

Social Deprivation, Inequality, and the Neighborhood-Level Incidence of Psychotic Syndromes in East London

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Although urban birth, upbringing, and living are associated with increased risk of nonaffective psychotic disorders, few studies have used appropriate multilevel techniques accounting for spatial dependency in risk to investigate social, economic, or physical determinants of psychosis incidence. We adopted Bayesian hierarchical modeling to investigate the sociospatial distribution of psychosis risk in East London for DSM-IV nonaffective and affective psychotic disorders, ascertained over a 2-year period in the East London first-episode psychosis study. We included individual and environmental data on 427 subjects experiencing first-episode psychosis to estimate the incidence of disorder across 56 neighborhoods, having standardized for age, sex, ethnicity, and socioeconomic status. A Bayesian model that included spatially structured neighborhood-level random effects identified substantial unexplained variation in nonaffective psychosis risk after controlling for individual-level factors. This variation was independently associated with greater levels of neighborhood income inequality (SD increase in inequality: Bayesian relative risks [RR]: 1.25; 95% CI: 1.04–1.49), absolute deprivation (RR: 1.28; 95% CI: 1.08–1.51) and population density (RR: 1.18; 95% CI: 1.00–1.41). Neighborhood ethnic composition effects were associated with incidence of nonaffective psychosis for people of black Caribbean and black African origin. No variation in the spatial distribution of the affective psychoses was identified, consistent with the possibility of differing etiological origins of affective and nonaffective psychoses. Our data suggest that both absolute and relative measures of neighborhood social composition are associated with the incidence of nonaffective psychosis. We suggest these associations are consistent with a role for social stressors in psychosis risk, particularly when people live in more unequal communities.

Key words: psychosis/epidemiology/urban/social environment/inequality/deprivation/Bayesian modeling/schizophrenia

Introduction

Urban birth, upbringing, and living appear to be associated with increased risk of nonaffective psychotic syndromes,^{1,2} but not affective psychoses.^{3–5} For nonaffective psychoses, the accumulated evidence with respect to urbanicity meets many of the Bradford-Hill criteria for causation.⁶ For example, this phenomenon has been frequently replicated,^{7–17} in studies of different designs,^{7,10,11,13,14} and persists after control for different patterns of confounders, including family history,^{7,10,11} parental age at birth,¹⁰ substance use,⁷ age,^{10–12,14–17} season of birth,¹⁰ sex,^{10,12,15–17} marital status,¹⁴ education,^{12,14} immigration status,^{12,14} and ethnicity.^{8,9,16} Risk also follows a dose-response relationship with both degree of urbanicity and time resident in urban environments.¹¹ Risk begins early in life and remains present through adolescence and into adulthood. Finally, familial liability to psychosis is modified after moving to or from more or less urbanized environments,^{12,18–20} suggesting an interplay between genetic and environmental factors influences psychosis risk.

Despite this, less is understood about the specific environmental factors that underpin these associations. Various factors have been hypothesized, including impaired nutrition,²¹ infection,²² and social stress.²³ Higher risk in urban environments may theoretically be explained by increased transmission of infections associated with increased schizophrenia risk,²² or insufficient exposure to sunlight and vitamin D early in life in densely populated environments.²⁴ In extreme conditions, such as famines, early life dietary malnutrition (particularly of key micronutrients related to normal one-carbon metabolism and brain development) appears to increase adulthood risk of psychotic disorders.^{25,26} It is unknown whether poor nutrition as a result of living in deprived, urban environments also increases psychosis risk. Furthermore, nutrition- and infection-related hypotheses assume that

the critical exposure window is early in life, but the association between schizophrenia and urbanicity persists into adolescence and adulthood.^{7,16}

Social environmental factors, which may operate throughout the life course, have received greater attention as an explanation of schizophrenia risk in urban areas. Incidence of schizophrenia is raised in more deprived communities,²⁷ where social and economic strains are likely to be more severe. Other studies have observed higher rates of schizophrenia in neighborhoods with less social support,^{13,15,28–31} conceptualized using measures including social cohesion, fragmentation, and residential mobility. Emerging research suggests that some social factors may operate in a relative rather than absolute fashion^{32–36}; eg, psychosis risk among migrants and their descendants appears to be partly conditional upon both the proportion of and the proximity to one's own ethnic group in the community.^{34–36} Neighborhood socioeconomic inequality (ie, relative rather than absolute deprivation) may also be associated with schizophrenia risk though no study has yet investigated this in a multilevel, population-based epidemiological study of first-episode psychosis (FEP).

While recent epidemiological studies of psychosis have used multilevel techniques to simultaneously model individual and neighborhood effects,^{4,13,28,30,35,36} only 1 study⁴ has additionally accounted for spatial patterning in incidence that might betray important underlying covariation in environmental risk. However, this study did not control for individual-level social class or investigate which neighborhood factors accounted for neighborhood variation in psychosis risk. Using appropriate Bayesian hierarchical spatial models, we therefore sought to investigate whether the incidence of psychotic disorders varied between neighborhoods in East London, after adjustment for individual-level age, sex, ethnicity, and social class. We tested whether such variation was associated with several neighborhood-level environmental factors, including absolute (deprivation, social fragmentation, social cohesion, and population density) and relative (inequality, ethnic density, and ethnic separation) measures.

Methods

Study Design

Data were obtained from the East London first-episode psychosis (ELFEP) study,^{37,38} a large population-based incidence study conducted in 3 neighboring inner-city, ethnically diverse and socially deprived boroughs of London, UK: City & Hackney, Newham, and Tower Hamlets. Ethical approval was obtained from the local research ethics committee in East London.

Subject Ascertainment

We identified everyone aged 18–64 years resident in our catchment area who made contact with mental health

services with a first episode of any probable psychosis, nonpsychotic mania, or bipolar disorder. The study took place over 24 months; December 1996–November 1998 in City & Hackney, December 1998–November 2000 in Newham and Tower Hamlets. Health services were contacted weekly to identify potential subjects. A leakage study was conducted to minimize missed subjects, using the method by Cooper et al.³⁹ Subjects completed several assessments, including the Schedules for Clinical Assessment in Neuropsychiatry (SCAN) and a sociodemographic schedule. Consensus diagnoses were made using the SCAN and all other available information, according to the Diagnostic and Statistical Manual (4th edition) (DSM-IV). Here, we investigated 2 outcomes; nonaffective psychoses (including schizophrenia, schizophreniform disorder, and schizoaffective disorder) (DSM-IV 295.xx, 297.xx, 298.8, 298.9) and the affective psychoses (DSM-IV 296.x4, 296.4, 296.89).

Residential address at first contact was geocoded to their respective statistical ward as the neighborhood unit of analysis. ELFEP included 60 such wards with a mean population size of 6195 (2001 census). We excluded 4 wards that covered London's main financial district, the City of London, because the resident population was sparse and atypical. No incident subjects were observed in these areas. Subjects without fixed abode, or who could not otherwise be geocoded were excluded.

Variable Generation

Individual-Level Variables. Age-at-first-contact was categorized into 5 age-bands; 18–24, 25–34, 35–44, 45–54, 55–64 years. Ethnicity was ascribed to 1 of 16 categories in the 2001 census using all available information, including self-ascription, place of birth, and parental place of birth. From this, we created a 10-category variable: white British, non-British white, black Caribbean, black African, mixed white and black Caribbean, other mixed ethnicities, Indian, Pakistani, Bangladeshi, and all other ethnic groups. Social class was based on main occupation, using decision rules provided for the Office for National Statistics' National Statistics-Socioeconomic Classification (NS-SEC).⁴⁰ We used a 7-category social class variable: managerial and professional, intermediate occupations, small account and own account workers (self-employed), lower supervisory and technical occupations, semiroutine and routine occupations, never worked and long-term unemployed, and "not classifiable" for other reasons (eg, students or people with inadequately stated occupations).

Neighborhood-Level Variables. We characterized statistical wards according to several a priori environmental factors collected from routine data sources immediately prior to, or during, the case ascertainment period (table 1). Deprivation was estimated from 2004 English Indices

Table 1. Overview of Neighborhood Variables in East London and England

Variable	Percentile			Mean (SD)	Mann-Whitney 2-Sample Test z-Score; P-Value
	10th	Median	90th		
Index of Multiple deprivation ^a					11.5; < 0.01
East London	35.6	42.5	51.5	43.8 (6.1)	
England	6.8	14.7	35.5	18.3 (11.9)	
Income inequality ^b					-6.7; < 0.01
East London	6.3	11.5	18.7	13.2 (6.6)	
England	8.1	21.1	35.1	21.5 (10.3)	
Population density (people per hectare)					11.8; < 0.01
East London	47.8	108.6	150.0	102.8 (37.7)	
England	0.6	13.1	49.9	20.9 (25.0)	
Social fragmentation index ^c					9.9; < 0.01
East London	1.9	4.0	5.6	3.9 (1.5)	
England	-3.2	-0.6	4.0	0.0 (3.1)	
Voter turnout (%) ^d					—
East London	26.4	33.4	39.1	32.9 (4.7)	
England	—	—	—	28.2 (-)	
Own-group ethnic density (%) ^e					
White British	23.9	42.3	53.6	40.4 (11.8)	—
Non-British white	5.0	7.9	15.9	10.0 (4.6)	—
Black Caribbean	2.5	8.0	15.1	8.3 (4.6)	—
Black African	2.9	10.1	17.4	9.9 (5.5)	—
Indian	1.4	2.6	17.8	6.0 (7.4)	—
Pakistani	0.5	1.2	11.7	3.5 (4.9)	—
Bangladeshi	2.5	7.1	40.1	14.1 (15.3)	—
Mixed, white & black Caribbean	0.5	1.2	1.7	1.2 (0.5)	—
Mixed, other	1.6	2.3	2.9	2.2 (0.5)	—
Other ethnicities	2.7	4.3	7.3	4.7 (2.2)	—
Own-group ethnic separation (%) ^e					
White British	14.0	21.0	30.3	21.3 (5.9)	—
Non-British white	11.1	18.2	27.1	18.4 (6.0)	—
Black Caribbean	13.8	19.4	28.7	20.2 (5.1)	—
Black African	17.1	25.3	32.7	24.9 (6.2)	—
Indian	20.8	29.6	38.1	29.5 (7.2)	—
Pakistani	21.5	43.8	64.3	43.1 (16.1)	—
Bangladeshi	23.7	34.2	44.1	33.8 (8.5)	—
Mixed, white & black Caribbean	23.7	32.0	54.5	35.1 (12.1)	—
Mixed, other	20.4	25.5	31.0	25.6 (4.3)	—
Other ethnicities	19.2	22.7	29.7	24.0 (4.5)	—

^aIMD 2004 scores are on a continuous scale where higher scores indicate greater deprivation.

^bWithin-neighborhood disparity in % of people classified as income deprived at SOA-level from IMD 2004, estimated by Gini coefficient. Summary statistics for England are approximate, based on 6,685 of 7,932 statistical ward, because scores could not be derived for statistical ward containing only a single SOA.

^cHigher scores indicate greater social fragmentation. Derived using the method of Allardyce et al. using aggregated z-scores for % unmarried, % living alone, % living at a different address in previous year, % of privately rented accommodation. Estimated from 2001 census.

^dLocal elections only held in 3,072 of 5271 wards in England in 1998. Average turnout = 28.2%.⁷³ No comparison between England & ELFEP was possible as ward-level turnout for England was unavailable.

^eEthnic density (% of own ethnic group in neighborhood) & separation scores (% segregation of own ethnic group from others) not provided for England for brevity.

of Deprivation (EID) using the Index of Multiple Deprivation, an aggregated deprivation score composed of 37 indicators obtained from routinely collected national surveys, generally estimated concomitantly with our case ascertainment period.⁴¹ Because deprivation scores were not published for wards directly but at the nested “lower

super output area” (SOA), we estimated ward-level deprivation as the mean of SOA deprivation in each statistical ward, weighted by SOA population size. Income deprivation was also obtained from the EID (% of people in each SOA classified as income deprived). From this we derived inequality in income deprivation across all SOAs in each

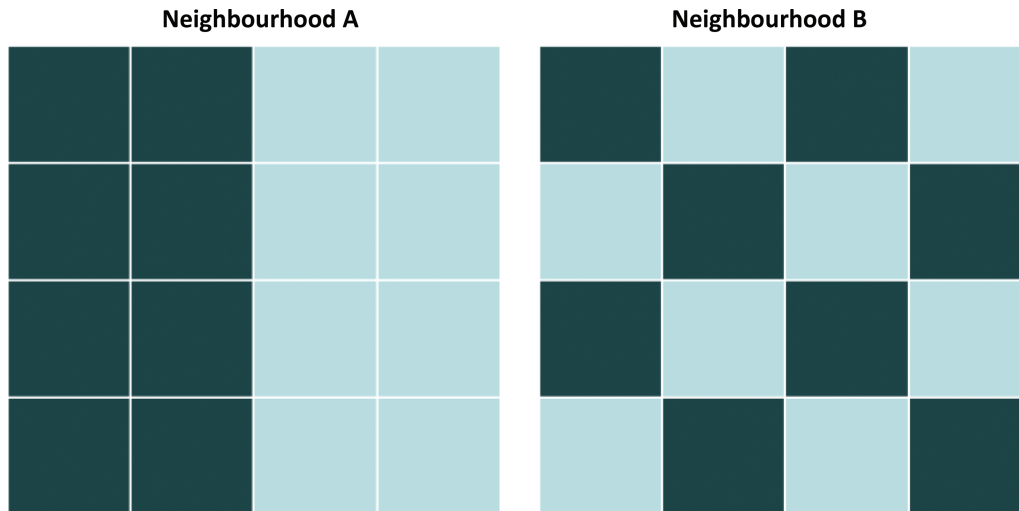


Fig. 1. Conceptualization of neighborhood-level ethnic density and ethnic separation. Neighborhoods A and B share the same level of ethnic density because the proportion of people shaded in the darker boxes is the same. However, the residential patterning—or *ethnic separation*—of this group differs in the 2 neighborhoods. In Neighborhood A there is perfect segregation of each ethnic group (ethnic separation = 1), while in Neighborhood B there is perfect integration of the 2 groups (ethnic separation = 0). For every statistical ward in the East London study region ($n = 56$), we estimated own-group ethnic density and ethnic separation for the 10 ethnic groups under study.

ward using the Gini coefficient (G).⁴² G gave the extent of dispersion in income deprivation (at SOA-level) away from a theoretical distribution where income deprivation took the same value for all SOA in each ward (ie, perfect equality). When G equalled zero, there was perfect equality and when G was 1, perfect inequality.

We used the 2001 census to estimate population density (people per hectare), own-group ethnic density, own-group ethnic separation, and social fragmentation. We defined own-group ethnic density in each statistical ward as the size of one’s own ethnic group as a proportion of total neighborhood population. Own-group ethnic separation is a related concept, but it measured the extent to which 1 ethnic group was segregated from other ethnic groups in a given neighborhood (see figure 1). We used the Index of Dissimilarity to measure ethnic separation,⁴³ where scores could vary from 0 (no segregation) to 1 (complete segregation). We used the method of Allardyce et al.¹⁵ to construct an index of social fragmentation. For each statistical ward, we estimated the proportion of unmarried persons, people living alone, people living at a different address 1 year ago, and proportion of privately rented accommodation using the 2001 census. We calculated z -scores for each variable, which we summed into an overall index. We obtained voter turnout statistics at 1998 local government elections as a proxy indicator of social cohesion, as previously described.⁴⁴ All neighborhood variables were standardized to have a mean of zero and SD of one.

Statistical Analyses

To identify neighborhood variation in psychosis risk, we first estimated standardized incidence ratios (SIR) for

each statistical ward, which gave the ratio of observed to expected subjects in each neighborhood compared with the overall ELFEP study region average, having indirectly standardized for age group, sex, ethnicity, and social class. SIRs greater than one indicate neighborhoods in the study region where risk was elevated, having standardized for individual-level factors.

Bayesian Hierarchical Modeling of Disease Risk. SIRs are problematic for the small-area study of rare diseases. First, observed counts are assumed to follow a Poisson distribution, but this is often violated in small area studies of rare disorders because sampling variability in the count of cases across neighborhoods exceeds the mean (overdispersion).⁴⁵ Second, neighborhoods closer together may have more similar SIR than neighborhoods further apart for important etiological reasons. This spatial autocorrelation must also be investigated and modeled.⁴⁶ We adopted Bayesian hierarchical modeling to simultaneously account for these issues in a multilevel framework. An overview of this approach is provided here, with further information and model specification provided in online [supplementary Appendix 1](#). As in a frequentist multilevel approach, Bayesian hierarchical models allow for the introduction of random effects to determine any variation attributable to the neighborhood level, independent of individual-level factors. However, under a Bayesian approach, all unknown parameters in the model, including random effects, are treated as stochastic, and are allowed to arise from a combination of prior beliefs—specified via prior distributions—and the data itself (see online [supplementary Appendix 1](#)).

This framework allowed us to fit several models with different prior assumptions about possible spatial dependency present in neighborhood-level random effects. This approach “smoothed” SIR in each statistical ward (Bayesian relative risks [RR]), by weighting them according to RR in neighboring areas, reducing sampling variability by borrowing information about psychosis risk from neighboring wards. We tested 4 different spatial models, each initially fitted without neighborhood covariates. Model 1 (M1) included an unstructured random effects term, which assumed no spatial autocorrelation and weighted RR in each statistical ward toward the overall study mean. Our second model (M2) included a structured variability term, which assumed RR in neighboring wards were more similar than in those further apart. The random effect was assumed to follow a conditional autoregressive normal prior distribution,⁴⁷ weighting risk estimates in each statistical ward by those in immediately adjacent wards, specified via an “adjacency matrix” to define all neighborhood connections. Model 3, known as a convolution model, included both these random effect terms.⁴⁷ Finally, we tested a mixture model (Model 4),⁴⁸ which extended model 3 to include a third “jump” random effect term to model discontinuities in risk between non-contiguous neighborhoods in the study area.⁴⁹

Model selection was based on several diagnostics, including mean absolute prediction error (MAPE), mean squared prediction error (MSPE), and the Deviance Information Criterion (DIC). MAPE and MSPE reported the average error between observed and fitted counts from each model. The DIC is a Bayesian equivalent to Akaike’s Information Criterion, which gives an estimate of model fit penalized for complexity. Lower scores indicated better model fit.⁵⁰ We inspected standardized residuals from all models to check they were distributed randomly. Final models did not exhibit evidence of non-normality (data available from authors).

For each psychotic outcome, we added all neighborhood-level covariates, except own-group ethnic density and own-group ethnic separation, to our best-fitting spatial model using a backward-fitting modeling approach to identify our most parsimonious model of psychosis risk. Because values of own-group ethnic density and separation varied by ethnic group, we could not include these in the overall (combined ethnic groups) analyses. Instead, we included these variables in separate analyses for ethnic groups with a sufficient sample size to investigate spatial effects ($n > 30$).

Implementation of Bayesian Models. Bayesian models use Monte Carlo Markov Chain (MCMC) simulation to combine the prior distribution with the data, leading to the posterior likelihood. Once all plausible values of the posterior distribution are thought to have been sampled,

the model is said to have converged. We assessed convergence using Gelman-Rubin plots. We ran the model for 10 000 iterations after convergence on 2 independent MCMC chains (20 000 total) to provide mean parameter estimates and corresponding 95% credible intervals (95% CI) for all parameters in our models, including RR in each ward and effect sizes for any neighborhood covariates. When neighborhood covariates were added to the model, RRs in each statistical ward represented any residual risk not explained by covariates. We also obtained the probability that RR in a given neighborhood exceeded unity over all MCMC iterations. Probabilities greater than 0.8 are regarded as strong evidence of excess risk.⁴⁶ Bayesian models were compared with the classical SIR approach (ISTDZE command, Stata version 11)⁵¹ and fitted in WinBUGS (version 1.4).⁵² Maps were constructed in GeoBUGS (version 1.2).⁵²

Results

Subject Sample

We identified 484 FEP subjects over more than 1.6m person-years at-risk as previously reported.^{37,38} We excluded 20 (4.1%) subjects of no fixed abode, 35 (7.2%) with insufficient postcode data to be geocoded, and 2 (0.4%) later found to be resident outside the catchment area. They did not differ on sociodemographic characteristics (data available from authors), but excluded subjects were more likely to be diagnosed with nonaffective psychosis (86% vs 73%; χ^2 -test $P = 0.04$). We included 313 (73%) subjects with nonaffective psychosis and 114 (27%) subjects with affective psychosis in the present analyses.

Neighborhood Variables

East London neighborhoods were characterized by higher levels of deprivation, population density, and social fragmentation although income inequality was lower compared with England as a whole (table 1). Own-group ethnic density and ethnic separation in East London varied according to ethnic group. Aside from the majority white British group, median neighborhood ethnic density was highest for people of black African (10.1%; 10th–90th percentile [10-90PC]: 2.9–17.4) and black Caribbean (8.0%; 10-90PC: 2.5–15.1) origin, and lowest for people of Pakistani (1.2%; 10-90PC: 0.5–11.7) and mixed white and black Caribbean ethnicities (1.2%; 10-90PC: 0.5–1.7). Pakistani (43.8%; 10-90PC: 21.5–64.3) and Bangladeshi (34.2%; 10-90PC: 23.7–44.1) groups had the highest median neighborhood levels of ethnic separation from other groups, with lowest separation (ie, greatest integration) observed for people of black Caribbean (19.4%; 10-90PC: 13.8–28.7) and non-British white ethnicities (18.2%; 10-90PC: 11.1–27.1).

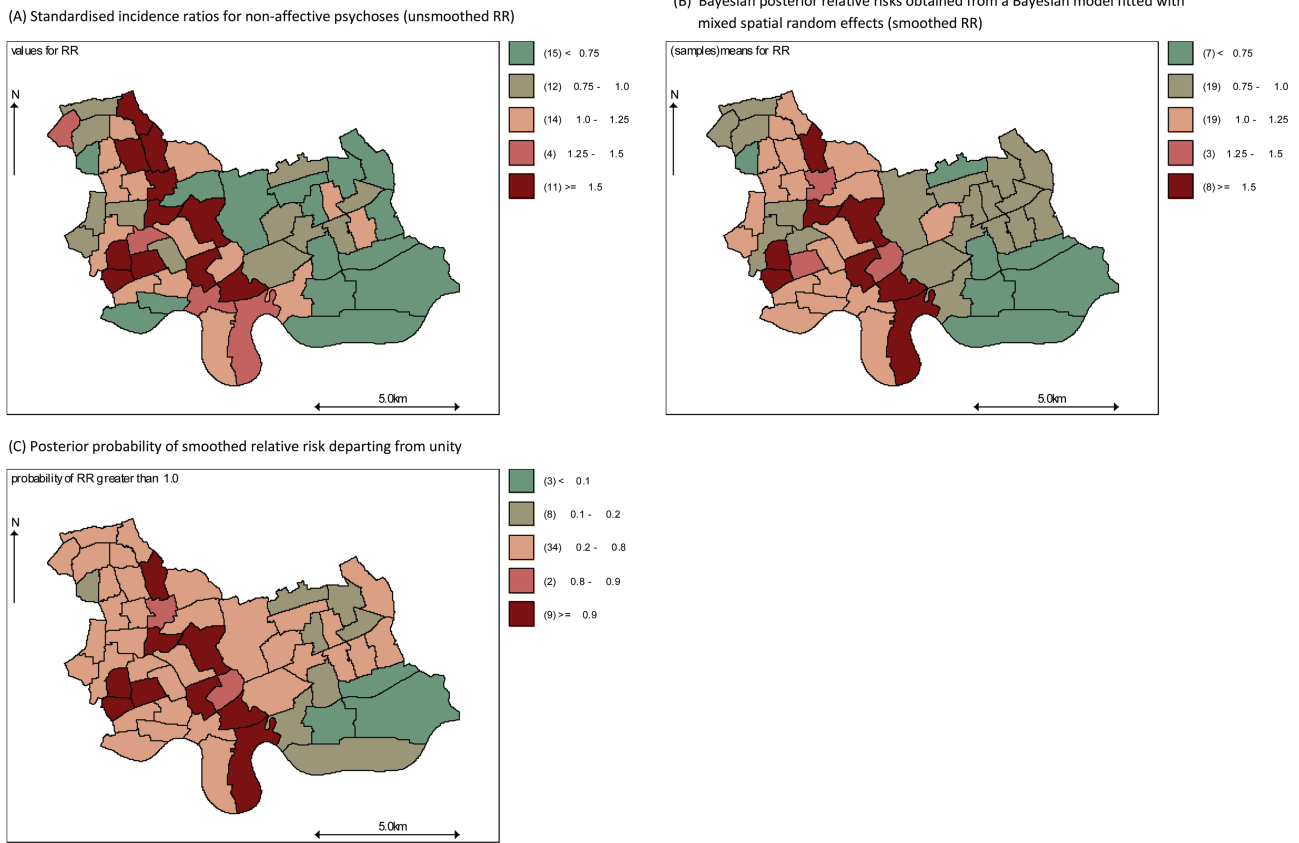


Fig. 2. Standardized incidence ratios (A), Bayesian posterior relative risks (B), and probabilities of risk departing from unity (C) from a Bayesian hierarchical mixed model for nonaffective psychotic disorders. Standardized incidence ratios for age, sex, ethnicity, and social class reveal variation in the risk of nonaffective psychoses across the study region (A). A Bayesian hierarchical model with mixed random effects (unstructured, structured, and jump) provides the best fit to the data given the prior information, resulting in smoothed RR (B). The posterior probability that RR depart from unity in the region are mapped in (C) and reveal evidence of excess rates in central parts of the study region, with high probabilities of significantly lower rates towards the southeast of the study region.

Bayesian Analyses of Nonaffective Psychotic Disorders

Spatial Patterning of Nonaffective Psychosis Risk. The count of subjects with nonaffective psychosis ranged from 0 ($N = 2$) to 14 ($N = 1$) across our neighborhoods and exhibited overdispersion (mean = 5.6, variance = 10.3). SIR varied from 0.00 to 3.24 across the study region (figure 2a). All Bayesian models reduced variance in RR between the 5th and 95th percentile (see table 2). A Bayesian-mixed model including unstructured, structured, and jump random effects (Model 4) fitted the data best according to all model fit diagnostics (see table 2). Having accounted for spatial dependency and overdispersion in the data and individual-level age, sex, ethnicity, and social class, this model indicated significant variation in RR remained at the neighborhood level (figure 2b), with high probabilities of RR greater than unity in several neighborhoods toward the centre and west of the study region (figure 2c). Neighborhoods toward the southeast of the study region had high probabilities of RR less than unity ie, lower risk of nonaffective psychosis.

Neighborhood-Level Influences on Psychosis Risk. We added neighborhood covariates to Model 4 using backward-fitting modeling to identify our most parsimonious model. This model suggested that a 1-SD increase in multiple deprivation (RR: 1.28; 95% CI: 1.08, 1.51), income deprivation inequality (RR: 1.25; 95% CI: 1.04, 1.49), and population density (RR: 1.18; 95% CI: 1.00, 1.41) was independently associated with increased incidence of nonaffective psychoses, after standardization for age, sex, ethnicity, and social class (table 3). No other neighborhood variables improved this model. Residual relative risk had been substantially reduced after modeling these neighborhood factors (see online supplemental figure 1), suggesting that these factors largely explained variance in RR for nonaffective disorders across our study region. We found no evidence of an interaction between inequality and deprivation (RR: 1.00; 95% CI: 0.87, 1.15) or of a nonlinear (quadratic) association between deprivation and psychosis (RR: 1.01; 95% CI: 0.87, 1.16).

Table 2. Spatial Variation in Unsmoothed and Smoothed Relative Risks of Psychotic Syndromes After Indirect Standardization

Outcome	Observed Subjects	Expected Subjects	Unsmoothed SIR ^a	Smoothed RR ^b			
				Model 1 (Unstructured)	Model 2 (Structured)	Model 3 (Convolution)	Model 4 (Mixed)
Nonaffective psychoses (F20-29)							
Minimum	0	2.90	0.00	0.46	0.54	0.48	0.44
5th percentile	1	3.71	0.18	0.49	0.62	0.48	0.54
Median	5.5	5.49	0.88	0.97	0.93	0.95	1.02
95th percentile	11	7.52	2.75	1.83	1.89	1.92	2.37
Maximum	14	8.26	3.24	2.02	2.04	2.05	2.41
Mean	5.59	5.59	1.03	1.03	1.02	1.03	1.11
DIC	—	—	—	279.54	273.40	273.88	262.62
MAPE	—	—	—	2.60	2.60	2.58	2.56
MSPE	—	—	—	11.39	11.30	11.18	11.03
Affective psychoses (F30-33)							
Minimum	0	1.14	0.00	0.96	0.93	0.91	0.95
5th percentile	0	1.41	0.00	0.97	0.94	0.91	0.95
Median	2	2.01	1.03	1.00	0.98	0.98	1.01
95th percentile	4	2.65	1.94	1.03	1.10	1.11	1.10
Maximum	5	2.78	2.42	1.05	1.11	1.16	1.16
Mean	2.04	2.04	0.98	1.00	0.99	0.99	1.02
DIC	—	—	—	182.83	182.24	183.24	182.79
MAPE	—	—	—	1.44	1.44	1.43	1.44
MSPE	—	—	—	3.45	3.42	3.41	3.43

Notes: DIC, Deviance Information Criterion; MAPE, Mean Absolute Prediction Error; MSPE, Mean Squared Prediction Error.

^aClassical (unsmoothed) Standardized Incidence Ratio—standardized for age, sex, ethnicity, and social class.

^bBayesian spatial modeling after standardization for age, sex, ethnicity, and social class. Type of random effects at neighborhood-level in parentheses.

Table 3. Association Between Neighborhood-Level Environmental Variables and Incidence of DSM-IV Nonaffective Psychosis

Variable (z-standardized) ^a	Control for	
	Individual-Level Factors ^b	Full Multivariate ^c
	RR (95% CI)	RR (95% CI)
IMD	1.20 (1.00, 1.43)	1.28 (1.08, 1.51)
Income inequality	1.09 (0.89, 1.28)	1.25 (1.04, 1.49)
Population density	1.15 (0.95, 1.39)	1.18 (1.00, 1.41)
SFI	1.05 (0.87, 1.26)	1.08 (0.87, 1.34)
Voter turnout	1.21 (0.98, 1.45)	1.12 (0.91, 1.35)

Notes: IMD, index of multiple deprivation; SFI, social fragmentation index.

^aRelative Risk (RR) report relative change in incidence associated with a 1 SD increase in value of neighborhood variable.

^bAfter indirect standardization for individual-level age group, sex, ethnicity, and socioeconomic status.

^cAdditional control for neighborhood-level variables included in final model: IMD, income inequality and population density. SFI & voter turnout not retained in final model given nonsignificance.

Own-Group Ethnic Density and Ethnic Separation for Specific Ethnic Groups. There was sufficient data to study ethnic density and separation effects on nonaffective psychosis in the white British ($n = 68$; 21.7%), non-British

white ($n = 38$; 12.1%), black Caribbean ($n = 55$; 17.6%), black African ($n = 49$; 15.7%), and Bangladeshi ($n = 53$; 16.9%) groups. For the black Caribbean group, there was evidence that incidence increased in neighborhoods where they were more segregated from people of other ethnic groups (RR: 1.54; 95% CI: 1.12, 2.03). A 1-SD increase in neighborhood-level own-group ethnic density among people of black African ethnicity was significantly associated with reduced risk of psychosis risk (RR: 0.70; 95% CI: 0.48, 0.99), after standardization for individual-level factors. No other statistically significant ethnic density or separation effects were observed (data available from authors).

Bayesian Analyses of Affective Psychotic Disorders

Spatial Patterning of Affective Psychosis Risk and Neighborhood-Level Influences. The mean number of subjects with affective psychosis per neighborhood was 2.04 with a variance of 1.71. SIR ranged from 0.00 to 2.42 (see online [supplemental figure 2a](#)), which was attenuated following Bayesian spatial modeling ([table 2](#)). All Bayesian models performed similarly ([table 2](#), affective psychoses). We did not observe significant variation in RR in the study region, after standardization for individual-level factors in any Bayesian model (Model 2: online [supplemental figures 2b and 2c](#)). No neighborhood

covariates improved the model or were significantly associated with the incidence of affective psychoses (data available from authors).

Discussion

Principal Findings

We identified marked spatial variation in the relative risk of nonaffective psychotic disorder in East London, not explained by individual-level factors including age, sex, ethnicity, and social class. Together with our observation that the incidence of affective psychoses showed no such spatial variation, our data replicate previous studies to have found such differences with respect to the environment.³⁻⁵ Our study extends these findings, however, by using appropriate spatial multilevel models to demonstrate that the incidence of nonaffective psychosis was independently associated with increased deprivation, income inequality, and population density. When we studied the influence of socioenvironmental factors in specific ethnic groups, we observed that ethnic separation and ethnic density were associated with nonaffective psychosis risk for people of black Caribbean and black African, respectively.

Strengths and Limitations

Our data set had sufficient power to detect variation in incidence of nonaffective psychoses at the statistical ward level, after standardizing for several individual-level variables. It is possible that our models were underpowered to detect such variation for the affective psychoses, given the smaller overall sample size and smaller counts per neighborhood for this outcome. Nevertheless, point estimates of RR for the affective psychoses were close to zero in each neighborhood after Bayesian modeling, and our findings are consistent with the previous literature.³⁻⁵ Furthermore, we were able to identify some variation in RR of nonaffective psychoses for specific ethnic groups where sample sizes were considerably smaller than for the affective psychoses overall.

We used data from a large, well-characterized FEP sample using a robust epidemiological methodology.^{37,38} Neighborhood-level variables were obtained from nationally collected data sources, reliable surveys, and official voter turnout statistics. Local election results were used to minimize contamination of wider national political factors affecting voting behavior. Derived variables, such as inequality, ethnic separation, and social fragmentation were all estimated using reliable, validated methodologies. Unlike previous studies of ethnic density in the UK that have inspected ethnic density for a single ethnic group,⁵³ or used the same composite measure for all ethnic minority groups,^{34,35} we estimated own-group ethnic density in several different ethnic groups.

We used environmental measures estimated prior to or during case ascertainment to minimize misclassification

of neighborhood exposures. Nonetheless, our results are cross-sectional limiting causal inference. Some of the association between our neighborhood factors and psychosis could have been attributable to downward drift although we believe this is unlikely to fully account for our findings, particularly because the duration of untreated psychosis appears to be unrelated to neighborhood residence at first contact.⁵⁴ Both social drift and causation may occur simultaneously, either independently of each other (ie, acting on different individuals) or in an interactive fashion (ie, acting on the same individual). Elucidation of such mechanisms requires testing in future studies.

This is the first neighborhood investigation of psychotic disorders to have fully considered spatial and multilevel effects. We used relatively uninformative prior distributions (see online [supplementary Appendix 1](#)) in our Bayesian models to allow the data to drive our findings. We included several major individual-level confounders in our analyses. While current social class may partially have reflected downward drift, any such misclassification would have only strengthened its confounding effect. It was only possible to control for up to 4 individual level variables in our analyses because census data could not be stratified more finely without leading to the possible identification of individuals. We were unable to control for individual-level substance misuse or family history of mental illness because these data were not available from denominator sources. Previous studies that included these variables suggest they do not fully confound associations between urbanicity and psychosis risk.^{7,10} We excluded 7.2% of subjects from our analyses because we could not determine their residential neighborhood at first contact. While these subjects did not differ significantly from the included sample on any individual-level characteristics, they could have biased neighborhood-level findings if they were more likely to come from certain parts of the catchment area than others. We believe this is unlikely to have substantially affected our results however, given the small number of subjects ($n = 35$; $n = 32$ for nonaffective psychosis) and given that any limited postal data available on these subjects ($n = 22$) suggested they lived in districts throughout the entire catchment area (data available from authors).

Although we adopted a spatial multilevel modeling strategy, the ecological fallacy still requires a caveat; we did not measure whether individuals who developed psychosis in certain neighborhoods were, themselves, “exposed” to the degree of deprivation, inequality, or population density observed at the neighborhood level. It is theoretically possible that some variance in incidence attributed to neighborhood-level (or individual-level) factors could have been due to other exposure levels, such as the family or school. Zammit et al.¹³ observed that neighborhood variation in nonaffective psychoses in Sweden could be mostly explained by school-level social fragmentation, raising the possibility that the exposure level at

which socioenvironmental factors shape psychosis risk is conditional upon stage of the life course. We also did not know whether residential neighborhoods constituted the relevant exposure neighborhood for risk; future studies will be required to investigate the role of and time spent in different environments on psychosis risk.

Interpretation of Findings

Our findings suggest that the spatial distribution of nonaffective psychotic disorders is not a compositional artifact of individual-level age, sex, ethnicity, or, importantly, social class. This decreases the possibility that the association with urban living close to onset is solely due to social drift. A recent publication from the Moving To Opportunities study has found moving to less deprived neighborhoods in adulthood increased subjective well-being,⁵⁵ suggesting that neighborhood environments can shape mental health over the entire life course. In our study variation in psychosis risk in East London was predominantly accounted for by 3 neighborhood factors: population density, deprivation, and income inequality. For some ethnic groups, exact psychosis risk was also related to neighborhood ethnic composition, in line with previous findings.^{3,34–36,53}

Although our study design limits causal inference, our findings are consistent with a role of environmental factors in the onset of nonaffective psychoses.¹ If true, we suggest our findings would be most parsimoniously interpreted as providing a role for the social rather than physical (ie, infections, nutrition) neighborhood environment during adulthood. Theoretically, both physical and social antecedents could be hypothesized to link psychosis risk to population density and deprivation. From a physical environment perspective, greater population density may facilitate greater transmission of infections relevant to later psychosis risk, or may present a marker for reduced exposure to sunlight and vitamin D.⁵⁶ Similarly, exposure to deprivation could involve biological antecedents in psychosis onset by restricting access to adequate nutrition at key periods of brain development over the life course.^{25,26} However, the critical period of risk for such factors is thought to operate early in life—and most likely in utero—making it harder to attribute exposures such as poor nutrition or infection to associations between adulthood environments and psychosis risk.

While it is possible that residency close to onset may reflect cumulative exposure to biological antecedents over the life course (absence of sunlight and poor nutrition, infection), we suggest any causal association between adulthood environment and psychosis risk is more likely to be due to social stressors. For example, living in more deprived neighborhoods may expose people to a range of stressful experiences, resultant from lower social and economic investment, including higher rates of crime and

antisocial behavior, poorer educational, leisure and health facilities, and more physical health problems, all of which may induce stress with further consequences for mental health.⁵⁷ Individuals who lack sufficient social or financial capital to offset exposure to these stressful events, either by drawing upon social support or by moving to less-deprived neighborhoods,⁵⁵ may face chronic exposure to such threats over the life course; several studies have observed an association between lower social support and psychosis risk.^{13,15,28–31} Interestingly, we did not observe a direct association between social fragmentation and nonaffective psychosis in the present study, having considered other neighborhood variables. However, social fragmentation was highly positively correlated with income inequality (correlation: 0.59; $P < 0.05$), suggesting that inequality could be an underlying driver of neighborhood social cohesiveness.

The association between population density and nonaffective psychosis risk observed in our study, independent of deprivation and inequality, is also open to a number of possible social interpretations. For example, it may reflect reduced opportunity to create meaningful social contacts in highly urban areas where social interaction is more transient. Alternatively, more dense environments may serve to increase paranoid ideation through exposing people susceptible to psychosis to a greater number of unknown individuals. Because humans tend to organize themselves into social hierarchies, another possibility is that more densely populated environments increase the number of levels in such hierarchies, increasing the propensity of exposure to chronic social defeat⁵⁸ in those individuals further down the hierarchy.

Our observation in regard to inequality is in line with a larger literature that has revealed several associations between inequality and many health and social outcomes, including all-cause mortality⁵⁹; cause-specific mortality,⁶⁰ including some cancers, cardiovascular, and respiratory diseases and nonmedical deaths including accidents and suicide; general self-rated health⁶¹; educational attainment and crime rates;⁶² and common mental disorders.⁶³ The association between inequality and psychotic disorders has received less attention to date, but our findings support and extend 2 ecological studies whose results were consistent with our own.^{32,33} Boydell et al.³³ found some evidence that schizophrenia incidence was greater in neighborhoods with higher income inequality, but only where absolute deprivation was already low. While we did not observe such an interaction, East London was universally deprived relative to the remainder of England, and our findings thus support the possibility that income inequality (although low in our study) may have particularly pronounced effects on psychosis risk in poor communities, where differences between those with and without access to resources may be most marked. Future studies are required to investigate the effect of inequality on psychosis risk across

broader levels of inequality. Inequality may invoke several pathways to stress, including increased risk of subordination (under a social defeat paradigm), or via marginalization of vulnerable individuals (ie, social isolation). Neighborhood ethnicity composition, which may reflect specific social inequalities for certain ethnic groups, also had distinct effects on nonaffective psychosis risk for black African and black Caribbean groups in our study.

In comparison to absolute measures of neighborhood exposure (population density and deprivation), it is more difficult to attribute relative exposures (inequality, ethnic density) to biological explanations of disease or reverse causation (social drift). First, under a biological paradigm, how malnourished one is relative to one's neighbor is irrelevant in relation to later psychosis risk; what matters here is absolute nutritional intake in governing brain development at key periods of the life course. Second, while it is plausible that individuals drift into more deprived neighborhoods during the onset of disorder, often unable to hold down employment or afford high-quality accommodation, we can think of fewer compelling reasons why such individuals would also drift into more unequal neighborhoods, unless the 2 were highly positively correlated. In our study, they were not (correlation: -0.32 , $P = 0.02$). We therefore suggest that the most plausible hypothesis to explain relationships between relative environmental exposures and psychosis risk will involve a social stress-orientated paradigm.⁵⁷

Our findings correspond to previous observations that the incidence of nonaffective psychotic disorder varies with respect to the environment, but affective psychoses do not.³⁻⁵ It is possible that the trajectories underlying each disorder differ, despite some shared genetic liability.⁶⁴ It has been suggested that additional neurodevelopmental insults following interactions with genetic factors⁶⁵ may lead to cognitive and social impairment and structural brain abnormalities,⁶⁶⁻⁷⁰ which lead some individuals toward schizophrenia rather than affective psychoses. This "second-hit" hypothesis,⁷⁰ suggests that those with early-life neurodevelopmental impairment are more likely to be propelled into adverse social environments, where exposure to negative life events and a lack of social support increase the risk of permanent, deleterious effects on functional brain processes, resulting in onset of psychotic symptoms.⁷¹ A recent finding from social neuroscience supports the possibility that the urban environment can modify brain function in adulthood in response to stress.⁷² Our findings suggest that inequality, absolute deprivation, and the experience of living in dense, urban environments in adulthood may contribute to psychosis risk. Continued efforts to integrate social neuroscience with social epidemiology should help reveal how and when environmental exposures over the life course have critical effects on brain processes that increase psychosis risk.

Supplementary Material

Supplementary material is available at <http://schizophreniabulletin.oxfordjournals.org>.

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