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Background: The association of urbanicity at birth and during upbringing with schizophrenia remains unexplained. Some studies have indicated that it may be driven by familial selective factors such as genetic liability. However, no studies have used measures of genetic risk other than family history to test this hypothesis with respect to urbanicity at birth. We assessed whether genetic liability to schizophrenia, measured by polygenic risk score (PRS), was associated with being born and raised in urban areas, and whether adjustment for PRS and parental history of mental disorder together explained the effect of urbanicity.

Methods: We conducted a nested case–control study using Danish population registry data. Cases in the Danish Psychiatric Central Register (n = 1692) who were born since 1981 and diagnosed with schizophrenia between 1994 and 2009 were matched to controls (n = 1724) with the same sex and birthdate. Genome-wide data were obtained from the Danish Neonatal Screening Biobank, and PRSs were calculated based on a Psychiatric Genomics Consortium meta-analysis that excluded Danish samples. PRSs were adjusted for ancestry using the first 10 principal components.

Results: PRS generally increased with increasing level of urbanicity. Those with higher PRSs were significantly more likely to reside in the capital compared to rural areas at age 15 (odds ratio [OR] = 1.19, 95% confidence interval [CI] =1.00–1.42) but not at birth (OR = 1.09, 95%CI = 0.94–1.26). Adjustment for PRS produced almost no change in the excess risk of schizophrenia associated with urbanicity at birth (relative risk [RR] = 1.67, 95%CI = 1.29–2.17). However, the RR associated with urbanicity at age 15 decreased from 1.58 (95%CI = 1.20–2.09) to 1.47 (95%CI = 1.10–1.97) after adjustment for PRS. After adjustment for PRS and parental history together, the association between urbanicity at birth and schizophrenia remained (P = .016) but that for urbanicity at age 15 was fully attenuated (P = .148).

Conclusion: We found evidence consistent with a role of selective migration in the association between urbanicity and schizophrenia, especially with respect to urbanicity during upbringing. However, our results failed to support the notion that genetic liability fully explains the excess risk of schizophrenia associated with urbanicity at birth. Additional research is needed to identify other familial mechanisms or constituent exposures that explain the robust association between urbanicity at birth and schizophrenia.

36. ARE FAMILIAL LIABILITY AND OBSTETRIC COMPLICATIONS INDEPENDENTLY ASSOCIATED WITH RISK OF A PSYCHOTIC ILLNESS, AFTER ADJUSTING FOR OTHER ENVIRONMENTAL STRESSORS?

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Background: Both maternal schizophrenia and obstetric complications have been implicated in risk for psychotic illness. Other environmental stressors (e.g., childhood abuse, social disadvantage, and discontinuity in parenting) have also been implicated. Previously (ICOSR 2015), we have shown that children of mothers with schizophrenia are more likely than comparison children to be exposed to these other environmental stressors. Our aim in this paper is to assess the independent contribution of maternal schizophrenia and obstetric complications to risk for developing a psychotic illness, covarying for exposure to other environmental stressors in childhood.

Methods: This cohort study used record-linkage across State-wide registers (including midwives, psychiatric, child protection, and mortality) to identify 1762 offspring born in Western Australia 1980–2001 to 889 mothers with a lifetime history of schizophrenia (case children) and compared them with 239 365 offspring born in the same period to 452 459 mothers with no known psychiatric history (comparison children).

Results: In total, 5.4% of children of mothers with schizophrenia developed a psychotic illness compared to 1.1% of comparison children. In bivariate analyses, maternal schizophrenia (HR 5.7, CI 4.7-7.0), pregnancy complications (HR 1.0, CI 1.0–1.2), and neonatal complications (HR 1.1, CI 1.1–1.2) were associated with risk of developing a psychotic illness. Other environmental exposures associated with risk of a psychotic illness included: being aboriginal, lower socioeconomic status, death of a parent and childhood abuse. Using multivariable modeling, maternal schizophrenia (HR 5.7, CI 4.6-7.0), pregnancy complications (HR, 1.1 CI 1.0–1.2), and neonatal complications (HR 1.0, CI 1.0–1.1) were independently associated with risk of a psychotic illness. When adjusted for other adverse environmental exposures, the risk associated with maternal schizophrenia dropped markedly (HR 3.9, CI 3.1–4.7) but was still high and significant. The HRs for pregnancy and neonatal complications were unchanged, but neonatal complications dropped out of significance.

Conclusion: Familial liability and obstetric complications are independently associated with risk of psychotic illness. The substantial decrease in risk associated with familial liability following adjustment for environmental stressors, some of which were potentially modifiable, indicates that risk can be lowered by interventions targeting these factors.

37. POSITIVE PSYCHOPATHOLOGY IN EXCEPTIONALLY CREATIVE ARTISTS?

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Background: As part of a broad investigation of brain and behavior, the UCLA Big C Project studied whether "Big C" (world-class) artistic and scientific creativity is linked with schizotypal and autistic features and whether temperamental traits are associated with creativity.

Methods: With the Schizotypal Personality Questionnaire (SPQ), Social Responsiveness Scale (SRS), and the International Personality Item Pool Representation of the NEO PI-R (IPIP-NEO), we examined more than 30 individuals each in 3 groups: Big C Visual Artists (VA), Big C Scientists (SCI), and a Smart Comparison Group (SCG) matched on age, sex, race/ethnicity, parental education, and IQ estimates. Big C groups had internationally prominent creative reputations and markedly higher Creative Achievement Questionnaire scores within their domains.

Results: VA reported higher levels of odd beliefs, ideas of reference, unusual perceptions, odd speech, and more socially divergent tendencies. Specifically, VA had significantly greater scores in Cognitive/Perception and Disorganization SPQ factors compared to SCI (and nominally greater scores than SCG). VA also had significantly higher scores in SRS subscales of Social Communication, Social Motivation, Restricted Interests and Repetitive Behavior compared to SCG.

All 3 groups had significant differences in openness (VA>SCI>SCG). Both Big C groups were significantly more achievement striving than SCG, with the scientists higher than the visual artists (SCI, VA > SCG). The Big C groups also reported significantly higher scores on activity and liberalism compared to SCG; the visual artists were higher than the scientists on these facets (VA, SCI > SCG). VA had significantly higher scores on imagination and artistic interests than SCI and SCG (VA > SCI, SCG).

Conclusion: These results complement and extend past findings of high magical ideation and openness associated with everyday creative achievement in healthy individuals. Compatible with the hypothesis of "subclinical" psychopathology associated with creativity, these results also open questions about whether there are valuable and potentially adaptive aspects of psychopathological traits and what may be driving this association in exceptional artistic creativity. These results also suggest that openness, imagination, and an active, achievement-focused lifestyle may mediate this link.