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# Neighbourhood-level effects on psychoses: re-examining the role of context

JAMES B. KIRKBRIDE[1]*, CRAIG MORGAN[2], PAUL FEARON[2], PAOLA DAZZAN[2], ROBIN M. MURRAY[2] AND PETER B. JONES[1]

[1] *Department of Psychiatry, University of Cambridge, Cambridge, UK*; [2] *Division of Psychological Medicine, Institute of Psychiatry, King's College, London, UK*

## ABSTRACT

**Background.** The incidence of schizophrenia varies by individual-level characteristics and neighbourhood-level attributes. Few specific socio-environmental risk factors (SERFs) have been identified at the neighbourhood level. Cross-level interactions are poorly understood. We investigated these issues using data from the Aetiology and Ethnicity in Schizophrenia and Other Psychoses (AESOP) study.

**Method.** All incidence cases of ICD-10 schizophrenia (F20) and other non-affective psychoses (F21–29), aged 16–64 years, across 33 wards in Southeast London were identified over a 2-year period (1997–1999). Census data provided the denominator for each ward. Multilevel Poisson regression simultaneously modelled individual- and neighbourhood-level SERFs, including socio-economic deprivation, voter turnout (proxy for social capital), ethnic fragmentation (segregation) and ethnic density.

**Results.** A total of 218 subjects were identified during 565 576 person-years at risk. Twenty-three per cent of variance in incidence of schizophrenia across wards could be attributed to neighbourhood-level risk factors [95% confidence interval (CI) 9·9–42·2]. Thus, 1% increases in voter turnout [incidence rate ratio (IRR) 0·95, 95% CI 0·92–0·99] and ethnic segregation (IRR 0·95, 95% CI 0·92–0·99) were both independently associated with a reduced incidence of 5%, independent of age, sex, ethnicity, deprivation and population density. This was similar for other non-affective psychoses. There was some evidence that ethnic minority individuals were at greater risk of schizophrenia in areas with smaller proportions of minority groups ($p = 0{\cdot}07$).

**Conclusion.** SERFs at individual and neighbourhood levels were implicated in the aetiology of psychosis, but we were unable to determine whether these associations were causal. Individual risk may be mediated by social capital, which could operate as a protective factor, perhaps moderating social stress in the onset of psychoses.

## INTRODUCTION

In 1939, Faris and Dunham demonstrated variation in the incidence of schizophrenia across the city of Chicago, with the highest rates in the most socially disorganized tracts. In stark contrast, the affective psychoses showed no such variation. Subsequently, social class was proposed as a candidate explanation for these findings (Hollingshead & Redlich, 1958), but methodological limitations of the ecological approach, combined with difficulties disentangling social drift from causation, contributed to a disengagement of research into psychosis from the social sciences (Pilgrim & Roger, 2005). Ecological research was superseded by an

* Address for correspondence: James B. Kirkbride, Ph.D., Department of Psychiatry, University of Cambridge, Box 189, Cambridge CB2 2QQ, UK.
(Email: jbk25@cam.ac.uk)

individualistic approach to disease causation, and a belief that place played little or no role in causation (Jablensky *et al.* 1992; Crow, 2000).

This paradigm prevailed throughout the 1990s, despite evidence of a dose–response relationship between urban birth and upbringing and adult onset of schizophrenia (Lewis *et al.* 1992; Takei *et al.* 1992; Mortensen *et al.* 1999; Pedersen & Mortensen, 2001), implying that social causation, and not social drift, was the most parsimonious explanation of urban–rural differences in the incidence of psychosis. Despite this, few specific environmental correlates of risk for non-affective psychoses have been unequivocally identified. More recently, neighbourhood-level socio-environmental risk factors (SERFs) have been associated with psychoses, including deprivation (Croudace *et al.* 2000), social mobility (Silver *et al.* 2002) and social capital (Allardyce *et al.* 2005). Social capital can be considered as a group of features related to the social organization of a neighbourhood that, collectively, 'facilitate coordination and cooperation for mutual benefit' (Putnam, 1993, p. 36). Despite these findings, two recent reviews have highlighted the paucity of empirical evidence linking social capital to psychoses (McKenzie *et al.* 2002; De Silva *et al.* 2005).

There is some evidence that individual-level risk may be conditional upon neighbourhood characteristics (van Os *et al.* 2000). For example, Boydell *et al.* (2001) observed that the risk of schizophrenia for black and minority ethnic (BME) individuals increased in neighbourhoods with smaller proportions of BME residents (ethnic density hypothesis). This finding has been replicated in The Hague (Veling *et al.* 2006), but was not found in an earlier study in the UK (Cochrane & Bal, 1988).

Recent findings from the Aetiology and Ethnicity in Schizophrenia and Other Psychoses (AESOP) study also support variation in the incidence of schizophrenia – both between and within cities – beyond that which can be explained by individual-level differences in age, sex and ethnicity (Kirkbride *et al.* 2006, in press). These findings do not exclude the possibility that SERFs at the neighbourhood level may be aetiologically relevant to the onset of psychosis, perhaps involving gene–environment or other interactions (Krabbendam & van Os, 2005). By contrast, we found no evidence to support corresponding variance for the affective psychoses, either between or within cities, after adjustment for individual-level confounders.

In this study, we attempted to quantify the proportion of variation in incidence rates of non-affective psychoses that could be attributed to neighbourhood-level factors using data from the AESOP study. Having controlled for individual-level age, sex and ethnicity, we then attempted to explain any variation at the neighbourhood level by modelling potentially relevant SERFs, including markers of social capital, socio-economic deprivation, ethnic density and the interaction between ethnic density and individual-level ethnic status.

## METHOD

The AESOP study is a large, population-based first-episode psychosis study conducted over 2 years in three UK centres: Southeast London, Nottingham and Bristol. We have previously provided a detailed methodology of the AESOP study as well as descriptive epidemiology of the dataset and between-centre differences in rates of psychoses (Kirkbride *et al.* 2006). In the current study, we considered within-centre differences in the incidence of non-affective psychoses and potential individual- and neighbourhood-level effects that may have explained this variation in Southeast London. We did not include data from Nottingham or Bristol because our sample sizes were underpowered to test for neighbourhood effects using multilevel models in these centres.

### Population at risk

The population 'at risk' was estimated from the 2001 Census of Great Britain; the temporally closest data to our AESOP study period (September 1997–August 1999). All people aged 16–64 years residing in our Southeast London centre at the time of the census (29 April 2001) were included. This area comprised 33 Census Area Statistic (CAS) wards covering the Local Authorities (LA) of Lambeth and the southern two-thirds of Southwark. CAS wards (mean size: 5880 people) were introduced in the 2001 census to supersede electoral wards. We used CAS wards to define our neighbourhoods because a large amount of neighbourhood-level information was available at this level from

routine data sources. The census population was doubled to account for the 2-year study period.

## Case ascertainment

We identified everyone aged 16–64 years living in Southeast London who made contact with mental health services because of a first episode of any probable psychosis. Initial inclusion criteria were based upon those used in the World Health Organization (WHO) ten-country study (Jablensky *et al*. 1992). For these analyses, we also excluded cases if an address at first presentation was not obtained.

All potential cases presenting to psychiatric services were screened. We contacted health service bases weekly to identify all potential contacts. A leakage study, based on the methods of Cooper *et al*. (1987), was conducted after the survey period to identify subjects missed by the screening process. Our case-finding procedure thus included as many potential service bases as possible in order to estimate the true incidence of psychoses in our study area (Kirkbride *et al*. 2006).

Subjects who passed the screen underwent the Schedules for Clinical Assessment in Neuropsychiatry (SCAN) and a modified Personal and Psychiatric History Schedule. We completed the SCAN Item Group Checklist for all subjects who declined an interview, based upon case-notes and information from clinical staff. ICD-10 diagnoses were made by consensus agreement from a panel of clinicians, including a principal investigator and the clinical researcher who conducted the individual assessments. Inter-rater reliability was high (Kirkbride *et al*. 2006).

We obtained sociodemographic data for our cases from a schedule developed for the AESOP study (Mallett, 1997). Case ethnicity was ascribed using self-ascription, place of birth and parental place of birth, rated independently by three researchers (J.B.K., C.M. and P.F.). Discrepant classifications were resolved by consensus with a fourth researcher (P.B.J.). Inter-rater reliability was high ($\kappa = 0{\cdot}92$).

## Socio-environmental risk factors (SERFs)

### *Individual-level SERFs*

We included three individual-level risk factors in our study: age, sex and ethnicity. For each CAS ward, we stratified our numerator and denominator by age (16–19, 20–29, 30–39, 40–49, 50–64 years), sex and ethnicity; the latter ascribed to one of 16 categories defined in the 2001 census. For our analyses, we collapsed ethnicity into a seven-category variable: White British, Black Caribbean (including Black Other groups), Black African, Asian (Indian, Pakistani, Bangladeshi), Mixed ethnicity, White Other (White Irish and White Other) and Other ethnic groups. A dichotomous ethnicity variable (BME *versus* White British) was also used to test the ethnic density hypothesis (discussed later).

### *Neighbourhood-level SERFs*

We obtained data on neighbourhood-level SERFs that we hypothesized may be associated with psychoses. Our choice was guided by the previous literature. These variables were predominantly obtained from nationally collected, routine sources. We used the 2001 census to measure population density (people per hectare, pph), ethnic density (BME population as a proportion of total population) and ethnic fragmentation, using the Index of Dissimilarity (ID), in each ward.

The ID is a geographical measure of the extent to which two groups are evenly distributed (or segregated) across a neighbourhood (ward), frequently used in the social sciences (Peach, 1981, 1996). Here, we interpreted the ID as a measure of ethnic segregation between White British and BME populations. ID scores may vary between 0 (no segregation) and 100 (total segregation). To obtain an ID score for each ward, it was necessary to know the distribution of the White British and BME populations within each ward. This was determined by considering the proportion of White British and BME groups within Output Areas (OAs) in each ward. OAs are nested within CAS wards and provide the finest spatial scale at which the census is enumerated. In Southeast London the mean number of OAs per ward was 42·6, with a mean population size of 286 people per OA. The ID was calculated using the following formula:

$$\mathrm{ID} = \frac{1}{2}\sum_{i=1}^{N}\left|\frac{b_i}{B} - \frac{w_i}{W}\right| \times 100,$$

where $b_i$ is the BME population of the $i$th OA, $B$ is the total BME population in the CAS ward

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for which the ID is being calculated, $w_i$ is the White British population of the $i$th OA, and $W$ is the total White British population in the CAS ward for which the ID is being calculated.

ID can be interpreted as the proportion of one population needed to move to a different OA (in the same ward) to achieve an even distribution of the two populations across the ward. More segregated wards would require a larger proportion of either population to move between OA to achieve an even population distribution. As segregation increases, each ethnic group becomes less fragmented. Here, we interpret high ID scores as markers of less ethnic fragmentation within the White British and BME groups.

We used the Index of Multiple Deprivation (IMD) (2004) to measure deprivation within each CAS ward. The IMD incorporates six domains (unemployment, education, health, barriers to housing and services, living unit environment, crime) drawing on a range of nationally collected datasets, predominantly collected between 1997 and 2001 (ODPM, 2004). The IMD was released at Super Output Area (SOA) and LA level, but not CAS ward level directly, which is nested between these two levels of geography. To obtain a ward-level measure of deprivation, we therefore calculated the mean SOA IMD score for each CAS ward, weighting each SOA IMD score by its relative population contribution to the total CAS ward population. As IMD scores are not directly interpretable, we standardized them to have a mean of zero and standard deviation of one.

We obtained data on voter turnout at local government elections as a proxy for social capital. We considered that these data provided a proxy measure of social capital, representing the extent to which people within each ward were motivated by local-level issues. Local elections are held to determine political control of the LA, with each ward electing a candidate to represent their constituency. We obtained voter turnout data at the 2002 local elections for each ward within our study area (Rawlings & Thrasher, 2002); temporally closest to our study period using CAS ward geography.

### Statistical analyses

We used multilevel Poisson regression with random effects to model the incidence of schizophrenia (F20) and other non-affective psychoses (F21–29) between CAS wards. By fitting random effects at ward level we were able to quantify the variation in the incidence of each outcome between neighbourhoods. We entered potential individual- and neighbourhood-level SERFs as fixed effects and included the logarithm of our denominator as an offset, to test whether there was an association between neighbourhood-level SERFs and the incidence of non-affective psychoses.

All potential SERFs were entered into a univariate analysis as fixed effects. We monitored change in residual variance (variance in incidence at the neighbourhood level not explained by fixed effects) from the null model (without fixed effects) after fitting each SERF. We then entered all individual-level variables into a multivariate model, followed by significant neighbourhood-level variables, entered according to the magnitude in change of residual variance identified in the univariate analysis. We re-entered non-significant variables to ensure that they did not improve our final model. We tested the ethnic density hypothesis by considering an a priori interaction between individual-level ethnic status and neighbourhood-level ethnic density. For both outcomes, we tested this interaction in two models containing identical covariates, but differing with respect to the individual-level ethnicity variable used. The first model contained our seven-category ethnicity variable, while the second model was fitted with our dichotomous ethnicity variable, in line with the previous literature (Boydell *et al.* 2001; Veling *et al.* 2006). Significance testing of fixed effects and their interactions was assessed by likelihood ratio tests (LRTs). We reported the level of residual variation at the neighbourhood level and incidence rate ratios (IRRs) for all fixed effects. Modelling was conducted using XTPOISSON in STATA version 9 (StataCorp, 2005).

## RESULTS

We identified 228 cases of non-affective disorder in Southeast London during 565 000 person-years of follow-up. Ten cases (4·4%) were excluded because an address at first presentation was not obtained, leaving 218 cases. Sixty-eight per cent received a diagnosis of schizophrenia ($n=148$) (ICD-10 F20), the remaining subjects

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Table 1. *Demographic characteristics of sample and denominator population*

| | Non-affective psychoses (F20–29) | Schizophrenia (F20) | Other non-affective psychoses (F21–29) | Denominator (person-years) |
|---|---|---|---|---|
| Total | 218 (100) | 148 (67·9) | 70 (32·1) | 565 576 |
| Sex | | | | |
| Men | 125 (57·3) | 91 (61·5) | 34 (48·6) | 277 196 (49·0) |
| Women | 93 (42·7) | 57 (38·5) | 36 (51·4) | 288 380 (51·0) |
| Age | | | | |
| Under 30 years | 109 (50·0) | 70 (47·3) | 39 (55·7) | 197 932 (35·0) |
| 30 years and older | 109 (50·0) | 78 (52·7) | 31 (44·3) | 367 644 (65·0) |
| Ethnicity | | | | |
| White | 55 (25·2) | 33 (22·3) | 22 (31·4) | 363 856 (64·3) |
| BME | 163 (74·8) | 115 (77·7) | 48 (68·6) | 201 720 (35·7) |

BME, Black and minority ethnic.
Values are given as *n* (%).

Table 2. *Correlation matrix for neighbourhood-level socio-environmental risk factors (SERFs)*

| Neighbourhood-level SERFs | 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|
| 1. Ethnic density | 1·00 | | | | |
| 2. Ethnic fragmentation | 0·22 | 1·00 | | | |
| 3. Voter turnout | −0·43* | 0·13 | 1·00 | | |
| 4. Population density | 0·67** | 0·38* | −0·31 | 1·00 | |
| 5. Deprivation | 0·84** | 0·11 | −0·43* | 0·49** | 1·00 |

* Significant at $p<0·05$. ** Significant at $p<0·01$.

having other non-affective psychoses ($n=70$). Fifty-seven per cent of our sample were men, 50% under 30 years of age, and 75% from a BME group (Table 1). In comparison, men comprised 49% of our denominator, 35% of the denominator were under 30 years of age, and 36% were from a BME group.

Neighbourhood-level SERFs were normally distributed with little evidence of skewness (data available on request). We observed considerable heterogeneity by ward in terms of ethnic density (24·8–74·3%) and population density (31·8–154·5 pph). The range of values was narrower for ethnic fragmentation (11·1–32·9%) and voter turnout (17·1–41·6%). The IMD was normally distributed across the wards in our study region. However, when we compared the rank position of our SOA IMD scores with all SOAs in England, there was evidence that our study area was relatively deprived; 95% of the 268 SOAs in our CAS wards were in the most deprived 50% of SOAs in England. We observed significant correlations between several neighbourhood-level SERFs (Table 2). Notably, socio-economic deprivation was highly positively correlated with the proportion of BME residents at the neighbourhood level (ethnic density) ($r^2=0·84$, $p<0·01$) and with population density ($r^2=0·49$, $p<0·01$). Voter turnout was negatively correlated with socio-economic deprivation ($r^2=-0·43$, $p<0·05$) and the proportion of BME residents at the neighbourhood level ($r^2=-0·43$, $p<0·05$).

## Schizophrenia

We observed considerable neighbourhood-level variance for schizophrenia in our null multilevel model [intraclass correlation coefficient (ICC) 0·36, 95% CI 0·15–0·82, $p<0·01$], representing 26·5% [0·36/(1+0·36)] of total variance (95% CI 13·0–45·1) (Table 3). Adjustment for individual age, sex and ethnicity led to a reduction in the proportion of variance attributable to the neighbourhood level, but this variation remained statistically significant (22·5%, 95% CI 9·9–42·2). There was evidence from univariate analyses that deprivation, ethnic fragmentation and voter turnout were associated with the incidence of schizophrenia. These variables were entered into our multivariate model.

Voter turnout and ethnic fragmentation were significantly associated with schizophrenia at the neighbourhood level after adjustment for individual-level age, sex and ethnicity and neighbourhood-level deprivation (multivariate model, Table 3). Thus, a 1% increase in voter turnout was associated with a reduction in the incidence of schizophrenia of 5% (IRR 0·95, 95% CI 0·92–0·99). Similarly, as wards became

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Table 3. *Modelling of individual- and neighbourhood-level socio-environmental risk factors for schizophrenia*

| | | Univariate | | | | | Multivariate, individual level adjusted analysis | |
|---|---|---|---|---|---|---|---|---|
| | | Fixed part of model | | Random part of model | | | Fixed part of the model | |
| Type of SERF | SERF | Unadjusted IRR (95% CI) | Wald *p* value | Level-2 neighbourhood variance (95% CI) | $\chi^2$ (1 df) | $\chi^2$ *p* value | Adjusted IRR (95% CI) | LRT *p* value |
| Individual-level | None | — | — | 0·36 (0·15–0·82) | 19·3 | <0·01 | — | — |
| | Sex (M *v.* F) | 1·68 (1·20–2·33) | <0·01 | 0·36 (0·16–0·83) | 19·7 | <0·01 | 1·85 (1·33–2·58) | <0·01[a] |
| | Age | | | | | | | |
| | 16–19 | 1 | — | 0·37 (0·16–0·84) | 20·2 | <0·01 | 1 | <0·01[a] |
| | 20–29 | 0·71 (0·41–1·24) | 0·23 | | | | 0·99 (0·57–1·72) | |
| | 30–39 | 0·59 (0·34–1·03) | 0·06 | | | | 0·72 (0·41–1·25) | |
| | 40–49 | 0·27 (0·13–0·55) | <0·01 | | | | 0·32 (0·16–0·66) | |
| | 50–64 | 0·37 (0·19–0·73) | <0·01 | | | | 0·49 (0·25–0·98) | |
| | Ethnicity | | | 0·27 (0·10–0·71) | 16·0 | <0·01 | | <0·01[a] |
| | White British | 1 | | | | | 1 | |
| | Black Caribbean | 6·06 (3·91–9·43) | <0·01 | | | | 6·42 (4·11–10·04) | |
| | Black African | 3·77 (2·30–6·17) | <0·01 | | | | 3·93 (2·39–6·46) | |
| | Asian | 0·92 (0·22–3·83) | 0·91 | | | | 0·86 (0·21–3·58) | |
| | Mixed ethnicity | 2·75 (1·15–6·57) | 0·02 | | | | 2·57 (1·07–6·16) | |
| | White Other | 2·01 (1·12–3·60) | 0·02 | | | | 2·04 (1·14–3·67) | |
| | Other ethnicity | 2·63 (1·10–6·30) | 0·03 | | | | 2·57 (1·07–6·15) | |
| Neighbourhood-level equivalent of individual level | Ethnic density[b] | 1·02 (1·00–1·04) | 0·10 | 0·30 (0·12–0·75) | 13·6 | <0·01 | 1·00 (0·97–1·03) | 0·94 |
| Neighbourhood-level | Ethnic fragmentation[b] | 0·95 (0·91–0·99) | 0·02 | 0·27 (0·11–0·69) | 13·2 | <0·01 | 0·95 (0·92–0·99) | 0·01[a] |
| | Voter turnout[b] | 0·93 (0·89–0·98) | <0·01 | 0·23 (0·08–0·64) | 9·7 | <0·01 | 0·95 (0·92–0·99) | 0·03[a] |
| | Population density[b] | 1·00 (0·99–1·01) | 0·77 | 0·36 (0·15–0·82) | 19·2 | <0·01 | 1·00 (0·99–1·01) | 0·49 |
| | Deprivation | | | 0·21 (0·07–0·63) | 8·9 | <0·01 | | |
| | Lowest third | 1 | | | | | 1 | 0·12[a] |
| | Middle third | 0·78 (0·43–1·42) | 0·42 | | | | 0·70 (0·39–1·24) | |
| | Highest third | 1·76 (1·02–3·04) | 0·04 | | | | 1·50 (0·74–3·01) | |
| Cross-level interaction between ethnic status and ethnic density | | | | — | — | — | — | 0·19 |
| Residual variance at level-2 after adjustment for individual-level SERFs | | | | 0·29 (0·11–0·73) | 13·2 | <0·01 | — | — |
| Residual variance at level-2 in multivariate model | | | | 0·04 (0·00–2·30) | 0·3 | 0·30 | — | — |

SERFs, Socio-environmental risk factors; IRR, incidence rate ratio; LRT, likelihood ratio test; df, degrees of freedom; CI, confidence interval.

[a] Individual- and neighbourhood-level variables included in final model.

[b] IRR reports change in risk of schizophrenia associated with a 1% increase in the neighbourhood-level variable.

less ethnically fragmented, the incidence of schizophrenia fell (IRR 0·95, 95% CI 0·92–0·99). These findings were independent of deprivation, which did not significantly improve the model (LRT $p=0·12$). Population density and ethnic density were not associated with schizophrenia and were removed from the final model. After fitting the final multivariate model, there was no evidence of significant residual variance at the neighbourhood level ($\chi^2$ $p=0·30$). IRRs for several ethnic groups were raised, as reported previously (Fearon *et al.* 2006), and were not significantly attenuated by adjustment for neighbourhood-level SERFs.

There was no evidence that the association between schizophrenia and ethnicity (seven-category) was modified by ethnic density in our final multivariate model (LRT $p=0·18$) (Table 3). However, when we tested this interaction using our dichotomous ethnicity variable, retaining the above variables in our final model (age, sex, deprivation, voter turnout and ethnic fragmentation), we observed some evidence (LRT $p=0·07$) that the risk of schizophrenia in the BME group was conditional upon the ethnic density of the neighbourhood. Thus, the risk of schizophrenia for BME individuals was highest in the third of wards with the smallest proportion of BME residents (IRR 6·51, 95% CI 3·01–14·08). This risk appeared to be lower for BME residents living in more ethnically dense neighbourhoods, despite some imprecision in our estimates (Table 4).

Table 4. *Interaction between individual-level BME status (dichotomous) and ethnic density for schizophrenia*

| Person–environment interaction ($p=0·07$) | IRR (BME *versus* White British) (95% CI)[a] |
|---|---|
| Ethnic density (% BME) | |
| Lowest third (24·8–47·1) | 6·50 (3·00–14·06) |
| Middle third (47·2–56·1) | 2·13 (1·17–3·88) |
| Upper third (56·4–74·3) | 3·81 (1·86–7·79) |

IRR, Incidence rate ratio; BME, black and minority ethnic; CI, confidence interval.

[a] After adjustment for age, sex, deprivation, ethnic fragmentation and voter turnout. Model identical to final model presented in Table 3, except a dichotomous ethnicity variable (BME *versus* White British) was fitted instead of the seven-category ethnicity variable to test the interaction with ethnic density.

### Other non-affective psychoses

We observed significant neighbourhood-level variance for other non-affective psychoses in our null model (23·1%, 95% CI 6·5–53·9, $p=0·02$) (Table 5). Adjustment for age, sex and ethnicity at the individual level led to some attenuation of this figure, with resulting imprecision in the estimate of variance attributable to neighbourhood-level characteristics (15·3%, 95% CI 2·9–55·0, $p=0·09$). Nevertheless, neighbourhood-level SERFs were significantly associated with the incidence of other non-affective psychoses in our multivariate model. As for schizophrenia, increased voter turnout was associated with a lower incidence of other non-affective psychoses (IRR 0·93, 95% CI 0·88–0·98), independent of age, sex, ethnicity, population density (IRR 0·98, 95% CI 0·97–0·99) and ethnic density (IRR 1·04, 95% CI 1·01–1·08). Ethnic fragmentation (LRT $p=0·70$) and deprivation (LRT $p=0·99$) were not significantly associated with other non-affective psychoses and were removed from the final multivariate model. There was no evidence that ethnic density interacted with individual-level ethnicity, using either the model fitted with our seven-category (LRT $p=0·43$) or the dichotomous ethnicity variable (LRT $p=0·43$). As for schizophrenia, the elevated incidence of other non-affective psychoses in BME groups was not attenuated by inclusion of neighbourhood-level characteristics (multivariate model, Table 5). After fitting our final model, there was no evidence of significant residual variance at the neighbourhood level.

## DISCUSSION

### Principal findings

The incidence of schizophrenia and other non-affective psychoses varied at both the individual and neighbourhood levels within Southeast London. Approximately 23% and 15% of variance of each disorder respectively could be attributed to neighbourhood-level risk factors after adjustment for age, sex and ethnicity.

Social capital, as measured by voter turnout, was a significant predictor of both outcomes, independent of age, sex, ethnicity and socioeconomic deprivation. The incidence of non-affective psychoses was higher in wards with lower voter turnout at local elections. We also observed a significant association between ethnic fragmentation and schizophrenia, suggesting

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Table 5. *Modelling of individual- and neighbourhood-level socio-environmental risk factors for other non-affective psychoses*

| | | Univariate | | | | | Multivariate, individual level adjusted analysis | |
|---|---|---|---|---|---|---|---|---|
| | | Fixed part of model | | Random part of model | | | Fixed part of the model | |
| Type of SERF | SERF | Unadjusted IRR (95% CI) | Wald *p* value | Level-2 neighbourhood variance (95% CI) | $\chi^2$ (1 df) | $\chi^2$ *p* value | Adjusted IRR (95% CI) | LRT *p* value |
| Individual-level | None | — | — | 0·30 (0·07–1·17) | 4·1 | 0·02 | — | — |
| | Sex (M *v.* F) | 0·99 (0·62–1·58) | 0·96 | 0·30 (0·07–1·17) | 4·1 | 0·02 | 1·07 (0·67–1·71) | 0·77[a] |
| | Age | | | | | | | |
| | 16–19 | 1 | — | 0·31 (0·08–1·19) | 4·4 | 0·02 | 1 | 0·01[a] |
| | 20–29 | 0·74 (0·35–1·57) | 0·44 | | | | 1·04 (0·49–2·22) | |
| | 30–39 | 0·41 (0·18–0·91) | 0·03 | | | | 0·48 (0·21–1·07) | |
| | 40–49 | 0·27 (0·10–0·72) | 0·01 | | | | 0·31 (0·11–0·83) | |
| | 50–64 | 0·26 (0·09–0·73) | 0·01 | | | | 0·33 (0·12–0·93) | |
| | Ethnicity | | | 0·17 (0·02–1·26) | 3·0 | 0·04 | | <0·01[a] |
| | White British | 1 | | | | | 1 | |
| | Black Caribbean | 4·96 (2·82–8·72) | <0·01 | | | | 4·78 (2·69–8·50) | |
| | Black African | 2·50 (1·27–4·91) | <0·01 | | | | 2·39 (1·20–4·76) | |
| | Asian | No cases | — | | | | No cases | |
| | Mixed ethnicity | 0·68 (0·09–5·08) | 0·71 | | | | 0·58 (0·08–4·33) | |
| | White Other | 0·70 (0·24–2·03) | 0·51 | | | | 0·69 (0·24–2·00) | |
| | Other ethnicity | 0·64 (0·09–4·79) | 0·67 | | | | 0·58 (0·08–4·33) | |
| Neighbourhood-level equivalent of individual level | Ethnic density[b] | 1·03 (1·01–1·06) | 0·02 | 0·13 (0·01–1·68) | 0·9 | 0·18 | 1·04 (1·01–1·08) | 0·01[a] |
| Neighbourhood-level | Ethnic fragmentation[b] | 0·97 (0·92–1·02) | 0·29 | 0·27 (0·06–1·15) | 3·5 | 0·03 | 0·99 (0·94–1·04) | 0·70 |
| | Voter turnout[b] | 0·92 (0·87–0·97) | <0·01 | 0·05 (0·00–10·41) | 0·2 | 0·34 | 0·93 (0·88–0·99) | 0·02[a] |
| | Population density[b] | 1·00 (0·99–1·01) | 0·66 | 0·29 (0·07–1·17) | 4·0 | 0·02 | 0·98 (0·97–0·99) | <0·01[a] |
| | Deprivation[c] | 1·44 (1·10–1·87) | <0·01 | 0·10 (0·01–2·13) | 0·6 | 0·22 | 1·00 (1·66–1·52) | 0·99 |
| Cross-level interaction between ethnic status and ethnic density | | | | — | — | — | — | 0·43 |
| Residual variance at level-2 after adjustment for individual-level SERFs | | | | 0·18 (0·03–1·23) | 3·4 | 0·09 | — | — |
| Residual variance at level-2 in multivariate model | | | | $9{\cdot}24 \times 10^{-8}$ | — | — | — | — |

SERF, Socio-environmental risk factors; IRR, incidence rate ratio; LRT, likelihood ratio test; df, degrees of freedom; CI, confidence interval.

[a] Individual- and neighbourhood-level variables included in final model.

[b] IRR reports change in risk of schizophrenia associated with a 1% increase in the neighbourhood-level variable.

[c] IRR reports change in risk of schizophrenia associated with 1 standard deviation increase in socio-economic deprivation at the neighbourhood-level variable.

that the incidence of schizophrenia was lower in wards where White British and BME individuals lived in more cohesive, less fragmented groups. We found no evidence of person–environment interaction between individual-level ethnic status and neighbourhood proportion of BME groups when we considered a broad ethnicity variable. However, when we used a dichotomous variable, we observed some evidence of an interaction, such that BME individuals were at greatest risk of schizophrenia in wards where they constituted a smaller proportion of the total population. These findings suggest that ethnic density is likely to interact with individual-level ethnicity in a more complex way than previously considered with regard to psychoses. Our results raise the possibility that contextual, neighbourhood-level factors operate somewhere along the causal pathway for both schizophrenia and other non-affective psychoses.

## Methodological considerations

### *Denominator population and individual-level risk factors*

We have previously discussed potential limitations of using the 2001 census to estimate our denominator population (Kirkbride *et al*. 2006). We acknowledge that the true, dynamic, population at risk over the survey period may have varied slightly, but we do not believe we have introduced systematic bias.

We may not have adequately controlled for all individual-level risk, which could have led to an overestimation of the effect of the neighbourhood. Specifically, we did not have data on family history of psychosis, the most important individual-level risk factor for disorder (Mortensen *et al*. 1999). This could have explained a degree of clustering of risk in some neighbourhoods, although the relationship between urbanicity and schizophrenia appears to persist independently of family history (Lewis *et al*. 1992; Mortensen *et al*. 1999). To minimize this problem in our study, we used incidence rather than prevalence data over a short study period. Furthermore, we conducted a sensitivity analysis to exclude any subsequent cases with the same postcode (as a potential marker for genetic clustering of cases within the same family). This did not change our results (data available on request).

We recognize that individual-level social class may have accounted for some of the variation we observed at the neighbourhood level (Silver *et al*. 2002). Unfortunately, we could not obtain denominator data stratified by a fourth variable (social class) at the ward level because of confidentiality issues in the 2001 census. We included its neighbourhood-level equivalent, socio-economic deprivation, given it is unclear at which level social class may operate (Diez-Roux *et al*. 1997). Furthermore, social class may only present an indicator of risk for other underlying SERFs (Jones *et al*. 1993; Cooper, 2005). Our data are consistent with that view.

Our findings could have been explained by downward social drift of psychotic individuals into poorer, less cohesive neighbourhoods. However, social causation appears to present a more parsimonious explanation of the 'urbanicity' effect than social drift (van Os, 2004). We measured place of residence at onset of psychosis rather than at birth or during upbringing (Lewis *et al*. 1992; Mortensen *et al*. 1999). It is plausible, however, that neighbourhood setting close to onset is relevant to the aetiology of psychoses. If prodromal or At-Risk Mental State (ARMS) individuals drift downwards (socially and geographically), neighbourhood risk factors (close to onset) may be sufficient to induce (or fail to 'buffer' against) psychotic disorders. Thus, downward social drift would operate synergistically with causal effects at the neighbourhood level. We would therefore expect to observe significant associations between the incidence of psychoses and neighbourhood-level effects close to the onset of disorder. Longitudinal studies are clearly required to elucidate the crucial exposure period(s) to 'urbanicity'.

### *Neighbourhood-level SERFs*

We obtained neighbourhood-level SERFs from a variety of routinely collected national datasets. Ethnic density and fragmentation were calculated from the 2001 census. Our ethnic density variable differed slightly from a previously used measure (Boydell *et al*. 2001) because we included the non-British white population in the BME group. We took this decision because a recent study has demonstrated that non-British white migrants may be at increased risk of

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schizophrenia compared to their British-born counterparts (Fearon *et al*. 2006).

The ID is a validated measure of segregation (Peach, 1981, 1996), to our knowledge applied here to psychiatric disorders for the first time. As absolute levels of segregation in Southeast London were low, and because the term segregation has negative connotations, it was helpful to consider this variable as a measure of residential fragmentation within the White British and BME groups in the same neighbourhood. Ethnic fragmentation and density appear to have distinct effects, providing some justification for inclusion of the ID. The ID measures the degree of residential segregation in an area, rather than the spatial structure of this patterning. A measure of ethnic fragmentation that takes into account the spatial clustering of residential patterns may provide further insights into the association between ethnic fragmentation and schizophrenia.

We recognize that voter turnout provides an imperfect measure of social capital. It may be influenced by a range of sociopolitical, sociocultural and sociodemographic factors (Smith & Dorling, 1996), not restricted to local-level issues. We attempted to minimize this by using data from local elections in an election year where national elections were not being held concurrently. In addition, voter turnout may have measured some factors that should have been specified at the individual level, such as social class or education. However, although voter turnout was our primary measure of social capital, ethnic fragmentation, which may also present a marker for social capital, was independently associated with the incidence of schizophrenia. Voter turnout is a reliable, routinely collected measure, but more detailed measures of social capital, such as the scales proposed by Sampson *et al*. (1997) or McCulloch (2001), may provide further aetiological clues to this potential association.

Voter turnout data were obtained from local elections in 2002 for our study area. This was the closest election to our study period based on the new census geography used in this study. We acknowledge that some of our neighbourhood SERF variables have been measured subsequent to our case ascertainment period, but we have attempted to minimize any error by using the temporally closest data available.

We consider that the IMD provided a suitable measure of deprivation. It incorporates a number of domains, meaning that it is unlikely that our findings could be explained by residual confounding, However, separate consideration of these domains may further our aetiological understanding of the role of socio-economic deprivation. Although released in 2004, the IMD used data collected close to, or during, the AESOP study period (ODPM, 2004). IMD was not significant in our multivariate model for other non-affective psychoses, but this is most likely to be due to its high positive correlation with ethnic density.

We chose to analyse neighbourhoods at the ward level. Wards are, however, unlikely to be conterminous with neighbourhoods as perceived by the people who live in them. If we could measure more 'ecological' neighbourhoods we would be better placed to understand the association between neighbourhood-level SERFs and psychoses. Here, however, our primary concern was to obtain reliable neighbourhood-level measures from routine sources. Inspection of ward boundaries suggested that they were not entirely arbitrary; several followed major roads, railways or parks, which may have some ecological validity.

### Meaning of findings

The above caveats notwithstanding, it remains important to discuss the potential causal pathways that may underlie the associations we have observed here, if such associations were genuinely causal.

Stress is associated with dopamine dysregulation (Howes *et al*. 2004), which may be important in the pathogenesis of schizophrenia. If city living increases exposure to social stressors, this may be a potential mechanism through which residency in urban areas induces psychotic symptoms, particularly in genetically vulnerable individuals or where there is no 'buffering' by external factors. It is plausible that social stress is mediated – or buffered – by social capital, such that in neighbourhoods with high social capital, individuals are able to dissipate social stress through access to actual or perceived support networks. By contrast, less cohesive neighbourhoods may foster paranoid attributional styles that, in some susceptible individuals, may eventually manifest as psychotic

symptoms (Garety *et al*. 2001; van Os *et al*. 2001). Having said this, we acknowledge the potential for reverse causality; a paranoid attributional style could lead to the formation of less cohesive neighbourhoods.

We have observed differential findings regarding the ethnic density hypothesis. When we operationalized the hypothesis as previously tested (interaction between dichotomous ethnicity variable and proportion of BME groups in a neighbourhood), we found weak evidence that the risk of schizophrenia for BME individuals was greatest when they constituted a smaller proportion of the overall neighbourhood population. This finding supports two recent studies that have observed similar interactions (Boydell *et al*. 2001; Veling *et al*. 2006), lending weight to the 'social capital as mediator' hypothesis outlined earlier. It appears that the risk of schizophrenia for BME individuals is greatest when they are more isolated from others who may share similar beliefs, cultures, values, attitudes or experiences of city life. Thus, there may be less opportunity for these individuals to mediate social stress by accessing social capital. This finding complements the independent association we observed between risk of schizophrenia and increasing ethnic fragmentation. It is possible that White British and BME groups that have formed more cohesive groups confer protection from psychotic illness by gaining increased access to social support.

When, however, we operationalized the ethnic density hypothesis using our seven-category ethnicity variable, we did not observe evidence of a significant interaction with ethnic density, for either outcome. We suggest three possible explanations for this absence of association. First, it may be genuine. Second, our study is likely to be underpowered to test cross-level interactions, particularly using a broad ethnicity variable. Third, our ethnic density variable may not be valid for different ethnic groups. As investigated here, ethnic density measured the proportion of White British residents in each ward. Conceivably, this may only be a relevant risk factor for psychoses in the White British group. The relevant 'ethnic density' variable for the Black Caribbean group may be the density of Black Caribbean residents in a neighbourhood, compared with all other groups. The relevant 'ethnic density' variable for non-British white migrants may be the density of non-British white, or more specifically Polish, Turkish, Italian or Portuguese migrants (et cetera) at the neighbourhood level. To investigate these pertinent issues, ethnic density needs to be tested for specific ethnic groups in larger samples.

### Comparison with previous studies

Multilevel studies investigating the respective roles of individual- and neighbourhood-level risk factors for schizophrenia remain sparse. However, one previous study, undertaken in Maastricht, reported that 12% of the variation in the incidence of schizophrenia could be attributed to neighbourhood-level factors (van Os *et al*. 2000). This figure was half that observed in our study, but both findings may be valid given the level of urbanization in Southeast London compared with Maastricht. Of note, neighbourhood variation in Maastricht does not appear to be associated with social capital (Drukker *et al*. 2006). However, Silver *et al*. (2002) found evidence to suggest the level of residential mobility was associated with the risk of schizophrenia, and a complementary finding has been observed by Allardyce *et al*. (2005) in Scotland with respect to social fragmentation. Our findings on voter turnout and ethnic fragmentation support the hypothesis that social capital mediates the relationship between exposure to stress and later psychoses (Myin-Germeys *et al*. 2001; McKenzie *et al*. 2002; Krabbendam & van Os, 2005).

It appears that the relationship between schizophrenia and social class is complicated, and factors allied to social class, such as social capital, may be more aetiologically relevant. This idea is not new; Faris & Dunham (1939) hypothesized that it was the most socially disorganized neighbourhoods, not the most socioeconomically deprived neighbourhoods *per se*, that had the highest incidence of schizophrenia. Our findings support this hypothesis.

## CONCLUSIONS

Variation in the incidence of schizophrenia and other psychoses in Southeast London cannot solely be explained by individual-level differences in age, sex and ethnicity. Neighbourhood-level

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socio-environmental risk factors appear to explain approximately 23% and 15% respectively of this variation. Voter turnout, ethnic density, ethnic fragmentation and socio-economic deprivation were significant predictors of risk at the neighbourhood level. Social capital is one mechanism that potentially binds these associations together, perhaps acting to mediate social stress. These findings support and extend Faris and Dunham's hypothesis that the highest rates of schizophrenia occurred in the most disorganized tracts of the city, not necessarily the poorest.

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## DECLARATION OF INTEREST

Robin M. Murray is an editor on the board of *Psychological Medicine*.

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