely solely on the interviewer’s observations and reports from care workers or family. This is a fundamental issue because these reports focus only on “observer ratings of social success and functioning” rather than the patient’s own experience of emotion, therefore distancing the PANSS from the patient’s lived experience of schizophrenia (Blanchard et al., 2011, p. 293).

This leads to arguably the most important fundamental issue with the PANSS: performance is determined by multiple factors other than the patient’s personal experience with schizophrenia. Blanchard et. al (2011) argue that these factors include the “lack of opportunity related to social and economic privation…lack of social skill, constraints on social and occupational activities imposed by housing requirements or disability payments, or social rejection related to family conflict or stigma” (Blanchard et al., 2011, p. 293). These factors may contribute to the perception that the patient may have impoverished social networks or occupational impairment, categorized as negative symptoms of schizophrenia; however, these factors are distinct from social or occupational failures that are the result of emotional or motivational deficits that are at the core of schizophrenia. Consequently, circumstances related to oppressions linked to racism, classism, and sexism not only fail to be recognized as structural forms of oppression, but are also further medicalized and categorized as symptoms of disorder.
The impact of the subjective interviewer reaches far beyond the rating of negative symptoms for a given patient. Without acknowledging the influence personal bias may have on their assessment of the patient, we cannot assign any level of objectivity, and therefore standardization, to the PANSS and other measures similar to the PANSS. Although Kay, Fiszbein, and Opler suggest that the process of assigning ratings is holistic because the rater first refers to the item’s definition to assess if such symptom is present, they fail to address their own bias in the creation of the rating system. In no place in the literature is there an examination of how they created the PANSS. Instead, the only insight regarding the process of validating the PANSS is the alarming fact that their subject pool had 70 men and 31 women, which can lead to sex-based discrepancy in the validation of the PANSS. Such drawbacks are further supported by Wallwork et al. (2012), who argue that the PANSS relies too heavily on a limited behavioral observation for some items and patient reports for others. Finally, the ordinal rating metric is also vague, and I would further argue that the decision between rating a 4 (moderate) or a 5 (moderate-severe) or between a 2 (minimal) or a 3 (mild) are arbitrary choices done at the whim of the researcher regardless of inter-rater reliability (Wallwork et al., 2012).

22 I mention this because I have administered the PANSS and have had the responsibility of rating the interview several days after its administration. How sure can I be of D.’s symptoms, for example, based on a 2-hour interaction? How can I be sure of her internal emotional state if, no matter how well I try to establish a space of non-judgement, D. is still affected by the stigma that she faces as a person with schizophrenia? Regardless of the training I have received, I must consider these factors while scoring.
I have given substantial space to discuss this because the implications of the assumed objectivity of the PANSS cannot be ignored. When tracing back to the contexts in which the PANSS was created, we see very little consideration of race, class, and gender in its creation. In fact, the only influence that was acknowledged in Kay, Fiszbein, and Opler’s article is the need for the PANSS due to Crow’s neurobiological model of schizophrenia. For the feminist scientist, this is not only alarming, but it also raises questions to the validity of the PANSS and related measures. How can we analyze data if the tool that is most commonly used is so broken? What can we reclaim from the data that would otherwise go unnoticed in the Scientific Method? In the next section, I will explore this reclamation, redefining what the Scientific Method of the data, results, discussion, and conclusion can look like under the feminist model of inquiry.
IV: Continuing the Feminist Scientific Method

Statistical Procedure

We left off right before the statistical procedure. In the traditional neuroscience thesis, multiple statistical methods would be used in order to analyze the several components of the testing battery. Because my analysis focuses exclusively on the PANSS, this section is rather short and simple. All statistical analyses were done using the IBM Statistical Package for the Social Sciences 23.0 (SPSS). A series of one-way analysis of variance (ANOVA) and chi-square tests were used to analyze between group-demographic, symptom, and functional differences before treatment. Chi-square tests were used to analyze between-group differences for demographic differences including sex, ethnicity, living status, and diagnosis (Arlia, 2019). Statistical significance was set at alpha 0.05 (p ≤ 0.05). Paired sample t-tests were conducted between three time periods to analyze durability: (1) baseline; (2) immediate follow-up; and (3) three-month follow-up. Another set of paired sample t-tests were conducted between three time periods: (1) between baseline and immediate follow-up; (2) between immediate follow-up and three-month follow-up; and (3) between baseline and three-month follow-up.
Results

Figure 1. Flow diagram of participant progression through the stages of the randomized control trial (Arlia, 2019).
Table 3. Chi-square tests were used to categorize demographic variables (Arlia, 2019).

Importantly, criticisms of the Scientific Method argue that although there are realities produced by science-making, those realities exist only in particular networks or systems of circulation. Science is often regarded as a method of describing reality, however, knowledge practices are also performative (Law, 2009). Arguing that knowledge practices are only successful if they create convincing knowledge that also fit already established knowledge, Law states, “…realities (as well as knowledge of realities) depend on practices that include or relate to a hinterland of other relevant practices – that in turn enact their own realities” (Law, 2009, p. 241). If science is performative, therefore, then truths are
not universal; as such, different realities are enacted in different places. Creating charts and figures, as McCabe and Castel (2008) suggest, further adds to a perception of credibility in the sciences (McCabe & Castel, 2008). Thus, it is essential to acknowledge that this study only presents one particular reality about schizophrenia that can transiently influence studies that offer an alternative account of schizophrenia. As such, these charts and figure do not simply describe the participants — whether that be their demographic data or the enumeration and analysis of their symptoms — they “help to enact the world that they describe” (Law, 2009, p. 249). This is why it is necessary to recognize the cultural dynamics of one’s study in the hypothesis, as it acknowledges that one particular study’s constructed reality of schizophrenia depends on the participant pool it recruits.

The Five-Factor Model for the PANSS

Thus, the following set of data describes the change in symptoms for participants analyzed according to the 5-Factor Model of the PANSS. The symptoms analyzed are as such: (1) Positive; (2) Negative; (3) Disorganized/Concrete; (4) Excited; and (5) Depressive. Each data set examines changes in symptoms across different time frames. As mentioned before, the first analysis examines changes in symptoms at individual time frames: (1) baseline; (2) immediate follow-up; and (3) 3-month follow-up. The second set of data examines the changes in symptoms at paired time frame between (1) baseline and immediate follow-up, (2) immediate follow-up and three-month follow-up, and (3) baseline and three-month follow-up.
Table 4. Mean Difference and P value between PSSCogRehab and CCT for Clinical Assessments at baseline testing. When $p \leq 0.05$, CCT was significantly more effective in treating the given symptom.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean Difference at Baseline</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>0.41667</td>
<td>0.841</td>
</tr>
<tr>
<td>Negative</td>
<td>1.635</td>
<td>0.602</td>
</tr>
<tr>
<td>Disorganized/Concrete</td>
<td>-2.58333</td>
<td>0.304</td>
</tr>
<tr>
<td>Excited</td>
<td>0.16667</td>
<td>0.834</td>
</tr>
<tr>
<td>Depressive</td>
<td>2.29167</td>
<td>0.338</td>
</tr>
</tbody>
</table>

Table 5. Mean Difference and P value between PSSCogRehab and CCT for Clinical Assessments at immediate follow-up. When $p \leq 0.05$, CCT was significantly more effective in treating the given symptom.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean Difference at Immediate Follow Up</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>2.40476</td>
<td>0.328</td>
</tr>
<tr>
<td>Negative</td>
<td>-1.95328</td>
<td>0.266</td>
</tr>
<tr>
<td>Disorganized/Concrete</td>
<td>0.47619</td>
<td>0.702</td>
</tr>
<tr>
<td>Excited</td>
<td>-0.16667</td>
<td>0.823</td>
</tr>
<tr>
<td>Depressive</td>
<td>3.85714</td>
<td>0.028*</td>
</tr>
</tbody>
</table>

Table numbers refer to the charts above it.
Table 6. Mean Difference and P value between PSSCogRehab and CCT for Clinical Assessments at 3-month follow-up. When $p \leq 0.05$, CCT was significantly more effective in treating the given symptom.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean Difference at 3-Month Follow-Up</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>3.16667</td>
<td>0.258</td>
</tr>
<tr>
<td>Negative</td>
<td>1.76667</td>
<td>0.499</td>
</tr>
<tr>
<td>Disorganized/Concrete</td>
<td>1.83333</td>
<td>0.307</td>
</tr>
<tr>
<td>Excited</td>
<td>-1.6667</td>
<td>0.734</td>
</tr>
<tr>
<td>Depressive</td>
<td>1.66667</td>
<td>0.509</td>
</tr>
</tbody>
</table>

We predicted that participants in the CCT condition would show larger improvements in functional outcome when compared to the PSSCogRehab condition. A t-test showed no significant difference between CCT and PSSCogRehab with the exception of depressive symptoms at the immediate follow-up ($p = 0.028$).
**II: Between PSSCogRehab and CCT at Paired Time Frames**

Table 7. Mean Difference and P Value between PSSCogRehab and CCT for Clinical Assessments between Baseline and Immediate Follow-Up assessments. When \( p \leq 0.05 \), CCT was significantly more effective in treating the given symptom.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean Difference Between Baseline and Immediate Follow-Up</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>1.52381</td>
<td>0.413</td>
</tr>
<tr>
<td>Negative</td>
<td>1.52381</td>
<td>0.254</td>
</tr>
<tr>
<td>Disorganized/Concrete</td>
<td>3.45238</td>
<td>0.199</td>
</tr>
<tr>
<td>Excited</td>
<td>-0.47619</td>
<td>0.604</td>
</tr>
<tr>
<td>Depressive</td>
<td>0.19048</td>
<td>0.783</td>
</tr>
</tbody>
</table>

Table 8. Mean Difference and P Value between PSSCogRehab and CCT for Clinical Assessments between Immediate Follow-Up and 3-Month Follow-Up assessments. When \( p \leq 0.05 \), CCT was significantly more effective in treating the given symptom.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean Difference Between Immediate Follow-Up and 3-Month Follow-Up</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>-0.6667</td>
<td>0.696</td>
</tr>
<tr>
<td>Negative</td>
<td>4.4</td>
<td><strong>0.021</strong>*</td>
</tr>
<tr>
<td>Disorganized/Concrete</td>
<td>0.8333</td>
<td>0.415</td>
</tr>
<tr>
<td>Excited</td>
<td>0.1667</td>
<td>0.817</td>
</tr>
<tr>
<td>Depressive</td>
<td>-2.16667</td>
<td>0.216</td>
</tr>
</tbody>
</table>

Table numbers refer to the charts above it.
Table 9. Mean Difference and P Value between PSSCogRehab and CCT for Clinical Assessments between Baseline and 3-Month Follow-Up assessments. When $p \leq 0.05$, CCT was significantly more effective in treating the given symptom.

We predicted that participants in the CCT condition would show larger improvements in functional outcome when compared to the PSSCogRehab condition. A paired-sample t-test showed no significant difference between CCT and PSSCogRehab with the exception of negative symptoms between the immediate and three-month follow-up ($p = 0.021$).
Discussion

This section of the Scientific Method is dedicated to examining the results in detail, where I ultimately assess the efficacy of CCT and PSSCogRehab. Statistical analyses revealed no effect between PSSCogRehab performance compared to CCT on symptomology as outlined by the five-factor model of the PANSS. CCT was significantly more effective in improving depressive symptoms at the immediate follow-up; CCT was also significantly more effective in improving negative symptoms between the immediate and three-month follow-up.

Limitations

Here, I discuss the limitations of the study. The small sample size of the study gives low statistical power and increases the chances of Type II error. Furthermore, a small sample size can be a limitation in a study’s ability to produce conclusive results. A factor that consistently contributed to the small sample size was the dropout rate of participants. In addition, the study’s ability to generalize our findings is limited because participant pool is majority-white and thus not racially balanced. Further, our participants are generally older, a limitation because it has been suggested that cognitive remediation therapies are more effective in younger participants, therefore, the high age-span of our participants is not consistent with the samples used in previous research (Kontis, Huddy, Reeder, Landau, & Wykes, 2013; Wykes et al., 2009).

Future Directions

The pilot study ended with its fourth cohort. With a grant approved by the National Institute of Mental Health (NIMH), an improved version of this study will be implemented at River Valley Services and the Institute of Living in Hartford,
CT. The testing battery has been modified, removing several assessments that are no longer suited for the participants’ best interests. Further, the testing battery includes the addition of the MATRICS battery. Another assessment period has also been implemented after the first half of the interventions. To address low morale in the control group, the waiting list has been replaced with computer games to improve motivation and engagement with the study. At the time of this thesis, the first cohort has completed baseline testing at IOL and began the intervention; recruitment is currently being organized at RVS.

**Conclusion**

The randomized control trial has successfully been completed with three cohorts. The fourth cohort did not complete the pilot, which was discontinued after the post-treatment test. It must be acknowledged that the nature of this study is exploratory, but it can be inferred from the data that the results are promising. With a larger sample size, it is expected that the effects will gain statistical power and analysis will produce more significant results.

This marks the end of the traditional neuroscience thesis. If I had followed the Scientific Method to the fullest, the results section would be more substantial; I would have completed an analysis with the entire testing battery, however, the conclusion would remain the same (Arlia, 2019). We would end hopeful for the future, depending on subsequent studies to suggest that CR is effective in treating the cognitive features of schizophrenias. However, because this is not a traditional neuroscience thesis, I am able to go beyond the bounds of the Scientific Method to interrogate what the dataset additionally offers. Although the data did not prove our hypothesis, the feminist model of inquiry would ask us to look beyond the lack of
significance of the data; in other words, just because the data did not fit within the established bounds of significance according to the p-value does not mean that this data cannot provide insightful conclusions about not only cognitive remediation, but also the treatment of schizophrenia. To see the true significance of the data, therefore, we must look beyond the traditional understandings of data to consider the less numerical, objective methods of psychiatric knowledge-making.

First, Roy asks us to consider if we, as the scientist, placed ourselves on a mutual footing with the subject of observation. In her case, the material was microbiological; in my case, the “material” was the clients I was sitting with, listening to their stories, seeing the impact a broken system had on their sense of self. There is a certain immaterial nature to a clinical neuropsychological study that the enumeration of symptoms and statistical analyses cannot quite grapple with. This can be seen with the testing battery itself. The research assistants are advised to follow a similar testing order to retain reliability across subjects, however, this measure taken to create standardization between subjects raises several questions. Recall that earlier in this chapter, I described my interactions with D., who, with each subsequent session, became more comfortable with me. For the purposes of the PANSS, the only valid data that can be used is within the bounds of its administration. If D. said something that contradicted her initial responses later on in the testing battery — even something that would fundamentally change the scoring on the PANSS — it was not valid. There is an incredible subjectivity to the PANSS, and perhaps if I had administered the PANSS on a later date, the responses I would have examined and scored may have changed.
Therefore, if I were to ask if I placed myself on a mutual footing with D., I would have to look beyond the testing battery and move towards not only my relationship with her, but also the way I discuss the data. As such, Roy ensured that she acknowledged the relationship she has with material. As she notes, even though she worked with neurons, her way of establishing a mutual footing with her subject was by acknowledging that the cells she was working with were alive (Roy 2004). An essential part of this process for my study is to avoid reductionist language that psychiatric literature often promotes. As such, I made sure to see D. not just for the purposes of the study, but as a person with a lived experience. I made sure to hear her when she wanted to share a part of her story, even if it did not relate to the study. Furthermore, by putting myself on a mutual footing with D. and the rest of the clients in the study, I am responsible for looking beyond the reported results and discuss the real-life implications of such a study. Although the data did not show significance in the ways we expected there is another way we can interpret the effectiveness of CR. I argue that the value of CR cannot be fully seen in the normative ways we understand significance in a research study. Instead, an interpretation based on the lived experiences of those with schizophrenia to analyze how therapies have positively impacted their lives is needed. This focus on the narrative is discussed in Chapter 3, where I will continue examining what an STS-derived conclusion on this data would look like.
Chapter Three: Reclaiming and Rehumanizing Psychiatry

Every Wednesday, I sit at my desk and wait for my therapist, Vincent, to FaceTime me. Over the past few weeks, our conversations have been similar — this is not surprising, considering the pandemic and a month of social distancing — and we’ve settled into a familiar cadence. I describe my week, all of which have become eerily similar, days blurring into weeks. He suggests that I go outside and perhaps exercise. We’ve been working together since January, with the first few months dedicated to deconstructing and reconstructing my sense of self. Naturally, my thesis has come up in conversation, as the “stressful” thing, as the “motivating” thing, but I’ve never discussed the project in full. This past Wednesday, I rant about how my thesis was one of the few sources of stability I had at the moment. *The world is on fire, but this is the one thing that I care about right now,* I say to him, acknowledging the extreme uncertainty of the present. I tell him my weekly plan — to write this chapter — and he asks me to describe what I wanted to do. I tell him that I’m frustrated that psychiatry is so numbers-focused, obsessed with labels and diagnostic tools that it forgets the person affected by it all. He replies that when he was younger, he was that person. He loved to categorize everything, including people, according to well-defined boundaries. He says that becoming a therapist showed him that those boundaries aren’t actually that well-defined; instead, he realized that he couldn’t rely on the DSM to define his patients. *Two people who have “bipolar” experience “bipolar disorder” very differently,* he says, *and by only treating the “bipolar,” you fail to help the person with bipolar disorder.* He argues that although medication is useful for treating symptoms, only relying on diagnostic
and descriptive tools forces you to view the person within the boundaries of the tool.

Recall in Chapter 1 that I argue that epidemiological studies can only offer an understanding of schizophrenia that is tied to the descriptive tools that define schizophrenia; these tools are influenced by the Scientific Method that are then influenced by the scientist producing psychiatric knowledge and the institutions that use science as a means of control. Moreover, in Chapter 2, I apply this understanding to my laboratory and conclude that had I followed the Scientific Method, I would have missed out on D.’s lived experience with schizophrenia. In this chapter, I will continue to highlight the lived experiences of schizophrenia by drawing on insights from memoirs written by people with schizophrenia who describe how they navigate through not only their internal experience with schizophrenia, but also the external institutions of treatment. I raise that focusing on the experience of the sufferer\textsuperscript{25} can reconcile psychiatry and approximate it towards the feminist science for which Roy advocates.

As discussed previously, one of the foundational shortcomings of neuropsychiatric research is the enumeration of symptoms. Although this diagnosis, as Wang (2019) writes, “…is comforting because it provides a framework – a community, a lineage—and, if luck is afoot, a treatment or cure. A diagnosis says that I am crazy, but in a particular way…” it also results in a loss of agency within medical institutions (Wang, 2019, p. 5). Wang’s work, \textit{The Collected}

\textsuperscript{25} And I don’t just mean sufferer of schizophrenia. I mean sufferer to encapsulate far beyond the diagnosis, because being diagnosed with schizophrenia entails suffering due to medicine, due to stigma, due to losing one’s autonomy.
Schizophrenias, explores the lived experience of schizophrenia and offers a powerful account of how she navigates her diagnosis of schizoaffective disorder. She states, “Schizophrenics are victims of the Russian word гибель (gibel), which is synonymous with ‘doom’ and ‘catastrophe — not necessarily death nor suicide, but a ruinous cessation of existence; we deteriorate in a way that is painful for others” (Wang, 2019, p. 3).

This cessation of experience is not equally experienced by people diagnosed with schizophrenia. Wang acknowledges her privilege as an Ivy-League educated woman, which afforded her to navigate society under a certain neurotypicality. She notes that even her psychiatrist was reluctant to diagnose her with schizoaffective disorder due to the stigma Wang would face from both her community and medical professionals. Acknowledging the power of the label of schizophrenia, she states, “Humans are the arbiters of which diagnoses are given to other humans — who are, in most cases, suffering, and at the mercy of doctors whose diagnostic decisions hold great power” (Wang, 2019, p. 13). Being “schizophrenic,” as she notes, changes the way a doctor views her medical history and thus changes the way she receives treatment. A DSM-5 diagnosis of schizophrenia, as she notes, “shrink-wraps the bloody circumstance with objectivity until the words are colorless” changing one’s self-perception and, more importantly, the level of agency afforded to them by psychiatry (Wang, 2019, 11).

The denial of agency is seen through the many powerful narrative memoirs written by people who are diagnosed with a mental disorder (Cahalan, 2012; Lukach, 2017; Redfield Jamison, 1995; Saks, 2007; Wang, 2019). Lukach’s (2017)
*My Lovely Wife in the Psych Ward* describes how his wife, Guilia, repeatedly lost her agency due to her psychotic behavior (Lukach, 2017). Involuntarily admitted to the psych ward under code 5250, Guilia was forced to take anti-psychotic medication against her will. Lukach recounts his guilt when he was convinced that a 5250 was the best for Guilia, stating,

> I closed my eyes and imagined three nurses walking into Guilia’s room, offering her pills, knowing she would refuse them, and then holding her down as they rolled up her hospital gown to expose her thigh for an injection…But the image of it, the nurses pressing down on her body, Guilia tensed and yelling and resisting whatever way she could, and them injecting her anyways, and knowing with certainty that it had happened, felt so tragic…(Lukach, 2017, 55).

Wang astutely notes that the involuntary hospitalization is not dissimilar to incarceration, noting “a confined person’s ability to control their life and their body is dramatically reduced; they are in the mercy of those in control…For those of us living with severe mental illness, the world is full of cages where we can be locked in” (Wang, 2019, p. 110).

The 5250, however, was not the only way that Guilia — and other patients who are involuntarily admitted — lose control. Often, the medication that they are forced to take also have profound impacts on their self-perception. Lukach notes that Guilia’s mind without medication was like a television that was stuck between two channels. Although the medication helped turn down the crackling noise, her psychosis convinced her that the medications were harming her (Lukach, 2017).
To reclaim her sense of self, Guilia began haphazardly taking her medication, which resulted in another set of 5250s. She was, once again, stripped of her agency and was involuntarily admitted. Recovery took several years of battling not only her psychosis, but the very institutions that reshaped her mind and body through pharmaceuticals (Lukach, 2017). Wang, who had also been involuntarily hospitalized, argues that there is no worse terror than not knowing when or if she would ever be able to leave the psych ward (Wang, 2019, p. 104). The alternative, as Saks (2007) experienced, is to give patients the agency to choose. She notes how her experience with hospitalization drastically differed when she was voluntarily admitted. She writes, “No one had ever locked me up against my will. I entered the hospital voluntarily. If I were going to be a mental patient, at least it would be by my choice and no one else’s” (Saks, 2007, p. 62). Moreover, she accounts how while in the UK, she was able to work alongside her doctors to create her treatment plan. Instead of forcing medication and therapy plans upon her, each step provided by her doctors was a recommendation, allowing her to choose what she was comfortable with. This allowed her to respond more positively to her treatment, stating, “…each time the decision was mine. Even at my craziest, I interpreted this as a demonstration of respect. When you’re really crazy, respect is like a lifeline someone’s throwing at you. Catch this and maybe you won’t drown” (Saks, 2007, pp. 79-80). This respect gave her the confidence to follow — and respond positively to — her treatment plan.

These narrative accounts problematize the ways that medicine has denied patient autonomy because patients’ experiences are often overshadowed by the
power of the *DSM* and conventional methods of describing disorder. Radical movements in psychiatry place an important emphasis on the power of the patient’s voice. Many of these movements focus on rehumanizing the scientific accounts of schizophrenia, thus calling for an emphasis on the narrative rather than the enumeration of symptoms. As Gambrill (2014) notes, this is especially important because the evolution of the DSM has resulted in the medicalization of distress that may arise from environmental factors such as poverty and the related political, social, moral, and economic effects of poverty (Gambrill, 2014). The *DSM-5*, in particular, has constructed even more diagnostic categories with looser boundaries which cause more people to fit the definition of mentally ill. Gambrill argues that this loosened medicalized classification system obscures the complexity of human behavior and, consequently, blames the individual for their oppression. She states,

> Biomedical psychiatry and pharmaceutical companies, with the help of the state, have been very successful in forwarding medical views of problems-in-living including transforming everyday behaviors, thoughts, and feelings into mental illnesses requiring medical solutions (medication), as illustrated by the ever-lengthening list of behaviors viewed as signs of mental illness and promotion of medical remedies (prescribed medication) (Gambrill, 2014, p. 14).

By doing so, biomedical psychiatry fails to acknowledge the oppressive structures that cause so-called disordered behavior; instead, the human experience is reduced and transformed into a diagnosable condition. Rectifying this requires a radical reframing of the definition of a disorder.
Among these movements is the Icarus Project, which aims to complicate the dominant biomedical model of mental illness (DuBrul, 2014). The Project emphasizes that labels such as “disorder,” “disease,” and “dysfunction” do not represent the fluid, complex experience of a mental disorder. Instead, the Project aims to envision a new culture and language that resonates with the lived experience of having a “mental illness.” DeBrul states,

How helpful is the modern psychiatric paradigm that revolves around medicine and mental disorders and how much of it is really just a function of powerful pharmaceutical corporations, public funding cuts, and a society that equates productivity with health? Are there other frameworks for understanding what it means to be “crazy?” Are there alternative ways to heal? How do we begin the process? (DuBrul, 2014, p. 264).

By rejecting the conventional framework of illness based on the enumeration of symptoms, the Icarus Project collaborates with those who are affected by diagnoses to reframe the oppression and alienation of conventional psychiatry. This reframing highlights humans as meaning makers, emphasizing that this meaning must be cultivated, envisioned through the emphasis of individual stories, allowing for an exploration of creativity, struggle and growth. As such, the Icarus Project creates community to “see our condition as a dangerous gift to be cultivated and taken care of rather than as a disease or disorder needing to be ‘cured.’” (DuBrul, 2014, p. 259)
Especially when viewed within the context of the Icarus Project, accounts like Saks’ and Wang’s allow for an interrogation of the normative process of involuntarily admission to the psychiatric ward. By writing how medical professionals often ignore somatic symptoms by labeling it as psychosis, these accounts further highlight how the medical community has failed its patients. Saks, who had been experiencing severe migraines, describes her multiple ER visits that resulted in immediate discharges due to her psychiatric history. Her symptoms were ignored, and she was instead told by doctors that she was simply having an “episode” (Saks 2007). It was only when she visited another ER that a doctor heard her concerns, and she was diagnosed with a subarachnoid hemorrhage, a brain bleed that would have been left undiagnosed had her family not intervened (Saks 2007). She argues that it was because of her psychiatric history that her experience was invalidated; her recovery amplified this bias further, as the psychologists who were evaluating her recovery suggested that her memory loss was not due to the hemorrhage, but because she was trying to look impaired due to her “disturbed thought process,” stating, “I was either faking it or crazy, or not eating properly. Somehow, the existence of an actual brain hemorrhage had disappeared in its importance” (Saks, 2007, pp. 236-237).

Patients with mental disorders are often ignored because, as Saks notes, the medical community does not take “mental patients” seriously (Saks, 2007, p. 233). The stigma of mental disorder, however, bleeds far beyond the psych ward, as patients who merely present psychotic symptoms are immediately diagnosed with a psychosis-spectrum disorder. Susannah Cahalan (2012), in her memoir Brain on
Fire: My Month of Madness, writes about her experience with such misdiagnoses. Cahalan had a rare form of encephalitis that caused hallucinations and disordered behavior. While hospitalized for a month, she was misdiagnosed multiple times, ranging from partying too hard to schizoaffective disorder. Eventually, a few physicians began seeing past her psychosis and began administering neurological tests to discover that she had Anti-NMDA receptor encephalitis (Cahalan, 2012). Cahalan writes that at the time, only ten percent of those with Anti-NMDA receptor encephalitis were correctly diagnosed and she could have very well died from it, highlighting the responsibility of medical professionals to treat patients without projecting the biases that is embedded within the medical institution.

These accounts serve as a powerful tool to demonstrate the importance of recognizing the lived experience of disorder when treating patients. Whether it is through the implementation or the creation of psychiatric knowledge, we must be aware of the often-subtle ways that bias bleeds into our interactions with those who are the most vulnerable. As Wang writes, “We are, in the end, linked by desperation based in suffering, and based on a system of conventional medicine that…has no method of alleviating that suffering” (Wang, 2019, 176). By focusing our understanding of disorder through the narrative rather than reductive descriptive tools, psychiatric institutions can begin reconciling years of suffering caused by oppressions linked to racism, classism, and the patriarchy. It is our responsibility as scientists to address the suffering that science-making has caused, especially to those who we observe in the name of science-making. We must push towards a method of science-making that views narrative accounts as potential data that can
teach much more about the experience of schizophrenia than descriptive tools ever can.

With all this being said, I don’t think psychiatry is inherently evil. Humans are curious beings, and we have sought to understand both the external world and our internal states for thousands of years; when the power to explore, however, is left to the hands of the few and powerful, that exploration results in systematic inequality. As scientists, we cannot ignore the flaws of psychiatry, the tools we use to diagnose and assess disorder, and the Scientific Method. When we do, the experiences of those who have been impacted by institutions of power — be it psychiatry, science, racism, politics, capitalism, or the patriarchy — are invalidated. This is not to say that psychiatry is a lost cause, either; instead, psychiatry must be radically changed to embed the lived experiences of those who experience mental disorder.

We must first begin, as Roy argues, with the tools that we use to create knowledge by distancing the sciences from its deterministic and essentializing language (Roy 2018). This radical reframing will require action that might be considered disruptive by normative scientists, but as Roy notes, such disruptions have resulted in our current understanding of scientific authority and knowledge production (Bose, 1902; Margulis, 2008; McClintock, 1950). To do so we, as scientists, must not simply judge a knowledge system that is not our own, but instead, we must actively engage within those systems. As Hacking notes, experimentation is not static; it is living and must be treated as such (Hacking & Hacking, 1983). Thus, a feminist science must reconfigure the relationship between the “scientific knower and what is to be known” (Roy, 2018, p. 11). This cannot be
executed without a radical transformation of *who* does the science, either. Rather, for a feminist science to thrive, feminists *themselves* must learn science and “face the nitty-gritty technical core of scientific knowledge production as well as the contradictions, tensions, and dilemmas that come with carrying out scientific experiments” (Roy, 2018, p. 135). Here, I consider both the tools used to define science and the scientist itself as tools of the Scientific Method because through my time as a researcher, I have seen how I am also a product of the co-production of psychiatric knowledge between the intersections of race, class, and gender and medicine. I am as guilty of falling into the trap of normative medicine as much as the scholars who I have criticized earlier. Roy is also aware of this, stating “such factors as one’s sex gender, sexuality, race, ethnicity, class, age, abilities, location, and more influence who gets to conduct research, which questions generally get asked, and what knowledge is ultimately produced” (Roy, 2018, p. 13). And it is with this acknowledgement that we can move forward and work towards a science that openly embeds this acknowledgement in its practice.

This radical reframing need not begin with sweeping institutional changes, but instead, can be accomplished by creating community that prioritize the involvement of people who are most affected by the flawed psychiatric institution.26 I argue that embedding both the scientist’s narrative as well as the narrative of those

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26 As much as I’d like to see the radical toppling of the Scientific Method, I fear that “new” systems of knowledge-making would be riddled with similar flaws because of science’s interactions with the powerful constructs I have discussed in Chapter 1. Instead, we must work within the current framework to make space for marginalized voices.
who are marginalized best allows for this reframing. This chapter has focused on the importance of narrative psychology, which as Vassilieva (2016) argues,

[The] narrative represents ‘a basic human strategy for coming to terms with time, process, and change— a strategy that contrasts with, but is in no way inferior to, “scientific” modes of explanation that characterize phenomena as instances of general covering laws’(Vassilieva, 2016, p. 2)

The narrative, as a result, combats the current state of psychiatry as a discipline that is absent from contemporary cultural, political, and philosophical debates, a consequence of the medicalization of psychiatry that I discussed in Chapter 1 (Gergen, 2001). By humanizing psychiatry, we are able to use the narrative to create a new kind of empirical knowledge that focuses on the raw experience rather than symptoms that are reduced down to scalar measures.

But most importantly, a method for psychiatric knowledge-making that allows for what Roy, Crenshaw, and Jasanoff call for requires a constant questioning of truth. As scientists, we cannot consider what we once thought was truth as such because we’ve seen that the objective “truths” of science are riddled with inconsistencies due to the many external influences that have shaped science. We can no longer operate in a binary between the so-called “hard” sciences and the social sciences; neither field should operate in its own universe because both disciplines have much to learn from each other. By embedding this thesis in the social sciences, I was able to view neuropsychiatric epidemiology within the lens of co-production and intersectionality; without these frameworks, I would be
unable to consider how race, class, and gender work with medicine to reproduce schizophrenia. We must redefine how we view the mind and its relationship with “disorder” to reconcile the ways that race, class, and gender have influenced that very definition. If we fail to redefine and reconstruct the very ways we create knowledge, neuroscience and psychology will continue to marginalize those who are often already marginalized by racism, classism, and the patriarchy. We will continue to allow these same powerful institutions to affect the everyday practices of science. As we’ve seen, psychiatric knowledge-making not only influences the DSM and diagnosis itself, but it bleeds into doctor-patient interactions and holds significant implications to institutions beyond psychiatry, making it much more imperative that we take the steps to improve science-making due to its foundational influence in these interactions.

My last day with D. was anti-climactic: after finishing up the last few tests that were left in the battery, I handed her a Walmart gift card. She signed a slip of paper to certify that she received the gift card, and I placed that slip into her file. We walked downstairs to the lobby together, where I thanked her for her time, and we parted ways. It was only until I began testing the following semester with another client did I see D. again. Walking down the hallway, I recognized her from her colorful outfit, but she didn’t recognize me. She continued walking, and I did the same.

I think about what would have happened if I decided to write a traditional neuroscience thesis. Perhaps, D. would have blurred into a line of digits that I would revisit two semesters later when I had to analyze this data. I would have concluded
that our data is promising — nothing more, nothing less — and would move on to the next study. It would feel nearly as anti-climactic as my last interaction with D., but perhaps I wouldn’t have noticed because I would have regarded the “nothing more, nothing less” as the normal way to end a thesis. But that isn’t the way this thesis was meant to end. Instead, we are left with a complexity and nuance that I have grown to both love and despise at the same time, a complexity and nuance that has allowed me to view myself as both a subject and agent in the Scientific Method, which I argue is the way it is supposed to be.
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