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Putting Madness in Its Place: Can the Environment Explain Schizophrenia's Hereditary Patterns?
Growing evidence points to birthplace as a risk factor for schizophrenia

Schizophrenia hides its heritability well. Although fewer than 1 percent of the general population will be diagnosed as schizophrenic based on symptoms such as hallucination and disorganized thought, for children of a schizophrenic parent, those odds jump to about one in 10. And yet the condition's genetic underpinnings have stubbornly resisted discovery. In the latest attempt, three crack teams of investigators pooled genomic data from 8,000 schizophrenics of European ancestry but could lay claim to only a handful of weak genetic risk markers.

Analyses such as these, which appeared online July 1 in *Nature* (*Scientific American* is part of the Nature Publishing Group), have led researchers to question the value of brute-force genomics for analyzing schizophrenia. "I think we need to pause and think through the risk pathways to disease more clearly," says Dolores Malaspina, director of the social and psychiatric initiatives program at New York University Langone Medical Center. In particular, devotees of genetics might want to cede a little ground to their colleagues in epidemiology, who over the past decade have amassed a provocative, interlocking set of studies implicating urban birthplace and migrant status as persistent risk factors.

Researchers believe the potential for schizophrenia starts to emerge during early brain development, beginning in the womb. Rates tick up slightly for offspring whose mothers were infected with influenza or undernourished during pregnancy, for newborns who suffered obstetric complications such as oxygen deprivation, and for offspring born in the winter or spring.

Starting in the 1990s, studies from Denmark, the Netherlands and Sweden began making the case for urban life as a distinct risk factor. In the largest of these, out of a cohort of 1.75 million Danes, being born in Copenhagen was associated with a 2.5-fold greater risk of schizophrenia than being born in rural areas. Danes who were born in smaller cities showed intermediate risk. Although the nature of the exposure remains obscure, researchers were able to narrow down its timing: Danes who lived in urban centers for the first 15 years of life had the most elevated risk.

A second wave of findings has documented that immigrants to European countries are at heightened risk of schizophrenia as compared with native-born residents. Second-generation immigrants show increased risk relative to their parents, and rates are highest among those of African heritage. In a study of three cities in the U.K., Afro-Caribbeans were nine times as likely as the general population to be treated for schizophrenia. Neighborhood composition seems to play a role. In South London epidemiologist James Kirkbride of the University of Cambridge and his colleagues at King's College London have found that in neighborhoods with higher measures of "social cohesion," such as voter turnout, the incidence of schizophrenia is proportionally lower.

Despite the consistency of the findings, epidemiologists who work in the field say scientific journals in the U.S. have shown reluctance to consider papers that explore the relation between race and schizophrenia. Hence, it was not until 2007 that Michaeline Bresnahan, Ezra Susser and their colleagues at the Columbia University Mailman School of Public Health cautiously published data from a cohort of 12,000 Californians enrolled in the Kaiser Permanente health plan, which showed that the rate of hospital admission for schizophrenia was twice as high for African-Americans as for whites, even after controlling for socioeconomic status of the parents. Because the cohort was part of the same health plan, reduced access to health services was unlikely to account for the discrepancy, Susser says.

Given that schizophrenia has no clear biological markers, skeptics may question whether diagnostic criteria have been applied rigorously across diverse cultural groups. For epidemiologists, such arguments miss the point. "The strategy is to identify important risk or protective factors within a given group," observes Dana March, a Ph.D. candidate in Susser's group.

March says her preliminary work shows that of Kaiser cohort members born in Oakland, Calif.,* those born into more densely populated neighborhoods are at twofold to threefold greater risk of schizophrenia than those born in less dense areas, irrespective of race. Residents of more run-down or overcrowded city neighborhoods could be more

exposed to toxic chemicals and infections, she says, and may have less access to social capital that would blunt the effects of a predisposition to mental illness acquired early in life.

In an attractive synthesis, such neighborhood-level risk factors might impart lasting epigenetic changes—the chemical overwriting of the genome in response to environmental cues. If true, the roots of schizophrenia would lie where geography and genetics meet.

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