

Update: Schizophrenia Across Cultures

Neely Lorenzo Myers

© Springer Science+Business Media, LLC 2011

Abstract The incidence of schizophrenia, as well as the symptoms, course, and outcomes for people so diagnosed seem to vary across some cultural contexts. The mechanisms by which cultural variations may protect one from or increase one's risk of developing schizophrenia remain unclear. Recent findings from transdisciplinary cross-cultural research, indicate ways that we may better understand how socioenvironmental and cultural variables interact with physiologic pathways relating psychosocial stress and psychotic symptoms, epigenetic changes, and people's use of culturally available tools to mitigate stress, in ways that may inform relevant, effective interventions for people diagnosed with psychotic disorders worldwide.

Keywords Schizophrenia · Psychosis · Psychosis continuum · Culture · Cross-cultural · Anthropology · Ethnography · Epidemiology · Epigenetics · Stress · Environment · Neurodevelopment · Migration · Urbanicity · Ethnic density · Vitamin D · Incidence · Family · Stigma · Early intervention · Social factors · Risk factors · Protective factors · Race · Family · Context · Global mental health · Psychotic disorder

Introduction

Provocative new research continues to indicate that the incidence of schizophrenia, as well as the symptoms, course, and outcomes for individuals so diagnosed seem

to vary across some cultural contexts. This article reviews literature concerning schizophrenia across cultures published since 2009. The mechanisms by which cultural variations may protect individuals from developing schizophrenia or increase their risk of developing schizophrenia remain unclear. Transdisciplinary cross-cultural research, recent findings suggest, may help us understand how socioenvironmental and cultural variables interact with physiologic pathways relating psychosocial stress and psychotic symptoms [1•], epigenetic changes, and people's use of culturally available tools to mitigate stress [2], which may then be used to inform relevant, effective interventions for individuals experiencing symptoms of schizophrenia worldwide [3••].

Culture and Symptoms

Culture and the Psychosis Continuum

Schizophrenia may be the end point of a “psychosis–proneness–persistence continuum,” with individuals who experience “nonclinical psychosis” at one end and multiple other diagnostic possibilities in between [4••]. The psychosis continuum concept builds on a survey of Western countries showing that 5% to 8% of the general population reported psychotic-like experiences (PLEs) that were typically transient, did not disrupt social functioning [4••], and met criteria for consideration as a “nonclinical psychosis” phenotype [5]. In a separate global survey, the percentage of people in the general population reporting at least one psychotic symptom varied dramatically from 1% in Vietnam to 45.8% in Nepal [6].

The neurodevelopmental expression of psychotic symptoms, some argue, may be understood as part of a

N. L. Myers (✉)
Center for the Study of Complementary and Alternative
Therapies, University of Virginia,
Charlottesville, VA 22908, USA
e-mail: neelymyers@gmail.com

common trajectory that only becomes pathological when an individual experiences repeated environmental insults that render him or her “prone” to the effects of adversity [4••]. For example, an individual who experienced childhood trauma and adolescent discrimination and who also has a genetic vulnerability may develop an overactive response to stress over time (“sensitization”) that leaves him or her more prone to psychotic experiences [1•]. In one recent longitudinal study, the persistence of auditory hallucinations (one type of PLE) for some Dutch adolescents from the general population at times led to delusional ideation, and then PLEs and delusional ideation together prompted a transition to psychosis [7]. However, one study found that even among an ultra high-risk group of young individuals who presented with psychotic symptoms, familial heritability for schizophrenia, and neuropsychological deficits associated with schizophrenia, only 19% transitioned to psychosis over an 18-month period, suggesting that even ultra high-risk groups may not be good targets for aggressive early interventions (EIs) [8]. More research will help clarify this proposed notion of a psychotic continuum and its implications.

Culture and Positive Symptoms

Positive symptoms of schizophrenia may also vary cross-culturally. In one study of 1,080 patients who met *DSM-IV* criteria for schizophrenia in 7 countries, the highest 1-year prevalence rates of auditory and visual hallucinations were identified among West African participants (e.g., 90.8% in Ghana, 85.4% in Nigeria, 53.9% in Ghana, and 50.8% in Nigeria, respectively) [9]. The lowest 1-year prevalence rate for auditory hallucinations in this sample was found in Austria (66.9%), and for visual hallucinations in Pakistan (3.9%) [9]. Hallucinations and delusions may even vary regionally within the same country, suggesting that cultural effects may not necessarily be delineated by geopolitical boundaries [10].

Culture and Negative Symptoms

Culture may also interact with the negative symptoms of schizophrenia. In another 20-year follow-up study in the United States of participants meeting criteria for “deficit schizophrenia,” or people who meet criteria for schizophrenia and also display multiple enduring negative symptoms for more than 1 year, only 13% showed one or more 1-year periods of global recovery compared with 63% of non-deficit schizophrenia patients [11•]. Another recent study found Swedish participants living independently more often and for longer periods of time than their cognitive and functional counterparts in New York because the process of obtaining and keeping housing was less cognitively demanding [12]. These findings, the authors claim, indicate

“divergent real-world outcomes among individuals who show evidence of the same levels of ability and potential” [12]. Taken together, findings from Chicago, New York, and Sweden raise interesting questions. For example, does one’s ability to recover from the effects of schizophrenia symptoms depend in part on how cognitively challenging one’s cultural context may be to navigate and the level of neurocognitive deficits an individual possesses? Further research into the interplay of cognitive capabilities and social context is needed. New technologies may help us revisit this old question in new ways.

Culture as Risk

Incidence, or an individual’s risk of developing schizophrenia over time, also appears to vary across cultural context. African Americans were recently found to be three times more likely to be diagnosed with schizophrenia than European Americans [13]. Migrants who are ethnic minorities in their host countries have a higher incidence of schizophrenia, with the highest increase in relative risk found among darker-skinned migrants to lighter-skinned nations [14••]. Increased risks in one meta-analysis held even for second-generation immigrants, strengthening arguments that social context rather than pre-migration events, the effects of migration itself, or even diagnostic bias might completely explain these findings [14••]. Diagnostic bias and errors, urbanicity (living in a city as opposed to a rural setting), migration, socioeconomic status, adverse life events, and cumulative social disadvantage may all play a role as cultural context imposes “risk” on individuals over time. Potential vitamin D deficiency among darker-skinned migrants to northern climates and its possible association with psychotic symptomatology also warrant discussion.

Ethnic Minorities and Diagnostic Errors?

Disproportionate rates of schizophrenia diagnoses in ethnic minority populations, some claim, may be explained by cultural norms that promote the erroneous reporting or diagnosis of psychotic experiences in ethnic minority groups. In *The Protest Psychosis*, for example, psychiatrist Metzl [15] questions the role of “institutional racism” in the increasing diagnosis of “schizophrenia” in the 1960s and 1970s among African American males, which occurred at the same time that prevalent cultural norms interpreted the African American struggle for civil rights as aggressive and paranoid [15]. Zandi and colleagues [16•] also recently questioned the role of clinician bias in the reportedly increased rates of psychosis among Moroccan immigrants to The Netherlands. Using a “more sensitive” and “culturally adapted” tool, Zandi and colleagues [16•] rated

symptoms of "Western" psychotic disorder such as hearing voices and seeing things or dead people as "not significantly present" if these experiences did not negatively impact the patient's functioning. Rather, the authors argued, such symptomatology was best attributed to a dissociative possession state common in Moroccan culture that has been widely misinterpreted by Western psychiatrists. Using their altered diagnostic tool, the statistical differences in the risk of developing schizophrenia between Moroccan immigrants and Dutch natives disappeared. More research confirming these findings is needed. In another relevant study, 10% of surveyed Latinos living in the United States self-reported psychotic symptoms such as delusions and hallucinations that were clinically meaningful (i.e., associated with increased suicidal ideation), but only 7% met Western diagnostic criteria (Structured Clinical Interview for *DSM-IV*) for psychotic disorders [11•]. Among Latino patients, the authors suggest, psychotic symptoms may best be interpreted as culturally acceptable ways to signal "interpersonal vulnerability" in individuals whose psychotic symptoms were typically associated with being unmarried and with exposure to trauma.

Psychiatrists' potential misunderstandings of the cultural salience of psychotic symptoms such as those documented by Metzl [15], Zandi and colleagues [16•], and Lewis-Fernandez and colleagues [17], suggest that increased rates of schizophrenia in ethnic minority populations may at times reflect errors in reporting or diagnosing psychotic experiences rather than variance in true disease expression. Clinicians should aim to be conscious of the cultural relevance of seeming psychotic symptoms in various ethnic groups that could lead to an inaccurate diagnosis of psychotic disorder based on insufficient information. Psychotic symptoms, while clinically significant, may be manifestations of culturally acceptable reactions to trauma exposure, dissociation, and anxiety [11•]. Clinicians should contextualize diagnoses with a strong orientation toward the patient's own cultural understandings of his or her symptomatology and the meanings that the symptoms have for that patient [18]. At this time, however, there is no conclusive evidence to suggest that all heightened incidence rates are solely a function of widespread misdiagnosis or clinician bias. A recent meta-analysis consistently finding increased risks of schizophrenia incidence in nearly every migrant group analyzed across a variety of migrant groups and settings suggests that social context likely plays a role in the variability of psychotic disorder across populations [14••].

Cultural Context and Gene–Environment Interactions

Others hypothesize that in various cultural contexts, interactions between genetic vulnerability and the multiple

risk factors posed by certain social and environmental conditions across the life course—including but not limited to discrimination—may lead to psychotic symptoms, with the combination of gene–environment interactions and their psychological and epigenetic effects (or alterations in gene expression during an individual's lifetime) being more potent than the influence of genes or environment alone [19]. For example, one study found that some aspects of neurocognitive functioning in African American families were both heritable and associated with schizophrenia [20•]. Perhaps heritable neurocognitive features associated with schizophrenia in combination with the challenging cultural context faced by some African Americans—and the epigenetic changes this may produce across one's life course—explain some of the elevated risk of psychosis beyond clinician bias and reporting errors (which likely at times play a role [15]) for this population. Furthermore, in a case–control study, cumulative social disadvantage (i.e., the adding up of instances of unemployment, discrimination, adverse life events, parental separation before age 16 years, and childhood trauma across the life course) was linked to higher rates of PLEs in the Black Caribbean, but not Black African, ethnic population [21]. These studies suggest that more than clinician bias against people of a specific skin color may be involved. A discussion of other hypothesized contextual risk factors from the literature follows, including ethnic density, socioeconomic status, urbanicity, adverse life events, and cumulative social disadvantage.

Ethnic Density

Living in areas of low "ethnic density" seems to increase an individual's risk of developing schizophrenia. For example, a recent study in the United Kingdom found that when black people comprised less than 25% of their neighborhood's population, their relative risk of developing schizophrenia increased nearly threefold, but that finding became statistically insignificant if their neighborhood was 25% or more black [22]. Similarly, in a Swedish multilevel, longitudinal study, Zammit and colleagues [23] found that foreign-born children whose school group had few other foreign-born children were also at higher risk of developing psychotic disorder, and that this risk diminished as their school group included more foreign-born individuals. The reverse applied to children with two Swedish parents who were in predominantly foreign-born groups, so that their risk of developing psychotic disorder increased as they seemed increasingly different from their peer group at school. Mexican immigrants to the United States also reported increased psychotic symptoms as they became more acculturated to the mainstream culture (e.g., no longer speaking Spanish) and more socially isolated from their ethnic communities [17]. All these studies indicate an

increased risk associated with living in a cultural context in which one may feel isolated from or seem different than the majority population. In 18 of 20 studies in one systematic review (all conducted in Western countries), urbanicity also increased an individual's risk of psychosis [24]. Migrants living in urban areas in which social groups may be more fragmented are likely to be at particularly increased risk, although more research is needed [24].

Socioeconomic Status

Children in Sweden without a biological risk of psychosis who were adopted into families with a disadvantaged socioeconomic position (e.g., parental unemployment, single-parent household, living in an apartment) had an increased risk of developing psychosis, and those with both genetic liability and a disadvantaged childhood socioeconomic situation [25] were substantially more at risk. Indebtedness also increased the risk of developing schizophrenia significantly, especially for men [26]. A recent study of socioeconomic status in a large Israeli cohort found no increased risk of schizophrenia across parental socioeconomic status or education level at birth except for a moderately increased risk for those having a father in the lowest socioeconomic bracket at birth [27]. The Israeli study suggests that there may be a compounding risk for those born into very poor circumstances in which adverse events are more common.

Adverse Life Events and Cumulative Social Disadvantage

Increased risk of psychotic disorder has long been associated with adverse life events and social disadvantage. In urban Tanzania, researchers recently found that experiencing two or more stressful life events in the past year led individuals to have an increased risk of developing schizophrenia [28]. Physical abuse in childhood among British women made them twice as likely as controls to report psychotic symptoms (with lesser effect for sexual abuse) [29]. Exposure to trauma in South Africa [30] and exposure to trauma with intention to harm (e.g., bullying) as a child led to an increased incidence of psychosis in Britain, even with controlled genetic liability for psychosis [31].

The Vitamin D Factor

Increased rates of psychosis found in darker-skinned migrants, who process vitamin D less efficiently due to the weak sunlight found in northern latitudes, may also reflect an association between vitamin D deficiency and psychotic symptoms. While “normal” levels of vitamin D have not been established, and the evidence remains inconclusive [32], a recent study conducted in Denmark

associated both low and very high levels of neonatal vitamin D with later risk of schizophrenia [33]. Based on their data, the authors predicted that optimizing vitamin D levels for pregnant mothers may prevent up to 44% of all schizophrenia cases in Denmark. However, this study accounts for an in utero effect and may not translate to the situation of darker-skinned migrants to northern climates since cultural factors also likely play a role.

Culture as Protective?

Families and Stigma as Risk

Rates of both anticipated and experienced stigma are consistently high across cultures with regard to mental illness [34]. Cross-cultural expressed emotion (EE) research in European American and Mexican American families suggests that when families are hostile and critical (high EE), the symptoms of their family member with schizophrenia increase and they experience higher rates of rehospitalization [35]. Anglo American families in the United States in which recovery outcomes from schizophrenia are less frequent seem to be typically more hostile and critical (high EE) [36].

Families and Protective Stigma

Families around the world may also protect their seemingly ill relatives via exertions motivated by their own stigma. For example, Yang and colleagues [37] described families' use of the term *xiang tai duo* (“excessive thinking”) as an alternative explanation for schizophrenia symptoms in Mandarin China. They argue that such linguistic devices may have “normalizing power” that allows families to recognize behaviors as socially unusual but not too abnormal (e.g., “sometimes we all think excessively”), so that psychotic behaviors would not be publicly interpreted as pathological. In southern India, where recovery outcomes typically have been high, families also used excessive thinking as an alternative explanation for schizophrenia [38]. Indeed, family members, the general public, and patients comfortably held multiple contradictory models of psychotic behaviors. For example, they might agree that there was something “abnormal in the brain” without calling it the result of a disease, or patients might refuse to accept the illness as something wrong with them and instead attribute it to external spiritual factors [38]. Such strategies may contribute to the increased recovery outcomes observed in some cultural contexts, as families work behind the scenes to offset stigma and preserve an ill family member's social standing and local “moral worth” [37, 38].

Families and Early Intervention

Family involvement in and stigmatization of psychiatric care is also relevant to the context of EI for psychosis, and some researchers speculate that families' alternative explanations of psychotic symptoms (e.g., excessive thinking) may prolong the seemingly critical duration of untreated psychosis (DUP). Longer DUP (although what constitutes long has yet to be determined) may be associated with poorer response to antipsychotic treatment and increased disability in both high-income (HI) [39] and low- to middle-income countries [40]. For example, a study in Vellore, India, identified a significant association between DUP and outcome, with longer DUP increasing the likelihood of remission over the course of 1 year [41].

Great debate has arisen over when and how to use EI programs (typically a combination of antipsychotic medications and psychoeducation) to possibly shorten DUP and ideally prevent full-blown psychosis. Some argue that intervening early is good evidence-based care [39], but there is no conclusive evidence to indicate that shortening DUP with EI works, or that shortening DUP improves prognoses. Others claim that EI may expose patients unnecessarily to the known risks of taking antipsychotic medications (e.g., the rapid weight gain associated with the increased risk of adult obesity, cardiovascular disease, and metabolic disorder; ongoing stigma despite the discontinuation of psychotic symptoms; debilitating side effects; and difficulties securing health insurance) [42, 43].

In addition, shortening DUP leaves people without options to explain their illness in nonpathologizing ways (e.g., excessive thinking) that may be protective over time. In one study of a woman with a 5-year DUP, we learned how the patient (an Indian migrant to Canada) used the extra time afforded her by a longer DUP to do the hard work of making sense of her psychotic symptoms with her husband in a way that strengthened their shared ability to understand her symptoms as abnormal but not pathological. This in turn strengthened their relationship, helped them feel confident about having children together, and made her no less likely to accept medications when the psychosis became overwhelming [18]. Arguably, a longer DUP was helpful for her and her husband when it came to sorting out these difficult, profoundly life-shaping issues before she received a diagnosis that may have otherwise greatly interrupted her life and marriage [18].

People diagnosed with schizophrenia in south India, who at times experience some of the best schizophrenia recovery rates in the world, may also have longer DUPs, although this needs to be confirmed with research. One recent study found that 50% of people diagnosed with schizophrenia in south India were untreated and living at home [44]. Would EI programs interrupt the care that exists for them now

thanks to nonpathologizing explanatory models that help lead to better social outcomes? For example, Bedouin families became more likely to abandon their family members once they were taught to understand schizophrenia as a brain disease; in contrast, thinking of psychotic disorders as spiritual afflictions inspired community members to help them [45]. Teaching biomedical models of mental illness also has recently been found to increase stigma in Spain, Germany, and South Africa [45, 46].

The implications of these findings should be carefully considered in the development of global mental health agendas. Existing explanatory models of psychosis developed between people as they struggle to make sense of a relative or friend's psychotic symptoms and the caregiving practices these meaning-making efforts produce may be more protective than initially recognized. Interrupting the subtle social processes that may encourage recovery in any context with EI or anti-stigma programs that are not carefully designed for specific cultural contexts may not be prudent.

Conclusions

With such variation in the cross-cultural experience of schizophrenia, the use of the term *schizophrenia* may not adequately account for the variety (and local specificity) of symptoms, experiences, explanatory models, and outcomes described here. The widespread idea that the 1% prevalence rate of "schizophrenia" does not vary significantly across cultures also may be interpreted with caution [47]. In addition, socioenvironmental factors in schizophrenia are more modifiable than genetic factors and have a greater potential for impacting public health through prevention efforts than any known genetic variant [48]. Certainly, many factors presented here may be addressed in future interventions.

This review also suggests that analyses of culture—understood as shorthand for complex assemblages of ways of life—may better account for the social order (e.g., institutions and relationships) and the "environment" (e.g., place) while capturing the ways individuals and their families make use of culturally available meanings to make sense of their illness experience and to find relief. Cultural analyses also raise our awareness of the host of value-laden commitments that people must learn to meet in order to thrive in sometimes highly-specified local moral worlds (e.g., the Bedouin with a psychiatric disability who is more valued if he or she is experiencing spiritual attacks than if he or she has a biological illness, or the Indian son or daughter whose psychiatric disability is tolerated once he or she is married, and especially if he or she has children).

To account for culture, or elements that make life more difficult or livable for people struggling with symptoms that can accumulate to become a serious disability, researchers and

policymakers might continue to engage the skills of ethnographers. Ethnographic research, the craft of anthropologists, requires a researcher to build long-term relationships (over at least 1 year) with participants and their support networks by visiting participants' homes, meeting their friends, and spending time engaged in activities that are meaningful to them. In this way, anthropologists participate with participants, and then generate richly detailed descriptions of their everyday experiences, activities, and interactions with others. In the process, anthropologists typically learn how an individual regularly pursues the tasks of his or her daily life, what is at stake for that person as he or she struggles to do so, and how pursuits and goals may shift or be shifted over time by contextually available explanatory frameworks, conditions, and events. Such data can greatly inform testable hypotheses about how to best prevent and treat psychosis. For example, a recent ethnographic analysis found that recovery outcomes may be higher in India because patients there have multiple therapeutic options available with which to address psychiatric distress (e.g., religious, ayurvedic, and allopathic), all of which appeared to work for some patients and not for others [49]. The epigenetic sequelae of key life events may also be of great importance, and ethnographers are well-suited to help prospectively document and retrospectively unpack these events [3••]. Ethnographic data also adds the voices of those being treated to debates surrounding what is best for them [18, 43, 49].

Transdisciplinary teams using ethnographers might, for example, identify potentially protective locally relevant cultural beliefs, practices, and rituals, and then test them to assess their potential as treatment. Indeed, cultural practices like meditation, yoga, dance, and ritual trance may alter the priming of stress reactions thought to trigger psychosis [1•]. Biomarkers related to stress, such as cardiovascular autonomic dysfunction (which is heritable, causes physiologic hyperactivity to stress, and appears in higher rates in people with schizophrenia and their relatives [50]), might be measured before and after seemingly helpful cultural practices to try to capture the ways culture may get “under the skin” to encourage recovery (e.g., an enhanced ability to relax in the face of stress [2]). In the future, this kind of transdisciplinary research could enable psychiatrists to identify and make use of culturally available mechanisms that facilitate recovery from psychotic disorders to provide the best contextually relevant care possible for people experiencing psychotic symptoms around the globe.

Acknowledgments The author would like to thank Steven Siegel, Kim Hopper, Tanya Luhrmann, and Constantin Tranulis for their comments on earlier versions of this manuscript. The project described was supported by grant no. 5-T32-AT000052 from the National Center for Complementary and Alternative Medicine at the National Institutes of Health. Its contents are solely the responsibility of the author and do not necessarily represent the

official views of the National Center for Complementary and Alternative Medicine.

Disclosure No potential conflict of interest relevant to this article was reported.

References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
 - Of major importance
1. • Howes OD, Kapur S. The dopamine hypothesis of schizophrenia: version III—the final common pathway. *Schizophr Bull.* 2009;35:549–62. *This article ties together several hypotheses relating social stress to neurophysiologic pathways.*
 2. Myers N. Culture, stress and recovery from schizophrenia: lessons from the field for global mental health. *Cult Med Psychiatry.* 2010;34:500–28.
 3. •• Kirkbride JB, Jones PB. The prevention of schizophrenia—what can we learn from eco-epidemiology? *Schizophrenia Bull.* 2011; 37:262–71. *In this article, the authors set the stage for future schizophrenia research with proposed transdisciplinary research that promises to break new ground in our understanding of psychotic disorders.*
 4. •• Van Os, J, Linscott, RJ, Myin-Germeys, I, et al.: A systematic review and meta-analysis of the psychosis continuum: evidence for a psychosis proneness-persistence-impairment model of psychotic disorder. *Psychol Med.* 2009; 39:179–95. *This article offers a new perspective on the ways we diagnose, classify, and treat psychotic disorders to provide a framework for a new generation of research.*
 5. Kelleher I, Cannon M. Psychotic-like experiences in the general population: characterizing a high-risk group for psychosis. *Psychol Med.* 2010; 1–6.
 6. Nuevo R, Chatterji S, Verdes E et al. The continuum of psychotic symptoms in the general population: a cross-national study. *Schizophr Bull* 2010; Sep 13.
 7. De Loore E, Gunther N, Drukker M, et al. Persistence and outcome of auditory hallucinations in adolescence: a longitudinal general population study of 1800 individuals. *Schizophr Res.* 2011;127:252–6.
 8. Ruhrmann S, Schultze-Lutter F, Salokangas RKR, et al. Prediction of psychosis in adolescents and young adults at high risk: results from the prospective European prediction of psychosis study. *Arch Gen Psychiatry.* 2010;67:241.
 9. Bauer S, Schanda H, Karakula H, et al. Culture and the prevalence of hallucinations in schizophrenia. *Compr Psychiatry.* 2011;52:319–25.
 10. Gecici O, Kuloglu M, Guler O, et al. Phenomenology of delusions and hallucinations in patients with schizophrenia. *Bull Clin Psychopharmacol.* 2010;20:204–12.
 11. • Strauss, GP, Harrow, M, Grossman, LS, et al.: Periods of recovery in deficit syndrome schizophrenia: a 20-year multi-follow-up longitudinal study. *Schizophr Bull.* 2010; 36:788–99. *This article accentuates the importance of considering negative symptoms when we try to account for who may be achieving recovery from schizophrenia and who is not.*
 12. Harvey PD, Helldin L, Bowie CR, et al. Performance-based measurement of functional disability in schizophrenia: a cross-national study in the United States and Sweden. *Am J Psychiatry.* 2009;166(7):821–7.

13. Bresnahan M, Begg MD, Brown A, et al. Race and risk of schizophrenia in a US birth cohort: another example of health disparity? *Int J Epidemiol.* 2007;36:751–8.
14. •• Bourque, F, van der Ven, E, Malla, A: A meta-analysis of the risk for psychotic disorders among first-and second-generation immigrants. *Psychol Med.* 2011; 41:897–910. *This article is the most thorough analysis to date on issues of race, migration, and psychosis.*
15. Metzl J. In: Metzl J, editor. *The protest psychosis: How schizophrenia became a black disease.* New York: Beacon; 2010.
16. • Zandi T, Havesaar JM et al. First contact incidence of psychotic disorders among native Dutch and Moroccan immigrants in the Netherlands: influence of diagnostic bias. *Schizophr Res.* 2010; 119:27–33. *This study demonstrates the importance of contextualizing diagnoses in the life worlds of individual patients and prompts provocative questions about the assumptions made when conducting research.*
17. Lewis-Fernandez R, Horvitz-Lennon M, Blanco C, et al. Significance of endorsement of psychotic symptoms by US Latinos. *J Nerv Ment Dis.* 2009;197:337–47.
18. Tranulis C, Park L, Delano L, Good B. Early intervention in psychosis: a case study on normal and pathological. *Cult Med Psychiatry.* 2009;33:608–22.
19. Van Winkel R, Esquivel G, Kenis G. Review: genome wide findings in schizophrenia and the role of gene-environment interplay. *CNS Neurosci Ther.* 2010;16:185–92.
20. • Calkins, ME, Tepper, P, Gur, R, et al: Project Among African-Americans to Explore Risks for Schizophrenia (PAARTNERS): evidence for impairment and heritability of neurocognitive functioning in families of schizophrenia patients. *Am J Psychiatry* 2010; 167:459–72. *This is a rare look at potential intersections between heritability and neurocognition, and implications for the development of psychotic disorder among African American populations.*
21. Morgan C, Fisher H, Hutchinson G, et al. Ethnicity, social disadvantage and psychotic-like experiences in a healthy population based sample. *Acta Psychiatr Scand.* 2009;119:226–35.
22. Schofield P, Ashworth M, Jones R. Ethnic isolation and psychosis: re-examining the ethnic density effect. *Psychol Med.* 2010; FirstView. 1–7.
23. Zammit S, Lewis G, Rasbash J, et al. Individuals, schools, and neighborhood: a multilevel longitudinal study of variation in incidence of psychotic disorders. *Arch Gen Psychiatry.* 2010;67:914–22.
24. March D, Hatch SL, Morgan C, et al. Psychosis and place. *Epidemiol Rev.* 2008;30:84–100.
25. Wicks S, Hjern A, Dalman C. Social risk or genetic liability for psychosis? A study of children born in Sweden and reared by adoptive parents. *Am J Psychiatry.* 2010;167:1240–6.
26. Jenkins R, Bhugra D, Bebbington P, et al. Debt, income and mental disorder in the general population. *Psychol Med.* 2008;38:1485–93.
27. Corcoran C, Perrin M, Harlap S, et al. Effect of socioeconomic status and parents' education at birth on risk of schizophrenia in offspring. *Soc Psychiatry Psychiatr Epidemiol.* 2009;44:265–71.
28. Jenkins R, Mbatia J, Singleton N, et al. Prevalence of psychotic symptoms and their risk factors in urban Tanzania. *Int J Environ Res Public Health.* 2010;7:2514–25.
29. Fisher H, Morgan C, Dazzan P, et al. Gender differences in the association between childhood abuse and psychosis. *Br J Psychiatry.* 2009;194:319.
30. Burns JK, Jhazbhay K, Esterhuizen T, et al. Exposure to trauma and the clinical presentation of first-episode psychosis in South Africa. *J Psychiatr Res.* 2010;45:179–84.
31. Arseneault L, Cannon M, Fisher HL, et al. Childhood trauma and children's emerging psychotic symptoms: a genetically sensitive longitudinal cohort study. *Am J Psychiatry.* 2010;168:65–72.
32. Eyles DW, Feron F, Cui X, et al. Developmental vitamin D deficiency causes abnormal brain development. *Psychoneuroendocrinology.* 2009;34:S247–57.
33. Morgan C, Fisher H, Hutchinson G, et al. Ethnicity, social disadvantage and psychotic-like experiences in a healthy population based sample. *Acta Psychiatr Scand.* 2009;119:226–35.
34. Thornicroft G, Brohan E, Rose D, et al. Global pattern of experienced and anticipated discrimination against people with schizophrenia: a cross-sectional survey. *Lancet.* 2009;373:408–15.
35. Aguilera A, Lopez SR, Breitborde N, et al. Expressed emotion and sociocultural moderation in the course of schizophrenia. *J Abnorm Psychol.* 2010;119:875–85.
36. Lopez SR, Ramirez Garcia JI, Ullman JB, et al. Cultural variability in the manifestation of expressed emotion. *Fam Process.* 2009;48:179–94.
37. Yang LH, Phillips MR, Lo G, et al. "Excessive thinking" as explanatory model for schizophrenia: impacts on stigma and "moral" status in Mainland China. *Schizophr Bull.* 2009;36:836–45.
38. Saravanan B, Jacob KS, Deepak MG, et al. Perceptions about psychosis and psychiatric services: a qualitative study from Vellore, India. *Soc Psychiatry Psychiatr Epidemiol.* 2008;43:231–8.
39. McGorry P, Johansson JO, Lewis S, et al. Early intervention in psychosis: keeping faith with evidence-based health care. *Psychol Med.* 2010;40:399–404.
40. Farooq S, Large M, Nielsens O, et al. The relationship between the duration of untreated psychosis and outcome in low-and-middle income countries: a systematic review and meta analysis. *Schizophr Res.* 2009;109:15–23.
41. Saravanan B, Jacob KS, Johnson S, et al. Outcome of first-episode schizophrenia in India: longitudinal study of effect of insight and psychopathology. *Br J Psychiatry.* 2010;196:454–9.
42. Correll CU, Manu P, Olshansky V, et al. Cardiometabolic risk of second-generation antipsychotic medications during first-time use in children and adolescents. *JAMA.* 2009;302:1765–73.
43. Jenkins J, Carpenter-Song E. Stigma despite recovery: strategies for living in the aftermath of psychosis. *Med Anthropol Q.* 2008;22:381–409.
44. • Keshavan M, Shrivastava A, Gangadhar B. Early intervention in psychotic disorders: challenges and relevance in the Indian context. *Indian J Psychiatry* 2010; 52:153–8. *This is an interesting look at the challenges of EI in global mental health.*
45. Sartorius N. Short-lived campaigns are not enough. *Nature.* 2010;468:163–5.
46. Angermeyer MC, Holzinger A, Matschinger H. Mental health literacy and attitude towards people with mental illness: a trend analysis based on population surveys in the eastern part of Germany. *Eur Psychiatry.* 2009;24:225–32.
47. van Os J, Kapur S. Schizophrenia. *Lancet.* 2009;374:635–45.
48. Kirkbride J, Coid JW, Morgan C, et al. Translating the epidemiology of psychosis into public mental health: evidence, challenges and future prospects. *J Public Ment Health.* 2010;9:4–14.
49. Halliburton M. *Mudpacks and prozac: Experiencing ayurvedic, biomedical, and religious healing.* Los Angeles: Left Coast; 2009.
50. Bar KJ, Berger S, Metzner M, et al. Autonomic dysfunction in unaffected first-degree relatives of patients suffering from schizophrenia. *Schizophr Bull.* 2010;36:1050–8.