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Urbanisation and psychosis

A recent study of urbanisation and incidence of psychosis was based on data from the excellent Swedish population registers (Sundquist *et al*, 2004). Unfortunately, there are methodological problems with the study.

Sundquist *et al* argue that the prospective nature of their data allowed them to calculate incidence rates of psychoses rather than prevalence rates. However, their study confuses readmission and incidence rates. In incidence studies, the event of interest is new occurrence of a specified disease (see e.g. Clayton & Hills, 1993). Sundquist *et al*'s cohort consists of people aged 25–64 years in 1996 (i.e. born 1932–1971). They analyse the data as incident data although they do not have this information. For persons who enter the study in old age, they claim that when admitted during the study period 1997–1999 this is the first admission ever of these people. They do, however, exclude people admitted 1992–1996, but this only partly solves this problem (e.g. for people born in 1932 in their study, the earliest possible age at onset of psychosis is 60 years). Let us say the earliest age at onset of psychosis is 25 years, the authors lack information on first admission for psychosis among people born 1966 or earlier. Roughly, they do not have information on first admission for psychosis among people born 1932–1966, about 85% of the study population. If the earliest age of first admission for psychosis is 15 years (Pedersen & Mortensen, 2001), the authors lack information on first admission for all people. This severely violates the assumptions of an incidence study (Breslow & Day, 1987), and the results may be extremely biased.

Sundquist *et al* measure urbanisation as the place where cohort members lived around 1996. Using this measure of urbanisation – in combination with the problems mentioned above – implies problems with causality; is the disease a result of the urbanisation or is the urbanisation a result of the disease?

Based on these methodological errors, it is difficult to interpret their findings. However, some possible biases include selective migration of people with psychosis after disease onset, and urban–rural differences in the readmission rates for people with psychosis.

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Authors' reply: We agree that in strict terms the definition of incidence rates is in accordance with the definition described by for example Clayton & Hills (1993) and Last (1998).

However, we disagree when Pedersen & Agerbo state that we claim 'this is the first admission ever of these people'. Nowhere in the article have we used the phrase 'first admission ever'. In the Method we clearly described that the 4.4 million women and men were followed from 1 January 1997 until 31 December 1999 for first admission to hospital for treatment of psychosis or depression (i.e. during the study period). Individuals with previous hospital admissions for treatment of psychosis or depression from 1992 to 1996 were excluded (i.e. the 'wash-out' period was 5 years). We calculated the urbanisation measure for the year 1996 (i.e. the year when we defined our study population).

Pedersen & Agerbo also say that our article implies problems with causality. However, we did not claim that we have solved the causality problem. In addition, we discussed that we were unable to adjust our results for selective migration (i.e. migration from urban to rural areas). However, we did adjust our model for migration between neighbourhoods and the results remained almost unaltered.

We agree that urban–rural differences in admission rates could exist if, for example, the distribution of psychiatric beds differed between urban and rural areas. We checked this possible bias and found that the number of psychiatric beds per 1000 inhabitants differed very little across Sweden.

To sum up, our large-scale study sheds new light on the inconsistencies in previous research since we used the comprehensive Swedish registers and adjusted our results for several possible confounders. In addition, the urbanisation measure was calculated as actual numbers of inhabitants per area unit, which should reflect the level of urbanisation in the most appropriate way.

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Anthropology and psychiatry

From the perspective of anthropological psychiatry, Harland *et al* (2004) suggest that the social change experienced by migrants may predispose to schizophrenia. The same may apply to other forms of social change, such as those changes which lead to revitalisation phenomena and new religious movements. Psychiatric anthropologists who have studied new religious movements have pointed out that new forms of belief system may originate in the hallucinatory or delusional experience of a single individual (Littlewood, 1984). Wallace (1956) applied the term 'mazeway resynthesis' to the change in belief system which occurs in prophets, the mazeway being to the individual what culture is to society, so that the prophet awakes to a new reality which he or she then tries to impart to followers; if successful, the prophet becomes the leader of a new religious movement; otherwise, he or she is alienated from the parent group and is likely to be labelled as mentally ill. Foulks (1977) compared the prophet to the patient with schizophrenia, and noted that they shared not only the capacity to undergo a radical change in belief system but also various 'premorbid' features.

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For many years population geneticists have been surprised that the genetic tendency to schizophrenia is maintained in spite of the reduced fertility of those affected (Crow, 1995). However, the fertility of cult leaders is often increased, owing to increased mating opportunities within the cult, and to the possibility of an 'adaptive radiation' in a new land following the inevitable social and geographical alienation of the cult from the parent group. If the genes responsible for schizophrenia and prophetic experience were the same, it is possible that the fecundity of successful cult leaders might balance the loss of genes both in people with schizophrenia and in unsuccessful cult leaders (Price & Stevens, 1998).

Such evolutionary speculations are independent of the proximal causes of schizophrenia, which might be a neurodevelopmental disorder or might even be part of the extended phenotype of a micro-organism. But they do suggest that if we find something in the brains of our patients, we should ask our psychiatric anthropological colleagues to look for the same thing in the brains of the founders of new religious movements.

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Case reports

Dr Williams (2004) courageously urges the Editor to think again regarding his predecessor's joy at hastening the demise of the case report.

Medicine generally is being dehumanised; if psychiatry follows suit, then we cannot complain that the masses are deserting us for alternative medicine. The bias of the *Journal* towards so-called 'pure science' while discarding the whole-person approach will accelerate the dehumanising process. Following the massive development of drug therapy from the mid-1950s there was a golden era when the so-called controlled experiments supported what we clinicians found in practice – that people improved with antidepressants and anxiolytics.

For the past 40 years or so the *Journal* has been full of further 'research' papers which have added little of note to our psychiatric knowledge. At the same time much fruitful research has occurred in the clinical field through the analysis of narrative and transference and the study of case reports.

Uncommon Psychiatric Syndromes (Enoch & Ball, 2001), described by Littlewood (2004) as a pioneering book, now in its fourth edition and translated into several languages, arose from one case report, albeit followed by a further 35 years' (continuing) research.

The pioneer Dr Rolf Strom Olsen, Superintendent of Runwell Hospital, encouraged every clinician to be a researcher; to think and to contribute to the advancement of our subject. Following one ward round he informed a young senior registrar that the case report that he had just presented was an example of the rare 'delusion of doubles' and insisted that the world literature be scanned for other examples. Little did we think at that time that the Capgras syndrome would become such a prominent condition throughout clinical psychiatry during the next 40 years. The same can be said for de Clérambault syndrome, now the explanation for about 50% of stalkers (the fashion disorder of the age), and folie à deux, which explains much of the mass phenomena of the past half century.

Peter Hobson psychiatrist, experimental psychologist and psychoanalyst, protests effectively that successive editors rejected his papers on dynamic psychopathology as not being scientific enough. Hobson illustrates 'how easy it is for the science to squeeze out the subjective, personal dimension of life in the quest for objectivity . . .' (Hobson, 2002).

The case history reminds us that the person is not merely a statistic but comprises body, mind and soul and that each must be taken into consideration for complete healing to occur.

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One hundred years ago

Types of alcoholic insanity

An important discussion on the above subject was held at the Boston Society of Psychiatry and Neurology, an account of which is published in the *Journal of Nervous and Mental Disease* for December, 1904. Dr. H. W. Mitchell of Boston introduced the discussion in a paper based on the study of 148 patients (excluding cases of true dipsomania which exhibited no insane symptoms) at the Danvers Hospital for the Insane, or 13 per cent of the male patients admitted. The cases were grouped as follows: delirium tremens, acute and

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