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# Introduction

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Schizophrenia is and is not a thing in the world.<sup>1</sup> To borrow a phrase from Steve Shapin, there is no such thing as schizophrenia, and this is its ethnography.<sup>2</sup> There are few medical labels that have been so firmly rejected—and for some good reasons. There is no specific genetic marker for the illness. It has no clear-cut trajectory, though most who experience it probably never return to the way they were before things went wrong. It has no unique symptoms—no symptoms specific to this disorder and not found in other disorders. The man who distinguished schizophrenia from bipolar disorder, Emil Kraepelin, did so on the basis of schizophrenia's progressively degenerative course—and now many argue that recovery from schizophrenia is possible. The man who gave the disorder its name, Eugen Bleuler, did not list hallucinations among his identifying features of the disorder—and these days, hallucinated voices are often assumed to be its primary symptom.<sup>3</sup> Schizophrenia may have been with us forever, but there are those who believe that the illness only emerged in the nineteenth century.<sup>4</sup> The difference between people who best fit the description of schizophrenia and those who similarly best fit the description of bipolar disorder or depression is striking—and yet more people seem to lie in the broad, gray, murky boundaries. The difficulty of finding specific neuroscientific markers for this (or, for that matter, any other) psychiatric illness has led the National Institute of Mental Health (NIMH), in recent years, to refuse to fund research based on diagnostic categories.<sup>5</sup>

And yet there certainly is a real and terrible disorder, the most devastating of all the psychiatric illnesses, that at its most severe has clearly recognizable

features and is found in nearly every corner of the world. In the modern era the disorder has consistently been understood as a combination of several groups of symptoms: first, the so-called positive symptoms of psychosis—the radical break with reality signaled by delusions, hallucinations, and incoherent speech; second, the so-called negative symptoms of emotional withdrawal, signaled by an unexpressive face and voice tone, often called “flat affect,” and mismatched emotion-cognition displays, like giggling when talking about something sad; and third, the so-called symptoms of cognitive dysfunction, signaled when someone’s life at work or at home seriously falls apart for a significant length of time. People with schizophrenia often hear voices talking to them, commanding them, sneering at them, cajoling them, sometimes so loudly it can be hard for them to hear anyone else, and the voices can continue for decades. The illness is terrifyingly common, claiming roughly one in a hundred people pretty much everywhere we have looked.<sup>6</sup>

This book examines the way this terrible madness is shaped by its social context: how life is lived with this madness in different settings, and what it is about those settings that alters the course of the illness, its outcome, and even the structure of its symptoms. We will call this madness “schizophrenia,” recognizing that the term is contested and its boundaries complicated, because the term points to the severe, persistent break with reality that is recognized around the world and is also identified in each of our case studies. It is a term with invisible scare quotes, but no other word does its job.

The question of how this madness is shaped by its social setting is a much bigger one than it was even a decade ago. Until recently, schizophrenia was perhaps our best example—our poster child—for the “bio-bio-bio” model of psychiatric illness: genetic cause, brain alteration, pharmacologic treatment.<sup>7</sup> The embrace of its fundamentally organic nature had arisen from new scientific research that swept in a biological psychiatry. The triumphant rethinking of psychiatric illness was heralded by books like Nancy Andreasen’s *The Broken Brain: The Biological Revolution in Psychiatry*, which took schizophrenia as its focus and as the best evidence for the disease-like nature of serious mental illness.<sup>8</sup> The 1990s became NIMH’s “Decade of the Brain.” Psychiatry was to be wrested away from its decades-long dependence on psychoanalysis and established as a field of medicine like any other.

That has happened. Psychiatry is no longer the field it was when psychoanalysis dominated the way psychiatrists thought. Most disorders are understood as diseases, and most of them are treated with medication. But in recent

years psychiatric disorders have become less culture free, *less* biological, if by “biological” we mean that they are understood to arise from our genes and to unfold independently from our social world. Increasingly we know that our genes interact with our environment and that this *epigenetic* interaction deeply shapes our lives.<sup>9</sup> This is true even of our most terrible madness. In the case of schizophrenia, we now have direct evidence that people are more likely to fall ill with schizophrenia in some social settings than in others, and more likely to recover in some social settings than in others. We know from the empirical research carried out by the new social epidemiology that something about the social world gets under the skin. The puzzle is to figure out what it is.

There is a new role for anthropology in the science of schizophrenia. Psychiatric science has learned—epidemiologically, empirically, quantitatively—that our social world makes a difference. But the highly structured, specific-variable analytic methods of standard psychiatric science cannot tell us what it is about culture that has that impact. Anthropology can. At least, the careful observation enabled by rich ethnography allows us to see in more detail what kinds of social and cultural features may make a difference to a life lived with schizophrenia.

This volume presents twelve case studies that help illustrate some of the variability in the social experience of schizophrenia. We sought cases that best illustrate the main hypotheses about the different experience of schizophrenia in the West and outside the West. Most of the authors are psychiatric anthropologists, that breed of ethnographer who takes mental illness as a central focus. Each was asked to tell the story of one person in the particular culture they studied who represents something important about the experience of schizophreniform disorder in that setting.

To be clear, the authors did not themselves conduct formal diagnostic interviews with their subjects. In each case, however, the author met the subject in a context in which caseworkers, clinicians, or the subjects themselves volunteered a diagnosis of schizophrenia or schizophreniform disorder (e.g., schizoaffective disorder). In all cases it was clear that the subject experienced “positive symptoms” like hallucinations or delusions; that the subject’s life had been seriously disrupted by their illness; and that they had struggled with the symptoms for more than six months. These are, in broad brushstrokes, the DSM criteria for schizophrenia. There is no question that each of our subjects has been very ill with a serious psychotic disorder.

There is, inevitably, a catch-as-catch-can quality to these case studies. Few anthropologists focus exclusively on schizophrenia. We reached out to find

people already working in the field who would have contact with possible cases.

Most of our cases come from the United States or from India, because the well-known findings that schizophrenia has a more benign course in India than it does in the West have led many anthropologists to look at schizophrenia in India. Our overrepresentation of work in India allows us to examine closely the factors that might account for this better outcome.

We also have more case studies about women. This is an accident based on our own gender—most of the contributors to this book are women. It is easier in many societies—and certainly in India—for female ethnographers to form close relationships with women. This accident may serve us well. Women are somewhat less likely than men to fall ill with schizophrenia: globally the incidence rate is roughly 1.4 men to 1 woman.<sup>10</sup> An emphasis on women may help us to see more clearly the social conditions that make someone vulnerable. And by looking primarily at women, we are at least comparing like with like.

We use our case studies to look closely at some basic problems in culture and schizophrenia: diagnosis and social identity; vulnerable transition points that may help trigger illness; a kind of psychosis, more common outside the West, in which people return to baseline after madness; immigrants who are more at risk of illness; a more benign voice-hearing experience; supernaturalist explanations of psychosis; the harsh institutional circuit that many with schizophrenia encounter in the United States; and recovery. At the end, we draw conclusions from these case studies and from other ethnographies, including an excellent collection by Janis Jenkins and Robert Barrett, *Schizophrenia, Culture, and Subjectivity*, that precedes us in the field. We believe that if we understood culture's impact more deeply, it could change the way we treat schizophrenia. That's particularly important because it turns out that schizophrenia is probably more common in a Western setting, and certainly more caustic.<sup>11</sup>

We call this approach “clinical ethnography.”<sup>12</sup> All of us were trained first and foremost as ethnographers. But many of us have had substantial clinical training (and Johanne Eliacin became fully licensed as a clinical psychologist while obtaining her scholarly degree). We read the psychiatric literature as well as the anthropological literature. We do not see our job, first and foremost, as criticizing mental health professionals as observers. We see ourselves as working alongside mental health and medical professionals to understand the illnesses humans confront and to contribute to the process of helping ease

their distress. We begin with the epidemiological puzzles and set out to research the patterns of local meaning that may help explain them.

There has been a shift in the way many anthropologists have been thinking about psychiatry in the past decade—away from a skeptical, even dismissive approach to clinicians to a more clinically engaged research process. These engaged anthropologists are more empathic with the struggles faced by clinicians, more collaborative with scientists and clinicians, more likely to publish in medical journals as well as in anthropology journals, and often more medically sophisticated. They are more likely to work in the trenches alongside clinicians.<sup>13</sup> In a recent essay, Rob Whitley calls this approach “no opposition without proposition.” He argues that psychiatric anthropologists should not only provide a meaningful critique of practices and beliefs within psychiatry, and not only illuminate the sociocultural, familial, and clinical contexts of illness, but also serve as a positive catalyst for change.<sup>14</sup> This is an engaged anthropology “in” medicine, as well as an anthropology “of” medicine. That is what we set out to accomplish here.

But first, we begin with an overview of our most troubling madness.

In the years when Benjamin Franklin defended the creation of the new America and Jean-Jacques Rousseau and others wrote tracts that set out the conditions for a just society, madness was imagined as a disease fomented by a world choking under the weight of its own civilization.<sup>15</sup> On his way to building his argument about the social contract, Rousseau invented a state of nature—part hypothetical, part based on travelers’ tales—that he saw as a state of grace and possibility. The real Europe around him he took to be corrupt and decadent, and he thought that it drove men mad. In *Emile*, he wrote that “Everything degenerates in the hands of man.” The nineteenth century—with the sense of social fragility after the French revolution, the rapid urbanization and social turmoil of the industrial revolution, the rising awareness of other ways of life through colonial expansion—led many European intellectuals to the conviction that European society was in trouble and decaying from within. In his end-of-the-century best seller, *Degeneration*, the Parisian Max Nordau pronounced that “We stand now in the midst of a severe mental epidemic; of a sort of black death of degeneration and hysteria.”<sup>16</sup>

As the nineteenth century turned into the twentieth, Émile Durkheim used statistics and census data to demonstrate, in *Suicide*, that as social cohesion loosened, more people killed themselves. In his models, primitive people were

so socially cohesive that they barely had any individuality at all. He thought that in such densely interdependent groups people might kill themselves for altruistic reasons, like World War II kamikaze pilots who deliberately crashed their bomb-loaded planes into enemy warships. But they would not kill themselves from anomie. I remember a class in graduate school in which our lecturer, sketching out Durkheim's theory on the blackboard, drew moderns as a crowd of stick figures with little round heads and primitives as one large oval head with dozens of little stick bodies poking out beneath. No one in that society, in this way of thinking, should ever have been psychiatrically ill.

That sensibility lingered on in Claude Lévi-Strauss, who in *Tristes Tropiques* wrote of a Europe suffocating "like some ageing animal whose thickening hide has formed an impermeable crust and, by no longer allowing the skin to breathe, is hastening the ageing process." The Amazonian Nambikwara, by contrast, he thought of as free. One evening, gazing at ocher-smeared families around a campfire, Lévi-Strauss wrote that an observer "can see in all of them an immense kindness, a profoundly carefree attitude, a naïve and charming animal satisfaction and—binding these various feelings together—something which might be called the most truthful and moving expression of human love."<sup>17</sup>

This happy vision was shattered by long-term fieldwork. It became clear that people like the Nambikwara did struggle with mental illness, and indeed with the same mental illnesses (in some broad sense) as those in the West. And yet it would also become clear, over time, that there was in fact something to these romantic views, although the contrast had been vastly overstated.

In the early twentieth century the colonial encounter had, of course, banished the myth of the Noble Savage—"gloriously glowing in rude but radiant physical health"<sup>18</sup>—but it had not entirely demolished the sense that fragile nerves and melancholy were the products of civilization.<sup>19</sup> Indeed, early anthropological reports seemed to confirm this. In 1929, C. G. Seligman, an anthropologist and physician, argued that serious mental illness did not exist in New Guinea, except where people had been deeply westernized.<sup>20</sup> This incensed a young British woman who came to Ghana in the 1930s and noticed a series of new shrines she thought were treating mental illness.

[T]here still lingers the idea that mental stress and mental illness are the prerogative of "over civilized" societies: that the simple savage may have Ancylostomiasis but cannot have Anxiety; that he may, in his innocence, believe his neighbour to be making bad magic against him, but he still sleeps like a top.<sup>21</sup>

M.J. Field could write. She returned home but came back to Ghana after the war, by that point trained as a psychiatrist, and settled in as an observer at a rural shrine. She recorded over 2,500 supplications. Most of these were about infertility and business trouble, but some seemed to be about serious psychiatric trouble.<sup>22</sup> She took remarkably detailed notes about those cases. At the end of her work, when drought and rising bus prices meant that fewer people were coming to the shrine, she went looking for what she called *chronic schizophrenia*—a term the British used more narrowly than Americans did<sup>23</sup>—and found forty-one individuals in twelve Ashanti country towns and villages with a population of 4,283 (in the 1948 census). These people, often located for her by a tribal elder, were obviously mad, talking *basu-basu*—for example, “an unkempt woman, with a baby on her back, dancing, singing, laughing and shouting.”<sup>24</sup>

Field’s *Search for Security* is a remarkable book, both because of its trenchant asides—“the latter Christian prophets owe their extinction to the poverty of their understanding and personality and the consequent inanity of their prophesying”<sup>25</sup>—and the unparalleled detail of her data. (She offers 144 examples of the mottos painted on the sides of local taxis.) She had no trouble recognizing in Ghana the serious psychiatric disorders she saw in her own society, although not all of the Ghanaians she thought were medically ill were seen as such by their fellow villagers. Nearly every person she thought could be diagnosed with depression came to the shrine accusing themselves of being witches.

Patients suffering from severe depression are, the world over, unshakably convinced of their own worthlessness and wickedness and irrationally accuse themselves of having committed every unforgivable sin. In Africa the worst sin they can imagine is witchcraft, and they insist that they have abundantly committed it.<sup>26</sup>

Field thought that local ideas about witchcraft could not have been sustained by these shrewd and common-sensical people without the rich evidence of those repeated confessions.

Her observations about schizophrenia are striking, and increasingly they are supported by later work. She thought that the basic rates were about the same as they were in Britain, but that they were higher for those who were literate, not because such people had complicated conflicts about being both traditional and Western, but because of frustrated aspirations—because the hard work of becoming literate often led to little economic gain.<sup>27</sup> We now



know that poverty and racism do increase the risk of psychotic illness. She sometimes saw a kind of psychosis in which people became suddenly and dramatically ill—and then just got better. We now know that there is a condition—non-affective acute remitting psychosis—that behaves this way, and that it is more common outside the West. She thought that the work demanded by farming—not only physical but intermittent, so that there were often unoccupied men sitting around the village—made it easier for a man with schizophrenia to pass as normal. She noticed that a third of the women with chronic schizophrenia became ill after menopause but that the stress of marriage could precipitate the illness. And she thought that on the whole, people with schizophrenia were better off in these villages, where they were known and cared for by relatives, than they would be in urban hospitals. This was not because rural Ghana was a place of timeless tranquility.<sup>28</sup> It was because in rural areas, people knew who the ill were, knew whether they would be violent, and tolerated them as part of the social world. The treatments did not always look kind. She saw people shackled to logs and locked in houses. But ill people stayed with those who knew them.<sup>29</sup>

A few years later a research team—headed by Alexander Leighton, a psychiatrist from Cornell, and Adeoye Lambo, the medical superintendent in the district where the study was done (and later a psychiatrist at University College, Ibadan, Nigeria)—came up with similar results, though in less detail. They set out to replicate in Yoruba country the same epidemiological survey that Leighton had done in his famous Stirling County study, which had found—as Durkheim had predicted—that people in more socially integrated communities were less ill.<sup>30</sup> Using the same diagnostic handbook, they found that while the general pattern of symptoms were quite similar in both settings—“we have not come upon any symptom patterns that are recognized by the Yoruba and are not recognized in [American] psychiatry”<sup>31</sup>—the overall level of impairment rose as one went from a rural African village (15%) to an African town (19%) and thence to Stirling County (33%). The Yoruba really did suffer from recognizable mental illness, but modernization seemed to make things worse.<sup>32</sup> This work also affirmed the observations of Field and of the anthropologist Robert Edgerton (in his study of psychosis in four African communities) that what mattered in a rural African setting was behavior, not inner experience.<sup>33</sup> In African villages, people were identified with serious mental illness when they shouted, stripped naked, and ran into the bush—not because they reported hallucinations. To be sure, the voices may well have



told them to do these things. But it was the behavior, not the voices, that concerned their peers.

These views were not, however, the dominant perspective on schizophrenia within anthropology. These were the years when psychoanalysis dominated psychiatry, and to some extent anthropology, in the United States. In the 1930s Margaret Mead, Ruth Benedict, Edward Sapir, Gregory Bateson, Ralph Linton, and others ran in psychoanalytic circles. They were part of an interdisciplinary seminar at Columbia with psychoanalysts like Abram Kardiner that continued for years.<sup>34</sup> From a psychoanalytic perspective, schizophrenia was a reaction to social experience—not a disease. Even if one conceded some organic process, there were no absolute standards against which people could be declared ill or out of place. What defined people as abnormal was what counted as normal, and that judgment was social and relative. As Ruth Benedict asserted in “Anthropology and the Abnormal,” “one of the most striking facts that emerge from a study of widely varying cultures is the ease with which our abnormals function in other cultures.”<sup>35</sup> Many anthropologists—and many romantic readers of anthropology—wanted to argue that people with the odd hallucinatory experiences the West called “schizophrenia” would thrive in a less modern setting as shamans. They would not even be identified as ill. Some protested that such a shaman would still be sick; as George Devereux, the most vehement of these voices, said, “Briefly stated, my position is that the shaman is mentally deranged.”<sup>36</sup> But he was arguing against a dominant position.

You still hear this argument that “our” schizophrenia is “their” shamanism. Compassionate clinicians, trying to make bad news sound better, sometimes tell patients that their ability to see and hear what others do not would be highly valued in other societies. Patients sometimes reach out to the idea of shamanism to make sense of their own sensory experiences or to repair an identity spoiled by the diagnosis—as John Hood does, in a case presented later in this volume. In general, those claims are wrong. In 1983 Richard Noll pointed out that what shamans experience is quite different from what those with schizophrenia experience. The shamanic “state” is willed, is often prosocial (the shaman saves souls and dances with spirits), and accords with local expectations of behavior appropriate for shamans. None of these is typically true of people with schizophrenia.<sup>37</sup> In fact, the shaman must make it clear that he is *not* mad.<sup>38</sup> Shamanism is now understood more as a dissociative process, a trance practice more akin to speaking in tongues and spirit possession than to psychosis. It is nonetheless also true that the relationship between

dissociation and psychosis has now become one of the most vexed questions in the study of psychosis and spirituality, as some of our case studies illustrate.<sup>39</sup>

Meanwhile, the way schizophrenia was understood during the mid-twentieth century would become famous as the most notorious misuse of psychoanalytic theory in American psychiatry. When psychoanalysis dominated American psychiatry, back before the biomedical revolution (roughly from World War II until the 1980s), the dominant American psychiatric perspective on schizophrenia held that the condition was the result of the patient's own emotional conflict. Such patients (it was thought) were unable to reconcile intense feelings of longing for intimacy with their fear of closeness. Neglect in early childhood and their subsequent intense resentment, fury, and violence drove them into an autistic self-preoccupation from which they yearned for contact but were too terrified to reach out for it. As Frieda Fromm-Reichmann—one of the most famous therapists of schizophrenia and the model for the fictional analyst in *I Never Promised You a Rose Garden*—wrote, “the schizophrenic’s partial emotional regression and his withdrawal from the outside world into an autistic private world, with its specific thought processes and modes of feeling and expression, is motivated by his fear of repetitional rejection, his distrust of others, and equally so by his own retaliative hostility, which he abhors, as well as the deep anxiety promoted by this hatred.”<sup>40</sup>

Often, clinicians blamed the mother for delivering conflicting messages of hope and rejection. She was “schizophrenogenic”: her own ambivalence paralyzed her child and drove him or her into the clinical impasse of the illness. The phrase was Fromm-Reichmann’s, although she appears to have used it only once in her own work: “the schizophrenic is painfully distrustful and resentful of other people, because of the severe early warp and rejection that he has encountered in important people of his infancy and childhood, as a rule, mainly in a schizophrenogenic mother.”<sup>41</sup> As the theory developed, schizophrenia became the endpoint of dominating, overprotective, but basically rejecting mothers who actually drove their children crazy. A 1949 article by Trude Tietze, a Viennese-educated psychiatrist, illustrates the genre well. Tietze interviewed the mothers of twenty-five hospitalized adult patients diagnosed with schizophrenia and concluded that they were the cause of their sons’ disturbance. “Once their superficial smiling front was broken through, one was appalled at the emotional emptiness one found. There was a lack of genuine

warmth. . . . It is this intuition or empathy with the child that appears to be missing or inadequately developed.”<sup>42</sup>

By the 1960s it was standard practice in American psychiatry to regard the mother as the cause of the child’s psychosis.<sup>43</sup> So entrenched did this view become that scholars made the most remarkable statements. One author wondered, in a particularly condemnatory essay describing those with schizophrenia as having been reared with “subtle malignancy,” whether schizophrenia as it is known today would exist “if women were impersonally impregnated and gave birth to infants who were reared by state nurses in a communal setting.”<sup>44</sup> The willingness of relatives to pay for hospital care was thought to arise from the guilt they felt for their role in the patient’s suffering. *The Mental Hospital*, the classic 1954 study of one of the best psychoanalytic hospitals, contains this remark: “In some cases it would be reasonably adequate to describe the ideal relative as a person who appeared, gave the history precisely, accurately and directly, and disappeared forever, except for paying his bills—by mail.”<sup>45</sup>

While, from a psychoanalytic perspective, all relationships are fraught by conflict, these relationships between a mother and her schizophrenic child were thought to be particularly torn. Gregory Bateson famously characterized their presumed destructive ambivalence as a “double bind.” The characteristic experience of schizophrenia, he argued, was one in which a mother would approach with a loving invitation; the child would respond, reaching out to give her a hug; the mother would flinch from the embrace; the child would withdraw; and the mother would then say, “Don’t you love me?” “The child is punished for discriminating accurately what she is expressing, and he is punished for discriminating inaccurately—he is caught in a double bind.”<sup>46</sup> The patient then becomes unable to assign what Bateson called “the correct communicational mode” to utterances. Bateson inferred the schizophrenic double bind from his observation that patients with schizophrenia often confused the literal with the metaphorical, but also from his own theory of communicational frames. That theory argued that communications have meaning in a context: an aggressive gesture after the indication “This is play” (“Let’s play pirates”) has a meaning quite different from that aggressive gesture in a non-play frame. He thought that people developed schizophrenia because when they were caught in a double bind—hug me, don’t touch me—frame sorting was emotionally impossible, and so they conflated communicative frames, the literal and the metaphorical, the explicit and the implied.

It was precisely because these patients seemed so conflicted, so incoherent, so sick, that psychiatrists found them to be the most interesting and most compelling patients of the era. In one of the most famous hospitals of the time, Mass Mental, the Massachusetts Mental Health Center where many future psychiatric leaders were trained in the 1950s and 1960s, to use psychoanalysis to treat people with schizophrenia became the ultimate professional challenge.<sup>47</sup> Perhaps the most dominant figure at Mass Mental in its heyday was Elvin Semrad, the legendary director of psychiatric residency. He took seriously Freud's dictum that psychoanalysis was a cure through love, and he taught that a doctor's ability to cure came from his ability to care. He taught that care meant to be able to sit with the patient and to bear with him the pain that the patient feared so much that he chose madness over recognition. To Semrad, a schizophrenic patient was the most exciting patient, the tough, difficult patient who made the doctor a "real" doctor because to connect emotionally with such a patient was so hard. As he wrote, "In order to engage a schizophrenic patient in therapy, the therapist's basic attitude must be an acceptance of the patient as he is—of his aims in life, his values and his modes of operating, even when they are different and very often at odds with his own. Loving the patient as he is, in his state of decompensation [his psychosis] is the therapist's primary concern in approaching the patient."<sup>48</sup> Not everyone agreed. Even at Mass Mental, at least some young psychiatrists concluded that these patients were struggling with a brain disorder, and left them alone. "It was nonsense," someone said to me thirty years after the fact. "You couldn't do anything with them."<sup>49</sup>

In the 1970s, for many reasons, psychiatry moved away from psychoanalysis. More and more people began using medical insurance, and insurers resisted reimbursing care for a condition that didn't resemble a disease. At the time, psychiatrists often treated diagnosis as an afterthought. Researchers began to demonstrate that different clinics gave different diagnoses to the same patient.<sup>50</sup> In a spectacularly embarrassing study published in *Science*, a Stanford psychologist revealed that twelve different hospitals, each with a trained medical team, had given diagnoses of schizophrenia to people who weren't ill at all, but said they'd heard a voice saying "thud."<sup>51</sup> R. D. Laing, a psychiatrist, had already published his *The Divided Self* (1960), in which he explained that psychiatric symptoms made sense: they were reasonable attempts to communicate anguish. Thomas Szasz, also a psychiatrist, had published *The Myth*

of *Mental Illness* (1961), in which he argued that there were no mental illnesses—that schizophrenia (for example) simply wasn't real in the way that cancer was real. Michel Foucault's passionate *Madness and Civilization*, which argued that psychiatry was modern society's attempt to corral and control inner experience, had been available in English since 1964. The anti-psychiatry movement was in full swing. The profession was in real danger of losing all credibility.

Psychiatry fought back. In 1980 the American Psychiatric Association published the *Diagnostic and Statistical Manual of Mental Disorders*, third edition, more commonly called "DSM-III." The two previous DSMs had been slight, spiral-bound notebooks not taken too seriously by clinicians. In those earlier volumes the diagnostic ancestors of the current psychiatric labels are clearly marked—but they are adjectives, not nouns. There is a "schizophrenic reaction," not "schizophrenia." The language is distinctly psychoanalytic. The "psycho-neurotic disorders," for instance, are "anxiety reaction," "obsessive-compulsive reaction," and "depressive reaction" rather than (as in *DSM-III*) "generalized anxiety disorder," "obsessive compulsive disorder," and "major depression." The 1952 manual (the first one) described all these problems this way: "The chief characteristic of these disorders is 'anxiety,' which may be directly felt and expressed or which may be unconsciously and automatically controlled by the utilization of various psychological defense mechanisms."<sup>52</sup>

*DSM-III* was a fat book. There were many more diagnoses, they were more precisely detailed, and they were presented with a panoply of science. The psychodynamics had been expunged. In their place stood clear-cut (well, more than before) lists of criteria, often with inclusion rules: five of the following nine, eight of the next sixteen. If a patient met the criteria, the patient had a mental illness. If the patient did not, she or he did not. The patient's personal history—his or her ambivalence, toilet training, basic trust, dependency, whatever—was irrelevant. From the vantage point of *DSM-III*, it didn't matter how the patient had become ill or why. What mattered was whether the patient met the necessary number of criteria, which could be determined (more or less) by a short interview. All of a sudden, there was a sharp, clean dividing line between mental health and illness.

And that line was determined by science. These diagnoses were based on what anyone could observe or determine in an initial interview (more or less—actually, using the manual required considerable skill), and the committee that came up with the lists of criteria went to great lengths to demonstrate that

different people would give the same diagnosis to the same patient. There was push-back. In a bracing book called *The Selling of DSM*, two social scientists accused Robert Spitzer, the leader of the task force that came up with the manual, of snowing the field with illusory statistics.<sup>53</sup> They undoubtedly had a piece of the truth. And yet, it is also clear that the new categories actually were more specific than the older ones. Here is the *DSM-II* overview of schizophrenia:

This large category includes a group of disorders manifested by characteristic disturbances of thinking, mood and behavior. Disturbances in thinking are marked by alterations of concept formation which may lead to misinterpretation of reality and sometimes to delusions and hallucination, which frequently appear psychologically self-protective. Corollary mood changes include ambivalent, constricted and inappropriate emotional responsiveness and loss of empathy with others. Behavior may be withdrawn, regressive and bizarre. The schizophrenias, in which the mental status is attributed primarily to a *thought* disorder, are to be distinguished from the *Major affective illnesses* . . . which are dominated by a *mood* disorder. The *Paranoid states* . . . are distinguished from schizophrenia by the narrowness of their distortions of reality and by the absence of other psychotic symptoms.

And schizophrenia, “simple type”:

This psychosis is characterized chiefly by a slow and insidious reduction of external attachments and interests and by apathy and indifference leading to impoverishment of interpersonal relations, mental deterioration, and adjustment on a lower level of functioning.

Now consider this one from *DSM-III*:

#### DIAGNOSTIC CRITERIA FOR A SCHIZOPHRENIC DISORDER

##### A. At least one of the following during a phase of the illness:

- (1) bizarre delusions (content is patently absurd and has no possible basis in fact), such as delusions of being controlled, through broadcasting, thought insertion, or thought withdrawal
- (2) somatic, grandiose, religious, nihilistic or other delusions without persecutory or jealous content if accompanied by hallucinations of any type
- (3) delusions with persecutory or jealous content if accompanied by hallucinations of any type
- (4) auditory hallucinations in which either a voice keeps up a running commentary on the individual's behavior or thoughts, or two or more voices converse with each other

- (5) auditory hallucination on several occasions with content or more than one or two words, having no apparent relation to depression or elation
- (6) incoherence, marked loosening of associations, markedly illogical thinking, or marked poverty of content of speech if associated with at least one of the following:
  - a. blunted, flat or inappropriate content
  - b. delusions or hallucinations
  - c. catatonic or other grossly disorganized behavior
- B. Deterioration from a previous level of functioning in such areas as work, social relations, and self-care.
- C. Duration: Continuous signs of the illness for at least six months at some time during the person's life, with some signs of the illness at present. The six month period must include an active phase during which there were symptoms from A, with or without a prodromal phase, as defined below.<sup>54</sup>

Then there were lists of symptoms for the prodromal and residual phases of the illness, and some inclusion and exclusion criteria.

It is clear that the *DSM-III* definition narrowed the gate for the diagnosis. Before 1980, people who might later be diagnosed with post-traumatic stress disorder or multiple personality disorder or, for that matter, borderline personality disorder—now all thought to have their origin in trauma, and all new diagnoses in *DSM-III*—could easily be diagnosed with schizophrenia. After 1980, “schizophrenia” would be reserved for people who were the most sick.

Ethnographic work contributed to this effort by demonstrating that severe madness—schizophrenia—appeared with the same patterns of symptoms (delusions, hallucinations, and significant long-term impairment in functioning) in non-Western settings and was recognized as madness (and not shamanism) in those settings. In 1976, in an article in *Science*, Jane Murphy took on then-popular “labeling theory,” which often invoked Benedict’s essay on the relativity of the normal/abnormal distinction. Labeling theory argued that what was labeled as mental illness in any particular setting was merely a deviation from the normal; that the norms differed in different groups; and that people so identified internalized the disapproval and rejection of others in their group, and so habituated and perpetuated the stigmatizing behavior.<sup>55</sup> The brain, in short, had nothing to do with psychiatric illness. Murphy presented fieldwork among Yupik-speaking Eskimos on an island in the Bering



Sea and among Egba Yoruba in West Africa, added data from elsewhere, and demonstrated with detail that “Almost everywhere a pattern composed of hallucinations, delusions, disorientations and behavioral aberrations appears to identify the idea of ‘losing one’s mind,’ even though the content of these manifestations is colored by cultural beliefs.” Moreover, she said, the rate of illness—which she identified as schizophrenia—seemed more or less similar. Her subjects, she pointed out, thought that shamans learned to behave as if they were out of their minds, but they knew that the shamans were not in fact sick. They distinguished between performing madness and being mad. “Rather than being simply violations of the social norms of particular groups, as labeling theory suggests, symptoms of mental illness are manifestations of a type of affliction shared by virtually all mankind.”<sup>56</sup>

With this shift, the psychodynamic blame associated with the schizophrenogenic mother was now seen as an unforgivable sin. Such mothers, psychiatrists realized, had not only had to struggle with losing a child to madness, but with the self-denigration and doubt that came from being told they had caused the misery in the first place. The pain of this realization still reverberates throughout the profession.<sup>57</sup> Many psychiatrists still think of themselves as fighting the battle against the idea of the schizophrenogenic family—in large part, of course, because families with schizophrenic children feel so awful about their child’s illness. And because the shift away from the schizophrenogenic mother had a moral push, the new biomedical model had a moral stance. It became not only incorrect, but morally wrong, to see the parents as responsible for their child’s illness.

By the late 1980s, psychiatrists routinely condemned the idea of the schizophrenogenic mother. Indeed, the moral horror of recognizing that their own profession had aggrieved and humiliated people it had been trying to help invited psychiatrists to talk about schizophrenia as random bad genetic luck, as controllable and predictable as being struck by lightning. By then, it was known that when one identical (monogenetic) twin developed schizophrenia, the other had a 50 percent chance of developing it as well. Genetic susceptibility was thus important but not determinative. Yet the other factors involved were not understood. It was known that if a first-degree relative had schizophrenia, the chance that another might was greatly increased, just as if you go outside during a storm it increases your chance of being struck by lightning.

But we think of lightning as being unpredictable bad luck, and that was the way many psychiatrists in the 1980s wanted patients to think about schizophrenia. Most people with schizophrenia, after all, do not have first-degree relatives with schizophrenia and do not know their own genetic vulnerability. In speaking with people diagnosed with schizophrenia and with their parents, then, clinicians—earnestly trying to ward off feelings of blame and guilt on the part of the parents—emphasized the accidental and unexpected, the bad luck that the disorder should strike your family, your son. Schizophrenia was described as something inflexible and evenhanded, something profoundly uncontextual. It just happened, and parents needed support and empathy, not blame.

This shift to a biomedical model has carried its own moral cost. As schizophrenia was biologized, a mother struggling with losing a child to madness no longer had to blame herself for the tragedy. This hostile, suspicious, terrifying stranger of a son was not her fault. But as she was freed from culpability, she was also stripped of the capacity to do anything about the train wreck that had been her beloved child. And so, to a large extent, were her child's psychiatrists, whatever they might offer in the way of medication. The patients who had been removed from the category by *DSM-III* were the ones thought not to be so ill; schizophrenia had now become the diagnosis of devastation. It was thought to have the inevitable degenerating course Kraepelin had outlined for it when he first described it as different from bipolar disorder primarily because patients did not improve. In the initial aftermath of the biomedical shift in psychiatry, many psychiatrists responded to the suggestion that a person with schizophrenia can get better with the comment that if a person gets better, he or she didn't have schizophrenia in the first place.

By the 1990s, what one could call the “lightning-bolt model” of schizophrenia dominated psychiatric thinking about the illness. It had been known for a long time that poverty is associated with schizophrenia, but even in the era of psychoanalytic dominance this had been understood as a consequence of the illness, and not associated with its cause. Individuals diagnosed with schizophrenia, people reasoned, would drop in social class because they would be unable to maintain a job with a secure income. This was social “drift” theory, or “social selection” theory, made famous in the 1950s as one study after another concluded that the illness led to declining income and not the other way around.<sup>58</sup>

It had also been known for many years that African-Americans were diagnosed with schizophrenia at a higher rate than whites, and this was often attributed to clinicians' racism, not to the patient's actual illness. In *The Protest Psychosis*, Jonathan Metzl shows that in the post–Civil Rights, Black Power period, ads for antipsychotics often displayed images of angry black men who needed to be controlled. A series of papers had argued that black men were overdiagnosed with schizophrenia, and that the symptoms that might lead a black man to be diagnosed with schizophrenia might lead a white man to be diagnosed as bipolar. The lower-status person was simply associated with the lower-status label.<sup>59</sup> It is true that one of the first papers pointed out that the apparent overdiagnosis of black men with schizophrenia might be explained either by clinician bias or by the African-American man's more florid presentation of psychosis.<sup>60</sup> But many later papers argued strongly for clinician bias.<sup>61</sup>

Work on the health status of immigrants seemed to further confirm this sense that if more schizophrenia is identified in some populations, that identification is the result of clinician bias, and not of medical reality. A famous epidemiological survey published in 1962 as the “Midtown Manhattan Study” had included Puerto Ricans in its database. Its author admittedly identified a high number of people as struggling with psychiatric illness (23% of all people were judged “impaired”). However, not a single first-generation Puerto Rican was judged to be “well.”<sup>62</sup> Then the “Epidemiological Catchment Area Study,” a major community survey of over 18,000 household residents and over 2,200 institutional residents in the 1980s, found no differences in the prevalence of schizophrenia across ethnic groups, at least across whites, Hispanics, and African-Americans. Those earlier 1962 findings were attributed to clinician bias.

But as a newly biomedical psychiatry has been stripping social origin from the cause of illness, medicine has been putting it back in. The new “social epidemiology” has demonstrated that there is a social gradient to health: your body's basic health rises, on average, as you rise through the social classes. Those results are not the consequences of poor health habits but of some complex mixture of status, neighborhood, income, education, and population. Social position affects both when you die and how sick you get: in general, the higher your social position, the healthier you are. It turns out that your felt sense of relative social rank—literally, where you draw a line on an abstract ladder to show where you are with respect to others—predicts many health

outcomes, including depression, sometimes even more powerfully than objective socioeconomic status alone.<sup>63</sup>

Moreover, in recent years a new model of treatment has arisen in the United States: the “Recovery Movement,” which holds that serious psychotic disorder is not a chronic condition, but that people can return to productive, functioning lives. (Joel Braslow has argued that the model gained traction because managed-care companies recognized the high cost of providing treatment and supportive community services to individuals with a chronic illness.)<sup>64</sup> To be clear, there is no expectation that everyone who falls ill with schizophrenia will return to paid work. As Larry Davidson and his colleagues define its goal, Recovery “identifies and builds upon each individual’s assets, strengths, and areas of health and competence to support the person in managing his or her condition while regaining a meaningful, constructive, sense of membership in the broader community.”<sup>65</sup> The Recovery Movement sets out to eschew what can be seen as a paternalistic, infantilizing attitude toward chronically ill patients, and promotes personal accountability. Typically, Recovery programs use client (or “peer”) counselors and leave judgments about life choices more in the hands of the clients rather than the clinicians.

Since the implementation of the Recovery model on a large scale is less than a decade old, it is not clear what the social costs and benefits of this paradigm will be for the lives of those with schizophrenia and those who care for them. Recent observers have pointed out that in practice, adopting a Recovery model can involve a superficial redescription of existing treatment—as Kim Hopper has pointed out, Recovery’s promise has been greater than its institutional imprint—and it has a somewhat naive disregard of the cultural expectations around independence and self-sufficiency.<sup>66</sup> Nevertheless, the fundamental commitment of the Recovery Movement is that the way we imagine and understand mental illness shapes the way those who are ill respond to their condition.

These days, the schizophrenogenic mother is long gone, and family dysfunction is seen as the natural result of having a wildly irrational and hostile child in the midst of an otherwise normal family. A group of researchers in England identified a pattern of family emotional style, called “expressed emotion,” which consisted of hostility, critical comments, emotional over-involvement, lack of warmth, and lack of positive comments and which, when identified in a family, significantly predicted the relapse of patients discharged to their

homes following hospitalization.<sup>67</sup> While some early observers argued that these kinds of hostile comments might generate a schizophrenic “response,” these days many observers believe that expressed emotion represents a consequence, rather than a cause, of schizophrenia.<sup>68</sup>

And now there is epidemiological evidence, mostly from Europe, that social factors increase the incidence of the diagnosed illness. Sophisticated studies, using the new, narrow, post-*DSM-III* diagnostic category or its equivalent, have shown that schizophrenia is associated with the social class of one’s father (and presumably of one’s birth), the risk increasing as the class declines.<sup>69</sup> It is associated as well with urban living.<sup>70</sup> The risk increases with what is called “ethnic density”: the incidence of schizophrenia among nonwhite people rises as their presence in their neighborhood begins to fall. If your skin is dark, your risk for schizophrenia rises as your neighborhood whitens, whether you live in the United States or in London.<sup>71</sup> Most strikingly, the risk of schizophrenia for immigrants to the United Kingdom rises sharply, an effect that—like these other effects—has now been shown in so many papers by so many researchers and with such methodological care that it cannot be explained away by clinicians’ racial bias.<sup>72</sup> Those who arrive in England from the Caribbean or whose parents were born in the Caribbean have a much higher incidence (number of new cases within a specific period) of schizophrenia and of other psychotic disorders than whites, even adjusting for social class and age.<sup>73</sup> Black Africans who immigrate to England have a similarly elevated risk, while South and East Asians have an elevated risk but a lower one.<sup>74</sup>

This is not genetics: the risk of schizophrenia in the countries of origin seems to be no higher than it is for whites in Europe.<sup>75</sup> It is not that only sick people migrate: the effect holds for Surinamese patients in the Netherlands, where nearly half the population of Surinam has arrived.<sup>76</sup> And the risk actually increases for the second generation of these immigrants.<sup>77</sup> Again: the anthropologist’s temptation is to look for clinical bias. But the sheer number of these studies, combined with the powerful evidence that social status affects health, should lead us to look not just for bias, but for the way that discrimination gets under the skin. As the editors of *Society and Psychosis* have remarked, with these findings psychiatry has “rediscovered its roots.”<sup>78</sup> Social conditions and experiences over the life course really count, even in the development of what seems to be among the most organic of psychiatric disorders.<sup>79</sup>

So it should not be surprising that cultural difference shapes the course and outcome of schizophrenia in developing and developed countries. In a

TABLE 1    EPIDEMIOLOGICALLY IDENTIFIED RISK FACTORS

<b>Immigration</b>	Risk is particularly high for immigrants to Europe from predominantly dark-skinned countries; risk is higher for second generation than for first generation; risk is independent of social class
<b>Economic social adversity in childhood</b>	Risk increases with parental unemployment, single-parent household, adults on social welfare benefits
<b>Socioeconomic status</b>	Risk increases with lower socioeconomic status at birth and even at parent's birth
<b>Urban living</b>	Risk increases with urban dwelling and seems to increase the longer time is spent in cities
<b>Ethnic density</b>	Risk increases as ethnic density declines
<b>Social adversity in childhood</b>	Risk increases with physical, emotional, and sexual childhood abuse

1973 World Health Organization (WHO) study, the “International Pilot Study of Schizophrenia” (or IPSS), researchers had found that two years after initial contact and identification, patients looked better in Africa and India than they did in sites scattered throughout the West. But the results were decades old, some of the data were dubious, people were identified at different stages of their illness, and clinicians had used an older and more capacious pre-*DSM-III* definition of schizophrenia. So the study was redone, this time with a treated incidence sample (the DOSMeD). That is, researchers set out to actively identify people presenting for the first time with the symptoms of serious psychotic disorder not only at mental health services, but also at primary care settings, police stations, jails and prisons, traditional healers, and religious shrines. There were twelve research sites in ten countries, a stricter diagnostic category, a clearer method, and a more careful analysis.<sup>80</sup> At the two-year follow-up, patients in developing countries had experienced significantly longer periods of unimpaired functioning, and complete clinical remission was far more common (although proportions of continuous unremitting illness were similar).

Results from a major reanalysis of both studies (along with several other study cohorts, from India, China, and Germany) were reported in 2001 (the ISOs).<sup>81</sup> A fuller account, under the editorship of the anthropologist Kim Hopper, was published in 2007. It involved original data collection with well over a thousand new interviews, eight hundred of which were with people who had been followed since their first episode of illness. The team used a rigorous method of case identification, common data-collection instruments, and

common analytic strategies across ten countries and fourteen sites. The results held up, despite the concerns, the criticisms, and the limitations of the data. No matter whether you look at symptoms, disability, clinical profile, or the ability to do productive work, roughly 50 percent more people do well after a diagnosis of schizophrenia in the developing world than they do in the developed world.<sup>82</sup>

But the “developing world” in this third reanalysis and study (the ISoS) was mostly India, and in particular two centers: Chandigarh in the north, which took part in the early WHO surveys; and Chennai in the south, which did not but had comparable data. The Chennai data are particularly impressive, not only because the researchers are consistent, the follow-up rates are excellent, and the diagnostic criteria are strict, but also because Chennai is not a romantic rural paradise. It is, as Hopper remarks, the “great, teeming, postcolonial, sectarian-riven complicated place that is India” at its most urban and chaotic.<sup>83</sup> Researchers identified ninety first-contact and first-episode patients who met ICD 9 criteria for schizophrenia (International Classification of Diseases; these are much like the *DSM-III* criteria, except that the period of disturbance need last only one month and not six). Ten years later, 76 patients remained in the sample (nine had died, four by suicide). Two-thirds of them were symptom free, and they remained symptom free and medication free even ten years after that, twenty years after first contact.<sup>84</sup> This was significantly higher than the overall rate for the ISoS study. (The most striking news here, to be clear, was the high rate of recovery in all groups, including those in the developed world.)<sup>85</sup>

Because of this focus on India in the third study and reanalysis, some still challenge the claim that schizophrenia has a better prognosis in the developing world.<sup>86</sup> Yet the observation that people with schizophrenia have an easier time of it outside the West has long been present in the ethnographic literature. M. J. Field thought that it was easier to be a person with schizophrenia in rural Africa than in London: people were less afraid of the ill person (because they knew him or her personally), and a man who didn’t work seemed less out of place in an agricultural community. Field was not alone. The great anthropologist Meyer Fortes and his wife Doris Mayer, a psychiatrist, returned to his field site in Ghana thirty years after his first visit. He thought that more of the Tallensi were seriously psychiatrically ill than on his first visit (when he remembered almost no one who was mad), but his wife was more impressed that the psychoses seemed more benign than the ones



she saw in Britain.<sup>87</sup> Nancy Waxler found that in Sri Lanka, psychoses seemed to be shorter-lived and more easily cured.<sup>88</sup>

There does appear to be a higher percentage of people in developing countries who do not really have schizophrenia, as it is commonly understood, but a form of psychosis from which people recover and return to baseline functioning.<sup>89</sup> Non-affective acute remitting psychosis (NARP), an illness characterized by acute onset and complete remission, resembles schizophrenia enough that a clinician might diagnose it as schizophrenia. Patients become suddenly and acutely psychotic, and then just get better. (Field noticed such patients among her Ashanti sample.) Moreover, it has also become clear that there are far more psychotic-like experiences, for example hallucinations, in the apparently normal population than we realized, and that the rates of these phenomena vary from culture to culture.<sup>90</sup> It may be more acceptable to respond to stress with psychotic hallucinations outside of a Western setting.

At the same time, NARP and brief psychotic reactions do not explain the WHO results. They do not explain the Chennai data, and investigators found that in the WHO studies, some of the developing-country patients who looked worst at the beginning were among the group that looked best at the end. “The more pointed challenge posed by ‘non-affective acute remitting psychosis’ . . . also failed to pan out.”<sup>91</sup> Hopper and Wanderling concluded from their reanalysis that NARP was indeed more common among the cases labeled schizophrenic in the developing than in the developed world.<sup>92</sup> Nonetheless, when subjects who experienced single-episode psychosis were dropped from the analysis entirely, the recovery rates dropped—but still favored the developing world.

So it does seem as if the WHO results are due to what we might call, following Janis Jenkins and Martin Karno, the “black box” of culture—the immensely complicated ways that people live in their skins in different social settings.<sup>93</sup> Hopper points out that in the discussions around the outcome differences, “culture” almost always refers to non-Western settings: as he remarks, “‘culture’ has been a mock-elegant way of referring to ‘there’ as opposed to ‘here.’”<sup>94</sup>

Why should people with schizophrenia and other serious psychoses do better in India? Among the factors most commonly discussed are these:

- a. In India, the family remains fully involved in the treatment, unlike in America.<sup>95</sup>

- b. In India, unlike in America, ascribed family roles are important to one's social status and sense of self; patients do not have to be primary breadwinners or primary caretakers to be considered valuable members of the household; people may live in joint families.<sup>96</sup>
- c. In India, unskilled and semiskilled work, such as agriculture and home-based artisan piecework, may be less stressful and less demanding than entry-level jobs in America, which are often in fast-paced, high-social-contact settings like McDonald's.<sup>97</sup>
- d. In India, fewer families exhibit expressed emotion than in America.<sup>98</sup>
- e. In India, while psychiatrists diagnose schizophrenia in patients, they do not use the label in interacting with the patients and the patients do not use the label for themselves.<sup>99</sup>
- f. In India, the auditory hallucinations of persons with serious psychotic disorder may be more benign.<sup>100</sup>
- g. In India, there are subtle psychological features that may shape an individual's reactivity: psychotic hallucinations may seem more similar to standard religious practice than they do in America; there may be a different understanding of self-coherence; there may be a different degree of stigma attached to mental illness as, for example, compared to divorce; there may be different expectations of professional achievement; and there may be different degrees of comfort with allopathic medicine.<sup>101</sup>

In a 1997 review of decades of ethnographic work, Byron Good argued that the following four hypotheses deserve particular attention:<sup>102</sup>

- a. *The local cultural interpretation of mental illness.* Is the illness understood to be inevitably chronic? A broken part of the essential self? Or a passing storm?
- b. *The presence of an extended family.* Is there another breadwinner? Are there other people at home to help? And is help provided, or is the person locked in a back room and kept from inquisitive eyes to preserve the family's honor?
- c. *Industrial-age labor vs. agricultural or nonwage labor.* Can the ill person work? Can he or she contribute? In a world in which wages reward performance, someone with illness will be less easy to employ.

- d. *The basic social environment.* The difference in living conditions—urban squalor as opposed to the traditional family home—may ultimately be more important to outcome than any actual treatment provided.

There is another interpretation, not widely discussed in the psychiatric literature of this debate but perhaps equally important: that the normative treatment for schizophrenia in American culture may significantly make things worse, and possibly even turn psychotic reactivity (the possibility of a brief psychotic reaction) into chronic clienthood, and that it does so by repeatedly creating the conditions for demoralization and despair, and for what we will call “social defeat.” In other words, the culture “here” may be as important as the cultural other “there.”

So the deep problem of the variable vulnerability to schizophrenia is embedded in the classic issues of anthropology: kinship, class, personhood, poverty, meaning. We have data and theory to contribute. And by attending to the ways in which central anthropological concerns about kinship, care, relationality, and cultural notions of selfhood and personhood shape the way that illness is identified, experienced, and treated, we hope to make it clear that schizophrenia cannot be understood fully without its cultural context.

Epidemiologists track numbers. Ethnographers use the only method that can reliably and validly identify the features of the social world that are real and salient for subjects. Our aim in this volume is to present case studies that give detail and depth to these hypotheses about our most troubling madness. We hope that by doing so, we will provide the material to help us tease out the complex ways in which culture shapes illness—and, perhaps, eventually nudge the treatment of serious mental illness toward an easier outcome. We believe that understanding the culture in which schizophrenia unfolds may have clear and consequential implications for treatment. We hope to show that here.

