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Life course trajectories of neighborhood social deprivation and population density before and after first diagnosis of psychotic disorders: a nested case-control study in Sweden

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Key points

Question: Do trajectories of exposure to neighborhood social environments before and after first diagnosis of a serious mental illness (SMI) differ between cases and matched controls?

Findings: In this nested case-control study of 26,729 cases diagnosed with SMI and 26,729 birth-year-sex matched controls, we observed gradients between living in more deprived neighborhoods during upbringing and subsequent risk of SMI; in contrast, risk was ameliorated in those who experienced early life upward mobility. Following diagnosis, few cases moved into more deprived areas; cases remained disproportionately exposed to higher levels of deprivation.

Meaning: Associations between deprivation, population density and psychotic disorder are partially explained by social causation, but exacerbated after diagnosis by social immobility; social drift does not play a strong role.

Abstract

Importance: People with psychosis are more likely to be born and live in densely populated and socioeconomically deprived environments, but it is unclear whether these associations are a cause or consequence of disorder.

Objective: To investigate whether trajectories of exposure to deprivation and population density before and after diagnosis were associated with psychotic disorders or non-psychotic bipolar disorder.

Design, Setting, and Participants: Nested case-control study of all individuals born in Sweden between January 1, 1982, and December 31, 2001, diagnosed for the first time with an International Classification of Diseases, Tenth Revision [ICD-10] psychotic disorder (F20-29, F30/1.2, F32/3.3) or non-psychotic bipolar disorder (F30/3.x) between their fifteenth birthday and cohort exit (December 31, 2016). We randomly selected one sex- and birth-year-matched control per case.

Exposures: Quintiles of neighbourhood-level deprivation and population density each year from: birth to age 14, and first diagnosis until cohort exit. Group-based trajectory modelling was used to derive trajectories of each exposure in each period. Logistic regression was used to examine associations with outcomes.

Results: We included 53,458 individuals (26,729 cases and 26,729 controls), of whom 30,746 (57.5%) were female. From birth to early adolescence, we observed gradients in exposure to deprivation and population density trajectories during upbringing and psychotic disorder, with those in the most- versus-least deprived (adjusted odds ratio [aOR]: 1.17; 95%CI: 1.08-1.28) and densely populated (aOR: 1.49; 95%CI: 1.34-1.66) trajectories at greatest risk. A strong upward mobility trajectory to less deprived neighborhoods was associated with similar risk to living in the least deprived trajectory (aOR: 1.01; 95%CI: 0.91-1.12). Following diagnosis, only 2.3% of participants experienced downward social drift; people with psychotic disorder were more likely to belong to this trajectory (aOR: 1.38;

95%CI: 1.16-1.65) or remain in the most deprived trajectory (aOR: 1.36; 95%CI: 1.24-1.48) relative to controls. Patterns were similar for non-psychotic bipolar disorder and deprivation, but weaker for population density.

Conclusions and Relevance: Greater exposure to deprivation during upbringing increased risk of serious mental illness (SMI), but upward mobility mitigated this. People with SMI disproportionately remained living in more deprived areas following diagnosis, highlighting issues of social immobility. Prevention and treatment should be proportionately located in deprived areas according to need.

Introduction

Elevated rates of serious mental illnesses (SMI), primarily non-affective psychotic disorders, have been consistently observed in more socially deprived and densely populated areas.[1-11] The causal direction of this association remains unclear. *Social causation* theory posits that exposure to socioenvironmental stressors cause increased psychosis risk.[12-15] Conversely, non-causal "*selection-drift*" theories propose that downwards social mobility explains the association between social adversity and SMI.[8,16] *Social drift* theory posits that psychosis negatively impact one's ability to sustain living standards, resulting in *intragenerational* drift into more deprived areas. *Social selection* theory proposes that individuals with genetic predisposition to psychosis are selected into such environments prior to psychosis onset due to *intergenerational* transmission of genetic liability to psychosis,[16-20] which may be an upstream common cause of other functional processes related to both exposure and outcome, such as cognition.[21] Cognitive impairment is more strongly associated with psychotic disorders than other SMIs such as bipolar disorder; this may explain the specificity of association between neighborhoood social environments and non-affective psychoses.[20,22,23]

Both *selection* and *drift* occur by actively moving into more adverse neighborhoods or lower socioeconomic positions. A related third process may also exist. Here, individuals with psychosis may remain in the same neighborhood or socioeconomic position, but experience social immobility relative to their unaffected peers, who are more likely to experience upward mobility by both place and status.

These causal and non-causal explanations are not mutually exclusive. Longitudinal evidence of a dose-response relationship between urbanicity at birth and upbringing with future risk of psychotic disorders excludes intragenerational drift as the sole underlying mechanism.[24,25] Some studies,[20,22] though not all,[26,27] have reported modest levels of social drift after psychosis onset, though whether this is due to social drift or relative social immobility amongst people with SMI remains unclear. Recently, genetically-informed studies have sought to untangle social causation from

intergenerational selection. Genetic susceptibility to schizophrenia, measured by polygenic risk scores (PRS) or shared familial influences, predicts subsequent residence in more deprived areas prior to onset, irrespective of SES at birth.[28,29] However, a social causation interpretation remains possible via mediated pleiotropy,[30] and not all studies have observed that PRS for schizophrenia predicts urban birth.[31] Further, urban birth and upbringing remain associated with later psychosis, despite adjustment for genetic risk.[31-34] No study to date has investigated the specificity of longitudinal associations between neighborhood social environments and various SMI outcomes, during upbringing and after first diagnosis, which would shed further light on social causation versus selection-drift-immobility hypotheses. The present study used Swedish national population-based register data to investigate these issues. Our aims were to:

1. Identify and describe latent trajectories of neighborhood-level deprivation and population density from (a) birth until age 14, and (b) year of SMI diagnosis in cases and matched controls until the end of follow-up.
2. Explore whether people diagnosed with psychotic disorder, non-psychotic bipolar disorder and controls differed in their deprivation and population density trajectories.

We hypothesised that social causation would mean that individuals diagnosed with a psychotic disorder were more likely to have lived in more deprived and densely populated areas prior to diagnosis, compared with controls. We also hypothesised that selection-drift-immobility would mean that, following diagnosis, individuals with psychotic disorders were more likely than controls to follow a downward trajectory to more deprived and densely populated areas. Finally, we hypothesised these patterns would be weaker for non-psychotic bipolar disorders, given previous evidence.[23,35]

Methods

Study Design

Using a nested case-control design, we matched cases and controls by birth year and sex, ensuring that trajectories of neighborhood change occurred during similar ages and time periods. We first identified a cohort of individuals born in Sweden between January 1, 1982, and December 31, 2001, through Psychiatry Sweden, a register linkage of national longitudinal registries of routine data, linked via a civic registration number assigned to all Swedish residents at birth. Data on Small Area Marketing Statistics (SAMS) neighborhoods were available from 1982 onwards. We followed the cohort from birth until censorship due to an SMI diagnosis, death, emigration, or the study end date (December 31, 2016), whichever came first. All individuals who died, emigrated, or were diagnosed with SMI before age 15 were excluded.

Selection of Cases and Controls

Within the base cohort, we excluded 49,784 (2.5%) individuals missing data on neighborhood of residence, and 97 (<0.1%) individuals missing covariate data (Figure 1). We then identified all cases with a first SMI diagnosis after 15 years old (earliest, January 1, 1997) recorded in the National Patient Register. SMI diagnoses were defined using the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10), and categorised into two groups: psychotic disorders (schizophrenia [F20], nonaffective psychoses [F21-29], or affective psychotic disorders [F30.2, F31.2, F31.5, F32.3, F33.3]); and non-psychotic bipolar disorder (F30.x, F31.x, excluding F30.2, F31.2, F31.5). People who received both diagnoses were categorised in the psychotic disorder group, consistent with previous research.[36] For each case, we randomly selected one sex- and birth-year-matched control without an SMI diagnosis.

Exposures

For each year of observation, we estimated area-level socioeconomic deprivation and population density, utilising the SAMS register.[29,37-39] Sweden is divided into 9,209 SAMS for administrative purposes. The register holds annual information on area-level characteristics of each SAMS. These are classified to be maximally socioeconomically homogeneous, but their deprivation and population density levels vary.[29] Socioeconomic deprivation was derived from measures of income, social benefits, unemployment, and crime (eMethods in Supplement). These were z-standardised and summed to calculate a deprivation index (higher scores specifying greater deprivation).[21,40] Population density was calculated as people per square kilometer in each SAMS. For each year, we calculated quintiles of deprivation and population density. Individuals were linked to their SAMS area and respective quintile values for each year of observation.

Covariates

We included the following confounders: biological parental history of SMI; parental migrant status; parental disposable income quintile at birth; number of residential moves from birth until age 14; and number of residential moves from index year until end of follow-up for post-diagnosis analyses (eMethods in Supplement).

Statistical Analyses

First, we conducted group-based trajectory modelling (GBTM) to identify latent groups that followed similar trajectories of deprivation and population density exposure over time (see eMethods in Supplement for full details).[41] GBTM was conducted separately for each exposure for two different time periods: *pre-diagnosis* (from birth year until 14th year of follow-up) and *post-diagnosis* (from index diagnosis year in cases until end of follow-up). For each model, we established the optimal number of trajectory groups and their shape, considering Bayesian Information Criterion values and

other statistics.[41-43] Each individual was then classified to a group according to the maximum posterior probability assignment rule.

Second, to determine the association between trajectory group membership and each SMI outcome, we conducted logistic regressions for *pre-diagnosis* and *post-diagnosis* periods, separately. We fitted univariable models for each exposure-outcome association, bivariable analyses (mutually adjusted for population density and deprivation trajectories), and multivariable models adjusted for all covariates. Reference categories for each exposure were the least deprived and least densely populated trajectory groups. We reported odds ratios (OR) with 95% confidence intervals (95% CI).

As post-hoc analyses, we conducted re-parameterized logistic regression models with the '*upward mobility'* and '*urban-rural movement'* trajectories as the reference categories, to investigate the presence of relative social immobility for people with SMI (i.e. remaining in more deprived or urban environments relative to their unaffected peers).

Given minimal missing data (2.5%), we conducted complete-case analyses,[44] and compared the characteristics of those with and without complete data. All modelling was conducted in Stata, version 17; GBTM was estimated using the *traj* user-written Stata package.[45]

This study was approved by the Stockholm Regional Ethical Review Board (2010/1185-31/5) and the UCL Research Ethics Committee (21019/001), and consent was waived.

Results

Sample characteristics

From the complete case sample of 1,949,374 individuals (97.5% of cohort; eTable 1 and eResults in Supplement), we identified 26,729 cases with a first SMI diagnosis (psychotic disorder: 12,947, 48.4%; non-psychotic bipolar disorder: 13,782, 51.6%), and selected 26,729 birth-year-sex matched controls (Figure 1; Table 1). Cases with psychotic disorder were more likely to be male, second-

generation immigrants, born in the most deprived and densely populated quintiles, and to have moved five or more times after index diagnosis year, compared with cases with non-psychotic bipolar disorder and controls (all p<0.001; Table 1). All cases were more likely to have a parental history of SMI, and to have moved between birth and age 14, consistent with previous findings.[39] Median years of post-diagnosis follow-up was 5 (interquartile range (IQR): 2-7) in both cases and controls.

Trajectory identification

Model fit statistics (eTable 2 and eTable 3 in Supplement) indicated that we obtained trajectory models with good model fit, as described below for each exposure and time period.

Pre-diagnosis

For deprivation, a 6-group model provided optimal fit to the data. Four trajectories indicated temporally stable levels of exposure to deprivation (from low to high; Figure 2A: trajectories 1, 2, 3, 4) between birth and age 14, accounting for 80.8% of the sample. Trajectories 5 (8.0%) and 6 (11.2%) depicted groups which moved from more to less deprived areas, which we termed '*strong upward mobility'* and *'moderate upward mobility'*, respectively.

For population density, we were unable to execute the trajectory modelling for the entire follow-up period due to convergence issues. Therefore, we restricted the model from birth to age 13, and selected a 6-group model. Population density remained stable for five trajectories (from low to high; Figure 2B: 1, 2, 3, 4, 5; 92.1% of the sample). Trajectory 6 (7.9%) depicted an *'urban-rural movement'* group which moved from more urban to rural environments in childhood.

Post-diagnosis

We chose a 7-group model for deprivation trajectories up to 19 years after the index diagnosis year (eTable 2). Deprivation remained stable for five trajectories (from low to high; Figure 2C: 1, 2, 3, 4, 5; 96.1% of the sample). Two further trajectories included an *'downward drift'* group (trajectory 6; 2.3%) moving from less to more deprived areas in the first 5-6 years following diagnosis, and conversely, an *'upward mobility'* group (trajectory 7; 1.6%).

We modelled population density using a 5-group model. Population density remained largely stable following diagnosis for four trajectories (from low to high; Figure 2D: 1, 2, 3, 4; 98.0% of the sample). Trajectory 5 (2.0%) represented an *'urban-rural movement'* group which moved from the most to least densely populated areas.

Association between trajectories and SMI outcomes

Pre-diagnosis

In unadjusted and bivariable models, we observed strong gradients between living in progressively greater deprivation trajectories from birth to age 14 and odds of psychotic disorder after age 15 (Table 2), which persisted in fully-adjusted models (i.e., trajectory 3: OR: 1.18, 95% CI: 1.09-1.29; trajectory 4: OR: 1.21, 95% CI: 1.11-1.31). A similar relationship was observed for non-psychotic bipolar disorder (i.e., trajectory 2: OR: 1.15, 95% CI: 1.06-1.24; trajectory 3: OR: 1.26, 95% CI: 1.16-1.37; trajectory 4: OR: 1.24, 95% CI: 1.14-1.35). In fully-adjusted models, odds of either outcome were ameliorated in the 'strong' and 'moderately' upward mobility trajectories (Table 2), with the strongest amelioration in the *'strong upward mobility'* group for both psychotic disorders (OR: 1.01, 95% CI: 0.91-1.12) and non-psychotic bipolar disorder (OR: 1.08, 95% CI: 0.97-1.19).

A gradated relationship was also observed between population density and psychotic disorder risk (i.e., trajectory 3: OR: 1.17, 95% CI: 1.05-1.31; trajectory 4: OR: 1.21, 95% CI: 1.08-1.34; trajectory 5:

OR: 1.49, 95% CI: 1.34-1.66; Table 2), but not non-psychotic bipolar disorder. Those in the *'urban-rural movement'* trajectory had increased odds of psychotic disorder (OR: 1.29, 95% CI: 1.13-1.47) and non-psychotic bipolar disorder (OR: 1.24, 95% CI: 1.09-1.41).

Post-diagnosis

Following diagnosis, cases of both psychotic disorders (trajectory 4: OR: 1.19, 95% CI: 1.09-130; trajectory 5: OR: 1.36, 95% CI: 1.24-1.48) and non-psychotic bipolar disorder (trajectory 4: OR: 1.21, 95% CI: 1.11-1.32; trajectory 5: OR: 1.39, 95% CI: 1.28-1.51) were at greater odds of living in more deprived trajectories relative to controls (Table 3). Cases with psychotic disorder were also more likely to belong to the '*downward drift'* trajectory than controls (OR: 1.38; 95% CI: 1.16-1.65), but not cases with non-psychotic bipolar disorder. *'Upward mobility'* was not associated with either outcome.

People with psychotic disorder were more likely to live in more densely populated post-diagnosis trajectories than controls, though no dose-response pattern was evident (trajectory 2: OR: 1.21, 95% CI: 1.10-1.32; trajectory 3: OR: 1.27, 95% CI: 1.18-1.38; trajectory 4: OR: 1.15, 95% CI: 1.07-1.25; Table 3). There was no association between the *'urban-rural movement'* trajectory and psychotic disorder, or between post-diagnosis population density trajectories and non-psychotic bipolar disorder.

Post-hoc analyses

Individuals with psychotic disorder were more likely to be in more deprived trajectories following diagnosis than in the *'upward mobility'* trajectory relative to controls (trajectory 4: OR: 1.30, 95% CI: 1.06-1.60; trajectory 5: OR: 1.49, 95% CI: 1.21-1.83; eTable 5), indicative of relative social immobility amongst people with a psychotic disorder. This was not observed for non-psychotic bipolar disorder,

nor for either outcome regarding population density with '*urban-rural movement*' as the reference category (eTable 5).

Discussion

Principal findings

From birth until early adolescence, we observed strong gradients between living in more deprived and densely populated areas and future odds of psychosis, congruent with social causation. These odds were ameliorated in proportion with the degree of upward mobility experienced during upbringing. Similar findings were observed with respect to deprivation, but not population density for non-psychotic bipolar disorder.

Following diagnosis, people with psychotic disorder were more likely than controls to drift downwards into more deprived areas, though this was only experienced by 2.7% of those with psychotic disorder. Relative social immobility was a bigger driver of exposure to deprivation following diagnosis than social drift, with people with SMI disproportionately remaining in the most deprived trajectory quintile.

Meaning of the findings

Our findings are consistent with research that shows elevated incidence of psychosis in those who are born or reside in deprived and densely populated areas prior to diagnosis.[3,8,46,47] Whilst early residential mobility may increase psychosis risk through disruption to social networks,[39,48] our findings show that upward mobility reduces future SMI risk, consistent with work from Denmark where children who moved to less urban areas during upbringing had a reduced schizophrenia risk.[3] We extend that work by showing this effect appears specific to deprivation, and was evident in early childhood for both psychotic disorders and non-psychotic bipolar disorder. Further, our trajectory

modelling approach suggests that social deprivation is a modifiable risk factor for SMI: the earlier participants experienced upward mobility, the lower their subsequent SMI risk. Potential mechanisms include both a critical window of susceptibility to deprivation in childhood, or a cumulative exposure hypothesis. It is also possible that threshold effects also exist. In our analyses, exposure to deprivation during childhood only increased the odds of psychosis in the two highest quintiles of persistent exposure to deprivation, consistent with earlier research.[49,50] Alternatively, a non-causal explanation would arise if cases and controls who experienced upward mobility were systematically different on unobserved confounders, including genetic liability to SMI, to those who remained in more deprived trajectories during upbringing. Nonetheless, we controlled for several covariates, including parental history of SMI, lending credence to a causal interpretation. If causal, our results indicate that socioeconomic interventions which lift people out of more deprived environments earlier in childhood will mitigate future SMI risk. Recent research provides potential clues, including evidence that children exposed to greater deprivation have lower total brain volumes and other structural brain differences,[51] that greater deprivation is associated with biomarkers of allostatic load,[52] and that cognition partially mediates the effect of deprivation on non-affective psychosis.[21]

We also identified gradients between population density during upbringing and later risk of psychotic disorders, as previously observed.[3,8,46] This was less evident for non-psychotic bipolar disorders, consistent with previous evidence.[23] Interestingly, we observed that those moving from more urban to rural areas remained at increased SMI risk, suggesting that early exposure to factors related to population density can have lasting impacts on mental health. Marcelis et al[53] also reported stronger effects of urban birth than later residency on schizophrenia risk. These findings suggest that deprivation and population density may have different critical windows or may operate differently to impact SMI risk. Further theoretical development and empirical studies are required to disentangle these potentially causal explanations.

Our study adds to the evidence base that limited social drift occurs following SMI diagnosis.[20,22] A recent Welsh study also did not observe such a process over a ten-year period,[26] but could not exclude relative social immobility (termed "passive social drift" in their paper), which has been demonstrated to be a more predominant social process in our study and in others.[20] People with psychotic disorders, but not non-psychotic bipolar disorder were more likely to live in more urban areas after diagnosis, but no dose-response relationship was observed; any urban area may offer better access to mental healthcare services than the most rural communities in our analyses.

Strengths and limitations

Using registry data, our sample was largely representative of the Swedish-born population, with a low likelihood of selection bias given minimal missing data (2.5%). Our exposures were well validated and prospectively measured, minimising recall bias.[21,40] Registry-based diagnostic codes have good concurrent validity with SMI diagnoses.[54] Using trajectory modelling allowed us to identify distinct longitudinal patterns of neighborhood-level exposures. While all participants had complete data on pre-diagnosis trajectories until age 14, our post-diagnosis trajectories included differential lengths of follow-up data, which became sparser beyond 15 years (Figure 2). Modelling quintile data may have captured less variability than possible through continuous data.

We controlled for several potential confounders, including age-period-cohort and sex effects by design, as well as parental migrant status, number of residential moves, and parental history of SMI. The latter, a marker of shared familial liability, did not substantively confound our findings. However, direct measures of genetic liability such as PRS for schizophrenia were unavailable. These have previously been associated with residence in more urban environments,[28] and thus intergenerational selection may explain our results.[29] Nonetheless, we believe this is unlikely as associations between neighborhood deprivation/urbanicity and psychosis have remained in several studies after

controlling for different genetic risk indices.[31-33] We also did not have data on other potential confounders such as individual-level socioeconomic status, birth order or adverse childhood experiences.[55-58]

Implications for policy, practice, and future research

For policymakers, our results highlight which population groups are most likely to experience psychotic disorders and non-psychotic bipolar disorders for the first time. We also observed that people with SMI tend to disproportionately remain living in the most deprived quintiles up to 20 years following diagnosis, and for people with psychotic disorders, in the most densely populated environments also. This can inform both provisions of early intervention for psychosis services and of healthcare resources in these communities, building on existing efforts to translate psychiatric epidemiology into effective resource allocation models.[59]

For public mental health, our results should guide prevention efforts that are preferentially located in more deprived and densely populated areas and linked with socioeconomic support.
Social deprivation appears to have its strongest influence on SMI risk in childhood and early adolescence, but our results crucially suggest its impact is modifiable through upward mobility. This provides vital clues for intervention research, and suggests that ambitious trials are now warranted to investigate whether moving people out of more deprived environments can ameliorate SMI risk. To our knowledge, no trial has tested such interventions regarding SMI, though the Moving to Opportunity trial has shown evidence that moving to higher quality neighbourhoods resulted in lower psychological distress in adolescence,[60] although this may also have introduced unintended harms for some groups, including increased mental health risks for boys.[61]

Our results also have implications for etiological research. We support calls for more interdisciplinary approaches to understand and target potential environmental risk factors that link early life

exposure to deprivation and urbanicity with later SMI risk.[62] Future studies should also investigate these trajectories in more diverse samples, including immigrant communities and settings outside the Global North, where emerging evidence suggests that the greater concentration of psychosis in urban areas may not hold.[63] We also need to better understand whether trajectories of exposure immediately prior to diagnosis are influenced by drift processes before onset. Whether SMI risk associated with exposure to different trajectories applies to all individuals, or may be stronger or weaker for some groups (such as by income, migrant, or ethnic status) also requires further investigation. Finally, future studies could investigate functional and clinical outcomes within each trajectory to identify those in greatest need of support.

Social causation and relative social immobility appear to play distinct roles in the onset and subsequent exposure to more deprived and urban environments for people with SMI. Importantly, our findings suggest upward social mobility may mitigate the impact of early life deprivation.

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Declarations of Interest

The authors have no conflicts of interest to declare.

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Table 1. Sample Characteristics by Case Status.

	Control N (%) (26,729; 50.0%)	**Cases N (%) (26,729; 50.0%)**		χ^2	*df*	**P-value**
Demographic characteristics		**Psychotic disorder (12,947; 48.4%)**	**Non-psychotic bipolar disorder (13,782; 51.6%)**			
Sex						
Male	11,356 (42.5%)	7,417 (57.3%)	3,939 (28.6%)	2251.4	1	<.001
Female	15,373 (57.5%)	5,530 (42.7%)	9,843 (71.4%)			
Parental migrant status						
Swedish born	22,324 (83.5%)	9,724 (75.1%)	11,241 (81.6%)	405.0	2	<.001
Migrant	4,405 (16.5%)	3,223 (24.9%)	2,541 (18.4%)			
Other Europe	1,894 (7.1%)	1,313 (10.1%)	1,308 (9.5%)			
Asia	134 (0.5%)	68 (0.5%)	22 (0.2%)			
N. Africa & Middle East	628 (2.4%)	375 (2.9%)	115 (0.8%)			
Sub-Saharan Africa	93 (0.4%)	130 (1.0%)	12 (0.1%)			
Mixed	1,570 (5.9%)	1,279 (9.9%)	1,043 (7.6%)			
Other	8.6 (0.3%)	58 (0.5%)	41 (0.3%)			
Parental history of SMI						
None	25,827 (96.6%)	11,418 (88.2%)	11,952 (86.7%)	1559.9	2	<.001
One	787 (2.9%)	1,312 (10.1%)	1,573 (11.4%)			
Both	115 (0.4%)	217 (1.7%)	257 (1.9%)			
Parental disposable income at birth						
1 (Lowest quintile)	4,814 (18.0%)	3,137 (24.2%)	2,967 (21.5%)	286.4	8	<.001
2	5,469 (20.5%)	2,730 (21.2%)	3,013 (21.9%)			
3	5,534 (20.7%)	2,370 (18.3%)	2,818 (20.5%)			
4	5,554 (20.8%)	2,423 (18.7%)	2,477 (18.0%)			
5 (Highest quintile)	5,358 (20.1%)	2,287 (17.7%)	2,507 (18.2%)			
Deprivation at birth						
1 (Lowest quintile)	4,464 (16.7%)	1,894 (14.6%)	2,016 (14.6%)	239.2	8	<.001
2	5,362 (20.1%)	2,291 (17.7%)	2,536 (18.4%)			
3	5,708 (21.4%)	2,546 (19.7%)	2,813 (20.4%)			
4	5,581 (20.9%)	2,696 (20.8%)	2,997 (21.8%)			
5 (Highest quintile)	5,614 (21.0%)	3,520 (27.2%)	3,420 (24.8%)			
Population density at birth						
1 (Lowest quintile)	2,703 (10.1%)	1,025 (7.9%)	1,357 (9.9%)	311.6	8	<.001
2	3,719 (13.9%)	1,462 (11.3%)	1,806 (13.1%)			
3	4,940 (18.5%)	2,120 (16.4%)	2,295 (16.7%)			
4	7,114 (26.6%)	3,259 (25.2%)	3,584 (26.0%)			
5 (Highest quintile)	8,253 (30.9%)	5,081 (39.2%)	4,740 (34.4%)			
Moves (birth year to 14th year of follow-up)						
0	11,761 (44.0%)	4,435 (33.6%)	4,502 (32.7%)	842.8	4	<.001
1 to 4	14,141 (52.9%)	7,825 (60.4%)	8,387 (60.9%)			
5 or more	827 (3.1%)	777 (6.0%)	893 (6.5%)			

Table 1. Sample Characteristics by Case Status. (continued).

	Control N (%) (26,729; 50.0%)	**Cases N (%) (26,729; 50.0%)**		χ^2	*df*	**P-value**
		Psychotic disorder (12,947; 48.4%)	**Non-psychotic bipolar disorder (13,782; 51.6%)**			
Moves (diagnosis year until end of follow-up)						
0	12,889 (48.2%)	6,441 (49.8%)	6,530 (47.4%)	39.1	4	<.001
1 to 4	13,125 (49.1%)	6,067 (46.9%)	6,879 (49.9%)			
5 or more	715 (2.7%)	439 (3.4%)	373 (2.7%)			
Clinical characteristics						
Diagnosis (ICD-10)						
Schizophrenia (F20) or schizoaffective disorders (F25)	-	2,942 (22.7%)	-	NA	NA	NA
Affective psychosis (F30-33)	-	3,369 (26.0%)	-			
Bipolar psychosis (F30-31)	-	1,153 (8.9%)	-			
Depressive psychosis (F32-33)	-	2,216 (17.1%)	-			
Other non-affective psychosis (F2X)	-	6,636 (51.3%)	-			
Bipolar/Mania w/o psychosis	-	-	13,782 (100%)			

Abbreviations: *df,* degrees of freedom; NA, not applicable; SMI, severe mental illness; N. Africa, North Africa.

Note: Data are presented as n/N (%) for categorical variables, where n is the number of participants within that category and N is the total number for whom data is available for that particular characteristic.

Table 2. Pre-diagnosis Logistic Regression Models for Psychotic Disorder and Non-psychotic Bipolar Disorder.

	OR (95% CI)					
	Psychotic Disorder			Non-psychotic Bipolar Disorder		
Exposures	**Univariable Model**	**Bivariable Model[a]**	**Multivariable Model[b]**	**Univariable Model**	**Bivariable Model[a]**	**Multivariable Model[b]**
Deprivation Index						
Trajectory 1 *(least deprived)*	(ref)	(ref)	(ref)	(ref)	(ref)	(ref)
Trajectory 2	1.10 (1.02-1.19)	1.16 (1.07-1.25)	1.03 (0.95-1.12)	1.21 (1.13-1.30)	1.26 (1.17-1.36)	1.14 (1.06-1.24)
Trajectory 3	1.38 (1.27-1.49)	1.45 (1.34-1.57)	1.17 (1.06-1.26)	1.42 (1.32-1.53)	1.51 (1.40-1.63)	1.25 (1.16-1.36)
Trajectory 4 *(most deprived)*	1.62 (1.50-1.75)	1.56 (1.43-1.69)	1.17 (1.08-1.28)	1.42 (1.32-1.53)	1.50 (1.39-1.63)	1.23 (1.13-1.34)
Trajectory 5 *(strong upward mobility)*	1.25 (1.12-1.38)	1.22 (1.10-1.35)	1.01 (0.91-1.12)	1.29 (1.17-1.42)	1.26 (1.14-1.39)	1.08 (0.97-1.19)
Trajectory 6 *(moderate upward mobility)*	1.46 (1.33-1.60)	1.46 (1.33-1.60)	1.11 (1.01-1.23)	1.50 (1.38-1.64)	1.53 (1.40-1.68)	1.21 (1.10-1.33)
Population Density						
Trajectory 1 *(least densely populated)*	(ref)	(ref)	(ref)	(ref)	(ref)	(ref)
Trajectory 2	1.14 (1.02-1.28)	1.22 (1.09-1.36)	1.08 (0.96-1.21)	1.13 (1.02-1.26)	1.19 (1.07-1.32)	1.07 (0.97-1.19)
Trajectory 3	1.26 (1.14-1.40)	1.42 (1.28-1.58)	1.17 (1.05-1.31)	1.12 (1.01-1.23)	1.25 (1.13-1.38)	1.06 (0.96-1.18)
Trajectory 4	1.36 (1.23-1.50)	1.55 (140-1.71)	1.21 (1.08-1.34)	1.23 (1.12-1.35)	1.41 (1.28-1.55)	1.16 (1.05-1.28)
Trajectory 5 *(most densely populated)*	1.96 (1.77-2.16)	1.98 (1.79-2.19)	1.49 (1.34-1.66)	1.23 (1.12-1.36)	1.27 (1.15-1.41)	1.08 (0.97-1.20)
Trajectory 6 *(urban-rural movement)*	1.69 (1.49-1.91)	1.82 (1.60-2.06)	1.29 (1.13-1.47)	1.58 (1.40-1.78)	1.69 (1.49-1.90)	1.24 (1.09-1.41)
Parental migrant status	1.73 (1.63-1.84)	-	1.43 (1.33-1.52)	1.12 (1.05-1.19)	-	1.02 (0.96-1.09)
Parental history of SMI	3.94 (3.52-4.40)	-	3.44 (3.08-3.85)	4.28 (3.86-4.75)	-	3.87 (3.48-4.30)
Parental disposable income at birth						
1 (Lowest quintile)	(ref)	-	(ref)	(ref)	-	(ref)
2	0.72 (0.67-0.78)	-	0.83 (0.77-0.90)	0.95 (0.88-1.02)	-	1.05 (0.97-1.14)
3	0.64 (0.59-0.69)	-	0.80 (0.74-0.87)	0.85 (0.79-0.91)	-	0.99 (0.92-1.07)
4	0.66 (0.61-0.71)	-	0.86 (0.79-0.93)	0.73 (0.68-0.79)	-	0.90 (0.83-0.97)
5 (Highest quintile)	0.63 (0.58-0.68)	-	0.83 (0.76-0.90)	0.79 (0.73-0.85)	-	1.01 (0.93-1.10)
Moves (birth to 14th year)	1.21 (1.19-1.24)	-	1.16 (1.14-1.18)	1.21 (1.19-1.23)	-	1.16 (1.14-1.18)

Abbreviation: ORs, odds ratios; ref, reference category; SMI, severe mental illness.

[a] = Adjusting for deprivation index and population density trajectory membership. We also controlled for birth year and sex by matching cases and controls.

[b] = Adjusting as above, and for parental migrant status, parental history of SMI, parental disposable income at birth, and number of moves (birth to 14th year).

Table 3. Post-diagnosis Logistic Regression Models for Psychotic Disorder and Non-psychotic Bipolar Disorder.

	OR (95% CI)					
	Psychotic Disorder			**Non-psychotic Bipolar Disorder**		
Exposures	**Univariable Model**	**Bivariable Model[a]**	**Multivariable Model[b]**	**Univariable Model**	**Bivariable Model[a]**	**Multivariable Model[b]**
Deprivation Index						
Trajectory 1 *(least deprived)*	(ref)	(ref)	(ref)	(ref)	(ref)	(ref)
Trajectory 2	1.00 (0.91-1.09)	1.03 (0.94-1.12)	1.02 (0.93-1.12)	1.04 (0.96-1.13)	1.04 (0.96-1.14)	1.04 (0.95-1.14)
Trajectory 3	1.03 (0.94-1.12)	1.07 (0.98-1.17)	1.04 (0.95-1.14)	1.16 (1.06-1.26)	1.16 (1.07-1.26)	1.11 (1.02-1.21)
Trajectory 4	1.23 (1.13-1.34)	1.27 (1.16-1.38)	1.19 (1.09-1.30)	1.28 (1.18-1.39)	1.29 (1.19-1.40)	1.21 (1.11-1.32)
Trajectory 5 *(most deprived)*	1.55 (1.43-1.69)	1.57 (1.44-1.70)	1.36 (1.24-1.48)	1.51 (1.39-1.64)	1.52 (1.40-1.65)	1.39 (1.28-1.51)
Trajectory 6 *(downward drift)*	1.29 (1.09-1.53)	1.28 (1.08-1.51)	1.38 (1.16-1.65)	1.28 (1.03-1.57)	1.28 (1.04-1.58)	1.06 (0.85-1.33)
Trajectory 7 (*upward mobility)*	0.85 (0.80-0.91)	0.86 (0.70-1.06)	0.91 (0.74-1.13)	1.29 (1.01-1.65)	1.30 (1.01-1.67)	1.11 (0.86-1.44)
Population Density						
Trajectory 1 *(least densely populated)*	(ref)	(ref)	(ref)	(ref)	(ref)	(ref)
Trajectory 2	1.25 (1.14-1.36)	1.25 (1.14-1.36)	1.21 (1.10-1.32)	1.03 (0.95-1.11)	1.03 (0.95-1.12)	1.02 (0.93-1.11)
Trajectory 3	1.34 (1.24-1.45)	1.33 (1.23-1.44)	1.27 (1.18-1.38)	1.02 (0.95-1.09)	1.02 (0.95-1.10)	0.99 (0.91-1.07)
Trajectory 4 *(most densely populated)*	1.35 (1.26-1.45)	1.26 (1.17-1.36)	1.15 (1.07-1.25)	1.06 (0.99-1.14)	1.01 (0.94-1.08)	0.97 (0.90-1.04)
Trajectory 5 *(urban-rural movement)*	1.15 (0.94-1.40)	1.13 (0.93-1.39)	1.15 (0.93-1.42)	1.06 (0.85-1.32)	0.99 (0.79-1.24)	0.87 (0.69-1.10)
Parental migrant status	1.73 (1.63-1.84)	-	1.51 (1.41-1.61)	1.12 (1.05-1.19)	-	1.03 (0.97-1.10)
Parental history of SMI	3.94 (3.52-4.40)	-	3.47 (3.10-3.88)	4.28 (3.86-4.75)	-	3.86 (3.47-4.29)
Parental disposable income at birth						
1 (Lowest quintile)	(ref)	-	(ref)	(ref)	-	(ref)
2	0.72 (0.67-0.78)	-	0.82 (0.76-0.89)	0.95 (0.88-1.02)	-	1.05 (0.97-1.13)
3	0.64 (0.59-0.69)	-	0.79 (0.73-0.86)	0.85 (0.79-0.91)	-	0.99 (0.92-1.07)
4	0.66 (0.61-0.71)	-	0.85 (0.78-0.92)	0.73 (0.68-0.79)	-	0.90 (0.83-0.97)
5 (Highest quintile)	0.63 (0.58-0.68)	-	0.83 (0.76-0.90)	0.79 (0.73-0.85)	-	1.01 (0.93-1.10)
Moves (birth to 14th year)	1.21 (1.19-1.24)	-	1.17 (1.15-1.19)	1.21 (1.19-1.23)	-	1.17 (1.15-1.19)
Moves (diagnosis to end of follow-up)	0.97 (0.95-0.99)	-	0.95 (0.94-0.97)	1.08 (1.06-1.09)	-	1.06 (1.04-1.08)

Abbreviation: ORs, odds ratios; ref, reference category; SMI, severe mental illness.

[a] = Adjusting for deprivation index and population density trajectory membership. We also controlled for birth year and sex by matching cases and controls.

[b] = Adjusting as above, and for parental migrant status, parental history of SMI, parental disposable income at birth, number of moves (birth to 14th year), and number of moves (diagnosis to end of follow-up).