

## 12 Reflections

### Hearing Voices How Social Context Shapes Psychiatric Symptoms

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From the first, psychiatry has been torn between understanding serious mental illness as a dramatic expression of the strains of ordinary living and as a disease, as something fundamentally different from the everyday, more like pellagra or diabetes than sadness.

Freud thought that mental illness arose out of emotional conflict. To be human was to live with impulses we could not acknowledge and losses we could not bear. Ordinary life was thus a habituated management of personal pain. “Depression” and “psychosis” were simply names we gave to patterns of self-management which were so woefully inadequate that those who used them could not function. Diagnosis was not particularly important. What mattered to Freud was the clinician’s ability to recognize particular patterns of interpreting emotions and responding to them – emotion–motivation–behavior bundles, if you will – and to help the person who enacted these patterns recognize them as patterns, as choices, which could be changed. The psychoanalyst empathized with a patient as a person like himself, struggling with similar burdens of thwarted love and future death, and the psychoanalyst understood that whatever medicine the person might take, what truly mattered in the end was the ability to help that person to recognize why they responded to people as they did.

Kraepelin was born the same year as Freud (1856) in a similar European milieu. But when Kraepelin looked at people with serious mental illness, he saw disease. He set out to identify specific diseases by identifying symptoms that marked people off as different, and distinguishing these diseases from one another by symptom cluster, illness trajectory, and outcome. He paid attention to family history, not because he thought that families might socialize similar responses to life’s challenges, but because he thought that these diseases might be heritable. He assumed that our hope of treating them was to distinguish them clearly enough to understand them. The biomedically oriented psychiatrist within the Kraepelinian tradition empathizes with a patient as a person different from himself, struggling with an alien and

unchosen burden, like someone who has been diagnosed with cancer. She understands that whatever compassionate care she may offer, her most important intervention is to prescribe the right medication. She puts her faith in science.

Freud's model dominated American psychiatry until the last decades of the twentieth century, when it was replaced more and more by a Kraepelinian one. And because the shift was born out of disappointment and frustration with an approach that, in the end, did not seem to cure serious mental illness, the biomedical psychiatry that emerged in response presented itself as an entirely new beginning. As Nancy Andreasen wrote in one of the manifestos of the period:

Psychiatry now recognizes that the serious mental illnesses are diseases in the same sense that cancer or high blood pressure are diseases. Mental illnesses are diseases that affect the brain, which is an organ of the body just as the heart or the stomach is. People who suffer from mental illness suffer from a sick or broken brain, not from a weak will, laziness, bad character or a bad upbringing. (Andreasen, 1984, p. 8)

This book, explained one of its blurbs, “chronicles a revolution in psychiatry that has returned this battered specialty to its birthplace in medicine.”

It is now clear that the simple biomedical approach to serious psychiatric illness has failed in turn. As Raz and McDonald point out, “decades of work on cognitive, molecular and systems neuroscience have taught most scientists a lesson in humility. Despite an enormous investment in research with an emphasis on the neural correlates of typical and atypical behavioral “phenotypes,” breakthroughs are sorely lacking.” At least, the confidence that these illnesses would be understood as brain disorders with clearly identifiable genetic causes and clear, targeted pharmacological interventions – what some researchers call the bio-bio-bio model: brain lesion, genetic cause, pharmacological cure – has faded into mist. To be sure, it would be too strong to say that we should no longer think of a condition such as schizophrenia as a brain disease. One often has a profound sense, when confronted with a person diagnosed with schizophrenia, that something has gone badly wrong with the brain.

Yet the outcome of two decades of serious psychiatric science is that schizophrenia now appears to be a complex outcome of many unrelated causes – the genes you inherit, but also whether your mom fell ill during her pregnancy, whether you got beaten up as a child or were stressed as an adolescent, even how much sun your skin has seen. It's not just about the brain. It's not just about genes. In fact, in terms of causation,

schizophrenia looks more and more like diabetes. A messy array of risk factors predisposes someone to develop diabetes: smoking, being overweight, collecting fat around the middle rather than on the hips, high blood pressure, and family history. These risk factors are not intrinsically connected to one another. Some of them have something to do with genes, but most of them do not. In fact they hang together so loosely that physicians now speak of a metabolic “syndrome,” something far looser and vaguer than an “illness,” let alone a “disease.” Psychiatric researchers increasingly think about schizophrenia and other psychiatric illnesses in similar terms.

And so the NIMH has shifted the scientific emphasis back to the ways in which people with serious mental illness are, in effect, more like the rest of us. The structure of the new approach to research is to search for specific alterations of specific human psychobiological symptoms, and to abandon (at least in the research) specific categories of disease. It is a remarkable experiment for a scientific field defined for decades by nosological categories.

Some see this new orientation as a mistake. Laurence Kirmayer and Daina Crafa, for example, describe it as impoverished and conceptually flawed:

Moving the search for mechanism back several steps in the causal chain to putative endophenotypes may increase the likelihood of finding certain lower-level mechanisms but it will not provide a complete explanation of how most symptoms are produced nor will it adequately address the role in psychopathology of processes of self-understanding, coping, and interpersonal communication or interaction with others. (Kirmayer & Crafa, 2014, p. 4)

This is an understandable response and many people are sympathetic to it – including many psychiatric scientists who have spent their careers focused on specific diseases and now see the scaffolding of their research ripped out from under them. Kirmayer and Crafa are particularly concerned that the emphasis on neuroscience will diminish the attention to cultural variation and social meaning in the study of mental illness.

My take is more optimistic. I think that this may be an opportunity not only for psychiatry, but for anthropology. From my reading, the RDoC criteria take seriously the basic insight of anthropology: that humans are fundamentally social animals and that the social gets under our skins in profound ways. It remains to be seen, of course, whether NIMH funding will acknowledge the contributions anthropology has to offer. Nevertheless, the shift invites us to think carefully about the specific ways in which social context shapes psychiatric illness.

Let me offer three examples of the way that social context may direct specific psychological processes and so shape psychiatric symptoms. The three chapters, other than Bilder's (Chapter 8), which form this section suggest that attention, emotion regulation, and early trauma shape symptom expression. The work that I and my colleagues have done with people with serious psychotic disorder suggests that social context may shape the way people attend to their voices; the inferences they draw about who is speaking to them, and thus, how they experience their voices emotionally; and, in general, the risk that someone will fall ill with psychosis.

When someone becomes psychotic and begins to hear voices, they often experience a wide range of auditory events (Tuttle, 1902). They hear bad voices and good voices, voices that command and voices that comment. They have internal events and external events and events that seem somehow in between. They hear hissing and murmuring and sounds that seem to fall off cars as they pass and resolve into voices. When I and my colleagues interviewed twenty people with serious psychotic disorder in each of Accra (Ghana); Chennai (India); and San Mateo (California), we found that they reported quite different experiences (Luhrmann, Padmavati, Tharoor, & Osei, in press; Luhrman, Padmavati, Tharoor, & Osei, 2014). In general, Americans hated their voices. They experienced their voices as assaults, and they reported that their voices were more violent. None of them reported their voice-hearing experiences to be primarily or exclusively positive. By contrast, many patients in Accra and Chennai described their voice-hearing experience as positive – half the patients in Accra, and over a third of those in Chennai. They more often talked about liking their voices, even when they said that the voices were mean. They did, to be sure, report voices that were caustic and told them to kill themselves – but less often.

Why would people with psychosis experience hallucinated voices differently in different social settings? There is no reason to think that the disease process of psychosis differs in these different settings. We concluded that local expectations shaped the ways that subjects were paying attention to the complex array of auditory events. In particular, we thought that local expectations about the mind – what we could call local theory of mind, the ways people understand thoughts and feelings – might alter the way people attend to sensory data. Americans imagine the mind as a separate, private place – as “bounded,” to use Charles Taylor's (2007) phrase, walled off from an external material world (D'Andrade, 1987). There are no cultural expectations that there are many supernatural beings who talk frequently. Even American Christians who embrace

a faith in which God speaks back need to learn that God will do so frequently, and they are not taught that God will speak back audibly (Luhrmann, 2012). The only meanings Americans tend to ascribe to their auditory hallucinations is that they are the signs of being “crazy,” and so it makes sense that Americans would dislike their voices, and feel them to be intrusions. This may lead them to attend more to their least pleasant experiences.

Ghanaians and South Asians, by contrast, live in social worlds in which spirits are widely understood to be present. It is not the case that the only meaning of hearing a voice is that one is crazy. The large majority of our Ghanaian subjects were Christian. They understood demons to be real, and they took God to be capable of controlling them. Our Chennai subjects were largely Hindu. They live within a social world in which Hindu spirits take command of human bodies and speak to them. There are distinctive class and caste expectations involved, but it is an evident part of this social world that invisible spirits can speak. Some of our Chennai subjects even experienced their voices as having the playfulness associated with some Hindu avatars. For both Accra and Chennai subjects, then, there would be more of a cultural invitation to attend to their most positive voices and to treat those voices as real invisible beings.

It is important to be clear that these differences in voice hearing are differences in emphasis. They are not absolute. There was no question that subjects in Chennai and Accra were ill. They knew they were ill and they would have preferred not to be ill. But they were much less critical of their voices – on average – than the Americans were.

Another striking difference in voice hearing was that in general, our American subjects did not know who was speaking to them. Subjects in Accra and Chennai were far more likely to say that they knew the real humans who spoke to them in this disembodied way. They were also more likely to say that the voices gave them good advice, and that they valued the advice – even if they did not like the voices. They were more likely to treat their voices as sources of comfort and aid during their illness, rather than as the cause of their illness, as the Americans often insisted. We concluded that differences in the culturally varied experience of the self may have shifted the way people incorporated voice hearing into their daily lives.

One of the most robust observations in cultural psychology and psychological anthropology is that Europeans and Americans imagine themselves as individuals – set apart and in contrast to others. The more “independent” emphasis of what we typically call the “West” and

the more “interdependent” emphasis of other societies has been demonstrated ethnographically and experimentally many times in many places – among them, India and Africa (Markus et al., 1997; Nisbett, 2003). This research does not suggest that people experience themselves in the same ways outside of the West; its point is that relationships with others are far more salient to the way non-Westerners (and certainly, South Asians and Africans) interpret their experience than they are to Westerners.

We believe that these social expectations about persons may shape the voice-hearing experience of those with serious psychotic disorder. The Chennai and Accra patients were more comfortable interpreting their voices as relationships, and not as the sign of a violated mind. Although there is robust evidence that emotional dysregulation contributes to the emergence of serious psychotic disorder, these data suggest that to some extent, the tendency to infer that voices are people may mitigate some of the distress. A number of studies have now suggested that psychosis has a more benign course and trajectory outside of the West, with the best data coming from India (Hopper et al., 2007). It is possible that these differences in voice hearing contribute to that more benign course and outcome.

It appears from our evidence that auditory hallucinations are not only construed differently in different cultural settings, but that their affective tone actually shifts – an observation in accord with the new cognitive-developmental model of psychotic hallucinations, which argues that cognitive bias – as well as cognitive deficit – shapes the rate, content, and phenomenology of psychotic hallucination. (Bentall et al., 2007) We suggest that everyday expectations determine (to some extent) the way subjects attend to the messy array of auditory events that occur for most people with serious psychotic disorder and, in consequence, alter those auditory phenomena: that everyday, socially shaped expectations alter not only how what is heard is interpreted, but what is actually heard. And it is possible that these differences have clinical implications (Connor & Birchwood, 2013).

The evidence that early-life adversity increases the risk of developing serious psychotic disorder, as well as suicide, is increasingly robust (Bentall et al., 2012). People who are humiliated and abused and bullied are more likely to fall ill with schizophrenia. People who are born poor or live poor are more likely to develop schizophrenia. People with dark skins are more likely to fall ill in white-skinned neighborhoods, probably (scientists think) because they feel their lesser social status more keenly. People who live urban, too, are more likely to fall ill with the condition, perhaps because they are more likely to feel socially

threatened. When life beats people up, they are at more risk of developing psychosis, and for the most part both our epidemiology and our ethnography suggest that the best way to understand social causation in schizophrenia is through the experience of social defeat: a social encounter in which one person physically or symbolically loses to another (see Morgan, McKenzie & Fearon, 2008, for summary; see also McKenzie & Shah, Chapter 13, *this volume*). And it is striking that back before the biomedical turn in psychiatry, anthropologists had argued that something like the bodily experience of social defeat explained why some societies had higher-than-average rates of schizophrenia. They saw a daily, constant grind of humiliation and rejection that – they thought – made people ill (Scheper-Hughes, 1979, Desjarlais, 1995, Hopper, 2003, Jenkins & Barrett, 2004). My own work on homeless psychotic women on the streets of Chicago makes it abundantly clear that homelessness is not the best way for our society to treat persons who are falling ill with psychosis, although evidence suggests that it is now normative for such people to experience bouts of homelessness in the United States. That work also suggests that the experience of living on the street with psychosis may exacerbate existing illness and heighten the symptoms of those who are already ill (Luhmann, 2007).

Indeed, in a forthcoming collection of case studies on the experience of psychosis, we suggest that the social patterning of increased risk of schizophrenia for some social worlds and better outcomes for others can be explained by different vulnerabilities to social defeat (Luhmann & Marrow, n.d.). Schizophrenia is not only more common in some social conditions, but its effects are mitigated in others, most notably in India and probably elsewhere in the developing world, where both its course and outcome are more benign. We argue that for all the pain in madness everywhere, there seems to be more opportunities for social defeat for a person with madness in the West.

The old bio-bio-bio world invited anthropologists to argue about whether diagnostic categories were real. The new RDoC approach invites us to think about psychiatric illness as a complex syndrome, and to ask how specific social patterns change them. That in turn invites us to think differently, not only about biology but about culture – to deessentialize it, just as we are deessentializing psychopathology. It invites us to ask not what is different about Ghanaian culture, but what it is about a specific sense of self that might be found in another culture, or an expectation about how the mind works, or what counts as trauma. It could be a remarkable opportunity to rethink our field.

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