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Schizophrenia and Afro-Caribbeans A Case-Control Study

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A case-control study was performed using 90% of all first-contact patients with a clinical diagnosis of schizophrenia residing in the London borough of Camberwell between 1965 and 1984. Cases and controls were obtained from the Camberwell psychiatric case register. Controls were those presenting with first episodes of non-psychotic disorders, matched for age, sex and period. The risk of schizophrenia was greater in those of Afro-Caribbean ethnicity, irrespective of age, gender or place of birth. This risk increased over the study period. The results cannot be explained by changes in the age, gender or ethnic structure of the local population. Effects of misdiagnosis or change in diagnostic practice were reduced by using uniform operational criteria. Possible explanations include maternal exposure to unfamiliar infective agents, a differential fall in the age at onset of illness, or worsening social adversity.

Several studies have recently suggested that rates of schizophrenia in the Afro-Caribbean population in the UK are higher than those found in non Afro-Caribbean samples (Rwegellera, 1977; Bebbington *et al*, 1981; Cochrane & Bal, 1989; Harvey *et al*, 1990; Castle *et al*, previous paper, this issue). These findings have recently been extended by reports that not only is this increase found in the children of Afro-Caribbean migrants, but the second generation have an even higher rate of schizophrenia than their parents (McGovern & Cope, 1987; Harrison *et al*, 1988).

However, no single study is satisfactory. Several studies depend upon the routinely collected data of the Mental Health Enquiry (Cochrane & Bal, 1989; Glover, 1989a). Such data have several limitations. Definitions of schizophrenia are unstandardised, place of birth is not always recorded, and ethnicity never, thus excluding all those of Afro-Caribbean ethnicity born in Britain. Finally, the data concern only hospital admissions.

McGovern & Cope (1987) recorded ethnicity as well as place of birth, but were still restricted to inpatients, and did not use operationally defined criteria for schizophrenia. Only Harrison et al(1988) and Harvey et al (1990) used direct interviews and standardised criteria, but only the former study was population-based, and its power was limited by the small sample size (Harrison, 1989). Of the 28 cases of schizophrenia in Afro-Caribbeans studied by Harrison et al, 18 were born in the UK; of the 10 born in the Caribbean, only two had come to the UK after the age of 18. Nevertheless, despite the sample size, the 95% confidence limit for the rate ratios for schizophrenia in Afro-Caribbeans compared with non-Afro-Caribbeans has a lower limit of six, a substantial excess (Harrison, 1990).

Problems also exist in calculating appropriate denominators. This is because no accurate figures exist for the size of the Afro-Caribbean population in the UK. Reliable statistics are available only for place of birth, not ethnicity, and even then it has been suggested that many young blacks of both British and Caribbean birth did not register for either the 1971 or 1981 census (Cruikshank & Beevers, 1989). One solution has been to use data concerning the head of household, and to assume that all those recorded as having been born in the UK but living in the household of someone recorded as being born in the Caribbean are second-generation Afro-Caribbeans. However, this may considerably underestimate the size of the second-generation population, since it assumes that all second-generation children remain in the same household, and that all those resident in the household are of similar ethnic origin. The absence of reliable data about the size and age structure of the Afro-Caribbean population means that it remains possible that the apparent increase in schizophrenia is at least in part due to increasing numbers of young Afro-Caribbeans in the population and therefore at the age of greatest risk, rather than a true increase in agestandardised rates.

Thus, there is now a variety of evidence suggesting that higher rates of schizophrenia are found in those of Afro-Caribbean ethnicity, but no convincing data on changes in risk over time. The current paper assumes that it will never be possible to obtain such data by comparing rate ratios, and that a different strategy is required. One possibility is to use the casecontrol study (Schlesselman, 1982) to compare samples of those with a particular illness ('cases') to those without ('controls'). We present the results of the first case-control study of changes in the risk of schizophrenia over time according to ethnic group.

Method

In this study, individuals with schizophrenia ('cases') are compared with those without schizophrenia but with other mental disorders ('controls'), with respect to a particular attribute (ethnicity).

The selection of cases has been described in detail in the previous paper (this issue, pp. 790–794). Briefly, it consists of all cases of first-contact schizophrenia and related conditions in Camberwell, London, between 1964 and 1984, as listed by the Camberwell psychiatric case register. Case notes were obtained on 90% of all those with a diagnosis of schizophrenia or related conditions, and rated using a uniform check-list allied to the OPCRIT computer program (McGuffin *et al*, 1991). Three diagnostic criteria are compared: the clinician's diagnosis of schizophrenia, which is based on the International Classification of Diseases; the Research Diagnostic Criteria (RDC); and the DSM-III-R criteria. As this is a study of risk, and not rates, no corrections are made for missing data.

Controls were selected from the same register. A control was the next person on the register to a case, matched for sex and age (to within five years). Controls were therefore also matched for time period.

Place of birth and ethnicity were obtained from case notes. The general standard of case records was high, and nearly all contained information on ethnicity in either the medical or nursing notes. In cases of doubt, further information was obtained either from the Maudsley Hospital computerised record system, which now records ethnicity, or general practitioner's records. Those in whom the parents were of different ethnic origins were classified according to the description contained in the notes - only two were found, both classified as Afro-Caribbean. No adequate data could be obtained on ethnicity for 19 controls, usually since the subjects had made only brief contact with one particular local out-patient department which had subsequently destroyed its records. These were replaced by new controls. Two checks were made on the reliability of the recording of ethnicity. As part of other studies, direct contemporary interviews that included a recording of ethnicity had been carried with 34 patients, and other data sources that directly recorded ethnicity were available for a further 21 patients. No misclassifications were found. Four controls fulfilled criteria for schizophrenia and were thus replaced by new controls.

Place of birth was classified as follows: UK and Eire; West Indies; Asia; Africa; other. Ethnicity was classified as follows: Caucasian; Afro-Caribbean; Asian; other. In this paper we present analyses of Afro-Caribbeans alone – either born in the West Indies or born in the UK to parents born in the West Indies. Any subjects born in Africa, and Asians born in the Caribbean, are not included as cases.

The data were analysed by four time periods (1965-69; 1970-74; 1975-79; 1980-84). The traditional method of calculation is illustrated in the upper half of Table 1. As the study is matched, only those pairs discordant for ethnicity contribute to the odds ratios (rows 2 and 3). Pairs concordant for ethnicity (rows 1 and 4), or pairs in which either case or control is neither Caucasian nor Afro-Caribbean (row 5), do not contribute further (Schlesselman, 1982). Odds ratios are therefore the ratio row 3:row 2. Confidence limits can be obtained in various ways, for example using the formula provided by Gardner & Altman (1989) together with standard tables for the binomial distribution (Neave, 1979).

However, matched case-control studies can also be analysed by the more powerful tool of conditional logistic regression available via the EGRET statistical package. The logistic regression method gives identical odds ratios for the entire study period (i.e. 1965-84), but the results for each stratum (i.e. 1965-69; 1970-74, etc.) are slightly different to those obtained without the aid of the statistical package. This is because logistic regression gives results that are mutually consistent across the study periods, the model in effect 'smoothing' both the odds ratios and confidence limits. We cite the odds ratios and confidence limits obtained by conditional logistic regression, but have confirmed that the more traditional approaches give similar results for all the analyses presented here, and in particular there is no tendency for either method to report consistently more liberal or conservative results. Each table also includes all the discordant pairs to permit calculation of the conventional odds ratios and confidence limits.

Tests for trends were also obtained using conditional logistic regression. Thus a possible trend in the risk of schizophrenia in Afro-Caribbeans over time was assessed by entering an interaction term between ethnicity and period, the resulting likelihood ratio statistic being treated as a χ^2 statistic on one degree of freedom. Again, a test for trend can be calculated directly, using a non-regression method (Kirkwood, 1989). Because this method uses a continuity correction, the results were generally more conservative (ignoring the continuity correction, as some statisticians advise, would give results very similar to those obtained by regression). Even with the correction, the results differed only substantially on one analysis (Table 3), for which both results have been quoted.

Unless otherwise stated, comparisons are made between those of Afro-Caribbean and Caucasian ethnicity only. However, results are also presented comparing Afro-Caribbeans with all other groups, thus eliminating row 5 of Table 1 and increasing the power (lower half of Table 1, and Table 3). For reasons of space, only discordant pairs (rows 2 and 3) are presented in Tables 2-5.

Results

A total of 130 patients of Afro-Caribbean ethnicity received a diagnosis of ICD-9 schizophrenia over the two decades. No evidence was found of an ethnicity-gender interaction (see below), so results are presented for both sexes combined.

The risk of the illness in Afro-Caribbeans is increasing across the study period (Table 1), particularly between 1964 and 1980. If only those cases fulfilling RDC or DSM-III-R (Table 2) criteria for schizophrenia or schizophreniform psychosis are used, similar trends are seen. That for RDC schizophrenia just fails to reach conventional statistical significance,

SCHIZOPHRENIA AND AFRO-CARIBBEANS

 Table 1

 Matching pairs and odds ratios for ICD schizophrenia, across study period

l able 2								
Matching	pairs	and	odds	ratios	for	RDC	and	DSM-III-R
	schiz	zoph	renia,	across	s sti	d vb.	eriod	

Pariod of procentation

	Period of presentation					
Matched pair	1965-69	1970-74	1975-79	1980-84		
Afro-Caribbean and C	aucasian1					
Caucasian case,						
Caucasian control	69	63	72	61		
Caucasian case, Afro-						
Caribbean control	6	6	2	4		
Afro-Caribbean case,						
Caucasian control	16	24	33	35		
Afro-Caribbean case,						
Afro-Caribbean						
control	2	2	1	11		
Other pairs	17	15	24	16		
Odds ratios (condition	al					
logistic regression)	3.0	4.1	10.5	11.6		
95% CI	1.0-6.8	0.8-10.5	5.0-153	3.3-72.4		
Afro-Caribbean and al	l other gro	ups				
'Other' case	-					
'other' control	83	74	95	74		
'Other' case, Afro-						
Caribbean control	8	7	3	4		
Afro-Caribbean case						
'other' control	16	27	33	38		
Afro-Caribbean case,						
Afro-Caribbean						
control	2	2	1	11		
Odds ratios (condition	al					
logistic regression)	2.1	3.8	15.7	9.1		
95% CI	0.9-4.7	1.2-11.8	3.0-82.9	2.4-35.0		

1. Test for trend: 5.48, 1 d.f., P=0.0019.

2. Test for trend: 7.12, 1 d.f., P = 0.008.

although this is achieved if the comparison is between Afro-Caribbeans and all other ethnic groups ($\chi^2 = 6.74$, P = 0.009). The particularly wide confidence limits for the period 1975-79 reflect the small numbers of pairs in which the case was Caucasian and the control Afro-Caribbean. Using ten-year time periods was considered, but, with the exception of Table 5 (see below), rejected because of the evidence of period-ethnicity interaction.

When the pairs were analysed by year of birth, rather than year of presentation, there was an increase in risk for later years of birth, most marked for RDC schizophrenia (Table 3), and least evident for ICD schizophrenia (χ^2 test for trend – 3.08, 1 d.f., P=0.08). Similar results were obtained when comparing only Afro-Caribbeans and Caucasians (data not shown). This was confirmed by a trend for an interaction between ethnicity and year of birth in the logistic model (likelihood ratio statistic=3.085, P=0.079).

Table 4 shows the risks for comparing those of Afro-Caribbean ethnicity born in the West Indies with all other groups. Second-generation Afro-Caribbeans now appear along with all other ethnic groups. This has resulted in a substantial fall in the odds ratio for the last time period. Again, the presence of only two cases born elsewhere for

	renou or presentation					
Discordant pairs	1965-69	1970-74	1975-79	1980-84		
RDC schizophrenia						
Caucasian case, Afro-						
Caribbean control	3	4	2	1		
Afro-Caribbean case,						
Caucasian control	6	17	24	22		
						
Odds ratios' (conditio	nal					
logistic regression)	2.0	4.2	17.4	33.0		
95% CI	0.8-5.3	1.2-18.8	2.4-52.2	3.6-246.5		
DSM-III-R schizonbre	nia and sc	hizonhrenif	orm disord	ar		
Caucasian case Afro-						
Caribbean control	2	3	2	1		
Afra Caribbaan control	2	5	2	•		
Arro-Caribbean case,	•	14	10	17		
Caucasian control	0	14	19	17		
Odds ratios (condition	al					
logistic regression)	1.8	4.7	11.0	18.0		
95% CI	0.6-9.0	0.8-31.3	1.4-72.8	1.5-193.0		

1. Test for trend: 3.32, 1 d.f., P=0.06.

2. Test for trend (regression) = 3.56, 1 d.f., P = 0.059.

3. Test for trend (continuity correction) = 1.86, 1 d.f., P = 0.17.

1975-79 makes the odds ratio for that period unstable. However, comparing the other three time periods suggests that there has not been a marked increase in risk for this group, and this is confirmed by the absence of a significant trend.

It was impossible to carry out the same analysis for Afro-Caribbeans born in the UK because of lack of discordant pairs, even if all time periods were combined. The odds ratio for first-generation Afro-Caribbeans over all other groups was 3.8 (rising to 4.3 if second-generation cases were excluded from contributing to other groups). However, although there were 30 second-generation Afro-Caribbean cases, and three second-generation controls, all the secondgeneration Afro-Caribbean controls were paired with second-generation cases, so the odds ratio was infinite. However, as the odds ratio could not be less than 10, the data

Table 3 Odds ratios for RDC schizophrenia by period of birth (Afro-Caribbean and all other groups)

Discordant pairs	Before 1930 1929 49		- 1950- 67	
'Other' case, Afro- Caribbean control Afro-Caribbean case.	5	4	3	
'other' control	8	29	36	
Odds ratios 95% Cl	1.6 0.3-8.1	7.3 1.8-51.5	12.0 3.6-38.0	

Test for trend: 5.58, 1 d.f., P=0.018.

Table 4 Odds ratios for ICD schizophrenia by place of birth (West Indies or elsewhere)

	Period of presentation					
Discordant pairs	1965-69	1970-74	1975-79	1980-84		
Cases born elsewhe controls born in West Indias	re, 8	7	2	7		
Cases born in West controls born	Indies		2	,		
elsewhere	19	25	25	23		
Odds ratios 95% Cl	2.4 0.9-4.7	3.6 1.1–11.7	12.0 2.3-63.7	3.4 1.0–11.3		

Test for trend: 1.9, P=0.274.

support other findings of a substantial elevation in the risk of schizophrenia in second-generation Afro-Caribbeans.

An alternative was to follow the example of McGovern & Cope (1987), and repeat the analysis with cases and controls including not only those of Afro-Caribbean ethnicity born in the UK, but also those who migrated as children, defined here as before 18 years of age. Two pairs were eliminated because of lack of information as to when the control entered the UK. Despite widening the definitions there were still no discordant pairs in the third period. Adjacent quinquennia were therefore combined into two ten-year bands. The results show that the odds ratios increased from 2.6 for 1965-74, to 8.8 for 1975-84, although not achieving significance ($\chi^2 = 3.21$; P = 0.075).

To overcome this problem of lack of power, the sample was simply divided into those born before and after 1955, a year chosen to approximate to the period when secondgeneration children began to be born. Comparing all those of Afro-Caribbean ethnicity against all other ethnic groups (Table 5) the risk of ICD-9 schizophrenia rose after 1955, but only slightly (likelihood ratio statistic = 0.82, P = 0.37). In contrast, comparing those born in the West Indies with all other places of birth the risk fell after 1955 (likelihood ratio statistic = 3.48, P = 0.06).

The influence of age of entry to the UK was assessed as follows. Looking only at those born in the West Indies, cases and controls were divided into those who came before age 18, and those who came aged 18 and over. Over the

Table 5 Influence of year of entry to UK on the odds ratios (95% CI) for ICD schizophrenia

	Before 1955	1955 and after
Afro-Caribbean ethnicity v. all other groups Place of birth: West Indies	4.5 (2.7-8.0)	7.6 (2.6-22.3)
v. all other places of birth As above, but excluding	4.8 (2.8-8.1)	1.7 (0.6-4.8)
all Afro-Caribbeans born in the UK	4.4 (2.5-8.4)	2.4 (0.8-5.6)

whole time period, the increase in the risk of schizophrenia for those born in the West Indies compared with all other places of birth was 4.1 (95% CI 2.4–7.3). For those entering aged 18 and above it was 4.9, and for those entering at younger ages it was 2.7. For these analyses Afro-Caribbeans born in the UK are included with all other places of birth. Excluding them did not alter the odds ratios for those entering as adults, but the risk for those arriving in the UK aged under 18 years increased from 2.7 to 3.5.

It has been suggested that the increased risk of schizophrenia in Afro-Caribbeans is restricted to males (Glover, 1989a), although this has not been a universal finding. The overall odds ratios for the excess of schizophrenia in Afro-Caribbean compared with Caucasian males was 9.0 (95% CI 4.1-19.6), and 4.1 (2.1-7.9) for females, a nonsignificant difference. No interaction was seen between time period and gender ($\chi^2 = 2.35$, P = 0.12).

Discussion

The current study avoids some of the methodological problems that have affected other studies. Uniform criteria minimise the effect of changing diagnostic habits over time. Using incident cases eliminates duration bias. Matching for age, sex and period controls for changes in the age or gender structure of the population.

Our findings are not due to an excess of the socalled 'brief reactive psychoses'. Although it has been suggested that individuals with these conditions are frequently misdiagnosed as schizophrenic in Afro-Caribbean samples, this has not been confirmed (Harvey *et al*, 1990). In the current analysis, similar trends were noted when using either RDC (which specifies a minimum period of illness of two weeks) or DSM-III-R criteria for schizophrenia (in which the minimum period of illness is six months).

However, other methodological explanations cannot be excluded. Using operational criteria helps to overcome idiosyncratic diagnostic habits and cultural biases, but may bring us closer to committing Kleinman's 'category error' (Kleinman, 1977). It is also true that the cross-cultural validity of the instruments used has not been specifically established. Nevertheless, we do not feel that such possibilities could explain all our findings, and in particular the time trends. Furthermore, there is no evidence that UK psychiatrists currently overdiagnose schizophrenia in Afro-Caribbeans (Lewis *et al*, 1990).

A second methodological explanation is that the age at onset of schizophrenia is differentially decreasing in the Afro-Caribbean population (Littlewood & Lipsedge, 1988). This would increase the number of cases towards the end of the study period and give a false impression of increasing risk. This explanation cannot be refused until all the cohort has passed through the period at risk and lifetime morbidity calculated.

A third possibility is that these results are affected by exposure bias. If this was a valid study, then the 'exposure' under investigation (ethnicity) would not influence the selection of cases and controls. This assumption may have been violated. Pathways to psychiatric care differ according to ethnic origin (Littlewood & Lipsedge, 1982). We believe that the current study uses a representative sample of schizophrenia in Camberwell, since nearly all schizophrenics make contact with the mental health services (Eaton, 1985). However, the controls may not be a representative sample of those with other mental illnesses. It is probable that different biases operate in the way in which non-psychotic individuals of different ethnic groups make contact with services. Some of these biases have been minimised, for example, by using all contacts, and not just admission rates. However, others remain. Thus the finding that Afro-Caribbeans have an increased risk of schizophrenia compared with other mental illnesses, although in keeping with other studies, is subject to bias.

This drawback was reduced by using four sampling periods. All of these are subject to the same bias, but there is little reason to suspect that these biases have changed during the period in question. More robust than the finding of a single elevated risk is that the risk of schizophrenia in Afro-Caribbeans is increasing. The use of controls matched by age, sex and period means that this is not due to changes in the age structure or ethnic composition of the Camberwell population. For it to be explained solely by methodological reasons, one must postulate not only a differential effect in the use of mental health services by Afro-Caribbeans, but one that is declining. The proportion of Afro-Caribbean controls rose from 7% to 12% between 1964 and 1984. Between 1961 and 1981 census data show that the proportion of the local population born in the West Indies rose from 2.5% to 6.6%, and we have estimated that, in 1981, 11.5% of the local population was of Afro-Caribbean ethnicity (see previous paper). This figure is very similar to the proportion of Afro-Caribbean controls in the last time period. While there is some evidence that the rate of increase in the number of controls of Afro-Caribbean ethnicity has not matched that of the Afro-Caribbean proportion of the local population, this is not substantial.

We thus report two findings. First, that Afro-Caribbeans are at elevated risk of schizophrenia, and, second, that this risk has increased between 1965 and 1980. The latter observation is at variance with reports from various countries of a decline in the incidence of schizophrenia (Der *et al*, 1990; Beiser & Iacono, 1990). In the previous paper we have shown that no such decline has occurred in Camberwell, and we now report that this is at least in part due to the high proportion of Afro-Caribbean residents.

One possible explanation for the different trends seen in the incidence of schizophrenia may be found in different experiences of social adversity. In contrast to those areas in which schizophrenia has been recorded as declining, such as New Zealand, north-east Scotland, Eire and Denmark, Camberwell is an area in which indices of social deprivation have worsened during the period in question.

It is beyond dispute that the ethnic minorities of the UK, and particularly those of Afro-Caribbean origin, are exposed to a wide range of social adversities. Being black is associated with unemployment, inadequate housing and low social class (Townsend et al, 1988), as well as disruptions and impairment of family functioning (Brown, 1984). The experience of racism has also been linked to increased rates of mental illness (Littlewood & Lipsedge, 1982). It is probable that any of these factors could directly result in a worse prognosis, and hence higher prevalence, of schizophrenia in the Afro-Caribbean population. It is also possible that such factors could affect the incidence of the disease, perhaps by precipitating illness in predisposed individuals. Jarman (personal communication), using district admission statistics provided by the Mental Health Enquiry, has confirmed that social adversity confounds the relationship between ethnicity and admission to mental hospital. Although being born in the West Indies is significantly associated with psychiatric hospital admission, once adjustment has been made for social deprivation, this association disappears.

Social adversity may also act as a proxy for other variables, of which plausible candidates include poor antenatal and perinatal care and infection. Recent work has suggested that obstetric complications are significantly associated with an increased risk of schizophrenia in offspring (Owen *et al*, 1988), while maternal viral infection during the second trimester of pregnancy has been associated with schizophrenia in offspring in two studies of the 1957 influenza epidemic, in England (O'Callaghan *et al*, 1991) and Finland (Mednick *et al*, 1989).

All these factors may be more marked in the Afro-Caribbean population. Maternal death rates are ten times higher in Jamaica than in England (Walker *et al*, 1986). Those brought up in the Caribbean are more likely to be seronegative to certain viruses such as rubella, since even in Jamaica the population is too small to maintain endemic rubella, and there was no immunisation programme for schoolgirls until 1978. As many young women migrated to the UK during the 1950s, first-generation Afro-Caribbean mothers were highly susceptible to rubella (Nicoll & Logan, 1989), which explains high rates of congenital rubella in their children (Parsons, 1963). A similar model has been proposed to explain increased rates of severe mental retardation in the offspring of Afro-Caribbean women recently arrived in this country (Wing, 1979). It is thus possible that another consequence of maternal exposure to viral illness, whether in the West Indies or the UK, is an increased risk of adult schizophrenia (Glover, 1989b; Harrison, 1990). It is also known that UK-born Afro-Caribbean babies have lower birth weights than either Caucasian or Asian infants, yet paradoxically rates of perinatal mortality are also lower (Terry et al, 1987; Pearson, 1991). Thus an increase in the number of infants of low birth weight surviving to adulthood may be a common factor linking various illnesses of neurodevelopmental origin. However, in contrast, it must also be pointed out that children born in the UK to mothers from the Caribbean have unusually low rates of stillbirth and perinatal mortality due to congenital and central nervous system abnormalities (Balarajan et al, 1989).

Such exposures may also change over time. Glover (1989a) has used hospital-admission data for two London regions to show that the increased risk of schizophrenia is most marked in men born in the Caribbean after 1950, and has proposed that these results reflect a cohort effect. It is possible that such an effect may be a result of epidemics during the same period, analogous to the effects of influenza epidemics reported in England and Finland. During the 1950s the Caribbean suffered a number of infectious epidemics - for example, epidemic poliomyelitis arrived in Jamaica in 1954 (Luck, 1966). We found that for those born in the West Indies, a peak was reached for those born in or before the early 1950s, and fell subsequently. At least part of this increase will be an artefact of emigration, since a proportion of those predisposed to develop schizophrenia may have become ill in the Caribbean, and thus not emigrated. This would differentially affect the older birth cohorts. Nevertheless, the data partially supports a cohort effect for young men born in the Caribbean between 1950 and 1960, as suggested by Glover (1989a). However, if one postulates exposure to an infective factor, then our results suggest increased maternal exposure to infection not only before migration, but also after.

Our evidence points to environmental factors affecting the risk of schizophrenia that continue to operate at all ages. Age at entry to the UK did not alter the risk of schizophrenia for those born in the West Indies, and thus those who entered as adults are no less at risk than those who enter as children. We thus conclude that several factors may be responsible for the increasing risk of schizophrenia in Afro-Caribbeans, and that these factors may operate at various periods, both pre-natally and in adult life. All these factors may in turn be related to social adversity and deprivation. Regrettably, as long as ethnicity and social adversity remain closely correlated, separating the various effects will be difficult.

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