

Severe mental illness and social factors

This chapter examines the evidence about the relationship between poverty and severe mental illness, by which we mean psychotic disorders. In Chapters 2 and 3 the nature of poverty in the UK in the twenty-first century is discussed in detail. It is important to recognise from the outset that the concept of poverty is complex. Individual income is difficult to measure, and may not be as relevant as relative income, perceived disadvantage or social exclusion. It is easy to inadvertently conflate these concepts, and this is apparent in parts of the literature. In this chapter, some work is cited that uses the concepts of social disadvantage and deprivation rather than poverty per se. The movement between discussion of different types of social adversity should not be taken to imply that the differences between them are irrelevant.

On the face of it, it appears self-evident that being poor is bad for people's health. A strong association between poverty and ill health is well established for nearly all types of disease and in all parts of the world (Black *et al.*, 1993). The association between material deprivation and mental illness is just as strong as it is for physical illness. However, it is by no means universally accepted that this relationship is causal; that exposure to poverty leads to mental ill health. Much of the discussion in the psychiatric literature has taken social factors to have a modulating effect on more profoundly causative bio-genetic factors. It is only recently that the possibility of a strictly causal role for social factors has been seriously addressed within the mainstream psychiatric research literature. There is a body of opinion that has long preferred the view that a reverse causation applies, that mental illness causes poverty. The suggestion is that mental illness undermines people's ability to work and to maintain their social position.

Interest in non-biological factors has sharply increased in recent years with regard to schizophrenia in particular. The association between this disorder and social factors is very strong and consistent. A particular type of social factor, namely growing up in inner-city deprivation, seems to be especially powerful in conferring a heightened risk of schizophreniform disorders. For this reason we shall concentrate here on the evidence regarding schizophrenia.

2 Chapter 1: Severe mental illness and social factors

Mental illness and social environment

The theoretical orientation of British psychiatric practice and research has tended to be stable over long periods of time compared with the radical shifts in stance seen in US psychiatry. In Britain there has been a strong biomedical emphasis, with an accompanying interest in social environment that may reflect a pragmatism in our national intellectual traditions. The contemporary American emphasis on the biological, memorably described by a president of the American Psychiatric Association as the *bio-bio-bio* model (Sharfstein, 2005), appears strident and unbalanced from our perspective. It is easy to forget that not so long ago US psychiatry was dominated by psychoanalytic ideas, and that prior to that the US pioneered social psychiatry. In the twentieth century, social psychiatry entered the UK through the influence of an American, Adolph Meyer, on an Australian, Sir Aubrey Lewis (Shepherd, 1996). The UK may lead social psychiatry research now, but the tradition was born in the USA.

The systematic study of the social epidemiology of mental illness starts in the 1930s with the seminal work by Faris and Dunham in Chicago (Faris & Dunham, 1939). Using routinely collected information, they found that schizophrenia was far more common in the 'disorganised areas near the centre of the city' than in the suburbs (respective prevalences of 362/100,000 versus 55.4/100,000). They found a similar but less marked difference in the prevalence of 'senile psychoses and psychosis with arteriosclerosis' (in other words, dementia). They found no such difference in the prevalence of 'manic-depressive insanity'. They were interested in social causation. They reported that catatonic presentations of schizophrenia were commoner in the 'foreign born and Negro slum areas where poverty and culture conflict are combined', whilst paranoid and hebephrenic presentations were commoner 'in the rooming house areas of the city where the primary group has broken down and individuals live in social isolation'. At the time there was much debate about the meaning of these findings, but the idea that gained greatest currency was that living in social isolation in an inner city had a special role in provoking schizophrenia.

In 1956, Edward Hare published a study of the distribution of people diagnosed as suffering from mental illness in Bristol (Hare, 1956). Nearly 60 years after its publication this stands out as a high quality piece of social psychiatry research that identifies many of the methodological problems and core theoretical issues inherent to this field of research. Hare essentially attempted to replicate the work of Faris and Dunham. He recognised that there were some advantages in studying the epidemiology of mental illness in the UK. At that time the degree of social inequality in the UK was less extreme than in the USA, and the existence of a National Health Service that was used by a large proportion of the whole population meant that routinely collected information was likely to be more inclusive.

Hare gained access to the records of the public and private psychiatric hospitals serving the city of Bristol. He collected information on everyone who was admitted

for the first time between 1949 and 1953. The period straddled 1951, which was a year when a national census was conducted. The returns from this census gave socio-economic information by electoral ward (the smallest geographical administrative unit in the UK). Hare took the clinical diagnoses given to the patients and put them into five categories: schizophrenia, manic-depressive psychosis (which included severe depression), neurosis, senile dementia, and other (including alcoholism, personality disorder and epileptic disorders). He correlated the prevalence of these diagnoses with three social factors by electoral ward and for three large 'natural communities' within Bristol. The factors were:

- The proportion of single person households in the area. This was taken to be a rough measure of social isolation.
- Mean rateable (property tax) value of property in the area. This was based on an estimate of the notional value of the property, and could be taken to measure the social desirability and the wealth of the area.
- Population density. At the time this could not be easily derived from census data, and Hare had to use a sophisticated method to allow for, for example, the presence of parks in areas of otherwise high population density.

Hare acknowledged the limitations inherent to his research method, and these have continued to be apparent in later research using similar approaches. He had to rely on routinely collected data. When this was analysed and made available by the Office for National Statistics, it was organised by electoral ward. Electoral wards are not natural communities, and often have marked social heterogeneity within them. This can obscure important differences within the ward or across wards. He had to accept clinical diagnoses that may not have been made in a consistent way; this was 1956, many years before the development of structured clinical interviews or diagnostic criteria. As the data depended on contact with services, it is possible that there were differences in the proportions of people with mental illness who presented for treatment in different areas, which would create an apparent difference in prevalence.

In keeping with every other rigorous study conducted in the developed world that has ever been published, Hare found a markedly higher rate of schizophrenia in the inner-city area than elsewhere. He found no differences between areas in the rates of manic-depression, dementia or neurosis. He found that there was a strong association between rates of schizophrenia and living alone. This held true in areas of low and high rateable value and high and low population density.

There is much in Hare's study that is of historical interest. For example, he found the lowest rate of schizophrenia in suburban council estates. This would be unlikely to be true today, as the social composition and structure of social housing estates has changed markedly. His central conclusion was that two factors might account for his findings. Firstly, there might be a causal factor related to social isolation. Secondly, people with schizophrenia might be drawn to live in single person accommodation, which is concentrated in the inner city. Hare did not regard these possibilities as mutually exclusive.

4 Chapter 1: Severe mental illness and social factors

The subsequent literature has not confirmed Hare's findings in their entirety, but debate has continued over his two explanatory possibilities: people with latent or florid schizophrenia drift into poor social conditions (social drift) or certain types of social environment create a vulnerability to, or cause, schizophrenia. Attention has tended to focus on schizophrenia, because the social effect has appeared greatest for this condition. Whilst there is evidence that social deprivation is associated with mental illness of nearly all types, findings are less consistent for other disorders.

Social drift and urban drift

For a long time most psychiatrists believed that the question of social causation of schizophrenia was settled. In part this was because a paper published in 1963 seemed to have convincingly demonstrated that the association between deprivation and schizophrenia was due to social drift (Goldberg & Morrison, 1963). This described an impressively rigorous study with two arms. At that time the General Register Office was routinely notified of the name, date of birth, occupation and diagnosis of every patient admitted to a mental hospital in England and Wales. From these records a sample was taken of men aged between 20 and 34 years who were admitted to hospital for the first time in 1956 with a diagnosis of schizophrenia. Their centrally held birth certificates were examined. These included a record of the father's occupation at the time of the patient's birth. Thus it was possible to compare patients' socio-economic class (SEC) at the time of admission with their fathers' SEC at the time of their birth. The distribution of the fathers' SEC closely matched that of the UK population as a whole. The distribution of the patients' SEC showed a strong skew towards SEC V (manual and unskilled). This appeared to offer strong support for the social drift hypothesis.

However, Goldberg and Morrison recognised that whilst they seemed to have demonstrated that social drift occurs in association with schizophrenia, their method could not cast any light on the process by which this had occurred. The social class of the patients' fathers was recorded 20–34 years before the patients' current SEC was recorded. The method could not distinguish whether it was the patient or their father who had experienced a social decline. If the parental generation had drifted down the social scale, it might have been a causal factor for their children to develop schizophrenia. This would not constitute social drift. Instead it would reflect social selection into a low SEC of those at risk of schizophrenia. The 'clinical arm' of the study was intended to tease out these possibilities.

A sample of about 80 consecutively admitted men aged 15–29 with an agreed clinical diagnosis of 'definite schizophrenia' were subject to very close history gathering. Hospital and school records were examined. Parents were interviewed and occupational histories were obtained for each extended family. The findings were similar to the 'documentary' arm of the study. The patients' fathers showed a distribution of SEC similar to that for the local area. The patients' distribution of SEC was skewed towards SEC V. Furthermore, when paternal work histories were

examined longitudinally, the majority of fathers had experienced an improvement in social position in the patient's lifetime. This social mobility was markedly less for fathers living in the more socially deprived of the two areas studied.

Findings for fathers were broadly reflected in the findings for other relatives, who were said to match the social characteristics of the general population in the areas where they lived. Finally, the patients' school attainment was as good as, or better than, their brothers'. However, their brothers were functioning at a higher occupational level in adulthood. A number of the patients were found to have experienced a sharp occupational decline in the period immediately prior to their first admission to mental hospital. On follow up, the whole sample of patients showed a continuing occupational decline as their illness progressed. There is brief mention of a comparison sample of patients diagnosed as 'definitely not schizophrenic', who were said to have experienced little or no comparable social decline.

Goldberg and Morrison's study is impressive, and it is not surprising that their conclusion '*These findings suggest that gross socio-economic deprivation is unlikely to be of aetiological significance in schizophrenia*' (page 802) was widely accepted. Indeed, the evidence appeared to be so unequivocal that for decades no one felt it was worth replicating the study. However, whilst it is likely that both social selection and social drift do occur, more recent work strongly suggests that the rejection of a causal role for social deprivation in schizophrenia was premature. The contradiction between Goldberg and Morrison's findings and more recent findings may, in part, be due to an assumption that drift down the social gradient and drift into urban areas amount to the same thing. Goldberg and Morrison may have demonstrated social decline following the onset of schizophrenia, but they did not demonstrate that people who were mentally ill moved into inner-city areas as a consequence.

A much more recent Australian study has looked at internal migration from rural areas of people with physical and mental health problems (Moorina *et al.*, 2006). There was no evidence of migration of people with physical health problems from rural areas into cities. In fact, physically ill people migrated to cities less than the healthy population did. There was increased migration from rural areas into cities by people with mental illness, but only small numbers were involved. The authors of the paper speculated that migration might be related to the fact that inpatient facilities were exclusively located in urban settings. The urban drift might therefore be an artefact of the particular social geography of Australia, where there is an exceptionally high degree of urban centralisation of services of all sorts. Furthermore, people admitted to centralised facilities may be discharged to the surrounding area to facilitate follow up.

The Australian study illustrates that a simple assumption that social decline and urban drift go hand in hand may not be valid. It appears self-evident that chronic health problems are likely to have adverse effects on people's income and economic productivity. Goldberg and Morrison's finding of social decline in people diagnosed with schizophrenia may reflect a more profound deleterious

6 Chapter 1: Severe mental illness and social factors

effect of being diagnosed with that particular disorder. However, social decline does not automatically lead people to move to inner cities where the incidence of schizophrenia is especially high. Families tend to look after their sick relatives. They tend to work hard to keep them nearby. A large proportion of all people with schizophrenia would have to migrate into inner cities ('urban drift') in order to produce a systematic and substantially higher prevalence in those areas.

Deprivation and utilisation of mental health services

In the UK, health care is largely funded by the state. In the past there have been significant efforts to match levels of funding for services to levels of need in different areas. Attempting to achieve this was ambitious. Imperfect proxy measures had to be used. 'Need' is generally modelled by measuring utilisation of services, and there are factors other than the prevalence of mental disorder in the local population that influence this. For example, it might be that depression is more readily identified and treated in middle-class areas than in deprived inner-city areas for reasons of education, culture or expectation. Similarly, in order to produce a model that would predict need, social deprivation had to be measured through centrally collected data that covered the entire population. This usually meant the national census, which is conducted every ten years.

Glover and colleagues developed such a model in order to guide the rational distribution of resources across different areas (Glover *et al.*, 1998). Centrally collated service utilisation data were compared to measures of social disadvantage from the 1991 UK census using regression analyses. Different population factors predicted admission rates in rural and urban areas. A number of different variables were used in an effort to overcome this, in order to generate a single index, usable across the whole country. The index was calculated from the proportion of adults who were single, widowed or divorced (a measure of social isolation), or lacked access to a car (a measure of poverty), or who were permanently sick or unemployed, or lived in households which were not self-contained, or were staying in boarding houses, hostels or hotels on the census night. As this information was available for every electoral ward in the UK, it could be used to generate a Mental Illness Needs Index (MINI) score for each ward.

The MINI score has been shown to model mental illness need more accurately than other health need indexes, such as the widely used Jarman Underprivileged Area score (Jarman, 1984). The national average MINI score was set as 100. A severely deprived inner-city area might have a score in excess of 135; a wealthy area might have a score under 80. The intention to use MINI scores to rationalise the distribution of resources was never achieved to any great extent. It was one thing to identify those impoverished areas where the need was likely to be greatest. It was quite another to take resources from wealthier areas (with an articulate and politically astute population) and move them to poorer areas. In any case, MINI did not model need perfectly, as was acknowledged by its designers.

Firstly, although MINI scores predicted high levels of need in very deprived areas, the index tended to underestimate the extent of need in those areas. This may be due to flaws in the modelling of demand, or it may be that the relationship between deprivation and the prevalence of mental illness is not linear, or both (Croudace *et al.*, 2000). There are good reasons to suppose that deprivation is not a single continuously distributed variable. It may be that there is a specific type of inner-city deprivation that has a large impact on rates of serious mental illness. MINI may also underestimate need in areas with very low scores, owing to relatively high rates of use of private sector psychiatric services, which would tend to make some need invisible within centrally collated statistics.

Secondly, the effect of a rising MINI score is uneven across different types of mental health problem. Glover and colleagues (Glover *et al.*, 1999) compared the effect of MINI score (and two other mental health need indices) on three different levels of mental health need. These levels of need were aggregated by the type of service that the person used: 'primary care level' need, 'general secondary mental health care level' need and 'forensic mental health care level' need, each level being taken to represent a step up in the severity of mental disorder. The study was primarily concerned with the distribution of resources between services. Despite some acknowledged problems in the data, this mainly statistical study did convincingly show that deprivation had a larger impact on utilisation of forensic secure services than on general psychiatric services, and, somewhat less convincingly, that deprivation had a larger impact on demand for secondary care services than primary care services. There are a number of different ways of interpreting the findings. It might be that offenders or people prone to behavioural disturbance drift into deprived areas. It might be that the effect of social deprivation is to make people more aggressive, and therefore in need of higher levels of care when they become mentally unwell. It might be that deprivation has a causal role in severe mental disorder, but not in less severe illnesses. Any or all of these may be true.

Causation cannot be determined through studies of service utilisation. Nonetheless, they do demonstrate the consistent association between deprivation (especially in inner-city environments) and the most disabling forms of mental illness. Furthermore, the MINI score and its updates have been shown to have a strong correlation with not just service utilisation, but also with prevalence of both psychosis (Croudace *et al.*, 2000), as measured through numbers diagnosed rather than numbers admitted, and common mental disorders as measured in a community survey (Fone *et al.*, 2007).

It seems that deprivation has an impact on a wide range of mental disorders, with a larger effect on more severe disorder. It is unlikely that this is mainly due to a difference in the proportion of people with these disorders who present for treatment in different localities. The deprivation effect is large and consistent in the face of considerable differences in patterns and levels of service provision across geographical areas.

8 Chapter 1: Severe mental illness and social factors

From the 1980s there was a strong (though not universal) psychiatric consensus that schizophrenia is a brain disease, predominantly associated with genetic risk factors. Social risk factors for developing the disorder tended to be regarded as stressors that provoke an illness that has biological causes. Causation is always difficult to firmly establish for mental illnesses, but there is a growing body of evidence that indicates a causal role for social factors. It would be wrong to claim that it is firmly established that schizophrenia is predominantly caused by social adversity. However, there is a realistic possibility that this is the case.

Growing up in the inner city

The reawakening of interest in the nature of the effect of social factors in schizophrenia in recent years has arisen in part from the controversies over the much higher rate of schizophrenia amongst people in the UK whose parents (or grandparents) migrated from the Caribbean (Harrison *et al.*, 1999). This has led researchers to examine the impact of social environment in an increasingly sophisticated way, whereby ethnicity, migration and deprivation have been disaggregated. For example, a well-conducted study from the Institute of Psychiatry confirmed previous findings that when the morbid risk for relatives of White and African–Caribbean people suffering from schizophrenia were compared, first-generation migrants did not differ from the rest of the UK population (Hutchinson *et al.*, 1996). However, the siblings of second-generation migrants with a diagnosis of schizophrenia had a morbid risk seven times as high as their White counterparts. The finding had a high statistical significance.

The likeliest explanation for this startling finding is that second-generation migrants are exposed to an environmental factor that has a massive effect on their risk of developing schizophrenia. The biggest difference between first-generation migrants and their offspring is the location where they spent their childhood. There is further evidence for a strong social factor in the high risk of schizophrenia amongst British Black people. It appears that the impact of being a second-generation African–Caribbean growing up in the UK is greatest in those areas where Black and Ethnic Minority people comprise a smaller proportion of the total population (Kirkbride *et al.*, 2007). In other words, living in areas with a high proportion of people of the same ethnicity appears to confer a degree of protection against the risk associated with being a Black person who has grown up in the UK. None of these findings are easily reconciled with the idea that schizophrenia is primarily caused by genetic factors.

There have been important studies that have examined the effect of social deprivation during childhood. Harrison and colleagues conducted a simple but convincing study in Nottingham as part of a larger project on first-episode psychosis (Harrison *et al.*, 2001a). The participants were 82 people with a research diagnosis of either ‘broadly defined schizophrenia’ or ‘non-schizophrenic psychotic illness’ who were born in Nottingham and still lived there. Their birth certificates were

obtained, from which was recorded their father's SEC at the time of their birth, and their mother's address. From birth records, 198 controls were identified, matched to the cases for gender, date of birth and area of birth registration.

In the 1970s, Nottingham City Council had developed an index of need within the city, based on 1971 census data. This index was taken as a measure of social deprivation. As 1971 was the median year of birth, it is reasonable to suppose that the score on the index for the mother's address indicated the level of deprivation of the area during the participant's childhood.

For the purpose of the study, areas of Nottingham were divided into two groups; above or below average level of deprivation. This was then combined with paternal SEC at birth to create a three-level childhood deprivation score:

- 0 if father was social class I–III and mother lived in a non-deprived area.
- 1 if father was social class IV–V *or* mother lived in a deprived area
- 2 if father was social class IV–V *and* mother lived in a deprived area

There was a consistent association of adult-onset psychosis with paternal social class and area level deprivation. However, the combined childhood deprivation score showed a highly significant linear association with all psychoses and with broad schizophrenia. The association for non-schizophrenic psychosis was weaker. The size of the effect was substantial. People over 25 years who had a childhood deprivation score of 2 were much more likely to suffer from broad schizophrenia as those with a score of 0.

The Harrison study attempted to control for possible confounding factors, including ethnicity, family history, and drift into deprived areas (to exclude social drift or selection or segregation effects). Controlling for African–Caribbean ethnicity did reduce the size of the effect somewhat, but it remained large. The study, though lacking methodological sophistication and sufficient statistical power to answer all the relevant questions, stands in contradiction to the Goldberg and Morrison work on social drift. Here we have striking evidence suggesting that the social environment you grow up in has a profound effect on the chances of developing a major mental illness in adulthood.

There have been very long-standing suspicions that city life might be bad for your mental health, with a history stretching back to the nineteenth century. Until recently the evidence with regard to the differential effects of urban and rural poverty has been equivocal. Some ambiguity remains (particularly concerning disorders other than schizophrenia), but the weight of evidence is that growing up in urban deprivation has a particularly toxic effect. A systematic review of the literature between 1950 and 2009 on factors affecting the incidence of schizophrenia in England found that the relationship with urbanicity was one of the few consistent findings (Kirkbride *et al.*, 2012a).

A credible study has suggested that the considerable difference in the number of people with schizophrenia using services in rural Dumfries and Galloway and urban Camberwell (61% higher in Camberwell) might be entirely due to the presence in Camberwell of a large non-White population with a high risk of mental illness (Allardyce *et al.*, 2001). The White populations in the two locations

10 Chapter 1: Severe mental illness and social factors

had similar rates of schizophrenia. This finding runs against most of the evidence (for example, the Nottingham study cited above). The authors point out that excluding the non-White population from the Camberwell sample in order to calculate prevalence in a White sub-sample may well have disproportionately eliminated people with lower social class. An American study has suggested that the urbanicity effect on the prevalence of schizophrenia has been evident since at least 1880 (Torrey & Bowler, 1990). Cities have always grown through migration, but this has often been due to a movement of the rural population within a nation. It is difficult to reconcile the persistence of the epidemiological pattern with the idea that the excess of people with a diagnosis of schizophrenia in urban areas is an artefact of the number of offspring of migrants from overseas living there.

Perhaps the most important evidence that early exposure to urban deprivation has an association with later mental illness, at least with respect to schizophrenia, comes from cohort studies. These are mostly based on Scandinavian record systems which allow data linkage in large populations. A Swedish cohort of 49,000 national service conscripts has been much studied, as medical and psychiatric evaluations on conscription aged 18 years could be linked to later health records. Findings from a study on this cohort showed an incidence of schizophrenia 1.65 times higher in those who grew up in cities compared to those who had a rural childhood (Lewis *et al.*, 1992).

A study of the whole Swedish population aged 25–65 years found a relationship between population density and risk of mental illness. Those living in the most densely populated areas had 68–77% more risk of developing psychosis and 12–20% more risk of developing severe depression leading to hospital admission (Sundquist *et al.*, 2004).

The most compelling evidence on urbanicity is a Danish population study showing a dose–response effect (Pedersen & Mortensen, 2001). The greater the number of years that individuals were exposed to an urban environment before their 15th birthday, whether continuously or intermittently, the higher the risk of developing schizophrenia later. Furthermore, the intensity of the urban environment also had an effect.

The toxic factor

Although there are some contradictions within the literature, it is quite certain that rates of diagnosis of mental illness of most types are higher amongst poor people than the rest of the population. In addition to this, it seems that there is a special association between inner-city deprivation in childhood and adult schizophrenia. To find firm evidence of social antecedents for a mental illness that has been so widely regarded as having a biological basis is contrary to expectation. Despite the strength of the evidence, it is possible that the effect of urban deprivation may yet prove to be due to some unknown confounding factor. This seems unlikely to us. Urban deprivation can be regarded as an established causal factor. It is worth