Prevalence of Psychotic Disorder and Community Level of Psychotic Symptoms

An Urban-Rural Comparison

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Background: Urban and rural populations have different rates of psychotic illness. If psychosis exists as a continuous phenotype in nature, urban-rural population differences in the rate of psychotic disorder should be accompanied by similar differences in the rate of abnormal mental states characterized by psychotic or psychosilike symptoms.

Methods: A random sample of 7076 individuals aged 18 to 64 years were interviewed by trained lay interviewers with the Composite International Diagnostic Interview. Approximately half of those with evidence of psychosis according to the Composite International Diagnostic Interview were additionally interviewed by clinicians. We investigated associations between a 5-level urbanicity rating and (1) any DSM-III-R diagnosis of psychotic disorder (sample prevalence, 1.5%), (2) any rating of hallucinations and/or delusions (sample prevalence, 4.2%), and (3) any rating of psychotic or psychosilike symptoms (sample prevalence, 17.5%).

Results: Level of urbanicity was associated not only with DSM-III-R psychotic disorder (adjusted odds ratio [OR] over 5 levels, 1.47; 95% confidence interval [CI], 1.25-1.72), but also, independently, with any rating of delusion and/or hallucination (adjusted OR, 1.28; 95% CI, 1.17-1.40; clinician-assessed psychotic symptoms only: OR, 1.30; 95% CI, 1.03-1.64) and any rating of psychosilike symptom (adjusted OR, 1.18; 95% CI, 1.13-1.24). Psychotic symptoms were strongly and independently associated with psychotic disorder, regardless of the level of urbanization.

Conclusions: Community level of psychotic and psychosilike symptoms may be inextricably linked to the prevalence of psychotic disorder. The prevalence of abnormal mental states that facilitate development to overt psychotic illness increases progressively with level of urbanization.

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Some risk factors for psychotic illness can be used to define populations with different levels of risk. For example, exposure to urban birth, upbringing, or residence increases the risk for later psychotic illness, suggesting that urban and rural populations have different lifetime risks. A plausible explanation for the urban-rural differences is that environmental factors associated with urban life make individuals more vulnerable to the development of psychotic states.

The factors that increase the risk for psychotic disorder in urban areas may contribute to a much larger pool of preclinical psychotic experiences, of which only a small proportion may continue to result in overt disorder. This hypothesis would be compatible with the suspected multifactorial origin of psychotic disorders, according to which it is unlikely that any multifactorial disease exists as a purely dichotomous entity in nature without less severe, nonpathologic manifestations of the phenotype. It is also compatible with accumulating evidence that schizotypal signs and psychosilike symptoms such as delusional ideation and isolated hallucinations are prevalent in the general population and show longitudinal, neuropysychological, psychopathologic, familial, neuroradiologic, epidemiologic, and risk factor continuities with clinical psychotic syndromes such as schizophrenia. All of these data suggest that, at the level of the general population, lesser psychotic states exist that are associated with the more severe clinical disorders that necessitate hospital admission.

Given these continuities, it is attractive to hypothesize that the higher level of psychotic disorders in urban areas is accompanied by a similar increase in the level of psychosilike symptoms. Population studies of minor psychiatric disorders

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have shown that the prevalence of disorder is linearly related to the mean number of psychiatric symptoms. A similar relationship between symptoms and disorder may exist in psychosis.

In the current study, we investigated to what degree the increase in risk for psychotic disorder associated with urban life is reflected in similar increases in the mean number of psychotic and psychosislike symptoms. We hypothesized that the mean level of symptoms would increase with the rate of disorder across increasingly urbanized areas. In addition, we hypothesized that the mean number of psychotic and psychosislike symptoms would increase with the rate of disorder across increasingly urbanized areas. If, however, not only the rate of disorder, but also the rate of symptoms, is higher in population A than in population B, a likely explanation would be that (1) the population level of vulnerability differs between population A and population B and (2) the psychosislike experiences are, at least in part, on a quantitative continuum with disorder. A graphic representation of this argument, using hypothetical data, is depicted in the Figure.

The sample consisted of 7076 individuals (46.6% male) with a mean age of 41.2 years (SD, 12.2). There were 936 individuals (13.2%) who were foreign-born or whose father or mother was foreign-born. The prevalences of the different CIDI ratings on the 17 psychosis items were as follows: any CIDI rating of 2, n=915 (12.9%); any CIDI rating of 3 or 4, n=39 (0.6%); any CIDI rating of 5, n=295 (4.2%); and any CIDI rating of 6, n=285 (4.0%). After training course in recruiting and interviewing, followed by a 4-day course at the World Health Organization–CIDI training center in Amsterdam, the Netherlands. Extensive monitoring and quality checks took place throughout the entire data collection period. B are essentially similar, except for the distribution of some rare cause affecting a few individuals. There is variation within populations. If, however, not only the rate of disorder, but also the rate of symptoms, is higher in population A than in population B, a likely explanation would be that (1) the population level of vulnerability differs between population A and population B and (2) the psychosislike experiences are, at least in part, on a quantitative continuum with disorder. A graphic representation of this argument, using hypothetical data, is depicted in the Figure.

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adjustment for age, sex, ethnic group, educational level, level of urbanicity, and presence of any psychotic or non-psychotic DSM-III-R diagnosis, all but one of the different CIDI ratings were independently (assessed by entering them together in the model) associated with a lifetime history of mental health service (rating 2: OR, 0.99; 95% CI, 0.82-1.19; rating 3 or 4: OR, 2.65; 95% CI, 1.30-5.44; rating 5: OR, 4.19; 95% CI, 3.23-5.44; rating 6: OR, 3.12; 95% CI, 2.39-4.06), and all were associated with a lower quality-of-life total score measured with the quality-of-life schedule of the 36-item Short Form Health Survey schedule (rating 2: multiple regression coefficient B = −1.99; 95% CI, −3.24 to −0.75; rating 3 or 4: B = −6.78; 95% CI, −12.25 to −1.13; rating 5: B = −6.79; 95% CI, −8.94 to −4.63; rating 6: B = −3.09; 95% CI, −5.25 to −0.94).

The lifetime prevalence of DSM-III-R schizophrenic symptoms broadly defined was 0.37% (26 cases), and the lifetime prevalence of affective psychosis (major depression or bipolar disorder with psychotic features) was 1.14% (81 cases), making a total of 107 cases (1.51%). The prevalence of psychotic symptoms broadly defined was 17.5% (n = 1237), and the prevalence of psychotic symptoms narrowly defined was 4.2% (n = 295).

PSYCHOSIS IN RELATION TO URBANICITY

The lifetime prevalences of DSM-III-R psychotic disorder, psychotic symptoms narrowly defined, and psychotic symptoms broadly defined increased in a monotonic fashion with level of urbanicity. Adjustment for age, sex, level of education, and country of birth of subject and parents changed the parameters only by a small amount (Table 1). Associations also remained after exclusion of individuals with psychotic disorder from the group with narrowly defined psychotic symptoms, and after exclusion of individuals with psychotic disorder and individuals with narrowly defined psychotic symptoms from the group with broadly defined psychotic symptoms (Table 1). There was no interaction with lifetime mental health treatment for any of the 3 groups (LR test psychotic disorder: χ² = 0.05, P = .83; LR test narrowly defined: χ² = 0.21, P = .64; LR test broadly defined: χ² = 0.00, P = .98).
SENSITIVITY ANALYSES

The following analyses were conducted. First, we repeated the analyses of association between urbanicity and the 3 groups of symptom ratings, adjusted for age, sex, level of education, and country of birth of subject and parents, excluding the 253 individuals who were eligible for clinical reinterview but who were not interviewed. Second, we repeated the same analyses restricted to the group who were interviewed by the psychiatric trainees (n=226). Third, we repeated the analyses assuming that all individuals who were eligible for reinterview, but were not reinterviewed, would have received a rating of 5 (true symptom) on all CIDI psychosis items if they had been reinterviewed. Finally, we repeated the same analyses assuming that these individuals would have received a rating of 1 (no symptom) on all ratings. The pattern of results for all these analyses was the same and was similar to the results in Table 1 (Table 2).

ASSOCIATIONS BETWEEN SYMPTOMS AND DISORDER AT DIFFERENT LEVELS OF URBANICITY

Psychotic symptoms, broadly and narrowly defined, were strongly associated with psychotic disorder (broadly defined: summary OR over 6 levels, 3.59; 95% CI, 3.17-4.06; narrowly defined: OR, 6.96; 95% CI, 5.57-8.68). The association between psychotic disorder and broadly de-
fined psychotic symptoms remained after adjustment for presence of narrowly defined psychotic symptoms (adjusted OR, 2.29; 95% CI, 1.93-2.73), indicating that broadly defined symptoms were associated with psychotic disorder independent of their association with narrow symptoms. There was no evidence that this association differed as a function of urbanicity (LR test broadly defined: $\chi^2 = 4.20, P = .04$; LR test narrowly defined: $\chi^2 = 4.51, P = .34$), or if the sample was restricted to those who had had clinical reinterviews ($n = 226; P = .54$ and $P = .95$, respectively).

### Table 2. Sensitivity Analysis

<table>
<thead>
<tr>
<th>Sensitivity Analysis *</th>
<th>Adjusted* Odds Ratio Linear Trend (95% Confidence Interval)†</th>
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<tbody>
<tr>
<td></td>
<td>Psychotic Disorder</td>
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<tr>
<td>Exclusion of individuals missed for reinterview</td>
<td>1.54 (1.26-1.89)</td>
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<tr>
<td>Restriction to individuals who were interviewed by psychiatrist</td>
<td>1.34 (1.04-1.74)</td>
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<tr>
<td>Assuming all CIDI psychosis items of individuals missed at reinterview would have been rated “5”</td>
<td>1.35 (1.19-1.53)</td>
</tr>
<tr>
<td>Assuming all CIDI psychosis items of individuals missed at reinterview would have been rated “1”</td>
<td>1.54 (1.26-1.88)</td>
</tr>
</tbody>
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*See the “Sensitivity Analyses” subsection of the “Subjects and Methods” section for explanation. CIDI indicates Composite International Diagnostic Interview.
†The sum increase in risk with 1-unit change in address density.

In a representative sample of 7076 subjects sampled from the general population, lifetime level of psychotic and psychosislike symptoms independently increased with level of urbanicity in the same manner as did DSM-III-R psychotic disorder. Initial response rate, clinical reinterview response rate, and change rate of CIDI psychosis items after clinical reinterview were not associated with level of urbanicity. At all levels of urbanicity, psychosislike symptoms were strongly associated with psychotic disorder. These findings therefore suggest that the increased prevalence of psychotic disorder in urban environments should be interpreted in light of increased levels of “psychosis proneness” in urban populations.

More generally, the results suggest that there is a link between community level of psychotic symptoms and rates of clinical disorder. As the association between symptoms and disorder did not differ as a function of urbanicity, the implication is that susceptibility to psychotic disorder varies between populations and can be demonstrated by comparing rates of psychosilike phenomena. Given that the rates of these phenomena are much higher than those for rare psychotic disorders, etiologic research may be served by focusing on these related phenotypes. Similarly, preventive action may be served by population interventions rather than, or in addition to, the high-risk strategies that are currently being explored.

The high rates of psychotic illness in urban environments may be the result of the influence of environmental factors. As the urban effect appears to have its impact during urban upbringing rather than during adult residence per se,1,12 developmental mechanisms ought to be considered. A possible developmental mechanism whereby social factors may create enduring liabilities for adult psychosis are the effects of the wider social environment, such as the neighborhood environment, on child and adolescent development.30 Mental states are reactive to experience, and differences in the level of deprivation and social isolation of the neighborhood environment in urban areas have been shown to be associated with variation in a range of mental health outcomes from problem behavior in children49 to incidence of neurosis and schizophrenia.49-50 High levels of deprivation and low levels of social capital51 in the wider social environment may enhance the development of “at-risk” mental states that in turn may facilitate the onset of clinical psychosis in adult life.

These results should be viewed in light of several possible limitations. First, we examined lifetime rates of disorder and symptoms in relation to current urban residence. Thus, one explanation for the findings is that symptomatic individuals could have “drifted” to urban areas. Although we cannot exclude this mechanism, a previous report by our group found that there was a high degree of lifetime stability of urban exposure status (around 75% of individuals living in urbanized areas had also been born there), indicating that current exposure is likely to reflect stable lifetime exposure in most cases.3 In addition, the association between psychosis and urbanicity did not differ as a function of lifetime mental health patient status. This suggests that it is unlikely that the findings can be explained solely by a process of urban drift of the most symptomatic individuals, in which case one would have expected associations to be stronger in the patient group. Second, the validity of the CIDI ratings of psychosis, such as a rating of 2 (symptom present but not clinically relevant) is not well researched. However, the associations with lifetime mental health service contact (no association with rating of 2 and significant associations with other ratings, especially rating of 3 for true psychotic symptom) and lower quality of life (weakest for rating of 2 and strongest for rating of 5), independent of each other and of any lifetime DSM-III-R diagnosis, provided some degree of validity for the conceptual contrast of the ratings as well as for their existence independent of psychiatric disorder. Third, psychotic symptom ratings were assessed by lay interviewers (CIDI ratings of 1, no symptom; 2, symptom present but not clinically relevant; 3, symptom the result of ingestion of drugs; and 4, symptom the result of somatic disease), whereas the other CIDI ratings (5, true psychiatric symptom; and 6, may not really be a symptom because there appears to be some plausible explanation for it) were as-

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sessed by clinicians through telephone interviews in approximately 50% of eligible cases. Although the sensitivity analyses showed that incomplete rates of reinterview by clinicians are unlikely to have biased the results, it is likely that, especially with lay interviewer ratings, a degree of misclassification did occur. However, for misclassification to explain the results, one would have to hypothesize that with greater degrees of urbanization, interviewers became progressively more likely to misclassify in one specific direction, and, furthermore, this influence of misclassification would have had to be the same for lay interviewers and clinicians. Although this cannot be excluded, it is unlikely also because the rate of change in CIDI ratings after clinician reinterview was not associated with urbanity and because the findings were similar when the sample was restricted to those who had been interviewed by a clinician.

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