Social experience and psychosis
Insights from studies of migrant and ethnic minority groups*

CRAIG MORGAN and PAUL FEARON

Abstract. In this paper we aim to provide an overview of initial findings from the UK ESOP study concerning ethnicity, social risk factors and psychosis, and to set the findings from this study within the context of other related research. Our focus is primarily on the UK African-Caribbean population. ESOP is a multi-centre population based incidence and case-control study of first episode psychosis, conducted initially over a three-year period. The study sample comprises: a) all patients with a first episode of psychosis who presented to secondary and tertiary services within tightly defined catchment areas in south-east London, Nottingham and Bristol, UK over defined time periods; and b) a random sample of healthy community controls. Findings from the ESOP study to date have confirmed that the African-Caribbean and Black African populations in the UK are at increased risk of schizophrenia and other psychoses, compared with the White British population. Analyses of data relating to social risk factors suggest that various forms of early childhood and adult adversity, and neighbourhood characteristics, including ethnic density, may be particularly important in contributing to increased risk in these populations. These data suggest that adverse social experiences may be aetiologically relevant in schizophrenia and other psychoses. A more complete understanding of these factors may help us to clarify why there are differences in rates between populations.

INTRODUCTION

Kraepelin was an early advocate of cross-cultural comparisons of mental disorder. He argued that: “There are two basic ways in which comparative studies may help advance our knowledge. On the one hand they can throw light on the causes of mental disorder, and on the other they provide a means of determining the influence which the patient’s personality exerts on the particular form his illness assumes” (Kraepelin, 1904). To these ends, during a trip to Java in 1904, Kraepelin collected accounts of mental disorder and observed hospitalised patients. Kraepelin’s basic rationale for making cross-cultural comparisons remains valid, and similar reasoning underpins much current comparative research. Research comparing the incidence of schizophrenia and other psychoses, for example, in migrant and ethnic minority groups with that in host and majority populations promises to shed new light on the aetiology of these disorders. In particular, such research is contributing to renewed interest in the role of adverse social experiences and circumstances in the onset of psychotic mental illnesses (e.g. Harland et al., 2004; Cooper, 2005; Selten & Cantor-Graae, 2005).

The high incidence of schizophrenia in migrant and ethnic minority groups is one of the most consistent findings in psychiatric epidemiology (Cantor-Graae & Selten, 2005), and the most striking, and perhaps well-known, example is that of the African-Caribbean population in the UK (Sharpley et al., 2001). In 1997 the UK Medical Research Council established the ESOP (aetiology and ethnicity in schizophrenia and other psychoses) study to investigate the apparent high rates of schizophrenia and other psychoses in this population, the aims being: 1) to establish a large population based, first contact case-control study of psychosis in which to test hypotheses concerning social and biological factors which might explain the increased incidence of schizophrenia in the African-Caribbean population; and 2) by determining the causes of the high incidence in this population, to throw light on the aetiology of schizophrenia in general. In this review, we set initial findings from this study within the context of other related research and argue that there is now increasingly robust evidence that social adversity across the life course contributes to the increased rates of psychosis in the African-Caribbean (and other migrant and ethnic minority) population.

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THE AESOP STUDY

AESOP is a multi-centre population based incidence and case-control study of first episode psychosis, conducted initially over a three-year period from September 1997 to August 2000. The study sample comprises: a) all patients with a first episode of psychosis (F20-F29 and F30-F39 (psychotic codings) in ICD-10) who presented to secondary and tertiary services within tightly defined catchment areas in south-east London, Nottingham and Bristol, UK over defined time periods; and b) a random sample of healthy community controls. Full details of the methodology can be found in Kirkbride et al. (2006), Fearon et al. (2006) and Morgan et al. (in press).

During the study period, we identified 592 cases (330 in south-east London; 205 in Nottingham; 57 over 9 months in Bristol), and 412 controls (183 in south-east London; 208 in Nottingham; 21 in Bristol), a total of 1004 subjects. Table I summarises the basic demographic characteristics of the study sample by case-control status and study centre, and Table II breaks the sample of cases down by diagnosis.

Table I. Basic characteristics of the AESOP sample.

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<thead>
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<th>London</th>
<th>Nottingham</th>
<th>Bristol</th>
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<tbody>
<tr>
<td></td>
<td>Cases</td>
<td>Controls</td>
<td>Cases</td>
</tr>
<tr>
<td>Age (mean ± SD)</td>
<td>31.0±10.5</td>
<td>36.1±11.3</td>
<td>30.3±11.2</td>
</tr>
<tr>
<td>Male, N (%)</td>
<td>186 (56.7)</td>
<td>67 (36.6)</td>
<td>122 (59.5)</td>
</tr>
<tr>
<td>White British, N (%)</td>
<td>78 (23.6)</td>
<td>76 (41.5)</td>
<td>151 (73.7)</td>
</tr>
<tr>
<td>African-Caribbean, N (%)</td>
<td>126 (38.2)</td>
<td>51 (27.9)</td>
<td>27 (13.2)</td>
</tr>
<tr>
<td>Black African, N (%)</td>
<td>66 (20.0)</td>
<td>21 (11.5)</td>
<td>3 (1.5)</td>
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</table>

Table II. Distribution of diagnoses in the AESOP patient sample.

<table>
<thead>
<tr>
<th></th>
<th>London</th>
<th>Nottingham</th>
<th>Bristol</th>
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<tbody>
<tr>
<td>Non-affective psychoses</td>
<td>248 (75.2)</td>
<td>140 (68.3)</td>
<td>40 (70.2)</td>
</tr>
<tr>
<td>Manic psychosis</td>
<td>45 (13.6)</td>
<td>26 (12.7)</td>
<td>6 (10.5)</td>
</tr>
<tr>
<td>Depressive psychosis</td>
<td>37 (11.2)</td>
<td>39 (19.0)</td>
<td>11 (19.3)</td>
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INCIDENCE RATES

The migration of substantial numbers of African-Caribbeans to the UK, particularly to urban centres such as London and Birmingham, occurred largely in the 1940's and 1950's. Post-war economic prosperity in the UK and the consequent promise of work and income was the primary factor driving the decision of many to migrate, often with family migrating later. The earliest studies reporting rates of mental illness in the UK among migrants from the Caribbean were published in the early to mid-1960's, and since then there have been close to twenty studies that have investigated the incidence of psychosis or, more specifically, schizophrenia in the African-Caribbean population (both first and subsequent generations). Without exception each has reported higher incidence rates than in the White population – between 2 and 14 times higher (Fearon & Morgan, 2006). However, for all the consistency, there have remained nagging doubts about the validity of the findings, mainly because of continuing concerns regarding a) the accuracy of denominator data for ethnic minority groups; b) completeness of case ascertainment; and c) diagnostic validity across ethnic groups.

The AESOP study has both replicated and extended these previous findings (Fearon et al., 2006). Using data from the 2001 UK census as the denominator, which has perhaps the most accurate population size estimates to date for ethnic minority groups in the UK, we found the incidence of all psychoses to be significantly higher in the African-Caribbean population across all three centres compared with the baseline White British population (African-Caribbeans: IRR (Incidence Rate Ratio) 6.7 (5.4-8.3)). This difference was most marked for narrowly defined schizophrenia (ICD-10 F20) and manic psychosis (ICD-10 F30-31). For example, after adjusting for age,
the incidence of schizophrenia across the three study centres was nine times higher in the African-Caribbean population (IRR 9.1 (6.6-12.6)). The incidence of manic psychosis was eight times higher (IRR 8.0 (4.3-14.8)). Further, in line with some previous studies, the incidence of all psychoses was also markedly elevated in the Black African population (IRR 4.1 (3.2-5.3)), again this being highest for schizophrenia (IRR 5.8 (3.9-8.4)) and manic psychosis (IRR 6.2 (3.1-12.1)). Black African refers to migrants from sub-Saharan Africa and their children; generally, this is a population of more recent migrants.

Intriguingly, the incidence rates for all psychoses were also raised for all other ethnic groups (other White, Asian, mixed, other) compared with the White British population, albeit much more modestly (IRRs for all psychoses ranged from 1.5 to 2.7). These findings mirror closely those of a meta-analytic review of all studies of migration and psychosis by Cantor-Graae & Selten (2005). They found an increased rate of psychosis for all migrant groups of around 2.7, with this increasing to around 4.8 for migrants (first and subsequent generation) from countries where the majority population is black.

PHENOMENOLOGY

Before moving on to address the question of why there are high rates, it is worth noting some preliminary findings from the ÆSOP study concerning the prevalence of specific symptoms across ethnic groups. It is interesting to ask whether there are any differences in the clinical profiles of cases from different ethnic groups diagnosed with a psychotic mental illness. In the ÆSOP sample, two intriguing differences are evident in preliminary analyses.

First, the prevalence of persecutory delusions in the White British, African-Caribbean and Black African samples varied markedly. Persecutory delusions were common in all groups, but they were more common among both the African-Caribbean and Black African cases, compared with White British cases. In the White British group, 58% experienced persecutory delusions during a first episode of psychosis compared with 70% of African-Caribbeans and 74% of Black Africans, both statistically significant differences. When the data are stratified by sex, age and diagnostic group, these differences persist (Demjaha et al., 2006). This finding requires confirmation in further analyses.

Second, when looking at the prevalence of Schneiderian first-rank symptoms (FRS) in each ethnic group by diagnosis, a further interesting difference emerges. We found that the prevalence of first-rank symptoms were more common among African-Caribbeans and Black Africans with a diagnosis of affective disorder than White British (Ihara et al., submitted for publication); the reverse was the case for schizophrenia. A caricature of these findings is that schizophrenia seemed less ‘Schneiderian’ in the black than in the white group, whereas the phenomenology of affective psychoses included FRSs more commonly in the black subjects. This is similar to what Kirov & Murray (1999) found in a small study of patients attending a lithium clinic.

These data hint at the possibility that there may be ethnic differences in the clinical profile or the phenomenology of psychosis. This is not a novel suggestion. There is some evidence from studies of the outcome of psychosis that African-Caribbeans are more likely to experience a remitting course, with fewer negative symptoms and more positive and manic symptoms (e.g. McKenzie et al., 1995). We will return to this.

ADVERSE SOCIAL EXPERIENCES AND PSYCHOSIS (1) BACKGROUND

If it is accepted that there are genuinely raised rates of schizophrenia and other psychoses in the African-Caribbean and Black African populations in the UK (and there are those who are still sceptical), the challenge is to understand why. The most likely explanation, according to most commentators, is that it is something to do with the social environment in the host country. In support of this broad conclusion a number of observations are often made, notably that there is no evidence that any of the more biological risk markers for schizophrenia (e.g. obstetric complications, viral infections) are more common or have a greater effect in the African-Caribbean population (Fearon & Morgan, 2006). A further notable observation frequently made is that studies of the incidence of schizophrenia in the Caribbean have not found rates as high as for the African-Caribbeans in the UK, the interpretation of this being that population differences in genetic risk are unlikely to account for the increased incidence in the UK. However, there is a need for some caution here. Table III provides a comparison of the incidence rates for schizophrenia found in the ÆSOP study for White British and African-Caribbean subjects, and the incidence rates found from the studies conducted in Jamaica (Hickling & Rodgers-Johnson, 1995), Trinidad (Bhugra et al., 1996) and Barbados (Mahy et al., 1999), the main Caribbean islands from which people migrated to the UK in the 1940’s and 1950’s.

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(2001)

An intriguing study by Boydell et al. (2004) found a median incidence of 1.5 per 10,000, based on 100 incidence studies.

Table III. – Reported incidence rates for narrowly defined schizophrenia per 10,000 per years.

<table>
<thead>
<tr>
<th>Area</th>
<th>Incidence Rate per 10,000 per years*</th>
<th>95% Confidence Interval</th>
</tr>
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<tbody>
<tr>
<td>AESOP: White British</td>
<td>0.7</td>
<td>(0.58-0.87)</td>
</tr>
<tr>
<td>AESOP: African-Caribbean (from Fearon et al., 2006)</td>
<td>7.1</td>
<td>(5.16-8.98)</td>
</tr>
<tr>
<td>Barbados (from Mahy et al., 1999)</td>
<td>2.8</td>
<td>(1.97-3.70)</td>
</tr>
<tr>
<td>Trinidad (from Bhugra et al., 1996)</td>
<td>1.6</td>
<td>(1.05-2.12)</td>
</tr>
<tr>
<td>Jamaica (from Hickling &amp; Rodgers-Johnson, 1995)</td>
<td>2.1</td>
<td>(Confidence intervals not given)</td>
</tr>
</tbody>
</table>

*The WHO Ten Country study found rates of narrowly defined schizophrenia that ranged from 0.7 to 1.4 per 10,000 per years (Jablensky et al., 1992). The meta-analysis by McGrath et al. (2004) found a median incidence of 1.5 per 10,000, based on 100 incidence studies.

The rates reported in the Caribbean are not as high as those for the African-Caribbean population in the UK. However, nor are they as low as for the White British population in the UK. They lie somewhere between. Of course, this does not necessarily hint at any one potential explanation, genetic or social, but what these figures suggest is that more cross-cultural comparisons are needed. Indeed, intriguing provisional findings from a more recent study in Trinidad, which used the same design as the AESOP study, suggests Afro-Trinidadians are at an increased risk of developing a psychotic mental illness compared with Indo-Trinidadians (Hutchinson & Murray, 2006). It may be that variations between non-White groups will prove particularly instructive in understanding why some groups are at such an increased risk; such variations merit much more detailed study.

What are the potential social factors that might account for the raised incidence of psychosis in the African-Caribbean (and other minority) population? A number have been proposed: socio-economic disadvantage; experiences of racism and discrimination; and social isolation. However, very little research has actually investigated hypotheses concerning specific risk factors directly. An intriguing study by Boydell et al. (2001) found that rates of schizophrenia were highest among African-Caribbeans when they lived in areas where they formed a relatively smaller proportion of the population; findings that hint at a possible role for social exclusion, discrimination and isolation. Recently, this finding has been replicated for migrant groups in the Netherlands (Veling et al., 2006).

Some further clues are offered by a small case-control study of first-episode schizophrenia, conducted in Camberwell and Ealing, UK, which was in effect the precursor and pilot for the AESOP study. In this study, Rosemarie Mallett and colleagues found that two factors were associated with an increased risk of schizophrenia in the African-Caribbean group in particular: unemployment and long-term separation from parents before the age of 17. They tentatively concluded from this: “... the impact of migration and urban living may act to fragment social networks and family life ... [leaving African-Caribbeans, in particular, more vulnerable to developing schizophrenia]” (Mallett et al., 2002). When Cooper (2005), in a recent editorial, considered the kinds of social pathogens that might be implicated, he identified both aspects of early childhood adversity and adult disadvantage, echoing the findings from Mallett and colleagues small scale study. In short, previous research points to the potential importance of early childhood and adult adversity, and to neighbourhood characteristics. Each of these has been investigated using data from the AESOP study.

ADVERSE SOCIAL EXPERIENCES AND PSYCHOSIS (2) AESOP FINDINGS

We examined the relationship between long-term separation from, and loss of, a parent before the age of 16 in the AESOP sample (Morgan et al., 2007). Two intriguing findings have emerged. First, in the whole AESOP sample, cases were 2-3 times more likely to have been separated from a parent because of family breakdown, or to have lost a parent, before the age of 16. These findings held independently of a number of potential confounders, including a parental history of mental illness. Secondly, the effect of separation on risk of adult psychosis was very similar in White and African-Caribbean subjects. However, African-Caribbean controls were more likely to have been separated from a parent early in life than were White British controls (African-Caribbean: 31% (n = 22) v. White British: 18% (n = 45) (χ² 4.98, p = 0.03)). Consequently, the population attributable risk for separation from parents was higher for African-Caribbeans (37%) than for White British (19%), a finding which suggests early separation may have a greater impact on rates of psychosis in the African-Caribbean population. Preliminary analyses of long-term markers of adult social adversity and exclusion (e.g. unemployment, living alone, limited social networks) show similar patterns, i.e.

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a basic association with an increased risk of psychosis and a greater prevalence in the African-Caribbean population. There was also clear evidence of a dose response relationship, such that the risk of psychosis increased linearly with increasing number of markers of adult adversity (Morgan et al., 2005). Further analyses contribute to the suggestion that socio-economic risk factors may be operating not just at the level of the individual, but also at the level of the community. For example, Kirkbride et al. (in press) found that risk of psychosis is highest for African-Caribbeans when they form a smaller proportion of the population, a finding that partially replicates an earlier finding by Boydell et al. (2001).

Starting with early separation from parents, it seems reasonable to suggest that what matters are the circumstances leading up to separation and the consequences of separation. That is, aberrant separation, e.g. resulting from family breakdown, may be a marker for: a) prior family discord or other difficulties; and b) subsequent adversity, be these related to financial and economic strains on the household or difficulties in forming attachments. Early separation may, therefore, be better thought of as a risk marker or indicator. Further, early childhood adversity of various kinds has been linked to the full range of mental illnesses, and it may be that such adversity or difficulties create a general vulnerability to later mental illness, the precise form depending on a range of other interacting factors, both social and biological. The precise mechanism aside, what these data suggest is that adversity linked to family breakdown during childhood may be important in understanding psychosis in general and the high rates of psychosis among African-Caribbeans in particular. This further ties in with other recent research linking various forms of early adversity and psychosis (see Morgan & Fisher, 2007).

Turning to adult social exclusion or isolation, proposing a link between social isolation or adversity and psychosis is not new. However, as with all studies linking adult social adversity and psychosis, there is the seemingly intractable problem of disentangling cause and effect. While the data analysed in AESOP relate to long term indicators of isolation, the relationship between these and psychosis is no doubt confounded by the illness prodrome, one well documented consequence of which is social withdrawal. This caveat notwithstanding, the patterns found in the data of a dose-response relationship and of a higher prevalence of exclusion in African-Caribbeans are arguably more suggestive of a causal role for these factors. Again, however, the mechanism is not clear. It may be that isolation and exclusion are vulnerability factors, indicating a lack of social resources for coping with adverse life events and, in the case of ethnic minorities, chronic discrimination.

CONCLUSIONS

For at least the past decade, the dominant view has been that schizophrenia is primarily a genetic brain disorder. On the basis of the social distribution of schizophrenia and other psychoses, the data presented here, and other recent work, however, it seems that social experience matters. The circumstances of our lives and the nature of our interactions with others and institutions can, it seems, increase or decrease our chances of developing a psychotic illness. This is not in any sense to deny the importance of non-social factors. What has become clear is that psychosis is the outcome of a series of interactions (biological and social) over the life course. It seems, however, that social experience may be a major factor in understanding why there are high rates among African-Caribbeans – and most likely other migrant and ethnic minority groups. Recently, there have been attempts to develop overarching theoretical frameworks for understanding the role of social factors in the aetiology of psychosis, particularly in relation to migrant groups: e.g. Selten & Cantor-Graae’s (2005) model of social defeat.

Let us finish with one final bit of speculation. A further intriguing question is whether psychosis caused predominantly by adverse social experiences over the life course has a distinct phenomenology. Does the continued experience of adversity, such as discrimination, particularly in the absence of coping resources, leave individuals more vulnerable to paranoia and persecutory delusions? Is there a greater affective component to psychoses with a significant social component, even in the presence of Schneiderian first-rank symptoms? The preliminary data presented above regarding the ethnic distribution of certain symptoms very tentatively supports this.

Returning to Kraepelin’s view that cross-cultural comparisons can throw light on the aetiology of mental illness, one product of studies of psychosis in migrant and ethnic minority groups is that the importance of social experience in a series of life course interactions is made clearer. Intriguingly, the involvement of different aetiological factors may give rise to different illness phenomenology. Together, these possibilities have important potential implications for treatment, most particularly for the treatment of patients from ethnic minorities. In an era of major population movements across the world, these are issues of urgent importance.

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APPENDIX

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