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Increased incidence of psychotic disorders in migrants from the Caribbean to the United Kingdom

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ABSTRACT

Background. Several studies have replicated the finding of increased incidence of schizophrenia and related psychoses in first and second generation migrants from the Caribbean. The finding has remained consistent in studies employing different methods, but concern has been expressed about indirect methods of calculating the population at risk. This study aims to overcome these shortcomings.

Method. A further prospective study was undertaken in Nottingham assembling an inception cohort of psychotic patients ($N = 168$) presenting from a defined catchment area. The 1991 census, which includes codings for self-ascribed ethnic origin, was used to calculate the denominator, employing correction factors for potential under-enumeration. Case-ascertainment was based upon all service contacts and subjects had in-depth assessments including the SCAN. Collateral history was obtained from informants.

Results. Subjects born in the Caribbean, or who had one or both parents born in the Caribbean, had a greatly elevated risk (incidence ratios above 7) for all psychotic disorders and for ICD-10 (DCR)-defined F20 Schizophrenia.

Conclusions. The size of the increase and the methodological safeguards employed support the validity of this now highly replicated finding. A personal or family history of migration from the Caribbean is a major risk factor for psychosis; the consistency of this finding justifies a systematic evaluation of potential aetiological factors. Any hypothesis derived from the evidence so far must explain: increased incidence in first *and* second generation migrants; increased risk for *all* psychoses (including affective psychoses); and an effect specifically associated with a migration history from the Caribbean to Northern Europe.

INTRODUCTION

Studies of migrant populations offer a unique opportunity to explore the relationship between environment and disease (Kennaway, 1944) and have illuminated the aetiology of conditions as diverse as multiple sclerosis, gastric carcinoma and cerebrovascular disease. Several studies have examined the relationship between migration and the incidence of psychotic disorders, but weaknesses in design and methodology have compromised their findings.

The difficulties of carrying out large scale studies of the relationship between migration and risk of schizophrenia are well recognized (Harrison, 1990). These include problems in defining and estimating the size of the populations at risk, imprecision in case definition (including those arising out of cross-cultural comparisons) and bias in case ascertainment. Nevertheless, the finding of increased risk of schizophrenia and related psychoses in first and second generation African-Caribbean migrant populations to the United Kingdom (Harrison *et al.* 1988) and the Netherlands (Selton & Sijben, 1994) has proved remarkably robust, having been replicated in studies relying upon different methodologies (reviewed by Harrison,

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1990; Wessely *et al.* 1991; Thomas *et al.* 1993). Since the publication of the 1991 census, and the availability of more accurate estimates of the populations at risk, two more research groups have reported incidence rates for schizophrenia and related psychoses among African-Caribbean migrants: van Os *et al.* (1996) employed extensive corrections for under-enumeration in the 1991 census and King *et al.* (1994) addressed the problem of bias in case ascertainment at secondary care level by finding cases in primary care and other community sources. Both these studies confirmed higher rates in migrants populations, especially for first and second generation African-Caribbeans.

The first Nottingham study (Harrison *et al.* 1988) overcame many, but not all, of the methodological difficulties which affected earlier studies of migrants. The authors employed a prospective design utilizing standardized assessments of mental state and operational diagnostic criteria, but relied upon indirect estimates of the population at risk. Following the publication of the 1991 census population data, which included self-ascribed ethnicity and place of birth, we decided to carry out a further prospective study of the incidence of psychosis in Nottingham based upon more reliable measures of the population at risk.

Nottingham has well circumscribed area boundaries with a relatively stable population of 600000, served by a single Provider of Mental Health Services. The tasks of assembling an inception cohort of psychotic patients making service contact was, therefore, considerably easier than for many large urban areas. We tested the hypothesis that there is no difference in the incidence of schizophrenia and other psychotic disorders, in first and second generation migrants to the United Kingdom from the Caribbean, assuming that previous reported differences are due to bias in case ascertainment and definition, or to errors in estimation of the denominator.

METHOD

The population at risk

Although self-ascribed ethnic group as 'African-Caribbean' is a reasonable indicator of personal migration history or of status as British born of African-Caribbean migrant parents, there are a number of problems concerning the validity of the 1991 census data for ethnic minority groups. The raw data, for example, clearly show under-enumeration of African-Caribbean males in the 20–35 age range. We decided, therefore, to carry our several modifications to the denominator, biasing the data toward the null hypothesis of no difference in rates of psychosis. First, the raw population data designated 'African-Caribbean' and 'other blacks' were adjusted for age and sex, based upon recommendations regarding potential under-enumeration of ethnic minority groups in the *OPCS 1991 User Guide* (1994) '*Undercoverage in Great Britain*'. These two groups were then added together on the assumption that most 'other blacks' had a personal or family history of migration from the Caribbean (this would not have been the case in centres such as London in the United Kingdom, but the intention here was to err on the side of caution in arriving at our estimate of the African-Caribbean population). The number of black males was then increased further to achieve parity with the number of black females (up to age 49). The overall effect was to increase the 1991 census estimate of African-Caribbean males in the age range 20–29 by 30%, and the total African-Caribbean population by 10%. Table 2 illustrates the (age specific) adjusted population data which formed the basis of rate calculations.

Case ascertainment and definition

The methods of case finding and the assessment instruments used have been described in detail elsewhere (Brewin *et al.* 1997). Briefly, over a 24-month period (1992–4) every case of potential psychosis making first contact with the mental health services in Nottingham was identified using an over-inclusive psychosis screen. Case ascertainment included all service contacts, in addition to first hospital admissions. All potential points of contact with the secondary psychiatric services were regularly surveyed by telephone and by personal contact; potential cases of psychosis were screened and entered into assessments where entry criteria were satisfied. A leakage study was carried out by checking all case notes for the study period in the local Provider unit for psychiatric services.

Personal and family migration history were established upon the basis of direct interview. Where study subjects refused interview, the

history was established on the basis of information in case notes, and whenever possible from informants. For the purposes of the study hypothesis, cases were defined as those having one or both parents born in the Caribbean; those of mixed parentage were included in the first stage analysis because hypotheses which might possibly explain differences in incidence rates (for example environment biological factors or socially mediated effects of racial discrimination) would not exclude them in the first instance. Rates for all psychoses and for schizophrenia were therefore calculated both including and excluding those of mixed parentage.

Informed consent was sought to carry out a number of assessments including the Schedules for Clinical Assessment in Neuropsychiatry (SCAN) (WHO, 1992); a modified Personal and Psychiatric History Schedule (PPHS); Scale for the Assessment of Negative Symptoms (SANS); and a schedule for coding substance and alcohol use and misuse (in addition to the relevant sections of the SCAN). Information was included from an informant wherever possible. Where patients refused consent for the SCAN examination, the Item Group Checklist (IGC) was coded according to SCAN rules, on the basis of case-notes and any other information available for the patient.

For each case a Diagnostic meeting was convened to consider information from all sources, including the item ratings in SCAN and collateral information from notes and informants; the meeting always included the researcher who carried out the patient assessments and either G.H. or I.M. Main, Alternative and Subsidiary consensus diagnoses were assigned by checking symptoms against criteria in the International Classification of Diseases (10th edition) Diagnostic Criteria for Research (DCR) (WHO 1993).

Training in assessments and reliability

Ratings of psychopathology were carried out by a team of four senior registrars who had completed a training course in the SCAN. Good pre-study reliability between the raters was achieved (Brewin *et al*. 1997).

Analyses

Incidence (first contact) rates were calculated for two diagnostic groups according to DCR criteria: all psychoses, based upon categories F20–29 (excluding F21 schizotypal disorder) and F30–39 where affective disorders presented with psychotic symptoms; and F20 Schizophrenia. Rates were calculated for first generation African-Caribbean migrants combined with second generation subjects and then also for the remainder of the population of Nottingham. Incidence rates for all psychoses and for F20 schizophrenia were adjusted for differences in the age structure of these two populations by direct standardization to the population of England and Wales according to the 1991 census, doubling the population estimates to account for the two year study period. These standardized rates (SIRs) are presented together with 95% confidence limits to allow comparison with other studies. Our null hypothesis of no difference in the rates of all psychoses and F20 schizophrenia in the two populations was tested by calculating rate ratios and 95% confidence limits, weighting each age stratum according to the size of the estimated population at risk. This method of adjusting for differences in age structure according to the methods of Mantel and Haentzel was used in preference to the ratio of directly standardized rates so as to avoid the bias which may arise from the latter method when rates are based upon small numbers of events (Breslow & Day, 1987). In fact, the two methods gave very similar results. The risk associated with African-Caribbean migration status was also expressed as the population attributable fraction making the assumption that some (as yet unknown) characteristics associated with this population might cause psychosis.

RESULTS

One hundred and sixty-eight cases of psychosis met our inclusion criteria and were included in the study. Of these, 124 were of European origin; 32 were first or second generation African-Caribbean migrants; three were African in origin and nine were south Asian. Because of the small numbers of patients in other ethnic minority groups, rates are reported only for those with a personal or family history of migration from the Caribbean compared with the remaining non-Caribbean cohort. Persons with a personal or family history of migration from the Caribbean are referred to as African-Caribbean subjects for

Table 1. *Sociodemographic and co-morbidity factors in African-Caribbean and the remaining population*

	African-Caribbean ($N = 32$)	Remaining population ($N = 137$)
Direct interviews	23 (72)*	97 (71)
Mean age at onset	31 (S.D. 10·85)	31 (S.D. 13·25)
Male : female ratio	1·7 : 1	1·3 : 1
Social class†		
Profess/skilled	10 (31)	59 (43)
Partial/unskilled	9 (28)	36 (26)
Unemployed	8 (25)	13 (10)
Other (inc. student)	3 (9)	17 (13)
Missing values	2 (7)	11 (8)
Unemployed in previous year‡	15 (47)	68 (50)
Substance use	10 (31)	56 (41)

* Figures in parentheses indicate percentages unless otherwise stated.

† Based upon respondents main occupation regardless of current employment status.

‡ Respondent has experienced one or more periods of unemployment lasting one month or more over past year.

the purposes of this study as all were assigned to this ethnic group according to the 1991 census classification by the person carrying out the assessments. There were no individuals judged to be of 'white' or 'Asian' origin migrating from this area to Nottingham. Of these 32 cases, 26 (81%) were born in the United Kingdom and six (19%) in the Caribbean (nearly all from Jamaica). Twenty-five subjects (78·1%) had both parents born in the Caribbean. Five subjects had mixed parental backgrounds and for two subjects information on place of birth of both parents was uncertain. Rates for all psychoses were calculated first including, and then excluding, these seven subjects; similarly rates for F20 ICD-10 schizophrenia were calculated including, and then excluding the one subject of mixed parentage in this diagnostic group.

Face-to-face interviews were achieved for 71% of the entire sample. For the remainder, clinical data were coded according to the SCAN system rules for the Item Group Checklist (IGC) based upon case-notes and informants. There were no differences between African-Caribbean migrants (first and second generation) and the remainder of the cohort for the proportion of patients who could not be interviewed directly (28·1% *v.* 29·2%) Table 1 illustrates the two groups compared for age at onset (defined as age at first contact), other sociodemographic indices and clinical features including co-morbidity for substance abuse. The sex ratio (m : f) for all psychoses was approximately 1·7 : 1 in African-Caribbean subjects compared with 1·3 : 1 in the remaining population; there were few differences in mode of onset or in sociodemographic indices. There were no differences in reported substance use but the African-Caribbean group reported significantly less substance misuse according to ICD-10 criteria. There were very similar proportions with affective and non-affective psychoses in both African-Caribbean subjects and the remainder, and within the affective psychoses (F30–39) category the proportion of depressive disorders was identical in both groups.

Table 2. *Rates of psychoses in African-Caribbean group and remaining Nottingham population*

Age	African-Caribbean population	Psychoses African-Caribbeans in 2-year period	Rate of psychoses per 100000 per year	Remaining Nottingham population	Psychoses Remaining population in 2-year period	Rate of psychoses per 100000 per year
16–19	820	2	122	31512	10	16
20–29	3385	18	266	102184	68	33
30–39	1907	6	157	83471	33	20
40–49	960	1	52	78926	15	10
50–59	1505	3	100	61815	4	3
60–64	600	2	167	30497	6	10
Total	9177	32	174 (123–246)	388405	136	18 (15–21)

SIR psychoses in African-Caribbean group per 100000 population = 149·6 (94·9–204·2).
SIR psychoses in Remaining population per 100000 population = 16·9 (14·1–19·8).
Age standardized rate ratio 8·8 (CIs 6·0–12·9).
Mantel–Haenszel rate ratio 8·73 (CIs 5·9–12·9).

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Table 3. *Rates of F20 schizophrenia in African-Caribbean group and Remaining Nottingham population*

Age	African-Caribbean population	F20 African-Caribbeans in 2-year period	Rate of F20 per 100000 per year	Remaining Nottingham population	F20 Remaining population in 2-year period	Rate of F20 per 100000 per year
16–19	820	0	0	31512	4	6
20–29	3385	8	118	102184	27	13
30–39	1907	2	52	83471	7	4
40–49	960	0	0	78926	5	3
50–59	1505	0	0	61815	3	2
60–64	600	1	83	30497	0	0
Total	9177	11	60 (33–108)	388405	46	6 (4–8)

SIR F20 African-Caribbean group per 100000 population = 46·7 (18·1–75·3).
SIR F20 Remaining population group per 100000 population = 5·7 (4·0–7·3).
Age standardized rate-ratio 8·1 (4·2–15·8).
Mantel–Haenszel rate ratio 8·5 (4·4–16·5).

The Standardized Incidence Rate (SIR) for all psychotic disorders was elevated in the African-Caribbean group (including those of mixed parental backgrounds) at 149·6 (94·9–204·2) per 100000 per year (Table 2); the Mantel–Haenszel Rate Ratio (RR_{mh}) was 8·73 (CI 6·13–12·28). The RR_{mh} for F20 schizophrenia was elevated (Table 3) at 8·5 (CI 4·4–16·5). Excluding those subjects with mixed parental backgrounds, the RR_{mh} for all psychoses was 7·37 (4·87–11·15) and for F20 schizophrenia was 7·7 (3·8–15·3). Because the census does not provide data for self-ascribed ethnicity by place of birth, it was not possible to calculate separate rates for the first and second generation migrant generations. However, it should be noted that 81% African-Caribbean subjects were born in the UK suggesting an increased rate of psychosis in both populations.

The RR_{mh} for males (F20 schizophrenia) was 10·75 (CI 4·94–22·36) and for females 4·46 (1·05–18·8) including all subjects. Sex-specific RRs indicated a stronger effect in males but the overlap of confidence intervals does not allow us to conclude firmly that there was an interaction. However, the lower limits for females suggests there may be a sex specific effect.

The population attributable fraction (17%) represents the percentage of first onset psychosis in Nottingham that might be attributable to the African-Caribbean migrant population (less than 3% of the population at risk), assuming that characteristics associated with this group are causal.

DISCUSSION

It is not possible to eliminate all possible sources of error in a study of this nature and there are several methodological considerations. First, small numbers of cases are clearly a limitation, although confidence intervals are well above one for all reported rate ratios. Further, the present study did not carry out case ascertainment at primary care level and selection biases may have operated in favour of psychotic African-Caribbean subjects if they have an increased risk of contact with the psychiatric services compared with the remaining population. It would be virtually impossible, however, for psychotic disorders of comparable severity to remain undetected in the general population on the scale required to eliminate the effect reported in this and in other studies. Population studies (Von Korff *et al.* 1985; Meltzer *et al.* 1995) also fail to support the notion of huge numbers of psychotic individuals remaining undetected by the services.

There may be errors in case definition given problems of carrying out standardized assessments in a cross-cultural setting. It is important however to avoid exaggerating this risk for one migrant group within the population. Virtually all the African-Caribbean patients were born

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and brought up in Nottingham, UK. It is notable that the proportion of cases in different diagnostic groups was similar, suggesting that biases in case definition would need to operate both at the level of the psychosis screen and at the level of the diagnostic process based upon standardized assessments and multiple sourcing of collateral data (including family informants).

The under-enumeration of young men in the 1991 census should sound a note of caution. However, the effect size reported here would allow further substantial increases in the size of the African-Caribbean population in Nottingham, in addition to the corrections already applied, and still identify an increased risk for psychosis in this migrant group.

We conclude, therefore, that taking into account these methodological considerations, the age adjusted rates for psychotic disorder, and for operationally defined schizophrenia, are up to eight times higher in the African-Caribbean first and second generation population, compared with the remaining population of Nottingham. These data are consistent with those from a series of studies over the past decade. The finding has been replicated by several groups employing different methods of sampling and case definition and applying various correction factors to the denominator. In the present study we have retained subjects of mixed parental background because there is no good reason for excluding them for all psychoses before more specific hypothesis are generated. Only one 'African-Caribbean' subject with ICD-10 Schizophrenia fell into this group and we also report the Rate Ratio with this individual eliminated.

Throughout the series of studies in this area, the direction of effect for migration status has proved remarkably consistent. We believe that the epidemic of schizophrenia and other psychotic disorders in this population may now be regarded as established beyond reasonable doubt and with sufficient validity to justify a concerted programme of research into aetiology. Sample sizes in studies carried out so far do not allow adjustment for socio-economic status and other potential risk factors for which the notion of 'migration status' or 'ethnicity' acts as a proxy variable. The unravelling of the risk factors associated with migration status will require much larger samples and case control designs. However, the fact that 17% of all first onset psychosis in Nottingham might be attributable to factors associated with migration suggest that such large scale investigations will be worthwhile.

It should be noted that the increase was not confined to schizophrenia but found across the broad range of psychotic disorders, including affective psychoses. When considering risk of psychosis in this migrant group, commentators have focused upon the finding for schizophrenia, probably because this challenged earlier notions that the increased risk of psychosis could be explained (and thereby eliminated) on the basis of mis-diagnoses of 'culturally' determined behavioural disturbances. However, our earlier report (Harrison *et al*. 1988) was entitled 'Severe mental disorder' in Afro-Caribbeans to reflect a similar finding of increased risk across all psychotic groups; the present data are therefore consistent with previous Nottingham findings, and with other data showing increased risk for mania and psychotic disorders in this migrant group (Leff *et al*. 1976; Bebbington *et al*. 1981 van Os *et al*. 1996).

There are considerable problems in devising a parsimonious explanation for these findings capable of addressing all of the available data. Any hypothesis must embrace: evidence for increased risk in first and second generation migrants; probable higher risk in second generation migrants compared with first; and an effect distributed across the range of psychotic illnesses, including the affective psychoses. One explanation might be that vulnerable individuals selectively migrated and either developed the illness or increased the risk of psychosis in the second generation by a process of assortive partnerships. However, the evidence so far (Sugarman & Crauford, 1994; Hutchinson *et al*. 1996), which requires replication, reports increased risk in siblings rather than in parents, suggesting an environmental rather than genetic factor. Further, the selection hypothesis is seriously weakened by data from the Netherlands (Selten & Sibjen, 1994) showing a five-fold excess in migrants from Surinam. As nearly half of the population migrated from Surinam to the Netherlands it is difficult to sustain an explanation based upon selective migration. The

notion of 'genetic ethnic vulnerability', or vulnerability based upon questionable notions of 'race', can also be excluded by evidence of much lower rates for schizophrenia reported from some parts of the Caribbean (Hickling & Rodgers-Johnson, 1995) and by data from the Netherlands where the Surinamese immigrants showing increased risk of schizophrenia were of both African and Asian origin (Selten, 1995).

The most likely explanation for this epidemic is a coincidence of risk factors arising from the experience of migration from the Caribbean to Europe. One candidate would be exposure to pre-natal and childhood infections linked with an idiosyncratic auto-immune response. In a multi-factorial model, a higher prevalence of such factors would be expected in comparison with non-psychotic African-Caribbean controls and non-migrant schizophrenic populations. Against this, it is difficult to explain the increased risk in both first and second generation migrants on the basis of biological factors. In contrast, both acute (life events) and chronic social adversity may operate across both generations and some components (e.g. limited opportunity) may have been intensified in second generation British born.

Several studies have reported poor outcome for schizophrenia in African-Caribbeans. Recently, McKenzie *et al.* (1995) reported better outcome for psychotic disorders (including schizophrenia), with ethnicity exerting a significantly favourable effect after controlling for age at onset and social class. Although these data suggest better outcome in terms of positive symptoms, negative symptoms and social disability were comparable at the point of follow up for both African-Caribbeans, and 'whites'. If such data showing better outcome in terms of positive symptoms are replicated, there may be some justification for concluding that these patients exhibit a subtype of psychosis precipitated by life events related to social adversity. Conversely, if negative symptoms of the illness are shown to persist in African-Caribbean's, it is possible that biological factors may have an equally prominent role. These questions remain unanswered.

In conclusion, the epidemiological finding of increased risk of psychosis in African-Caribbean migrants has proved remarkably consistent. It has been replicated in sufficiently diverse geographical and treatment settings, and in studies employing a range of methodologies, to allow the conclusion that its validity is beyond reasonable doubt. Although migration status is a highly confounded proxy variable, this finding underlines the potential role of environmental risk factors in the aetiology of psychosis.

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