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Schizophrenia and City Residence

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It has long been thought that rates of mental illness are higher in cities than elsewhere, because of crowding and resultant stress. In the case of schizophrenia, there are some marked exceptions to generally higher prevalence rates in industrialised cities. Factors such as migration, culture, infectious disease, demographic rates, and other social processes may affect geographical differences in rates. The excess of schizophrenia in central city areas has been given two opposing explanations – the 'breeder' hypothesis and 'social drift'. Data on incidence from three cities are compared, but do not reveal a clear common picture. Environmental factors connected with urban living are of two main types – social and non-social – which are not mutually exclusive; 'urban' may also have a variety of meanings. Rather than 'urbanicity' being an independent aetiological factor in schizophrenia, its effect may be largely explained in terms of migration and social class.

Theories linking increased rates of mental illness to the crowding and resultant stress which are regarded as typical of cities have a long history (Torrey & Bowler, 1991). They were popular in the 19th century, with speculation, for instance, that such geographical disparities were consequent on "the stresses incident to active competition" (White, 1903). A general association has in fact been found between city life and poorer mental health, both in children (Rutter, 1981) – though not relating specifically to schizophrenia – and adults (Blazer *et al*, 1985). For instance, schizophrenic patients living in urban boarding homes demonstrated more serious psychopathology than those in rural ones (Davies, 1989), and in North American long-term follow-up studies, outcome was better in rural than in urban settings (McGlashan, 1989). Prevalence rates of schizophrenia reported from urban areas, both in Europe and the US, have generally been significantly higher than those of rural areas, though with some marked exceptions, such as the Istrian peninsula and isolated areas of Norway (Andersen, 1975) and Iceland (Helgason, 1964). The prevalence of schizophrenia and rates of first admission for it are said to be higher than average in modern industrialised cities (Lewis *et al*, 1992). Paradoxically, the highest prevalence ever measured (17.4 per 1000 population) was reported from a thinly populated rural area of County Roscommon in the West of Ireland (Torrey, 1987), though there seems to be no evidence that this represented a higher-than-average incidence rate. What these anomalous rural areas may have as a common factor is a high proportion of unmarried males, living as farmers or rural labourers (Keatinge, 1987). Thornicroft *et al* (1993) found that a rural area of the Veneto had twice the treated incidence and prevalence of urban South Verona, but offered no explanation for this. On the other hand, neurotic and personality disorders have generally been found to have a random distribution in cities (Giggs, 1980; Stefansson, 1984), though the findings about them from Mannheim, discussed below, were different.

Prevalence rates are likely to be influenced by factors such as migration, infectious disease, fertility and mortality rates, culture, and other social processes (Eaton, 1986). Cooper & Sartorius (1977), seeking an explanation for the emergence of schizophrenia as a major disorder in the 19th century, pointed out that large populations had accumulated then on a hitherto unprecedented scale, mainly through migration. As a result, cases of severe mental illness, which would previously have remained scattered in rural societies, became aggregated in such numbers that they represented a major public health problem. Furthermore, because of the demands of industrialisation, many families became unable to manage psychotic relatives at home, where conditions were often very overcrowded, although this would have been possible in their previous agrarian settings. More recent relevant social processes include 'deinstitutionalisation'; there has been a widespread failure in Western countries to provide adequate community residences for schizophrenic patients leaving mental hospitals, so that they tend to drift into the poorest parts of cities, or even be resettled there by public services (Goldman, 1983).

However, from the point of view of aetiological factors, data on incidence are more valuable than those on prevalence – if more difficult to determine. Henderson (1988) points out that the non-random

distribution of the incidence of a disorder might be due to (a) environmental factors having a direct effect; (b) the disorder or its prodromata influencing where people live; or (c) a third set of factors prior to both (a) and (b). All these possibilities need to be examined in relation to schizophrenia.

Explanations for geographical differences in the frequency of schizophrenia have, in many cases, made use of a pair of opposite tendencies – 'social drift' and 'social residue'; these are both varieties of the social selection hypothesis, and both represent forms of social segregation. 'Social drift' is the migration of those affected by psychiatric morbidity to areas of a particular kind, where social demands on them may be less; it has been considered particularly relevant to schizophrenics, though alcoholics, drug addicts, people with marked personality disorders, and others with reduced social competence and affiliative bonds show the same tendency. Inner-city areas are the ones to which such affected individuals are most likely to migrate in Western countries, since these tend to contain both cheap, single-person accommodation and opportunities for casual but lowly paid work, while lacking any well-knit social structure which would make them feel unwelcome. At the same time, though, these environments are lacking in social support, which may operate as an adverse factor on mental health for some people. In many studies, the higher incidence of schizophrenia in central city areas has been more marked in men than in women, which suggests that social influences affect the two sexes differently in this disorder, e.g. that men may be more likely to drift centrally. However, a study in Greater Stockholm (Widerlöv *et al*, 1989) showed a stronger effect in females (see below).

On the basis of this view, the reason for the onset of schizophrenia is not that those affected live in environmentally unfavourable areas, but that people with schizophrenia, like others who are socially marginal, become unable to live in better residential districts. This would result in them moving to the central parts of a city, where they experience few social or emotional demands. Thus, being resident in a poor environmental area is not an aetiological factor in itself, but rather one associated with the development of the disorder. The 'drift hypothesis' has been used to describe, firstly, movement down the socio-economic scale (intra- and inter-generational), and secondly, geographical movement to more deprived urban areas; both processes have been related to the presence of schizophrenia. Goldberg & Morrison's theory of inter-generational 'social drift' (1963) was a hypothesis of great heuristic importance. However, it has been claimed (Dauncey *et al*, 1993) that this does not provide an adequate explanation for schizophrenics tending to be concentrated in central city areas, because the occupational classification that Goldberg & Morrison used is no longer appropriate, and because accurate data were missing at that time on the subjects' area of residence. These seem relatively minor objections, though.

The reverse of the social drift process, but a similar hypothesis, is that of 'social residue', i.e. that the mentally healthy migrate away from socially and environmentally undesirable areas, leaving the relatively incompetent behind (Freeman & Alpert, 1986*a*). The result of this differential migration, in which those affected by morbidity of any kind fail to participate in a general movement to better environmental areas, is that levels of morbidity rise in the central areas and remain low in suburban or new communities. One example was in British new towns, in one of which a relatively low rate of serious psychiatric morbidity was reported, but misinterpreted, in the earlier years (Taylor & Chave, 1964); it was in fact a consequence of the processes by which the town population was selected. Another example, showing the opposite aspect of the process, is the dramatic population fall of the industrial city of Salford, beginning in 1928, which was the sharpest for any local authority in England (Freeman, 1984); this resulted predominantly from the movement outwards of smaller family groups containing employed adults. Such a differential fall tends to leave behind those who are below average in social mobility, including the mentally and physically disabled, single-parent families, and the elderly; they have been described by Stromgren (1987) as a 'residual population'. As an example, Lei *et al* (1974) found in a Californian city that the highest concentrations of mental retardation were in areas where the population had been longest established.

The 'breeder' or social causation hypothesis, on the other hand, assumed that environmental factors are either causative of schizophrenia or have to be present for a predisposed individual to become ill. Dunham (1965) stated that this hypothesis was dependent on a series of propositions.

(a) Symbolic communication is essential for normal development, and lack of it leads to mental breakdown.
(b) In an urban community, certain areas have a greater amount of disorganisation than others.
(c) Social disorganisation is characterised by excessive mobility, ethnic conflict, breaks in communication, and lack of consensus.
(d) Seclusiveness is a key trait in schizophrenia.

(e) Persons who develop seclusive traits do so as a result of isolation or breaks in the communication process.
(f) These conditions are found in certain areas identified as being disorganised.

These propositions were described by Dunham as "not completely substantiated", which very much understates the objections to them. He added that such aetiological theories were "assumed to be adequate" and that the epidemiological evidence was then "often regarded as inferentially supportive of the original untested assumption". This is clearly very unsatisfactory from the scientific point of view.

Lewis *et al* (1992) describe the social drift hypothesis as having been little challenged for 25 years, in spite of the evidence for it being weak. However, it had in fact been questioned by several workers. Lapouse *et al* (1956) examined the movement of schizophrenic patients within a city over 20 years before the onset of psychotic illness, but found little evidence of downward drift, while a control group did not move significantly up the social scale, relative to them. Dunham (1969) stated that whereas the incidence of schizophrenia tended to be concentrated in central areas of Detroit in the 1930s, this was no longer so in the 1950s; the distribution of cases had become more dispersed. In Hamburg, Klusmann & Angermeyer (1987) found no concentration of first-admission schizophrenic patients in the central areas, though this was the case for readmissions. The difference between their findings and the original ones from Chicago was explained by the different historical development of the two cities, and by a trend towards greater environmental uniformity in urban areas. The Hamburg findings do in fact support the relevance of social drift for established cases of schizophrenia, but the latest data from Nottingham (Dauncey *et al*, 1993) are not confirmatory of the hypothesis in general.

Migration, however, is a difficult issue in relation to psychiatric morbidity, not least because there is evidence that both good and bad mental health may provoke a higher rate of mobility than the average, though for opposite reasons (Giggs, 1984). The two concepts of drift and residue make this dichotomy clear, and both may operate at the same time and place. The high prevalence recorded both for inner-city Salford (Freeman & Alpert, 1986*a*) and for rural areas of the west of Ireland (Torrey, 1987) may well have resulted to a significant extent from migration; in both places, the population fell to half an earlier level over a period of about 50 years, though the Irish fall began much earlier. Since economic migration of this kind inevitably has a differential character, it will tend to alter levels of morbidity in the populations affected by it. Another factor which may have to be considered is that foreign immigrants tend to concentrate significantly in the central areas of large cities (Herbert & Johnston, 1976).

Evidence from a number of different places will now be examined, with the object of throwing further light on the possible aetiological role of 'urbanicity'.

Evidence from Swedish national data

The hypothesis that the environment of cities can be directly related to a higher-than-average prevalence of schizophrenia has been proposed by Lewis *et al* (1992). They claim that studies supporting the drift hypothesis have failed to take account of the nature of the population at risk – the single, mobile, young people who make up a greater proportion of urban populations than of others. To test it, they investigated the association beetween place of upbringing and the incidence of schizophrenia, using data from a cohort of 49 191 male Swedish conscripts, 21% of whom had been brought up in one of the three urban centres of Sweden. These data came from the Swedish National Register of Psychiatric Care, which recorded 83% of all admissions in 1973, but was closed in 1983. Incidence rates were calculated for a follow-up period of 14 years. Diagnoses of neurosis, personality disorder, and alcoholism, as well as cannabis use and reports of 'feeling nervous', were commoner among those brought up in cities.

The incidence of schizophrenia among men brought up in cities was found to be 1.65 times the rate in men who had had a rural upbringing, despite adjustment for some other factors associated with city life but independent of the illness, such as use of cannabis, parental divorce, and family history of psychiatric disorder. The association of schizophrenia with urban life was also independent of family poverty, which in Sweden was commoner in rural areas.

To assess whether the effect of city upbringing was specific to schizophrenia, the strength of the association between urban upbringing and the development of the other psychoses was measured, and showed a similar, though weaker, trend. For schizophrenia, a strong linear trend was observed, with the highest rate in the cities, intermediate rates in large and small towns, and lowest rates in country areas. Since the *prevalence* of schizophrenia in Swedish cities is only about 1.5 times that in rural areas, genetic factors were considered very unlikely to explain this result. However, these findings did not distinguish between place of birth and place of upbringing.

Lewis *et al* (1992) state that their study design eliminated the possibility that geographical drift could explain any observed association between city life and schizophrenia; in that case, undetermined environmental factors, believed to be present in cities, must be assumed to have increased the risk. They seem in fact to have returned to a modified version of the original 'breeder' hypothesis.

Evidence from the United States

In the United States, Torrey & Bowler (1991) examined the geographical distribution of 'insanity' and schizophrenia for nine separate years between 1880 and 1963. The prevalence in north-eastern and Pacific Coast States was approximately three times that of those with a low rate (south-eastern and south central); this difference was remarkably consistent over the 83 years, despite changes in diagnosis (insanity or schizophrenia) and in the categories recorded (in-patients plus resident in the community, in-patients only, first admissions only). These authors also compared urban/rural ratios (percentage of population living in towns of 2500 people or more) in the different States with rates of insanity/schizophrenia, for nine years between 1880 and 1963; they found high correlations between mental illness and urbanicity (i.e. residence in an urban area (as defined)) for all years except 1963. Data on socio-economic status, ethnicity, and temperature for each State were also compared with first-admission rates and the resident hospitalisation rate (1963) for schizophrenia. These rates correlated positively with socio-economic status as well as with urbanicity, but not with the other two factors. This positive correlation between higher socio-economic status and admission rates was unexpected, but since high regional correlations between urbanicity and socio-economic status were also found, there may have been a confounding effect between the two variables. As in Sweden, the per capita income of people living in American cities is higher than it is for those living in rural areas.

Since the variation in rates between States was greater than that for affective, substance abuse, anxiety/somatoform, or antisocial personality disorders (Burnam *et al*, 1987), diagnostic differences would have to be remarkably consistent over a very long period to provide the explanation for this geographical effect. Malzberg (1955) had noted "a general progressive increase in rates from the rural areas through the urban groupings" (i.e. the larger the city, the higher the first-admission rate for schizophrenia), and Frumkin's (1954) study of first admissions to Ohio State mental hospitals reported that the urban rate was 1.9 times the rural. The only American study that did not find a clear urban excess of schizophrenia was by Lemert (1948) in Michigan, where not only Detroit, but several rural counties, had a high rate of admissions for schizophrenia.

Torrey & Bowler state that social drift is very unlikely to explain the State and regional differences in hospitalisation rates for schizophrenia that they found over this very long period. From the sociological point of view, a study of mental illness in Arkansas (Stewart, 1953) suggested that community pressure in rural areas makes it more difficult to commit people to State hospitals, while the nuclear family is stronger there and more likely to have accommodation for a psychotic member. However, New York State hospital admission data for 1949–1951 showed no difference in the duration of illness before admission for schizophrenics from rural and urban areas (Malzberg, 1955), and the belief that bizarre people are more easily accepted by the community in rural areas remains unproven. Such social factors, though, would not affect studies of prevalence rates which included both in-patient and community cases. Torrey & Bowler conclude that if a model of genetic predisposition combined with stress factors was to account for the geographical distribution of schizophrenia, it would be necessary to postulate either a selective migration of genetically predisposed individuals to both north-eastern and Pacific Coast States – and this was discarded as improbable – or else an urban stress factor that elicited the disease in genetically predisposed individuals in cities. That stress factor apparently remains unidentified.

The main methodological question raised by Torrey & Bowler's study is their criterion for 'urbanicity': the minimum population of 2500 is little more than a large village, so that the conflation of all populated areas above that size – including the largest cities – may be concealing important differences between subgroups with different orders of population. This has been a persistent problem in all studies attempting to link psychiatric disorder with urban living, and it is still unresolved.

Evidence from Nagasaki

Turning to the incidence of schizophrenia, data have been published from several cities, of very different characteristics, which will be discussed in turn. Firstly, an ecological study in Nagasaki by Ohta *et al* (1992) examined the possible influence of social class. Characteristics of residents (employment status, occupational and academic career, household status, length of residence, housing tenure, and the

proportion of young males in the population) were related statistically to the city's ecological structure. Contrary to some studies in Western cities, no significant differences were observed between different areas, and no significant relationship was found between incidence rates of schizophrenia and social class. However, schizophrenic men tended to have lived in the central parts for a long time and to have relatively low socio-economic status, whereas incidence rates were low in the areas where residents could afford to own private dwellings. As in many other studies, the findings for women were not as consistent as those for men. Ohta *et al* state that in Japan, differences in social class are not as marked as in Europe or the US, and those between residential areas not so clear. Japanese cities have not had long developmental backgrounds; since demolition and rebuilding have been frequent, areas containing poor housing have not had time to develop into residential districts with an established character. This work seems to indicate that both the environmental characteristics of Japanese cities and the social structure of Japanese society are so different from those of Western cities that conclusions about the frequency of schizophrenia cannot reliably be extrapolated from the one setting to the other.

Evidence from Camberwell

In Camberwell – an inner-city area of South London – for all first-contact cases of schizophrenia recorded between 1965 and 1984, patients were compared with matched non-psychotic patients and investigated for place of birth and paternal occupation (Castle *et al*, 1993). Compared with controls, the schizophrenic patients were more likely to have been born in the socially deprived area and to have had fathers with manual, as opposed to non-manual, occupations. The sample included the British-born children of Afro-Caribbean immigrants, but controlling for ethnicity did not alter the trends recorded. The results suggested, therefore, that individuals who develop schizophrenia are more likely than non-psychotic controls to have been born into socially deprived households. This would be associated with a greater risk of disadvantage both *in utero* and in early life. The favoured explanation was that some environmental factor of aetiological importance in schizophrenia is more likely to affect those born into households (a) of lower socio-economic status and (b) in the inner city.

Evidence from Chicago

In their classic study, Faris & Dunham (1939) found that first admissions for schizophrenia were concentrated in the city centre, and that the rate decreased in all directions towards the periphery. However, later findings in Detroit by Dunham (1965) did not confirm this finding, so that his view changed largely from a social causation to a social selection hypothesis. In later studies in Chicago, Levy & Rowitz (1973) found no clear-cut pattern for first admissions for schizophrenia; areas with high rates were scattered throughout the city. On the other hand, readmissions showed a marked excess in the poorer central areas, providing strong evidence for the 'social drift' explanation. These authors speculated that Faris & Dunham's findings might have been contaminated by the lack of general hospital psychiatric beds at the time their research was done, so that most middle-class schizophrenics would not have appeared in the statistics – being treated either at home or in distant private hospitals. The absence of these cases from the data might thus have led to a misleading impression of a central excess of new cases.

Evidence from Nottingham

In Nottingham, enumeration districts were statistically grouped into low-status and high-status clusters respectively; social deprivation emerged as mainly concentrated in the areas of post-1919 public housing and in some inner-city districts of privately rented housing. New cases of schizophrenia had been found to be significantly more likely to come from low-status areas, in contrast to manic–depressive cases, which were evenly distributed over the two types of district (Cooper *et al*, 1987). Dauncey *et al* (1993) related the lifetime geographical mobility of a group of 67 patients suffering from schizophrenia to the ecological structure of the city: a skewed ecological distribution was found to begin early in the lives of the patients, and to be well established at least five years before initial contact was made with the psychiatric services.

The sample consisted of virtually all new cases of schizophrenia during 1978–1980. Most patients were male and presenting in the 15–34 age range; 40% were either born outside the UK or had one or both parents born outside, but no particular migrant group predominated. The annual incidence rate in the most deprived area (0.18 per 1000) was three times that in the least deprived, so that the earlier findings on the same group of patients had persisted and could not be accounted for by the age structure of the population. If there was a process of geographical 'drift' affecting incidence, it would occur in the 'prodromal' period before the first psychiatric contact, but in fact, five years before presentation, there was still a trend (non-significant)

towards an excess of patients living in the most deprived areas, which contained only 18% of the at-risk population, but 33% of the patients – a statistically significant difference. The few patients who moved into the city in the five years before first presentation did not contribute to the skewed distribution. Of the 27 patients born in the central part of the catchment area, 23 originated in areas of social deprivation, this relationship being independent of ethnicity.

This study of a geographically defined and representative sample of patients making their first contact with psychiatric services was said to show that the incidence of schizophrenia correlated closely with levels of social and urban 'deprivation'; most of the poor zones were central, but rates also remained high in the more scattered pockets of deprivation in peripheral areas. Though some degree of 'drift' was likely, the baseline from which it took place was an already skewed pattern of residence, in that most cases originated in socially deprived areas. However, as in Hamburg, social drift could still be relevant to the distribution of established cases.

Evidence from Greater Stockholm

In the county of Stockholm, comparisons were made between urban, suburban, and rural districts in relation to all patients with long-term functional psychoses, including schizophrenia (Widerlöv *et al*, 1989). The urban district was characterised by a high rate of social problems, and had a younger population than the other two. The study cohort consisted of people aged 18–64 years who had been affected by a non-organic psychosis continuously for at least six months, showing psychotic features or residual symptoms during 1984, and who had lived in the catchment area during that year. The one-year prevalence rates of psychosis in the rural, suburban, and urban districts were 3.4, 5.6, and 6.6 per 1000 respectively.

There was also a gradient in the use of in-patient care between the different areas, with a rising level towards the city centre. Patients from the urban area spent a longer time in hospital than those from the other areas, and had more antipsychotic medication. However, involuntary admission was much less frequent in the urban area, where continuity of care was better than in the rural and suburban ones. The rural–urban gradient found in this study, particularly among females, was interpreted mainly in terms of migration – either of the social drift or social selection type. A possible, but untested, explanation of these differences was that affected individuals demonstrated more severe pathology in a more urban setting. However, there would also be a tendency for patients to be admitted from the rural district only in the more serious cases, because of the district's relatively long distance from the psychiatric in-patient unit.

Evidence from Salford

Salford, in the Greater Manchester conurbation, is a very old industrial area with a highly developed network of psychiatric facilities. Since 1968, the community has had a population-based case register, linked with all specialist psychiatric agencies serving the population. For each patient, it records – on a cumulative basis – the total use of psychiatric services, including long-term follow-up.

Freeman & Alpert (1986*a*) found higher than expected prevalence rates of schizophrenia for 1974, compared with other industrialised countries (Babigian, 1985; Jablensky, 1986; Häfner & an der Heiden, 1986). These were most marked in the middle-aged, and a relationship was proposed with selective population movements, combined with the low mortality and frequently chronic course of the disorder. Data on out-patients, in-patients, visits by mental health professionals, depot injections, etc. were derived both from the case register and from enquiries to general practitioners (GPs). These prevalence figures were then analysed in terms of distribution by electoral wards (Freeman & Alpert, 1986*b*); although in general this ecological method does not provide a satisfactory basis for epidemiological studies, it was felt to be useful in the case of Salford because both the social-class distribution of the population and characteristics of housing within its sub-areas were relatively homogeneous, compared with most cities. The data showed the city to be clearly divided on this basis into two separate zones – an inner one, in which all wards had a rate of schizophrenia above 5 per 1000 general population, and an outer one in which all wards had a rate below that figure. Although the outer zone contained the three wards with the highest social-class level in the city, this separation was by no means clearly along such lines, since the same zone also included several wards of low socio-economic status. Thus, ecological analysis showed no direct relationship between prevalence of schizophrenia and social class, but the group of wards with higher rates were those closest to the centre of the conurbation.

When the 1974 prevalence study was repeated for 1984, the point-prevalence rate of treated schizophrenia within this urban community had increased from 4.56 to 6.26 per 1000 adult population

during the decade (Bamrah *et al*, 1991). In 1974, the peak period-prevalence rate was for those aged 45–54 years, but ten years later, the rates peaked for both sexes in the 55–64-year age group – much more sharply in women. This is in contrast to earlier studies, which showed peaks at younger ages (Shur, 1988), but the same as Torrey's finding in Roscommon (1987). Thus, in some populations at least, the prevalence rate of schizophrenia is currently increasing, even if inception rates are average. In Salford, emphasis on reducing hospital stay had been compensated for by increased admission rates (mostly short-term) and greater support for patients living in the general community.

A number of factors might be responsible for such an increase in prevalence. In the first place, it seems likely that schizophrenic patients currently have a longer life-span than previously, possibly owing to improved care. Since this rate mainly takes into account individuals with established illnesses, short-term influences would probably be social and environmental, rather than biological. The general population fell by 25% in the decade, though by 1982–1984, some relative stability had occurred in this secular decline. The increase may therefore have reflected a continuing selective migration of healthy individuals out of the inner city. Whether or not this was the case, since the absolute numbers of schizophrenics present fell substantially between 1974 and 1984 (from 624–557), selective in-migration of previously affected individuals does not appear to have been a significant factor. People suffering from schizophrenia who are left behind in the inner city may be becoming progressively older and their cases more chronic.

The 'total' inception rate (i.e. of first contact with the case register) appeared to have decreased in the decade. This resulted from more patients maintaining contact with their carers than was previously the case, possibly because fewer were being discharged from follow-up or were defaulting. This greater continuity of care was made possible by the wide range of community services available, better co-ordination between them, and the precision with which cases were recorded. Both in-patient and out-patient facilities had improved, and there had been a major expansion in the community psychiatric nursing service, particularly within primary care, presumably improving liaison between psychiatrists and GPs. Although such systematic follow-up adds to the impression of chronicity of the sample, the case register had been fully operational for six years before the 1974 study, so that the results are unlikely to have been influenced by administrative factors.

This rise in prevalence appears to conflict with some recent findings which suggest that the frequency of schizophrenia is on the decline. However, such studies have dealt with first admissions or first contacts, and relate to populations which were different in character. In Salford, the population structure had altered considerably in the study period, with a sizeable reduction in overall numbers and a further increase in social deprivation and unemployment, thus enhancing its 'inner-city' character that has long been – though for largely unexplained reasons – associated with high rates of schizophrenia. If the inception rate had in fact fallen, this would add emphasis to the effect of differential outward migration; any effect of the disease process on prevalence, related to the accumulation of chronic cases, would be attenuated in this way.

Evidence from Mannheim

Häfner & Reimann (1970) determined the incidence of all psychiatric disorders for 20 areas within the city of Mannheim for 1965; significantly higher rates were found in socially disorganised areas, mainly in the centre, with lowest rates on the periphery, particularly in villages with strong community activities. Similar data for the period 1974–1980 were examined by Weyerer & Häfner (1989), who found that the ecological distribution of disorders had remained very stable, in spite of large urban renewal programmes in the inner city. Since 1965, the spatial concentration in the inner zone increased not only for schizophrenia, but also for neuroses and personality disorders – contrary to the findings of some other studies; however, there was no such concentration of affective psychoses. Alcoholism and other addictions were also strongly concentrated in the city centre during both periods. Using the factor ecology method, Maylath *et al* (1989) divided the 23 districts of Mannheim into five areas that were as homogeneous as possible for two principal components (segregation, density). All psychiatric disorders except affective psychoses and neurotic depression in 1974–1980 were found to be concentrated in areas that were centrally located and characterised by high population density, poor housing, low social status, and a high proportion of foreign residents.

Weyerer & Häfner (1992) examined whether the excess morbidity in the inner city in 1974–1980 could be explained by the high proportion of immigrants there. In fact, the high concentration in the inner zone was solely due to the German residents. This picture was similar to that of the prevalence data for 1974–1977, which showed that the prevalence of all psychiatric disorders was about twice as high among

the German population as among foreign residents, in the whole city. However, it contrasted with the findings of Giggs (1983) that in Nottingham, the incidence of schizophrenia among the foreign-born followed a classically zonal form, with the highest concentration in the central city. In Mannheim, the annual incidence of schizophrenia among the foreign-born in the inner zone (1.08 per 1000) was little different from that in the outer zone (0.98 per 1000) whereas for German residents, the figures were respectively 1.68 and 0.78 per 1000, and those for the intermediate zone were midway between these rates. Weyerer & Häfner point out that if environmental conditions in the inner zone have an aetiological effect in psychiatric disorders, "an explanation is needed of why they should only exert an effect on the German but not on the foreign residents". Between 1970 and 1978, a large proportion of the inner-city German residents moved out; although it is likely that these had a better health status than those who remained, there are no data to test this proposition. Unfortunately, the closure of the Mannheim psychiatric case register for political reasons has prevented these trends from being followed in more recent years.

Discussion

If there is an environmental factor, or group of factors, which is present more in cities than elsewhere and which has a direct positive effect on the incidence of schizophrenia, then there is a particular need to try and identify what it could be. Theoretically, such knowledge could provide opportunities both for primary prevention and for providing treatment at earlier stages of the illness.

Studies attempting to explain the uneven distribution of schizophrenia, both in environments and among the social classes, have mostly focused more on individual than on environmental factors, though individual genetic predisposition can be linked to the process of social segregation (Eaton, 1980). However, theories emphasising that environmental influences – both psychological stressors and poor physical health – relate to the experience of deprived inner-city living need not exclude the importance of biological factors. Similarly, hypotheses of a neurodevelopmental basis for schizophrenia (Murray *et al*, 1988; Castle & Murray, 1991), as well as the linking of maternal illness and birth trauma with schizophrenia are not incompatible with some environmental influence.

Jablensky (1988) has pointed out the difficulty which arises from the lack of a clear-cut distinction between 'social' and 'non-social' (or biological) environmental factors. However, since the action of the latter is mediated by social circumstances, the two types are not mutually exclusive, but interact with one another. Non-social environmental factors connected with urban living include exposure to lead or other heavy metals, air-polluting gases, toxic waste sites, and industrial effluents, as well as birth complications and a higher rate of infectious diseases, especially where there are many persons per household and/or room (Torrey & Bowler, 1991). Hollingshead & Redlich (1958) drew attention to the excess prevalence of schizophrenia in the socio-economic class V population of New Haven, many of whom were living in crowded tenements. Machon *et al* (1983) found that, in the case of their high-risk cohort, the season-of-birth effect was stronger in urban than in rural settings, and speculated that this was because urban mothers were more likely to acquire viral infections which could damage the foetal nervous system. Similar findings have been reported from Camberwell (Takei *et al*, 1992) and from Ireland, where the effect was more marked among females (O'Callaghan *et al*, 1992). Damage to the central nervous system from head injuries, which are more common in cities, may also be a factor increasing the incidence of schizophrenia (Wilcox & Nasrallah, 1987).

Social environmental factors which are more common in cities include stressful life-events, which represent a risk factor for the development of many psychiatric disorders, including schizophrenia (Brown & Birley, 1968; Brown & Prudo, 1981). Other urban factors that have been implicated include social isolation (Jaco, 1954), lack of control over the living environment – both isolation and overcrowding (Magaziner, 1988), and social overstimulation, which can provoke psychotic breakdown in vulnerable individuals (Wing, 1989). So far as crowding is concerned, though central city areas tend to contain more crowded accommodation than peripheral districts, any influence of density or crowding on schizophrenia or other psychiatric disorders remains controversial. This is partly through frequent confusion between the concepts of density (number of persons resident per square unit of ground) and crowding (number of persons per living unit); a high rate of one does not necessarily indicate a similar rate of the other. Lawrence (1974) did not find evidence for any simple causal relationship between density and socio- or psycho-pathology. Galle *et al* (1972) found that rooms per housing unit were an important predictor of admission to mental hospital, but this merely reflected the fact that people with more serious mental illness tended to live in single-room hotels, with many rooms per housing unit.

Schweitzer & Su (1977), who examined data on persons per acre, structures per acre, persons per household, and persons per room, in parts of Brooklyn, concluded that rates of household and family contact correlated significantly with rates of mental-hospital use. However, none of these studies was specifically concerned with schizophrenia, separately from other disorders, and none provides evidence on incidence. Scientific interest in the subject has fallen off because of methodological difficulties, including the need for longitudinal rather than cross-sectional studies and the narrow range of densities in modern industrial cities (Freeman, 1984).

As mentioned above, in the scientific study of these questions, the meaning of 'urban' or even 'city' also needs much clearer definition, because of the changes that have occurred worldwide since the 1950s. A fairly homogeneous community of 100 000 cannot be equated with a sprawling megalopolis of perhaps 15 million; settlements of the latter kind have been previously unknown in human history. Harpham (1993) points out that very little information is available on intra-urban differences in mental health, and that research on the social and economic characteristics of particular populations may be more productive than examination of their physical environments. The Japanese study by Ohta *et al* (1992) seems to add weight to this view. However, evidence from the US, reviewed above, indicates that – in that country at least – the relationship between schizophrenia and social class varies according to the size of the urban area. Kohn (1968) reported that the larger the city, the stronger the correlation between rates of schizophrenia and indices of lower social class, whereas this was not the case in small cities or suburban areas. However, in spite of the strong association between schizophrenia and a low social-class position, it has not so far been established that this is an aetiological relationship. This ecological correlation of schizophrenia with lower social class, demonstrated in the US only within large cities, would be at least partly explained by a double drift of individuals with schizophrenia – into the lowest socio-economic class and into a particular kind of urban area. No studies, though, have compared socio-economic status and prevalence rates in both rural and urban areas up to now.

Murphy (1976) suggested that any lack of evidence for significant urban/rural differences in psychiatric morbidity may not mean that the hypothesis of additional social stress in cities is wrong, but rather that the usual urban/rural dichotomy does not represent it. The reason suggested is that each large rural or urban area is too diverse to have the same impact from this point of view on all inhabitants. That point, however, was not made specifically about schizophrenia.

Though an association has been found between place of birth and the later development of schizophrenia, only the study of Astrup & Odegard (1961) distinguished between place of birth and that of later upbringing. Their results indicated only a weak effect of birthplace among those who had moved since birth, and so the effect of a city upbringing was said to be more likely to be important than that of place of birth.

If living in more urbanised areas is a risk factor for schizophrenia, then its prevalence might rise in developing nations that are currently undergoing rapid urbanisation, though no firm evidence of such a change has yet been obtained. Similarly, if industrialisation is also a major factor, one might expect prevalence to increase with modernisation (Eaton, 1974), but in Taiwan, at a time when this process was occurring rapidly, Lin *et al* (1969) found an actual decrease in schizophrenia over 15 years, despite an overall increase in psychiatric morbidity. It should not be assumed, though, that the full effects of urbanisation are necessarily seen in the short term: an incubation period might extend over several generations, during which the environment continues to change, so that there could be a persisting cultural lag (Swedish Government, 1971). Henderson (1988) points out that urbanisation, modernisation, regional variation, and secular change have shared components, and that it is very difficult to identify clearly defined factors within them. However, unless this is done, attempting to establish independent effects for any of them must remain controversial. Similarly, Murphy (1976) emphasised that discovering the social causes of psychiatric disorder means not only developing case-finding methods for the dependent variables, but also locating those events in society which are genuinely antecedent to the disorders, and are therefore independent variables. The evidence discussed above indicates that the first of these tasks is being adequately accomplished for schizophrenia, but that the second remains much more difficult.

The question discussed here is part of the wider issue of the role of social factors in the aetiology of schizophrenia. Jablensky (1986) concluded that there is only weak evidence for them having a direct effect of this kind: "Although social stress may be a trigger of the symptoms, schizophrenia does not have the characteristics of a stress reaction, or of a pattern of learned responses to threatening social situations." If that carefully supported view is accepted, then we would expect to see variations in prevalence between ecologically different areas for schizophrenia, but

little difference in incidence. In the view of most investigators, the balance of evidence leads to that conclusion, in which case 'urbanicity' would seem unlikely to survive as an independent variable in the aetiology of schizophrenia.

If there has in fact been a secular fall in the incidence and/or severity of schizophrenia in the last 30 years or so – though this is by no means generally accepted (Jablensky, 1993; Harrison & Mason, 1993) – then this might have taken a differential form in terms of sex, age-group, social class, etc. Waddington & Youssef (1993) found that in a rural area of Ireland, the morbid risk for schizophrenia was 37% less in those born during the period 1940–1969 than in those born during 1920–1939. However, the fall was much greater in females (−56%) than in males (−19%). A similar differential fall was recorded on the Danish island of Bornholm, when the rates for 1935 were compared with those for 1983 (Stromgren, 1987). If these findings are supported, any relationship of schizophrenia to city living might have altered over time as a result of such changes, thus confounding even more an already highly complex question.

Conclusion

The evidence from various situations that has been reviewed above does not lead clearly in any one direction. Dauncey *et al* (1993) suggest that such data are best explained as combining elements of the two social processes of 'breeding' and 'drifting'. In their view, the support that these data provide for a significant relationship between schizophrenia and a tendency for patients to originate in areas of urban deprivation points to a link between this disorder and an early environment of socio-economic deprivation. Similar findings have come from Camberwell (Castle *et al*, 1993). However, this need not be only the result of social influences, since the indices of deprivation involve a broad range of 'non-social' variables, such as obstetric complications, low birth weight, and maternal viral infections (Jablensky, 1988). Both maternal influenza and maternal dietary deficiency during pregnancy appear to be a much greater risk factor for subsequent schizophrenia in females than in males (O'Callaghan *et al*, 1991). So far as immigration is concerned, Harrison *et al* (1989) found that area of residence did not explain the higher rates of schizophrenia for Afro-Caribbeans in an urban setting, and in Mannheim there was no evidence of an environmental effect on the foreign-born.

The view that a factor of 'urbanicity' – the precise nature of which so far remains unknown – could be a significant aetiological influence in schizophrenia needs very careful appraisal. In the first place, it does not seem to take adequate account of the effects of migration, though that process is less relevant to incidence than to prevalence. For instance, because of the greater mobility in Swedish society since World War II, it is quite possible that social-residue processes would have reduced the healthy population (in all senses) within that country's cities, correspondingly raising the proportion living there who have some form of morbidity. The young men in the sample of Lewis *et al* (1992) who were born in cities may therefore have come disproportionately from families with either genetic or environmental handicaps – or both. Secondly, social class has not been fully separated from other influences; Lewis *et al* (1992) refer to low socio-economic status as an 'environmental' factor, which confuses the issue. Although 'inner city' areas are nearly always characterised by a social-class composition that is skewed heavily towards the poorest groups, urban populations in many countries have a more favourable socio-economic level than rural populations, as indicated in American and Swedish studies, for instance. Cabot (1990) states that epidemiological studies of schizophrenia have focused so much on urban areas (and to some extent on middle-class patients) that, "We know very little about first contact rates . . . among the rural poor of any country", though it is in precisely such areas that the high schizophrenic morbidity of Ireland is suspected to be. Thirdly, the influence of schizophrenic suicides, particularly in the younger age-groups, deserves further examination; if these occurred at different rates in urban and rural areas, prevalence rates could be affected. It is also likely that co-morbidity with alcohol and drug abuse, which appears to be an increasing problem in many countries, occurs at a higher rate in cities.

It may well be that when their effects have been fully explored, the two factors of migration and social class will provide much of the explanation for any urban excess of schizophrenia. Nevertheless, Hammer *et al* (1978) offered a warning that "migration, social class and ethnic marginality are too gross as social variables and too complex as social phenomena to implicate any particular mechanism that would account for their association with psychopathology". She and some other authors, however, have identified adverse change in people's social networks as a possible common mechanism underlying these processes, and this hypothesis remains to be fully examined.

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References

ANDERSEN, T. (1975) Physical and mental illness in a Lapp and Norwegian population. *Acta Psychiatrica Scandinavica*, suppl. **263**, 47–56.

ASTRUP, C. & ODEGARD, O. (1961) Internal migration and mental illness in Norway. *Psychiatric Quarterly*, **34**, 116–130.

BABIGIAN, H. M. (1985) Schizophrenia epidemiology. In *Comprehensive Textbook of Psychiatry IV* (eds H. I. Kaplan & B. J. Saddock). Baltimore: Williams & Wilkins.

BAMRAH, J. S., FREEMAN, H. L. & GOLDBERG, D. P. (1991) Epidemiology of schizophrenia in Salford, 1974–84: changes in an urban community over ten years. *British Journal of Psychiatry*, **159**, 802–810.

BLAZER, D., GEORGE, L. K., LANDERMAN, R., *et al* (1985) Psychiatric disorders: a rural/urban comparison. *Archives of General Psychiatry*, **42**, 651–656.

BROWN, G. W. & BIRLEY, J. L. T. (1968) Crisis and life changes and the onset of schizophrenia. *Journal of Health and Social Behaviour*, **9**, 203–214.

—— & PRUDO, R. (1981) Psychiatric disorder in a rural and an urban population. 1. Aetiology of depression. *Psychological Medicine*, **11**, 581–599.

BURNAM, M. A., HOUGH, R. L., ESCOBAR, J. I., *et al* (1987) Six-month prevalence of specific psychiatric disorders among Mexican Americans and non-Hispanic whites in Los Angeles. *Archives of General Psychiatry*, **44**, 687–694.

CABOT, M. R. (1990) The incidence and prevalence of schizophrenia in the Republic of Ireland. *Social Psychiatry and Psychiatric Epidemiology*, **25**, 210–215.

CASTLE, D. & MURRAY, R. M. (1991) The neurodevelopmental basis of sex differences in schizophrenia. *Psychological Medicine*, **21**, 565–575.

——, SCOTT, K., WESSELY, S., *et al* (1993) Does social deprivation during gestation and early life predispose to later schizophrenia? *Social Psychiatry and Psychiatric Epidemiology*, **28**, 1–4.

COOPER, J. E. & SARTORIUS, N. (1977) Cultural and temporal variations in schizophrenia: a speculation on the importance of industrialisation. *British Journal of Psychiatry*, **130**, 50–55.

——, GIGGS, J. & HOWAT, J. (1987) Schizophrenia and the ecological structure of Nottingham. *British Journal of Psychiatry*, **151**, 627–633.

DAUNCEY, K., GIGGS, J., BAKER, K., *et al* (1993) Schizophrenia in Nottingham: lifelong residential mobility of a cohort. *British Journal of Psychiatry*, **163**, 613–619.

DAVIES, M. A. (1989) Community adjustment of chronic schizophrenic patients in urban and rural settings. *Hospital and Community Psychiatry*, **8**, 824–830.

DUNHAM, H. W. (1965) *Community of Schizophrenia*. Detroit: Wayne State University Press.

—— (1969) City core and suburban fringe distribution patterns of mental illness. In *Changing Perspectives in Mental Illness* (eds S. C. Plog & R. B. Edgerton), pp. 337–363. New York: Holt, Reinhardt & Winston.

EATON, W. W. (1974) Residence, social class and schizophrenia. *Journal of Health and Social Behavior*, **102**, 627–633.

—— (1980) A formal theory of selection for schizophrenia. *American Journal of Sociology*, **86**, 149–158.

—— (1986) Epidemiology of schizophrenia. *Epidemiological Review*, **7**, 105–126.

FARIS, R. E. L. & DUNHAM, H. W. (1939) *Mental Disorders in Urban Areas*. Chicago: University of Chicago Press.

FREEMAN, H. L. (1984) The scientific background. In *Mental Health and the Environment* (ed. H. L. Freeman). London: Churchill Livingstone.

—— & ALPERT, M. (1986*a*) Prevalence of schizophrenia in an urban population. *British Journal of Psychiatry*, **149**, 603–611.

—— & —— (1986*b*) Prevalence of schizophrenia: geographical variations in an urban population. *British Journal of Clinical and Social Psychiatry*, **4**, 67–75.

FRUMKIN, R. M. (1954) Comparative rates of mental illnesses for urban and rural populations in Ohio. *Rural Sociology*, **19**, 70–72.

GALLE, O., GOVE, W. & MACPHERSON, J. (1972) Population density and pathology: what are the relationships for Man? *Science*, **176**, 23–30.

GIGGS, J. A. (1980) Mental health and the environment. In *Environmental Medicine* (eds G. M. Howe & J. A. Loraine). London: Heinemann Medical.

—— (1983) Schizophrenia and ecological structure in Nottingham. In *Geographical Aspects of Health* (eds N. D. McGlashan & J. R. Blunden), pp. 197–222. London: Academic Press.

—— (1984) Residential mobility and mental health. In *Mental Health and the Environment* (ed. H. L. Freeman). London: Churchill Livingstone.

GOLDBERG, E. M. & MORRISON, S. L. (1963) Schizophrenia and social class. *British Journal of Psychiatry*, **108**, 785–802.

GOLDMAN, H. H. (ed.) (1983) International perspectives in deinstitutionalization. *International Journal of Mental Health*, **11**, 3–165.

HÄFNER, H. & REIMANN, H. (1970) Spatial distribution of mental disorders in Mannheim, 1965. In *Psychiatric Epidemiology* (eds E. H. Hare & J. K. Wing). Oxford: Oxford University Press.

—— & AN DER HEIDEN (1986) The contribution of European case registers to research on schizophrenia. *Schizophrenia Bulletin*, **12**, 26–51.

HAMMER, M., MAKIESKY-BARRON, S. & GUTWIRTH, L. (1978) Social networks and schizophrenia. *Schizophrenia Bulletin*, **4**, 522–545.

HARPHAM, T. (1993) Urbanization and mental disorder. In *Principles of Social Psychiatry*, (eds D. Bhugra & J. Leff). Oxford: Blackwell.

HARRISON, G., HOLTEN, A., NEILSON, D., *et al* (1989) Severe mental disorders in Afro-Caribbean patients: some social, demographic, and service factors. *Psychological Medicine*, **19**, 683–696.

—— & MASON, P. (1993) Schizophrenia – falling incidence and better outcome? *British Journal of Psychiatry*, **163**, 535–541.

HELGASON, T. (1964) Epidemiology of mental disorders in Iceland. *Acta Psychiatrica Scandinavica*, **173** (suppl. 40), 1–258.

HENDERSON, A. S. (1988) *Introduction to Social Psychiatry*. Oxford: Oxford University Press.

HERBERT, D. T. & JOHNSTON, R. J. (eds) (1976) *Social Areas in Cities*. Chichester: Wiley.

HOLLINGSHEAD, A. B. & REDLICH, F. C. (1958) *Social Class and Mental Illness: a Community Study*. New York: Wiley.

JABLENSKY, A. (1986) Epidemiology of schizophrenia: a European perspective. *Schizophrenia Bulletin*, **12**, 52–73.

—— (1988) Schizophrenia and environment. In *Handbook of Social Psychiatry* (eds A. S. Henderson & G. D. Burrows). Amsterdam: Elsevier.

—— (1993) The epidemiology of schizophrenia. *Current Opinion in Psychiatry*, **6**, 43–52.

JACO, E. G. (1954) *The Social Epidemiology of Mental Disorders: a Psychiatric Study of Texas*. New York: Russell Sage Foundation.

KEATINGE, C. (1987) Schizophrenia in rural Ireland: a case of service overutilisation. *International Journal of Social Psychiatry*, **33**, 186–194.

KLUSMANN, D. & ANGERMEYER, M. C. (1987) Urban ecology and psychiatric admission rates: results from a study in the city of Hamburg. In *From Social Class to Social Stress* (ed. M. C. Angermeyer), pp. 16–45. Berlin: Springer-Verlag.

KOHN, M. L. (1976) The interaction of social class and other factors in the aetiology of schizophrenia. *American Journal of Psychiatry*, **133**, 177–180.

LAPOUSE, R., MONK, M. & TERRIS, M. (1956) The drift hypothesis in sociological differentials in schizophrenia. *American Journal of Public Health*, **46**, 978–986.

LAWRENCE, J. E. S. (1974) Science and sentiment: overview of respect on crowding and human behaviour. *Psychological Bulletin*, **81**, 712–720.

LEI, T. J., BUTLER, E. W., ROWITZ, L., *et al* (1974) An ecological study of agency labelled retardates. *American Journal of Mental Deficiency*, **79**, 22–31.

LEMERT, E. M. (1948) An exploratory study of mental disorder in a rural problem area. *Rural Society*, **13**, 48–64.

LEVY, L. & ROWITZ, L. (1973) *The Ecology of Mental Disorder*. New York: Behavioral Publications.

LEWIS, G., DAVID, A., ANDREASSON, S., *et al* (1992) Schizophrenia and city life. *Lancet*, **340**, 137–140.

LIN, T. Y., RIN, H., YEH, E. K., *et al* (1969) Mental disorders in Taiwan, fifteen years later. In *Mental Health Research in Asia and the Pacific* (eds W. Caudill & T. Y. Lin). Honolulu: East-West Center Press.

MACHON, R. A., MEDNICK, S. F. & SCHULSINGER, F. (1983) The interaction of seasonality, place of birth, genetic risk and subsequent schizophrenia in a high-risk sample. *British Journal of Psychiatry*, **143**, 383–388.

MAGAZINER, J. (1988) Living density and psychopathology: a re-examination of the negative model. *Psychological Medicine*, **18**, 419–431.

MALZBERG, B. (1955) The distribution of mental diseases in New York State 1949–1951. *Psychiatric Quarterly*, **29** (suppl. 2), 209–238.

MAYLATH, E., WEYERER, S. & HÄFNER, H. (1989) Spatial concentration of the incidence of treated psychiatric disorders in Mannheim. *Acta Psychiatrica Scandinavica*, **80**, 650–656.

MCGLASHAN, T. H. (1989) A selective review of recent North American long-term follow-up studies of schizophrenia. *Schizophrenia Bulletin*, **14**, 515–542.

MURPHY, J. E. (1976) Social causes: the independent variable. In *Further Explorations in Social Psychiatry*, (eds B. H. Kaplan, R. N. Wilson & A. H. Leighton). New York: Basic Books.

MURRAY, R. M., LEWIS, S. W., OWEN, M. J., *et al* (1988) The neurodevelopmental origins of dementia praecox. In *Schizophrenia: the Major Issues* (eds P. Bebbington & P. McGuffin). London: Heinemann.

O'CALLAGHAN, E., SHAM, P., TAKEI, N., *et al* (1991) Schizophrenia after prenatal exposure to 1957 A2 influenza epidemic. *Lancet*, **337**, 1248–1250.

———, COLGAN, K., COTTER, D., *et al* (1992) Evidence for confinement of winter birth excess to those born in cities. *Schizophrenia Research*, **6**, 102.

OHTA, Y., NAKANE, Y., NISHIHARA, J., *et al* (1992) Ecological structure and incidence rates of schizophrenia in Nagasaki City. *Acta Psychiatrica Scandinavica*, **86**, 113–120.

PASAMANICK, B. & KNOBLOCH, H. (1961) Epidemiologic studies on the complications of pregnancy and the birth process. In *Prevention of Mental Disorders in Children* (ed. G. Coplan). New York: Basic Books.

RUTTER, M. L. (1981) The city and the child. *American Journal of Orthopsychiatry*, **51**, 610–625.

SCHWEITZER, L. & SU, W. H. (1977) Population density and the rate of mental illness. *American Journal of Public Health*, **67**, 1165–1172.

SHUR, E. (1988) The epidemiology of schizophrenia. *British Journal of Hospital Medicine*, **7**, 38–45.

STEFANSSON, C. G. (1984) Map analysis of psychiatric services: the application of a computerized psychiatric case register to geographical analysis. *Acta Psychiatrica Scandinavica*, **70**, 515–522.

STEWART, D. D. (1953) A note on mental illness in rural Arkansas. *Social Problems*, **1**, 57–60.

STROMGREN, E. (1987) Changes in the incidence of schizophrenia? *British Journal of Psychiatry*, **150**, 1–7.

SWEDISH GOVERNMENT (1971) Urban conglomerates as psycho-social human stressors. Report to the UN Conference on the Human Environment, Stockholm.

TAKEI, N., O'CALLAGHAN, E., SHAM, P., *et al* (1992) Winter birth excess in schizophrenia: its relationship to place of birth. *Schizophrenia Research*, **6** (special issue), 102.

TAYLOR, S. & CHAVE, S. W. P. (1964) *Mental Health and Environment*. London: Longmans.

———, BISOFFI, G., DE SALVIA, D., *et al* (1993) Urban–rural differences in the associations between social deprivation and psychiatric service utilization in schizophrenia and all diagnoses: a case-register study in Northern Italy. *Psychological Medicine*, **23**, 487–496.

TORREY, E. F. (1987) Prevalence studies of schizophrenia. *British Journal of Psychiatry*, **150**, 598–608.

——— & BOWLER, A. (1991) Geographical distribution of insanity in America: evidence for an urban factor. *Schizophrenia Bulletin*, **16**, 591–604.

WADDINGTON, J. L. & YOUSSEF, H. A. (1993) Evidence for a gender-specific decline in rate of occurrence of schizophrenia in rural Ireland over a fifty-year period. *British Journal of Psychiatry*, **164**, 171–176.

WEYERER, S. & HÄFNER, H. (1989) The stability of the ecological distribution of the incidence of treated mental disorders in the city of Mannheim. *Social Psychiatry and Psychiatric Epidemiology*, **24**, 57–62.

——— & ——— (1992) The high incidence of treated disorders in the inner city of Mannheim. *Social Psychiatry and Psychiatric Epidemiology*, **27**, 142–146.

WHITE, W. A. (1903) The geographical distribution of insanity in the United States. *Journal of Nervous and Mental Disease*, **30**, 257–279.

WIDERLÖV, B., BORGÅ, P., CULLBERG, J., *et al* (1989) Epidemiology of long-term functional psychosis in three different areas in Stockholm County. *Acta Psychiatrica Scandinavica*, **80**, 40–46.

WILCOX, J. A. & NASRALLAH, H. A. (1987) Childhood head trauma and psychosis. *Psychiatry Research*, **21**, 303–307.

WING, J. K. (1989) The concept of negative symptoms. *British Journal of Psychiatry*, **155**, 10–14.

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