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Neighbourhood deprivation and intersectional inequalities in biomarkers of healthy ageing in England

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ABSTRACT

While social and spatial determinants of biomarkers have been reported, no previous study has examined both together within an intersectional perspective. We present a novel extension of quantitative intersectional analyses using cross-classified multilevel models to explore how intersectional positions and neighbourhood deprivation are associated with biomarkers, using baseline UK Biobank data (collected from 2006 to 2010). Our results suggest intersectional inequalities in biomarkers of healthy ageing are mostly established by age 40–49, but different intersections show different relationships with deprivation. Our study suggests that certain biosocial pathways are more strongly implicated in how neighbourhoods and intersectional positions affect healthy ageing than others.

1. Background

Tackling health inequalities between places is a key government public health priority in England (NHS England, 2019; Public Health England, 2017). There is a 7.7 and 6.1 year gap in life expectancy for males and females respectively between lower-layer super output areas (LSOAs) in the most and least deprived quintiles (Marmot et al., 2020). These inequalities have been persistent over time and across multiple health outcomes. The last 30 years of research has dissected the numerous pathways and mechanisms through which spatial context impacts health and wellbeing. While diverse individual, demographic, social and spatial determinants have been identified, much of this research focuses on their overall effect and does not fully consider how place-related effects may vary across population subgroups, or how health may vary between subgroups of the population living in the same locations. There has been recent renewed interest in this topic following the publication of the much-critiqued Sewell Report which claimed that many ethnic inequalities are solely explained by deprivation (Commission on Race and Ethnic Disparities, 2021), ignoring processes of racialisation and racial discrimination that are deeply embedded in society. Further, the UK government's £4.8b Levelling up Fund (HM Treasury, 2021) aimed at tackling place-based inequality has been criticised for not taking into account deprivation in allocating funding despite its importance for health outcomes (The King's Fund, 2021).

Research has evaluated whether spatial context matters for health beyond an individual's personal (or compositional) circumstances, and findings are generally consistent in demonstrating that contextual effects matter independent of compositional ones (Arcaya et al., 2016; Pickett and Pearl, 2001; Riva et al., 2007; van Ham et al., 2012). However, it is important to think about these factors holistically. Macintyre (2007) argues that the interaction of context *and* composition produces greater disadvantage for some groups. For example, while individuals with low income may not be able to afford a car to access health services, this issue is compounded if they live in an area with poor public transport links or without services within walking distance. While this argument is appealing, few studies have robustly explored it beyond single combinations of characteristics that may not adequately capture the full context of an individual's situation.

Neighbourhoods influence health because they constitute both *physical* and *social environments* (Roux and Mair, 2010). Physical environmental factors include traffic, public transport, walkability, housing, green space and access to services and healthy foods (Green et al., 2018; Roux and Mair, 2010). Social environmental factors include cohesion, trust, segregation, crime and safety. Some neighbourhood features have

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a direct physiological effect such as air pollution. Otherwise, both physical and social environmental factors influence health through two main pathways (Roux and Mair, 2010). The first is through psychosocial stress. Some features of neighbourhoods can induce stress including violent crime, low social cohesion, lack of green space or cramped housing. The second pathway is behavioural. Neighbourhood features such as healthy food access or walkability can have a direct effect on health-related behaviours. In addition, other factors, such as high levels of crime, can lead to health-damaging behaviours – such as alcohol use or smoking – as coping mechanisms via the stress pathway (Green et al., 2018; Roux and Mair, 2010). Residential segregation and inequalities in resources across neighbourhoods act as structural mechanisms that produce differential exposures to neighbourhood physical and social environments (Roux and Mair, 2010).

We have argued previously (Green et al., 2017; Holman et al., 2020; Holman and Walker, 2020) that to fully understand the contextual and compositional determinants of health inequalities we need to embrace 'intersectionality' approaches. Originating in Black Feminist thought (Collins, 2008; Crenshaw, 1989), the approach focuses on how systems of social discrimination (e.g. sexism, racism, ageism) cannot be viewed independently but layer and interact to shape the experiences of people based on their multi-dimensional intersectional positions. Different intersectional positions (defined by combinations of social and spatial characteristics) are potentially associated with differential: access to power, prestige and resources, social identities and exposure to and experiences of structural and inter-personal discrimination. To understand the social determinants of health inequalities, we need to consider how they impact health across these multi-dimensional intersectional positions.

Whilst intersectional thinking is not new to health geography (Hopkins, 2019; Valentine, 2007), geographers have tended to under-theorise place, space and scale in intersectional thinking (Bambra, 2022). Conversely, although intersectionality is rooted in a concern with contexts, structures and institutions, quantitative intersectional analyses of place-as-context are rare (Evans, 2019). Considering place as an aspect of intersectionality presents significant theoretical challenges particularly with regards to issues of power and identity (Bambra, 2022). Place operates at multiple scales – locally, as well as regionally and (inter)nationally – suggesting a need to integrate understanding of how power structures, processes and institutions operate across contexts vertically (Bambra, 2022). Social identities are similarly affected by scale; they operate within neighbourhoods, and are shaped by city, regional and national processes. One potential operationalisation is to consider how the same intersectional position is experienced differently, and affects health differently, in different places (Evans, 2019; Hopkins, 2019). Black Feminist literature offers a springboard for this kind of approach given its rich and established theorising on how social divisions have varied consequences for health inequalities across space and time (Bowleg, 2021). Bringing in a geographical perspective here adds insight around how social identities are differentially affected by place. Place is a source of social identity and belonging, or in/exclusion, which depends on our social characteristics (Antonsich, 2010). Following Yuval-Davis' (2006) insight that different social divisions are ontologically distinct, being rooted in different assumptions, processes and boundaries, place is not 'just another' intersectional axis of inequality but one that also has a firmly ecological basis; place is therefore both intersectional and contextual. For this reason, neighbourhood deprivation (an established way of operationalising place effects relating to, for instance, cohesion, trust, segregation, crime and safety) is likely to be associated with health both because of how it is associated with differential physical exposures and access to resources, but also because neighbourhoods are social environments, entailing processes of identity, power relationships, social roles, stigma and exclusion. Places are inherently political – reflecting macro axes of power – and are a key hallmark of social divisions.

A key concern of Black Feminist and intersectional literature is the way in which social categories result in unfair treatment i.e. social discrimination. Discrimination is therefore central to an intersectional health perspective (Gkiouleka et al., 2018). Discrimination is a strong driver of health because it shapes access to various types of resources, including material, educational, occupational, political, and healthcare; It is also a source of social stress via diminished access to social status, stigmatised identity, and experience of covert and overt insults, manifested as verbal abuse and physical attacks or brief and commonplace microaggressions. Discrimination therefore drives psychological well-being and health behaviours (Krieger, 2014). Discrimination is shaped by both spatial and temporal context (Holman and Walker, 2020), helping to explain why different neighbourhoods might result in different intersectional health outcomes. From a life-course perspective, persistent exposure to discrimination and other sources of social stress can result in 'weathering'. Originally conceived by Geronimus (1992) to explain the health deterioration of Black groups at younger ages than their White counterparts in the United States, weathering is thought to result from chronic exposure to social and economic adversity and disadvantage over the life-course. Persistent coping gradually wears down the body across multiple physiological systems (Geronimus et al., 2006). An intersectional, eco-epidemiological (Krieger, 2001) lens opens the possibility that those in different intersectional positions, and in neighbourhoods with differing level of deprivation, experience different rates of weathering. Intersectional inequalities in weathering potentially follow the three classic patterns over the life course: age-as-leveller (inequalities shrink between intersectional subgroups over time), persistent inequality (inequalities remain constant) or cumulative disadvantage (inequalities widen) (Brown et al., 2012).

A recent systematic review found evidence for weathering across contexts measured via multiple biomarkers, though effects varied by biomarker (Forde et al., 2019). The review included studies which found that weathering is more pronounced for Black populations living in lower-socioeconomic-status neighbourhoods, defined by individual or neighbourhood poverty, education, and neighbourhood segregation. Existing studies are typically limited by the use of composite biomarker measures. For example, in the UK context, a recent study found evidence that 'allostatic load', a composite measure of biological response to stress, mediates the pathway between neighbourhood deprivation and health (Prior et al., 2018). A multi-cohort study of English, Portuguese and Swiss data also found an association between neighbourhood deprivation and allostatic load (Ribeiro et al., 2019). Yet the features of neighbourhoods outlined above are likely to affect different biological systems – and their biomarkers – in different ways. For example, a high concentration of fast-food outlets might be expected to particularly affect obesity and HbA1c, whilst persistent social stress might instead influence low level inflammation. This would ultimately be expected to result in different chronic disease and healthy ageing outcomes reflecting damage to different bodily systems (Mathers et al., 2015). In the present study we therefore analyse different biomarkers of healthy ageing to shed light on the mechanisms linking intersectional positions and features of neighbourhoods with later life health.

The main aim of our study is to analyse neighbourhood deprivation as an axis of intersectional later life health inequality (as measured by biomarkers of healthy ageing). Sub-aims are to (i) Examine the relative contribution of individual intersectional position and neighbourhood deprivation to biomarkers of healthy ageing, and (ii) Compare age groups to explore potential intersectional age/cohort patterning of biomarkers of healthy ageing. In comparing the results for different age groups, we discuss which of the three aforementioned competing hypotheses of life course health inequality the results are consistent with.

2. Methods

2.1. Data and variables

The UK Biobank is a prospective cohort study of over 500,000 adults

aged 40–69 when they were recruited at baseline (Sudlow et al., 2015). The cross-sectional baseline data were collected from 2006 to 2010 and are analysed in the present study. It contains a range of health-related measures as well as socio-demographic information, including age, gender, educational qualifications, ethnicity and neighbourhood deprivation (measured using Index of Multiple Deprivation (IMD)). Given that IMD scores are not comparable between the UK nations, we restricted the sample to those living in England (n = 430,516). Of these, 9260 were missing data on educational qualifications and 2476 on ethnicity. Given the low proportion of missing data for socio-demographic variables, complete case analysis was used with respect to these variables. Of the remaining 419,773 cases, 33,298 did not belong to one of the ethnic categories of interest (see below), leaving an analytical sample of 386,475.

The UK Biobank is unrepresentative of the UK population. Participants are less deprived, older, more likely to be female, and less likely to be obese, smoke, drink alcohol daily and have self-reported health conditions (Fry et al., 2017). Recent analyses have shown the associations between health behaviours and disease and mortality outcomes are mostly consistent with those from representative samples (with some exceptions) (Stamatakis et al., 2021). For the purpose of the current analysis, which is to understand the relevance of neighbourhoods and neighbourhood deprivation to intersectional patterning in biomarkers of healthy ageing and disease, the unrepresentativeness of the UK Biobank is less problematic given the socio-demographic variation in the sample, but the estimates are only indicative and are not generalisable to the English population. Given the lack of alternative large scale datasets containing biomarker data, we use the UK Biobank with these limitations in mind.

The UK Biobank methodology for matching IMD scores is available online [https://biobank.ndph.ox.ac.uk/showcase/showcase/docs/imd_baseline.pdf], and involved matching participants' lower super output area (LSOA), as indicated by their postcode at baseline, to the version of the IMD (2004, 2007, or 2010) that was closest to (and preceding) their year of recruitment. Quintiles were calculated using the IMD range for England in the corresponding year. While recognising that ethnic categorisations necessarily obscure important elements of intra-group diversity (e.g. relating to migratory history, religion, or language), we employed those that show important patterning in health outcomes and for which sample sizes were sufficiently large: White British, Chinese, Indian, Pakistani, Black Caribbean and Black African. We chose educational qualifications as our indicator of socioeconomic position instead of household income or occupation as qualifications are typically more accurately and completely measured, are an individual level indicator, relevant to participants of all working statuses. Further, formal education is mostly acquired early in life and is therefore most relevant to lifelong healthy ageing. Qualifications were categorised into high (College or University Degree), medium (any qualification except College or University Degree) and low (No listed qualification). Age was split into ten-year bands. We defined the intersections in this study following common conventions used in quantitative intersectional analysis, using different combinations of age, gender, ethnicity, socio-economic position (educational qualifications). We additionally included neighbourhood deprivation (IMD quintile) to represent place. Neighbourhoods were defined using LSOAs, a statistical unit containing ~1500 people. In the sample there were 13,715 LSOAs each with an average of 46 individuals.

We selected biomarkers associated with different bodily systems, processes and pathologies which would therefore be expected to bear the imprint of geographical and intersectional inequalities in different ways, entailing the mechanisms we have outlined above (Table 1). To keep the analysis manageable, we analysed vitality and locomotion aspects of intrinsic capacity (Cesari et al., 2018). Alternative biomarkers available in the analysed data included cholesterol, BMI and lung function. HbA1c was chosen over cholesterol; both are molecular metabolic markers but the former is directly related to a disease (diabetes). Waist circumference was chosen over BMI because the former is better associated with cardiometabolic risk factors and chronic disease (Jayedi et al., 2020). Grip was chosen over lung function; both are measures of vitality but the former is a well-established indicator of overall healthy ageing and frailty (Ho et al., 2019).

Missingness for biomarker data ranged from 0.32% for waist circumference to 9.78% for HbA1c (Table 1). We used multiple imputation to test whether the results were sensitive to missing data. We followed best practice guidelines (Sterne et al., 2009) and used chained equations, including all biomarkers in the model, with all socio-demographic variables as predictors, as well as assessment centre and self-rated health as auxiliary variables. The number of imputations was set to 10, the % missing for the variable with the most missing data (HbA1c) (Sterne et al., 2009). We compared point estimates between models using imputed and non-imputed data using main effect regression models (supplementary material). Although processor and software limitations meant we were unable to run our main models on imputed data, the differences we found with the main effects were negligible and

Table 1
Biomarkers of healthy ageing.

Biomarker	Health implications	Potential neighbourhood pathway	Cut-off
Hba1c (mmol/mol)[1]	A measure of blood glucose concentration over the past two to three months used to diagnose type 2 diabetes and is (World Health Organization, 2011a).	Behavioural (primary); psychosocial (secondary)	>48 mmol/mol indicates diabetes and >42 mmol/mol pre-diabetes (Diabetes UK).
C-reactive protein (mg/L)[2]	A measure of body inflammation and is associated with a range of chronic disease including type 2 diabetes, obesity and metabolic syndrome (Medzhitov, 2008).	Psychosocial (primary); behavioural (secondary)	≥3 (mg/L) indicates systemic inflammation (Benzeval et al., 2014).
Systolic blood pressure (mm Hg)	A well-known marker of cardiovascular disease (Mourad, 2008).	Psychosocial and behavioural	>140 mm HG indicates hypertension.
Waist circumference (cm)	Significantly predictive of general health, type 2 diabetes and osteoarthritis (Darsini et al., 2020).	Behavioural (primary); psychosocial (secondary)	Waist circumference cut-offs are different for men and women due to differences in biological developmental (World Health Organization, 2011b): for men >94 cm is associated with increased risk of metabolic complications, for women >80 cm >102 and > 88 indicate substantially increased risk.
Grip (kg)	Associated with cardiovascular disease, respiratory diseases and cancer (Ho et al., 2019) and is a key marker of sarcopenia and frailty.	Psychosocial and behavioural	Reflects physiological differences between men and women and hence has different cut-offs for what is considered clinically relevant 'weak grip strength'. We follow Dodds et al. (2014) who calculated this figure as <32 kg for men and <19 kg for women.

1 Values of ≥200 mmol/mol were considered outliers and removed.
2 As CRP was highly negatively skewed, we transformed it by adding one before applying a log transformation following Taheri et al. (2007), and transformed it back when presenting the analytical results by exponentiating estimates then subtracting 1. ≥10 (mg/L) were removed as this indicates recent infection (Benzeval et al., 2014).

likely inconsequential with respect to the substantive findings given the relatively small amount of missingness.

2.2. Analysis

Our analysis is deliberately exploratory in nature: we are aiming to explore intersectional and geographical patterns, rather than aiming to test specific hypotheses. We used the multilevel analysis of individual heterogeneity and discriminatory accuracy (MAIHDA) method for analysing inter-categorical intersectionality (Bell et al., 2019; Evans et al., 2018; Jones et al., 2016; Merlo, 2018). Although still an emerging method, MAIHDA has been used to analyse a wide set of outcomes, including biomarkers of healthy ageing using English national survey data (Holman et al., 2020). Rather than nest individuals within higher level units such as neighbourhoods, schools or countries, MAIHDA nests individuals within population subgroups, or intersections, which are defined by combinations of social characteristics, mostly typically gender, socioeconomic position (SEP) and ethnicity (Persmark et al., 2019). Whilst conventional multilevel models differentiate between the variance in a particular outcome that exists at both the individual and the neighbourhood level, for example, MAIHDA models differentiate individual and intersectional variance, or in other words, the extent to which intersectional clustering accounts for the variance in a particular outcome. They are therefore appropriate when the aim is to examine health inequalities at the intersection of multiple social positions (Evans et al., 2018), which conventional multilevel models do not allow for. Although other approaches to modelling intersectional effects exist, such as including interaction terms in regression models, the advantages of MAIHDA are that it goes some way towards solving the issue of multiple testing via shrinkage inherent in multilevel models (Bell et al., 2019) (an important consideration, given the potentially large number of intersectional subgroups). The model is also more scalable, parsimonious and interpretable than conventional approaches involving interaction terms (Green et al., 2017) and performed well in a recent comparison with other quantitative intersectional methods (Mahendran et al., 2022).

In a 2-level null model with intersections specified as random effects at level 2, the intra-class correlation coefficient (ICC) expresses the proportion of the total variance that is explained by intersectional differences. This is usually followed by a model with the social characteristics included as (fixed) main effects in the fixed part of the model. In this model, those fixed effects explain some of the intersectional variance, so the ICC now expresses the proportion of the variance – excluding that caused by main effects – explained by *multiplicative* intersectional clustering. In other words, these differences cannot be explained by the additive effects of the social characteristics used to define the intersections. Further, intersection-level residuals in this model now represent multiplicative divergence from those simple additive effects, which opens the possibility that certain intersections may be associated with synergistic effects for a particular outcome while others may have effects in line with what would be expected given the additive (i.e. the main) effects. We discuss the substantive implications of such effects when discussing our findings further below.

In this paper we make a novel extension to MAIHDA that partitions variance between individual, intersectional and neighbourhood (LSOA) levels. Cross-classified models were used because intersections are not nested within neighbourhoods (nor vice-versa), making a conventional strictly-hierarchical, three-level model inappropriate. Previous studies have demonstrated the utility of cross-classified MAIHDA models (Evans, 2019; Khalaf et al., 2020; Rodriguez-Lopez et al., 2020). Neighbourhood is included as an additional, separate level in the model to account for neighbourhood level variance other than in IMD. However, even with a large sample size of the UK Biobank, including LSOAs as an intersectional variable in the fixed part of the model (as per stage two of the MAIHDA approach described above) is unfeasible given the large number of neighbourhoods, which would also make interpretation challenging. We are, however, able to include a neighbourhood level variable, in this case IMD quintiles, to define the intersections – we do this in some but not all models, as explained below. Our purpose is to start by separating out neighbourhood effects to assess their relative contribution, and then including IMD to see how it intersects with conventional social characteristics. As pointed out by Evans (2019), a model that only cross-classifies by neighbourhood would be unable to analyse how the same intersectional position has different outcomes across different types of neighbourhoods. A key contribution here is to explicitly model neighbourhood context in the form of deprivation and consider how intersectional outcomes vary by different levels of deprivation rather than examining how each intersection could operate differently in each and every neighbourhood (LSOA).

The analysis involved the following modelling steps:

- Model 1 is a two-level null model which nests individuals in neighbourhoods to test the extent to which biomarkers vary by neighbourhoods (via the neighbourhood ICC).
- Model 2 is the same as model 1 but includes IMD quintiles as main (or fixed) effects to test the extent to which IMD explain neighbourhood variation (via reduction in the neighbourhood variance/ICC).
- Model 3 specifies a three-level null model with intersections and neighbourhoods as cross-classified higher levels, entered as random effects. As such, this model builds on model 1 by adding a cross-classified intersection level. In this model, intersections are defined without using IMD quintiles. This model tests the extent to which the outcomes vary, net of neighbourhood variation, by intersections defined by gender, ethnicity, education, age only (via the intersectional ICC).
- Model 4 is the same as model 3 but includes intersection-level main effects (gender, ethnicity, education, age), to see the extent to which model 3's intersectional effects are additive (and so explained by those main effects), or multiplicative.
- Model 5 is a cross-classified null model, similar to model 3, which additionally uses IMD to define the intersections, and tests the total intersectional variation via the intersection ICC.
- Finally model 6 is the fully specified model. It is the same as model 5 but includes main effects (gender, ethnicity, education, age, IMD). It is the best fitting specification from which we generate the graphs. The intersectional ICC tests for multiplicative effects, and is a useful comparison to model 4, to see the role of IMD in the intersectional effects.

Table 2 describes what each variance parameter/level means in each model. In the supplementary material we include a research note on the modelling strategy, specifically in terms of the approach to modelling main effects. As a robustness check, we ran model 6 without the neighbourhood level included (i.e., as a conventional two-level model MAIHDA model) and found both the model parameters and the resulting graphs to be almost identical (see supplementary material). It is important to note however that it is not necessarily the case that the empirical results from these models will be identical, for example if particular intersectional groups are clustered in neighbourhoods with particular characteristics that are related to the outcome. For instance, if fast-food outlet density was a significant risk factor for high levels of HbA1c, and black women were particularly clustered in neighbourhoods with high fast-food outlet density, we would expect the standard MAIHDA model to include that as intersectional differences, whilst our cross-classified model would include it as a neighbourhood effect. This is not to say that such an effect is not intersectional (indeed, how individuals cluster in particular areas is inherently intersectional), but the interpretation of the intersection-level variance in the two models is different and the comparison between the two substantively meaningful where the results differ. Neighbourhood variance is often of substantive interest in itself, and failure to account for neighbourhood differences could lead to incorrect inference, particularly if the interest is

Table 2
Description of multilevel intersectional model parameters.

Model	1	2	3	4	5	6
Description	Two level (individuals nested in neighbourhoods) null model with no predictors in fixed part of the model.	As model 1, but with IMD quintiles as fixed effects.	Three level cross-classified (individuals nested in neighbourhoods and strata). Strata defined without IMD. Null model with no predictors.	As model 3 but with additional main effects that define strata (not including IMD).	As model 3 but intersectional strata are additionally defined by IMD quintiles.	As model 5 but with additional main effects that define strata (including IMD quintiles).
Intersection level	–	–	The proportion of variance that occurs between intersectional strata. This is a mix of additive and multiplicative effects of the variables that define the intersections. Neighbourhood differences are controlled in this model.	The proportion of variance that occurs between intersectional strata once main effects have been controlled for. This model therefore has multiplicative effects only of the variables that define the intersections. The comparison with model 3 will reveal the extent of the additive vs multiplicative intersectionality. Neighbourhood differences are controlled in this model.	As model 3, but now the variance includes variability between IMD quintiles, such that the strata are divided further. Compared to model 3, it reveals how including IMD in the definition of intersections affects the extent of (combined multiplicative and additive) intersectionality.	Compared to model 5, this variance reveals the extent of multiplicative (compared to additive) intersectionality (this is a similar comparison to model 3 vs 4). Compared to model 4, it will reveal the extent to which the inclusion of IMD in the definition of intersections affects the extent of multiplicative intersectionality.
Neighbourhood level	Proportion of variance in Y at neighbourhood level – this could include selection effects caused by intersectional strata clustering in particular neighbourhoods, as well as other selection effects and neighbourhood effects.	Proportion of variance in Y at neighbourhood level once IMD has been controlled for – this could include selection effects caused by intersectional strata clustering in particular neighbourhoods, and other unmeasured neighbourhood variables unrelated to IMD. Compared to model 1, it show how much neighbourhood variance IMD explains.	Proportion of variance in Y at neighbourhood level once IMD has been controlled for – this will not include selection effects caused by intersectional strata clustering in particular neighbourhoods (but could be produced by other selection effects). Compared to model 1, this will reveal the extent to which intersectional clustering explains neighbourhood differences.	As model 3	Compared to model 3, this variance now excludes IMD, since this will be included in the intersectional level above. It will reveal the extent of neighbourhood variance that is net of IMD and other intersectional differences (including other neighbourhood and selection effects).	As model 5
Individual level	Proportion of variance occurring within neighbourhoods – that is, how different people are in the same neighbourhood.	As model 1	Proportion of variance occurring within neighbourhood-strata units – that is, how different people are within a given neighbourhood-strata combination.	As model 3	As model 3	As model 3

neighbourhood-level variables.

All models used 5000 burn-in iterations and 50,000 iterations as sufficient stability was achieved with this number according to the Effective Sample Size (greater than 400 for all parameters) and a visual inspection of parameter chains. MCMC estimation was used with non-informative priors generated using IGLS models (Bell et al., 2019). Models were run using MLwiN v3.05 (Charlton et al., 2020), called from Stata with the runmlwin package (Leckie and Charlton, 2013).

To present the results graphically we plot residual effects from model 6 combining the additive and multiplicative estimates to generate estimates for each intersection together with measures of uncertainty. We display outcomes for 60–69 year olds, as biological weathering has likely had the greatest effect for those in older age groups, and they are also more likely to have lived in their neighbourhoods for longer. However, we compare graphs to those for younger age groups (supplementary material) to show how potential weathering effects might operate, though note that because we use cross-sectional data we cannot disentangle age and generation effects (Bell, 2020).

3. Results

Table 3 shows that the analytical sample is weighted towards women, older age groups, White British people and those in higher SEPs, reflecting the non-representativeness of the UK Biobank (Fry et al., 2017). Mean values for the biomarker outcomes are also given in Table 3. Generated intersections based on the characteristics in Table 3 ranged in size from 1 to 14,900, with a mean of 6000 (SD 3454).

Fixed effects estimates from the fully specified models (model 6) are given in Table 4. Biomarkers were higher in older age groups, with the strongest association seen for SBP and the weakest for CRP. Men had higher biomarker levels, except for CRP which was higher in women. Ethnic inequalities were observed and their nature varied by biomarker.

Table 3
Sample characteristics.

Intersectional characteristics – % (n)	% (n)
Full Sample	(n = 386,475)
Age 40-49	22.59 (87,322)
Age 50-59	33.11 (127,968)
Age 60-69	44.29 (171,185)
Women	54.23 (209,578)
White British	96.07 (371,295)
Chinese	0.33 (1282)
Indian	1.37 (5285)
Pakistani	0.39 (1498)
Black Caribbean	1.06 (4097)
Black African	0.78 (3018)
Low education	17.42 (67,336)
Medium education	51.61 (199,477)
High education	30.96 (119,662)
V. low neighbourhood deprivation	29.97 (115,831)
Low neighbourhood deprivation	23.66 (91,422)
Medium neighbourhood deprivation	18.10 (69,933)
High neighbourhood deprivation	16.01 (61,861)
V. high neighbourhood deprivation	12.27 (47,428)
Biomarkers	**Mean (SD)**
HbA1c (mmol/mol)	36.09 (6.57)
Missing – % (n)	*6.21 (23,997)*
CRP (mg/L)	1.89 (1.83)
Missing – % (n)	*9.78 (37,782)*
SBP (mm Hg)	139.90 (19.65)
Missing – % (n)	*2.89 (11,181)*
Waist circumference (cm)	90.39 (13.47)
Missing – % (n)	*0.32 (1244)*
Grip strength (kg)	30.56 (11.01)
Missing – % (n)	*0.77 (2958)*

For example, while each ethnic group had higher Hb1Ac compared to White British groups, patterns for the other biomarkers were more complex. Higher education and lower neighbourhood deprivation were associated with worse health for nearly all biomarkers except for SBP, where those with vey high deprivation had low average SBP, and for waist circumference where those with medium education had the smallest average value.

Results for the MAIHDA models are given in Table 5. Overall, as indicated by ICC values from model 1, 1.41–2.99% of the variation in biomarkers was observed at neighbourhood level. Adding neighbourhood deprivation to model 1 (see model 2) explained varying amounts of neighbourhood variation, from half of the variation in HbA1c, to no variation in SBP. When accounting for intersectional compositional differences (see models 3 and 4), the amount of neighbourhood variation explained by these differences varied by biomarker – around half for HbA1c and CRP, to a fifth for waist circumference. For grip strength, neighbourhood variance was larger in model 4. For HbA1c, CRP and waist, model 6 suggested that intersectional compositional differences and IMD both had independent effects in explaining neighbourhood variation as this model saw the greatest decreases in neighbourhood variance. However, for SBP, including IMD in the model made little difference, consistent with the fixed effects results in Table 4, and for grip strength, the suppressor effect of the intersectional compositional variables was also evident in the fully specified model.

The intersectional ICC values from model 5 suggest that a significant proportion of the variance in biomarker outcomes was at the intersectional-IMD level – highest for grip at 59.7% and lowest for CRP at 8.4%. Including the main effects in model 6 suggests that remaining multiplicative effects present as indicated by the ICC values varied by outcome, from 0.68% for CRP to 3.44% for HbA1c. Comparing the multiplicative effects remaining in model 6 with model 4 as indicated by the intersectional ICC values suggests that multiplicativity is not driven in particular by IMD, with similar levels of multiplicative effects present whether or not IMD was used to define the intersections.

The standard MAIDHA comparisons here (between models 3 and 4 and between models 5 and 6) show that intersectional variance is largely additive but with a small multiplicative variance remaining in models 4/6 for all outcomes.

We now plot the intersectional predicted effects of each outcome by combining the main effect and multiplicative effects residuals of the fully specified model 6 to visualise the intersectional inequalities. The graphs show gender x ethnicity groupings (the main x axis), with individual (qualifications) and neighbourhood (IMD) advantage/disadvantage being visualised within these groupings.

3.1. HbA1c

We observed social gradients in HbA1c by deprivation and education, but the nature of these inequalities varied by gender and ethnicity (Fig. 1). White British ethnic groups had the narrowest deprivation and education inequalities and the lowest levels of Hba1c. None of the White British or Chinese intersections on average reached the cut-off for prediabetes. For the Indian, Caribbean and African intersections, whether

Table 4
Fixed effect estimates.

	HbA1c (mmol/mol)	CRP (mg/L)	SBP (mm Hg)	Waist (cm)	Grip strength (kg)
Age 50-59	Ref	Ref	Ref	Ref	Ref
Age 60-69	2.98 (2.63–3.32)	.070 (.052–.087)	7.92 (7.28–8.56)	2.33 (1.75–2.94)	−2.64 (−2.93–−2.34)
Age 70-79	4.32 (3.94–4.71)	.102 (.082–.121)	14.86 (14.45–15.55)	3.65 (3.02–4.30)	−5.57 (−5.89–−5.25)
Women	Ref	Ref	Ref	Ref	Ref
Men	.980 (.687–1.27)	−.089 (−.104–−.075)	4.47 (3.89–5.01)	8.38 (7.85–8.91)	15.44 (15.18–15.71)
White British	Ref	Ref	Ref	Ref	Ref
Chinese	2.13 (1.59–2.66)	−.253 (−.277–−.228)	−2.07 (−3.28–−.854)	−8.38 (−9.33–−7.45)	−3.10 (−3.61–−2.60)
Indian	4.46 (4.05–4.87)	.099 (.077–.121)	−.314 (−1.08–.464)	−.216 (−.932–.486)	−5.55 (−5.91–−5.20)
Pakistani	6.37 (5.85–6.90)	.179 (.141–.217)	−1.47 (−2.62–.312	2.92 (2.00–3.84)	−5.07 (−5.56–−4.58)
Caribbean	4.20 (3.76–4.66)	−.027 (−.049–−.004)	2.29 (1.43–3.15)	1.02 (.239–−1.78)	2.21 (1.81–2.59)
African	3.76 (3.25–4.27)	.036 (.009–.064)	5.05 (4.08–6.01)	3.33 (2.47–4.17	−.874 (−1.31–−.445)
Low education	Ref	Ref	Ref	Ref	Ref
Med. education	−.990 (−1.36–−.629)	−.068 (−.084–−.051)	−1.28 (−1.98–−.575)	−1.64 (−2.29–−.951)	1.42 (1.10–1.77)
High education	−1.49 (−1.87–−1.10)	−.132 (−.148–−.115)	−3.05 (−3.77–−2.33)	1.63 (.774–2.47)	1.77 (1.43–2.12)
V. low neigh. dep.	Ref	Ref	Ref	Ref	Ref
Low neigh.	.035 (−.459–.518)	.018 (−.006–.042)	.408 (-.554–1.39)	.933 (.098–1.78)	−.218 (−.640–.206)
Med. neigh. dep.	.549 (.073–1.02)	.042 (.018–.066)	.482 (-.396–1.38)	1.63 (.774–2.47)	−.475 (−.908–−.047)
High neigh. dep.	1.06 (.593–1.55)	.066 (.041–.091)	.902 (.016–1.79)	2.44 (1.61–3.27)	−1.09 (−1.51–−.670)
V. high neigh. dep.	1.47 (.994–1.95)	.122 (.096–.148)	.222 (-.667–1.13)	3.16 (2.34–4.00)	−1.91 (−2.33–−1.49)
n	362495	348710	375312	385250	383536

Estimates are from fully specified model 6.
CRP transformed back by exponentiating estimates then subtracting 1.

Table 5
Multilevel intersectional model estimates.

	Neighbourhood variance	Intersectional variance	Individual variance	ICC neighbourhood	ICC intersection	DIC
Hba1c, model 1	.738 (.674–.803)	–	42.43 (42.23–42.63)	1.71%	–	2391100
Hba1c, model 2	.395 (.345–.448)	–	42.44 (42.25–42.64)	0.92%	–	2389876
Hba1c, model 3	.326 (.277–.377)	10.27 (7.74–13.57)	40.17 (39.98–40.36)	0.64%	20.23%	2369779
Hba1c, model 4	.324 (.273–.372)	.838 (.540–1.24)	40.17 (39.98–40.36)	0.78%	2.03%	2369763
Hba1c, model 5	.146 (.109–.185)	12.21 (10.51–14.17)	40.10 (39.91–40.29)	0.28%	23.28%	2368397
Hba1c, model 6	.150 (.107–.195)	1.44 (1.06–1.89)	40.11 (39.93–40.30)	0.36%	3.44%	2368384
CRP, model 1	.006 (.005–.007)	–	.268 (.267–.269)	2.32%	–	534741
CRP, model 2	.004 (.003–.004)	–	.268 (.266–.269)	1.35%	–	533267
CRP, model 3	.004 (.003–.004)	.032 (.023–.043)	.262 (.261–.263)	1.18%	10.69%	525644
CRP, model 4	.003 (.003–.004)	.003 (.002–.005)	.262 (.261–.263)	1.30%	1.19%	525619
CRP, model 5	.002 (.002–.002)	.024 (.020–.029)	.262 (.260–.263)	0.68%	8.40%	524370
CRP, model 6	.002 (.002–.002)	.002 (.001–.003)	.262 (.260–.263)	0.73%	0.68%	524174
SBP, model 1	9.25 (8.68–9.83)	–	377.07 (375.34–378.81)	2.39%	–	3296405
SBP, model 2	9.22 (8.66–9.79)	–	377.07 (375.34–378.79)	2.39%	–	3296400
SBP, model 3	6.48 (6.02–6.94)	54.21 (40.59–72.05)	333.85 (332.33–335.39)	1.64%	13.74%	3250197
SBP, model 4	6.48 (6.04–6.94)	4.15 (2.61–6.26)	333.86 (332.33–335.39)	1.88%	1.20%	3250177
SBP, model 5	6.40 (5.95–6.86)	52.53 (44.92–61.14)	333.78 (332.26–335.31)	1.63%	13.38%	3250379
SBP, model 6	6.42 (5.96–6.87)	3.30 (2.41–4.42)	333.81 (332.28–335.36)	1.87%	0.96%	3250210
Waist, model 1	5.43 (5.12–5.74)	–	176.28 (175.48–177.08)	2.99%	–	3091338
Waist, model 2	3.22 (2.98–3.46)	–	176.21 (175.42–177.01)	1.79%	–	3089782
Waist, model 3	4.06 (3.82–4.31)	39.94 (30.11–52.73)	135.84 (135.22–136.46)	2.26%	22.21%	2990961
Waist, model 4	4.06 (3.82–4.30)	5.64 (3.97–7.89)	135.84 (135.23–136.46)	2.79%	3.87%	2990951
Waist, model 5	2.69 (2.50–2.89)	38.75 (33.51–44.72)	135.59 (134.97–136.21)	1.52%	21.89%	2989466
Waist, model 6	2.69 (2.49–2.89)	4.35 (3.48–5.35)	135.59 (134.97–136.21)	1.88%	3.05%	2989290
Grip, model 1	1.71 (1.57–1.85)	–	119.61 (119.07–120.16)	1.41%	–	2926849
Grip, model 2	1.50 (1.36–1.64)	–	119.61 (119.07–120.15)	1.24%	–	2926531
Grip, model 3	2.33 (2.22–2.44)	72.04 (54.95–94.29)	48.30 (48.09–48.53)	1.90%	58.72%	2582500
Grip, model 4	2.33 (2.22–2.44)	1.29 (.860–1.88)	48.31 (48.09–48.53)	4.49%	2.49%	2582487
Grip, model 5	1.99 (1.89–2.09)	74.37 (65.41–84.52)	48.21 (47.99–48.43)	1.60%	59.70%	2581708
Grip, model 6	1.99 (1.89–2.09)	.897 (.657–1.19)	48.23 (48.01–48.45)	3.89%	1.76%	2581552

Analysis of CRP on transformed variable.
Model 1 = Two-level null neighbourhood model.
Model 2 = Model 1 + IMD quintiles as main effects.
Model 3 = Cross-classified null model, IMD not used to define intersections.
Model 4 = Cross-classified main effects model (gender, ethnicity, education, age), IMD not used to define intersections.
Model 5 = Cross-classified null model, IMD used to define intersections.
Model 6 = Cross-classified main effects model (gender, ethnicity, education, age, IMD), IMD used to define intersections.

they on average reached the cut-off for prediabetes depended much on SEP, both individual (educational qualifications) and area-level (IMD). For Pakistani intersections, nearly all intersections were on average prediabetic, regardless of SEP. Some male Pakistani intersections nearly reach the cut-off for diabetes on average. For the other ethnic groups the patterns with respect to education and deprivation were somewhat inconsistent: in most cases, higher deprivation was associated with higher HbA1c, but in some cases the pattern was unclear. The deprivation gradient was steepest for Pakistani women, especially those with low education. For Pakistani men, those with low education had elevated levels. Pakistani men with high education and high levels of deprivation had amongst the highest HbA1c levels in the whole sample.

Intersectional patterning was mostly consistent by age groups (supplementary material). This suggests that ethnic and socioeconomic inequalities in HbA1c are produced in an individual's formative years, and the pattern of inequality continues into later years, consistent with the persistent inequality hypothesis.

3.2. C-reactive protein

Patterns by gender were consistent across deprivation, education and ethnicity, with women having slightly higher levels of C-reactive protein than their male counterparts on average (Fig. 2). No intersections on average reached the cut-off for systemic inflammation, though female Pakistani intersections with the lowest SEP were close. The patterning by education and deprivation were mostly consistent across gender-ethnic subgroups. There was some variation however, with CRP levels for Pakistani women more spread out by IMD quintiles than White British or Chinese groups. Overall, the Pakistani group had the highest levels of CRP followed by the Indian and African groups. The White British group had slightly elevated levels compared with the Caribbean group, and the Chinese group had the lowest levels by some margin.

There were few differences in intersectional patterning in CRP across age groups (supplementary material), except for 60–69 year olds the values were more tightly clustered together. This was mostly consistent with the persistent inequality hypothesis but there was some suggestion of age-as-leveller effect for the oldest age group.

3.2.1. Systolic blood pressure

Overall, men had higher SBP than women. Chinese and Pakistani women with the highest levels of education had the lowest blood pressure whilst African and especially Caribbean men with low and medium levels of education had the highest levels (Fig. 3). Nearly all intersections on average met, and some significantly exceeded, the cut-off for hypertension. Those with higher SEPs had lower blood pressure, though the pattern was not clear across all gender-ethnic subgroups, being most pronounced for White British women.

Comparing intersectional patterns across age groups (supplementary material) suggests that there is some degree of flattening out of intersectional inequalities with age. At age 40–49, men had higher blood pressure than men, and Caribbean and especially African groups had the highest blood pressure. By ages 60–69, these differences had reduced, especially with respect to gender. This is consistent with the age-as-leveller hypothesis.

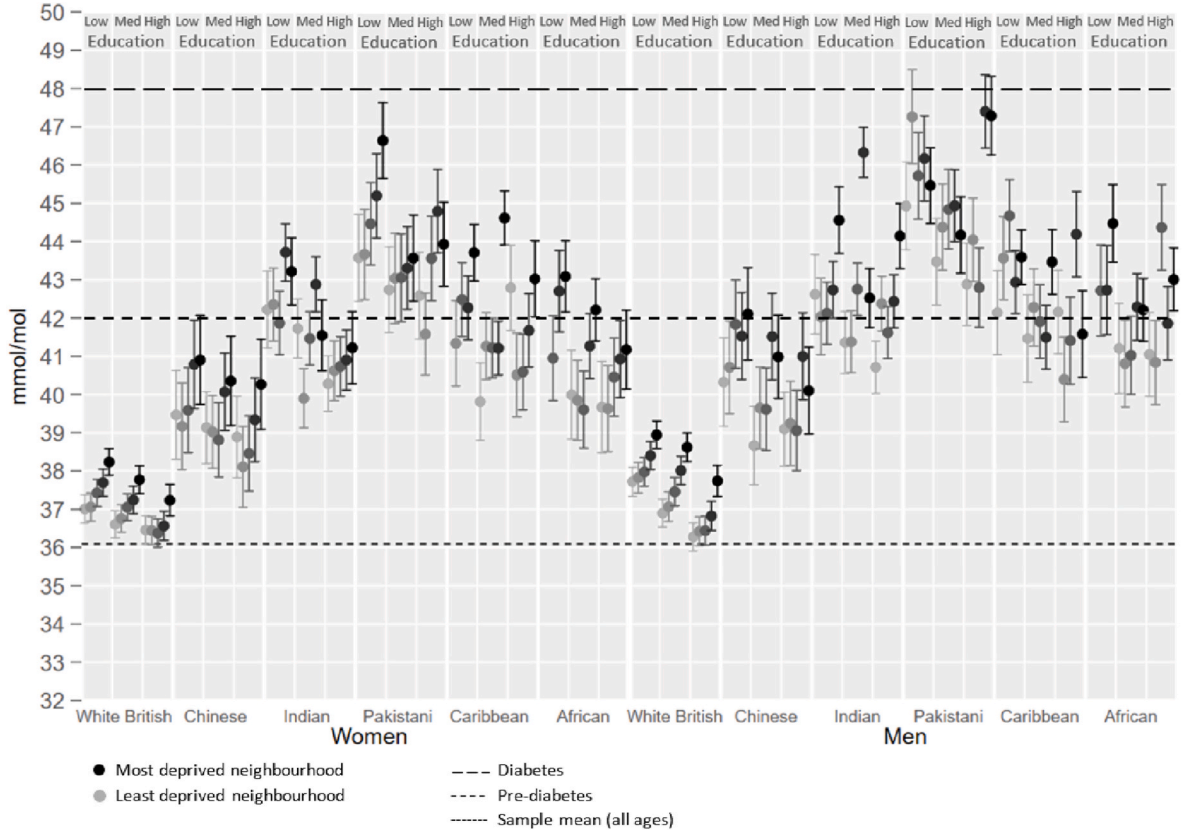

Fig. 1. Intersectional inequalities in HbA1c, age 60-69.

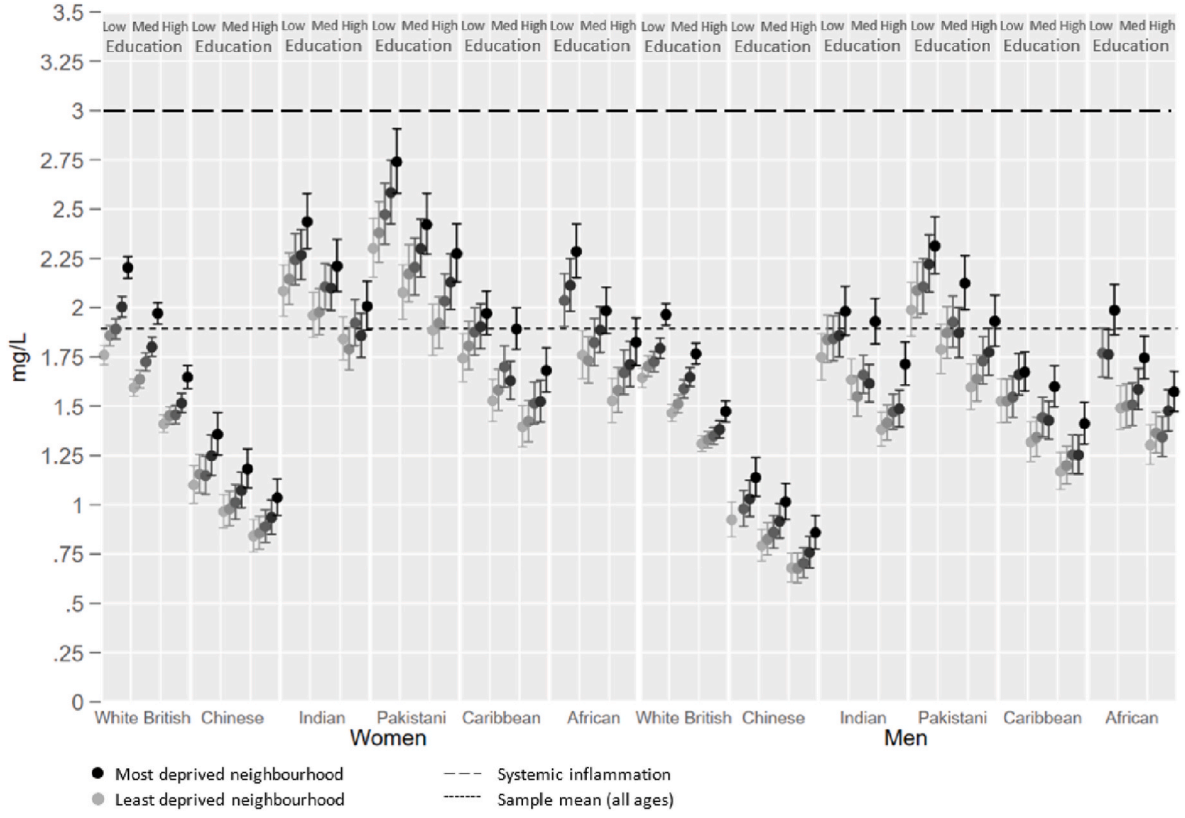

Fig. 2. Intersectional inequalities in C-reactive protein, age 60-69.

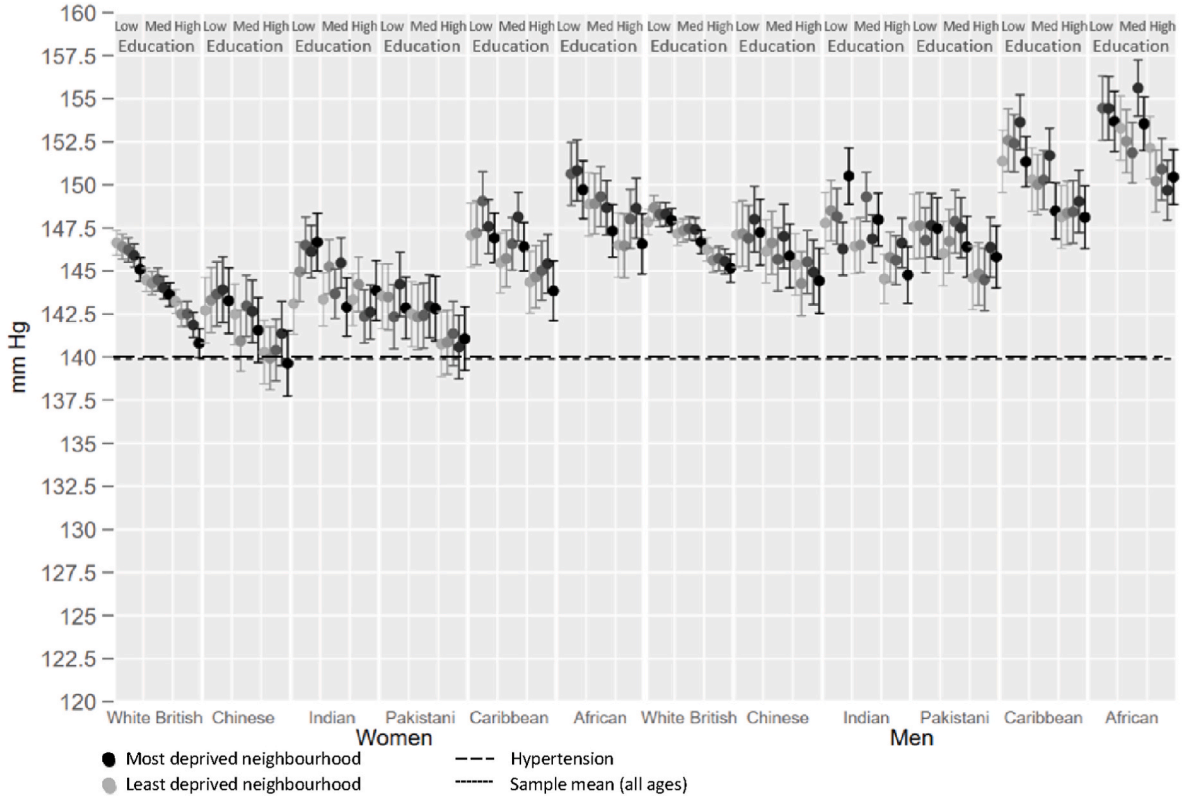

Fig. 3. Intersectional inequalities in systolic blood pressure, age 60-69.

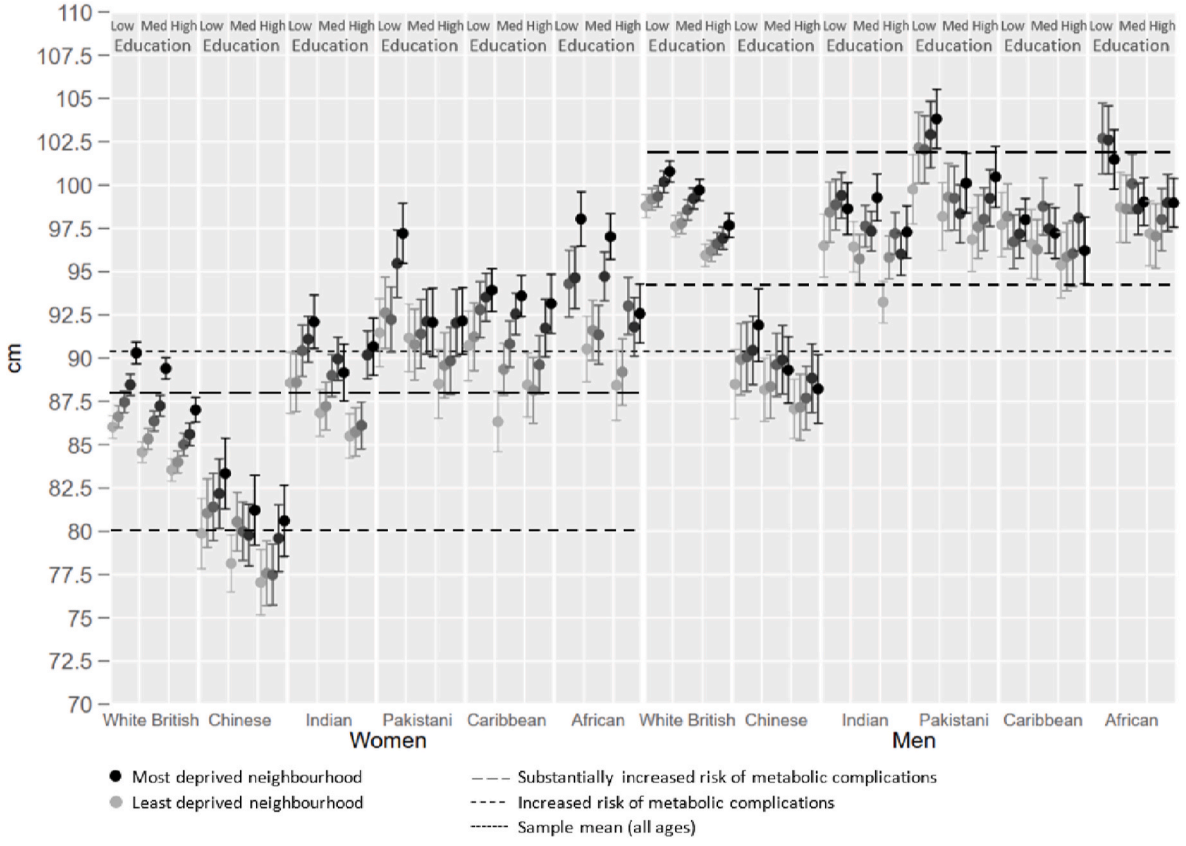

Fig. 4. Intersectional inequalities in waist circumference, age 60-69.

3.3. Waist circumference

Men had overall larger waist circumferences (Fig. 4). All female intersections on average reached the cut off for increased risk of metabolic complications, except for the female Chinese intersections where deprivation and education were the deciding factors. Most female Pakistani, Caribbean and African intersections on average reached the cut-off for substantially increased risk of metabolic complications, the exception being those with high education and low deprivation. For White British women, only the intersections with low or medium education and high or very high deprivation on average reached the cut-off. All male intersections on average reached the cut-off for increased risk of metabolic complications, except Chinese men. Pakistani and African with low education on average reached the cut-off for substantial risk. The overall association between deprivation and waist circumference was stronger for women than men.

There was little difference in intersectional patterning across age groups (supplementary material), which suggests that intersectional inequalities in waist circumference are established in earlier years and mostly persist into later years. This is consistent with the persistent inequality hypothesis.

3.4. Grip strength

Gender differences in grip strength were consistent across all intersections (Fig. 5). However, for White British women, deprivation had almost no effect, in contrast to its effect across other gender-ethnic subgroups, which was small but mostly followed a clear social gradient. Neighbourhood deprivation differences for Caribbean and African women were also smaller than for Caribbean and African men. Caribbean men, especially those in less deprived neighbourhoods, had the highest levels of grip strength. Indian and Pakistani women, especially those in the most deprived neighbourhoods, had the lowest grip strength, reaching the cut-off for weak grip, alongside Chinese intersections. For men, only Indian and Pakistani groups on average reached the cut-off for weak grip strength, except Pakistani men with the highest individual and neighbourhood SEP. Chinese men with low education and in deprived neighbourhoods also had weak grip on average.

Comparing intersectional effects across age groups (supplementary material), lower ages were associated with lower grip strength, consistent with a gradual age decline. The intersectional patterning was mostly consistent across ages. However, there was a greater gender-ethnic subgroup spread in younger age groups, especially for the White British group. This is consistent with the age-as-leveller hypothesis where the intersectional effects are washed out by age effects over time.

4. Discussion

In this study we have made several novel additions to the literature. First, we analysed a large dataset – the UK Biobank – allowing us to calculate estimates for granular subgroups defined by both spatial and intersectional factors. Second we used relatively specific ethnic categories, in contrast to existing studies which often collapse ethnicity into broad groups (e.g., White vs non-White) and thereby combine diverse populations and obscure heterogeneity in social determinants and health outcomes. Third we analysed novel biomarker data which enabled us to unpack the various potential pathways through which spatial and intersectional aspects of social context 'get under the skin'. Finally, we extended an intersectional multi-level modelling framework to incorporate neighbourhood deprivation types (operationalised via IMD quintiles) in a cross-classified model to evaluate the relative contribution of spatial and intersectional factors to the patterning of biomarkers of healthy ageing.

The results show complex inequalities in biomarkers of chronic disease and healthy ageing. We found the relative importance of neighbourhood deprivation, and the way it intersects with gender, ethnicity and education, depends on the biomarker in question. For systolic blood pressure and grip strength neighbourhood deprivation revealed no

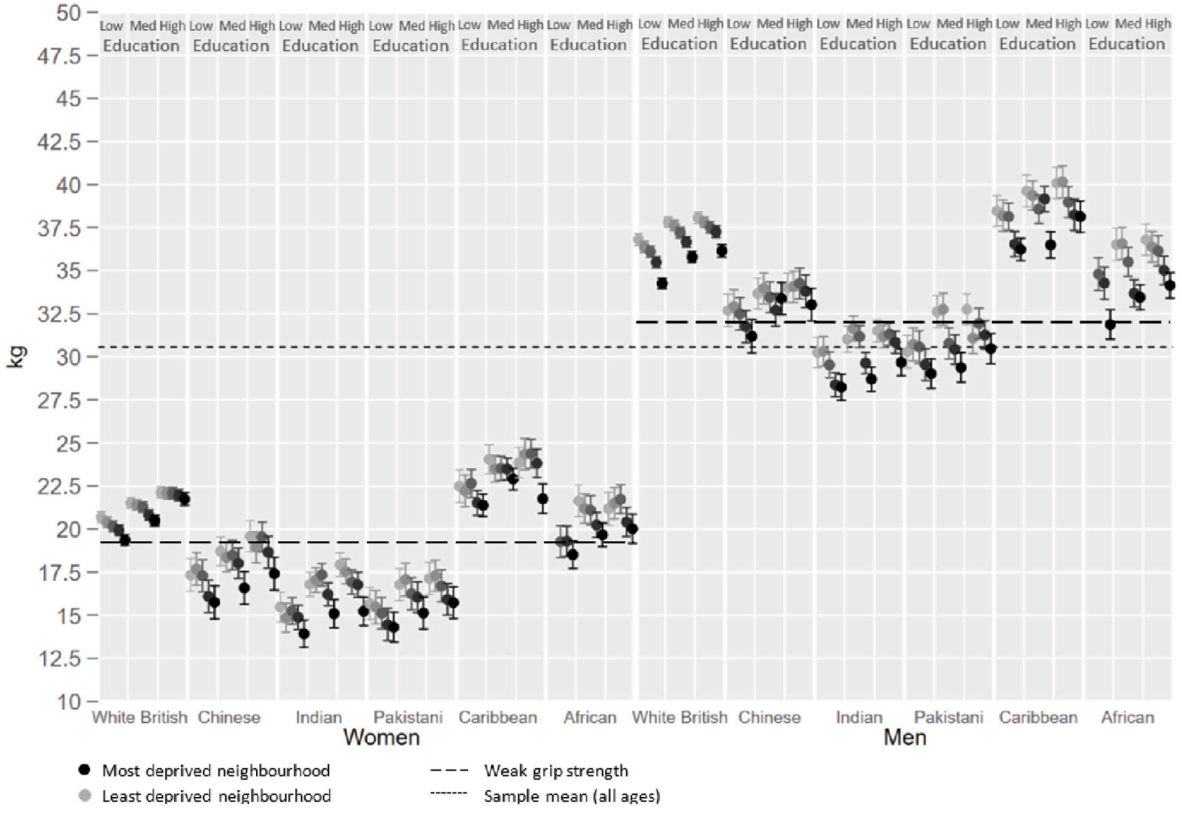

Fig. 5. Intersectional inequalities in grip strength, age 60-69.

additional intersectional heterogeneity when gender, ethnicity, education and age were already included in the model. Conversely, including deprivation as an intersectional factor revealed pronounced inequalities in HbA1c for all ethnic groups except White British. Intersectional inequalities were mainly driven by additive effects, with multiplicative effects greatest, though still small, for HbA1c and waist circumference. Despite the differences we observed, overall conventional intersectional factors – age, gender, ethnicity and education – explained more variance in the outcomes than neighbourhood deprivation. Overall, our findings caution against simplistic narratives around the effects of gender, ethnicity, education and neighbourhood deprivation alone which non-intersectional approaches might promote.

Our findings pose a direct challenge to the narrative that ethnic inequalities in health are simply explained by differences in socioeconomic factors. By comparing educational and neighbourhood deprivation patterning within each ethnic group we show that for most outcomes there is an additional health penalty of being both in a lower SEP and from a minority ethnic background. Put another way, those from a minority ethnic background in high SEP intersections often had biomarker-indicated levels of health around the same as medium SEP White British intersections, and the lowest SEP White British intersections often had around the same health as minority ethnic groups with medium SEP. There are three caveats to note here. First, intersectional inequalities vary by biomarker. For some biomarkers such as SBP, ethnic differences are overall small. Second, specific minority ethnic groups have quite divergent health outcomes, and in fact, in some cases, some groups have a health advantage for some biomarkers, such as Chinese groups having the best health according to CRP and waist circumference. Third, biomarker-indicated health may have different healthy ageing implications for different minority ethnic groups. For example, there is ongoing debate regarding whether HbA1c cut-offs should vary by ethnicity (Sacks, 2016; Selvin, 2016). Nonetheless, our findings make clear that any attempts to 'explain away' minority ethnic differences in health by reference to socioeconomic factors simply do not account for the way in which health is patterned by combinations of ethnicity and SEP. Neither as noted do they account for processes of racialisation and racial discrimination, including in how different ethnic groups occupy different SEPs.

Similarly, gender differences in waist circumference and grip strength are thought to be the result of physiological sex differences between men and women (Dodds et al., 2014; World Health Organization, 2011b), notwithstanding the ways in which social factors become biologically embedded over historical time and reflect complex culture-biology interplays. Gender differences in intersectional patterning of grip strength were minimal, though men exhibited slightly more variation in terms of neighbourhood deprivation, whereas deprivation differences in waist circumference were greater for women than men. We also found that neighbourhood deprivation differences in SBP were larger for White British women than their male counterparts. Existing research examining the role of gender in the relationship between neighbourhood deprivation and biomarkers has tended to use a measure of allostatic load, obscuring the biomarker-specific differences. Bird et al. (2010) found in a US sample that the relationship between neighbourhood deprivation and allostatic load was consistent for men and women. Chaparro et al. (2018) did analyse biomarkers separately, but in relation to neighbourhood physical environment factors in particular, finding that associations were mostly consistent by gender.

The health profiles of neighbourhoods may vary both due to the people who live in them (compositional effects), as well as the characteristics of neighbourhoods themselves (contextual effects). Our findings point to the interplay of compositional and contextual factors given that the effect of individual measures vary in different contexts of neighbourhood deprivation. Further, in contrast to studies which attempt to measure compositional effects by considering single categories of difference at a time e.g. ethnicity or SEP, we account for intersectional composition by including well-known socio-demographic determinants of health *as well as their interactions*. We found that total neighbourhood variation was reduced most for HbA1c and CRP in the full models; including intersectional composition accounted for around half the neighbourhood variance in these outcomes. Further reductions were seen when also including neighbourhood deprivation (measured as IMD). Remaining neighbourhood variation is likely driven by both contextual factors (both physical and social) and unmodelled compositional factors, as well as their interplay. For example, IMD does not capture social networks and participation, yet in a qualitative analysis Grewal et al. (2004) found that these play significant roles in the quality of life for older minority ethnic people. Another important strand of work here is on the health effects of neighbourhood ethnic density which might play an important role in intersectional neighbourhood effects (Bécares et al., 2012).

Given that we analysed a range of biomarkers associated with different physiological systems, our findings also suggest that intersecting inequalities, including those relating to place, get 'under the skin' in different ways. As HbA1c is strongly related to diet and physical activity, the large neighbourhood deprivation differences we observed for minority ethnic groups suggest these groups are sensitive to the neighbourhood food and physical activity environment. CRP provides a strong contrast to this patterning, with neighbourhood deprivation affecting all intersections more or less equally. This finding suggests a universal dose-response relationship between neighbourhood deprivation and inflammation that is not mediated by individual characteristics and behaviours. Given that low grade systemic CRP elevation is a measure of accumulated social stress and adversity (Chiang et al., 2019), this suggests that neighbourhood deprivation affects all residents via the psychosocial pathway, regardless of their social characteristics. Although systolic blood pressure showed a similar level of neighbourhood variation to the other biomarkers, this was not explained by neighbourhood deprivation, suggesting that other neighbourhood-level factors explain this variation. Results for waist circumference to some extent mirror those for HbA1c, with larger neighbourhood deprivation differences for non-White British groups (especially for women). This suggests that the deprivation > health behaviour pathway might be particularly important for minority ethnic groups, though given the exploratory nature of our analysis this should be tested in further research. Lastly, grip strength differences in terms of neighbourhood deprivation are small and mostly consistent between gender x ethnicity groupings, suggesting that the effects of neighbourhood deprivation on frailty are not modified by individual social characteristics but have a universal effect. Overall, the health behaviour pathway appears to be central to intersectional neighbourhood deprivation differences in biomarkers of ageing, though our analysis is only suggestive and further is unable to tease out the interrelationship between psychosocial stress and health behaviours.

With respect to age differences, our findings are mostly consistent with the persistent inequality hypothesis, with some biomarkers showing evidence of an age-as-leveller effect. This finding suggests that while biological weathering may be a life-long process, inequalities in healthy ageing, including those driven by neighbourhood deprivation, appear to be established early in the lifespan and remain similar over time. However, it is important to note people were only measured from age 40 in the sample and, further, the data are not longitudinal, so we are unable to disentangle age from cohort effects. For example, those in minority ethnic groups in different age groups will have different migratory histories which could have significant effects on health. This highlights the need for further research to understand at which point in the life course such inequalities are established to aid policy and intervention efforts. Research is also needed, both quantitative and qualitative, to understand the mechanisms and processes behind the intersectional patterning we observed, and particularly why some intersectional groups are more, or less, vulnerable to neighbourhood deprivation.

5. Limitations

Our data were cross-sectional, and while analysing intersectional inequalities by age is informative, we are unable to determine whether the differences were due to age or cohort (generational) effects. We were also unable to measure neighbourhood deprivation over time, yet a recent review suggested that accumulated exposure over the life course damages later life health (Jivraj et al., 2020). We deliberately do not control for confounders, mediators or moderators because this approach is best handled by other methods e.g. SEM approaches. In our analysis, we seek to describe the overall social patterning of biomarkers (such as HbA1c) rather than explain causal pathways (for instance, the diagnosis, medication use and management of diabetes). Future research could investigate these mechanisms from an intersectional perspective.

As noted, given that the UK Biobank is unrepresentative the results require replication in other samples. MAIHDA models only partly correct for multiple testing (Bell et al., 2019). Further work in this area could include migratory histories given their importance for intersectional positioning. As LSOAs were not identifiable we were unable to map the results geographically which might help to both visualise the results and be useful from a policy perspective. Further work might consider further levels of analysis such as Travel to Work Areas or Super Output areas because these are likely associated with wider-level determinants of health. Finally our analysis is a first step in attempting to capture the complexity of how neighbourhood deprivation intersects with more traditional intersectional axes; this is a complex endeavour, both theoretically and methodologically, which we hope others will build on.

6. Conclusion

Our novel approach to modelling neighbourhood deprivation as an intersectional factor has allowed us to compare its contribution to healthy ageing biomarkers relative to conventional intersectional characteristics. It has further revealed granular patterning in these outcomes, including in the degree to which neighbourhood deprivation matters for different intersectional subgroups. Results varied by biomarker, suggesting various social-biological pathways by which social causes of disease get under the skin. Future research should exploit longitudinal data to further understand how the timing of neighbourhood deprivation exposure matters for intersectional inequalities in healthy ageing, and qualitative work is needed to further understand mechanisms including differential experiences and exposures.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.healthplace.2022.102871.

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