Individual- and neighbourhood-level predictors of psychotic symptom dimensions in West London

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Thesis declaration form

I confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

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Overview

Part one is a systematic review of the association between income inequality and adult mental health at the subnational level. It considers two alternative hypotheses: (i) the Income Inequality Hypothesis (IIH), which predicts an association between *higher* inequality and *poorer* mental health, and (ii) the Mixed Neighbourhood Hypothesis (MNH), which predicts a reversed association, i.e. between *higher* inequality and *better* mental health.

Part two is a quantitative, empirical study into the Individual- and neighbourhood-level predictors of psychotic symptom dimensions. It involves a secondary analysis of data originally gathered from a group of participants presenting to services in West London with First Episode Psychosis (FEP).

Part three is a critical appraisal of the process of undertaking the research described in parts one and two. It includes a series of reflections on various stages of the research process, in addition to a consideration of some of the broader questions and issues it raised.

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Part 1: Literature Review

A systematic review of the association between

income inequality and adult mental health at the subnational level.

Abstract

Aims: To determine whether existing research supports an association between adult mental health and income inequality (measured at the subnational level), and further, to determine the direction of any such association. Thus, the Income Inequality Hypothesis (IIH) predicts an association between *higher* inequality and *poorer* mental health, whilst the Mixed Neighbourhood Hypothesis (MNH) predicts a reversed association, i.e. between *higher* inequality and *better* mental health.

Methods: A systematic search of PsychInfo, Medline and Web of Science databases was undertaken for all relevant studies from first publication to October 2016.

Results: Twenty-one studies meeting criteria for inclusion were identified, representing data from over 1.3 million individuals and nine countries. Whilst 47.62% of these (n=10) were found to be either partially or wholly supportive of the IIH, only 4.76% (n=1) were supportive of the MNH; 9.52% reported mixed findings (n=2) and 38.1% reported null results only (n=8). These findings did not depend on the spatial scale of analysis, e.g. neighbourhood or state, nor the quality of studies included.

Conclusions: In line with the IIH, these findings support the notion of an association between *higher* inequality and *poorer* mental health. In addition, they highlight the importance of further research in this field if an understanding of the social determinants of mental health is to be integrated into mental health policy and practice.

Introduction

Data taken from the Global Burden of Diseases, Injuries and Risk Factors Study (2010) indicate that psychological disorders are the leading cause of years lived with disability worldwide (Whiteford et al., 2013). Further, the effects of physical and psychological health are difficult to disentangle. In a meta-analysis of 91 studies undertaken in the US, it was estimated that patients accessing psychological therapy cost 20% less in physical healthcare than un-treated control participants with comparable psychological conditions (Chiles, Lambert, & Hatch, 2006). Findings of this kind have led to campaigns for greater investment in psychological therapies (Layard, 2015), with putative economic savings to be gained from a reduced burden on the National Health System (NHS) as well as reduced benefits claims, lower levels of unemployment, sickness absence and premature mortality (The Centre for Economic Performance's Mental Health Policy Group, 2012).

This approach to mental health treatment was clearly evident in 2007 with the government's unrolling of the improving access to psychological therapies (IAPT) scheme. IAPT services were designed to increase the provision of evidence-based treatments -primarily Cognitive Behavioural Therapy (CBT)- for people with common mental health conditions, e.g. anxiety and depression (Clark, 2011). Since its inception, IAPT's remit has continued to expand, with demonstration sites currently exploring its effectiveness in treating Children and Young People (CYP IAPT), as well as individuals with Severe Mental Illness (IAPT-SMI), e.g. psychosis, bipolar disorder and personality disorders, long-term health conditions and medically unexplained symptoms (NHS England, 2016).

The IAPT approach, and its focus on individual therapy, has had a number of detractors. One of the most commonly leveled criticisms is that it does not take into consideration the socioeconomic contexts in which mental illness occurs, and as

such, removes the onus on government for broader social and economic reform (Harper, 2016). For example, in a recent, controversial report that will form part of an upcoming book on the 'Origins of Happiness', Clark, Fleche, Layard, Powdthavee, and Ward (2016) claimed that psychological treatments rather than economic reform should represent the primary focus of government policy to tackle misery and promote psychological health (*"not 'wealth creation' but 'wellbeing creation'"*). In support of this position, the authors presented evidence to suggest that mental health has a greater impact on *life satisfaction* than does income inequality -i.e. disparity between the rich and the poor- and further, is cheaper to address than other factors such as poverty, unemployment or physical health; see Table 4 in Clark, Fleche, Layard, Powdthavee, and Ward (2016).

In response to the 'Origins of Happiness report, a number of professionals expressed their disagreement; see England (2016) for popular press coverage of the debate. For example, Psychologists Against Austerity (PAA) -an alliance of applied psychologists set up in 2014 to campaign on issues of social justice- published a statement arguing that the report failed to consider the full complexity of interactions between mental health and deprivation, and further, claimed that it overlooked the broader body of evidence into the social and socioeconomic predictors of mental illness (Psychologists Against Austerity, 2016). Whilst the debate has become highly politicized -as well as polarized one might argue- the putative association between mental illness and socioeconomic factors such as deprivation and inequality is an important area for research, with potential implications for the commissioning, design and delivery of psychological services.

The association between life expectancy and income

The association between income and health is well established, with poverty having been linked to a range of poor health outcomes. For example, the World Bank

published information from over 100 countries, examining the association between life expectancy and gross national product (GNP) between 1900 and 1990 (World Bank, 1993). These data showed that the strength of association between national GNP and life expectancy depended on the wealth bracket of the countries examined. The association was in fact asymptotic: whilst small increases in GNP were associated with large increases in life expectancy in comparisons across countries with relatively low GNP, the association was much weaker for countries at higher levels of GNP. Above approximately $5,000 per capita, in fact, increases in GNP made relatively little impact on life expectancy (World Bank, 1993). One interpretation of these findings is that amongst poorer countries income is crucial to health outcomes since poverty limits access to scarce resources such as food and clean water, i.e. poverty is associated with *material* deprivation. In contrast, in countries above a certain threshold of wealth, these factors become less important as basic amenities become more widely available.

Looking at data *within* a country, rather than *between* countries, income re-emerges as an important predictor of health outcomes. For example, in a study of the US between 1969 and 1989, it was found that an individual's likelihood of dying within a five-year period was negatively associated with their annual, total household income, i.e. the incidence of mortality was higher for lower income households (McDonough, Duncan, Williams, & House, 1997). One possible explanation of this pattern, is that whilst income is an index of access to basic amenities in comparisons *across* countries, *within* a country, income becomes an indicator of social position or socioeconomic status. This is important, because a large body of research has shown that socioeconomic status (SES) is inversely related to unhealthy behaviours. For example, smoking, physical inactivity and poor nutrition become increasingly common the lower an individual's SES; see Pampel, Krueger, and Denney (2010) for a review. Further, these social gradients in health-related

behaviours appear to operate across the entire spectrum of SES. Even amongst relatively affluent members of a society, small differences in SES are associated with disparities in health behaviours (Marmot, 2015). Thus, unhealthy behaviours may, at least in part, mediate some of the effects of income deprivation on poor health outcomes, with poorer individuals exhibiting more unhealthy behaviours.

Thus, there is an association between lower income and poorer health. In studies undertaken *between* countries, however, this effect may be lost above some threshold of GNP, i.e. once access to essential resources and basic amenities is no longer limited.

The association between life expectancy and income inequality

According to the Income Inequality Hypothesis (IIH) (Kragten & Rözer, 2017), another critical factor in health outcomes is *relative* income, i.e. a person's income in comparison to others' in a reference group. This is related to the level of inequality in a society, such that within a hypothetical society with zero income inequality, there would be no relative income differences. The role of income inequality on health outcomes may be particularly relevant at higher levels of affluence where material deprivation is no longer a limiting factor / determinant of health.

Popular interest in the IIH exploded in 2009, with the publication of a controversial book called 'The Spirit Level' (Wilkinson & Pickett, 2009). The authors presented a wealth of data -the majority of which had been presented previously in peer-reviewed journals- from richer countries of the world, plotting measures of income inequality against an aggregate index of health and social problems, as well as related individual indices such as obesity (Pickett, Kelly, Brunner, Lobstein, & Wilkinson, 2005), life expectancy (Wilkinson, 1990), incarceration, homicide rates, education and levels of childhood conflict (Wilkinson & Pickett, 2009, 2007). All of

these data showed significant correlations with inequality, i.e. countries characterised by *higher* levels of income inequality were consistently associated with *poorer* health, as well as *higher* levels of mortality and social problems. The authors also described a similar pattern at the subnational level, when comparing data across US states. Once again, *higher* levels of income inequality were associated with *higher* levels of health and social problems.

A number of criticisms have been raised against Wilkinson and colleagues' analyses, including accusations that the authors cherry-picked data from countries that supported their thesis, as well as the observation that potential confounders - such as absolute deprivation- were often not built into their analyses; see Saunders (2010) for example. This led one author to publish a book-length critique of the 'The Spirit Level', entitled the 'The Spirit Level Delusion: Fact-Checking the Left's New Theory of Everything' (Snowdon, 2010); see Wilkinson and Pickett (2017) for a response to some of these criticisms however. Although a detailed exploration of this debate and the underlying research lies beyond the remit of this thesis, it is worth noting that several attempts have been made to synthesize the relevant data by other groups. For example, reviewing the existing literature, the Joseph Rowntree Foundation, an independent development and research charity, concluded that an association *did* in fact exist between higher inequality and poorer health and social outcomes, although the effect was small (Rowlingson, 2011). The same conclusion was also supported by a number of systematic reviews (Macinko, Shi, Starfield, & Wulu, 2003; Subramanian & Kawachi, 2004; Torre & Myrskylä, 2014; Wilkinson & Pickett, 2006) and one meta-analysis (Kondo et al., 2009), which employed strict inclusion criteria including appropriate controls for *absolute* levels of income.

Thus, although somewhat controversial, there is a growing body of evidence in support of the IIH, which points to the existence of a small but significant association between income inequality and poor health outcomes. Further, this association seems to hold for multiple geographical scales, e.g. at the cross-national level as well as at the subnational level.

The association between mental health and income

Compared to available information on *general* health outcomes across countries, there is a relative dearth of validated data on *mental* health. For example, in a review of nearly 77,000 population-based epidemiological studies of mental disorders from around the world, fewer than 1% included or facilitated calculation of basic indicators such as incidence, prevalence, remission and mortality (Baxter, Patton, Scott, Degenhardt, & Whiteford, 2013). Four of the 21 world regions explored therein lacked *any* data on *mental* health. Further, mental health and psychiatric illness are more complex constructs to define and operationalize than indices such as life expectancy, and different cultures may conceptualise, express and/or respond to distress in different ways; see Agbayani-Siewert, Takeuchi, and Pangan (1999) for discussion.

Nonetheless, there is some evidence to suggest that mental health may show a similar pattern to physical health in its association with income. For example, in the 'Spirit Level', the authors presented data from 60 countries gathered between 1995 and 2001 by the European Values Study Group (2002). This showed the association between national income per person and the percentage of individuals who described themselves as 'quite happy' or 'very happy' (as opposed to 'not very happy' or 'not at all happy') on a four-point Lickert scale (Wilkinson & Pickett, 2009). Although not a measure of mental illness *per se*, these data showed a similar asymptotic relationship to that described for GNP against life expectancy (World

Bank, 1993). Happiness increased steeply for poorer countries, but much less steeply for richer countries, suggesting that beyond a certain threshold of wealth, GNP comes to play a decreasing role as a determinant of mental health outcomes, just as it does for physical health.

The association between income and mental health *within* a country, rather than *between* countries, is better characterised, although most of the data in this field is drawn from richer countries only. For example, the WHO reported mental health prevalence rates from seven countries of the world (Canada, USA, Brazil, Mexico, Germany, Netherlands and Turkey), based on general population probability samples (World Health Organisation, 2000). These data showed that the prevalence of anxiety, mood and substance-use disorders (defined using standardised diagnostic criteria) were positively correlated with indices of socioeconomic deprivation such as low income, low educational attainment and unemployment. Similar patterns of findings have also been reported for other mental health conditions, e.g. the psychotic disorders, in studies using a range of different diagnostic tools, indices of socioeconomic deprivation and experimental methodologies; see Muntaner, Eaton, Miech, and O'Campo (2004) and Murali and Oyebode (2004) for discussion.

Taken together, these data suggest that income may show a similar association with mental health as it does for physical health, with higher levels of deprivation being associated with poorer outcomes, an effect that may not be seen, however, in cross-national comparisons of *higher* income countries.

The association between mental health and income inequality

In their book, 'The Spirit Level', Wilkinson and Pickett (2009) also presented data on the association between mental health and income *inequality*. Drawing together

information from the World Health Organisation (WHO) as well as three national surveys, they analysed data from 12 of the richer countries (Belgium, France, Germany, Italy, Japan, the Netherlands, New Zealand, Spain and the USA) and demonstrated a strong association between income inequality and the 12-month incidence of mental illness (Pickett & Wilkinson, 2010; Wilkinson & Pickett, 2009, 2007a).

Once again, these analyses have been hotly contested (Snowdon, 2010). One of the primary criticisms raised is that the authors did not include mental health data from key countries that contradict their thesis. For example, Wilkinson and colleagues presented no data for the Scandinavian countries, which despite relatively *low* levels of inequality have prevalence rates of mental health difficulties that are broadly comparable to the US, which in contrast has *high* levels of inequality (Kringlen, Torgersen, & Cramer, 2001; Munk-Jørgensen et al., 2006). The authors justify this exclusion on the basis that the WHO has not, to date, produced directly comparable data for these countries (Wilkinson & Pickett, 2017).

The pattern of association between income inequality and mental health at the subnational level, i.e. when mental health outcomes are compared across geographical regions smaller than the country, is even less clear. For example, when Wilkinson and Pickett (2009) searched for a relationship between income inequality and mental illness across US states they found a significant association for females, but not male segments of the population. Other studies undertaken at different spatial scales -e.g. at the level of the neighbourhood, city or state- and in different countries have also raised contradictory findings. Thus, whilst some studies report *no* association between income inequality and mental health, others have even reported an *inverted* pattern of association, i.e. between *higher* income

inequality and *better* health, e.g. Marshall, Jivraj, Nazroo, Tampubolon, and Vanhoutte (2014).

In trying to make sense of apparent inconsistencies within the literature, Pickett and Wilkinson (2010) have argued that the effects of social inequality are unlikely to be found at scales smaller than the national -or certainly state- level, since, social comparisons mediate the effects of deprivation on health, and operate across a broader reference frame than the state (Drukker, Feron, & van Os, 2004). For example, the authors suggest that individuals make social comparisons by judging their own income relative to their perception of the nationwide average, rather than comparisons made more locally. The authors do not provide evidence for this assertion, but suggest that television -and presumably the broader media and social media- may play a role in this process. Nonetheless, this hypothesis lends itself to a clear prediction: that the association between income inequality and poor health outcomes *within* a country should fall as the scale of the geographical comparison area gets smaller, or else disappear below a certain threshold of geographical scale.

Thus, whilst there is some evidence to suggest that income inequality is associated with mental health outcomes when comparisons are made *between* countries (a claim that has itself been contested), it is even less clear over what spatial scales these effects operate at the subnational level, if at all.

Possible mechanisms of interaction between income inequality and mental health

Three main theories have been proposed to account for the putative association between (higher) income inequality and (poorer) health at the sub-national level (Layte, 2012). Whilst these are often discussed with respect to physical health, they are equally relevant in thinking about the association with mental health.

The *Social Capital Hypothesis* posits that the association between income inequality and health is mediated by social capital. Social capital is a poorly operationalised construct, which nonetheless attempts to capture the extent of interaction, trust and integration that exists between members of a community or society, i.e. something of value ('capital') that emerges from social connections and interactions; see McKenzie, Whitely, and Weich, (2002) for a discussion. According to the Social Capital Hypothesis, when individuals or groups of individuals differ greatly in their incomes, i.e. conditions of high inequality, they are less likely to trust one another, or to interact and form cohesive social networks, with resulting effects on mental health (Layte, 2012). There may be many reasons for this putative effect on mental health; for example, a society characterized by higher levels of trust may be less stressful (Takahashi et al., 2005) and engender greater levels of reciprocity and practical support (Coleman, 1988). In support of the SCH, a systematic review of 21 relevant studies found moderate evidence for an association between lower social capital and higher levels of common mental health difficulties (De Silva, McKenzie, Harpham, & Huttly, 2005). Further, when Kragten and Rözer (2017) drew upon the World Values Survey (WVS) and European Values Study (EVS) to analyse data from 80 countries, they found that social trust mediated the effects of income inequality on self-rated health.

The *Status Anxiety Hypothesis* holds that income inequality leads to greater social comparison between the rich and poor. This process of social comparison is thought to be *inherently* stressful and detrimental to health (Pham-Kanter, 2009). It has been hypothesized that this stress is a result of the perceived -and potentially real- lack of control over life and work that is associated with low social status (Marmot, 2004). Relatedly, there is a wealth of psychological literature on 'relative deprivation' as a cognitive construct, i.e. *perceived* social status and its effects on the individual.

Research in the field typically explores the sense of inferiority engendered in those of a lower perceived status, and its connection to putatively related constructs such as resentment, frustration and trust; see Smith, Pettigrew, Pippin, and Bialosiewicz (2012) for a review.

The *Neomaterialist Hypothesis*, in contrast, takes a more sociological perspective. It posits that when levels of inequality are high, less investment is made into public infrastructure and welfare services, e.g. gyms, parks, hospitals and schools, which in turn, leads to poorer health outcomes; see Smith (1996) for discussion. Thus, when disparities are high, there is less incentive for the rich to invest in social resources that are likely to redistribute the wealth. Consistent with this theory there is evidence that US states characterized by higher inequality spend proportionally less on education and public health, and have poorer educational outcomes and higher levels of adult mortality (Dunn, Burgess, & Ross, 2005; Kaplan, Pamuk, Lynch, Cohen, & Balfour, 1996; Ronzio, Pamuk, & Squires, 2004).

The Mixed Neighbourhood Hypothesis

A number of studies have reported an *inverted* pattern of association between income inequality and health, i.e. a correlation between *higher* income inequality and *better* health, e.g. Marshall et al. (2014). These findings are instead consistent with an alternative hypothesis: the Mixed Neighbourhood Hypothesis (MNH); see Manley, VanHam, and Doherty (2012), Musterd and Andersson (2005) and Ostendorf, Musterd, and De Vos (2001) for discussion. According to the MNH, income inequality may actually be *good* for health. Thus, whilst neighbourhoods of *homogeneous* poverty, i.e. areas of high deprivation but *low* inequality, may become mired by a lack of social opportunities and cultures of crime, substance use and joblessness, the theory proposes that these effects can be partially ameliorated by integration and mixing with individuals of a higher socioeconomic status, i.e. areas

of high deprivation but *high* inequality also. On a purely pragmatic level, poorer members of the community may benefit from the increased investment in local infrastructure and resources that such heterogeneity brings. In some areas, particularly within the US, this has led to the adoption of mixed-income housing development schemes, e.g. the HOPE VI project (Popkin et al., 2004). However, this is a highly controversial approach, which some have argued is founded on insufficient evidence at present (Cheshire, 2012; Monk, Clarke, & Tang, 2011). See Levy, McDade, and Dumlaom (2010) for a review of the literature on the impact of mixed-income communities on poor families for example.

Previous reviews

To the author's knowledge, no single review has specifically explored the association between income inequality and *mental* health. In reviewing the literature on the association between major mental disorders and socioeconomic position, one article (over 10 years old) identified five studies that focused on income *inequality* rather than *absolute* income as a sub-set of its analysis (Muntaner et al., 2004). These involved studies undertaken at the level of US metropolitan areas (n=2), US states (n=2) and UK regions (n=1). In their conclusions, the authors reported inconsistent findings, with two out of the fives studies showing no association between income inequality and mental health (Gresenz, Sturm, & Tang, 2001; Sturm & Gresenz, 2002). Thus, to date, there has been little to no systematic review of the data on the association between income inequality and mental health, despite a growing interest –academic, political and popular- in the field; see Campbell (2016), Griffiths (2016) and Wilkinson and Pickett (2017) for recent examples.

Aims

The aims of this thesis are to address an identified gap in the literature and determine: (a) whether income inequality is associated with the prevalence / incidence and/or severity of adult mental illness at the subnational level; (b) the direction of this effect (if it is found to exist), since two competing hypothesis will be considered: first, that income *inequality* is detrimental to mental health (consistent with the IIH), and second, that income inequality is beneficial to mental health (consistent with the MNH); (c) whether any association between income inequality and mental illness holds across spatial scales of the geographical comparison area, e.g. neighbourhood or borough, and (d) whether any such association is consistent across different mental health conditions.

Methods

Relevant literature was reviewed using a systematic review approach; for the purposes of reproducibility and transparency, methods and results are reported in line with published (PRISMA) guidelines (Liberati et al., 2009). A meta-analytic approach was *not* adopted since aggregation of effect sizes is inappropriate when studies differ markedly in terms of sample characteristics, outcome variables, methodologies and analytic approaches, and further, may mask important differences in findings (Cooper & Harris, 2003; Hinshaw, 2009; Sharpe, 1997).

Search strategy

Relevant articles were identified by searching PsychInfo, Medline and Web of Science databases from first publication to October 2016. Additional articles were identified by reviewing reference lists of key papers.

Search terms used were based on the two key concepts of 'income inequality' and 'mental health'. Synonyms for income inequality (e.g. 'relative deprivation') and specific indices of income inequality (e.g. 'Gini coefficient', 'pietra ratio') were derived from a review paper of measures of income inequality (De Maio, 2007). All terms described therein were included.

In order to identify studies involving data on specific diagnostic categories and symptoms as well as mental health more generally, a list of relevant synonyms for mental health (e.g. 'psychological disorder', 'mental illness') and diagnostic categories (e.g. 'schizophrenia', 'anxiety', 'depression') were derived from a number of sources including: published search terms from previous meta-analyses of epidemiological studies into mental health, e.g. Barratt et al. (2016), the diagnostic classification systems of the DSM-IV (American Psychiatric Association, 2000), DSM-5 (American Psychiatric Association, 2013) and ICD-10 (World Health Organisation, 1992), as well as diagnostic terms used in the Improving Access to Psychological Therapies (IAPT) and IAPT for Severe Mental Illness services (Health and Social Care Information Centre, 2014).

This generated 11 search terms for income inequality (combined using the OR operator) and 52 search terms for mental health / diagnostic categories (combined using the OR operator), which were in turn combined using the AND operator. See Appendix 1 for full details.

Inclusion and exclusion criteria

Studies were deemed suitable for inclusion if they met the following criteria: (i) they included a measure of mental illness incidence, prevalence or symptom severity, defined using a diagnostic tool, screening instrument or symptom scale; (ii) they included an objective measure of income inequality derived at the subnational level

(see below); (iii) they focused on *adult* mental health, i.e. were not *limited* to a sample population of children / adolescents (defined as <18 years); (iv) were written in English; and (v) were published in peer-reviewed journals.

Studies were excluded for the following reasons: (i) if the measure of income inequality included was based on *subjective* inequality, i.e. perceived inequality assessed through self-report questionnaires, rather than *objective* inequality, i.e. based on economic indices; (ii) if the measure of 'mental health' was in fact a measure of life satisfaction or health-care use; (iii) if the focus of the study was on neurodevelopmental disorders, learning disabilities, degenerative diseases, e.g. dementia, or studies of behaviour, e.g. suicide, aggression, drugs or alcohol use, rather than mental health *per se*; (iv) if the sample population was based on a *highly* specialized population sample for which findings might not be expected to generalise, e.g. HIV+ prisoners (Lincoln et al., 2015).

Selection and coding

To determine whether inclusion criteria were met all records were screened in two phases. In the first, the title and abstract of all studies identified in the search were read to screen for basic relevance including a focus on adult mental health and objective inequality. Where insufficient information could be gleaned in this way, the full article was obtained and the Methods section reviewed. In the second phase, all remaining articles were read in order to exclude studies that did not meet criteria defined above.

Remaining studies (to be included in the review) were then coded for key measures in order to facilitate synthesis of findings and assessment of study quality; see Table 1. These included: the scale of the region of interest, e.g. neighbourhood or borough, the mean population size of the region of interest, the data sample size (at

the individual and higher-order level – e.g. numbers of participants and number of neighbourhoods studied), the type of analyses undertaken, predictors built into statistical analyses, the significance or otherwise of any findings, at an alpha criterion level of 0.05, as well as an index of study quality (see below). Additional variables of interest are also presented in Appendix 2 Supplementary Table 1, including male-to-female ratio, method of data collection and sampling strategy.

Where data were *not* specified in a given study, e.g. the mean population size of the geographical area of interest, this information was sought from original sources, e.g. government reports and national statistics, and/or attempts were made to request the information directly from the authors.

Quality Assessment

Following the approach of Uphoff et al. (2013), studies were scored for *quality* rather than *risk of bias*, the former being more appropriate for a critical appraisal of large-scale cross-sectional and/or ecological data. Thus, most recommended criteria for assessing bias, e.g. random allocation, allocation concealment, participant blinding and blinding of outcome assessment (Higgins & Green, 2011), are not relevant to natural experiments of the kind reviewed here. In line with *a priori* hypotheses, epidemiological studies often *purposely* over-sample –i.e. introduce bias- in order to increase power and facilitate detection of effects in small population groups, e.g. ethnic minorities. Therefore, the criteria identified in Uphoff et al. (2013) were used to assess quality: (i) validity of key measures, and (ii) sample size. In order to afford a more stringent assessment of quality, two additional criteria were also used: (iii) inclusion of appropriate control variables, and (iv) optimal statistical analyses.

Key measures (measures of income inequality and mental illness) were deemed valid and suitable if they accurately mapped onto the stated research question and had been validated in previous peer-reviewed research. A score of one was given if *both* key measures were deemed valid.

For a single-level regression analysis or partial correlation the sample size was deemed appropriate (score of one) if it met the 'one in ten' heuristic, which defines a minimum sample size of ten observations per predictor variable (Austin & Steyerberg, 2015; Vittinghoff & McCulloch, 2007). For multi-level analyses, an additional criteria of a minimum of 30 units, e.g. states or neighbourhoods, at the higher level also had to be met (Maas & Hox, 2004). A score of one was given if *both* these criteria were met.

With respect to appropriate control variables, studies were given a score of one if analyses controlled for *absolute* deprivation at the individual / household level *as well as* at the superordinate level (e.g. neighbourhood), but a score of zero if they controlled for only one or neither of these.

Finally, an additional score of one was given if multi-level analyses were used, with data included at an individual level, e.g. variables capturing age, gender and individual income, as well as at a higher geographical level, e.g. variables capturing neighbourhood income inequality and deprivation.

Therefore, individual studies were given a quality score that could range from zero to four.

Results

Figure 1 shows the sequence by which studies were identified, screened and reviewed. Five hundred and thirty-eight studies were initially identified using defined search terms; only 21 of these met all criteria for inclusion. Table 1 shows the full list of these 21 studies along with key coded variables; additional data are presented in Appendix 2, Supplementary Table 1. The authors of six studies included were contacted to request information that was not available in the publication itself or related sources, e.g. region mean population size; of these, four responded to the requests and provided missing data.

Of the 21 studies included, ten involved data gathered in the US (47.62%) and four in the UK (19.05%), with individual studies contributing data from Australia, South Africa, the Netherlands, Mexico, Wales, Indonesia and Korea (Table 1, column 3).

With respect to the psychological condition examined, eleven of the 21 involved research into depression (52.38%), five into general mental health (23.81%), three into psychosis (14.29%) and two into anxiety and depression (9.52%) (Table 1, column 7).

With respect to the method of collecting individual- level data, of the 21 studies included, one gathered data from distributed questionnaires *only* (4.76%), one from phone-based interviews *and* postal questionnaires (4.76%), two from clinical records *only* (9.52%), three from phone-based *and* face-to-face interviews (14.29%), four from phone-based interviews *only* (19.05%) and ten from face-to-face interviews *only* (47.62%) (Appendix 2, Supplementary Table 1, column 11).

With respect to sampling strategies, 17 studies used some form of probability sampling (80.95%) stratified by geography, e.g. randomly sampling a subset of

households within defined states or municipalities; of these 17, seven also stratified sampling by demographics such as age or ethnicity. This enabled over-sampling of specific populations so that inferences could be made about low incidence groups, e.g. Latinos (Zimmerman & Bell, 2006), or to facilitate a focus on particular groups of interest to the research hypothesis, e.g. individuals with mental health difficulties (Gresenz et al., 2001). Four studies used convenience samples defined by individuals (or the parents of individuals) presenting to services (19.05%) (Appendix 2, Supplementary Table 1, column 12).

Finally, only one study out of the 21 (4.76%) used a longitudinal analysis; the remaining 20 (95.24%) involved cross-sectional analyses (Appendix 2, Supplementary Table 1, column 13).

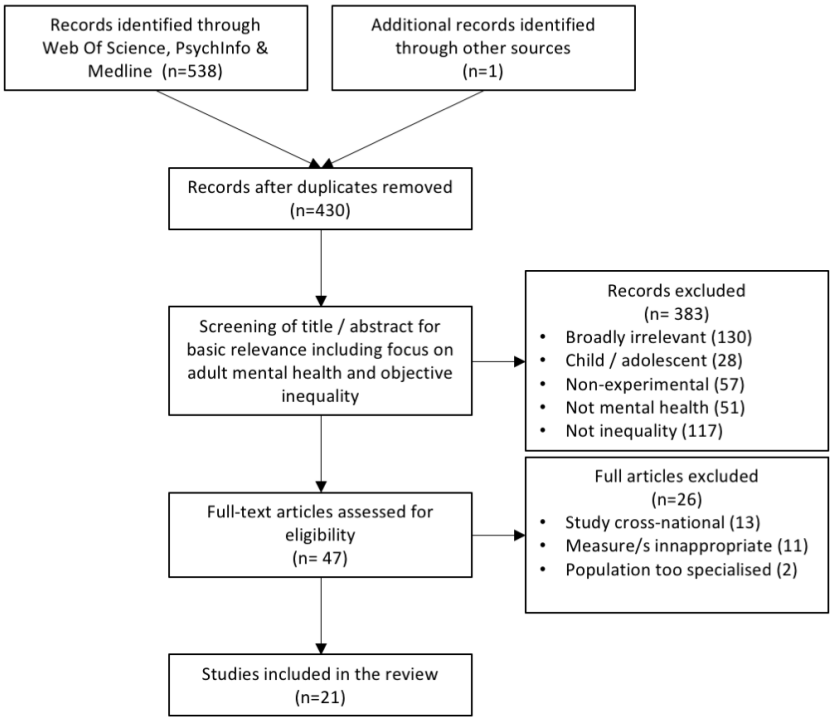

Figure 1 Study inclusion flow diagram. Flow diagram showing sequence by which studies were identified, screened and reviewed.

Table 1 Studies included in the review. The full list of studies included in this review is presented along with corresponding key measures: study authors and date of publication (study), years over which data were gathered (data year), country / focus of study, geographical area of interest (area of interest), region mean population size, inequality measure/s used, mental health variable/s, sample size (N; individual level, with higher level in brackets), type/s of analyses used, lower and higher level predictors, conclusions drawn and assigned quality index (QI). MH = Mental Health; NA = data not available; OR = odds ratio; IRR = incident rate ratio.

Study	Data year	Country / focus of study	Area of interest	Region mean pop size	Inequality measure	MH variable	MH tool	N	Analyses	Lower level predictors	Higher level predictors	Conclusion	QI
(Ahern & Galea, 2006)	2000-2002	US / New York City	Community district	125,000	Gini	Caseness for depression	National Women's Study (NWS) depression module	1355 (59)	Multi-level logistic regression	Age, ethnicity, individual income	Income, inequality	Association between higher inequality & depression (low income participants only) (beta=35.02, p<0.01)	4
(Bechtel, Lordan, & Rao, 2012)	2001	Australia / nationwide	Neighbourhood, city, major statistical region	NA	Gini and others	General mental health Sxs	Short Form Health Survey (SF-36)	67305 (488)	Poisson regression	Age, age-squared, dependents, region of birth, education, household income	Inequality	No association (beta=1.16, p>0.1)	2
(Boydell, van Os, McKenzie, & Murray, 2004)	1988-1997	UK / South London	Electoral ward	10,000	Median deviation from median deprivation	Treated incidence of psychosis	Operational Criteria Checklist for Psychotic Illness (OCPCI)	222 (15)	Multi-level poisson regression	Age, sex, ethnicity	Deprivation, inequality, proportion ethnic minority	Association between higher inequality and FEP (most deprived wards only) (IRR=3.79, p=0.019)	1
(Burns & Esterhuizen, 2008)	2001-2005	South Africa / district of uMgungundlovu	Municipality	72,611	Ratio of mean income of highest to lowest decile earners	Treated incidence of First Episode Psychosis	Meeting DSM-IV criteria	160 (7)	Partial correlation	None	Deprivation, inequality	Association between higher inequality and FEP (r=0.84, p=0.036)	0
(Drukker et al., 2004)	1998-2002	Netherlands / City of Maastricht	Neighbourhood	3,337	Ratio of low to high incomes, house price stdev	General MH Sxs	World Health Organization Quality of Life Assessment (WHOQOL-BREF)	1082 (36)	Multi-level linear regression	Age, sex, occupation, education, welfare recipient, single-parent	Deprivation, inequality	No association (beta=-0.03, p>0.05)	2

Study	Data year	Country / focus of study	Area of interest	Region mean pop size	Inequality measure	MH variable	MH tool	N	Analyses	Lower level predictors	Higher level predictors	Conclusion	QI
(Fernandez-Nino, Manrique-Espinoza, Bojorquez-Chapela, & Salinas-Rodriguez, 2014)	2010	Mexico / nationwide	Locality, municipality, state	45,616	Gini	Caseness for depression	Centre for Epidemiological Studies Depression Scale (CES-D)	8,874 (2,456)	Multi-level logistic regression	Age, sex, civil status, education, employment, household assets, household rurality, household deprivation + 6 more	Municipality and state deprivation, inequality	No association at the municipality (OR=1.68, p>0.1) or state (OR=0.45, p>0.1) level	4
(Fiscella & Franks, 2000)	1982-1987	US / nationwide	Primary Sampling Unit	NA	Proportion of total income earned by poorest 50%	Sxs of depression	General Well Being (GWB) Schedule	14,407 (105)	Linear regression	Age, sex, household income	Inequality	Association between higher inequality & depression (beta=-0.21, p<0.05)	1
(Fone et al., 2013)	2003-2010	Wales / nationwide	Lower Layer Super Output Area, Unitary Authority	1558; 135,000	Gini	General MH Sxs (& caseness)	Short Form Health Survey (SF-36)	88,623 (1887/22)	Multi-level linear & logistic regression	Age, sex, education, employment, housing tenure, household socioeconomic level	Deprivation, inequality	Association between higher inequality & better mental health at LSOA level (low deprivation areas only) (beta=0.7, p=0.04); association between higher inequality & poorer mental health at UA level (beta=-1.35, p=0.01)	4
(Gresenz et al., 2001)	1990-1998	US / nationwide	State, Community	4,961,421	Gini and others	Caseness for anxiety or depression, general MH Sxs	Short Form Health Survey (SF-36), Composite International Diagnostic Interview (CIDI)	6925 (60)	Multi-level linear & logistic regression	Individual & family income	Income, inequality	No association at community (beta=-0.45, p>0.1) or state (beta=1.27, p>0.1) level	4
(Hanandita & Tampubolon, 2014)	2007	Indonesia / nationwide	District	1,471	Gini	General MH Sxs (& caseness)	20-item Self-Reporting Questionnaire (SRQ)	577,548 (440)	Linear & poisson regression	Age, sex, civil status, education, employment, household consumption + 6 more	Precipitation level, deprivation, inequality	Association between higher inequality & poorer general mental health (beta=3.59, p<0.01)	3

Study	Data year	Country / focus of study	Area of interest	Region mean pop size	Inequality measure	MH variable	MH tool	N	Analyses	Lower level predictors	Higher level predictors	Conclusion	QI
(Henderson, Liu, Diez Roux, Link, & Hasin, 2004)	1990-1992	US / nationwide	State	4,961,421	Gini	Sxs of depression & alcohol dependence (& caseness)	Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS)	42,862 (48)	Logistic regression	Age, ethnicity, education, household family size, urbanicity, household income	Income, inequality	No association for males (OR=0.9, p>0.05) or females (OR=1.09, p>0.05)	3
(Kahn, Wise, Kennedy, & Kawachi, 2000)	1990-1991	US / nationwide	State	4,961,421	Gini	Caseness for depression	Centre for Epidemiological Studies Depression Scale (CES-D)	8,060 (50)	Logistic regression	Age, civil status, education, ethnicity, household no., household income	Inequality	Association between higher inequality & depression (strongest effect in low income participants) (OR=1.3, p>0.05)	2
(Kirkbride, Jones, Ullrich, & Coid, 2014)	1996-2004	UK / East London	Statistical ward	6195	Gini	Treated incidence of psychosis	Schedules for Clinical Assessment in Neuropsychiatry (SCAN)	427 (56)	Multi-level Bayesian modeling	Age, sex, ethnicity, socioeconomic level	Social fragmentation, cohesion, pop density, ethnic separation, deprivation, inequality	Association between higher inequality & non-affective psychosis (RR=1.25, p<0.05) but not for affective psychosis	4
(Lee & Park, 2015)	2009	Korea / nationwide	Community	NA	Gini	Caseness for depression	Centre for Epidemiological Studies Depression Scale (CES-D)	230,715 (253)	Multi-level logistic regression	Age, sex, education, no. of illnesses, living alone, family income	Income, inequality	No association (OR=0.87, p>0.05)	4
(Marshall et al., 2014)	2002-2004	UK / nationwide	Middle Superior Output Area	7200	Gini (based on house prices)	Caseness for depression	Centre for Epidemiological Studies Depression Scale (CES-D)	10,644 (2000+)	Multi-level logistic regression	Age, sex, ethnicity, education, household wealth, economic activity	Deprivation, inequality	Association between higher inequality and lower levels of depression (strongest effect in low income participants) (OR=0.81, p<0.05)	4
(Messias, Eaton, & Grooms, 2011)	2006-2008	US / nationwide	State	5,425,007	Gini	Prevalence of depression	Physical Health Questionnaire (PHQ-8)	235,067 (45)	Linear regression	None	Income, inequality, % with a college degree, % over 65	Association between higher inequality and depression (unstandardized beta=43.67, p<0.001)	2

Study	Data year	Country / focus of study	Area of interest	Region mean pop size	Inequality measure	MH variable	MH tool	N	Analyses	Lower level predictors	Higher level predictors	Conclusion	QI
(Muramatsu, 2003)	1990-1994	US / nationwide	County	100,000	Gini	Sxs of depression	Centre for Epidemiological Studies Depression Scale (CES-D)	6,640 (211)	Multi-level linear regression	Age, sex, ethnicity, socioeconomic level, marital status, physical health	Income, inequality	Association between higher inequality & lower depression (beta=2.59, p<0.01)	4
(Pabayo, Kawachi, & Gilman, 2015)	2001-2005	US / nationwide	State	5,616,997	Gini	3-year incidence of depression	Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS)	34653 (50)	Multi-level logistic regression	Age, sex, ethnicity, education, marital status, hx of depression, past-year life events, household income	Income, inequality	Association between higher inequality & depression for women (OR=1.5, p<0.05) but not for men	4
(Sturm & Gresenz, 2002)	1990-1998	US / nationwide	Metropolitan area / economic area	NA	Gini	Caseness for depression or anxiety	Composite International Diagnostic Interview (CIDI)	8,235 (60)	Logistic regression	Age, sex, ethnicity, education, family size, family income	Income, inequality	No association (p>0.1)	2
(Weich, Lewis, & Jenkins, 2001)	1991	England, Wales & Scotland / nationwide	Region	3,000,000	Gini	Caseness for general MH	General Health Questionnaire (GHQ)	8,191 (18)	Logistic regression	Age, sex, ethnicity, employment, social class, physical health problems, housing tenure, household income	Income, inequality	Association between higher inequality & poorer MH in wealthier participants (OR=1.31, p=0.02); higher inequality and better MH in poorer participants (OR=0.42, p<0.001)	2
(Zimmerman & Bell, 2006)	1984-2000	US / nationwide	County	100,000	Percentage of households with income over threshold	Caseness for depression	Centre for Epidemiological Studies Depression Scale (CES-D)	4,81 (NA)	Logistic regression	Sex, ethnicity, education, employment, possession of health insurance, lone living, region of residence, household income	Social capital, affordable housing, unskilled wages, unemployment, proportion ethnic minority, education, psychiatric and health service availability, income, inequality	No association (OR=0.74, p>0.05)	NA

Table 2 Breakdown of studies by quality, controlled variables and mean population size. The number of studies that are wholly or partially supportive of the Income Inequality Hypothesis (IIH), wholly or partially supportive of the Mixed Neighbourhood Hypothesis (MNH), or else unsupportive of either theory, is presented for: (i) all studies, (ii) higher quality studies only (assessed using the Quality Index), (iii) studies that controlled for absolute deprivation only (at the higher- order level *as well as* at both the individual- and higher- order levels), and (iv) studies stratified by the mean population size of the geographical area of interest (X<10,000; 10,000<X<1million; X>1million; NA=data not available). For these data, percentages of total studies (row total) are also presented in brackets.

		Wholly Supportive of the Income Inequality Hypothesis (only sig '+' findings)	Partially supportive of the Income Inequality Hypothesis (some sig '+' & some null findings)	Unsupportive of either theory (no sig findings, or sig '+' & '-' findings)	Partially supportive of the Mixed Neighbourhood Hypothesis (only sig '–' & some null findings)	Wholly supportive of the Mixed Neighbourhood Hypothesis (only sig - findings)	Total
(i) All studies		6 (28.57%)	4 (19.04%)	10 (47.62%)	0 (0%)	1 (4.76%)	21 (100%)
(ii) Higher quality studies only		2 (18.18%)	3 (27.27%)	5 (45.45%)	0 (0%)	1 (9.09%)	11 (100%)
(iii) Studies that controlled for absolute deprivation	**At higher- order level**	4 (23.53%)	4 (23.53%)	8 (47.06%)	0 (0%)	1 (5.88%)	17 (100%)
	At individual- and higher-order levels	2 (15.38%)	3 (23.08%)	7 (53.85%)	0 (0%)	1 (7.69%)	13 (100%)
(iv) Studies stratified by mean population size	**Statistical ward, neighbourhood (<10,000)**	1 (20%)	2 (40%)	1 (20%)	0 (0%)	1 (20%)	5 (100%)
	County, district, municipality or authority (>10,000)	2 (33.33%)	1 (16.67%)	3 (50%)	0 (0%)	0 (0%)	6 (100%)
	State or region (> one million)	2 (33.33%)	1 (16.67%)	3 (50%)	0 (0%)	0 (0%)	6 (100%)
	NA	1 (25%)	0 (0%)	3 (75%)	0 (0%)	0 (0%)	4 (100%)

Table 3 Breakdown of studies by mental health condition. The number of studies that are wholly or partially supportive of the Income Inequality Hypothesis (IIH), wholly or partially supportive of the Mixed Neighbourhood Hypothesis (MNH), or else unsupportive of either theory, is presented for: (i) all studies, (ii) studies stratified by mental health condition (general mental health, depression and psychosis), and (iii) studies focusing on all mental conditions *except* general mental health. For these data, percentages of total studies (row total) are also presented in brackets.

		Wholly Supportive of the Income Inequality Hypothesis (only sig '+' findings)	Partially supportive of the Income Inequality Hypothesis (some sig '+' & some null findings)	Unsupportive of either theory (no sig findings, or sig '+' & '-' findings)	Partially supportive of the Mixed Neighbourhood Hypothesis (only sig '–' & some null findings)	Wholly supportive of the Mixed Neighbourhood Hypothesis (only sig - findings)	Total
(i) All studies		6 (28.57%)	4 (19.05%)	10 (47.62%)	0 (0%)	1 (4.76%)	21 (100%)
(ii) Studies stratified by mental health condition	**General mental health**	1 (14.29%)	0 (0%)	6 (85.71%)	0 (0%)	0 (0%)	7 (100%)
	Depression	4 (36.36%)	2 (18.18%)	4 (36.36%)	0 (0%)	1 (9.09%)	11 (100%)
	Psychosis	1 (33.33%)	2 (66.67%)	0 (0%)	0 (0%)	0 (0%)	3 (100%)
(iii) All mental health conditions except *general* mental health		5 (35.71%)	4 (28.57%)	4 (28.57%)	0 (0%)	1 (7.14%)	14 (100%)

Quality analysis

The mean and standard deviation score on the quality index was 2.8 (±1.28) out of a maximum possible score of four. Of the 21 studies included in the analysis, only 11 scored in the higher range (>=3). Exploring the reasons for this: four studies lost a point for inadequate sample sizes, five for low relevance / validity of key measures, eight lost a point for a lack of adequate control variables, i.e. measures of absolute deprivation (at the individual and/or neighbourhood level) were not built into the model, and ten lost points for non-optimal analyses, i.e. single-level rather than multi-level analyses; see Appendix 2, Supplementary Table 1 for further details in relation to specific studies.

Evidence for the Income Inequality Hypothesis vs. the Mixed Neighbourhood Hypothesis

Table 2 shows a breakdown of the number of studies that were found to be partially or wholly supportive of the IIH, partially or wholly supportive of the MNH, or else unsupportive of either. Considering *all* the data first (top row), 47.62% (n=10) were found to be either wholly or partially supportive of the IIH, whereas only 4.76% (n=1) were found to be supportive of the MNH. In contrast, 47.62% of the studies (n=10) were found to be unsupportive of *either* hypothesis. Of these ten, two showed mixed findings, i.e. different sub-analyses supporting the MNH and IIH, whereas only eight (38.1%) were due to null findings.

A near-identical pattern of results also emerged when a subset of studies that were of a higher quality were analysed separately, i.e. those scoring three or four on the quality index (Table 2). Out of the 11 higher quality studies included, 45.45% (n=5) were found to be either wholly or partially supportive of the IIH, whereas only one (9.09%) was found to be supportive of the MNH. In turn, 45.45% (n=5) of studies

were found to be unsupportive of either hypothesis, of which four (36.36%) were due to null results.

A similar pattern also emerged when only studies that controlled for absolute levels of deprivation at the higher- order level were analysed. Of these 17 studies, 47.06% (n= 8) were found to be either wholly or partially supportive of the IIH, whilst only one (5.88%) was found to be supportive of the MNH. However, 47.06% (n=8) of studies were found to be unsupportive of either hypothesis (35.29% due to null results).

Finally, broadly similar -though slightly less pronounced- results were *also* seen when absolute levels of deprivation were controlled for at *both* the individual- and higher- order levels. Of these 13 studies, 38.46% (n= 5) were found to be either wholly or partially supportive of the IIH, whilst only one (7.69%) was found to be supportive of the MNH. However, 53.85% (n=7) of studies were found to be unsupportive of either hypothesis (45.45% due to null results).

Taken together, these findings provide support for the IIH over the MNH, with evidence of an association between poorer mental health and higher income inequality, over and above the effects of absolute deprivation.

Effects of geographical scale

Following the approach of Wilkinson and Pickett (2006), the data were also analysed with studies stratified according to the mean population size of the geographical area of interest (Table 2): fewer than 10,000 (e.g. statistical wards and neighbourhoods), greater than 10,000 but fewer than one million (e.g. counties, districts, municipalities and authorities) and greater than one million (e.g. states and regions). These cut-offs were designed to generate an approximately equal number

of studies at each level in order to facilitate comparison. For four of the studies, no information about geographical scale was available.

There was little to suggest that the association between inequality and mental health was linked to geographical scale. Across the three different levels the vast majority of the studies (80-100%) were found to be either supportive of the IIH or else unsupportive of either hypothesis (Table 2).

Pattern across conditions

In Table 3 data were re-analysed by mental health condition. Two studies that examined caseness for anxiety and depression were lumped together with the studies of general mental health, since measures of the latter, e.g. the SF-36, GHQ and SRQ, in fact tended to focus on anxiety and depression.

With respect to studies of *general* mental health, the vast majority (6 out of 7; 85.71%) were unsupportive of either the IIH or the MNH. In fact, only one study, undertaken in Indonesia, found a straightforward association between inequality and mental health (Hanandita & Tampubolon, 2014), showing that *higher* inequality was linked to *poorer* mental health. Of these six studies that were unsupportive of either the IIH or the MNH, four showed no significant effects and two showed equal support for the two theories in sub-analyses undertaken.

With respect to studies of depression, 54.55% (n=6) were found to be supportive (either wholly or partially) of the IIH, whilst only a single study (9.09%) was supportive of the MNH (Marshall et al., 2014). A further 36.36% of the studies (n=4) were found to be unsupportive of either hypothesis, all due to null effects.

Finally, whilst there were only three studies of psychosis, all were either wholly or partially supportive of the IIH, whilst none were supportive of the MNH.

Taken together, these findings suggest that there may be differences in the extent to which different mental health conditions are associated with inequality. Thus, associations between higher inequality and poorer mental health were most evident in studies of depression, and possibly psychosis.

Interactions between inequality and absolute deprivation

Ten of the 21 studies included in the analyses reported that they examined interactions between inequality and *absolute* deprivation, either at the individual / household level or at a higher level, e.g. neighbourhood. These were typically undertaken by adding interaction terms to a regression model, or by splitting data into low and high deprivation groups and analysing the data separately.

Of the ten studies that explored interactions between inequality and absolute deprivation, four suggested that individuals of a lower SES are negatively affected by inequality, and/or individuals of a higher SES are positively impacted upon (assuming a causal association). Thus, two studies reported higher levels of depression amongst poorer participants living in high inequality areas (Ahern & Galea, 2006; Kahn et al., 2000). Boydell et al. (2004) reported a higher treated incidence of psychosis in areas high in poverty *and* inequality, and Fone et al. (2013) reported better general mental health in richer areas characterized by high inequality, although this was only evident at one spatial scale of analysis.

In contrast, of the ten studies that explored interactions between inequality and absolute deprivation, two identified a positive effect of inequality on individuals of a lower SES and/or a negative effect on individuals of a higher SES. Thus, Marshall et

al. (2014) reported lower levels of depression amongst poorer participants living in areas of high inequality. Weich et al. (2001) found an association between higher inequality and poorer general mental health in wealthier participants and an association between higher inequality and better general mental health amongst the poor.

Finally, of the ten studies that explored interactions between inequality and absolute deprivation, four reported *no* significant interactions (Gresenz et al., 2001; Henderson et al., 2004; Muramatsu, 2003; Sturm & Gresenz, 2002).

Discussion

The studies included in this review provide clear evidence for the IIH over the MNH. Irrespective of how the data were analysed, the overwhelming majority of studies that provided evidence for one of these two theories supported an association between *higher* inequality and *poorer* mental health, consistent with the IIH. Further, although there is a risk of over-interpreting subtler patterns within the data given the relatively small sample of studies included, this pattern was not dependent on the spatial scale of the geographical area of analysis or the quality of the studies included. However, there was some suggestion of a possible dependence on the nature of the mental health condition. Whilst studies of depression and psychosis supported the IIH, the majority of studies of *general* mental health included showed *no* association with inequality.

These findings suggest that the MNH can be rejected as an explanation of *mental* health inequalities at the *subnational* level. However, given the high proportion of studies that reported *no* significant association between inequality and mental illness (38.1% for all studies, rising to 45.45% when *absolute* deprivation was controlled for), to what extent can these findings be interpreted as positive evidence

for the IIH? To explain such a high level of null findings one might posit two alternative explanations. One possibility is that findings supportive of the IIH have arisen purely by chance, but are over-represented in the literature due to publication bias (Nieuwenhuis, 2016; Rosenthal & Robert, 1979). An alternative possibility is that the association between higher inequality and mental health is real, but statistically small and/or potentially dependent on other moderating variables that are as yet unidentified.

There are a number of reasons to support the second explanation, i.e. the existence of a real -but potentially small- effect of inequality. First, if the pattern of findings reported was merely due to publication bias one would have to explain why these biases are operating to selectively suppress studies supportive of the MNH, rather than null findings, since the latter were in fact well represented. Even if this were the case, however, and findings supportive of the MNH were found to be largely excluded from the published literature, this would not explain why nearly 50% of the remaining studies showed significant effects supportive of the IIH, as opposed to the 2.5% one might expect to follow this pattern by chance alone (the type one error rate for an alpha criterion of 0.05 at one tail of the distribution). Finally, the likelihood that any genuine effect of the IIH on mental health is relatively small -and hence easily missed in studies (type two error)- is supported by the demonstration of a relatively weak association of inequality with *physical* health in a previous meta-analysis (Kondo et al., 2009), as well as a browsing of the reported odds ratios and coefficients presented in Table 1.

Considering the more specific claims of the IIH, the effects of inequality should not be felt by the poorer members of society *only*, but should affect the richer too (Wilkinson & Pickett, 2009). The findings reported here are consistent with this hypothesis: the association between inequality and mental health persisted in a sub-

analysis of studies that had controlled for individual and higher level income / deprivation. A number of studies also explicitly looked for an interaction between inequality and absolute deprivation. Of these, twice as many supported a detrimental effect of inequality on the poor and/or a positive effect on the rich, rather than the reverse, i.e. a positive effect on the poor and/or a negative effect on the rich. However, there were too few studies in this sub-analysis to draw any firm conclusions. Consequently, in the absence of further evidence, there is little to suggest that inequality uniquely affects people of lower or higher SES.

A more recent claim made by Pickett and Wilkinson (2010) in relation to the IIH is that the effects of inequality become less pronounced at smaller spatial scales, such that it essentially disappears below the national -or certainly state- level. Proponents of this view propose that this is because the spatial scale over which social comparisons operate is large (Drukker et al., 2004), e.g. across a nation rather than across a neighbourhood. In contrast, the data reviewed indicate an association between higher inequality and poorer mental health that operates at a sub-national level, with no evidence to suggest that the effect disappears at smaller spatial scales. These data are instead consistent with research on social comparison (Festinger, 1954) and social rank (Gilbert, 2000) theories, which has shown that the negative effects of social comparisons operate across multiple reference groups and spatial scales. For example, analysing data from 19,000 participants across 24 countries, Clark and Senik (2010) reported that the importance people attach to social comparisons about their income relative to friends' and colleagues' negatively predicted their self-reported level of happiness; i.e. intense social comparisons were associated with being less happy. Goerke and Pannenberg (2013) found a similar pattern with respect to income comparisons made in relation to friends, co-workers and others in the same occupation. Although studies of this kind do not demonstrate a causal relationship between social comparisons and mental health, they suggest

that people make meaningful social comparisons with others in their immediate social networks.

It is important to note, that much of the evidence to support Wilkinson and colleagues' claims is based on *physical* health data; see Wilkinson and Pickett (2006) for a review. Whilst the authors posit that these effects are mediated primarily by psychosocial factors (consistent with the Status Anxiety Hypothesis), it is possible that partially separable mechanisms are involved in mediating the effects of inequality on *mental* and *physical* health (see (Zimmerman & Bell, 2006) for example), and further, that these might be differentially affected by spatial scale. For example, one might imagine how reduced investment in public infrastructure and services in areas of high inequality (consistent with the Neomaterialist Hypothesis) might operate over larger spatial scales than adverse social comparisons, and have a greater impact on physical health. Hence, the association between inequality and *physical* health may in fact collapse at smaller spatial scales and be partially dissociable from its association with *mental* health.

With respect to the effects of inequality on different mental health conditions, the IIH makes no clear predictions in this regard. In relation to the findings reported here, whilst it is difficult to comment on psychosis given the paucity of relevant studies, the data tentatively suggest that the association between higher inequality and poorer mental health is more pronounced in studies of depression than in studies of *general* mental health. One possible explanation for this finding is that the effects of inequality on mental health may be mediated, at least in part, by depression itself, such that tools specifically designed to detect the symptoms of depression, e.g. the CES-D, are more sensitive to inequality-associated distress. This notion, essentially a variant of the Status Anxiety Hypothesis, would propose that social comparisons and a lack of social cohesion, heightened under conditions of high inequality, are

inherently depressogenic. However, as with the well-documented association between *absolute* deprivation and depression (see Lorant et al. (2003) for a meta-analysis), the direction of causality cannot be inferred in the absence of more sophisticated experimental paradigms; see Hudson (2005) for example. Thus, individuals experiencing depression might be more likely to migrate to areas characterized by high inequality (social drift hypothesis), or alternatively, social inequality might *itself* represent a risk factor for the development of depression (social causation hypothesis). See Goldman (1994) for discussion.

Limitations of the review and included studies

With respect to the weaknesses of this review and studies therein, there are several. First, the number of papers meeting inclusion criteria was relatively small, such that conclusions drawn must be tentative, particularly in relation to sub-analyses. Second, all but one of the studies included in the review are based on cross-sectional analyses, so that causation cannot be inferred. Third, no measure of potential bias was used to assess included studies. However, in the service of the authors' stated research aims and hypotheses, several studies purposely over-sampled specific groups or regions, and others used convenience sampling, e.g. all individuals presenting to services. These were perfectly legitimate approaches, without which, for example, statistically significant conclusions could not be drawn about low incidence groups. Nonetheless, these biases in sampling may limit the generalizability of the findings, i.e. the extent to which they can be extrapolated to the *general* population.

Finally, no attempt was made to integrate effect sizes across the studies. This is because any aggregate effect size based on such heterogeneous data would be largely meaningless (Cooper & Harris, 2003; Hinshaw, 2009; Sharpe, 1997). Thus, included studies differed with respect to: country of interest, geographical scale, key

measures and outcomes (e.g. symptom severity, prevalence and incidence), clinical condition, statistical method and controlled variables. As a result, however, the real-world significance of the effects reported is difficult to gauge. In addition, it was not possible to test for publication bias directly, i.e. through a funnel plot.

Future research

This review has highlighted the need for further research into the effects of inequality on mental health. Thus, only 21 studies were found to meet inclusion criteria, and this across the full range of mental health conditions and spatial scales examined. Further, the quality analysis undertaken highlighted a number of limitations in the existing research, most notably a lack of appropriate control variables / potential confounders, i.e. measures of absolute deprivation, and non-optimal (single-level) study designs; these issues should be addressed in future studies.

In addition, future research should attempt to determine the direction of causality and underlying mechanisms that drive the association between inequality and mental health, as well as the real-world clinical significance of any such association. To this end, future studies should ideally incorporate longitudinal designs and additional predictor variables, e.g. measures of status anxiety, social capital and local investment, that directly map on to hypothetical causal models. See Layte (2012) for example.

Broader implications

The findings of this thesis contribute to a growing body of research into the environmental predictors of psychological disorders, and as such, have a number of implications for the commissioning, design and implementation of mental health services.

At the level of service commissioning, the role of environmental factors in the etiology and/or maintenance of mental health difficulties raises a number of key questions about where to focus investment, particularly in the context of limited resources. For example, national guidelines for implementation of Early Intervention Psychosis (EIP) services in the UK (NICE, 2016) state that commissioning "should be underpinned by estimated local incidence of psychosis, derived to incorporate a range of demographic features such as ethnicity, age, population density and deprivation" (p.6). At a broader level, it is not clear to what extent expenditure should be focused on the development and provision of mental health services that work with the individual to target symptom reduction (Clark et al., 2016) over investments into a wider range of services as part of a more systemic preventative approach (Dahlgren & Whitehead, 1991; World Health Organisation, 2004). For example, Marmot (2004) has argued for the importance of focusing on "early child development and education, work environments, building healthy communities and supporting active social engagement of older people" in overcoming the effects of social inequality on health (p.153).

A parallel argument might also be made for tackling inequality more directly, i.e. as a primary causal factor in health difficulties, as part of government policy. Thus, whilst a review of relevant economic theory lies beyond the remits of this thesis, it is worth noting that a number of academics, including economists, e.g. Stiglitz (2013), and epidemiologists, e.g. Marmot (2004), have argued that trends for rising inequality can in fact be reversed through targeted changes in social policy, without sacrificing overall economic growth (Cingano, 2014). Proven tools in this regard include progressive taxation and focused expenditure aimed at improving education and reducing hunger and poverty (OECD, 2015).

The demonstration of an association between income inequality and mental health may also have a number of implications for ways of working within the field of Clinical Psychology. Thus, the findings reported might be seen as part of a broader body of research that highlights the role that social and socioeconomic factors play in the etiology and perpetuation of psychological distress. At the most basic level, an understanding of how deprivation, unemployment, housing instability, food insecurity and other stressors contribute to mental health should be considered at formulation, treatment and outcome evaluation stages. For example, psychological interventions have been found to be more effective in treating individuals from a low socioeconomic status when these are designed to address poverty-related issues (Grote et al., 2009; Miranda, Chung, et al., 2003; Miranda, Azocar, Organista, Dwyer, & Areane, 2003). Finally, although controversial, there is ongoing debate, particularly within the field of Community Psychology, as to the broader roles of a Clinical Psychologist, and the extent to which this should involve influencing and changing social policy; see Wolff and Tom (2014) for example.

Conclusions

This systematic review highlights an association between higher levels of income inequality and poorer adult mental health at the subnational level. Whilst the review could not identify the mechanisms or direction of this association, the conclusions drawn reinforce the importance of further research into the social predictors of psychological distress, as well as the potential value of considering these issues further in the commissioning, design and delivery of psychological services. Irrespective of the outcome of future research in the field, the importance of understanding the drivers and consequences of inequality is only likely to grow in the future. In a recent report entitled ‘Britain in the 2020s’, the Institute for Public Policy Research, an independent charitable think-tank (Lawrence, 2016), predicted that inequality will “surge” in the coming decade (p.12), with the income of the rich

forecasted to rise 11 times faster than the incomes of the poor, and an extra 3.6 million predicted to fall into poverty within this time-frame.

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Part 2: Empirical Paper

Individual- and neighbourhood-level predictors

of psychotic symptom dimensions in West London

Abstract

Aims: To determine whether environmental factors such as deprivation and inequality predict significant variance in psychotic symptoms / symptom dimensions after controlling for individual- level variables.

Methods: The study represents a secondary analysis of data gathered from 345 first episode psychosis patients presenting to services in the West London region. Symptom dimension scores, derived from principle components analyses of the SAPS and SANS, were regressed on individual- and neighbourhood- level predictors using multi-level regression analyses.

Results: Neighbourhood- level effects explained a significant proportion of variance in global symptom severity (9.3%, p=0.02) after controlling for individual- level demographics (age, gender and socioeconomic status). Further, *higher* income inequality was associated with *lower* negative symptom scores (p=0.01) and *higher* levels of ethnic segregation were associated with *lower* positive symptom scores (p=0.02), even after controlling for individual- level demographics, other symptoms scores and *absolute* levels of deprivation.

Conclusion: These findings contribute to a growing body of evidence that implicates specific associations between psychotic symptom dimensions and neighbourhood- level social / socioeconomic factors. Further, they implicate the need for longitudinal studies that can begin to tease apart patterns and directions of causation.

Introduction

The psychotic disorders represent a broad family of psychological conditions that are characterised by cognitive, affective, perceptual, behavioural, and social symptoms (American Psychiatric Association, 2013; World Health Organisation, 1992). Despite more than a century of research in the field, however, there is no known *single* cause for psychosis; see Murray and Fearon (1999) for discussion. Instead, a growing body of research points towards a complex, multi-factorial etiology (Assen Jablensky, 2000), with a broad range of risk factors putatively acting –and interacting- at the level of the individual, the inter-personal as well as the societal; see Shah, Mizrahi, and McKenzie (2011) and Van Os, Rutten, and Kenis (2010) for conceptual frameworks of how these might connect.

Studies exploring geographical variation in psychosis incidence have highlighted a number of adverse characteristics of environments that are associated with an increased risk of psychosis. These include: high population density (e.g. Kirkbride, Jones, Ullrich, and Coid (2014)), high urbanicity (see Heinz, Deserno, and Reininghaus (2013) for a review), high income deprivation (see O'Donoghue, Roche, and Lane (2016) for a review), and more recently, high income *inequality*, i.e. socioeconomic variance (Burns & Esterhuizen, 2008; Burns, Tomita, & Kapadia, 2014; Kirkbride et al., 2014). Further, a number of studies have begun to explore the extent to which the quality of social interactions within a community, e.g. trust, support and civic engagement, predict psychosis. Variably referred to as 'social capital' (McKenzie, Whitely, & Weich, 2002), 'social cohesion' (Kirkbride et al., 2007), or its inverse, 'social fragmentation' (Allardyce et al., 2005), there is a relative paucity of research in this area as it relates to psychosis, and further, ongoing debate with little consensus as to how best to define and operationalize these putatively related constructs; see Orford (2008) for a review and discussion.

Whilst large-scale epidemiological research has played an important role in the identification of these environmental risk factors, research in the field has typically relied upon studies of psychosis *incidence* or *prevalence*, i.e. the search for factors that predict the likelihood that an individual will -or will not- receive (/have received) a diagnosis. However, there are a number of reasons –theoretical and empirical- why a *categorical* approach of this kind might not be optimal (Allardyce, McCreadie, Morrison, & van Os, 2007). First, the theoretical underpinnings of a categorical / diagnostic approach to the classification of psychosis has *itself* been called into question. A number of authors have argued that the psychotic disorders represent a heterogeneous collection of phenomena, with diagnostic categories demonstrating poor validity, reliability and etiological specificity; see Allardyce, McCreadie, et al. (2007), Bentall (2004) and The British Psychological Society (2014) for discussion. For example, within a single diagnosis, e.g. schizophrenia, large differences in symptoms, clinical course, cognitive ability and treatment outcome are commonly observed (Jablensky, 2006; Joyce, Hutton, Mutsatsa, & Barnes, 2005; Stroup, 2007). Conversely, distinct diagnoses, e.g. schizophrenia and bipolar disorder, may share many features, including patterns of genetic, biochemical and neuroanatomical abnormalities (Craddock, O'Donovan, & Owen, 2006; McDonald et al., 2005; Murray et al., 2004; Post, 1999).

As an alternative to the categorical approach, dimensional (or 'factor') based approaches posit that psychopathology, including psychosis (Allardyce, Suppes, & Van Os, 2007; Van Os, 2015), may be better formulated with respect to multiple, continuous symptom dimensions (Chmielewski, 2012; Widiger, 2005). This is a view that has gained strength within mental health research and clinical practice. For example, whilst the latest version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) (American Psychiatric Association, 2013) has retained a predominantly diagnostic approach to the classification of psychopathology, it

includes a rating tool called the Clinician-Rated Dimensions of Psychosis Symptom Severity, which is designed to aid treatment planning, decision-making and research (Parker, 2014). In 2008, the National Institute of Mental Health (NIMH) launched the Research Domain Criteria (RDoC) project, which advocates research that is not constrained by diagnostic categories, but instead, attempts to identify and explore domains of functioning and associated dimensional constructs (Morris & Cuthbert, 2012). Further, in a report entitled 'Understanding Psychosis and Schizophrenia', the British Psychological Society (BPS) took a critical stance towards diagnostic approaches to psychosis, and instead emphasized the importance of formulation of presenting difficulties and symptoms (The British Psychological Society, 2014).

Within a dimensional approach to the study of psychosis, symptom dimensions are typically derived from large-sample data using the statistical method of factor analysis or principal component analysis (PCA), e.g. Russo et al. (2014). Whilst studies of this kind have rendered a variable number of psychotic symptom dimensions (John, Khanna, Thennarasu, & Reddy, 2003; Klimidis, Stuart, Minas, Copolov, & Singh, 1993; Peralta & Cuesta, 1999; Peralta, Cuesta, & Farre, 1997), at least three core dimensions repeatedly emerge, reflecting the positive, negative and disorganized symptoms of psychosis (Andreasen, Arndt, Alliger, Miller, & Flaum, 1995). There is evidence to suggest that certain documented risk factors in psychosis may exhibit some specificity in their association with these dimensions, with defined adverse experiences and/or environmental conditions being associated with an elevated risk of specific symptoms within the syndrome of psychosis; see Bentall, Wickham, Shevlin, and Varese (2012) for a review. Further, these associations may be less pronounced / harder to detect in analyses undertaken at the level of incidence or prevalence.

For example, it has been suggested that certain types of environments, e.g. densely populated areas of urban deprivation, may play a *specific* casual role in the development and expression of *positive* symptoms, e.g. paranoid delusions and hallucinations (Ellett, Freeman, & Garety, 2008; Veling, Pot-Kolder, Counotte, van Os, & van der Gaag, 2016). Proposed mechanisms for this association include the putative mediating role of stressful life experiences such as trauma (Bentall et al., 2012), discrimination (Janssen et al., 2003) and victimization (Schreier et al., 2009), which may be particularly common in these environments; see Oher et al. (2014) for discussion. Thus, according to cognitive models of psychosis, e.g. Garety, Kuipers, Fowler, Freeman, and Bebbington (2001), early and ongoing exposure to stressors such as these results in the development of negative beliefs about the self and others, in addition to a number of cognitive biases, e.g. external attribution biases and a tendency to 'jump to conclusions' (Janssen et al., 2006; Moritz, Van Quaquebeke, & Lincoln, 2012; Romm et al., 2011). As a result of these biases, ambiguous stimuli and events may come to be appraised as negative, externally driven and beyond the individual's control, key ingredients for the development of paranoid delusions (Bentall et al., 2009). Further, once triggered, paranoid thinking may be exacerbated by the ongoing experience of deprivation and a perceived - and/or real- lack of social status relative to others in one's community (Wickham, Shryane, Lyons, Dickins, & Bentall, 2014), an experience that may be particularly common in urban areas characterized by high social and economic inequality. With respect to hallucinations, the link with social adversity and deprivation may be even more direct. Thus, trauma -particularly abuse- is a strong risk factor for hallucinations and other anomalous perceptual experiences (Freeman & Fowler, 2009; Janssen et al., 2004), leading several researchers to explore the possible links between hallucinations in psychosis, and the trauma-related intrusions that are characteristic of Post-Traumatic Stress Disorder (PTSD), e.g. Morrison, Frame, and Larkin (2003).

A number of epidemiological studies have begun to use a dimensional / non-categorical approach to psychopathology to explore the association between environmental indices of social adversity and specific psychotic symptoms and/or symptom dimensions. However, relatively few studies have employed this method to date, and the findings reported are complex. For example, in a recent study of first episode psychosis (FEP), the authors set out to test the hypothesis that the positive symptoms of psychosis would be associated with population density, an index they used as a proxy for urbanicity (Oher et al., 2014). Whilst the authors indeed found a positive association between population density and *hallucinatory* symptoms (Oher et al., 2014), they found no such association (contrary to their prediction) with paranoia. In contrast, in a study of 7,353 individuals sampled from the *general* population, the authors found a positive association between neighbourhood-level deprivation and paranoia, but *not* hallucinations (Wickham, Taylor, Shevlin, & Bentall, 2014). Further, both studies found that environmental risk factors predicted some negative symptoms *as well as* positive symptoms (Oher et al., 2014; Wickham et al., 2014). Taken together, these studies provide contradictory evidence as to a possible link between neighbourhood deprivation and specific symptoms within the positive symptom dimension, e.g. paranoia.

Thus, whilst there is growing evidence to suggest a role for environmental risk factors in psychosis, e.g. deprivation and population density, it is unclear to what extent these effects reflect *specific* causal pathways impacting on defined symptoms and/or symptom dimensions, e.g. positive symptoms (Bentall et al., 2012), versus more *general* effects operating on *multiple* symptom dimensions through a common mechanism. In the *general* case, individual risk factors are not associated with *defined* symptoms or symptom dimensions in a one-to-one mapping; instead, they contribute to *general* stress on the individual, e.g. through chronic activation of

bodily systems associated with the stress response, i.e. the nervous, endocrine and immune systems (Corcoran, Mujica-Parodi, Yale, Leitman, & Malaspina, 2002). With an accumulation of these stressors, some threshold is eventually reached, at which point the systems become dysregulated, and symptoms emerge. For example, according to more recent versions of the diathesis-stress model of psychosis (Nuechterlein & Dawson, 1984), an accumulation of environmental stressors triggers a dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis in individuals with predisposing vulnerabilities (Pruessner, Cullen, Aas, & Walker, 2017; van Winkel, Stefanis, & Myin-Germeys, 2008). Critically, in addition to its involvement in the stress response, the HPA axis has been linked to the regulation of dopamine transmission, a neurotransmitter that has been heavily implicated in a range of psychotic symptoms, although this a hotly contested area of research; see Howes, McCutcheon, Owen, and Murray (2017) for a recent review.

Another line of research that speaks to the *generality* versus *specificity* of symptom-stressor associations involves a recent factor analytic study of psychological symptoms in over 1000 participants, which uncovered a *general* psychopathology factor (factor p) that was associated with an increased propensity to develop *any* mental health condition (Caspi et al., 2014). According to the model developed therein, an accumulation of stressors over the course of a lifetime increases the risk of *many* symptoms, with psychosis simply reflecting an extreme outcome of a developmental progression in severity (Caspi et al., 2014). Consistent with this hypothesis, the authors note that many adverse experiences, e.g. childhood maltreatment, increase the risk of *multiple* psychiatric conditions, e.g. mood, anxiety, behavioural and substance-use disorders, *as well as* psychosis (Green et al., 2010; Scott, Smith, & Ellis, 2010; Varese et al., 2012). A similar argument might also be made for a number of environmental risk factors that have been linked to an array of psychiatric conditions, e.g. neighbourhood deprivation (Caspi et al., 2000; Kirkbride

et al., 2014; Schneiders et al., 2003; Wainwright & Surtees, 2004). Thus, the *generality* versus *specificity* of association between psychosis and environmental risk factors remains to be elucidated.

In order to explore the nature of the association between environmental risk factors and specific symptoms / symptom dimensions in psychosis, a secondary analysis was undertaken on a pre-existing data-set that explored psychotic symptoms in FEP patients presenting to services in the West London region. Specifically, psychotic symptom dimension scores were regressed on individual- and neighbourhood- level indices of deprivation, inequality and social capital using multi-level linear and logistic regression.

Aims

The over-arching aim of this paper was to determine the individual- level and neighbourhood- level social and socioeconomic predictors of psychotic symptoms / symptom dimensions in FEP. Specifically, the primary hypotheses tested were that: (H1) a significant proportion of variance in symptom levels would be explained by participants living in different neighbourhoods once individual- level demographic variables (age, gender and socioeconomic status) were controlled for (*test of environmental influence*), and (H2) that distinct symptom dimensions would exhibit independent patterns of association with neighbourhood- level environmental factors (*test of specificity).* In addition, in order to address existing inconsistencies in the literature (Oher et al., 2014; Wickham et al., 2014), the following more specific hypotheses were assessed in relation to defined symptoms and symptom dimensions: first, that indices of environmental adversity (high population density, deprivation, inequality and/or low social capital) would be associated with higher levels of positive symptoms (H3; *dimension level analysis*), particularly paranoia, i.e. persecutory delusions (H4; *item-level analysis*).

Methods

All data presented were gathered at baseline as part of the West London First Episode Psychosis study (WLFEP), which aimed to explore the clinical, cognitive and neuropsychological predictors of psychosis symptoms, illness trajectory and outcome in FEP; see Barnes et al. (2000) and Joyce et al. (2005) for example. Participants were recruited from individuals presenting to secondary care services, i.e. Community Mental Health Teams (CMHTs) and inpatient units, within the London boroughs of Ealing, Hammersmith and Fulham, Wandsworth, Kingston, Richmond, Merton, Sutton and Hounslow, between the years of 1998 and 2006. Individuals were *not* recruited from primary, tertiary or GP services. Participation was dependent on informed consent, such that the population sample does not represent a geographic cohort but a subset of all cases that presented to services.

As part of the WLFEP, information was obtained with consent (see Appendix 3) from participant case-notes and clinical interview, as well as interviews with the participants' carers and relatives, where possible. Data were gathered at the time of first presentation to psychiatric services and included basic demographic information, e.g. date of birth, gender, occupation and address, as well as responses / performance on a broad array of clinical, cognitive and neuropsychological assessments. Variables included in this study therefore represent a sub-set of available data, selected on the basis of identified research hypotheses.

For individuals to be included in the study they had to be 16 years or older, experiencing their first psychotic episode, and have a sufficient command of the English language to facilitate participation in the assessment process. In addition, they had to have received fewer than 12 weeks of antipsychotic medication.

Potential participants were initially screened using the World Health Organization Psychosis Screen (Jablensky et al., 1992), and subsequently diagnosed by two psychiatric research nurses trained in delivering a structured interview known as the diagnostic module of the Diagnostic Interview for Psychosis (Castle et al., 2006). A computer algorithm (OPCRIT 4 windows, MRC Social Genetic and Developmental Psychiatry Centre, n.d.) was then used to generate diagnoses according to multiple classification systems including the Diagnostic and Statistical Manual of Mental Disorders (3rd ed.; DSM-III; American Psychiatric Association, 1980) and International Statistical Classification of Diseases and Related Health Problems (10th ed.; World Health Organization, 2006). These were then converted into DSM-IV categories by cross-referencing with DSM-IV criteria (American Psychiatric Association, 1994).

Ethical approval was obtained from local ethics committees of all boroughs detailed above; see Appendix 4.

Individual- level measures

Psychological symptoms. Psychotic symptoms were assessed using the Scale for the Assessment of Positive Symptoms (SAPS) (Andreasen, 1990) and a revised version of the Scale for the Assessment of Negative Symptoms (SANS) (SANS-R). The SAPS is a 34-item clinician-administered questionnaire, which divides symptoms into five sub-scales (hallucinations, delusions, bizarre behaviour and positive formal thought disorder), each of which is also given a global symptom severity score by the rater. The purpose of the global ratings is to summarize the severity of all symptoms in the relevant subscale. Global ratings also take into consideration the functional impact of the symptoms on the participant and/or the contexts in which they are located (e.g. SAPS-B5: *'global rating of bizarre behaviour – this rating should reflect the type of and the extent to which it deviates from social*

norms'). The SANS-R is a 24-item clinician-administered questionnaire, which divides symptoms into four sub-scales (affective flattening or blunting, alogia, avolition-apathy, anhedonia-asociality, attention); these are also given a global score (e.g. SANS-22: '*global rating of anhedonia / asociality – this rating should reflect overall severity, taking into account the patient's age, family status, etc.'*). The SANS-R is identical to the full 25-item SANS (Andreasen, 1990), except that it does not include the item SANS-6 (inappropriate affect), which was removed as it was found to reduce the fit of a factor analytic model of the scale (Peralta & Cuesta, 1995). The measures have been validated in FEP (Peralta et al., 1999), and correlate well with other well-established psychotic symptom measures, e.g. the Positive and Negative Syndrome Scale for Schizophrenia (PANSS; r=0.71-0.84) (van Erp et al., 2014). Higher scores represent more severe symptoms.

Variables defining the time-course of symptom onset were included in the analysis since this has been shown to independently predict greater symptom severity (positive and negative) as well as poorer social function at one-year follow-up (Barnes et al., 2008). The duration of untreated psychosis (DUP) was calculated as the period from psychosis onset to the start of treatment, and the duration of untreated illness (DUI) was calculated as the DUP plus the duration of prodrome. These were estimated using the Nottingham Onset Scale (NOS), a short guided interview and rating schedule with high inter-rater (ICC=0.62-0.8) and test-retest reliability (ICC=0.67-0.89) (Singh et al., 2005).

To determine whether identified predictors were specific to psychotic symptoms or whether associations were also shared with affective symptoms (Wickham et al., 2014), a measure of mood / depression was also included in the analyses. Mood was assessed using the 10-item Montgomery–Åsberg Depression Rating Scale (MADRS) (Montgomery & Asberg, 1979) in the initial stages of the project; however,

at a later stage, a switch was made to using the more extensive 24-item Hamilton Rating Scale for Depression (HRSD) (Hamilton, 1960). Nonetheless, the MADRS was designed as an adjunct to the HRSD, and scores on the two measures are highly correlated (r=0.8-0.88) (Heo, Murphy, & Meyers, 2007). To standardize depression scores across measures they were re-coded as (1) absent, (2) mild, (3) moderate, and (4) severe according to published thresholds (Hamilton, 1960; Herrmann, Black, Lawrence, Szekely, & Szalai, 1998; Snaith, Harrop, Newby, & Teale, 1986).

Social function. A measure of individual-level social function was included in the study since it was hypothesized that a deficit in this area might limit the individual's capacity to benefit from ward- level social capital. Social function was assessed using the Social Function Scale (Birchwood, Smith, Cochrane, Wetton, & Copestake, 1990), a 76-item self-administered, structured questionnaire, specifically developed for use with people with schizophrenia. The SFS assesses social functioning in seven different domains: social engagement / withdrawal, interpersonal behaviour, pro-social activity, recreation, independence-competence, independence-performance and employment / occupation, and has good validity and reliability (mean test-retest reliability=0.72). Higher scores on the SFS indicate a higher level of social functioning.

Socioeconomic status. A measure of individual-level socioeconomic status (SES) was included in the analysis since various indices of SES have been shown to predict levels of mental health difficulties, including psychosis; see Werner, Malaspina, and Rabinowitz, (2006) for example. In addition, it is critical that a measure of individual-level SES be included, since without one, individual-level deprivation may confound the effects of putative ward- level effects such as neighbourhood deprivation (Kondo et al., 2009).

Whilst a measure of individual *income* was not available, a measure of individual-level *SES* was derived on the basis of occupational information gathered from each participant at assessment. Measures of SES / class are thought to be more stable indices of life-course earnings than income *per se*, since the latter has been shown to be subject to large fluctuations over time, a process known as income 'churning' (Jarvis & Jenkins, 1998, p.32). Participants were retrospectively assigned to one of five socioeconomic categories on the basis of their occupation using the National Statistics Socio-Economic Classification system (NS-SEC) (Rose & Pevalin, 2005), an approach that has been used previously (Kirkbride et al., 2008, 2010, 2015): (1) managerial and professional occupations, (2) intermediate occupations, (3) routine and manual occupations, (4) unemployed and (5) student. Assignment was undertaken using an online Occupation Code Search Tool, developed by the Office for National Statistics (ONS, 2016).

Neighbourhood- level measures

Each participant's postcode was used to identify the Census Area Statistics (CAS) ward in which they lived at first contact using databases produced by the ONS (Office for National Statistics, n.d.). The CAS ward is an intermediate geographical level at which UK census data is produced every ten years. CAS wards were created in 2003 from the statistical ward (itself based on electoral ward boundaries), except that smaller wards (<40 households or 100 residents in size) were merged with others in order to preserve confidentiality of the inhabitants (n=18 merged wards). In 2003 there were 8,850 CAS wards in England and Wales (7,932 in England alone).

Participants included in the study reported residencies from across 118 wards (median population: 10,068; interquartile range (IQR): 9,447-12,804) distributed across 15 boroughs. For each of these wards a number of indices of urbanicity,

deprivation, inequality and social capital were identified; unless specified otherwise, all these data were obtained from the 2001 census (Office for National Statistics, n.d.). Henceforth, the terms 'ward' and 'neighbourhood' will be used interchangeably to indicate CAS ward.

Urbanicity, deprivation and inequality. Following the work of others, e.g. Kaymaz et al. (2006), population density was used as a proxy for urbanicity.

Two indices of deprivation were included in the study: (i) income deprivation (ID): the percentage of individuals who were living in a household with an income of less than 60% of the median, and (ii) the Index of Multiple Deprivation (IMD), an aggregate measure of deprivation comprised of 37 indicators within seven domains (income deprivation, employment and deprivation, health deprivation and disability, education, skills and training deprivation, barriers to housing and services, living environment deprivation and crime) obtained from national surveys.

The ID and IMD were obtained from The English Indices of Deprivation (2004), data published by the Office of the Deputy Prime Minister (Noble et al., 2004). Since these data were available for nested geographical areas smaller than the CAS ward (lower layer super output areas (LSOA)), ward-level indices were calculated as the sum of composite LSOA values, with each weighted by its population size, as described previously (Kirkbride et al., 2014). High scores on the ID and IMD denote high levels of deprivation.

In addition, it was *also* possible to derive indices of *inequality* (i.e. the dispersion of deprivation). Thus, for both the ID and IMD, a corresponding Gini coefficient was calculated for each ward (GINI-ID and GINI-IMD), based on the distribution of deprivation across its composite LSOAs, as described previously by Kirkbride et al.

(2014). A Gini coefficient of zero represents perfect equality (indicating that all composite LSOAs are equally deprived), whereas Gini coefficients approaching one indicate maximum inequality. See Appendix 5 for full details on how Gini coefficients were calculated.

Social capital - cohesion and fragmentation. A number of measures of social fragmentation and social cohesion were also analysed since it is thought that the quality of social interactions within a geographical region may be protective against mental health difficulties, see De Silva, McKenzie, Harpham, and Huttly (2005) for a systematic review. Also, a lack of social capital may mediate the effects of income inequality and deprivation on health outcomes, including mental health (Fone et al., 2007; Kawachi & Kennedy, 1999).

To calculate an index of social fragmentation (SFI) a composite of four separate 2001 census measures were used, as described previously (Allardyce et al., 2005; Congdon, 1996): (i) the percentage of people who were aged 16 years or over and single, i.e. falling into one of the following categories: single (never married), separated (but still legally married) divorced or widowed; (ii) the percentage of households that were single-occupancy, i.e. resident to one person (under the age of 65) only; (iii) the percentage of households that were rented, irrespective of whether from a local authority, housing association or private landlord / letting agency; and (iv) the percentage of people who were mobile in the 12 months leading up to the census date, i.e. having moved into, out of, or within the area. For each ward these individual measures were Z-transformed and summed, thereby creating a single index of social fragmentation (SFI), with high scores indicating high social fragmentation. (Note: Z-score transformation was based on all wards included in the analysis rather than nationwide data).

As a proxy for social cohesion voter turnout during the 2002 local elections (percentage of the electorate who cast valid ballots) was obtained for each ward (Rallings & Thrasher, 2002) as described previously (Kirkbride et al., 2007; Oher et al., 2014). The reasoning behind this approach is that voter turnout reflects people's engagement with local- level issues and involvement with the community. Higher scores on this social cohesion index (SCI) reflect higher levels of social cohesion.

Ethnicity. Ethnic segregation was estimated using the Index of Dissimilarity (IDS), since high ethnic segregation has been shown to predict a low incidence of psychosis (Kirkbride et al., 2007). In addition, ethnic segregation might be thought of as a dimension of social capital, since fragmented communities are likely characterised by lower levels of trust and social cohesion (Tesei, 2014). The IDS, as applied here, measures the extent to which two populations are segregated within a target region (CAS ward) comprised of lower level areas (LSOAs). It is calculated using the following formula:

$$ID = \frac{1}{2}\sum_{i=1}^{n} \left| \frac{a_i}{A_T} - \frac{b_i}{B_T} \right|$$

where n is the total number of LSOAs in the ward, a_i is the number of individuals of ethnic group a in LSOAi, A_T is the *total* number of individuals of ethnic group a in ward$i$, b_i is the number of individuals of ethnic group b in LSOAi and B_T is the total number of individuals of ethnic group b in ward$i$. IDS scores therefore range between 0 (no segregation) to 1 (total segregation), with intermediate scores indicating the proportion of one of the two populations that would have to move for complete integration to occur. Note that as segregation within a ward increases, fragmentation *within* an ethnic group decreases.

The index of dissimilarity was calculated at the ward level, measuring the extent to which white and BME populations are segregated across Lower Output Areas (LSOAs) within each ward (IDS-BME). BME was defined as any ethnicity other than white, i.e. Asian or Asian British, Black or Black British, Chinese or Other, but not mixed ethnicities. In addition, BME ethnic density was calculated for each ward (DEN-BME) as the proportion of BME individuals relative to the total population in each ward.

Data collection

Participants' postcodes were manually transferred from library cards (stored in a data archive at the Institute of Cognitive Neuroscience) to an electronic database for further analyses. Individual postcodes were then manually checked using online resources (UK Data Service, n.d.), and coded as being either: (i) usable (U), if they were valid and fell within the study's catchment area (defined as any borough within the boundaries of the M25); (ii) non-specific (NS), if they were found to be too broad / covering multiple wards; (iii) non-applicable (NA), if they were found to be invalid or outside the study's catchment area. In addition, two participants were coded as having no fixed abode at the time of testing (NF). Any participants falling into these three latter categories (NS, NA or NF) were excluded from analysis (8.97% of the total data), since environmental predictors would be unavailable for these cases. See Figure 1.

Look up tables (LUT) were then downloaded from the UK Data Service, facilitating conversion of individual postcodes into CAS ward codes (The UK Data Service, n.d.).

Note: in order to preserve anonymity postcode data were kept separate from individual participant data at all stages; individual- and neighbourhood- level data

were only combined in a single database once the latter had been converted into ward- level indices such as inequality and deprivation, which could not facilitate identification of the individual.

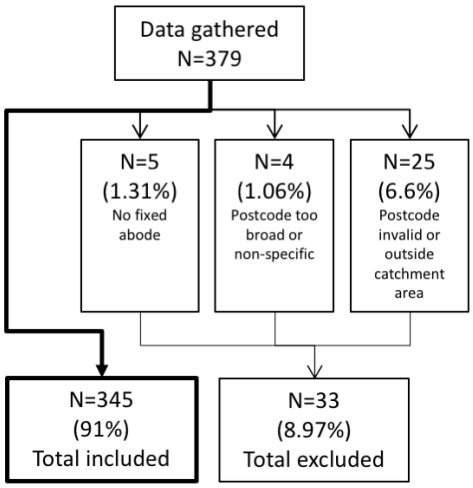

Figure 1 Data inclusion flow diagram. The number and percentage of participants for whom usable postcode data were included / available are shown alongside the number and percentage of participants who were excluded from analyses. Data were excluded if a participant had no fixed abode at the time of testing, or if they provided too broad / non-specific a postcode, i.e. one that encompassed multiple wards. Data were also excluded if recorded postcodes were invalid, fell outside the catchment area or else were otherwise invalid.

Analyses

Data extraction, e.g. from NOS and participant databases, integration / cross-referencing *between* databases and calculation of all ward- level predictors was undertaken using custom-written scripts generated in Matlab (MathWorks, Cambridge, MA), with the exception of the Gini coefficients, which had been calculated previously for all UK wards by a collaborator (JK) using Stata. All other analyses were undertaken in SPSS (version 22; SPSS Inc., Chicago, IL) or Stata (Version 14; StataCorp LP, College Station, TX).

Pre-processing. Data were pre-processed using a custom-written Matlab program. Extreme outliers (>3 Z-scores from the mean) were discarded from all ordinal and ratio data. In addition, outcome variables used in linear regression analyses were transformed in order to normalize the data; this entailed subjecting scores to square-root, cube-root, logarithmic and inverse transformations, with an optimal transformation selected as that which minimized skew. Where data were negatively skewed, these were reflected -by subtraction from the maximum value plus one- both before and after transformation, thereby preserving the sign of the variables' entries. Transformed scores were then re-expressed as standard scores (Z-transformed) in order to aid interpretation of analyses. Independent variables were *not* transformed.

Principal component analysis of symptom data. Participants' SAPS and SANS scores were subjected to a series of PCAs in order to derive symptom dimension scores to be used as outcome variables in the second stage of the analysis (multi-level regression). A two-step process was undertaken following the methods of Peralta et al. (1997), in which an item-level, first-order PCA was first undertaken on raw SAPS and SANS scores, followed by a second-order PCA on the principle component scores extracted from the first-order analysis. A PCA

approach was adopted since previous studies have highlighted that a simple dichotomy of positive and negative symptoms is not supported by analyses of the data; specifically, the positive symptoms can at least be broken down further to include a disorganized symptom dimension (John et al., 2003; Klimidis et al., 1993; Peralta & Cuesta, 1999; Peralta et al., 1997). A *second-order* PCA was deemed necessary, since previous studies have shown that a single item-level PCA of the SAPS and SANS typically generates a large number of components (Minas, Klimidis, Stuart, Copolov, & Singh, 1994; Victor Peralta et al., 1999), which would lead to an inflation of the probability of a type 1 error if all were independently tested for association with predictor variables.

For the first-order, item-level analysis, data from 49 individual items of the SAPS and SANS were included; the nine *global* rating scores included in the measure were not built into the model, since these exhibited high correlations with other items (some >0.9) and overlapped with individual item constructs, resulting in non-independence. Since PCA does not require variables to be normally distributed, these item-level data were not transformed prior to analysis. Principle components were extracted if they had an eigenvector value >1, since this is the most reliable method given the number of variables to be included in the model (Peralta et al., 1997). Principle component rotation was undertaken using an oblique rotation (OBLIMIN) method, since, on the basis of previous studies, e.g. Peralta et al. (1997), correlations were expected between symptom dimensions at this stage of analysis. In addition, this made sense clinically, since participants included in the study would be expected to present to services with symptoms characteristic of multiple symptom dimensions.

For the second-order PCA, which was undertaken on the principle component scores extracted from the first-order analysis, a VARIMAX rotation was used so that

orthogonal symptom dimensions might be identified for use as *independent* outcome variables in the multi-level linear regression.

Multi-level regression. A series of multi-level regression analyses were undertaken to determine individual and ward- level predictors of psychotic symptoms and depression scores (H1; *test of environmental influence*). Multi-level models are ideally suited to the research hypotheses as well as this specific data-set, for a number of reasons. First, the data are inherently clustered (individuals at the lower- level are clustered within wards at the higher- level), such that a multi-level model allows the variance in outcome attributed to this clustering structure to be ascertained. Second, multi-level models do not assume that *all* variance in outcome can be explained by lower- and higher- level *fixed* effects, but instead, include estimates of unmeasured variance (*random* effects) at the higher- level/s. In this example, random effects measure between-ward variation that is not captured by environmental predictors included in the model. Third, multi-level models do not suffer from several of the problems that would affect single-level analyses of the same data. For example, if individual- level data were instead aggregated to the neighbourhood- level, e.g. *mean* symptom scores for each ward regressed on ward-level environmental predictors, information about associations between individual-level measures would be lost (loss of within-group information) (Gelman & Hill, 2007). Alternatively, if ward- level data were disaggregated, e.g. individual participants coded for each of the ward- level predictors, this would fail to take into consideration the fact that individuals *within* a ward may be more similar to one another than individuals *between* wards, leading to a violation of the assumption of independence (Gelman & Hill, 2007).

To determine the relevant predictors of *overall* psychotic symptoms, the sum of SAPS and SANS global rating scores were calculated for each participant, and

treated as an outcome variable in a multi-level *linear* regression. Since the rating of the SAPS and SANS *global* ratings takes into consideration the functional impact of the symptoms as well as the contexts in which they occur, they are well suited to assessing *overall* symptom severity.

To determine the predictors of *specific* symptom dimensions (H2; *test of specificity* and H3; *dimension level analysis*), principle component scores derived from the second-order symptom- level PCA were used as outcome variables in a series of multi-level *linear* regressions. This approach was deemed most appropriate since symptom- level PCAs retain the richness of the full symptom data-set, and make no prior assumptions about the clustering of –and associations between- individual symptoms.

In order to explore the predictors of paranoia (H4; *item- level analysis*), participants' responses on the persecutory delusions item of the SAPS (SAPS-D1) were regressed on individual- and neighbourhood- level variables. Since the distribution of participants' responses for item SAPS-D1 could not be normalized through transformation, they were instead re-coded as a binary variable, with scores of 0-2 coded as absent-to-mild and scores of 3-5 coded as moderate-to-severe paranoia. Multi-level *logistic* regression was used, since *linear* regression cannot be used with a categorical outcome variable.

Finally, the predictors of depression were also explored using multi-level *logistic* regression, since depression scores also could not be normalized. Depression scores were instead re-coded as a binary variable, with scores of 0-2 coded as absent-to-mild and scores of 3-5 coded as moderate –to-severe depression.

For each of the regression analyses (linear and logistic), a null multi-level model was run first to determine the proportion of variance explained by ward- level random effects. Basic demographic information (age at assessment, sex and SES) were then added as potential *a priori* confounders. Additional individual-level predictors were then assessed for inclusion in a multistage-stage process using forward step-wise selection. First, predictors were discarded if they did not independently improve the model ($p>0.05$); this was assessed using a likelihood ratio test (LRT), which uses maximum likelihood estimation (MLE) to assess the extent to which the fit of a model is improved by adding parameters, whilst penalizing the more complex model for the resulting increase in degrees of freedom (Lewis, Butler, & Gilbert, 2011). Remaining predictors were then *sequentially* added to the model according to the strength of their association with the outcome variable (assessed using Akaike's Information Criterion (AIC)) and retained if they significantly increased the percentage of variance explained (LRT) ($p<0.05$).

Ward-level social / socioeconomic predictors were then added to the model using this same two-stage process, with the exception that the individual level predictors were already in place (as *a priori* confounders). Ward- level fixed effects were assessed irrespective of whether or not ward- level random effects were found to be significant (relative to a null model), since a previous study using Monte Carlo simulation of multi- level data has shown, that whilst estimates of higher- level fixed effects tend to be unbiased and robust to changes in sample size, estimates of higher- level random effects are comparatively unreliable, and tend to be biased towards under-estimation (Bryan & Jenkins, 2016). This is particularly the case when sample sizes at the higher- (i.e. ward-) level are small.

Following the methods of Oher et al. (2014), all regression analyses were re-run with scores from the *other* symptom measures included; i.e. depression scores and

symptom dimension scores (positive, negative and disorganized) were added simultaneously to the model. This approach was used to test for confounding effects due to inter-correlations between symptom dimensions, and further, to facilitate a more stringent test of specificity of association. Thus, if a given ward- level predictor is *specifically* and *uniquely* associated with a given symptom dimension, one would expect the association to hold after controlling for scores on *other* symptom dimensions.

Finally, where ward- level predictors were found to predict significant variance in a symptom dimension, the relevant regression analysis was *re*-run with *absolute* deprivation (ID / IMD) included in the model. This was undertaken in order to test for the confounding effects of *absolute deprivation* in indices such as inequality and ethnic density, which might be expected to co-vary; see Jivraj and Khan (2013) for example.

The principle assumptions of linear regression are: (i) additivity and linearity, i.e. the outcome variable can be expressed as a linear combination of predictor variables, (ii) independence of errors, (iii) homoscedasticity, i.e. the variance in residuals does not differ as a function of the predictor values, and (iv) normal distribution of residuals (Gelman & Hill, 2007).

Linearity was assessed by eye, with individual predictors plotted against the outcome variable in order to detect any large deviations from linearity. In addition, where a deviation from linearity might be thought to exist on an *a priori* theoretical basis, for example, indices of health are often found to decline at an accelerating rate as a function of age: i.e. following age^2 rather than age, selection was made on the basis of which version of the variable gave the best fit, determined using the AIC test. With respect to independence of errors, as discussed above, the multi-level

approach allows for violations of independence, e.g. similarities between participants' scores *within* a ward, by modelling the inherently clustered structure of the data (Gelman & Hill, 2007)

Normality of residuals was assessed using the one-sample Kolmogorov-Smirnov test. Homoscedasticity was assessed using the Breusch–Pagan test (Breusch & Pagan, 1979), which regresses squared residuals from the regression model on the predictor variables; a significant p value (<0.05) indicates that the null hypothesis of homoscedasticity can be rejected and implicates a systematic association between at least one of the predictor variables and the squared residuals. This was run using custom-written code in Stata.

The principle assumptions of logistic regression are the following: (i) low levels of multi-collinearity, i.e. predictor variables should not be highly correlated; (ii) a linear relationship between the natural log odds of the outcome variable and the predictor variables; (iii) independence of errors and (iv) a large sample size, e.g. >30 cases per predictor (Stoltzfus, 2011).

Collinearity -assumption (i)- was assessed *post hoc* through bivariate correlation, with any large correlations between variables included in the model (Spearman's Rho (r_s)>=0.5) reported. Assumption (ii) was assessed using the Box-Tidwell test. This was executed in Stata using the 'boxtid' function, which was downloaded from an online bibliographic database (Royston, n.d.). As described, the assumption of independence of errors could be disregarded as the spatial clustering of data was modelled, and finally, the sample size used here clearly met the last assumption.

A number of variables had missing cases. In order to retain sufficient power and minimize the risk of introducing bias, therefore, any predictors for which >10% of the

cases were missing were discarded from analyses. As a result of missing cases in retained predictors however, the final number of observations differed slightly in each regression model. To ensure that predictor value parameters were not biased by missing cases, each variable included in the multi-level model was exposed to a t-test (ordinal / ratio data) or Chi-square test (nominal data), comparing the total data-set (all cases for that variable) to the reduced data-set retained in the model. In addition, all primary analyses were re-run using a *complete case* analysis approach, i.e. all data from any participant without a full data-set, e.g. missing a single score for one measure, were discarded.

Any violations of the criteria reported are discussed within the relevant results section and implications for interpretation discussed.

For the linear regression analyses, the reported coefficients and their estimated 95% confidence intervals indicate the change in outcome variable (expressed as units of standard deviation) associated with a unit change in predictor.

In order to aid interpretation, the output of logistic regression analyses is provided in the form of odds ratios (OR). For a categorical predictor, the OR represents the probability that an 'event' will occur, for example, that a participant will have depression, for a given 'exposure', e.g. they are female, compared to the odds of the same 'event' occurring given an alternative 'exposure', e.g. they are male (Szumilas, 2010). For a *non*-categorical variable, the OR represents the odds ratio associated with a one-unit increase in the predictor variable, for example, the relative probability that the participant is depressed at 23, compared to the probability that they are depressed at 22 years of age.

Comparing test data to national average data. In order to characterize the study region, analyses were undertaken on each environmental variable, e.g. deprivation and inequality, comparing scores for *each* unique ward included in the analyses (n=118) against national data. In other words, the following question was asked for each ward- level predictor: are scores in the study catchment area typical of the pattern seen across England as a whole? To facilitate statistical comparison, for each higher- level variable, a random sample of 118 scores were taken from the nationwide pool of wards (n=7,932 in England). These two data-sets were then compared using the Wilcoxon Signed-Rank test, which makes no assumptions about the underlying distribution of the data.

Results

Missing and excluded data

A number of cases were missing for variables included in the final analyses (Table 1, column 9). This was due to missing entries at the data collection stage (Table 1, column 3) as well as extreme outliers excluded during the pre-processing stage (Table1, column 6). For each individual- level variable, the number and percentage of entries *missing* from the final data-set were: age at assessment (n=6 of 345, 1.74%), age at onset (n=17, 4.93%), DUP (n=18, 5.22%), gender (n=2, 0.58%), NS-SEC (n=3, 0.87%), diagnosis (n=6, 1.74%), SFS (n=26, 7.54%), SAPS (n=9, 2.61%), SANS (n=3, 0.87%) and depression symptom scores (n=8, 2.32%). With respect to neighbourhood-level variables (Table 2, column 8), the only indices for which one or more entries were missing were: SCI (n=3, 2.54%), IDS-BME (n=2 of 118, 1.69%) and DEN-BME (n=2 of 118, 1.69%). As a result, a *complete* data-set was only available for 255 of 345 participants; these data were used for the complete case analyses.

Individual level data - descriptive analyses

The ratio of male to female participants in the data-set was 1.96 (227 males to 116 females; two unrecorded). The median age of participants at assessment was 24 years with a median age of onset of 23 years, reflecting a median duration of untreated psychosis (DUP) of 12 weeks (Table 1). Of the 345 participants included, 265 (76.81%) had diagnoses of non-affective psychoses, 74 (21.45%) had diagnoses of affective psychoses and six (1.74%) were not sub-classified. Due to the relatively low number of individuals with affective psychoses, analyses were not undertaken separately as a function of diagnosis type. The median SAPS and SANS total scores were 32 (maximum score = 150) and 18 (maximum score = 100), respectively. The median depression score was two, i.e. in the mild range.

Table 1 Key statistics for individual- level variables. Statistics provided include the number of cases (N), the median and Z-score skew (Skew Z) before and after removal of extreme outliers, where applied (raw and processed data, respectively). In addition, the number (n) and percentage of entries missing for each variable in the final data-set are also included (missing or excluded data). Age at Ax=age at assessment; NS-SEC= National Statistics Socio-Economic Classification system; DUP=duration of untreated psychosis (in weeks); SFS=social functioning scale; SAPS= Scales for the Assessment of Positive symptoms; SANS= Scales for the Assessment of Negative symptoms.

		Raw data			Processed data			
Variable	Level	N	Median (range)	Skew Z	N	Median (range)	Skew Z	Missing or excluded n (%)
Age at Ax		343	24.06 (16-63.53)	12	339	24(16-53.87)	9.67	6 (1.74)
Age at Onset		335	23 (11-63)	12.53	328	22 (11-48)	8.95	17 (4.93)
DUP		339	12 (0-504)	24.41	327	12 (0-288)	18.35	18 (5.22)
Gender	All	343	-	-	-	-	-	2 (0.58)
	Male	227	-	-	-	-	-	-
	Female	116	-	-	-	-	-	-
NS-SEC	All	342	-	-	-	-	-	3 (0.87)
	Managerial and professional	18	-	-	-	-	-	-
	Intermediate occupations	22	-	-	-	-	-	-
	Routine and manual	53	-	-	-	-	-	-
	Student	197	-	-	-	-	-	-
	Unemployed	52	-	-	-	-	-	-
Diagnosis	All	339	-	-	-	-	-	6 (1.74)
	Affective	74	-	-	-	-	-	-
	Non-affective	265	-	-	-	-	-	-
SFS		321	112.43 (65.86-135.21)	-3.6	319	112.43 (82.64-135.21)	-1.63	26 (7.54)
SAPS total		336	32 (0-85)	3.97	-	-	-	9 (2.61)
SANS total		342	18 (0-81)	6.02	-	-	-	3 (0.87)
Depression		337	2 (1-4)	1.74	-	-	-	8 (2.32)

Neighbourhood level data - descriptive and correlational analyses

The 345 participants included in the analyses were distributed across 118 wards. Of these, 325 reported residencies within the boroughs from which they were recruited (n=8 boroughs), and 20 reported residencies within seven surrounding boroughs (Harrow, Westminster, Kensington and Chelsea, Lambeth, Lewisham, Croydon, Elmbridge). (See Figure 2 and Appendix 6, Supplementary Table 2). The mean number of LSOAs per ward was 7.11 (std=1.45; range=2-11), and the mean number of participants per ward was 2.9 (std=2.08). Comparing these 118 wards to national data, i.e. all wards in England, the study region was characterised by higher population density / urbanicity ($Z=10.04$, $p<0.001$), higher social fragmentation ($Z=8.7$, $p<0.001$), higher BME ethnic density ($Z=11$, $p<0.001$) and lower BME ethnic segregation ($Z=-4.31$, $p<0.01$) (Table 2). The region did *not* differ with respect to deprivation (ID and IMD; $ps>0.05$) or inequality (GINI-ID and GINI-IMD; $ps>0.05$). Social cohesion, as measured by voter turnout was extremely close to the national average, 33.61% compared to 33.3%, respectively.

In order to characterise the nature of these wards further, the pattern of associations between key measures was explored using simple bivariate correlations. Rather than correlate data at the participant level, which would lead to replication of data points in the case of individuals living in the same neighbourhood, and hence an artificial inflation of power, data were analysed at the level of the neighbourhood itself, i.e. n=188 unique wards / data-points included per measure.

Analyses (Table 3) indicated that, even after *Bonferroni* correction for nine multiple comparisons (corrected alpha=0.006), neighbourhoods characterised by higher levels of *absolute* deprivation (IMD) were associated with higher social fragmentation (Spearman's Rho (r_s)=0.3, $p<0.001$), lower social cohesion (r_s =-0.59, $p<0.001$), higher levels of ethnic segregation (r_s=0.29, $p<0.006$) and a higher density

of ethnic minorities (r_s =0.76, p<0.001). However, they were also associated with *lower* levels of inequality (r_s=-0.36, p<0.001).

Further, the results show that, areas characterised by *higher* inequality (GINI-IMD), were associated with *lower* levels of absolute deprivation (r_s=-0.36, p<0.001), lower social fragmentation (r_s=-0.28, p<0.006), higher social cohesion (r_s=0.44, p<0.001) and a lower density of ethnic minorities (r_s=-0.3, p<0.001).

Figure 2 Study catchment area. Participants were recruited from services within the London Boroughs of Ealing, Hammersmith and Fulham, Wandsworth, Kingston, Richmond, Merton, Sutton and Hounslow (shaded dark grey). Participants reported residencies from these boroughs as well as seven surrounding boroughs however (shaded light grey). H&F=Hammersmith and Fulham; K&C=Kensington and Chelsea; TH=Tower Hamlets; B&D=Barking and Dagenham; WF=Waltham Forest.

Table 2 Key statistics for ward- level variables (N=118 unique wards included in the analysis). Statistics provided include the number of entries (N), the median, range and Z-score skew (Skew Z) before and after removal of extreme outliers (raw and processed data, respectively); in addition, the number (n) and percentage of entries missing for each variable in the final data-set are included. The same data are also shown for all wards in England (N=7932) for comparison, or else, all wards with more than one composite LSOA (N=6685) where this was required to calculate an index (e.g. GINI-ID). The outcome of a series of Wilcoxon Signed-Rank tests are also shown, comparing study data to a random sample of 118 wards taken from the national-level data. Population density=general population density in people per hectare; ID=income deprivation; IMD=index of multiple deprivation; GINI-ID=Gini coefficient based on income deprivation; GINI-IMD=Gini coefficient based on index of multiple deprivation; SFI=social fragmentation index; SCI=social cohesion index; IDS-BME=index of dissimilarity for BME versus white populations; DEN-BME=BME ethnic density (people per hectare).

	Study data (raw)			Study data (processed)			Missing or excluded data	National data			Wilcoxon Signed-Rank Test	
	N	Median (range)	Skew Z	N	Median (range)	Skew Z	n (%)	N	Median (range)	Skew Z	Z	p
Pop density	118	63.9 (10.45-236.05)	6.31	116	63.7 (10.45-202.30)	5.29	2 (1.69)	7932	12.01 (0.02-236.05)	89.65	10.04	<0.001
ID	118	11.13 (2.63-31.5)	2.81	118	11.13 (2.63-31.5)	2.81	0 (0)	7932	8.66 (1.03-61.71)	60.52	2.42	>0.05
IMD	118	16.75 (3.04-41.47)	2.2	118	16.75 (3.04-41.47)	2.2	0 (0)	7932	14.68 (1.1-79.81)	51.48	0.53	0.52
GINI-ID	118	0.24 (0.06-0.52)	2.37	118	0.24 (0.06-0.52)	2.37	0 (0)	6685	0.21 (0-0.57)	8.57	2.17	0.08
GINI-IMD	118	0.17 (0.04-0.37)	2.88	118	0.17 (0.04-0.37)	2.88	0 (0)	6685	0.17 (0-0.65)	14.83	0.43	0.52
SFI	118	4.83 (-3.42-12.27)	0	118	4.83 (-3.42-12.27)	0	0 (0)	7932	-0.83 (-6.7-22.48)	59.59	8.7	<0.001
SCI	115	33 (23.3-49.2)	1.19	115	33 (23.3-49.2)	1.19	3 (2.54)	7932	33.3	-	-	-
IDS-BME	118	0.13 (0.04-0.36)	4.01	116	0.13 (0.04-0.3)	2.33	2 (1.69)	6685	0.21 (0-0.79)	33.53	-4.31	<0.001
DEN-BME	118	0.17 (0.04-0.86)	8.34	116	0.17 (0.04-0.71)	7	2 (1.69)	7932	0.01 (0-0.86)	144.93	11	<0.001

Table 3 Spearman's correlations between ward-level variables (n=118 unique wards). Significant correlations at an alpha of 0.006 (corrected for nine comparisons) are shown in bold. GINI-IMD=Gini coefficient calculated for the index of multiple deprivation; SFI=social fragmentation index; SCI=social cohesion index (voter turnout); IDS-BME=index of dissimilarity with respect to BME versus white populations; DEN-BME=BME ethnic density.

	GINI -IMD	SFI	SCI	IDS-BME	DEN-BME
IMD	**-0.36 (<0.001)**	**0.3 (<0.001)**	**-0.59 (<0.001)**	**0.29 (<0.00)**	**0.76 (<0.001)**
GINI-IMD	-	**-0.28 (<0.006)**	**0.44 (<0.001)**	0.05 (0.58)	**-0.3 (<0.001)**

First-order principal component analysis of the SAPS and SANS

A first-order item-level PCA was undertaken on raw symptom scores. A number of checks were undertaken to determine the suitability of a PCA analysis, i.e. data factorability. Inspection of the correlation matrix indicated that 40 out of the 49 individual variables initially included in the analysis showed a medium sized correlation or greater (i.e. $r>0.3$) with at least one other variable. Further, no excessively large correlations ($r>0.9$) were seen. However, three values on the diagonals of the anti-image correlation matrix were <0.5, indicating items that were insufficiently correlated with extracted components to warrant inclusion in the analysis. Consequently, these three items were excluded from the analysis (SAPS-D1 – persecutory delusions; SAPS-D3 – delusions of sin or guilt and SAPS-D6 - somatic delusions) and the analysis was rerun without them.

Finally, a Kaiser-Meyer-Olkin value of 0.86 was obtained (above the recommended value of 0.5 (Kaiser, 1974)), indicating an adequate sample size, and Bartlett's Test of Sphericity was found to be significant ($\text{chi-squared}_{(1035)}=7267.99$, $P<0.001$). Consequently, a PCA was deemed appropriate for this data-set.

The first-order item-level PCA resulted in the extraction of 11 components with eigenvectors >1, which together explained 63.15% of the variance in the data (compared to Peralta and Cuesta (1999), who extracted 12 components that explained 66% of the total variance). Table 4 shows the PCA structure with variable loadings. Variables with component loadings >0.4 are presented in full contrast. The majority of the components extracted were easy to interpret. Naming of each component is based both on the size and nature of item loadings.

The first component (negative symptoms) includes 11 items from the SANS: SANS-1 (unchanging facial expression), SANS-2 (decreased spontaneous movement),

SANS-3 (paucity of expressive gestures), SANS-4 (poor eye contact), SANS-5 (affective non-responsiveness), SANS-7 (lack of vocal inflections), SANS-9 (poverty of speech), SANS-11 (blocking), SANS-12 (increased latency of response), SANS-16 (physical anergia) and SANS-21 (relationships with friends and peers).

The second component (thought disorder) included all items from the domain of positive formal thought disorder, i.e. SAPS-P1-P8, which includes derailment, tangentiality, incoherence (i.e. word salad or schizophasia), illogicality, circumstantiality, pressure of speech, distractible speech and clanging. The component also included a number of items from the SANS: SANS-10 (poverty of speech content), and SANS-24 (inattentiveness during testing).

The third component (delusions) included six items from the domain of delusions, including SAPS-D7 (delusions of reference), SAPS-D8 (delusions of being controlled), SAPS-D9 (mind-reading), SAPS-D10 (thought broadcasting), SAPS-D11 (thought insertion), SAPS-D12 (thought withdrawal).

The fourth component (social dysfunction) included eight items from the apathy / avolition, anhedonia / asociality and attention domains. These included SANS-S14 (grooming and hygiene), SANS-S15 (impersistence at work or school), SANS-S16 (physical anergia), SANS-S18 (recreational interests and activities), SANS-S19 (sexual interest and activity), SANS-S20 (ability to feel intimacy and closeness), SANS-S21 (relationships with friends and peers) and SANS-S23 (social inattention).

The fifth component (bizarre behaviour) was comprised of two items from the bizarre behaviour domain: SAPS-B2 (social and sexual behaviour) and SAPS-B3 (aggressive and agitated behaviour).

The sixth component (auditory hallucinations) was comprised of SAPS-H1 (auditory hallucinations), SAPS-H2 (voices commenting) and SAPS-H3 (voices conversing), as well as SAPS-D10 (delusions of thought broadcast).

The seventh component (grandiose delusions) was comprised of SAPS-D4 (grandiose delusions), SAPS-D5 (religious delusions), but also showed a loading from the item SAPS-B2 (social and sexual behaviour).

The eighth component (other hallucinations) was comprised of SAPS-H4 (somatic or tactile hallucinations), SAPS-H5 (olfactory hallucinations) and SAPS-H6 (visual hallucinations).

The ninth component was comprised of only a single loading, SAPS-D2 (delusions of jealousy).

The tenth component (alogia) was comprised of loadings from all 4 items from the alogia domain, SANS-9 (poverty of speech), SANS-10 (poverty of content of speech), SANS-S11 (blocking) and SANS-S12 (increased latency of response), as well as the two items from the attention domain, SANS-23 (social inattentiveness) and SANS-24 (inattentiveness during testing).

Finally, the eleventh component (other bizarre behaviour) included items from several domains across both the SAPS and SANS (bizarre behaviour, positive formal thought disorder and avolition / apathy). Thus, individual loadings were observed from SAPS-B1 (clothing and appearance), SAPS-B4 (repetitive or stereotyped behaviour), SAPS-P3 (incoherence) and SANS-14 (grooming and hygiene).

Table 4 Component loadings (structure) matrix for all 45 variables included in the first-order PCA. Loadings >0.4 in magnitude are shown in full contrast.

			F1	F2	F3	F4	F5	F6	F7	F8	F9	F10	F11
Hallucinations	SAPS-H1	Auditory	0.03	-0.08	0.29	-0.10	-0.09	-0.81	0.04	0.23	-0.09	0.04	0.10
	SAPS-H2	Voices commenting	0.07	-0.01	0.29	-0.15	-0.04	-0.84	0.01	0.23	0.08	0.01	-0.03
	SAPS-H3	Voices conversing	0.08	-0.03	0.27	-0.09	-0.09	-0.82	-0.09	0.14	0.06	0.05	-0.02
	SAPS-H4	Somatic or Tactile	-0.12	-0.02	0.17	-0.03	-0.36	-0.38	-0.25	0.45	0.11	0.07	-0.08
	SAPS-H5	Olfactory	-0.02	0.02	0.30	-0.06	0.03	-0.14	-0.03	0.72	0.11	0.01	0.00
	SAPS-H6	Visual	0.01	0.03	0.14	-0.01	-0.08	-0.28	-0.04	0.75	0.03	-0.02	0.00
Delusions	SAPS-D2	Jealous	-0.02	-0.05	0.14	0.01	0.06	-0.04	0.03	0.12	0.77	-0.01	-0.06
	SAPS-D4	Grandiose	-0.15	0.21	0.10	0.12	0.30	0.01	-0.69	0.01	0.07	-0.06	-0.08
	SAPS-D5	Religious	-0.07	0.03	0.14	0.09	-0.09	-0.01	-0.79	0.05	-0.09	-0.04	-0.03
	SAPS-D7	Of reference	0.00	0.02	0.45	-0.14	-0.10	-0.25	-0.25	-0.05	0.33	0.13	0.34
	SAPS-D8	Of being controlled	0.04	0.07	0.65	-0.11	-0.25	-0.22	-0.32	0.21	0.05	0.15	0.03
	SAPS-D9	Of mind reading	0.05	0.11	0.69	-0.05	-0.11	-0.17	-0.13	0.10	0.25	0.11	0.08
	SAPS-D10	Thought broadcasting	0.05	0.07	0.72	-0.05	0.03	-0.43	-0.09	0.22	0.13	0.19	-0.01
	SAPS-D11	Thought insertion	0.10	-0.04	0.72	-0.03	-0.07	-0.32	-0.09	0.20	0.00	0.05	-0.10
	SAPS-D12	Thought withdrawal	0.13	0.12	0.76	-0.08	-0.01	-0.20	-0.01	0.28	0.01	-0.03	-0.06
Bizarre behaviour	SAPS-B1	Appearance	-0.09	0.11	-0.14	0.05	0.33	-0.09	-0.34	-0.07	0.05	-0.06	-0.59
	SAPS-B2	Social / sexual	0.05	0.19	0.00	0.03	0.51	0.10	-0.42	0.16	0.03	0.08	-0.18
	SAPS-B3	Aggressive / agitated	-0.02	0.01	-0.10	0.07	0.77	0.06	-0.01	-0.08	0.02	0.03	-0.08
	SAPS-B4	Repetitive / stereotyped	0.16	0.13	0.30	-0.05	-0.19	0.14	0.00	0.25	0.07	0.19	-0.52
Positive formal thought disorder	SAPS-P1	Derailment	0.12	0.85	0.10	-0.15	0.01	0.04	-0.07	0.02	-0.02	0.29	-0.13
	SAPS-P2	Tangentiality	0.15	0.83	0.05	-0.24	-0.02	0.01	-0.05	-0.05	0.06	0.31	-0.16
	SAPS-P3	Incoherence	0.13	0.46	0.15	-0.13	0.10	-0.05	0.01	-0.09	0.02	0.15	-0.57
	SAPS-P4	Illogicality	0.19	0.79	0.18	-0.24	0.10	0.05	-0.04	0.10	-0.05	0.24	-0.19
	SAPS-P5	Circumstantiality	0.17	0.77	0.15	-0.21	-0.03	0.00	-0.18	0.03	-0.02	0.16	-0.09
	SAPS-P6	Pressure of Speech	-0.19	0.59	-0.20	0.04	0.25	0.20	-0.21	-0.02	-0.08	-0.20	-0.21
	SAPS-P7	Distractible Speech	0.03	0.61	0.06	-0.03	0.35	0.04	-0.06	0.08	-0.24	0.24	-0.08
	SAPS-P8	Clanging	0.10	0.61	0.16	-0.15	0.19	0.07	-0.02	0.30	-0.31	0.00	-0.12

			F1	F2	F3	F4	F5	F6	F7	F8	F9	F10	F11
Affective flattening / blunting	SANS-1	Facial expression	0.88	0.09	0.08	-0.37	-0.06	-0.02	0.14	-0.05	-0.09	0.32	-0.03
	SANS-2	Spontaneous movements	0.86	0.12	0.13	-0.27	-0.07	-0.07	0.05	-0.02	-0.13	0.19	-0.09
	SANS-3	Expressive gestures	0.93	0.12	0.06	-0.36	-0.03	0.00	0.09	-0.01	-0.10	0.28	-0.07
	SANS-4	Eye contact	0.56	0.04	0.01	-0.24	0.09	-0.06	0.08	0.01	-0.08	0.37	-0.29
	SANS-5	Nonresponsiveness	0.87	0.17	0.13	-0.35	0.07	-0.02	0.13	0.07	-0.02	0.29	-0.11
	SANS-7	Vocal Inflections	0.87	0.05	0.15	-0.36	0.02	-0.08	0.12	0.02	-0.02	0.30	-0.10
Alogia	SANS-9	Poverty of speech	0.72	-0.07	0.00	-0.19	0.05	-0.11	0.22	0.03	-0.15	0.57	0.00
	SANS-10	Poverty of speech content	0.36	0.52	0.01	-0.23	-0.05	0.07	0.00	0.05	-0.09	0.53	-0.12
	SANS-11	Blocking	0.55	0.26	0.26	0.03	0.07	0.02	0.15	0.15	-0.12	0.64	-0.01
	SANS-12	Latency of response	0.68	0.18	0.12	-0.04	0.05	-0.08	0.10	0.06	-0.19	0.61	0.04
Avolition / apathy	SANS-14	Grooming / hygiene	0.34	0.13	0.00	-0.53	0.26	-0.05	-0.07	-0.09	-0.36	0.27	-0.43
	SANS-15	Impersistence	0.23	0.24	0.11	-0.68	0.23	-0.09	-0.11	-0.02	-0.17	0.25	0.04
	SANS-16	Physical anergia	0.52	0.14	0.16	-0.63	-0.06	-0.15	0.02	0.00	-0.34	0.27	-0.06
Anhedonia / asociality	SANS-18	Recreational interest / activity	0.35	0.10	0.08	-0.70	-0.19	-0.20	0.21	-0.03	-0.19	0.18	-0.01
	SANS-19	Sexual interest / activity	0.25	0.08	0.03	-0.64	-0.25	-0.15	0.09	0.15	0.00	-0.04	0.04
	SANS-20	Ability to feel intimacy	0.31	0.18	0.12	-0.75	-0.06	-0.04	0.19	0.04	0.21	0.20	-0.17
	SANS-21	Relationships	0.45	0.24	0.06	-0.81	-0.02	-0.05	0.16	0.07	0.14	0.32	-0.08
Attention	SANS-23	Social inattentiveness	0.29	0.35	0.13	-0.45	0.10	0.00	0.02	-0.04	-0.02	0.73	-0.16
	SANS-24	Inattentiveness during testing	0.39	0.44	0.04	-0.39	0.10	0.02	0.03	-0.02	-0.09	0.66	-0.29

Second-order principal component analysis of the SAPS and SANS

With respect to the second-order item-level PCA, a number of checks were again undertaken to determine the factorability of the data-set. First, inspection of the correlation matrix indicated that all individual variables included in the analysis showed a small sized correlation or greater (i.e. r>0.1) with at least one other variable, and three out of 11 showed a medium sized correlation or greater (i.e. r>0.3). Further, no excessively large correlations (r>0.9) were seen. All values on the diagonals of the anti-image correlation matrix exceeded 0.5, indicating items were sufficiently correlated with extracted components to warrant inclusion in the analysis. Finally, a Kaiser-Meyer-Olkin value of 0.59 was obtained (above the recommended value of 0.5 (Kaiser, 1974)), indicating an adequate sample size, and Bartlett's Test of Sphericity was found to be significant ($chi\text{-}squared_{(55)}$=209.2, P<0.001). Consequently, a PCA was deemed appropriate for this data-set.

Three components were extracted with eigenvectors >1, which together explained 41.46% of the variance in the data (compared to Peralta and Cuesta (1999), who extracted four components that explained 54% of the total variance). Table 5 shows the PCA structure with variable loadings.

In order to aid interpretation of Table 5, the sign of the coefficients for components 4, 6, 7 and 11 (social dysfunction, auditory hallucinations, grandiose delusions and other bizarre behaviour, respectively) have been inverted. This is appropriate, since in the first-order PCA, the loadings associated with these four components were negative, i.e. of a different sign to the other components.

The first component (negative symptoms), which explained 16.39% of the variance, showed loadings from the negative symptoms, social dysfunction and alogia components derived from the first-order PCA.

The second component (positive symptoms), which explained 13.33% of the variance, showed loadings from delusions, auditory hallucinations and other hallucinations components derived from the first-order PCA.

Finally, the third component (disorganized symptoms), which explained 11.74% of the variance, showed loadings from thought disorder, bizarre behaviour, grandiose delusions and other bizarre behaviour components derived from the first-order PCA.

These three components closely resembled the first three components extracted by Peralta and Cuesta (1999), as well as those described previously by others (Dollfus et al., 1993; Dollfus & Petit, 1995; John et al., 2003; Klimidis et al., 1993; Peralta et al., 1997). In order to assess the robustness / reproducibility of these findings a third PCA was undertaken on the global symptom severity scores from the SAPS and SANS. This followed the methods of John et al. (2003), and also generated a stable three-component solution that could be described as positive, negative and disorganized symptoms (see Appendix 7, Supplementary Table 3). The stability of these findings indicates the suitability of using symptom dimensions derived from the PCAs as outcome variables for the regression analyses.

Table 5 Component loadings (rotated matrix) for all 11 variables included in the second-order PCA. The coefficients of components 4, 6, 7 and 11 have been inverted to aid interpretation; see main text for explanation. Loadings >0.4 in magnitude are shown in full contrast.

	F1	F2	F3
(1) Negative Symptoms	0.78	-0.02	-0.01
(2) Thought Disorder	0.29	0.04	0.62
(3) Delusions	0.16	0.72	0.05
(4) Social dysfunction	0.63	0.10	0.00
(5) Bizarre behaviour	-0.08	-0.25	0.57
(6) Auditory hallucinations	0.09	0.62	-0.17
(7) Grandiose delusions	-0.34	0.32	0.51
(8) Other hallucinations	0.00	0.57	0.09
(9) Jealous delusions	-0.22	0.29	-0.11
(10) Alogia	0.64	0.06	0.17
(11) Other bizarre behaviour	0.12	-0.02	0.55

Regression– global psychotic symptoms

Global psychotic symptom severity scores were regressed on all individual- and ward- level predictors (multi-level linear regression; see Table 6 and Figure 3). A null multi-level regression model indicated that ward- level random effects explained 9.3% of the variance in global psychotic symptom severity and significantly improved the fit of the model, relative to a basic intercept only model (chi-squared$_{(1)}$=4.43, p=0.02) (Table 7). Controlling for basic demographic variables (age, gender and SES) slightly reduced the amount of variance that could be attributed to ward- level random effects (8.83%), although this was still a significant proportion (chi-squared$_{(1)}$=4.12, p=0.02). Univariate analyses indicated that the SFS (coefficient=-0.01, CI=-0.02, -0.0003, p=0.04), GINI-ID (-1.34, CI=-2.61, -0.06, p=0.04) and GINI-IMD (coefficient=-1.78 CI=-3.35, -1.96, p=0.03) were significantly associated with global symptom severity, after controlling for basic demographics, such that these were put forward for inclusion in multivariate analyses.

Table 6 Key statistics for outcome variables in the regression analyses. Statistics provided include the number of entries (N), the mean (M), standard deviation (STD), and Z-score skew (Skew Z) before and after transformation / removal of extreme outliers (raw and processed data, respectively). Details are also provided of the optimal transformations used and types of analyses undertaken. GLOB-SXS=total global symptom scores; NEG-SXS=scores on the negative symptoms component to emerge from second- order principal component analysis; POS-SXS=scores on the positive symptoms component; DIS-SXS=scores on the disorganized symptoms component; Depression=depression scores; SAPS-D1=SAPS score on the persecutory delusions item; ML=multi-level.

	Raw data			Processed data				
Variable	N	M (STD)	Skew Z	N	M (STD)	Skew Z	Transformation	Analyses
GLOB-SXS	344	20.04 (8.67)	3.2	344	~0 (1)	-0.21	$x^{1/3}$	Linear ML
NEG-SXS	335	~0 (1)	6.75	332	~0 (1)	0.2	$\text{Log}_{10}\, x$	Linear ML
POS-SXS	335	~0 (1)	5.69	333	~0 (1)	-0.5	$x^{1/3}$	Linear ML
DIS-SXS	335	~0 (1)	5.94	332	~0 (1)	0.7	$\text{Log}_{10}\, x$	Linear ML
Depression	337	2.36 (1.06)	1.74	-	-	-	Binary split	Logistic ML
SAPS-D1	344	2.92 (1.73)	-4.74	-	-	-	Binary split	Logistic ML

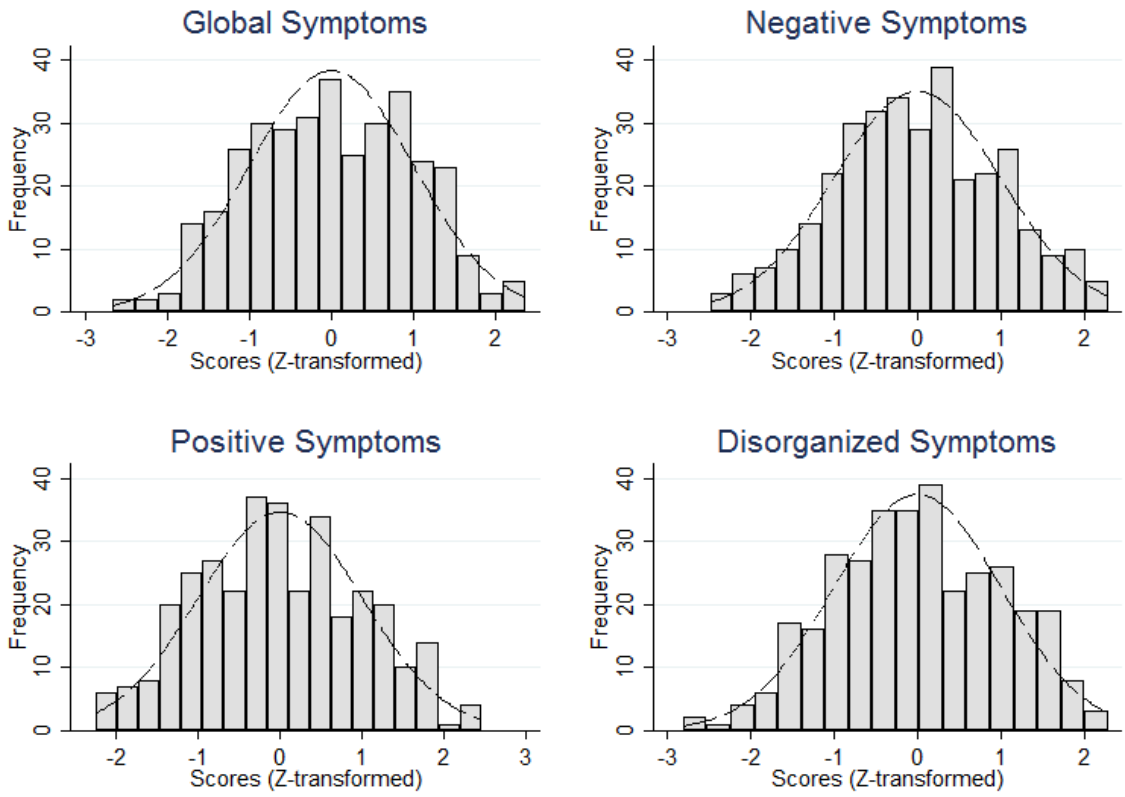

Figure 3 Distribution of symptom measure scores used as outcome variables in the multi-level linear regression analyses. The distribution of global, negative, positive and disorganized symptom scores are shown following removal of extreme outliers and Z-transformation. Dotted lines indicate best-fitting normal distributions.

Model One (Table 7), which was based on 311 participants, included SFS and GINI-IMD (as well as basic demographics) and was highly significant (chi-squared$_{(8)}$=27.87, p<0.001). GINI-ID was not included in the final model since it did not significantly improve the fit when variables were sequentially added to the model according to AIC scores; this was due to a positive high correlation with GINI-IMD scores (Spearman's $r_{s(116)=}$0.79, p<0.001), which was added to the model first. This is unsurprising given the overlap in the two constructs. With respect to SES, global psychotic symptoms were statistically less severe for those belonging to the managerial class relative to the unemployed (coefficient=-0.55, CI=-1.07, -0.03, p=0.04). Further, global-symptom severity was less marked for individuals who were socially higher functioning (SFS: coefficient=-0.01, CI=-0.02, -0.001, p=0.04). With respect to *ward-level* predictors, inequality (GINI-IMD) was the only variable to independently predict a significant amount of unique variance in global psychotic symptom severity (coefficient=-1.9, CI=-3.47, -0.32, p=0.02). Thus, individuals living in wards characterised by *higher* inequality were associated with *less* severe symptoms.

When scores for specific symptoms dimensions (positive, negative and disorganized) and depression were added to the model (Model two; planned analysis) the effect of SES remained significant (coefficient=-0.33, CI=-0.62, -0.03, p=0.03), whilst the effects of SFS (coefficient=0.01, CI-0.001, 0.01, p=0.1) and GINI-IMD disappeared (coefficient=-0.72, CI=-1.75, 0.32, p=0.18) (Table 7, model two).

Finally, in order to determine if the effects of inequality were confounded with ward-level deprivation, model one was re-run whilst controlling for *absolute* deprivation (planned analysis). This either abolished the effect (ID; coefficient=-1.79, CI=-3.49, -0.1 p=0.04) or reduced it (IMD; coefficient=-1.67, CI= -3.39, 0.06, p=0.06), depending on the measure of deprivation used (ID or IMD). In addition, to see if

there was an *interaction* between inequality and absolute deprivation, model 1 was re-run independently for participants living in areas of low deprivation (n=155) and those living in areas of high deprivation (n=156) (median split on IMD scores; planned analysis). Neither analysis was found to be significant; thus, GINI-IMD did not predict global symptom scores in either the low deprivation (coefficient=-1.46, CI=-3.8, 0.89, p=0.22) or high deprivation (coefficient=-1.39, CI=-4.01, 1.23, p=0.3) sub-group.

Table 7 Predictors of total global psychotic symptom severity scores. The top line of the table indicates a random effects only model. Columns three and four show the results of a series of univariate analyses, examining the effects of adding a single predictor (individual- or ward- level) to a basic model that controls for core demographics only (age, gender and SES). Model one shows the results of a multivariate analysis that includes all individual- and ward- level variables selected for inclusion. Model two is identical to model one except that negative, positive, disorganized and depression symptoms are also controlled for. Ward level variables are in italics. Significant variables are in bold.

		Univariate (controlling for basic demographics only)					Model 1 - Multivariate (basic)		Model 2 - Multivariate (controlling for other Sxs also)	
		Fixed part of model		Random part of model			Fixed part of the model		Fixed part of the model	
Predictor	Level	Coefficient (95% CIs)	Wald p value / LR test	Ward-level variance	Chi-squared (1 df)	Chi-squared p value	Coefficient (95% CIs)	Wald p value	Coefficient (95% CIs)	Wald p value
Random only		-	-	9.3	4.43	0.02	-	-	-	-
Age		-0.002 (-0.02, 0.01)	0.74	8.83	4.12	0.02	-0.003 (-0.02, 0.01)	0.66	-0.01 (-0.02, 0.002)	0.14
Gender	Female	-0.25 (-0.48, -0.02)	**0.03**	8.83	4.12	0.02	-0.22 (-0.46,0.02)	0.07	0.06 (-0.07, 0.2)	0.37
NS-SEC	Managerial	-0.68 (-1.19, -0.18)	**0.01**	8.83	4.12	0.02	-0.55 (-1.07, -0.03)	**0.04**	-0.33 (-0.62, -0.03)	**0.03**
	Intermediate	-0.19 (-0.62, 0.24)	0.39	8.83	4.12	0.02	-0.07 (-0.52, 0.37)	0.74	-0.08 (-0.33, 0.17)	0.53
	Routine	-0.28 (-0.58, 0.01)	0.06	8.83	4.12	0.02	-0.18 (-0.5, 0.15)	0.28	-0.1 (-0.28, 0.09)	0.31
	Student	-0.12 (-0.43, 0.2)	0.47	8.83	4.12	0.02	-0.06 (-0.38, 0.26)	0.71	-0.16 (-0.34, 0.02)	0.08
SFS		-0.01 (-0.02, -0.0003)	**0.04**	7.07	2.25	0.07	-0.01 (-0.02, -0.001)	**0.04**	0.01 (-0.001, 0.01)	0.1
DUP		0.001 (-0.001, 0.003)	0.41	7.45	2.78	<0.05	-	-	-	-
Pop Den		0.0003 (-0.003, 0.004)	0.84	9.67	4.55	0.02	-	-	-	-
ID		0.01 (-0.01, 0.03)	0.25	8.42	3.75	0.03	-	-	-	-
IMD		0.01 (-0.002, 0.02)	0.12	8.04	3.41	0.03	-	-	-	-
GINI-ID		-1.34 (-2.61, -0.06)	**0.04**	6.45	2.16	0.07	-	-	-	-
GINI-IMD		-1.78 (-3.35, -1.96)	**0.03**	6.34	2.13	0.07	-1.9 (-3.47, -0.32)	**0.02**	-0.72 (-1.75, 0.32)	0.18
SFI		-0.01 (-0.04, 0.02)	0.61	8.77	4.12	0.02	-	-	-	-
SCI		-0.02 (-0.04, 0.005)	0.14	7.72	2.99	0.04	-	-	-	-
IDS-BME		-0.94 (-3.08, 1.19)	0.39	8.49	3.89	0.02	-	-	-	-
DEN-BME		0.75 (-0.04, 1.54)	0.07	6.58	2.17	0.07	-	-	-	-

Regression – negative symptoms

A null multi-level regression model indicated that ward- level random effects explained only 0.86% of the variance in negative symptom scores (derived from second-order PCA) and did not significantly improve the fit of the model, relative to a basic intercept model (chi-squared$_{(1)}$=0.04, p=0.42) (Table 8). Controlling for basic demographic variables (age, gender and SES) further reduced the amount of variance that could be attributed to ward- level effects (<0.001%) (chi-squared$_{(1)}$=0, p=1). Univariate analyses indicated that the SFS (coefficient=-0.04, CI=-0.05, -0.02, p<0.001), DUP (coefficient=0.002, CI=-0.64, 0.22, p<0.05) and GINI-ID (coefficient=-1.42, CI=-2.6, -0.23, p=0.02) were significantly associated with negative symptoms (after controlling for basic demographics), such that these were put forward for inclusion in multivariate analyses.

The primary multi-level model (Table 8, model one) included 300 participants and was highly significant (chi-squared$_{(8)}$=64.66, p<0.001). Being female (coefficient=-0.25, CI=-0.48, -0.03, p=0.03) and scoring more highly on the SFS (coefficient=-0.04, CI=-0.05, -0.03, p<0.001) were both associated with less severe symptoms. At the ward- level there was an association between *higher* income inequality (GINI-ID) and *less* severe negative symptoms (coefficient=-1.49, CI=-2.66, -0.31, p=0.01). Adding other symptoms to the model (Table 8, model two), i.e. disorganized, positive and depressive symptoms, did not affect the findings: the effects of gender (coefficient=-0.29, CI=-0.53, -0.06, p=0.01), SFS (coefficient=-0.03, CI=-0.05, -0.02, p<0.001) and GINI-ID (coefficient=-1.54, CI=-2.74, -0.34, p=0.01) remained significant.

Finally, in order to determine if the effects of inequality (GINI-ID) were confounded with ward- level deprivation, model one was re-run whilst controlling for *absolute*

deprivation. This did not abolish the effect, irrespective of which measure of deprivation was used, i.e. IMD (coefficient=-1.82, CI=-3.06, -0.58, p=0.004) or ID (coefficient=-1.79, CI=-3.02, -0.57, p=0.004). In addition, to see if there was an *interaction* between inequality and absolute deprivation, model one was re-run independently for participants living in areas of *low* deprivation (n=148) and those living in areas of *high* deprivation (n=152) (median split on IMD scores). Neither analysis was found to be significant, though both were marginal ($p<0.1$); thus, GINI-ID did not predict negative symptom scores in either the low (coefficient=-1.62, CI=-3.47, 0.23, p=0.09) deprivation or high deprivation (coefficient=-1.6, CI=-3.27, 0.07, p=0.06) sub-group.

Table 8 Negative symptoms, derived from second-order principal component analysis, regressed on predictor variables. The top line of the table indicates a random effects only model. Columns three and four show the results of a series of univariate analyses, examining the effects of adding a single predictor (individual- or ward- level) to a basic model that controls for core demographics only (age, gender and SES). Model one shows the results of a multivariate analysis which includes all individual- and ward- level variables selected for inclusion. Model two is identical to model one except that other symptoms (positive, disorganized and depressive symptoms) are also controlled for. Ward level variables are in italics. Significant variables are in bold.

		Univariate (controlling for basic demographics only)					Model 1 - Multivariate (basic)		Model 2 - Multivariate (controlling for other Sxs also)	
		Fixed part of model		Random part of model			Fixed part of the model		Fixed part of the model	
Predictor	Level	Coefficient (95% CIs)	Wald p value	Ward-level variance	Chi-squared (1 df)	Chi-squared p value	Coefficient (95% CIs)	Wald p value	Coefficient (95% CIs)	Wald p value
Random only		-		0.86	0.04	0.42	-	-	-	-
Age		0.01 (-0.003, 0.03)	0.13	0	0	1	0.01 (-0.01, 0.02)	0.38	0.01 (-0.01, 0.02)	0.35
Gender	Female	-0.34 (-0.58, -0.11)	**0.004**	0	0	1	-0.25 (-0.48, -0.03)	**0.03**	-0.29 (-0.53, -0.06)	**0.01**
NS-SEC	Managerial	-0.39 (-0.9, 0.11)	0.13	0	0	1	0.03 (-0.46, 0.53)	0.9	-0.03 (-0.53, 0.47)	0.9
	Intermediate	-0.33 (-0.76, 0.11)	0.14	0	0	1	0.07 (-0.36, 0.49)	0.76	0.03 (-0.4, 0.46)	0.9
	Routine	-0.33 (-0.63, -0.03)	0.03	0	0	1	0.03(-0.28, 0.35)	0.85	0.06 (-0.26, 0.38)	0.72
	Student	0.11 (-0.2, 0.43)	0.5	0	0	1	0.22 (-0.09, 0.52)	0.16	0.17 (-0.14, 0.48)	0.28
SFS		-0.04 (-0.05, -0.02)	**<0.001**	1.07	0.06	0.41	-0.04 (-0.05, -0.03)	**<0.001**	-0.03 (-0.05, -0.02)	**<0.001**
DUP		0.002 (-0.64, 0.22)	**<0.05**	0	0	1	-	-	-	-
Pop Den		-0.002 (-0.005, 0.001)	0.26	0.52	0.01	0.45	-	-	-	-
ID		-0.01 (-0.02, 0.01)	0.41	0	0	1	-	-	-	-
IMD		-0.003 (-0.01, 0.01)	0.6	0	0	1	-	-	-	-
GINI-ID		-1.42 (-2.6, -0.23)	**0.02**	0	0	1	-1.49 (-2.66, -0.31)	**0.01**	-1.54 (-2.74, -0.34)	**0.01**
GINI-IMD		-1.41 (-2.89, 0.06)	0.06	0	0	1	-	-	-	-
SFI		-0.01 (-0.04, 0.02)	0.35	0	0	1	-	-	-	-
SCI		0.001 (-0.02, 0.02)	0.95	0	0	1	-	-	-	-
IDS-BME		-1.28 (-3.22, 0.66)	0.2	0	0	1	-	-	-	-
DEN-BME		-0.07 (-0.82, 0.68)	0.85	0	0	1	-	-	-	-

Regression - positive symptoms

A null multi-level regression model indicated that ward- level random effects explained negligible variance (<0.001%) in positive symptom scores and did not significantly improve the fit of the model, relative to a basic intercept only model (chi-squared$_{(1)}$=0, p=1) (Table 9). Controlling for basic demographic variables (age, gender and SES) did not alter this pattern (chi-squared$_{(1)}$=0, p=1). Nonetheless, univariate analyses indicated that IDS-BME (coefficient=-2.42, CI=-4.39, -0.45, p=0.02) was significantly associated with positive symptom severity, such that this was put forward for inclusion in the multivariate analyses.

The primary multi-level multivariate model (Table 9, model one) included 321 participants and just failed to reach significance (chi-squared$_{(7)}$=13.62, p=0.06). Being of an intermediate SES was associated with elevated positive symptoms, relative to the unemployed (coefficient=0.55, CI=0.11, 0.99, p=0.02). In addition, wards characterised by *higher* segregation of ethnic minorities were associated with *less* severe positive symptoms (coefficient=-2.42, CI=-4.31, -0.45, p=0.02). None of the findings were affected by the inclusion of other symptoms to the model (Model two), i.e. negative, disorganized and depressive symptoms: thus, intermediate SES (coefficient=0.52, CI=0.08, 0.96, p=0.02) and IDS-BME (coefficient=-2.67, CI=-4.65, -0.68, p=0.01) remained significant.

Finally, in order to determine if the effects of IDS-BME were confounded with ward-level deprivation, model one was re-run whilst controlling for *absolute* deprivation. This did not abolish the effect, irrespective of which measure of deprivation was used: IMD (coefficient=-2.29, CI=-4.31, -0.28, p=0.03) or ID (coefficient=-2.33, CI=-4.33, -0.32, p=0.02). In addition, to see if there was an *interaction* between inequality and absolute deprivation, model one was re-run independently for participants living in areas of low deprivation (n=165) and those living in areas of

high deprivation (n=156) (median split on IMD scores). The effect of IDS-BME was not predictive of positive symptoms in participants living in areas of low deprivation (coefficient=-2.41, CI=-5.82, 0.99, p=0.17), nor within areas of high deprivation (coefficient=-2.68, CI=-5.36, -0.02, p=0.05), although the latter fell on the boundary of significance.

Table 9 Positive symptoms, derived from second-order principal component analysis, regressed on predictor variables. The top line of the table indicates a random effects only model. Columns three and four show the results of a series of univariate analyses, examining the effects of adding a single predictor (individual- or ward- level) to a basic model that controls for core demographics only (age, gender and SES). Model one shows the results of a multivariate analysis which includes all individual- and ward- level variables selected for inclusion. Model two is identical to model one except that other symptoms (negative, disorganized and depressive symptoms) are also controlled for. Ward level variables are in italics. Significant variables are in bold.

Predictor	Level	Univariate (controlling for basic demographics only)					Model 1 - Multivariate (basic)		Model 2 - Multivariate (controlling for other Sxs also)	
		Fixed part of model		Random part of model			Fixed part of the model		Fixed part of the model	
		Coefficient (95% CIs)	Wald p value	Ward-level variance	Chi-squared (1 df)	Chi-squared p value	Coefficient (95% CIs)	Wald p value	Coefficient (95% CIs)	Wald p value
Random only		-	-	0	0	1	-	-	-	-
Age		-0.01 (-0.02, 0.01)	0.29	0	0	1	-0.01 (-0.02, 0.01)	0.26	-0.01 (-0.02, 0.01)	0.25
Gender	Female	0.01 (-0.23, 0.25)	0.95	0	0	1	0.03 (-0.21, 0.27)	0.82	0.01 (-0.24, 0.25)	0.96
NS-SEC	Managerial	-0.01 (-0.53, 0.51)	0.98	0	0	1	0.05 (-0.47, 0.57)	0.84	0.01 (-0.51, 0.53)	0.96
	Intermediate	0.52 (0.08, 0.97)	**0.02**	0	0	1	0.55 (0.11, 0.99)	**0.02**	0.52 (0.08, 0.96)	**0.02**
	Routine	0.16 (-0.15, 0.47)	0.32	0	0	1	0.16 (-0.15, 0.47)	0.3	0.15 (-0.16, 0.47)	0.34
	Student	0.15 (-0.17, 0.47)	0.35	0	0	1	0.11 (-0.21, 0.43)	0.49	0.07 (-0.25, 0.4)	0.66
SFS		-0.01 (-0.02, 0.004)	0.2	0	0	0.5	-	-	-	-
DUP		0.001 (-0.001, 004)	0.25	0	0	1	-	-	-	-
Pop Den		-0.0004 (-0.003, 0.003)	0.77	0	0	1	-	-	-	-
ID		0.24 (-0.24, 0.72)	0.36	0	0	1	-	-	-	-
IMD		-0.01 (-0.2, 0.01)	0.3	0	0	1	-	-	-	-
GINI-ID		0.22 (-0.1, 1.44)	0.72	0	0	1	-	-	-	-
GINI-IMD		0.34 (-1.18, 1.85)	0.66	0	0	1	-	-	-	-
SFI		0.004 (-0.03, 0.03)	0.81	0	0	1	-	-	-	-
SCI		0.02 (-0.002, 0.04)	0.08	0	0	1	-	-	-	-
IDS-BME		-2.42 (-4.39, -0.45)	**0.02**	0	0	1	-2.42 (-4.31, -0.45)	**0.02**	-2.67 (-4.65, -0.68)	**0.01**
DEN-BME		-0.55 (-1.31, 0.21)	0.16	0	0	1	-	-	-	-

Regression - disorganized symptoms

A null multi-level regression model indicated that ward- level random effects accounted for only 2.87% of the variance in disorganized symptoms (derived from second-order PCA) and did *not* significantly improve the fit, relative to a basic intercept only model (LR test: chi-squared$_{(1)}$=0.35, p=0.28) (Table 10). Controlling for basic demographic variables (age, gender and SES) further reduced the amount of variance that could be attributed to ward- level effects (0.44%; chi-squared$_{(1)}$=0.01, p=0.46). Nonetheless, univariate analyses indicated that SFS (coefficient=0.02, 0.004, 0.03, p=0.01) and DUP (coefficient= -0.003, CI=-0.01, -0.0003, p=0.03) were significantly associated with disorganized symptom severity, after controlling for basic demographics, such that these were put forward for inclusion in the multivariate analyses.

The primary multivariate multi-level model (Table 10, model one) included 283 participants and was significant (chi-squared$_{(8)}$=18.24, p=0.02). With respect to SES, being in routine employment was associated with reduced disorganized symptoms relative to being unemployed (coefficient=-0.36, CI=-0.72, -0.02, p=0.04). Interestingly, higher scores on the SFS (coefficient=0.02, CI=0.003, 0.03, p=0.02) and a shorter DUP (coefficient=-0.003, CI=-0.01, -0.0003, p=0.02) were associated with *higher* disorganized symptom scores. No ward-level predictors were significant.

Finally, addition of other symptoms to the model (i.e. negative, positive and depressive symptoms) rendered the effects of SES (coefficient=-0.34, CI=-0.69, -0.003, p=0.05) and SFS (coefficient=0.01, CI=0.003, 0.02, p=0.12) non-significant (Table 10, model two). However, the effect of DUP remained significant (coefficient=-0.003, CI=-0.01, -0.0003, p=0.03).

Table 10 Disorganized symptoms, derived from second-order principal component analysis, regressed on predictor variables. The top line of the table indicates a random effects only model. Columns three and four show the results of a series of univariate analyses, examining the effects of adding a single predictor (individual- or ward- level) to a basic model that controls for core demographics only (age, gender and SES). Model one shows the results of a multivariate analysis which includes all individual- and ward- level variables selected for inclusion. Model two is identical to model one except that other symptoms (positive, negative and depressive symptoms) are also controlled for. Ward level variables are in italics. Significant variables are in bold.

		Univariate (controlling for basic demographics only)					Model 1 - Multivariate (basic)		Model 2 - Multivariate (controlling for other Sxs also)	
		Fixed part of model		Random part of model			Fixed part of the model		Fixed part of the model	
Predictor	Level	Coefficient (95% CIs)	Wald p value	Ward-level variance	Chi-squared (1 df)	Chi-squared p value	Coefficient (95% CIs)	Wald p value	Coefficient (95% CIs)	Wald p value
Random only		-	-	2.87	0.35	0.28	-	-	-	-
Age		-0.01 (-0.02, 0.01)	0.26	0.44	0.01	0.46	-0.01 (-0.02, 0.01)	0.43	-0.003 (-0.02, 0.01)	0.76
Gender	*Female*	0.01 (-0.23, 0.25)	0.94	0.44	0.01	0.46	-0.14 (-0.39, 0.11)	0.28	-0.08 (-0.34, 0.18)	0.55
NS-SEC	*Managerial*	-0.04 (-0.56, 0.48)	0.89	0.44	0.01	0.46	-0.48 (-1.04, 0.08)	0.09	-0.46 (-1.01, 0.09)	0.1
	Intermediate	0.51 (0.06, 0.95)	**0.03**	0.44	0.01	0.46	-0.16 (-0.62, 0.31)	0.51	-0.18 (-0.64, 0.29)	0.46
	Routine	0.13 (-0.18, 0.45)	0.4	0.44	0.01	0.46	-0.36 (-0.71, -0.01)	**0.04**	-0.34 (-0.69, -0.003)	0.05
	Student	0.11 (-0.22, 0.44)	0.51	0.44	0.01	0.46	-0.3 (-0.62, 0.03)	0.07	-0.26 (-0.59, 0.07)	0.12
SFS		0.02 (0.004, 0.03)	**0.01**	0	0	1	0.02 (0.003, 0.03)	**0.02**	0.01 (0.003, 0.02)	0.12
DUP		-0.003 (-0.01, -0.0003)	**0.03**	0	0	1	-0.003 (-0.01, -0.0003)	**0.02**	-0.003 (-0.01, -0.0003)	**0.03**
Pop Den		0.001 (-0.002, 0.004)	0.55	0.59	0.01	0.45	-	-	-	-
ID		0.01 (-0.01, 0.02)	0.29	0.35	0.01	0.47	-	-	-	-
IMD		0.01 (-0.004, 0.02)	0.23	0.13	0	0.49	-	-	-	-
GINI-ID		-0.73 (-1.94, 0.49)	0.24	0	0	1	-	-	-	-
GINI-IMD		-0.87 (-2.38, 0.64)	0.26	0	0	1	-	-	-	-
SFI		0.005 (-0.03, 0.04)	0.76	0.39	0.01	0.47	-	-	-	-
SCI		-0.02 (-0.04, 0.003)	0.1	0	0	1	-	-	-	-
IDS-BME		-0.11 (-2.1, 1.88)	0.92	0.53	0.01	0.46	-	-	-	-
DEN-BME		0.52 (-0.23, 1.28)	0.18	0	0	1	-	-	-	-

Regression – persecutory delusions

Scores on the persecutory delusions item of the SAPS (SAPS-D1) were regressed on to individual- and ward- level predictors (after conversion into a binary variable: absent / mild versus moderate / severe) using multi-level logistic regression. Ward-level random effects explained 7.7% of the variance in outcome but did *not* significantly improve the fit relative to an intercept only model (LR test: chi-squared$_{(1)}$=1.48, p=0.11).

The primary multi-level model (Model 1) included 335 participants and was not significant (chi-squared$_{(8)}$=8.19, p=0.22) (Table 11). This included only one variable that predicted significant levels of unique variance in persecutory delusions: thus, belonging to the managerial class was associated with *less severe* symptoms relative to being unemployed (OR=0.27, CI= p=0.02). Further, this persisted after controlling for other symptoms, i.e. positive, negative, disorganized and depression (OR=0.23, CI=0.07, 0.75, p=0.02).

Table 11 Item D1 of the SAPS (persecutory delusions) regressed on predictor variables. Model one shows the results of a multivariate analysis which includes basic demographics only, since no other individual- or ward- level predictors were found to be significant. Model two is identical to model one except that other symptoms (negative, positive, disorganized and depressive symptoms) are also controlled for. Significant variables are in bold. OR=odds ratio.

Model	Predictor	Level	OR (95% CIs)	Wald p value
Model 1 (basic)	Age		1.03 (0.99, 1.06)	0.15
	Gender	Female	0.97 (0.57, 1.64)	0.9
	NS-SEC	Managerial	0.28 (0.09, 0.84)	**0.02**
		Intermediate	1.04 (0.37, 2.94)	0.94
		Routine	0.77 (04, 1.5)	0.45
		Student	0.7 (0.35, 1.38)	0.31
Model 2 (controlling for other Sxs also)	Age		1.03 (0.99, 1.07)	0.1
	Gender	Female	0.95 (0.53, 1.68)	0.85
	NS-SEC	Managerial	0.23 (0.07, 0.75)	**0.02**
		Intermediate	0.75 (0.26, 2.17)	0.59
		Routine	0.71 (0.35, 1.47)	0.36
		Student	0.53 (0.26, 1.09)	0.08

Regression – depression

Binary depression scores were regressed on to individual- and ward- level predictors (after conversion into a binary variable: absent / mild versus moderate / severe) using multi-level logistic regression (Table 12).

Ward- level random effects explained ~0% of the variance in outcome and did *not* significantly improve the fit relative to an intercept only model (LR test: chi-squared$_{(1)}$=~0, p=~1). The primary multi-level model (Table 12, model one) included 305 participants and was not quite significant (chi-squared$_{(8)}$=13.94, p=0.05).

The only significant predictors of depression scores were SES and SFS. Thus, higher social functioning was associated with lower levels of depression (OR=0.96, CI=0.93, 0.99, p=0.003), whilst belonging to the managerial class (relative to being unemployed) was associated with greater symptoms of depression (OR=3.58, CI=1.06, 12.15, p=0.04). There were no significant ward- level predictors.

Finally, inclusion of global psychotic symptom scores in the model (Table 12, model two) abolished the effect of SES (OR=2.95, CI=0.86, 10.12, p=0.09), whilst the effect of SFS remained significant (OR=0.97, CI=0.94, 1, p=0.03).

Table 12 Depression scores regressed on predictor variables. Model one shows the results of a multivariate analysis which includes all individual- and ward- level variables selected for inclusion. Model two is identical to model one except that global psychotic symptoms are controlled for. Significant variables are in bold. OR=odds ratio.

Model	Predictor	Level	OR (95% CIs)	Wald p value
Model 1 (basic)	Age		1 (0.97, 1.03)	0.98
	Gender	Female	1.45 (0.84, 2.5)	0.18
	NS-SEC	Managerial	3.58 (1.06, 12.15)	**0.04**
		Intermediate	2.47 (0.89, 6.82)	0.08
		Routine	1.17 (0.55, 2.49)	0.69
		Student	1.29 (0.64, 2.64)	0.48
	SFS		0.96 (0.93, 0.99)	**0.003**
Model 2 (controlling for other Sxs also)	Age		1 (0.97, 1.04)	0.87
	Gender	Female	1.58 (0.9, 2.77)	0.11
	NS-SEC	Managerial	2.95 (0.86, 10.12)	0.09
		Intermediate	1.86 (0.66, 5.21)	0.24
		Routine	1 (0.45, 2.2)	1
		Student	1.06 (0.52, 2.18)	0.87
	SFS		0.97 (0.94, 1)	**0.03**

Regression – complete case analyses

Finally, all primary regression analyses were re-run using a complete case analysis approach, as described in the Methods (n=255 participants and n=99 wards). Table 13 therefore summarises the findings from the regression of individual symptom dimension scores (global, negative, positive and disorganized) on individual- and ward- level predictors, whilst controlling for basic demographics (equivalent to model 1 in Table 7). The findings closely support the primary analyses: higher inequality (GINI-IMD) was associated with lower global symptom severity (coefficient=-2.33, CI=-4.08, -0.57, p=0.01), higher scores on the GINI-ID predicted lower negative symptom scores (coefficient=-1.7, CI=-2.98, -0.42, p=0.01), and higher ethnic segregation (IDS-BME) was significantly associated with less severe positive symptoms (coefficient=-3.02, CI=-5.17, -0.87, p=0.01). Data are not shown for the regression of depression and persecutory delusion scores since as *per* primary analyses, no ward- level predictors were found to predict significant levels of variance in outcome.

Table 13 Summary of complete case analyses. Individual symptom dimensions, derived from second-order principal component analysis, were regressed on predictor variables using multi-level regression analyses. Data reported are from multivariate models that were run whilst controlling for basic demographic information (age, gender and NS-SEC). Ward level variables are in italics. Significant variables are in bold.

		Global symptoms		Negative symptoms		Positive symptoms		Disorganized symptoms	
		Fixed part of the model		Fixed part of the model		Fixed part of the model		Fixed part of the model	
Predictor	Level	Coefficient (95% CIs)	Wald p value	Coefficient (95% CIs)	Wald p value	Coefficient (95% CIs)	Wald p value	Coefficient (95% CIs)	Wald p value
Age		-0.004 (-0.02, 0.01)	0.58	0.01 (-0.01, 0.02)	0.31	-0.01 (-0.03, 0.01)	0.21	-0.003 (-0.02, 0.02)	0.77
Gender	Female	-0.24 (-0.5, 0.01)	0.06	-0.25 (-0.49, -0.005)	**<0.05**	0.03 (-0.23, 0.29)	0.8	-0.14 (-0.41, 0.13)	0.3
NS-SEC	Managerial	0.56 (-1.08, -0.04)	**0.04**	-0.09 (-0.6, 0.42)	0.73	0.21 (-0.33, 0.75)	0.44	-0.51 (-1.07, 0.06)	0.08
	Intermediate	0.01 (-0.45, 0.48)	0.95	0.13 (-0.33, 0.58)	0.59	0.55 (0.07, 1.03)	**0.02**	-0.18 (-0.68, 0.32)	0.48
	Routine	-0.3 (-0.63, 0.03)	0.08	0.04 (-0.29, 0.37)	0.81	0.05 (-0.29, 0.39)	0.77	-0.34 (-0.7, 0.02)	0.07
	Student	0.02 (-0.32, 0.36)	0.91	0.3 (-0.22, 0.61)	0.07	0.12 (-0.22, 0.47)	0.48	-0.27 (-0.62, 0.07)	0.12
SFS		-	-	-0.04 (-0.05, -0.02)	**<0.001**	-	-	0.02 (0.004, 0.03)	**0.01**
DUP		-	-	-	-	-	-	-0.003 (-0.01, -0.0004)	0.04
Pop Den		-	-	-	-	-	-	-	-
ID		-	-	-	-	-	-	-	-
IMD		-	-	-	-	-	-	-	-
GINI-ID		-	-	-1.7 (-2.98, -0.42)	**0.01**	-	-	-	-
GINI-IMD		-2.33 (-4.08, -0.57)	**0.01**	-	-	-	-	-	-
SFI		-	-	-	-	-	-	-	-
SCI		-	-	-	-	-	-	-	-
IDS-BME		-	-	-	-	-3.02 (-5.17, -0.87)	**0.01**	-	-
DEN-BME		-	-	-	-	-	-	-	-

Discussion

Principle findings

Consistent with hypothesis one (*H1: test of environmental influence*), regression analyses indicated that ward- level random effects predicted a significant proportion of variance in global psychotic symptom severity after controlling for individual- level demographics (age, sex and SES). These findings are consistent with a growing body of literature that highlights the importance of environmental factors in psychosis; see Dean and Murray (2005) for discussion. Further, they suggest that the association between environmental factors and psychosis can be detected at the level of symptoms as well as at the level of incidence / prevalence, the latter having been the focus of the majority of previous research in this field.

In contrast, ward- level random effects *did not* predict significant amounts of variance in individual symptom dimensions. However, estimates of neighbourhood-level random effects have previously been shown to be comparatively unreliable and biased towards under-estimation (Bryan & Jenkins, 2016). Consequently, and given specified *a priori* hypotheses (e.g. *H2: test of specificity*), ward- level fixed effects were modelled, irrespective of the overall significance of random effects.

In support of hypotheses two (*H2: test of specificity*), each symptom dimension was associated with a distinct pattern of ward- level fixed effects, even after controlling for other symptom dimensions and depression. Thus, *higher* levels of inequality were associated with *lower* negative symptoms and greater ethnic segregation was associated with *less* severe positive symptoms. Further, once scores on individual symptom dimensions were controlled for, ward- level fixed effects did not predict unique variance in *global* symptom scores. These findings suggest, therefore, that distinct environmental factors may act as specific risk factors in the etiology of

defined symptoms / symptom dimensions, rather than acting more generally, e.g. as general stressors that exacerbate all symptoms; see later discussion on direction of causality however.

With respect to hypothesis three (*H3: dimension- level analysis*), there was no evidence to suggest that the positive symptoms of psychosis were higher in areas of *socioeconomic* deprivation, after controlling for basic demographics; thus, there was no association between positive symptom scores and ward- level deprivation or inequality. Nor were there any associations between positive symptoms and ward-level social cohesion, fragmentation or population density. However, as mentioned above, there *was* an association between less severe positive symptoms and *higher* segregation of ethnic minorities.

Finally, with respect to hypothesis four, there was no evidence to suggest that persecutory delusions -in particular of the positive symptoms- were associated with social or socioeconomic deprivation. Thus, no single ward- level predictor explained significant amounts of variance in paranoia.

Significant associations between symptoms / symptom dimensions and individual- and ward- level predictors will now be discussed in greater depth below; only those findings that persisted after controlling for *other* symptoms as well as basic demographics are described since this represents a more stringent test of specificity of association.

Ward- level effects

Considering ward- level fixed effects first, an association was found between *higher* inequality and *less* severe negative symptom scores (GINI-ID). Thus, participants living in wards characterised by higher inequality, i.e. higher disparity between the

rich and the poor, typically had *less* severe negative symptoms. Further, this effect emerged after controlling for individual- level SES, and survived after controlling for ward- level deprivation. The effect was also present in the complete case analysis. This finding is inconsistent with the Income Inequality Hypothesis (Wilkinson & Pickett, 2009), which posits that highly unequal neighbourhoods will be characterised by poorer health outcomes; see thesis *Part One: Literature Review*, which includes a more in-depth explanation of the IIH and related theories. Further, it is difficult to reconcile with the existing literature. Thus, higher inequality has been associated with an *increased* incidence of FEP in East London (Kirkbride et al., 2014) and South London (most deprived wards only (Boydell, van Os, McKenzie, & Murray, 2004)), as well as within municipalities of South Africa (Burns & Esterhuizen, 2008). However, no such association was seen with respect to psychotic *symptoms* in a study of FEP undertaken in South East London and Nottinghamshire (Oher et al., 2014).

In searching for reasons for this discrepancy one might look for differences in either the population sample or the geographical location examined. With respect to the former, there was no reason to suspect that the participants recruited here were unrepresentative of FEP: they were in fact highly typical of this participant group in terms of age, gender split, DUP and symptom severity; see Hovington, Bodnar, Joober, Malla, and Lepage (2012) and Skeate, Jackson, Birchwood, and Jones (2002) for example. With respect to the socio-political geography of the region, a review of Table 3 -which shows the pattern of associations between ward- level predictors- indicates that *more* unequal wards were actually *less* deprived, and characterised by *higher* levels of social cohesion. Seen in this way, a picture emerges of highly unequal areas that are defined by relative *extremes of affluence* rather than extremes of poverty. Consistent with this hypothesis, Appendix 8 Supplementary Figure 2 shows that the distribution of deprivation scores in *higher*

inequality wards was slightly biased towards *lower* levels of deprivation. Thus, whilst the Gini coefficient (by itself) captures *variance* in incomes, it says little about the *distribution* of those incomes. However, Appendix 8 Supplementary Figure 2 *also* shows that this pattern is broadly consistent with nationwide data.

Given the observed association between higher inequality and lower deprivation / high social cohesion within the wards studied, it is perhaps unsurprising that symptoms were not elevated in the more unequal wards. According to one major hypothesis, the Social Capital Hypothesis (McKenzie et al., 2002), areas that are relatively more equal tend to have better health outcomes because they are characterised by greater trust and a sense of shared community; see thesis *Part One: Literature Review* for discussion. In support of this hypothesis, data from 80 countries taken from the World Values Survey (WVS) and European Values Study (EVS) indicated that social trust mediated the effects of income inequality on self-rated health (Kragten & Rözer, 2017). In the area studied here, however, although trust was not measured directly, social cohesion (a related construct) was in fact greater in wards characterised by *higher* inequality. Although, a significant association was not seen between social cohesion and psychotic / depressive symptoms, it is possible that more sensitive indices of social capital, or measures that tap into different facets of this complex construct, might uncover such an association (Siegler, 2014). Although merely speculative, such a finding would indicate that the Social Capital Hypothesis may be relevant to this population group, it is just that the social capital shows an unexpected pattern of association with inequality here.

An alternative possible explanation of the observed association between high inequality and more severe negative symptoms can be found in the Mixed

Neighbourhood Hypothesis (MNH); see thesis *Part One: Literature Review* as well as Manley, VanHam, and Doherty (2012), Musterd and Andersson (2005) and Ostendorf, Musterd, and De Vos (2001) for discussion. According to the MNH, the mixing of individuals from different socioeconomic backgrounds within areas of high inequality may be protective against some of the difficulties that can arise within areas of *homogenous* deprivation, e.g. cultures of crime, substance use, joblessness and a lack of social opportunity. Further, the presence of the wealthy in an area of high inequality may also bring higher investment in local infrastructure and resources, e.g. parks, schools and health services. With respect to this particular data-set, one might hypothesise how some of the more social symptoms to have loaded onto component one (negative symptoms) -e.g. social inattention (SANS-S22), a lack of relationships with friends and peers (SANS-S20), impersistence at work or school (SANS-S14) and impoverished recreational interests and activities (SANS-S17)- might be partially exacerbated in areas characterised by low investment in community services and a scarcity of public resources**.** However, in the absence of further research this remains purely speculative.

The only other significant ward- level factor to predict variation in participants' symptom scores was ethnic segregation (IDS-BME). Thus, participants living in wards that were characterised by highly segregated BME communities were found to be associated with *less* severe positive symptoms. This effect persisted after controlling for ward- level deprivation as well as other symptoms. These findings are consistent with a previous study undertaken in South East London, which reported a lower incidence of psychosis in areas characterised by higher (BME) ethnic segregation, even after controlling for age, sex, ethnicity, deprivation and population density (Kirkbride et al., 2007), as well as research showing that the risk of psychosis reduces as the proportion of one's own ethnic group increases within a

neighbourhood (Boydell et al., 2001; Das-Munshi et al., 2012; Kirkbride et al., 2007; Veling et al., 2008). However, to the author's knowledge, no single study to date has explored the association between ethnic segregation and psychotic *symptoms*, rather than incidence / prevalence.

To explain this association between *higher* ethnic segregation and *lower* positive symptoms one must recall that whilst a high index of dissimilarity indicates high segregation *between* populations, *within* a given ethnic group it implies *reduced* fragmentation. The Social Capital Hypothesis might be relevant here; thus, a distinction is often made between *binding* social capital, which describes links that exist *between* groups, and *bonding* social capital, which exists *within* a group (Orford, 2008). Being closely connected to and embedded within one's own ethnic community might facilitate *bonding* social capital (Becares & Nazroo, 2013), an asset that may in turn be protective against the positive symptoms of psychosis, e.g. hallucinations and delusions (Das-Munshi et al., 2012). For example, one might hypothesize how individual symptoms that loaded onto component 2 (positive symptoms) –e.g. delusions of reference (SAPS-D7), delusions of control (SAPS-D8), voices commenting (SAPS-H2) and voices conversing (SAPS-H3)- might be exacerbated if a person felt lonely or isolated from their own community, i.e. conditions conducive to the development of paranoia (Lamster, Lincoln, Nittel, Rief, & Mehl, 2017). However, if this is the case, one might expect ethnic segregation to predict variation in paranoia, i.e. persecutory delusions, also (H4; *item level analysis*). This was not found to be the case. Nonetheless, item-level analyses are likely to be less robust than a dimension-level analysis, since the former, by definition, depends on the accurate coding of a single response.

Individual- level effects

Although not the primary focus of the thesis, a number of individual- level predictors were also tested for association with symptoms. This included basic demographics (age, gender and SES), which were built into all models as *a priori* potential confounders, as well as other individual variables that were tested for association (DUP and SFS).

With respect to basic demographics, gender was found to predict negative symptom severity, with less severe symptoms seen in female participants. Previous studies have generated inconsistent findings in relation to gender differences in symptom expression; whilst many have found no differences, others have found *higher negative* but *lower affective* symptom scores in males; see Ochoa et al. (2012) for a review.

Socioeconomic status was also found to predict variance in symptom ratings. Thus, relative to unemployment, belonging to the managerial class (the highest class on the NS-SEC measure) was associated with reduced global symptom severity and lower scores on the persecutory delusions item of the SANS. However, in parallel, being of an intermediate SES was associated with elevated positive symptoms (relative to being unemployed), a finding that is difficult to interpret / reconcile with previous studies. Interestingly, in a review of the evidence for an association between social class at birth and risk of psychosis, the authors identified a shifting trend across time, with an association between low SES and high incidence of psychosis emerging in studies undertaken after 2001, i.e. around the time that data for this study were gathered, but not in studies undertaken before this period (Kwok, 2014). However, the authors offer no explanation for this finding.

With respect to social functioning, higher scores on the SFS, i.e. better social functioning, were associated with less severe negative symptoms and lower levels of depression. This is consistent with a study of individuals at high risk of psychosis (Corcoran et al., 2011), which found that poor social functioning correlated with negative, disorganized and global psychotic symptoms as well as depressive symptoms. However, in their study, the association with negative symptoms was the only one to survive regression analyses once other symptoms were controlled for (Corcoran et al., 2011). More generally, the finding is also consistent with a broader body of literature that highlights the protective role of social capital and social support in depression, i.e. irrespective of a diagnosis of schizophrenia; see Gariépy, Honkaniemi, and Quesnel-Vallée (2016) for a review.

Finally, an association was also seen between DUP and disorganized symptoms; thus, a *longer* period without treatment was associated with *less* severe disorganized symptoms. This is somewhat counter-intuitive at first glance, since the existing evidence would suggest that treatment outcome suffers if treatment is delayed; see Penttilä, Jääskeläinen, Hirvonen, Isohanni, and Miettunen (2014) for a review. Further, a meta-analysis of 43 relevant papers found that at the start of treatment, a *longer* DUP in FEP was associated with *more* severe negative symptoms; however, it did *not* predict severity of positive symptoms, general psychopathology or neurocognition. Further, the authors did not explore any association with disorganized symptoms (Perkins, Gu, Boteva, & Lieberman, 2005).

Interestingly, in the data reported here, participants' disorganized symptom scores *negatively* correlated with their depression scores (r_s=-0.21, p<0.001); see Appendix 9, Supplementary Table 4. Thus, clients who exhibited highly disorganized symptoms were *less* likely to be depressed or show negative symptoms. This finding is consistent with a PCA study of psychotic symptoms in schizophrenia

(Dollfus & Petit, 1995), which demonstrated a similar negative association between 'conceptual disorganisation' and depression during the acute phase of the illness, an association that reversed in sign once the acute phase had passed; see Dollfus et al. (1993) also. The authors hypothesized that individuals might only be able to feel depressed once the acute phase has passed and the disorganized symptoms are less prevalent, or else, that the depressive / negative symptoms may be harder to detect in the context of florid psychosis / disorganized symptoms (Knights & Hirsch, 1981). Relatedly, the Insight Paradox describes a phenomenon in schizophrenia whereby the more insight an individual has into the nature of their illness, the more pronounced their depressive symptoms (Belvederi Murri et al., 2015).

Turning these scenarios on their head, might it be that the disorganized symptoms, many of which related to speech, e.g. derailment, tangentiality, incoherence, word salad, illogicality, circumstantiality, pressure of speech etc., were harder to detect in the context of high negative symptoms, e.g. paucity of expressive gesture, poverty of speech, social inattentiveness etc.? Put simply, perhaps the content of the communication (disorganized) is somewhat overlooked when communication is itself reduced and the participant is highly withdrawn (high negative / depressive symptoms). Returning to the data presented here, this might explain the counter-intuitive association between *higher* disorganized symptom scores and a *shorter* DUP. Thus, perhaps the disorganized symptoms of individuals who presented to services for the first time after a long period without treatment (long DUP) were masked by the prevalence of severe negative and/or depressive symptoms. However, in the absence of further data, this remains purely speculative.

Limitations of the study

There are a number of limitations to the study. Perhaps the greatest is its cross-sectional design, which means that no causal inferences could be made. For example, with respect to the reported associations between ethnic segregation and positive symptoms scores, the data cannot distinguish between hypotheses based on theories of social drift, i.e. do individuals with more severe symptoms tend to drift out of ethnically integrated communities, versus theories of causation, i.e. is a fragmented community a causal *risk factor* for psychotic symptoms; see Cooper (2005) for a related discussion. To begin to establish a direction of causality one must instead employ a longitudinal design. Although longitudinal data were available for this population sample, participant drop-out at follow-up was high such that statistical power would have been severely compromised. Further, a longitudinal approach was not deemed necessary to address the core hypotheses.

Another major limitation is that the data were collected between a decade or two ago, i.e. between 1998 and 2006. In the intervening period since data collection the area is likely to have undergone many social, socio-political and socioeconomic changes, e.g. changing patterns of migration and government investment, as well as service restructure, e.g. the introduction of early intervention services (EIS). Whilst this may limit the extent to which the findings are representative of the West London region as it stands today, it does not necessarily compromise the findings with respect to the associations identified between psychotic symptoms and *defined* environmental variables. These associations may be equally relevant to other regions (and/or time periods), although further research and an integration of findings from multiple studies is needed to determine if this is the case.

Other limitations relate to sampling and possible bias in sampling. Thus, whilst the population sample used here may have been typical of individuals presenting to

services, it will inevitably have missed harder-to-reach individuals who were not known to services, including those who may have been too unwell to engage. Similarly, individuals who consented to take part in the research also represent a self-selecting sub-sample of the broader population of individuals with FEP, and necessary inclusion / exclusion criteria, e.g. fluency in English, will have further limited the broader ecological validity of the study. Finally, another complication with data sampling / collection is that individual- level data and ward- level indices were not gathered simultaneously. Nonetheless, efforts were made to ensure that all indices were derived from data taken as coincidentally as possible, e.g. voter turnout was based on 2002 election data, whilst the IMD was based on the 2004 English Indices of Deprivation. Further, there is no reason to suppose that this temporal noise will have introduced any *systematic* bias into the data.

With respect to the variables available for analysis, whilst ethnicity was controlled for at the ward- level, individual- level ethnicity was not built into the model since these data were missing for a large proportion of the participants. Consequently, individual- level ethnicity may have confounded some of the effects of ward- level indices, particularly those relating directly to ethnicity, e.g. the effect of IDS-BME on positive symptoms. Thus, there is ample evidence that the *incidence* / *prevalence* of psychosis is elevated in migrant and minority ethnic populations, an effect that seems to persist even after controlling for individual- level SES (Kirkbride et al., 2012). Further, psychotic *symptoms* may also be elevated in individuals from these populations (King et al., 2005). A number of other, potentially important variables could not be included in the analyses as potential confounders or predictors, e.g. substance use and family history of psychosis, since these were not collected consistently throughout the entire course of the project. To avoid this pitfall, future studies in the field should gather this information as routine practice.

Another limitation to the study is the use of voter turnout as a proxy for social cohesion. Whilst there is evidence to suggest that voter turnout does indeed correlate with self-reported interpersonal and societal trust, e.g. Arora, Mendoza, and Kim (2016), a number of criticisms of the approach can –and have been- raised. First, social cohesion is a complex and multi-faceted construct, and as such, is unlikely to be captured in its entirety by such a crude, population-level aggregate measure, i.e. it has relatively low content validity; see Orford (2008) for a review. Second, voter turnout, by definition, cannot represent individuals who are denied access to the electoral role, for example, individuals under the voting age (<16years), non-citizen migrants, refugees and asylum seekers. This is particularly relevant to the field of psychosis research, since migrant groups have an elevated risk of experiencing psychotic symptoms; see Parrett and Mason (2010) for a review. Despite these limitations however, the Organisation for Economic Co-operation and Development (OECD) has included voter turnout as an indicator of civic and political participation in its National Well-being Wheel of Measures (Self, 2014).

Finally, the Gini coefficient used here was not the most commonly reported in the literature, although to date, there has been little consensus across studies of FEP as to which measure of inequality to use; see Boydell et al. (2004), Burns and Esterhuizen (2008) and Kirkbride et al. (2014) for example. Thus, a Gini coefficient based on variation in neighbourhood- level mean income is more commonly reported in the literature than the indices calculated here, which were based on indices of deprivation / multiple deprivation. However, previous research has shown that the choice of income inequality measure has little impact on findings, and there is a high degree of correlation across measures (Bechtel, Lordan, & Rao, 2012; Gresenz, Sturm, & Tang, 2001; Kawachi & Kennedy, 1997). Further, the measures of inequality chosen here have been used previously in a highly relevant study of

FEP (Kirkbride et al., 2014), thereby facilitating direct comparison of findings. Further, one might argue that a measure of inequality based on a broader range of indices of deprivation (the IMD) may be more sensitive than one based on a single dimension of wealth / deprivation, since deprivation is itself a multi-faceted construct.

Conclusions

This study is one of very few to explore ecological predictors of psychotic *symptoms* in FEP*,* whilst controlling for individual- level factors using appropriate multi- level modeling techniques that are optimized to clustered data of this kind. The findings show that several ward- level variables predicted unique and significant variance in specific symptom dimensions. Specifically, lower negative symptoms were associated with higher income inequality, whilst lower positive symptoms were associated with greater levels of (*between-groups*) ethnic segregation, even after controlling for *absolute* levels of deprivation and other symptoms. These findings reinforce the importance of studying environmental factors in psychosis, as well as the need for longitudinal studies that can begin to identify patterns of causation, and explore the interaction of key factors at the individual-, interpersonal- and societal level. Finally, the findings reported also highlight the limitations of reducing the complex social and socioeconomic profile of a geographical region to a set of unidimensional, aggregate indices that fail to capture the broader context. For example, the unexpected finding that individuals in *more* unequal areas actually experienced *less* severe negative symptoms -in seeming contradiction with previous studies- only began to make sense once the broader association *between* environmental factors, i.e. surrounding context, was explored. Thus, the most unequal areas were actually found to be the *least* deprived and *most* socially cohesive. This indicates the need for future studies of this kind to incorporate a fuller characterisation of their geographical region of interest, as well as the importance of

replicating the research within different contexts and using distinct population samples.

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Part 3: Critical Appraisal

The following represents a critical reflection on my experience of undertaking research for the review and empirical sections of the thesis. I begin with an outline of my background and the role this played in my selection of a project, before moving on to a series of reflections on the various stages of the research process. Finally, I explore some of the broader questions that arose for me whilst working on the thesis and through exploration of the broader literature within the field.

Background

Prior to clinical training the bulk of my professional experience was spent in academia undertaking research in the field of visual neuroscience and experimental psychology. I spent more than ten years using visual psychophysics and brain imaging techniques to characterise the way people with neurological and psychiatric conditions such as autism and schizophrenia perceive the world. With respect to *clinical* experience prior to training, I had relatively little. I'd spent some time doing bank-work as a support worker on a forensic ward, and one year as an assistant psychologist working with people with a dual diagnosis (psychosis and substance use), also in a forensic service. Thus, I came to clinical training with considerable research experience, but relatively little experience of direct clinical work.

Selection of a project

When thinking about a potential topic for a thesis I initially explored the possibility of undertaking a qualitative study into the role of music in the identity of men in forensic populations. This was driven by a long-standing passion for music and conviction in its therapeutic potential, as well as the experience of setting up and facilitating participatory music groups on forensic wards. The appeal of such a project was that it represented something very different to my previous research experience, but also, the fact that it would involve learning a completely different

(i.e. *qualitative*) experimental approach, which might open up novel avenues for research after training.

In parallel however, I wondered whether I would be a fool *not* to work to my previous experience and strengths. My reasoning was that clinical skills were what I really lacked, and hence this should represent my main focus during training. In the end, I approached my eventual primary supervisor (VH) to ask if he knew of any existing large data-sets in the field of psychosis that I might be able to work on. In this way, I reasoned, I'd be able to draw –and build- upon previous experience in psychosis research and quantitative approaches, whilst learning new, more advanced statistical techniques that are made possible by working with large data-sets. As luck would have it VH said that he *did* have just such a project.

The research process – empirical paper

Data collection / pre-processing

Although I did not have to collect the raw data for the study, getting it into a state that was ready for analysis proved to be more challenging and time-consuming than I had envisaged. The individual- level data were stored on two large databases at Queen Square, which had to be tidied and integrated. Each held a huge amount of clinical and demographic information, symptom scores, results from tests of cognitive and neuropsychological function, as well as data from –or relating to- ERP and fMRI recordings. Further, these were not stored in identical formats. Simply identifying and familiarising myself with all of the relevant measures, many of which were coded in the form of acronyms only, took considerable time, as did identifying gaps in the data and relevant measures that had a sufficient number of cases to warrant inclusion.

Participants' addresses and postcodes were held separately on a series of hand-written library cards, with individuals only identifiable by anonymised, multi-digit codes. To preserve anonymity and confidentiality these data had to be entered into another database, where they could be stored separately from all other individual-level information; the two data-sets were then only integrated once addresses had been converted into neighbourhood level indices such as deprivation and inequality, which did not include any identifiable information.

Getting the ward- level indices into a useable format *also* proved more challenging than I had envisaged, at least initially. These data were held in various different forms across multiple websites, e.g. Nomis (Office for National Statistics, n.d.) and Infuse (UK Data Service Census Support, n.d.), some of which were archived and not stored in an immediately intuitive form. The data then had to be cross-referenced with a series of look-up tables that facilitated conversion of postcodes into relevant statistical ward codes, so that appropriate scores for the various indices of interest, e.g. deprivation and population size, could be extracted. In retrospect, this is not a particularly complex task; however, there was a lot of trial-and-error and sifting through government websites before I identified the relevant databases and most efficient methods to access / transfer the data. One mistake that I made at the outset was to try and extract data *only* for the regions of England that I was interested in. I had thought that downloading and processing data for the *whole* country would be too cumbersome and result in regular software crashes, since I ran all the analyses from home by remote connection to UCL computers. In fact, this was not the case, and in the end I had to extract data for the *whole* of England anyway, as I wanted to get a sense of how typical the study area was as a region, i.e. relative to the rest of the nation.

Analyses

Running the analyses themselves was not *overly* complex, as I have a fair amount of experience in statistics, programming and the use of statistical software packages. I also had support from an external supervisor with extensive experience in multi-level modelling (JK).

What I found more challenging, however, was the myriad of small -but what seemed like potentially critical- decisions that had to be made at each stage of the analyses; for example, which rotation method to use to extract components in the principal components analysis (PCA) or which variables to add *a priori* to regression analysis models. In theory, the 'correct' choice *should* fall neatly out of the identified research hypotheses and structure of the data. In practice, however, my experience is that more often than not, each option available at a given decision point in the research process typically presents a number of advantages and disadvantages that have to be carefully weighed against one another. Consequently, these decisions are often more arbitrary, or at least more open to debate, than one might hope. This is particularly true for more complex procedures with multiple stages of analyses.

Through my reading and discussion with JK I learned a number of more principled approaches to making some of these decisions, for example, the optimal way to determine the order in which variables should be added to a regression model. I also checked to see if analysing the data in *different* ways would affect the results. For example, as reported in the empirical paper, I undertook a PCA of item- level SAPS and SANS scores *as well as* a PCA of global symptom severity ratings; I also re-ran all regressions using complete case analyses. Whilst analysing the same data in a number of different ways inflates the likelihood of a type I error, i.e. a false positive, the purpose of this approach was not to 'hunt' for significant effects, but instead, to openly report any divergence or convergence in findings and, in the

process, get a sense of their stability. Reassuringly, the main findings were extremely robust and emerged irrespective of which variant of the analyses was applied.

In retrospect, I think that if I was beginning the process of conducting research for the thesis afresh, I would seek face-to-face support from my external supervisor (JK) -whose expertise lies in the field of epidemiological statistics- earlier on during the process. Due to my background in research, I felt confident working independently, and did not seek much support at the start. This was completely my own error, however, since JK and VH were both extremely supportive and available for advice or assistance whenever I did contact them.

Write-up

Having worked in academia previously I have had to become quite self-disciplined; my experience is that there are fewer externally-imposed pressures and structures in academia than in most other working environments, at least as a PhD student or postdoctoral researcher. Consequently, I am used to setting my own short-term goals and following self-imposed dead-lines. In fact, the main challenge I found, particularly as deadlines approached, was working from home. Whilst I enjoyed the freedom this entailed, as well as the lack of a commute, I think I struggled with the lack of a defined end to the working day or a clear distinction between home- and work-life. I often found myself working overly long hours, forgetting to eat and failing to take adequate breaks. By the end of the day I was often working beyond the window within which I was truly effective, and occasionally, struggled to get to sleep at night, my head whirring with thoughts.

This experience is not unique to this context however. I have had to reflect on a similar process at play in a number of areas of my life. I know that I have a relatively

obsessive personality, and that I tend to become passionate and engaged with whatever it is I am working on -or interested in- at the time. The *advantages* of this are that I enjoy what I do, plus it pushes me to learn more. The disadvantages are that it can leave me feeling over-stretched and susceptible to 'burn-out' at times. Over the years, I have learnt a number of ways to manage this, e.g. daily meditation, time spent outdoors, regular breaks and playing music to relax, although the more challenging aspect seems to be following my own advice.

Another thing that I found quite challenging during the process of working on the thesis was managing all the other competing demands in parallel: clinical work, case reports, revision, exams and background reading, although these became fewer towards the end of the final year. As I mentioned, I like to *submerge* myself in whatever work I am doing, and as a result, I often found myself wishing that I had longer chunks of time to focus on the thesis. I find that switching between tasks entails a loss of time as I have to reacquaint myself with the relevant material and get back into a particular frame of mind required for each task. In terms of managing this however, I found keeping a research log invaluable, as this allowed me to get back up to speed with what I was doing much faster following a transition between tasks. I only wish I had been more diligent in the use of a logbook as a PhD student / postdoctoral worker, and it is definitely something I will be taking forward with me in future research.

Other reflections

When I initially embarked upon the thesis I think I was quite unbiased with regards to the relative role that inequality and other economic factors play in the etiology of mental health difficulties. I was acquainted with -and been interested by- some of the social constructionist critiques of medical models of psychosis, e.g. the work of Mary Boyle (2002), and was aware of some of the literature on the role of trauma,

minority status and urbanicity in schizophrenia (see Van Os, Rutten, and Kenis (2010) for discussion), but had read very little knowledge of the research on the wider impact of environmental factors on mental health. As described, I came to the topic quite circuitously through an interest in psychosis, and therefore started from a position of relative ignorance with respect to the broader field.

As I read more of the literature on inequality, particularly the writings of Wilkinson and Pickett (2009), however, I found myself becoming increasingly drawn towards one side of the debate, i.e. in support of the Income Inequality Hypothesis (IIH). I was drawn to this side primarily, I suspect, because it resonated with my own political views and sense of justice, rather than because of the strength of the arguments made by Wilkinson and other researchers. In other words, I was falling prey to the confirmation bias (Nickerson, 1998), i.e. I was more receptive to -and less critical of- arguments that supported my own pre-existing beliefs. After reading a number of critiques of the work of Wilkinson and colleagues, e.g. Snowdon (2010), however, I found that my position began to shift. I slowly became convinced that, in an attempt to bolster their argument -whether consciously or unconsciously- Wilkinson and colleagues had presented a selective picture of the data, presumably reflecting their own susceptibility to the confirmation bias. Ironically, I think that this triggered a different type of error in my thinking: the fallacy fallacy (Pope & Vasquez, 2003). The fallacy fallacy involves the outright rejection of the *conclusions* of an argument solely because the argument contains a flaw. Thus, I began to confound limitations in Wilkinson and Pickett's (2009) methodology with an assumption that the hypothesis they advocated must *therefore* be incorrect.

Ultimately, however, after submerging myself in the *original* studies for many months, my position shifted once more, and I became convinced by the weight of the evidence. I came to the conclusion that the IIH is in all likelihood *essentially*

correct, *in spite of* the limitations of some of the arguments raised in its service. Reassuringly, a similar conclusion seems to have been reached by a number of independent researchers who have reviewed related data on health outcomes and inequality; see Rowlingson (2011) for example.

Reflecting back on my own shifting position throughout the process of undertaking the research, a number of issues and thoughts come to mind. First, it brings into focus the challenges involved in trying to remain truly impartial, i.e. unbiased, in the research process. This seems to be particularly true in the context of subject matter that is so politically loaded and hence likely to trigger strong beliefs and emotions (Jung, Wranke, Hamburger, & Knauff, 2014). I don't think this reflects a fundamental flaw in the scientific method so much as the simple reality that it is undertaken by imperfect humans with all the cognitive biases and blind-spots that psychology has shown plagues our thinking (Kahneman, 2011). Instead, it reinforces the need for a more critical and nuanced appraisal of the research base, as well as the importance of checks and balances that are used to identify and minimise bias at *all* stages of the research process, from pre-registration of trials to double-blind methodologies and blinded reviewing of grant applications and manuscript submissions (Pannucci & Wilkins, 2010).

Finally, the social implications of the field of research that I chose as the subject of my thesis has brought me into contact with a much more socially engaged and politically active body of literature, e.g. the writings of the Psychologists Against Austerity, now called Psychologists for Social Change (Psychologists for Social Change, n.d.). This has forced me to think more deeply about the wider roles, responsibilities and ethics of working as a psychologist *and* researcher. For example, the extent to which psychologists should –or should not- be taking a more active role in shaping policy; see McGrath, Walker, and Jones (2016). I have mixed

feelings about this. On the one hand I find the idea of using my training to be an active driver of social change exciting and invigorating. I also think that a convincing argument can be made that it is impossible *not* to take position; to do nothing is to be complicit with the system within which you are working, for example. However, I am also vaguely troubled by the idea of a psychological profession that becomes *overly* enmeshed with politics, and wonder whether our responsibility to be socially and politically active lies in our roles as citizens rather than as professionals; see Haeny (2014) for discussion.

I also wonder whether a politicisation of psychology would threaten its objectivity, an issue that seems all the more relevant in this "post-truth" era of "alternative facts". For example, a number of researchers have highlighted a growing lack of political diversity within psychology, with the political left coming to dominant the social sciences and humanities (Duarte et al., 2014). In the US in the 1920's, the likelihood that a psychology professor self-identified as conservative or liberal was roughly equal; by 2006, 84% identified as liberal compared to just 8% who identified as conservative, see Figure 1 in Duarte et al. (2014, p.3). The authors suggest that this growing homogeneity of viewpoint threatens the research process, particularly in areas that are of interest to the political left, e.g. gender, race, power and *inequality.* A specific risk that they identify is that researchers may concentrate on topics that validate the "liberal progress narrative" (Duarte et al., 2014, p.5). Whilst I probably subscribe to this narrative of liberal progress, I believe that policy should be driven by facts and evidence of what works rather than ideology, no matter how well intentioned the underlying ideology, i.e. "rational politics" rather than party politics (Tsipursky, 2017). Thus, recent research suggests that liberals remain as firmly entrenched in their ideological bubbles as conservatives (Frimer, Skitka, & Motyl, 2017).

Conclusions

Reflecting back on the process of undertaking research for the empirical and review papers, I am pleased with the topics I chose. The experience of working in these areas has broadened my knowledge of statistical methodologies and introduced me to a whole new body of literature that I would probably not have engaged with otherwise, or certainly not to the same degree. More profoundly, however, I think that the experience made me reflect more on the broader roles of a clinical psychologist, as well as the responsibilities and ethical dilemmas that come with clinical practice *and* applied research.

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Appendix 1– Full List of Search Terms Used

[“income inequality” OR “relative deprivation” OR “gini coefficient” OR “generalised entropy” OR “Atkinson index” OR “decile ratio” OR “kakwani progressivity” OR “proportion of total income earned” OR “Robin Hood index” OR “pietra ratio” OR “sen poverty measure”]

AND

[“mental disorder” OR “mental illness” OR “mental health” OR “psychological disorder” OR “personality disorder” OR “schizophrenia” OR “schizophrenic” OR “psychosis” OR “psychotic” OR ”bipolar” OR “schizoaffective” OR “manic depression” OR “affective disorder” OR “depression” OR “anxiety” OR “PTSD” OR “post traumatic stress disorder” OR “eating disorder” OR “social phobia” OR “social anxiety” OR “panic” OR “generalised anxiety disorder” OR “GAD” OR “obsessive compulsive disorder” OR “OCD” OR “psychological disturbance” OR “emotional problem” OR “schizophreniform” OR “separation anxiety” OR “agoraphobia” OR “body dysmorphic” OR “body dysmorphia” OR “bulimia” OR “binge-eating” OR “hypomania” OR “mania” OR “dysthymia” OR “cyclothymia” OR “paranoia“ OR “paranoid“ OR “delusional“ OR “schizotypal“ OR “schizoid“ OR “dissocial“ OR “emotionally unstable“ OR “histrionic“ OR “anankastic“ OR “avoidant“ OR “dependent personality disorder” OR “dissociative“ OR “antisocial“ OR “borderline“].

Appendix 2 – Additional information on studies included in the review

Supplementary Table 1 Studies included in the review – additional information. The full list of studies included in this review is presented along with a number of additional variables not included in the main body of the thesis: male to female ratio, Q1 (validity of variables), Q2 (adequate sample size), Q3 (appropriate control variables), Q4 (optimal analyses), QI (quality index, i.e. the sum of Q1-4), method of data collection, sampling strategy, longitudinal study (0=no, 1=yes), level of analysis (1=individual-level analysis, 2=higher-order level analysis, 3=multi-level analysis). NA=data not available.

Study	Country / focus of study	MH variable	Male to female ratio	Age	Q1	Q2	Q3	Q4	QI	Method of data collection	Sampling strategy	Longitudinal study	Level of analysis
(Ahern & Galea, 2006)	US / New York City	6-month prevalence of depression	0.78	>=18	1	1	1	1	4	Phone-based interview	Probability sample (random dial; stratified by geography); over-sampling of World Trade Centre (WTC) site	0	3
(Bechtel et al., 2012)	Australia / nationwide	General mental health Sxs	NA	>=15	1	1	0	0	2	Face-to-face interview	Probability sample (stratified by geography); under-sampling of remote areas	0	1
(Boydell et al., 2004)	UK / South London	10-year Incidence of psychosis	1.31	NA	0	0	0	1	1	Review of clinical records	Convenience sample (all incident cases presenting to services)	0	3
(Burns & Esterhuizen, 2008)	South Africa / district of uMgungundlovu	One-year incidence of FEP	2.4	15-49	0	0	0	0	0	Review of clinical records	Convenience sample (all incident cases presenting to services)	0	2
(Drukker et al., 2004)	Netherlands / City of Maastricht	General MH Sxs	NA	35-45	0	1	0	1	2	Distributed questionnaires	Convenience sample / cohort study (parents of children presenting to services)	0	3
(Fernandez-Nino et al., 2014)	Mexico / nationwide	Caseness for depression	0.87	>=60	1	1	1	1	4	Face-to-face interview	Probability sample (stratified by geography)	0	3
(Fiscella & Franks, 2000)	US / nationwide	Sxs of depression	NA	25-74	0	1	0	0	1	Face-to-face interview	Probability sample (stratified by geography & demographics); over-sampling of poor areas, women of child-bearing age & the elderly	1	1
(Fone et al., 2013)	Wales / nationwide	General MH Sxs (& caseness)	0.87	18-74	1	1	1	1	4	Face-to-face interview	Probability sample (stratified by geography)	0	3

Study	Country / focus of study	MH variable	Male to female ratio	Age	Q1	Q2	Q3	Q4	QI	Method of data collection	Sampling strategy	Longitudinal study	Level of analysis
(Gresenz et al., 2001)	US / nationwide	Caseness for anxiety or depression, general MH Sxs	NA	<65	1	1	1	1	4	Phone-based interview	Probability sample (stratified by geography & demographics); over-sampling of individuals with mental illness &/or low income	0	3
(Hanandita & Tampubolon, 2014)	Indonesia / nationwide	General MH Sxs (& caseness)	0.93	>=15	1	1	1	0	3	Face-to-face interview	Probability sample (stratified by geography)	0	1
(Henderson et al., 2004)	US / nationwide	Sxs of depression (& caseness)	0.71	18+	1	1	1	0	3	Face-to-face interview	Probability sample (stratified by geography & demographics); over-sampling of black people & 18-29 year olds	0	1
(Kahn et al., 2000)	US / nationwide	Caseness for depression	0	>=15	1	1	0	0	2	Phone-based interview & postal questionnaire	Probability sample (stratified by geography & demographics); over-sampling of mothers of black & low birth-weight infants	0	1
(Kirkbride et al., 2014)	UK / East London	Psychosis incidence	1.54	18-64	1	1	1	1	4	Face-to-face interview	Convenience sample (all incident cases presenting to services)	0	3
(Lee & Park, 2015)	Korea / nationwide	Caseness for depression	0.87	19-60+	1	1	1	1	4	Face-to-face interview	Probability sample (stratified by geography & demographics)	0	3
(Marshall et al., 2014)	UK / nationwide	Caseness for depression	0.83	>=50	1	1	1	1	4	Face-to-face interview	Probability sample (stratified by geography)	0	3
(Messias et al., 2011)	US / nationwide	Caseness for depression	NA	NA	1	1	0	0	2	Phone-based interview	Probability sample (random dial; stratified by geography)	0	2
(Muramatsu, 2003)	US / nationwide	Sxs of depression	0.61	70-103	1	1	1	1	4	Face-to-face and phone-based interview	Probability sample (stratified by geography & demographics); over-sampling of residents of Florida & minority groups	0	3
(Pabayo et al., 2015)	US / nationwide	Incidence of depression	0.72	>=18	1	1	1	1	4	Face-to-face interview	Probability sample (stratified by geography)	1	3
(Sturm & Gresenz, 2002)	US / nationwide	Caseness for depression or anxiety	NA	NA	1	1	0	0	2	Phone-based interview	Probability sample (random dial; stratified by geography)	0	1

Study	Country / focus of study	MH variable	Male to female ratio	Age	Q1	Q2	Q3	Q4	QI	Method of data collection	Sampling strategy	Longitudinal study	Level of analysis
(Weich et al., 2001)	England, Wales & Scotland	Caseness for general MH	NA	16-75	1	0	1	0	2	Face-to-face and phone-based interview	Probability sample (stratified by geography)	0	1
(Zimmerman & Bell, 2006)	US / nationwide	Caseness for depression	NA	40-45	0	NA	1	0	NA	Face-to-face and phone-based interview	Probability sample (stratified by geography & demographics); over-sampling of black people & Latinos	0	1

Appendix 3 – Original consent form

WEST LONDON FIRST EPISODE STUDY

[redacted]

Version 2.02

RESEARCH CONSENT FORM

Title of project: OUTCOME STUDY OF FIRST EPISODE PSYCHOSIS

Please circle 'YES' or 'NO'

Have you read the patient information sheet? **YES NO**

Have you had the opportunity to ask questions and discuss the study? **YES NO**

Have you received satisfactory answers to your questions? **YES NO**

Who have you spoken to?

Do you understand that your participation is entirely voluntary, that you are free to withdraw from the study at any time, and that if you choose not to enter the study, your medical care and legal rights will not be affected in any way? **YES NO**

Are you willing to allow access to your medical records for the purposes of this study, and for this access to continue should you decide to take part in the actual research interviews? **YES NO**

Do you agree to take part in the study? **YES NO**

Name of patient	Date	Signature
Name of person taking consent (if different from researcher)	Date	Signature
Researcher	Date	Signature

Appendix 4 – Ethics committee response letter

RIVERSIDE RESEARCH ETHICS COMMITTEE

Pharmacy Offices Lower Ground Floor
CHELSEA WESTMINSTER HOSPITAL
369 Fulham Road London SW10 9NH
Tel: 020 8846 6855 Fax: 020 8846 6860
Email: riverside.ethics@chelwest.nhs.uk

Thursday, April 25, 2002

Dr Eileen Joyce

Imperial College School of Medicine
Charing Cross Campus
St Dunstan's Road
London SW6 8RP

Dear Dr Joyce,

RREC 3006 - Cognitive and Neuroimaging Abnormalities in First-Episode Psychosis: The West London Longitudinal First Episode Study.

Thank you for your application. The Chairman of the Riverside Research Ethics Committee, Dr Charles Mackworth-Young, has asked me to write to inform you that the above study has now been approved.

Please note the following conditions which form part of this approval:

[1] Your study has been assigned a unique reference number. This number must be quoted in any correspondence with the Committee concerning this study.

[2] This approval is for a limited period only. A letter from the principal investigator will be required in order to extend this period of approval.

[3] Any changes to the protocol or investigator team must be notified to the Committee. Such changes may not be implemented without the Committee's approval.

[4] Any revised study documents submitted must be given a new version number/date.

[5] For projects with an expected duration of more than one year, an annual report from the principal investigator will be required. This will enable the Committee to maintain a full record of research.

[6] The Committee must be advised when a project is concluded and should be sent one copy of any publication arising from your study, or a summary if there is to be no publication.

[7] The Committee should be notified immediately of any serious adverse events that are believed to be study drug related or if the entire study is terminated prematurely.

[8] Please note that research conducted on NHS Trust premises must receive the approval of the relevant Research and Development department. Approval by the Committee for your project does not remove your responsibility to obtain this approval.

[9] You are responsible for consulting with colleagues and/or other groups who may be involved or affected by the research, e.g., extra work for laboratories. Approval by the Committee for your project does not remove your responsibility to negotiate such factors with your colleagues.

[10] You must ensure that nursing and other staff are made aware that research in progress on patients with whom they are concerned has been approved by the Committee.

[11] Pharmacy must be told about any drugs and all drug trials, and must be given the responsibility of receiving and dispensing any trial drug.

[12] All documents relating to the study, including Consent Forms for each patient (if applicable), must be stored securely and in such a way that they are readily identifiable and accessible. The Committee will be conducting random checks on the conduct of studies, and these will include inspection of documents.

May I take this opportunity to wish you well in your research. If any doubts or problems of an unexpected nature arise, please feel free to contact me at any time.

Yours sincerely

[redacted]

Miss Katherine Bolton
Administrator
Riverside Research Ethics Committee
(On behalf of the Chairman, Dr C G Mackworth-Young MA MD FRCP)

The Riverside Research Ethics Committee has approved the following:

RREC 3006 - Cognitive and Neuroimaging Abnormalities in First-Episode Psychosis: The West London Longitudinal First Episode Study.
Dr E Joyce; Prof T Barnes; Prof M Ron; Dr G Barker; Prof T Burns; Mr S Mutsatsa
This study was considered by the full Committee.
This study was first approved on the: 25/04/2002.
Approval for this study expires on the: 25/04/2003.

Study History:

	Comments
Application Form (15/04/02)	Approved 25/04/02
Patient Information Sheet (Version 3: April 2002)	
Control Group Information Sheet (Version 3: April 2002)	
Research Consent Form (Version 1.02)	
Advertisement (Page 1, Page 2)	
Protocol (15/01/02)	
Correspondence from Dr Joyce (15/04/02)	

Appendix 5 – Calculating indices of inequality

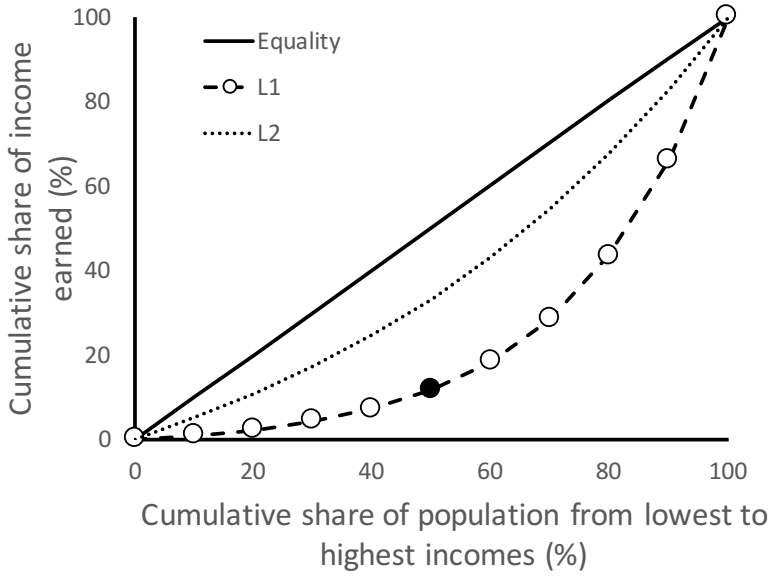
The Gini coefficient is the most commonly used measure of inequality, and is derived from the Lorenz curve; see Supplementary Figure 1. Consider a simple example in which the aim is to calculate the Gini coefficient for the household income of ten homes. The Lorenz curve plots the cumulative share of the population across the households after they have been ordered from lowest to highest income (along the abscissa), against the cumulative share of income earned by these households (along the ordinate axis). In a perfectly equal society where each household earns the same amount, the Lorenz curve follows the straight line of equality (solid black line). Thus, 50% of the lowest income houses would together earn 50% of the total income.

In an unequal society, however, with income disparity between the rich and the poor, the Lorenz curve will deviate from equality. For instance, consider curve L1 (dashed line) in Supplementary Figure 1: the black data-point indicates that the 50% lowest income houses together only earn around 10% the total income. By inference therefore, the top 50% together earn 90% of the total.

The Gini coefficient measures the extent to which this Lorenz curve deviates from equality, i.e. the area between L1 and the line of equality in the previous example. A Gini coefficient of zero represents perfect equality, i.e. all houses earn the same amount, whereas a Gini coefficient that approximates 1 represents maximum inequality. In this example, L1 is associated with a Gini coefficient of 0.28. For reference, another hypothetical data-set with an intermediate level of inequality (Gini coefficient=0.11) is also presented in the form of a second Lorenz curve (L2; dotted line).

The Lorenz curve and Gini coefficient can be used to generate a measure of inequality using any number of different indices of wealth / deprivation, and can be

estimated at different levels of analysis. In this thesis, Gini coefficients were calculated using income deprivation (ID) and the Index of Multiple Deprivation (IMD) rather than household income. Lower layer super output areas (LSOAs) were used as the unit of analysis, so that estimates of inequality were derived for each ward based on the distribution of deprivation across its composite LSOAs, as described previously by Kirkbride et al. (2014).



Supplementary Figure 1 Theoretical Lorenz curves for the household incomes of ten houses. The solid black line represents the line of equality, where all households have the same incomes (Gini coefficient=0). The dashed (L1) and dotted lines (L2) represent Lorenz curves with associated Gini coefficients of 0.28 and 0.11, respectively.

Appendix 6 – Boroughs included

Supplementary Table 2 Boroughs included in the study. All boroughs included in the study are broken down by the number of wards (total n=118) and number of participants (total n=345) in each, based on the self-reported residency of each participant at the time of first presentation to services. Note: whilst the majority of the participants reported residencies within boroughs in which they were recruited (recruitment boroughs; n=325, 94.2%) a subset of participants reported residencies that were in surrounding boroughs (n=20, 5.8%).

Borough	Borough	No. wards	No. participants
Recruitment boroughs	Ealing	21	64
	Hammersmith and Fulham	15	61
	Hounslow	3	6
	Kingston Upon Thames	14	41
	Merton	13	15
	Richmond Upon Thames	18	92
	Sutton	2	3
	Wandsworth	15	43
Surrounding boroughs	Croydon	1	1
	Elmbridge	4	7
	Harrow	1	1
	Kensington and Chelsea	5	5
	Lambeth	3	3
	Lewisham	1	1
	Westminster	2	2
	Total	118	345

Appendix 7 - Global symptom severity rating level analysis

To assess the robustness of the primary PCAs undertaken, a third PCA was undertaken using participant global symptom severity rating scores. This followed the methods described by John et al. (2003), and analysed global rating scores for the nine different dimensions of the SAPS (hallucinations, delusions, bizarre behaviour and positive formal thought disorder) and SANS (affective flattening of blunting, alogia, avolition / apathy, anhedonia / asociality and attention). A VARIMAX rotation was used since this level of analysis can be thought of as broadly analagous to the second (high-order) analysis described by Peralta et al. (1997).

Once again, the factorability of the data was assessed. Inspection of the correlation matrix indicated that all individual variables included in the analysis showed a small sized correlation or greater, i.e. $r>0.1$, with at least one other variable, and seven out of nine showed a medium sized correlation, i.e. $r>0.3$. Further, no excessively large correlations ($r>0.9$) were seen. All values on the diagonals of the anti-image correlation matrix exceeded 0.5, indicating that items were sufficiently correlated with extracted components to warrant inclusion in the analysis. Finally, a Kaiser-Meyer-Olkin value of 0.76 was obtained (above the recommended value of 0.5 (Kaiser, 1974)), indicating an adequate sample size, and Bartlett's Test of Sphericity was found to be significant ($\text{chi-squared}_{(36)}=738.77$, $P<0.001$). Consequently, a PCA was deemed appropriate for this data-set.

The PCA resulted in the extraction of three components with eigenvectors >1, which together explained 63.73% of the variance in the data. Supplementary Table 3 shows the PCA structure with variable loadings.

Extracted components were relatively straightforward to interpret. The first component (negative symptoms), which explained 34.26% of the variance in the data was comprised of loadings from affective flattening, alogia, avolition, anhedonia

and attention. The second component (disorganized symptoms), which explained 16% of the variance in the data, was comprised of loadings from delusions, bizarre behaviour and positive formal thought disorder. Finally, the third component (positive symptoms), which explained 13.46% of the variance in the data, was comprised of loadings from hallucinations and delusions.

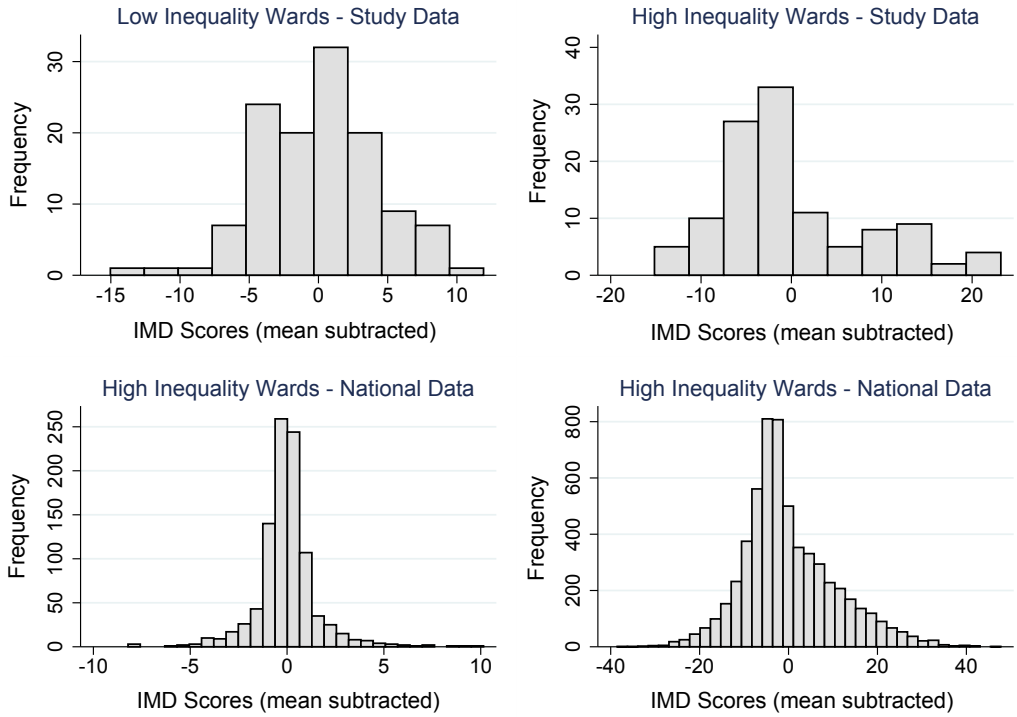
Supplementary Table 3 Component loadings (rotated matrix) for all nine variables included in the global symptom severity rating level principal component analysis. Loadings >0.4 in magnitude are shown in full contrast.

	F1	F2	F3
(1) Hallucinations	0.12	-0.19	0.82
(2) Delusions	-0.06	0.41	0.71
(3) Bizarre behaviour	0.02	0.82	0.14
(4) Positive formal thought disorder	0.26	0.74	-0.11
(5) Affective flattening	0.80	0.01	0.11
(6) Alogia	0.78	0.17	-0.04
(7) Avolition / apathy	0.68	0.27	0.16
(8) Anhedonia / asociality	0.74	-0.14	0.05
(9) Attention	0.69	0.29	-0.17

Appendix 8 - Characterisation of wards included in the study

Supplementary Figure 2 plots the distribution of IMD scores (pooled) for all composite LSOAs in low inequality wards (<1Z from the mean; left panels) and high inequality wards (>1Z from the mean; right panels), for wards included in the analyses (upper panels; study data), as well as national data (lower panels). LSOA IMD scores have been centred about zero by subtracting individual scores (for each ward) from the ward mean, in order to facilitate comparison of distributions. Only wards with more than one composite LSOA are included.

For the study data, there were 18 low inequality wards (n=123 composite LSOAs) and 17 high inequality wards (n=114 LSOAs); in the high inequality distribution, the modal IMD value was less than zero, with 64.91% of the wards falling below zero. For the national data, there were 385 low inequality wards (n=969 LSOAs) and 1100 high inequality wards (n=5852 LSOAs); for the high inequality distribution, once again, the modal IMD score was less than zero, with 60.2% of the wards falling below zero. In contrast, for both the study and national data, low inequality distributions were broadly centred about zero.



Supplementary Figure 2 Distribution of LSOA-level deprivation scores. The distributions of LSOA-level deprivation scores are shown for low inequality and high inequality wards within wards included in the analyses (study data) as well as all wards in England (national data).

Appendix 9 - Pair-wise correlations

Supplementary Table 4 Pair-wise (simple) correlations between symptom dimension scores. Values provided represent Spearman's Rho (r_s) and associated (uncorrected) p-values (in parentheses). Significant correlations at a corrected alpha level of 0.01 are shown in bold (corrected for seven multiple comparisons).

	Negative	Positive	Disorganized	Depressive
Negative	1			
Positive	0.06 (0.26)	1		
Disorganized	-0.08 (0.13)	0.01 (0.89)	1	
Depressive	**0.16 (<0.01)**	0.14 (0.01)	**-0.21(<0.001)**	1