Chapter 8

Is urbanicity an environmental risk-factor for psychiatric disorders?

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The link between urbanicity and the development of psychiatric disorders is well established. Recently, Kristina Sundquist and colleagues (2004) showed once again, with a strong study design, that there is a link between degree of urbanisation and first admission for psychosis. For men, the risk of first admission for psychosis was 68% higher in the most densely populated areas of Sweden than in the least densely populated areas (on a scale with five categories of urbanisation). For women, the risk was 77% higher. These results were all independent of age, marital status, education, and immigrant status. The results for psychosis contrast with the weak correlations found for depression (12% higher for men and 20% higher for women). Possible explanations given by the investigators for the increased risk in cities compared with rural areas are differences in social support, stressful life events, and familial liability.

Sundquist and colleagues' findings (2004) for psychosis confirm previous findings for schizophrenia in, for instance, Denmark (Pedersen & Mortensen, 2001a), the USA (Torrey & Bowler, 1990), and the Netherlands (Dekker et al., 1997; Peen & Dekker, 1997; van Os et al., 2001; Peen & Dekker, 2003).
In his review, Freeman (1994) speculated whether the link often found between urbanicity and schizophrenia is not, ultimately, the result of migration effects and social class. If results of previous studies were to be completely controlled for these variables, the urbanicity effect might disappear.

Against this background, we focus on the confounders corrected for by Sundquist and colleagues (2004) to assess the robustness of the findings. In the Sundquist study, data for men and women were corrected for age, marital status, education, and immigrant status. Unfortunately, it was not possible to correct for income, a factor which, in combination with education, provides a better picture of socioeconomic status. Furthermore, it was not possible to make an entirely accurate assessment of marital status with a distinction between living alone and married or cohabiting. Couples cohabiting without children were not registered as such, but as living alone. This means that the high hazard rate found for living alone compared with married or cohabiting for psychosis (6.02 for men, for instance)

will probably be even higher in reality. Another possible limitation of the study is that it deals with first admissions for psychosis or depression in residential health care. This type of care is the final filter used by Goldberg and Huxley (1980). Sundquist and colleagues have indicated that the risk of admission could not have been influenced by the available bed capacity, which was the same in rural areas as in large cities. We wonder whether the risk of admission for severe depression depends on the pressure on inpatient facilities for, in particular, psychotic patients. We think it plausible that the ratio of depressed patients to psychotic patients is lower in city wards, the explanation being that alternatives outside the clinic are used more often for depressed patients who are not highly suicidal. Possible filter effects of this kind might have reduced the urban/rural differences found for depression by Sundquist and colleagues. Nevertheless, despite the limitations of the Swedish study, we think it unlikely that these limitations can explain the link found between psychosis and urbanisation.

Urban/rural differences have now been found for psychosis in Sweden (Sundquist et al., 2004), Denmark (Pedersen & Mortensen, 2001), and the Netherlands (Dekker et al., 1997; Peen & Dekker, 1997; van Os et al., 2001; Peen & Dekker, 2003). Findings for the USA (Torrey & Bowler, 1990; Kessler et al., 1994) and the UK (Allardyce et al., 2001; Paykell et al., 2003) are inconsistent. We think it possible that variations of this kind between countries are related to differences in the organisation of mental health-care (when looked at in case-register studies; Sytema et al., 1996). A second possible explanation might be the differences in migration patterns in for instance the USA and Western Europe in terms of the formation of urban and rural communities. These migration processes might also affect the general genetic vulnerability of a population, and therefore the incidence of schizophrenia. Jablensky and Kalaydjieva (2003) refer to the high levels of rural-urban migration in the Netherlands in the 1970s, which might have resulted indirectly in the higher rates of schizophrenia found later in those born in urban settings (Marcelis et al., 1998). However, at present, there is no evidence supporting these hypothetical urban/rural differences in genetic liability.

Putting aside the inconsistency in findings between countries, some interesting openings have been found in recent years for an environmental explanation of the link between schizophrenia and urbanicity. First, for instance, the rates of schizophrenia in cities in singles (van Os et al., 2000) and in non-white ethnic minorities (Boydell et al., 2001) are higher when such groups make up a smaller proportion of the local population. This fact might be especially important in segregated urban communities. Second, the dose-response relation found (Pedersen & Mortensen, 2001b) in Denmark between schizophrenia and urbanicity indicated that urban exposure during upbringing and in later life was directly related to the risk of schizophrenia. Third, the risk of schizophrenia in people with

a family history of the disorder is higher when they live in urban areas. There would seem to be synergy between vulnerability and urbanicity (van Os et al., 2003). In addition to these important environmental links, possible urban/rural differences in genetic liability cannot be excluded. However, we believe that it is more likely that future research into environmental variables, such as daily-life stress and social cohesion, will elucidate the exact risk factors making up the general notion of urbanicity.

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