

Modelling the Effects of Material Deprivation on Social and Occupational Outcomes in Healthy and Psychosis Experiencing Individuals

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Abstract

Purpose

Psychosis is associated with changes in social participation (SP) and occupational function (OF). These outcomes are influenced by a substantial number of environmental factors. Here, we aim to model the relationship between material deprivation and SP/OF outcomes in the UK Biobank and investigate the mediating role of the social brain network (SBN) and general intelligence.

Methods

SP was defined by frequency of friend/family visits and leisure/social activities, while OF was based on employment status. Material deprivation was measured using the Townsend Deprivation Index (TDI). The mediating effects of SBN measures and general intelligence were investigated using structural equation modelling, while the moderating effects of psychosis were also investigated.

Results

TDI was negatively associated with SP (Std. β =-0.05; $p < 0.001$) and OF (Std. β =-0.066; $p < 0.001$). SBN cortical volume, subcortical volume and general intelligence significantly mediated this relationship. Psychosis moderated the relationship between mediating factors and SP and OF outcomes.

Conclusion

This suggests that region-based environmental resources play a direct and indirect role in psychosocial outcomes in older adults. In this relationship, there may be fundamental differences in the role of structural brain measures and cognition in individuals with psychosis. This highlights the potential for future economic and policy interventions aimed at improving social and occupational outcomes.

Introduction

Psychosis is a key feature of schizophrenia and is present in other psychiatric disorders including bipolar disorder [1] and major depressive disorder [2]. Experience of psychosis is associated with changes in social and occupational functioning [3,4], which leads to a decrease in quality of life for the individual and loss of economic productivity for society [1,2,5]. The ability to predict individuals who are most at risk of developing a diminished social and occupational capacity would facilitate the prioritisation of established interventional resources [6] and promote targeting of at-risk individuals earlier in their illness trajectory.

Environmental factors and social and occupational outcomes

An individual's social participation (SP) is highly dependent on personal and community circumstances [7]. The degree to which an individual may be capable and willing to participate in social activities is dependent on many factors, including access to commercial [8] and recreational [9,10] resources, degree of support from family or friends [11,12], access to independent [13] and public [13,10,12] transportation options, neighbourhood security [14,9] and neighbourhood user-friendliness [13,15]. Environmental predictors of occupational outcomes are rarely studied in healthy individuals and many studies focus on the employment of individuals with disorders or disabilities that act as a barrier to maintaining healthy occupational engagement [16]. Although studying employment characteristics in a clinical sample can be meaningful, it has shortcomings: 1) it may not be informative for nominally healthy, prodromal or subclinical populations and 2) it does not consider factors that could indicate good occupational functioning (OF) outside of paid employment including volunteering, enrolment in educational programmes or the ability to look after the home or family members. Accordingly, many factors associated with SP are related to deprivation, particularly those related to nearby resources, infrastructure and security within a community. Townsend [17] proposed the Townsend deprivation index (TDI) to index region-specific material deprivation. The TDI can be calculated from census data and assigned to individuals based on postcode. Measures of area-based deprivation play an important role in allocating government funding [18] making measures such as the TDI modifiable targets via changes in economic and public health-related policy.

Brain imaging factors and social and occupational outcomes

Another important consideration is whether the deprivation-based factors found to be associated with SP [7] or OF are related directly to the abundance of resources. It is likely the case that the deficit of resources directly prohibits individuals from taking part in social scenarios. However, the outcome of long-term exposure to deprivation on the brain, whether it is impacting social and occupational outcomes, is unclear. Johnson et al. [19] discuss the effect of deprivation on the developing brain and resultant cognitive processes. They argue that areas of the brain with defined roles in stress response, language skills and executive function including in the temporal lobe, the amygdala, hippocampus and prefrontal cortex are particularly vulnerable to deprivation stressors in the environment. If these structures and processes also determine an individual's social and occupational outcomes, indirect effects may be present. Structural variation in subcortical regions such as the amygdala [20,21] as well as cortical structures such as the orbitofrontal cortex [22] has been identified as associated with measures of social participation. Furthermore, white matter microstructure in frontotemporal tracts [23], inferior longitudinal fasciculus [21] and the corpus callosum [24] are implicated in social functioning. Several resting state networks have also been linked with social outcomes including the default mode, limbic, and frontoparietal networks [25,26]. Much like with community or environmental factors, brain MRI-derived determinants of occupational functioning are not well understood in healthy individuals. Rather, focus is often on the neuroplastic changes associated the demands or expertise associated with specific occupations [27].

However, even in social outcomes where there is a substantial body of evidence studying brain-based determinants of social outcomes, results can be inconsistent in both findings and methodologies [28] and studies are largely based on predefined region-of-interest (ROI) approaches, which may compound biases over time – especially if ROIs are not made explicitly clear before analysis [29]. Recent work by our group [30] has addressed this shortcoming by defining the multimodal structural and functional social brain network using a data-driven unbiased feature selection approach. Herein, we made use of this predefined social brain network (SBN).

Cognitive ability, the brain and social and occupational outcomes

Measures of cognitive ability, which are known to be directly derived from brain properties [31-33] also play a role in a social relationship [34]. The cognitive demands of social relationships may be a key aspect of why humans have such large [35] and metabolically costly [36] brains. Although little work has detailed the impact of cognitive ability on OF in healthy individuals, general cognitive ability, processing speed and memory are all associated with several aspects of job complexity [37], which could indicate a generally higher degree of function. There is evidence that both social cognition as well as general cognition may predict social network size in healthy individuals and those at clinical high-risk for psychosis [38]. Further, in early psychosis, general cognitive ability and social cognition are predictors of both social and occupational outcomes [3].

Here, we build on previous work by this group [30,39] aiming to gain a better understanding of social and occupational outcomes in the population. We investigate several hypotheses based on previously published work. First, that a region-based measure of material deprivation negatively affects social participation – a question we further extend to occupational function. Second, based on the relationships between deprivation and brain development and the relationship between brain signatures and social outcomes, we test the hypothesis that brain measures and cognitive ability may mediate this effect. Finally, based on the underlying social and occupational changes found in psychosis, we test the hypothesis that measures of psychosis moderate these relationships.

Methods

Participants

Participants used in this study are from the UK Biobank (UKB; <https://www.ukbiobank.ac.uk/>) cohort. We defined an overall healthy control cohort as individuals who have no diagnoses of neurodegenerative, demyelinating or neoplastic brain diseases, are not part of the psychosis cohort (defined next), and have valid social and occupational data after recoding (see section on *Social and occupational outcome phenotypes*) at baseline (instance 0). The full cohort was further refined for imaging analyses by removing individuals without at least one imaging subtype, without social or occupational data at imaging (instance 2), missing valid T2-weighted MR preprocessing data and individuals without White British ancestry (p22006). Finally, this cohort was split into a 25:75 ratio with 25% being used to

determine the social brain network as described in Casburn et al. [30] and 75% referred to hereafter as the “Test cohort” in which the models were tested.

We defined our psychosis cohort as any individual meeting one or more of the following criteria: An ICD summary diagnosis (p41270) in “F20-F29 Schizophrenia, schizotypal and delusional disorders”; “F30.2 Mania with psychotic symptoms”; “F31.2 Bipolar affective disorder, current episode manic with psychotic symptoms”; “F31.5 Bipolar affective disorder, current episode severe depression with psychotic symptoms”; “F32.3 Severe depressive episode with psychotic symptoms”; “F33.3 Recurrent depressive disorder, current episode severe with psychotic symptoms”. “Mental health conditions ever diagnosed by a professional” (p29000) responses “Schizophrenia” or “Any other type of psychosis or psychotic illness”.

Social and occupational outcome phenotypes

Initial attempts to construct social participation (SP) and occupational function (OF) latent variables were unsuccessful based on unacceptable fit indices and poor factor loadings. Thus, SP was indexed using measures present at the imaging visit (instance 2) of two social support variables: Frequency of friend/family visits (p1031) and Leisure/social activities (p6160). Frequency of friend/family visits was recoded from 0-5 based on the following key: No friends/family outside household = 0; Never or almost never = 0; Once every few months = 1; About once a month = 2; About once a week = 3; 2-4 times a week = 4; Almost daily = 5. Leisure/social activities was recoded 0-5 based on the number of social activities from “Sports club or gym”; “Pub or social club”; “Religious group”; “Adult education class”; “Other group activity” attended once a week or more often. Both scores were added together resulting in a numerical scale ranging from 0 (lowest SP) to 10 (highest SP). OF was derived from employment status (p6142) at the imaging visit (instance 2) variable where individuals were free to select multiple options. Individuals would receive +1 for each of the following selections: “In paid employment or self-employed”; “Looking after home and/or family”; “Doing unpaid or voluntary work” and “Full or part-time student”. Individuals would receive -1 for each of the following selections: “Unable to work because of sickness or disability” and “Unemployed”. Individuals selecting the “retired” option were excluded from the analysis of OF. This formed a scale ranging from -2 (lowest OF) to +4 (highest OF). Finally, not in education, employment or training (NEET) status was coded from the same employment status variable as OF (p6142) where individuals meeting any of the following criteria were non-NEET: “In paid employment or self-employed”; “Doing unpaid or voluntary work” or “Full or part-time student” while individuals not meeting any of these criteria were classified as NEET.

Brain imaging data

We used pre-processed brain imaging-derived phenotypes (IDP) from a workflow developed and run on behalf of UKB [40] from the September 2022 (v1.9) data release in this study. We used pre-processed T1-weighted structural measures indexing volume of subcortical structures (FreeSurfer subcortical

volumetric segmentation; p190); volume of cortical structures (Freesurfer Destrieux (a2009s) parcellation; p197); Tract-Based Spatial Statistics (TBSS)-derived mean fractional anisotropy (FA) values from diffusion-weighted MRI (p134), functional connectivity measures from 25-dimension partial correlations of ICA-derived resting-state functional MRI networks (p111).

Definition of social brain network (SBN)

The SBN was previously defined in Casburn et al. [30] using data-driven feature selection involving recursive feature elimination (RFE). For each set of IDPs (cortical volume, subcortical volume, diffusion-derived FA and functional connectivity (FC) measures), scaled IDP values were assessed for multicollinearity and highly correlated ($R^2 > 0.8$) sets of IDPs were represented using an arithmetic mean. RFE feature selection was performed on each brain measure-type set in a 25% subset of individuals with imaging data and using 10-fold cross-validation and 5 repeats to control for overfitting. Each feature selection process was performed 100 times and IDPs retained as predictors in $\geq 50\%$ of iterations were considered stable components of the SBN. Mean scaled values of selected IDPs within each sub modality were calculated to establish a single value from each: SBN mean cortical volume; SBN mean subcortical volume; SBN mean FA and SBN mean FC. The set of SBN IDPs generated by Casburn et al. [30] comprised 29 cortical volumes, 11 subcortical volumes, 9 diffusion FAs and 49 resting state functional MRI (rfMRI) FC measures in total.

Generating general intelligence factor

A general intelligence latent factor, “g”, was established based on the method of Cox et al. [31], in which four cognitive factors were used: fluid intelligence (FL; p20016); matrix pattern completion (MAT; p6373); symbol-digit substitution (SDS; p23324); and alphanumeric trail making (TMT; p6350). Confirmatory factor analysis (CFA) was used to estimate and predict a latent factor of general intelligence in the individuals in the test cohort. Only individuals with complete data for the 4 cognitive measures above were included in the CFA. Fit indices thresholds of confirmatory fit index > 0.9 , Tucker-Lewis index > 0.9 , standardized root mean square residual < 0.06 and root mean square error of approximation < 0.06 were considered acceptable.

Statistical analyses

Baseline differences in SP, OF and NEET status between psychosis and healthy control populations were assessed using t-test and chi-squared tests as appropriate. The relationship between TDI and social and occupational outcomes was assessed using linear regression for quantitative outcomes (SP/OF) and logistic regression for binary outcomes (NEET) including age and sex as covariates. Regression coefficients were standardised and reported as std. β . Pearson correlation coefficient and corresponding test statistic p-value were used to assess correlation. Structural equation modelling (SEM) and CFA were

performed using the lavaan [41] R package. The robust diagonally weighed least squares (WLSMV) estimator was used for SP and OF outcomes in SEM. Statistical analysis was performed using R version 4.4 [42].

Results

Descriptive statistics and cohort summary

Our test cohort comprised of 28,317 individuals, of whom, 140 had psychosis. Statistical comparisons of the social and occupational outcomes between psychosis and the general population in the test cohort are shown in Table 1 below (See: Experience of psychosis is associated with changes in social and occupational function and higher material deprivation).

		General population	Psychosis
		mean (SD)	mean (SD)
N	Overall Cohort	474,795	3,103
	Test Cohort	28,317	140
Age	Overall Cohort	56.5 (8.1)	55.4 (8.3)
	Test Cohort	64.5 (7.7)	63.9 (8.2)
% Female	Overall Cohort	54.5	52.3
	Test Cohort	51.7	52.9
Mean SP	Overall Cohort	4.23 (1.48)	4.10 (1.73)
	Test Cohort	4.48 (1.50)	4.21 (1.77)
Mean OF	Overall Cohort	0.90 (0.58)	0.15 (1.06)
	Test Cohort	0.58 (0.58)	0.51 (0.84)
% NEET	Overall Cohort	12.1	50.1
	Test Cohort	49.5	32.7
TDI	Overall Cohort	-1.34 (3.07)	0.51 (3.72)
	Test Cohort	-2.03 (2.64)	-1.13 (2.83)

< Table 1: MS_Table1.docx >

Higher material deprivation is associated with decreased social and occupational function

Linear regression with age and sex as covariates showed that increased TDI was associated with decreased SP (Std.β=-0.051, p<2.2e-16) and decreased OF (Std.β=-0.066, p=1.11e-11), but was not significantly associated with NEET status as assessed by logistic regression (β=0.014, p=0.285). The mean SP (Fig. 1A) and OF (Fig. 1B) per decile of TDI is shown below. Full regression results can be found in Supplementary Table 1.

< Fig. 1: MS_Fig1.TIFF>

Predicted general intelligence is correlated with mean brain MRI measures

Scores in the Matrix pattern completion (MAT; 0.56), Symbol-digit substitution (SDS; 0.61), Fluid intelligence/Verbal numerical reasoning (FL; 0.52) assessments had significant positive loadings on g, while alphanumeric trail making time (TMT-B; -0.55) had a negative loading. Loadings are visualised in Supplementary Fig. 1. Full CFA results can be found in Supplementary Table 2.

All measures of SBN mean volume and SBN mean FA were significantly positively correlated with predicted intelligence while accounting for the effects of age and sex (Supplementary Fig. 2). The strongest correlation was between the volumetric measures of SBN mean cortical and SBN subcortical volume (Pearson R=0.66). The SBN mean FC measure was significantly negatively correlated with all other measures, however, was not significantly correlated with general intelligence (Supplementary Fig. 2).

Volumetric measures in the social brain network partially mediate the relationship between material deprivation and social and occupational outcomes

Brain MRI factors mediate the relationship between TDI and SP/OF outcomes (Fig. 2). Only SBN mean cortical and SBN mean subcortical volume had significant relationships with both TDI and SP in the SEM model. SBN mean cortical volume was negatively associated with TDI (Fig. 2A; Std.β=-0.022; p<0.001) and positively associated with SP (Fig. 2A; Std.β=0.038; p<0.001). Similarly, SBN mean subcortical volume was negatively associated with TDI (Fig. 2C; Std.β=-0.029; p<0.001) and positively associated with SP (Fig. 2C; Std.β=0.035; p<0.001). In the mediation analyses, both SBN mean cortical volume (Fig. 2B; Std.β=0.00083; p<0.001) and SBN mean subcortical volume (Fig. 2C; Std.β=-0.001; p<0.001), but neither mean SBN FA (Fig. 2E; Std.β=0; p=NA) nor SBN mean FC (Fig 2G; Std.β=0; p=NA) had a significant mediating effect on the relationship between TDI and SP. SBN mean cortical volume (Fig. 2B; Std.β=0.08; p<0.001), SBN mean subcortical volume (Fig. 2D; Std.β=0.08; p<0.001) and SBN mean FA (Fig. 2F; Std.β=0.06; p<0.001) were all positively associated with OF, while only SBN mean cortical volume (Fig. 2B; Std.β=-0.0017; p=0.026) and SBN mean subcortical volume (Fig. 2D; Std.β=-0.0022;

p=0.0093) had a significant mediating effect between TDI and OF. Full brain MRI mediation results can be found in Supplementary Table 3.

< Fig. 2: MS_Fig2.TIFF >

General intelligence partially mediates the relationship between material deprivation and social and occupational outcomes

The predicted general intelligence factor (g) was negatively associated with TDI in both SP (Std.β=-0.061; p<2.2e-16, Fig. 3A) and OF (Std.β=-0.077; p=1.89e-13, Fig. 3B) models, while predicted g was positively associated with both SP (Std.β=0.028; p=3.63e-4) and OF (Std.β=0.12; p=1.94e-9) outcomes. General intelligence partially mediated the relationship between TDI and SP (Fig. 3A; Std.β=-0.0017; p<0.001) and between TDI and OF (Fig. 3B Std.β=-0.0093; p<0.001) indicating approximately 3% and 10% of the total relationship was mediated respectively. Full general intelligence mediation results can be found in Supplementary Table 4.

< Fig. 3: MS_Fig3.TIFF >

Experience of psychosis is associated with changes in social and occupational function and higher material deprivation

At baseline, individuals with psychosis had lower SP t(477896)=4.91, p=8.992e-07, lower OF t(309190)=57.707, p<2.2e-16; a higher proportion of NEET status χ^2 (1, 309192) = 2858.8, p<2.2e-16 and higher TDI t(477303)=-34.25, p<2.2e-16 than the general population. Lower SP t(28455)=2.117, p<0.05; lower OF t(10613)=9.644, p<2.2e-16 and higher TDI t(28431)=-3.99, p=6.67e-05 were also seen in the test cohort. However, unlike in the overall cohort, individuals experiencing psychosis were less likely to be of NEET status χ^2 (1, 10613)=44.852, p<2.1e-11 than healthy controls.

Moderating effects of psychosis experience

Models with significant mediation effects were brought forward for investigation of moderation by psychosis. Psychosis status did not have a significant moderating effect on the relationship between TDI and SBN mean cortical (F(1,27916)=0.47; p=0.494) or SBN mean subcortical (F(1,27921)=0.21; p=0.648) volume (Supplementary Fig.3).

Conversely, psychosis experience significantly moderated the relationship between SBN mean cortical volume and SP (F(1,27877)=2.03; p=0.046) and OF (F(1,10455)=21.67; p<0.001) outcomes as well as

between SBN mean subcortical volume and SP ($F(1,27876)=6.30$; $p=0.012$) and OF ($F(1,10455)=7.42$; $p=0.006$) outcomes. For SP, the moderating effect was apparent at higher SBN brain volumes (Fig. 4A and 4C), while for OF this was primarily at lower SBN brain volumes (Fig. 4B and 4D).

< Fig. 4: SCAN_MS_Fig4.TIFF >

Psychosis did not moderate the relationship between material deprivation and predicted g. However, psychosis had a moderating role in the relationship between predicted intelligence and SP ($F(1,19909)=6.20$; $p=0.013$) as well as OF ($F(1,7308)=6.85$; $p=0.009$). The effect of psychosis as a moderator was more evident at higher predicted g values for SP (Supplementary Fig. 4B) and lower predicted g values for OF (Supplementary Fig. 4C) outcomes.

Polygenic liability for schizophrenia moderates the relationship between TDI and SBN mean volume measures

As an alternative to psychosis experience available in all individuals in the test cohort, polygenic liability for schizophrenia (SCZ PGS), which explains variance in SCZ case/control status [43] was investigated as a moderating variable. Logistic regression using sex and age as covariates showed that polygenic liability for SCZ significantly predicted psychosis experience in the test cohort ($n=28457$; $\beta=0.354$; $p=3.72e-05$). Explained variance (Nagelkerke Pseudo R^2) was 1.0% in the test cohort. When decile 10 (Highest genetic liability) was compared to decile 1 (lowest genetic liability), there was a 4.9-fold higher proportion of psychosis (Supplementary Fig. 5B).

Although psychosis did not moderate the relationship between TDI and SBN mean volumetric measures, SCZ PGS in healthy controls significantly moderated the relationship between TDI and SBN mean cortical (Supplementary Fig. 6A; $F(1,27695)=6.22$; $p=0.013$) and subcortical volume (Supplementary Fig. 6B; $F(1,27694)=5.43$; $p=0.020$). In both cases, high SCZ PGS was associated with a weaker relationship between TDI and SBN brain volume measures.

Discussion

Greater material deprivation was significantly associated with decreased SP and OF outcomes. Bowling and Stafford [44] showed that better social functioning is linked to both objective and perceived neighbourhood affluence, using similar outcomes and a sample with a comparable age profile to the UKB cohort used here. In line with conventional theory, geographical areas with fewer resources have a shortfall in social capital which lowers the capacity for social interactions [45]. As well as being an important index of function and quality of life, some work has reinforced the direct importance of social disconnection in health outcomes, namely an increased risk of all-cause mortality [46] comparable in extent to known mortality factors such as smoking [47].

In our mediation analysis, we saw a small but significant role of the SBN's mean cortical and subcortical volume, but not fractional anisotropic or functional connectivity in mediating the relationship between TDI and the SP and OF outcomes. Neuroanatomically, the social brain cortical volume comprised somatomotor, visual, orbitofrontal/inferior and limbic/paralimbic cortices while the subcortical volume included amygdala, thalamus and basal ganglia as well as white matter volumes of the corpus callosum and cerebellum. Although causality cannot be determined in these relationships, a potential lifelong exposure to increased material deprivation could lead to changes in brain structure important in social and occupational functioning. Thus, the mediating effects of the SBN mean cortical and subcortical volumes suggest that pathways important in motor, sensory and limbic function may be involved in translating material deprivation stressors to changes in behaviour. There is evidence that regions important in visual and sensorimotor are particularly sensitive to material deprivation in adolescents [48]. Deprivation during childhood is associated with cortical volume and thickness changes later in life [49], specifically thickness in the precentral and lateral orbitofrontal cortical regions is related to SES [50]. Although this relationship may not be as evident in studies of adult subcortical structures [49], the hippocampus and amygdala may be sensitive to poverty-related stress exposure during childhood [51–53]. Further, there is substantive evidence that brain structure may serve as a mediator between deprivation and behavioural outcomes. Total brain volume may mediate the relationship between deprivation and cognitive impairment and behavioural disorder outcomes [49]. In the context of social disorder, Holz et al. [54] showed that the impact of poverty exposure on conduct disorder symptomology was mediated by structural changes in the orbitofrontal cortex in a 25-year longitudinal study. When looking at material deprivation and stress-induced cognitive and behavioural outcomes, Johnson et al. [19] demonstrate a mediating role of prefrontal cortex structure.

General cognitive ability (g) also significantly mediated the relationship between TDI and the SP and OF outcomes. Although this was a partial mediation, this was stronger than that found with SBN mean cortical and subcortical volume. In a systematic review, [55] find moderate-to-strong evidence for the association between neighbourhood SES and cognitive ability and moderate evidence for neighbourhood design, accessibility and demographics. In a longitudinal cohort (Lothian Birth Cohort 1936), Baranyi et al. [56] show that area deprivation in mid-late adulthood was associated with more severe cognitive decline and that deprivation during childhood was associated with decreased late-life cognitive ability, possibly mediated by education and residential mobility factors. Given the cognitive demands of social activity [35] and employment in occupational functioning [57, 58], any cognitive decline resulting from a long-term challenging environment or upbringing could impair an individual's ability to maintain psychosocial function. We found that predicted g correlated with all SBN mean structural measures, which is consistent with intelligence being associated with variation in measurable structural or neuroanatomical properties of the brain [31–33] and may contribute to the detected mediating relationship.

Experience of psychosis was associated with lower social and occupational functioning in our cohorts. This suggests individuals with psychosis, on average, are less likely to participate in objective social scenarios and less likely to be occupationally engaged including employment, volunteering, studying or

looking after the home or family. Individuals with psychosis are more likely to face social exclusion and rejection [59] and employment discrimination [60], which could lead to being involuntarily inhibited from participating. In addition, impaired general and social cognitive abilities are key in psychosis and given the established role of these factors in psychosocial functioning [3] such individuals may be at a disadvantage. Smaller numbers of social connections in psychosis associated with more severe overall- and negative-symptom outcomes [61], which could in turn lead to more limited participation, emphasising the particular importance of establishing correlates of social and occupational outcomes in this subgroup of the population.

Investigating the moderation effects of psychosis experience revealed several interesting phenomena. First, the relationship between TDI and SBN volumetric brain measures appeared to be mediated by schizophrenia genetic liability, but not psychosis experience. Unusually, in the case of this analysis, a lower genetic liability for schizophrenia was associated with a stronger negative relationship between deprivation and brain SBN mean volume measures. Structural brain IDPs are relatively heritable [62] and this may suggest an underlying gene-by-environment effect in which a predefined genetic burden may interact with environmental stressors such as material deprivation. Second, psychosis experience had the opposite effect in its relationship with SP as it did with OF: psychosis status was associated with an inverse relationship between brain measures and SP while for OF, a stronger positive relationship compared to healthy controls was detected. This may indicate that measures of social brain network volume play a more important role in determining occupational outcomes in those with psychosis compared to controls. This was also true for the relationship between general cognitive ability and SP and OF outcomes.

This study has several strengths including 1) the use of the large sample size available in the UK Biobank 2) the use of an independent test cohort within the UK Biobank for running SEM and 3) The use of multivariate statistics including several domains of factors (e.g. environmental, cognitive, brain MRI, genetic) in order to study social and occupational outcomes. There are a number of limitations including 1) The cross-section nature of the data leaves causality and the presence of bidirectional effects impossible to determine 2) The psychosis cohort is small, leading to a limited ability to detect moderating effects and limited generalisability in detected effects 3) The UKB imaging cohort are biased toward being older, healthier and more intelligent than the general population, limiting the generalisability of our results and 4) The outcomes, although objective, may oversimplify the measurement of social and occupational functioning.

In this work we highlight that material deprivation may be directly and indirectly linked to social and occupational function outcomes. Although the mediating effect of social brain network volumetric measures and cognitive ability is small in this older sample, it highlights that these factors may be involved in the translation of increased deprivation to poorer psychosocial outcomes. However, longitudinal analyses are crucial to understand the directionality and robustness of these relationships. Furthermore, our results suggest that increased SBN volume may provide a more pronounced occupational function advantage for those with psychosis compared to general population. Even small

effects of deprivation may have substantial downstream consequences once spread over a large, aging population. Material deprivation is a modifiable life factor, through targeted economic and policy shift and this work, followed by extension of findings to a younger cohort, highlights a role for economic and policy interventions aimed at improving psychosocial outcomes.

Declarations

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Statements and Declarations

The authors declare no competing interests.

Ethics statement

All UK Biobank participants provided informed consent at recruitment. The UK Biobank received ethical approval from the Northwest Multi-center Research Ethics committee. Individuals with complete data withdrawal requests by 18th August 2025 were excluded from all analyses. Research was conducted under UK Biobank application number 98153 (<https://biobank.ndph.ox.ac.uk/showcase/app.cgi?id=98153>).

Author contributions

Aodán Laighneach: Writing – original draft, Methodology, Investigation, Formal analysis, Data curation. **Mia Casburn:** Writing – review & editing, Methodology, Investigation, Formal analysis, Data curation. **Fergus Quilligan:** Writing – review & editing, Methodology, Data curation. **Evie Doherty:** Writing – review & editing, Formal analysis, Methodology, Data curation. **Emma Corley:** Writing – review & editing, Methodology. **Pilib Ó Broin:** Writing – review & editing, Methodology. **Gary Donohoe:** Writing – review & editing, Supervision, Resources, Project administration, Funding acquisition, Conceptualization. **Derek W. Morris:** Writing – review & editing, Supervision, Resources, Project administration, Funding acquisition, Conceptualization. **Dara M. Cannon:** Writing – review & editing, Supervision, Resources, Project administration, Funding acquisition, Conceptualization.

Data availability statement

Data for the UK Biobank are available at <https://www.ukbiobank.ac.uk/>. All code used in this work can be made available upon request.

References

1. Geddes JR, Miklowitz DJ (2013) Treatment of bipolar disorder. *Lancet* 381 (9878):1672-1682. doi:10.1016/S0140-6736(13)60857-0
2. Kupfer DJ, Frank E, Phillips ML (2012) Major depressive disorder: new clinical, neurobiological, and treatment perspectives. *Lancet* 379 (9820):1045-1055. doi:10.1016/S0140-6736(11)60602-8
3. Cowman M, Godfrey E, Walsh T, Frawley E, Fowler D, Alvarez-Jimenez M, O'Connor K, Wykes T, Birchwood M, Donohoe G (2024) Measures of Social and Occupational Function in Early Psychosis: A Systematic Review and Meta-analysis. *Schizophr Bull* 50 (2):266-285. doi:10.1093/schbul/sbad062
4. Organisation WH (2017) Depression and other common mental disorders: global health estimates.
5. Evensen S, Wisloff T, Lystad JU, Bull H, Ueland T, Falkum E (2016) Prevalence, Employment Rate, and Cost of Schizophrenia in a High-Income Welfare Society: A Population-Based Study Using Comprehensive Health and Welfare Registers. *Schizophr Bull* 42 (2):476-483. doi:10.1093/schbul/sbv141
6. Frawley E, Cowman M, Lepage M, Donohoe G (2023) Social and occupational recovery in early psychosis: a systematic review and meta-analysis of psychosocial interventions. *Psychol Med* 53 (5):1787-1798. doi:10.1017/S003329172100341X
7. Levasseur M, Genereux M, Bruneau JF, Vanasse A, Chabot E, Beaulac C, Bedard MM (2015) Importance of proximity to resources, social support, transportation and neighborhood security for mobility and social participation in older adults: results from a scoping study. *BMC Public Health* 15:503. doi:10.1186/s12889-015-1824-0
8. Levasseur M, Gauvin L, Richard L, Kestens Y, Daniel M, Payette H, NuAge Study G (2011) Associations between perceived proximity to neighborhood resources, disability, and social participation among community-dwelling older adults: results from the VoisiNuAge study. *Arch Phys Med Rehabil* 92 (12):1979-1986. doi:10.1016/j.apmr.2011.06.035
9. Fristedt S, Björklund A, Wretstrand A, Falkmer T (2011) Continuing mobility and occupational participation outside the home in old age is an act of negotiation. *Activities, Adaptation & Aging* 35 (4):275-297
10. Michael YL, Green MK, Farquhar SA (2006) Neighborhood design and active aging. *Health Place* 12 (4):734-740. doi:10.1016/j.healthplace.2005.08.002
11. Keysor JJ, Jette AM, Coster W, Bettger JP, Haley SM (2006) Association of environmental factors with levels of home and community participation in an adult rehabilitation cohort. *Arch Phys Med Rehabil* 87 (12):1566-1575. doi:10.1016/j.apmr.2006.08.347
12. Andonian L, MacRae A (2011) Well older adults within an urban context: Strategies to create and maintain social participation. *British Journal of Occupational Therapy* 74 (1):2-11
13. Richard L, Gauvin L, Gosselin C, Laforest S (2009) Staying connected: neighbourhood correlates of social participation among older adults living in an urban environment in Montreal, Quebec. *Health Promot Int* 24 (1):46-57. doi:10.1093/heapro/dan039

14. Clarke PJ, Ailshire JA, Nieuwenhuijsen ER, de Kleijn-de Vrankrijker MW (2011) Participation among adults with disability: the role of the urban environment. *Soc Sci Med* 72 (10):1674-1684. doi:10.1016/j.socscimed.2011.03.025
15. Chaudhury H, Mahmood A, Michael YL, Campo M, Hay K (2012) The influence of neighborhood residential density, physical and social environments on older adults' physical activity: An exploratory study in two metropolitan areas. *J Aging Stud* 26 (1):35-43. doi:10.1016/j.jaging.2011.07.001
16. Fortune N, Curryer B, Badland H, Smith-Merry J, Devine A, Stancliffe RJ, Emerson E, Llewellyn G (2022) Do Area-Level Environmental Factors Influence Employment for People with Disability? A Scoping Review. *Int J Environ Res Public Health* 19 (15). doi:10.3390/ijerph19159082
17. Townsend P (1987) Deprivation. *Journal of social policy* 16 (2):125-146
18. Lloyd CD, Norman PD, McLennan D (2023) Deprivation in England, 1971-2020. *Appl Spat Anal Policy* 16 (1):461-484. doi:10.1007/s12061-022-09486-8
19. Johnson SB, Riis JL, Noble KG (2016) State of the Art Review: Poverty and the Developing Brain. *Pediatrics* 137 (4). doi:10.1542/peds.2015-3075
20. Bickart KC, Wright CI, Dautoff RJ, Dickerson BC, Barrett LF (2011) Amygdala volume and social network size in humans. *Nat Neurosci* 14 (2):163-164. doi:10.1038/nn.2724
21. Noonan MP, Mars RB, Sallet J, Dunbar RIM, Fellows LK (2018) The structural and functional brain networks that support human social networks. *Behav Brain Res* 355:12-23. doi:10.1016/j.bbr.2018.02.019
22. Powell J, Lewis PA, Roberts N, Garcia-Finana M, Dunbar RI (2012) Orbital prefrontal cortex volume predicts social network size: an imaging study of individual differences in humans. *Proc Biol Sci* 279 (1736):2157-2162. doi:10.1098/rspb.2011.2574
23. Hampton WH, Unger A, Von Der Heide RJ, Olson IR (2016) Neural connections foster social connections: a diffusion-weighted imaging study of social networks. *Soc Cogn Affect Neurosci* 11 (5):721-727. doi:10.1093/scan/nsv153
24. Molesworth T, Sheu LK, Cohen S, Gianaros PJ, Verstynen TD (2015) Social network diversity and white matter microstructural integrity in humans. *Soc Cogn Affect Neurosci* 10 (9):1169-1176. doi:10.1093/scan/nsv001
25. Kieckhaefer C, Schilbach L, Bzdok D (2023) Social belonging: brain structure and function is linked to membership in sports teams, religious groups, and social clubs. *Cereb Cortex* 33 (8):4405-4420. doi:10.1093/cercor/bhac351
26. Pillemer S, Holtzer R, Blumen HM (2017) Functional connectivity associated with social networks in older adults: A resting-state fMRI study. *Soc Neurosci* 12 (3):242-252. doi:10.1080/17470919.2016.1176599
27. Wu H, Yan H, Yang Y, Xu M, Shi Y, Zeng W, Li J, Zhang J, Chang C, Wang N (2020) Occupational Neuroplasticity in the Human Brain: A Critical Review and Meta-Analysis of Neuroimaging Studies. *Front Hum Neurosci* 14:215. doi:10.3389/fnhum.2020.00215

28. Lin C, Keles U, Tyszka JM, Gallo M, Paul L, Adolphs R (2020) No strong evidence that social network index is associated with gray matter volume from a data-driven investigation. *Cortex* 125:307-317
29. Gentili C, Cecchetti L, Handjaras G, Lettieri G, Cristea IA (2021) The case for preregistering all region of interest (ROI) analyses in neuroimaging research. *Eur J Neurosci* 53 (2):357-361. doi:10.1111/ejn.14954
30. Casburn M, Laighneach A, Doherty E, Quilligan F, Fernandes A, Fahey S, Donohue G, Morris D, Cannon D (2025) 543. Investigating the Brain Network Involved in Social Participation Using a Large-Scale Multi-Acquisition, Data-Driven Approach. *Biological Psychiatry* 97 (9):S321-S322. doi:10.1016/j.biopsych.2025.02.782
31. Cox SR, Ritchie SJ, Fawns-Ritchie C, Tucker-Drob EM, Deary IJ (2019) Structural brain imaging correlates of general intelligence in UK Biobank. *Intelligence* 76:101376. doi:10.1016/j.intell.2019.101376
32. Pietschnig J, Penke L, Wicherts JM, Zeiler M, Voracek M (2015) Meta-analysis of associations between human brain volume and intelligence differences: How strong are they and what do they mean? *Neurosci Biobehav Rev* 57:411-432. doi:10.1016/j.neubiorev.2015.09.017
33. Gignac GE, Bates TC (2017) Brain volume and intelligence: The moderating role of intelligence measurement quality. *Intelligence* 64:18-29
34. Wascher CAF, Kulahci IG, Langley EJG, Shaw RC (2018) How does cognition shape social relationships? *Philos Trans R Soc Lond B Biol Sci* 373 (1756). doi:10.1098/rstb.2017.0293
35. Dunbar RI (2003) The social brain: mind, language, and society in evolutionary perspective. *Annual review of Anthropology* 32 (1):163-181
36. Jamadar SD, Behler A, Deery H, Breakspear M (2025) The metabolic costs of cognition. *Trends Cogn Sci* 29 (6):541-555. doi:10.1016/j.tics.2024.11.010
37. Smart EL, Gow AJ, Deary IJ (2014) Occupational complexity and lifetime cognitive abilities. *Neurology* 83 (24):2285-2291. doi:10.1212/WNL.0000000000001075
38. Linares N, Pratt DN, Damme KSF, Mittal VA (2025) Cognition as a predictor of social network size and early communication in clinically high risk for psychosis individuals. *Schizophr Res Cogn* 41:100365. doi:10.1016/j.scog.2025.100365
39. Doherty E, Laighneach A, Casburn M, Quilligan F, Donohoe G, Cannon DM, Morris DW (2025) Genome-wide association studies of social participation and occupational engagement in the UK Biobank. medRxiv:2025.2005.2015.25327674. doi:10.1101/2025.05.15.25327674
40. Alfaro-Almagro F, Jenkinson M, Bangerter NK, Andersson JLR, Griffanti L, Douaud G, Sotiropoulos SN, Jbabdi S, Hernandez-Fernandez M, Vallee E, Vidaurre D, Webster M, McCarthy P, Rorden C, Daducci A, Alexander DC, Zhang H, Dragonu I, Matthews PM, Miller KL, Smith SM (2018) Image processing and Quality Control for the first 10,000 brain imaging datasets from UK Biobank. *Neuroimage* 166:400-424. doi:10.1016/j.neuroimage.2017.10.034
41. Rosseel Y (2012) lavaan: An R package for structural equation modeling. *Journal of statistical software* 48 (1):1-36

42. Team RC (2021) R: A language and environment for statistical computing. R foundation for statistical computing, Vienna, Austria
43. Trubetskoy V, Pardinas AF, Qi T, Panagiotaropoulou G, Awasthi S, Bigdeli TB, Bryois J, Chen CY, Dennison CA, Hall LS, Lam M, Watanabe K, Frei O, Ge T, Harwood JC, Koopmans F, Magnusson S, Richards AL, Sidorenko J, Wu Y, Zeng J, Grove J, Kim M, Li Z, Voloudakis G, Zhang W, Adams M, Agartz I, Atkinson EG, Agerbo E, Al Eissa M, Albus M, Alexander M, Alizadeh BZ, Alptekin K, Als TD, Amin F, Arolt V, Arrojo M, Athanasiu L, Azevedo MH, Bacanu SA, Bass NJ, Begemann M, Belliveau RA, Bene J, Benyamin B, Bergen SE, Blasi G, Bobes J, Bonassi S, Braun A, Bressan RA, Bromet EJ, Bruggeman R, Buckley PF, Buckner RL, Bybjerg-Grauholm J, Cahn W, Cairns MJ, Calkins ME, Carr VJ, Castle D, Catts SV, Chambert KD, Chan RCK, Chaumette B, Cheng W, Cheung EFC, Chong SA, Cohen D, Consoli A, Cordeiro Q, Costas J, Curtis C, Davidson M, Davis KL, de Haan L, Degenhardt F, DeLisi LE, Demontis D, Dickerson F, Dikeos D, Dinan T, Djurovic S, Duan J, Ducci G, Dudbridge F, Eriksson JG, Fananas L, Faraone SV, Fiorentino A, Forstner A, Frank J, Freimer NB, Fromer M, Frustaci A, Gadelha A, Genovese G, Gershon ES, Giannitelli M, Giegling I, Giusti-Rodriguez P, Godard S, Goldstein JL, Gonzalez Penas J, Gonzalez-Pinto A, Gopal S, Gratten J, Green MF, Greenwood TA, Guillin O, Guloksuz S, Gur RE, Gur RC, Gutierrez B, Hahn E, Hakonarson H, Haroutunian V, Hartmann AM, Harvey C, Hayward C, Henskens FA, Herms S, Hoffmann P, Howrigan DP, Ikeda M, Iyegbe C, Joa I, Julia A, Kahler AK, Kam-Thong T, Kamatani Y, Karachanak-Yankova S, Kebir O, Keller MC, Kelly BJ, Khrunin A, Kim SW, Klovins J, Kondratiev N, Konte B, Kraft J, Kubo M, Kucinskas V, Kucinskiene ZA, Kusumawardhani A, Kuzelova-Ptackova H, Landi S, Lazzeroni LC, Lee PH, Legge SE, Lehrer DS, Lencer R, Lerer B, Li M, Lieberman J, Light GA, Limborska S, Liu CM, Lonnqvist J, Loughland CM, Lubinski J, Luykx JJ, Lynham A, Macek M, Jr., Mackinnon A, Magnusson PKE, Maher BS, Maier W, Malaspina D, Mallet J, Marder SR, Marsal S, Martin AR, Martorell L, Mattheisen M, McCarley RW, McDonald C, McGrath JJ, Medeiros H, Meier S, Melegh B, Melle I, Meshulam-Gately RI, Metspalu A, Michie PT, Milani L, Milanova V, Mitjans M, Molden E, Molina E, Molto MD, Mondelli V, Moreno C, Morley CP, Muntane G, Murphy KC, Myin-Germeys I, Nenadic I, Nestadt G, Nikitina-Zake L, Noto C, Nuechterlein KH, O'Brien NL, O'Neill FA, Oh SY, Olincy A, Ota VK, Pantelis C, Papadimitriou GN, Parellada M, Paunio T, Pellegrino R, Periyasamy S, Perkins DO, Pfuhlmann B, Pietilainen O, Pimm J, Porteous D, Powell J, Quattrone D, Quested D, Radant AD, Rampino A, Rapaport MH, Rautanen A, Reichenberg A, Roe C, Roffman JL, Roth J, Rothermundt M, Rutten BPF, Saker-Delye S, Salomaa V, Sanjuan J, Santoro ML, Savitz A, Schall U, Scott RJ, Seidman LJ, Sharp SI, Shi J, Siever LJ, Sigurdsson E, Sim K, Skarabis N, Slominsky P, So HC, Sobell JL, Soderman E, Stain HJ, Steen NE, Steixner-Kumar AA, Stogmann E, Stone WS, Straub RE, Streit F, Strengman E, Stroup TS, Subramaniam M, Sugar CA, Suvisaari J, Svrakic DM, Swerdlow NR, Szatkiewicz JP, Ta TMT, Takahashi A, Terao C, Thibaut F, Toncheva D, Tooney PA, Torretta S, Tosato S, Tura GB, Turetsky BI, Uçok A, Vaaler A, van Amelsvoort T, van Winkel R, Veijola J, Waddington J, Walter H, Waterreus A, Webb BT, Weiser M, Williams NM, Witt SH, Wormley BK, Wu JQ, Xu Z, Yolken R, Zai CC, Zhou W, Zhu F, Zimprich F, Atbasoglu EC, Ayub M, Benner C, Bertolino A, Black DW, Bray NJ, Breen G, Buccola NG, Byerley WF, Chen WJ, Cloninger CR, Crespo-Facorro B, Donohoe G, Freedman R, Galletly C, Gandal MJ, Gennarelli M, Hougaard DM, Hwu HG, Jablensky AV, McCarroll SA, Moran JL, Mors O, Mortensen

- PB, Muller-Myhsok B, Neil AL, Nordentoft M, Pato MT, Petryshen TL, Pirinen M, Pulver AE, Schulze TG, Silverman JM, Smoller JW, Stahl EA, Tsuang DW, Vilella E, Wang SH, Xu S, Indonesia Schizophrenia C, PsychEncode, Psychosis Endophenotypes International C, Syn GOC, Adolfsson R, Arango C, Baune BT, Belangero SI, Borglum AD, Braff D, Bramer E, Buxbaum JD, Campion D, Cervilla JA, Cichon S, Collier DA, Corvin A, Curtis D, Forti MD, Domenici E, Ehrenreich H, Escott-Price V, Esko T, Fanous AH, Gareeva A, Gawlik M, Gejman PV, Gill M, Glatt SJ, Golimbet V, Hong KS, Hultman CM, Hyman SE, Iwata N, Jonsson EG, Kahn RS, Kennedy JL, Khusnutdinova E, Kirov G, Knowles JA, Krebs MO, Laurent-Levinson C, Lee J, Lencz T, Levinson DF, Li QS, Liu J, Malhotra AK, Malhotra D, McIntosh A, McQuillin A, Menezes PR, Morgan VA, Morris DW, Mowry BJ, Murray RM, Nimgaonkar V, Nothen MM, Ophoff RA, Paciga SA, Palotie A, Pato CN, Qin S, Rietschel M, Riley BP, Rivera M, Rujescu D, Saka MC, Sanders AR, Schwab SG, Serretti A, Sham PC, Shi Y, St Clair D, Stefansson H, Stefansson K, Tsuang MT, van Os J, Vawter MP, Weinberger DR, Werge T, Wildenauer DB, Yu X, Yue W, Holmans PA, Pocklington AJ, Roussos P, Vassos E, Verhage M, Visscher PM, Yang J, Posthuma D, Andreassen OA, Kendler KS, Owen MJ, Wray NR, Daly MJ, Huang H, Neale BM, Sullivan PF, Ripke S, Walters JTR, O'Donovan MC, Schizophrenia Working Group of the Psychiatric Genomics C (2022) Mapping genomic loci implicates genes and synaptic biology in schizophrenia. *Nature* 604 (7906):502-508. doi:10.1038/s41586-022-04434-5
44. Bowling A, Stafford M (2007) How do objective and subjective assessments of neighbourhood influence social and physical functioning in older age? Findings from a British survey of ageing. *Soc Sci Med* 64 (12):2533-2549. doi:10.1016/j.socscimed.2007.03.009
 45. Wen M, Browning CR, Cagney KA (2003) Poverty, affluence, and income inequality: neighborhood economic structure and its implications for health. *Soc Sci Med* 57 (5):843-860. doi:10.1016/s0277-9536(02)00457-4
 46. Foster HME, Gill JMR, Mair FS, Celis-Morales CA, Jani BD, Nicholl BI, Lee D, O'Donnell CA (2023) Social connection and mortality in UK Biobank: a prospective cohort analysis. *BMC Med* 21 (1):384. doi:10.1186/s12916-023-03055-7
 47. Holt-Lunstad J, Smith TB, Layton JB (2010) Social relationships and mortality risk: a meta-analytic review. *PLoS Med* 7 (7):e1000316. doi:10.1371/journal.pmed.1000316
 48. Thomas M, Whittle S, Cropley V (2025) The influence of deprivation on cortical development and psychotic symptoms in youth. *J Child Psychol Psychiatry* 66 (8):1249-1262. doi:10.1111/jcpp.14150
 49. Mackes NK, Golm D, Sarkar S, Kumsta R, Rutter M, Fairchild G, Mehta MA, Sonuga-Barke EJS, team ERAYAF-u (2020) Early childhood deprivation is associated with alterations in adult brain structure despite subsequent environmental enrichment. *Proc Natl Acad Sci U S A* 117 (1):641-649. doi:10.1073/pnas.1911264116
 50. Tan CH, Tan JJX (2023) Low neighborhood deprivation buffers against hippocampal neurodegeneration, white matter hyperintensities, and poorer cognition. *Geroscience* 45 (3):2027-2036. doi:10.1007/s11357-023-00780-y
 51. Luby J, Belden A, Botteron K, Marrus N, Harms MP, Babb C, Nishino T, Barch D (2013) The effects of poverty on childhood brain development: the mediating effect of caregiving and stressful life events.

- JAMA Pediatr 167 (12):1135-1142. doi:10.1001/jamapediatrics.2013.3139
52. Hanson JL, Chandra A, Wolfe BL, Pollak SD (2011) Association between income and the hippocampus. PLoS One 6 (5):e18712. doi:10.1371/journal.pone.0018712
53. Noble KG, Houston SM, Kan E, Sowell ER (2012) Neural correlates of socioeconomic status in the developing human brain. Dev Sci 15 (4):516-527. doi:10.1111/j.1467-7687.2012.01147.x
54. Holz NE, Boecker R, Hohm E, Zohsel K, Buchmann AF, Blomeyer D, Jennen-Steinmetz C, Baumeister S, Hohmann S, Wolf I, Plichta MM, Esser G, Schmidt M, Meyer-Lindenberg A, Banaschewski T, Brandeis D, Laucht M (2015) The long-term impact of early life poverty on orbitofrontal cortex volume in adulthood: results from a prospective study over 25 years. Neuropsychopharmacology 40 (4):996-1004. doi:10.1038/npp.2014.277
55. Besser LM, McDonald NC, Song Y, Kukull WA, Rodriguez DA (2017) Neighborhood Environment and Cognition in Older Adults: A Systematic Review. Am J Prev Med 53 (2):241-251. doi:10.1016/j.amepre.2017.02.013
56. Baranyi G, Conte F, Deary IJ, Shortt N, Thompson CW, Cox SR, Pearce J (2023) Neighbourhood deprivation across eight decades and late-life cognitive function in the Lothian Birth Cohort 1936: a life-course study. Age Ageing 52 (4). doi:10.1093/ageing/afad056
57. Potter GG, Helms MJ, Plassman BL (2008) Associations of job demands and intelligence with cognitive performance among men in late life. Neurology 70 (19 Pt 2):1803-1808. doi:10.1212/01.wnl.0000295506.58497.7e
58. Vélez-Coto M, Rute-Pérez S, Pérez-García M, Caracuel A (2021) Unemployment and general cognitive ability: A review and meta-analysis. Journal of Economic Psychology 87:102430
59. Best MW, Bowie CR (2022) Social exclusion in psychotic disorders: An interactional processing model. Schizophr Res 244:91-100. doi:10.1016/j.schres.2022.05.016
60. Crestois N, Kempton MJ, Twumasi R (2025) A systematic review and meta-analysis of employer discrimination towards people living with psychosis. Schizophr Res 278:35-46. doi:10.1016/j.schres.2025.03.018
61. Degnan A, Berry K, Sweet D, Abel K, Crossley N, Edge D (2018) Social networks and symptomatic and functional outcomes in schizophrenia: a systematic review and meta-analysis. Soc Psychiatry Psychiatr Epidemiol 53 (9):873-888. doi:10.1007/s00127-018-1552-8
62. Elliott LT, Sharp K, Alfaro-Almagro F, Shi S, Miller KL, Douaud G, Marchini J, Smith SM (2018) Genome-wide association studies of brain imaging phenotypes in UK Biobank. Nature 562 (7726):210-216. doi:10.1038/s41586-018-0571-7

Figures

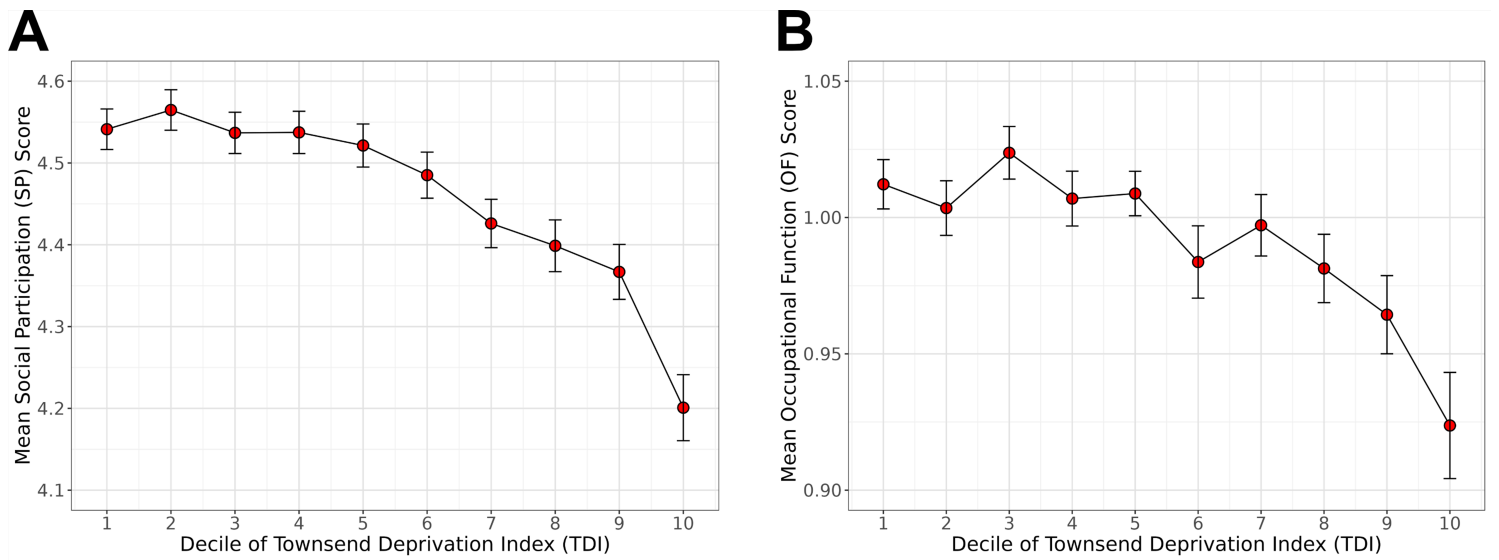


Figure 1

Material deprivation is negatively associated with social and occupational functioning. Mean SP (A) and OF (B) score per decile of TDI in test cohort. Retired individuals excluded from OF measurement. Data presented as mean score $\pm$ SEM.

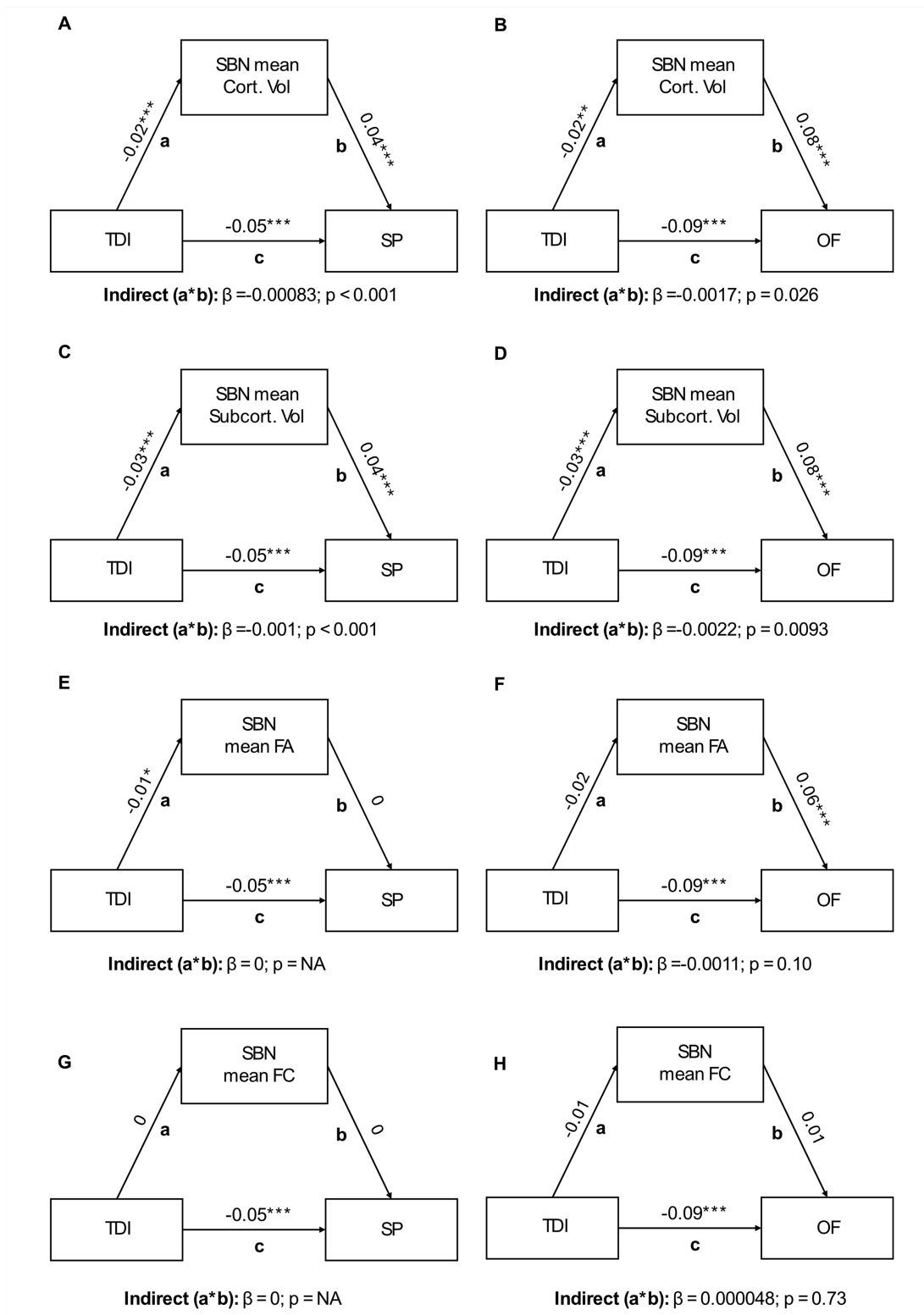


Figure 2

Mediating effects of Social Brain Network (SBN) mean RFE-selected MRI features. Standardised beta coefficients (β) and p-values reported above each path: *** = $p < 0.001$; ** = $p < 0.01$; * = $p < 0.05$. Overall mediating β and p-value reported below each path diagram (A-H). SP = Social participation; OF = Occupational function; TDI = Townsend deprivation index. SBN = Social brain network; FA = Fractional Isotropy; FC = Functional Connectivity.

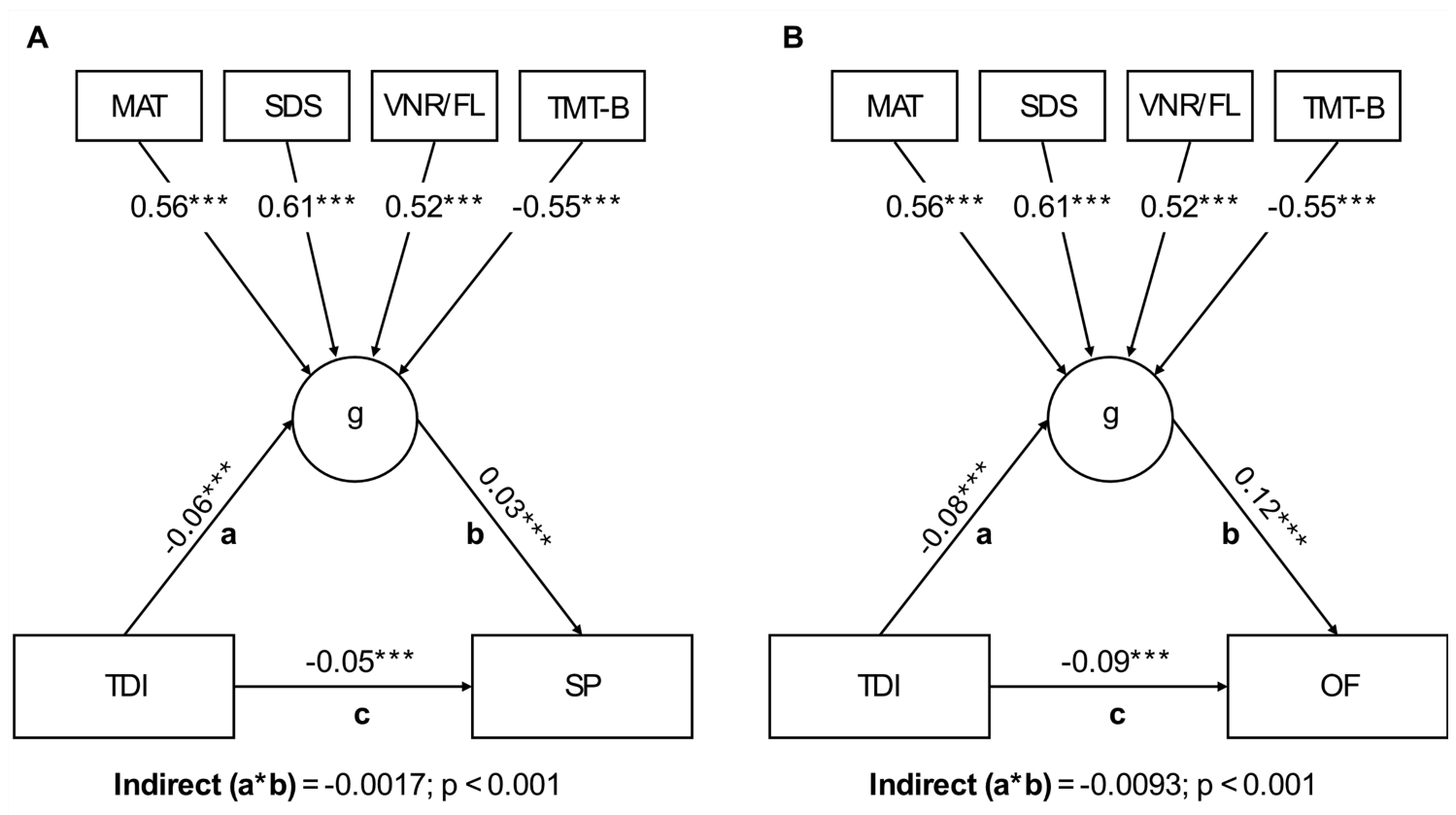


Figure 3

Mediation effects of predicted intelligence (g) on the relationship between material deprivation and social and occupational outcomes. Standardised beta coefficients (β) and p-values reported above each path: *** = $p < 0.001$; ** = $p < 0.01$; * = $p < 0.05$. TDI = Townsend Deprivation index; g = predicted general intelligence; SP = Social participation; OF = Occupational Function.

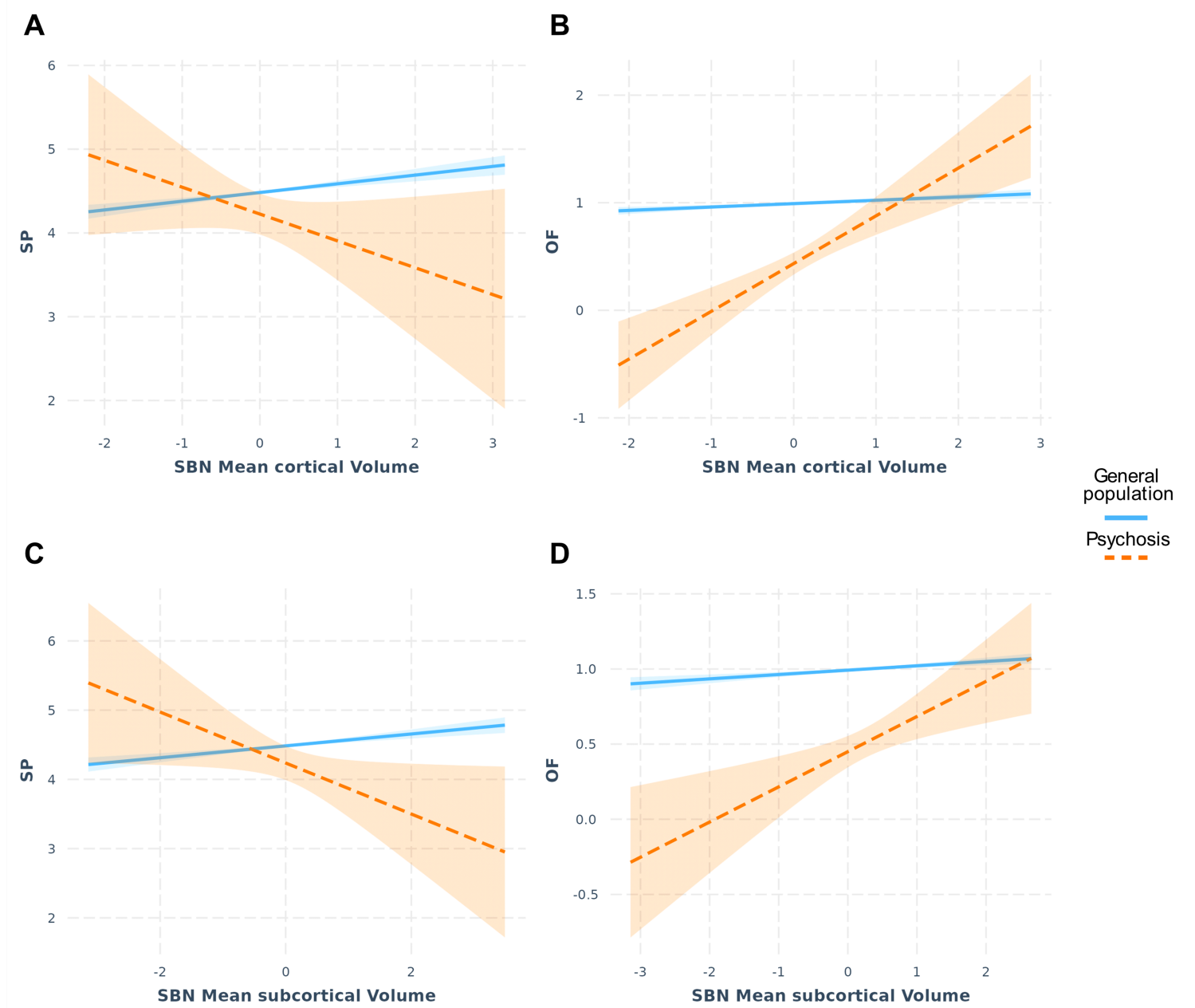


Figure 4

Moderating effects of psychosis experience between brain MRI volume measures and social/occupational outcomes. Bootstrapped (1000-fold) standardised effect size with 95% confidence interval for the conditional relationships between brain volumetric measures and SP and OF outcomes. Panels represent (A) SBN mean cortical volume vs. SP, (B) SBN mean cortical volume vs. OF, (C) SBN mean subcortical volume vs. SP, and (D) SBN mean subcortical volume vs. OF in healthy controls and individuals with psychosis.

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