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Ex ante inequality of opportunity in health, decomposition and distributional analysis of biomarkers

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Abstract

We use a set of biomarkers to measure inequality of opportunity (IOp) in the risk of major chronic conditions in the UK. Applying a direct *ex ante* IOp approach, we find that inequalities in biomarkers attributed to circumstances account for a non-trivial part of the total variation. For example, observed circumstances account for 20% of the total inequalities in our composite measure of multi-system health risk, allostatic load. We propose an extension to the decomposition of *ex ante* IOp to complement the mean-based approach, analysing the contribution of circumstances across the quantiles of the biomarker distributions. Shapley decompositions show that, for most of the biomarkers, the percentage contribution of socioeconomic circumstances (education and childhood socioeconomic status), relative to differences attributable to age and gender, increase towards the right tail of the biomarker distribution, where health risks are more pronounced.

Keywords: biomarkers; equality of opportunity; non-communicable diseases; Shapley decomposition; unconditional quantile regression

JEL codes: C1, D63, I12, I14.

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1 Introduction

Health inequality has many sources, not all of which are equally objectionable. The existing literature focuses on socio-economic inequalities in health and variations associated with differences in living conditions, access to health care, and health-related lifestyle (e.g., Contoyannis and Jones, 2004; Baum and Ruhm, 2009). This literature implicitly suggests a distinction between legitimate and illegitimate inequalities. Building on Roemer's (1998, 2002) influential formalisation of the concept of inequality of opportunity (IOp), the "egalitarian" framework does not necessarily indicate equality of the distribution of outcomes *per se* but emphasises the role of individual responsibility in defining a "fair" distribution (Fleurbaey, 2008; Fleurbaey and Schokkaert, 2009, 2012; Ramos and Van de gaer, 2016; Roemer and Trannoy, 2016).

IOp has influenced the policy agenda in recent years (World Bank, 2005; NHS England, 2017) and a growing literature has addressed the measurement of IOp in health (e.g., Fleurbaey and Schokkaert, 2009; Garcia-Gomez et al., 2015; Jones et al., 2012, 2014; Jusot et al., 2013; Li Donni et al., 2014, 2015; Rosa Dias, 2009, 2010; Trannoy et al., 2010). However, many of the existing studies employ subjective self-assessed health (SAH) measures that are inherently categorical and ordinal. This was recently acknowledged by Carrieri and Jones (2018), who propose a semiparametric approach to decompose *ex post* IOp into the direct contribution of efforts and the direct and indirect contribution of circumstances, using ratio-scaled objectively measured blood-based biomarkers in the Health Survey for England.

The IOp approach aims at disentangling inequalities by circumstances, for which individuals cannot be considered responsible, and efforts, for which individuals should be considered responsible. As will be discussed later, there are two approaches to IOp: the ex-ante and the ex-post approach (eg., Fleurbaey and Schokkaert, 2009; Fleurbaey and Peragine, 2013; Li Donni et al., 2014). The ex post approach seeks equality of outcomes among people who have exerted the same degree of effort, regardless of their circumstances (Roemer, 1998). The ex ante approach to IOp is based on the principle that there is equality of opportunity if all individuals face the same opportunity set, prior to their efforts and outcomes being realised (Fleurbaey and Schokkaert, 2009; Van de Gaer, 1993). The ex-ante approach suggests that all individuals have equal opportunities if (in expectation) no differences in outcomes emerge from having different circumstances. The expectation over outcomes within a circumstances type can be taken with a simple mean (utilitarian reward) or with some inequality aversion (Van de Gaer, 1993; Ooghe and Schokkaert, 2007). In light of the context for UK health policy, in this study we adopt an ex ante approach that focuses on inequality in the distribution of outcomes across social types, defined by their circumstances. We draw on the legal and health policy context to support this approach.

With respect to the UK policy setting, a recent NHS Scotland policy report states that health inequalities go against the principle of social justice because they are avoidable (NHS Health Scotland, 2015). In particular, it is argued that "health inequalities do not

occur randomly or by chance, but are socially determined by circumstances largely beyond an individual's control". More broadly, NHS England draws on the international definition of health inequalities, as adopted by the World Health Organisation (WHO); health inequalities are defined as avoidable inequalities in health between groups of people within countries and between countries (NHS England, 2017). NHS England laid out the public sector equality duty to reduce health inequalities, which are set out in the Health and Social Care Act 2012. These health inequalities "can cut across a range of social and demographic indicators including socio-economic status, occupation, geographical location and the protected characteristics laid out in the Equality Act 2010" (NHS England, 2017).

These obligations to address health inequalities flow down to specific chronic conditions such as those directly relevant to the biomarker data used in this study; for example, through the Healthier You: NHS Diabetes Prevention Programme that was launched in 2016. Given that diabetes is one of the long-term conditions that may be defined as a disability under the Equality Act 2010, addressing diabetes prevalence is both an equalities issue and a health inequalities issue (NHS England, 2017). In line with the WHO's targeting of cardio-vascular (CVD) health (WHO, 2007), the recent NHS Long-term Plan highlights timely access to treatment and improving treatment of high-risk conditions including hypertension and high cholesterol, which are linked to CVD disease (NHS, 2019). Cardiovascular conditions are one of these Long-term Plan priorities, being largely preventable, more common in areas of deprivation and a major cause of health *inequalities* (NHS, 2019); the Long-term Plan identifies air pollution, smoking, obesity and poor diet as risk factors for CVD.

Reducing health inequalities is therefore at the centre of the policy agenda (Bleich et al., 2012), with the WHO calling for a global action at different levels of policy making to tackle health inequalities and promote the health of the more vulnerable population groups (Marmot et al., 2012; WHO, 2017). More specifically, in the context of the UK health policy, the guidance for those allocating health care resources at a local level is to '…pay particular attention to groups or sections of society where improvements in health and life expectancy are not keeping pace with the rest of the population' (NHS England, 2015a). In the case of major chronic health conditions that are relevant to this study, such as cardiovascular conditions, diabetes and obesity, these transfers could be achieved through targeting of fiscal, environmental, health promotion and public health policies such as clean air zones, medical control of blood pressure and cholesterol, health education, preventative interventions and taxes on tobacco or sugary drinks (Marmot et al., 2012; NHS England, 2015a; Thomson et al., 2018).

Distributive justice encompasses the fair distribution of health, since health is an important component of well-being and contributes to an individual's capability to function (Zheng, 2011). The literature discusses the theoretical considerations on how the inequality concept, as initially applied to the income domain, can be transferred to the health domain (e.g., Culyer, 2014; Zheng, 2011). However, one of the major complications of the measurement of health inequality, as opposed to income inequality, is the measurement of health itself (e.g., Costa-Font and Cowell, 2019; Nesson and Robinson,

2019; Zheng, 2011). Most of the social science surveys used to measure (socioeconomic) inequalities in health are limited to SAH measures. SAH measures are qualitative, subjective assessments of individual's health and are typically ordinal in nature. Several approaches have been introduced to cardinalise SAH facilitating the use of conventional inequality measures, however the cardinalisation methods have been criticised as an important source of bias (Costa Font and Hernández-Quevedo, 2013), with the relevant inequality results being sensitive to the cardinalisation method used (Ziebarth, 2010). Bond and Lang (2019) show that between-individual comparisons of well-being measures are fragile to different cardinalisations of the inherently ordinal data. Another complication with the self-reported measures of health is that they are subject to significant misreporting, with the reporting bias varying systematically with individual's socioeconomic characteristics, posing significant implications for the robustness of earlier inequality studies (e.g., Bago d'Uva et al., 2008; Dowd and Zajacova, 2010; Jürges, 2007). Availability of ratio scale, continuous, biomarker measures may offer an attractive alternative here.

In this study, we use nationally representative UK data (Understanding Society: the UK Household Longitudinal Study; UKHLS) to provide a comprehensive analysis of ex ante IOp and its underlining sources using objective health indicators. A contribution of our paper is that we are using nurse-collected and blood-based biomarkers that are each directly relevant to diagnosis, monitoring and the clinical management of specific chronic health conditions: obesity, high blood pressure, inflammation, diabetes and high cholesterol. Higher biomarker values indicate the staging or severity of the particular condition of interest. Over and above being objectively measured, our biomarkers are continuous, ratio-scale measures; thus, our inequality analysis does not depend on cardinalising the health outcome of interest. We use each biomarker separately and we also construct a composite score as a proxy measure of the cumulative wear and tear on the body, reflecting how repeated exposures to adverse circumstances (social, economic and environmental stressors) may get "under the skin" (e.g., McEwen, 2015; Seeman et al., 2004; Turner et al., 2016); similar composite health measures are often called allostatic load and have recently been used in health inequalities research (Nesson and Robinson, 2019).

Given our biomarker measures, the IOp analysis in this study should be interpreted in terms of redistribution of the health burden and functioning and, consequently, social welfare with respect to the particular, preventable health condition (and its severity) that is directly relevant to each of our biomarkers.

A key question is whether the Pigou-Dalton principle holds in this case. The original Pigou-Dalton principle states that a mean-preserving income transfer from a richer to a poorer person should increase the sum of social welfare. The intuition behind the Pigou-Dalton transfer principle is not based on income as such but, more broadly, on individuals' and society's welfare, which is determined (at least partially) by income. In this utilitarian framework, it has been assumed that utility is an increasing and concave function of income, with diminishing marginal utility of income (Atkinson and Brandolini, 2015; Schwartz and Winship, 1980). For example, taking a dollar away from a richer person and

giving it to a poorer person, we decrease the first person's welfare by less than we increase the poorer person's welfare and, thus, we achieve an increase in the total welfare (as the Pigou-Dalton principle assumes). In a parallel concept, assuming that utility is an increasing concave function of good health, we may consider the association between social welfare and elevated, preventable morbidity and mortality risks as reflected in higher biomarker scores. As we will discuss later, medical research shows a positive and typically convex relationship between our biomarker scores and elevated morbidity and mortality. This indicates that a reduction in the spread of the biomarker value by a hypothetical reduction (-δ) in the biomarker score from someone with a high initial biomarker score (i.e., someone with poor health initially and, hence, higher marginal utility of improving health) that is offset by an increase (+δ) in the biomarker score for someone with a low biomarker score (someone who is more healthy initially and hence with lower marginal utility of health), would increase the first persons' welfare by more than we reduce the healthier person welfare; this is because utility is assumed to be an increasing concave function of health and that the medical evidence suggests that the harm associated with the biomarkers we use tends to be steeper for higher biomarker scores (those in poorer health). Overall, based on the evidence from the clinical literature presented below, we maintain the assumption that the Pigou-Dalton principle holds for our biomarkers and the analysis in our paper.

We use the objective health measures to estimate both absolute measures of the level of IOp and measures that express IOp as a fraction of the overall inequality in our health measures. We adopt the direct *ex ante* parametric approach proposed by Ferreira and Gignoux (2011, 2013). The advantage of the parametric approach is that, unlike nonparametric tests for IOp (e.g., Lefranc et al., 2009; Rosa Dias, 2009), it does not suffer from a curse of dimensionality, due to insufficient sample sizes for social types (groups of people sharing identical circumstances).[1] Moreover, even in the presence of unobserved circumstances, our IOp measures can be interpreted as the lower-bound estimates of overall IOp, i.e., of the inequality due to all circumstances, not only those that are observed (Ferreira and Gignoux, 2011).

We decompose the direct *ex ante* measure of IOp in our health measures into its sources. Shapley-decomposition techniques allow us to identify which circumstances are more relevant to shaping IOp in our biomarker measures. Given that age and gender are principal drivers of variations in health, the sample is then split by gender and by age groups to analyse the extent of IOp that is relevant to circumstance variables apart from age or gender and how these inequalities may vary by gender and across the adult lifespan[2].

[1] Maintaining a reasonable number of observations within each social type is a challenging issue given the usual sample sizes of social-science datasets and the relatively large number of circumstances that empirical researchers wish to use to partition the population by types (Ferreira and Gignoux, 2011; Carrieri and Jones, 2018).

[2] For example, it has been shown that the association between education and health may follow heterogeneous patterns by age and gender (e.g., Davillas et al., 2017; Baum and Ruhm, 2009). We extend this literature to the IOp context by exploring differential patterns by age and gender.

Finally, we relax the assumption of inequality neutrality within types, that is implied by the conventional parametric approach, and extend the literature on the decomposition of *ex ante* IOp, capitalising on the continuous nature of our health outcomes. We use the recentered influence function (RIF) approach to distributional analysis (Firpo et al., 2009), to explore how the contribution of circumstances may vary across the distribution of biomarkers. Shapley decompositions are implemented at different quantiles of the biomarker distribution to explore the underlying sources of these inequalities, with a particular focus on the right tails, where clinical concerns are typically focused. We again split the sample by gender and by age groups to analyse the contribution of socioeconomic circumstances (apart from age and gender) at different biomarker quantiles, spanning the whole distribution of our health measures.

2 Methods

Roemer (1998) assumes a responsibility cut by which factors associated with individual attainments can be partitioned into: a) effort factors, for which individuals should be held partially responsible, and b) circumstances which are beyond individuals' control. Following the IOp in health literature (Carrieri and Jones, 2018; Jusot et al., 2013; Rosa Dias, 2010), a generalised health production function for the health outcome (y_i) for each individual (i) can be defined as a function of a vector of circumstances (C_i) and of efforts (E_i). Assuming that circumstances are not affected by efforts, while efforts may be influenced by circumstances (Bourguignon et al., 2007; Ferreira and Gignoux, 2011; Roemer, 1998, 2002), we can write:

$$y_i = h(C_i, E(C_i, v_i), u_i) \tag{1}$$

where v_i and u_i are unobserved error terms which capture the random variation in the realised outcomes, sometimes labelled as 'luck' in the IOp literature (Lefranc et al., 2009; Lefranc and Trannoy, 2017).[3] To be specific, v_i represents random variation in effort that is independent of C and u_i represents random variation in the outcome that is independent of C and E.

In principle, the structural form (1) can be used in an *ex post* framework to decompose the direct and indirect contribution of circumstances and efforts. Here we adopt an *ex ante* approach and are interested in measuring overall IOp as a share of total inequality. Then, assuming additive separability and linearity of $h(.)$ and $E(.)$, a linear reduced form can be derived:

[3] In the Roemerian framework, the partial correlations between C and E should also be treated as circumstances, embodying the indirect effect of the unjust circumstances on health that is channelled through effort (see Ferreira and Gignoux, 2011; Rosa Dias, 2009). This is embodied in the reduced form coefficients that capture both direct and indirect effects. However, the ethical stance of the Roemer concept is open to debate. Examples of empirical methods to compare the Roemer view with other more liberal perspectives are available elsewhere (Jusot et al., 2013).

$$y_i = C_i\psi + \varepsilon_i \quad (2)$$

where the coefficients ψ reflect the total contribution of circumstances and include both the direct effect of circumstances on health, and the indirect effect of circumstances through efforts.

The *ex ante* approach to IOp is based on the principle that there is equality of opportunity if all individuals face the same opportunity set regardless of their circumstances and prior to the realisation of effort and the outcomes. The *ex ante* approach can be implemented empirically using information on observed circumstances and does not require measures of effort. These circumstances are used to measure the opportunity set for each individual (e.g., Aaberge et al., 2011; Fleurbaey and Peragine, 2013; Ooghe et al., 2007; Van de gaer, 1993). Two approaches have been adopted to do this:

i. The first uses the mean of outcomes within types, $E(y_i|C_i)$. This corresponds to the approach suggested by Roemer (1998, 2002) and has been termed "utilitarian reward". This implies inequality neutrality within types. Jones et al. (2014) note the equivalence between the mean and the area to the left of the distribution function, $F(y_i|C_i)$, and they therefore use this as their criterion for health policy evaluation. This can therefore be considered as a 'mean-based' approach, where equality of opportunity corresponds to equality of mean outcomes across types (e.g., Ferreira and Gignoux, 2011, 2013; Lefranc et al., 2009; Van de gaer, 1993).

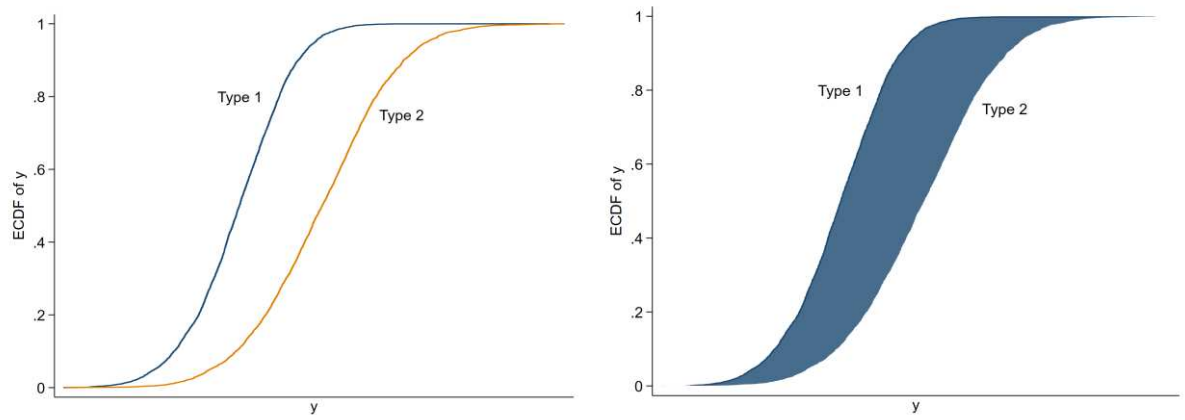
ii. The second approach uses a more general definition that interprets the full type-specific conditional outcome distribution $F(y_i|C_i)$ as the opportunity set (e.g., Lefranc et al., 2009; Lefranc and Trannoy, 2017; Ooghe et al., 2007; Ramos and Van de gaer, 2016). This goes beyond the mean-based approach, focusing on differences in distributions across types, and allows for the possibility of inequality aversion within types such that, for example, more significance is attached to inequalities across types at worse levels of health outcomes. The implications of this second approach are explored using a distributional regression approach that is described below.

Figure 1 illustrates these concepts for the case where there are just two values of circumstances, represented by types 1 and 2. The *ex ante* approach compares the distribution of outcomes conditional on type, as shown in the left-hand panel, and interprets these distributions as the opportunity set for each type. In particular, the mean-based approach focuses on differences in means across types, given by the area to the left of the distribution functions and hence, in this case, shown by the dark shaded area in the right-hand panel. Differences in means is regarded as a weak test for IOp. A stronger test takes account of the shape of the distribution within types and, depending on the degree of inequality aversion within types, may give more weight to horizontal differences between the distributions at different levels of the outcome[4]. This motivates

[4] In the context of our application to biomarkers the notion of inequality aversion within types may be motivated by the fact that these are measured in physical units, y_i, which may not correspond to their social value, say $\omega(y_i)$. For example, rather than being linear, the function $\omega(.)$ may give

our analysis of the contribution of circumstances at different points of the biomarker distribution.

Figure 1. *Ex ante* IOp and distribution functions.



We begin with the mean-based framework. The direct approach, as in Ferreira and Gignoux (2011), measures inequality in a counterfactual in which all inequalities are attributable to circumstances. This involves defining a smoothed distribution from the distribution of (health) outcomes (y_i) and a partition of ($k = 1,2..K$) types by replacing each individual health outcome y_i^k with the relevant type-specific mean (μ_i^k) and, then, using inequality indexes to measure IOp (Ferreira and Gignoux, 2011).[5] In practice, the mean-based direct parametric approach to measure *ex ante* IOp is based on using predictions of $E(y_i|C_i)$ from the reduced form as the counterfactual outcome:

$$\tilde{y_i} = C_i\hat{\psi} \tag{3}$$

where $\hat{\psi}$ represents the OLS estimates of the coefficients in equation (2) (Checci and Peragine, 2010; Rosa Dias, 2010; Trannoy et al., 2010; Ferreira and Gignoux, 2011, Li Donni et al., 2014; Abatemarco, 2015). The predicted health outcomes are the same for all individuals with identical circumstances (Ferreira and Gignoux, 2011). Thus, IOp can be estimated using an inequality measure ($I(.)$) applied to $\tilde{y_i}$:

$$\theta_a = \mathrm{I}(\tilde{y_i}). \tag{4}$$

different weight to outcomes above or below the clinical risk thresholds that are associated with some of the biomarkers.

[5] The indirect approach (as in, for example, Bourguignon et al., 2007) uses the difference between inequality in actual outcomes and a counterfactual in which there is no IOp, calculated using a reference level of circumstances. We have opted for the direct approach in our analysis. Parametric estimation shows that the direct and indirect approaches result in similar, but not exactly identical, results (Ferreira and Gignoux, 2011); this is typical, given the functional form assumptions that are relevant to parametric estimation models.

A relative measure of IOp, expressing IOp as a fraction of the overall health inequality ($I(y_i)$), can be obtained by:

$$\theta_r = \frac{\mathrm{I}(\tilde{y}_i)}{I(y_i)}. \quad (5)$$

Following Ferreira and Gignoux (2011), we use the mean logarithmic deviation (MLD) inequality index as our principal measure of inequality I(.). They adopt the MLD because of its path-independent decomposability properties (Checchi and Peragine, 2010; Ferreira and Gignoux, 2011; Ramos and Van de Gaer, 2016; Wendelspeiss Chávez Juárez and Soloaga, 2014). If I(.) needs to satisfy the path-independent decomposability axiom in addition to other typical axiomatic properties relevant to the measurement of inequality literature, i.e., symmetry, transfer principle, scale invariance and population replication this restricts the eligible "path-independent decomposable" class of inequality measures for the case of ratio-scale outcomes (as with our biomarkers) to a single measure, the MLD (Ferreira and Gignoux, 2011). The main advantage of the decomposability axiom for the purpose of our analysis is that it allows us to decompose total inequality into the share attributed to circumstances and a residual (eq. 3, 4 and 5) (Checchi, and Peragine, 2010; Ferreira and Gignoux, 2011). To check the robustness of our findings we complement our results by adding a sensitivity analysis to show how our decomposition of the MLD compares to the variance share. The variance share is the share of the total variance in our biomarkers that is attributed to circumstances and is a relative IOp measure that satisfy all standard axioms and the axiom of the path independent decomposability, but is more appropriate for outcomes that are not ratio-scaled, unlike our biomarker data (Ferreira and Gignoux, 2013; Wendelspeiss Chávez Juárez and Soloaga, 2014).[6]

The MLD is zero when there is no inequality, and takes on larger positive values as ill-health is distributed more unequally. It should be explicitly noted here that our analysis does not account for unobserved circumstances that are not available in the dataset. However, it has been shown that equations (4)-(5) can be interpreted at least as the lower-bound estimates of inequality due to all predetermined circumstances (Ferreira and Gignoux, 2011).

Decompositions of IOp

We use the Shapley decomposition to explore the contribution of each of the circumstances to the total IOp in health (Fajardo-Gonzalez, 2016; Shorrocks, 2013; Wendelspeiss Chávez Juárez and Soloaga, 2014). Specifically, inequality measures for all possible permutations of the circumstance variables are estimated and, then, the average marginal effect of each circumstance variable on the total IOp is calculated. The key advantage of the Shapley-decomposition, unlike other decomposition methods, is that it is both path independent and exactly additive, with the different components sum up exactly to the total IOp (Fortin et al., 2011; Wendelspeiss Chávez Juárez and Soloaga, 2014). This makes the

[6] However, inequality measures based on the variance have been used in the existing IOp (in health) literature (e.g., Carrieri and Jones, 2018; Ferreira & Gignoux, 2013; Garcia-Gomez et al., 2015; Jusot et al., 2013).

additive decomposability property of the MLD less of a distinctive advantage and we apply the Shapley decomposition to the variance in our robustness checks.

Given that age and gender are the main sources of variation in health, we then use a split sample analysis to further analyse gender and age differentials in IOp. This involves estimating IOp separately for each population sub-group (i.e., by gender and across age groups), exploring the role of circumstance variables apart from age or gender.

Using distributional regressions to relax inequality neutrality within types

The methods described so far measure and decompose overall IOp in health using linear parametric regression specifications and a counterfactual based on the conditional mean. As noted above, this implies inequality neutrality within types. In our context this may be too restrictive and, for example, we may wish to give greater weight to the contribution of circumstances in the upper tail of the distribution of biomarkers, where individuals are at greater risk of developing chronic health problems.

To assess the implications of relaxing inequality neutrality we propose a method of decomposing the contribution of circumstances to the overall IOp at different points in the distribution of the outcome. This makes use of unconditional quantile regression (UQR) based on the RIF approach (Firpo et al., 2009) to decompose the contribution of circumstances at specific quantiles of the biomarker distributions.

The RIF method works by providing a linear approximation of the unconditional quantiles of each biomarker. Subsequently, the law of iterated expectations is applied to the approximated quantile and used to estimate the marginal effect of circumstances through a regression of the RIF on the circumstance variables. The total contribution of circumstances, at each quantile q_τ, can be then obtained by:

$$RIF(y_i; q_\tau) = C_i\alpha^\tau + \varepsilon_i^\tau \tag{6}$$

where α^τ are the coefficients at different quantiles and ε_i^τ stands for the error term. This is illustrated by Figure 2, which shows how the RIF regressions aim to capture differences attributable to circumstances at specific quantiles of the distribution, represented here by the horizontal line at the 60th percentile.

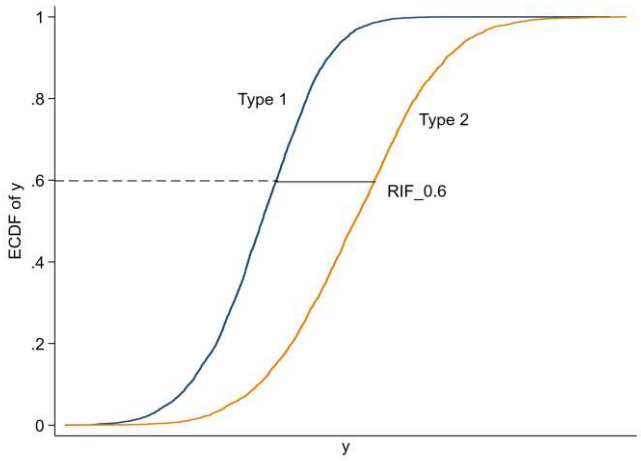
Then the counterfactuals used in the direct approach are given by:

$$\widetilde{y_i^\tau} = C_i\widehat{\alpha^\tau} \tag{7}$$

The variation in these fitted values, which capture the role of circumstances since counterfactuals ($\widetilde{y_i^\tau}$) are the same for all individuals with identical circumstances (in line with the concept of the direct *ex ante* IOp), can be summarized using an inequality index (here we again use the MLD, as in equation 4). As the RIF equations are additive and linear, the Shapley decomposition and split sample analysis can be then applied to this

index to explore the contribution of each circumstance variable as well as differentials in their contribution by gender and age at different quantiles of biomarker distribution.

Figure 2. *D*istributional regressions.



3 Data

The data come from UKHLS, a longitudinal, nationally representative study of the UK. We use the General Population Sample (GPS) component of UKHLS, a random sample of the general population. As part of wave 2 (2010-2011), nurse-measured and non-fasted blood-based biomarkers were collected for the GPS. The wave 2 nurse visits included 15,632 respondents, while blood-based biomarkers impose further restrictions on the sample since they require the successful collection and processing of blood samples.[7] Exclusion of missing data on covariates reduces the potential sample to a maximum of 14,068 and 9,005 individuals with valid nurse-collected measurements and blood-based biomarkers, respectively.

Biomarkers

We focus on physical measurements and blood-based biomarkers that are directly relevant to major chronic conditions such central obesity, high blood pressure, inflammation, diabetes and high cholesterol. Our nurse-collected measurements are the waist-to-height ratio (WHR), defined as waist circumference over height, being a validated measure of central adiposity (Swainson et al., 2017) and systolic blood pressure. Our

[7] Respondents were eligible for nurse visits if they were aged 16+, lived in England, Wales, or Scotland, and were not pregnant. Blood sample collections were further restricted to those who had no clotting disorders and no history of fits.

blood-based biomarkers reflect 'fat in the blood', 'sugar in the blood' and markers for inflammation. The cholesterol ratio, the ratio of total cholesterol over high-density lipoprotein, is our 'fat in the blood' biomarker used for diagnosis, staging and monitoring of high cholesterol. High levels of the cholesterol ratio are associated with increased risk of cardiovascular conditions. Glycated haemoglobin (HbA1c) is a standard diagnostic test for diabetes. Given that no single inflammatory biomarker can capture all aspects of inflammation because of the complexity of the inflammatory profiles (Castagné et al., 2016; Davillas et al., 2017), two inflammatory biomarkers are used in our analysis due to data availability: C-reactive protein (CRP) and fibrinogen[8]. CRP rises as part of the immune response to infection. Fibrinogen is a glycoprotein that aids the body to stop bleeding by promoting blood clotting, and is regarded as an inflammatory biomarker (Davillas et al., 2017).

In addition to each of the specific markers, we also combine them in a composite measure, which gives an overall assessment of a respondent's physiological condition. We construct an index of multi-system risk, often called allostatic load (e.g., Davillas and Pudney, 2017). Allostatic load is elevated when a person's biological systems are affected by repeated stressors, resulting in persistently elevated or altered levels of a number of biomarkers associated with 'chronic stress'. Allostatic load is, thus, a measure of cumulative wear and tear in a number of physiological systems. As such, it is an ideal health measure for the purpose of the measurement of IOp because it captures chronic physiological responses that are associated with social and environmental stress and, thus, can be considered as more proximal outcomes in the process through which economic and social circumstances may get "under the skin" across the lifespan (Davillas et al., 2017; McEwen, 2015; Seeman et al., 2004).

The literature on the measurement of allostatic load has shown that a single additive measure of the different biomarkers is sufficient to measure allostatic load (Howard and Sparks, 2016).[9] Specifically, our composite measure combines all six nurse-collected and blood-based biomarkers considered in our study: WHR, systolic blood pressure, HbA1c, CRP, fibrinogen and cholesterol ratio. We then transform each of these biomarkers into standard deviation units and sum them (Davillas and Pudney, 2017; Howard and Sparks, 2016; Seeman et al., 2001). Higher values of allostatic load indicate worse health.

It should be noted here that the biomarkers used in our analysis are surrogate markers of cardiovascular and total mortality risks, being at the centre of causal pathogenic

[8] It has been shown that relying only on one inflammatory marker makes generalisations of the results to inflammatory burden uncertain. For example, because of the natural complexity of the inflammatory mechanisms, such as synergistic or offsetting effects, analysis needs to account for more inflammatory biomarkers rather than rely on CRP alone (Castagné et al., 2016; Davillas et al., 2017). Moreover, there are concerns whether CRP captures recent infections rather than systematic inflammation (Povoa et al., 2005), which indicates that results on CRP may be contaminated by recent infections.

[9]It has been shown that individual biomarkers commonly used to calculate allostatic load do not differ in their strength of association, or factor loadings, to a single underlying factor structure of allostatic load. Thus, simple, additive measures of allostatic load are sufficient to model allostatic load (Howard and Sparks, 2016; Seeman et al., 2001).

mechanisms[10]. To highlight the shape of the association between our biomarkers and all-cause mortality risks, we summarise some of the existing evidence from the medical literature. Mortality hazards are an increasing function of WHR across its distribution, with a steeper risk profile at the higher quantiles of the distribution (Ashwell et al., 2014). Systolic blood pressure is considered as more relevant to cardiovascular mortality risks as opposed to diastolic blood pressure, with mortality risks being steeper at higher levels of systolic blood pressure (Bundy et al., 2017). The cholesterol ratio has the advantage that it involves the opposing effects of "bad" and "good" cholesterol varieties on health and is considered as a stronger predictor of all-cause mortality risks than each of the individual cholesterol concentrations (Prospective Studies Collaboration, 2007); it has been shown that cardiovascular mortality risks gradually increase, with a weaker associations at the lower levels of cholesterol ratio and steeper associations at higher levels (Prospective Studies Collaboration, 2007). Implications on individual's health are not homogeneous across the HbA1c distribution, with higher values being an increasing function of diabetes severity, morbidity and mortality risk. There is a J-shaped association between HbA1c and cardiovascular disease and cancer mortality risks (Zhong et al., 2016). Turning to our inflammatory biomarkers, CRP has a J-shaped association with cardiovascular and non-cardiovascular mortality risks, with the association being steeper at the higher CRP levels (Emerging Risk Factors Collaboration, 2010). All cases mortality rates are a progressively increasing function of fibrinogen, with stronger associations at the higher fibrinogen quantiles (Acevedo et al., 2002; Danesh et al., 2005). Our composite health measures, allostatic load, is a strong predictor of future mortality risks with a "dose-response" association that is more pronounced and steeper at higher allostatic load values (Castagné et al., 2018; Seeman et al, 2004).

Note that our biomarkers are ratio scaled, i.e., they have a true zero value and their measurement scale is unique up to a proportional scaling factor. Specifically, they are measured in natural units: CRP (mg/L), cholesterol ratio (both total cholesterol and high-density lipoprotein cholesterol are measured in mmol/L), fibrinogen (g/L), HbA1c (mmol/mol), systolic blood pressure (mmHg) and WHR (both waist circumference and height are measured in cm). Our measure of allostatic load is the sum of these biomarkers, standardised by their standard deviations, and is invariant to rescaling of the individual biomarkers.[11]

[10] A few biomarkers can achieve surrogate endpoint status as they need to satisfy a number of criteria to ensure causal links to the pathogenic mechanisms to the elevated mortality risks (Aronson, 2005; Biomarkers Definitions Working Group, 2001). Our biomarkers are carefully selected as good surrogate measures of mortality risks in several clinical trials and medical research. For example, blood pressure, cholesterol levels and CRP are considered as good surrogate measures of a number of life-threatening events (Weintraub et al., 2015; Welsh et al., 2017).

[11] Given the absence of longitudinal data on biomarkers, our results should not be viewed and interpreted as a lifetime health achievements approach to measure and analyse IOp in health. Recall however, as a potential way to alleviate these concerns, that our biomarkers reflect the risk for chronic conditions and that allostatic load measures the cumulative wear and tear of several physiological systems due to repeated exposure to social, economic and environmental disadvantages.

Circumstances

The choice of measured circumstance factors follows the recent empirical literature, informed by the normative framework for health equity and the UK policy and legal context (Carrieri and Jones, 2018; Rosa Dias, 2009, 2010; Jusot et al., 2013). Our circumstance variables embody the ethical position of the responsibility cut, defining illegitimate sources of health inequality.

Specifically, we have adopted Roemer's pragmatic approach of drawing on the socio-legal context of the analysis to inform the responsibility cut and to define relevant circumstances. Our paper addresses IOp in health in the United Kingdom and hence the legal duties with respect to health inequality that face the National Health Service (NHS) help to define that context. For example, NHS England's inequality aims include: "advance equality of opportunity between persons who share a relevant protected characteristic and persons who do not share it" (NHS England, 2015b), where the protected characteristics are defined by the Equality Act 2010 and include age, sex and race. This is reinforced by the evidence on differential healthcare and social care treatment by age and gender regardless of healthcare need (Department of Health, 2012; Oliver, 2009). One way in which these aims are put into practice is through NHS England's 2015 guidance on monitoring equality and health inequalities (NHS England, 2015b); the categories for collecting information on equality include the protected characteristics under the Equality Act 2010, such as age, sex and race, as well as information on socio-economic factors and educational attainment.

The Equality Act of 2010 defines protected characteristics that include age, sex and race. We treat sex and age (dummies for 10-year intervals between 16 and 75 and a dummy for 75+) as circumstances (Carrieri and Jones, 2018). Nationality and linguistic background is proxied by a dummy for speaking English at home during childhood. It should be noted however that it is unlikely to achieve universal agreement. In the IOp in health literature existing studies treat age and gender as either legitimate or illegitimate sources of health inequalities. Either way, the demographic variables are included in the reduced form regressions for health outcomes in all of these studies (e.g., Carrieri and Jones, 2018; Garcia Gomez et al., 2015; Jusot et al., 2013; Li Donni et al., 2014; Trannoy et al., 2010). However, the way that these variables are handled in subsequent decomposition or counterfactual analyses differs depending on whether they are treated as demographic controls (and, thus, should be included in the regression analysis but not treated as circumstances in the measurement and decomposition of IOp) or, at least to some extent, as circumstances. Garcia-Gomez et al. (2015), in their review analysis on legitimate or illegitimate sources of health inequalities, argue that the normative status of any age- and gender-specific standardisation is not clear in the case of the IOp literature and they remain agnostic regarding whether age and gender should to be treated as either legitimate or illegitimate sources of health inequalities. Specifically, the association of age and gender with the biomarkers cannot be considered as entirely "irremediable" and may vary over time, depend on (health) policy and be influenced by socially constructed roles and relationships (Connell, 2012; Doyal, 2001; Garcia-Gomez et al., 2015; Rieker and Bird, 2005). The advantage of our Shapley decompositions is that the results can be interpreted under either perspective, as the analysis allows us to identify the contributions of age and

gender separate from the other circumstance variables. In the spirit of Garcia-Gomez et al. (2015), this analysis allows the reader to draw their own conclusions regarding the responsibility cut and, for example, to disregard the contribution of age or gender if they wish. Nevertheless, we note that the decomposition only provides upper and lower bounds on the contribution of age and gender.

The Equality Act 2010 does not directly encompass socioeconomic status (SES) among its protected characteristics but this has been a concern of the existing literature on IOp. Childhood SES is regarded as an important source of IOp in health, being beyond individual's control and exerting a lasting effect on individual's adult health (Jusot et al., 2013; Rosa Dias, 2009, 2010). We use both parental occupational status and education to proxy childhood SES. The occupational status of the respondent's mother and father, when the respondent was aged 14, is measured using two categorical variables (one for each parent) with six categories: not working (reference category), four occupation skill levels (based on the skill level structure of the Standard Occupational Classification 2010) and a category for missing data. Given the high correlation between mother's and father's education, we combine them creating a measure capturing the highest parental education level (Kenkel et al., 2006). This is a five category variable measured as: left school with no/some qualification (reference category), post-school qualification/certificate (e.g., an apprenticeship), degree (university or other higher-education degree) and a missing data category.[12]

The legal and policy context in the UK also frames the notion of an age of responsibility. This age varies across different dimensions such as criminal responsibility and age of consent. Young people aged over 18 are treated as an adult by the law. Here we make the normative assumption that the level of secondary schooling achieved by age 18 is beyond individual's responsibility, influenced by parental and environmental factors during individual's earlier life, and therefore individuals' own education constitutes a circumstance (Jones et al., 2012; Carrieri and Jones, 2018). Education is measured as: no/basic qualification (reference), O-Level, A-Level/post-secondary and degree. Descriptive statistics for circumstances and biomarkers are available in Tables A1 and A2 (Appendix).

[12] Comparison of summary statistics for the biomarkers reveals similar results for our working sample and the sample restricted to non-missing parental information (Table A2, appendix), suggesting that the use of missing categories or the exclusion of missing data on parental characteristics should have limited implications for our analysis.

4 Results

4.1. Mean-based measures of *ex ante* IOp

Table 1 presents inequality results for the different biomarkers and for allostatic load. Column [a] shows the total inequality, measured by the MLD. Observed circumstances account for a non-trivial part of the total inequalities as our results from the mean-based *ex ante* IOp measures show (columns [b]-[c]). The contribution of measured circumstances to the total inequality is lowest for CRP (4%), it is higher for cholesterol ratio, fibrinogen (11%) and waist-to-height ratio (17%) and around 20% for systolic blood pressure, HbA1c and allostatic load.

Table 1. Total inequality and IOp (MLD indexes).

	Total inequality [a]	IOp		Sample size
		Absolute IOp [b]	% of total inequality [c=b/a]	
Waist-to-height ratio	0.0116*** (0.0001)	0.0020*** (0.00007)	16.9%	14,068
Systolic blood pressure	0.0087*** (0.0001)	0.0017*** (0.00007)	19.8%	11,865
Cholesterol ratio	0.0583*** (0.0010)	0.0064*** (0.0004)	11.0%	9,005
HbA1c	0.0153*** (0.0006)	0.0030*** (0.0002)	19.5%	8,468
CRP	0.4244*** (0.005)	0.0161*** (0.0020)	3.9%	8,311
Fibrinogen	0.0218*** (0.0004)	0.0023*** (0.0002)	10.7%	8,964
Allostatic load	0.0074*** (0.0001)	0.0016*** (0.0001)	21.8%	6,166

Bootstrapped standard errors in parenthesis (500 replications).
***P<0.01

Specifically, Table 1 shows systematic total inequalities that follow heterogeneous patterns across biomarkers (column a). Beyond CRP, for which the total inequality is much larger compared to all other biomarkers, overall inequality is higher for our fat in the blood biomarker (cholesterol ratio), fibrinogen, diabetes biomarker (HbA1c) and for our measure of adiposity. The contribution of measured circumstances to the total inequality ranges between 11-20% for most of the biomarkers (Table 1, columns b and c)[13] and is broadly in line with previous studies on the presence of considerable social and economic patterning in several cardiovascular risks, such as obesity, diabetes, hypertension and cholesterol (e.g., Baum and Ruhm, 2009; Brunello et al., 2013; Cohen et al., 2010).

[13] Although different techniques and biomarkers are employed by Carrieri and Jones (2018), our results regarding HbA1c (the only biomarker in common) are comparable, with IOp accounting for up to 19% of the total (including the unexplained) inequalities in HbA1c in their analysis of the Health Survey for England.

It should be noted that CRP is a clear outlier here, having a very large overall inequality, while the contribution of the observed circumstances to the total inequality is the lowest (about 4%). The large total CRP inequality may reflect the fact that CRP is an acute-phase biomarker, which may increase up to 1,000-fold in the presence of infection or inflammation (e.g., Sproston and Ashworth, 2018); this indicates that CRP values vary considerably between the healthy and less healthy sub-populations resulting in large overall inequalities. We need to recap here that CRP may capture recent infections, rather than chronic systematic processes, which may be less relevant to circumstances and more likely due to unobserved factors. This may explain why circumstances account for such as small part of the total inequalities, unlike the results for our alterative inflammatory biomarker (fibrinogen).[14]

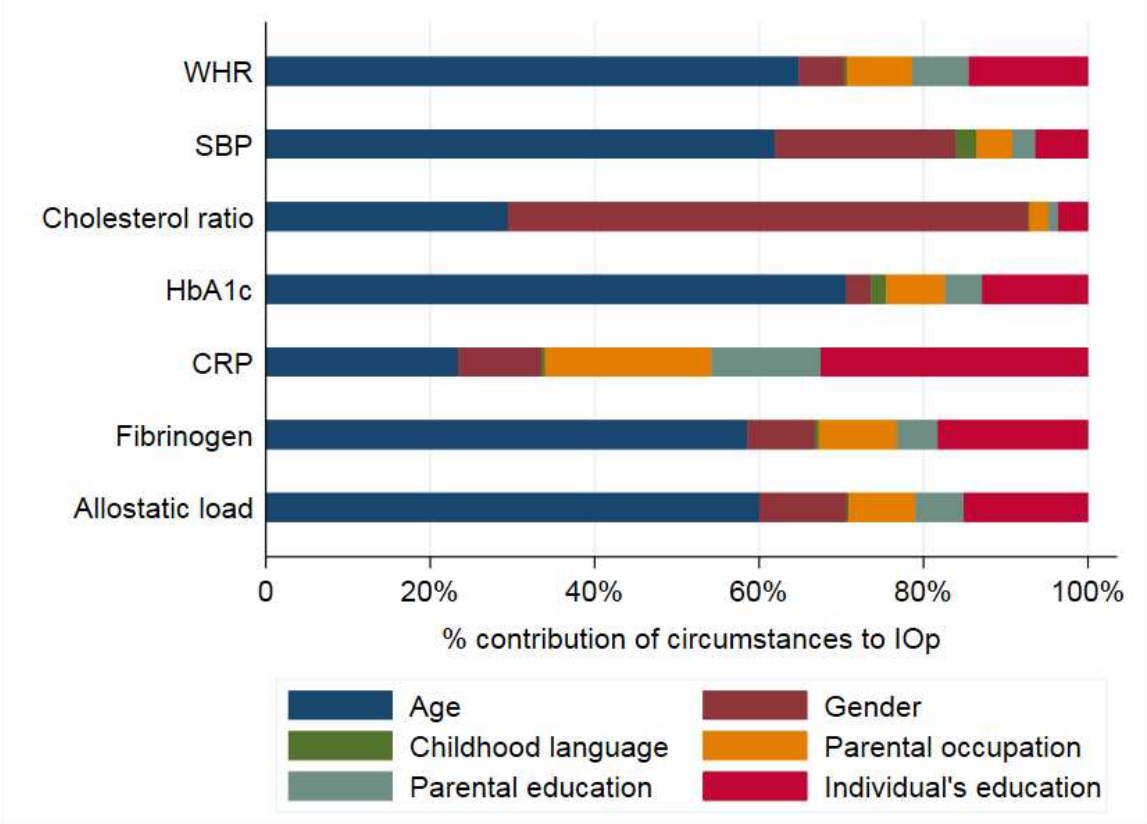
We then explore the contribution of each of the circumstances to IOp using the Shapley-decomposition (Figure 3).[15] Age and gender (in combination) account for the largest part of the IOp in almost all the biomarkers (except CRP), and age accounts for the dominant contribution; this is in line with literature on the role of age and gender on explaining variations in health (e.g., Baum and Ruhm, 2009). Individuals' education and parental occupational status are the second and third sources of IOp, while parental education is the fourth contributor. Respondents' education, parental occupation and parental education account for 15%, 8% and 6% of the total IOp in allostatic load, respectively.[16]

[14] CRP may reflect recent infections rather than solely systematic/chronic inflammation. Although in this study we have excluded those individuals with CRP levels over 10 mg/dL to partially account for this, there is no consensus regarding how (or if it is feasible) to eliminate the potential role of recent infections in CRP concentrations (Povoa et al., 2005). In any case, exclusive reliance on CRP cannot capture all aspects of inflammation (Castagné et al., 2016; Davillas et al., 2017) and, thus, given the aforementioned shortcomings, our results on fibrinogen are of particular importance here.

[15] It should be noted here that the Shapley decomposition analysis may facilitate a standardisation of the health outcomes, which could be equivalent to an "indirect" age and gender standardisation of the health outcomes (O'Donnell et al., 2008). The advantage of the Shapley decomposition is that it allows us to identify the contributions of age and gender separate from the other circumstance variables. In line with our discussion in the circumstances sub-section, this analysis allows the reader to draw their own conclusions regarding the responsibility cut. For example, if someone assumes the pure "control" view, the contributions of age and gender could be deducted from the measure of IOp and this could be used in policy evaluation. Future research that accounts and compares alternative standardisation methods may be useful in case that someone takes the view that the role of age and gender is purely "natural".

[16] As it has been shown by Ferreira and Gignoux (2011), our analysis should be viewed as a lower bound of the share of inequality associated with all circumstances, both observed and unobserved. In the spirit of Ferreira's and Gignoux's (2011) arguments, if age and gender are not included in our set of circumstances, then, the share of inequality attributed to the other circumstances included in our analysis may be over-/under-estimated (depending on their correlation with the omitted age and gender variables); but, our IOp approach still would be considered as a lower bound of the total true IOp, attributed to observed and unobserved (in our analysis) circumstances. By including age and gender in our analysis, following all previous literature of IOp in health, we are able to quantify the contribution of all socio-economic circumstances net of the potential (direct and indirect) role of age and gender. Still, however, our absolute and relative IOp measures should be viewed as lower bounds of the possible true measures of (ex-ante) IOp.

Figure 3. Shapley decomposition of circumstances to IOp.



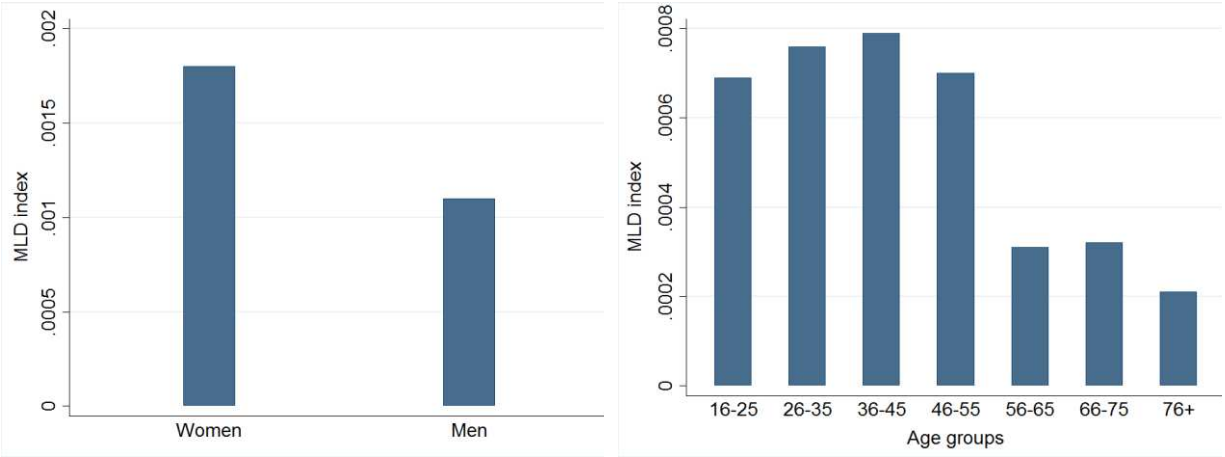
As discussed earlier, we conducted sensitivity analysis to show how our results based on the MLD index compare to the variance share. Table A3 (appendix) shows that our results presented above, based on the MLD index, and those on the variance share (in terms of both the relative IOp measures and the contribution of circumstances to IOp) are practically identical. Given that the MLD is scale invariant but not translation invariant, while the variance share is both scale and translation invariant (Ferreira and Gignoux, 2013), these sensitivity analysis results may alleviate concerns regarding the robustness of our finding with respect to the measurement of inequality.

Given that age and gender are the main sources of variation in health, a split sample analysis is used to explore differentials in the IOp in allostatic load between women and men and across age groups (Figure 4). IOp is evident for both genders, being higher for women than for men (p-value for gender IOp difference: 0.000). These results are in line with recent evidence suggesting that low socio-economic position is more consistently associated with a worse profile of biomarkers for cardiovascular health for women rather than for men (Kavanagh et al., 2010).

Figure 4 also presents the results of the corresponding split sample analysis of IOp in allostatic load by age group. We find that IOp in allostatic load varies substantially across the adult lifespan, being higher for the 26-35 and 36-45 age groups compared to younger and older ages. These results highlight the role of circumstances other than age on

shaping IOp in health and reflect the IOp patterns across the adult life span. Our mean-based analysis shows that the cross sectional health inequalities increase with age up to a limit and then inequality begins to narrow most likely due to the age-as-leveller hypothesis (Baum and Ruhm, 2009; Davillas et al, 2017); the latter suggests that, as far as analysis at the mean of the allostatic load distribution is considered, unavoidable biological processes may dominate the social determinants on health at older ages.

Figure 4. IOp in allostatic load by gender and age.



4.2. Distributional analysis of the contribution of circumstances

Unconditional quantile regression (UQR) models are estimated to measure the contribution of measured circumstances across the biomarker distribution and Shapley decomposition analysis is then used to explore the contribution of circumstances at each quantile (Table 2).

Our results show the presence of systematic variation attributable to circumstances for all health outcomes across the whole distribution (as shown by the MLD indexes). The most striking result from the Shapley decomposition shows that the percentage contribution of socioeconomic circumstances, measured by parental occupation, education and individual's education, increases towards the right tail of the biomarker distribution for most of the biomarkers. For example, the contribution of parental occupation for allostatic load increases from 7% (25th quantile) to 17.4% (95th quantile). It is also notable that, in most cases, the relative contribution of age and sex declines, relative to that attributed to socioeconomic factors, in the right hand tails, where individuals are most at risk of health problems. For example, the joint contribution of age and gender for allostatic load at the 25th quantile is 78%, while socioeconomic circumstances (parental occupation, parental education and own education) account for 21%; the corresponding contributions are almost equal (around 50%) at the top quantiles (Q90, Q95).

Table 2. Contribution of circumstances (MLD) at different biomarker quantiles and Shapley decomposition.

Waist to height ratio	**Q25**	**Q50**	**Q75**	**Q90**	**Q95**
MLD index	0.0040***	0.0025***	0.0014***	0.0011***	0.0008***
		% contribution to IOp			
Age	68.89%	61.81%	52.93%	36.32%	34.00%
Gender	12.73%	7.75%	1.27%	0.55%	1.99%
Childhood language	0.40%	0.44%	0.35%	0.28%	0.27%
Parental occupation	5.64%	9.37%	12.77%	15.71%	15.41%
Parental education	5.29%	6.90%	7.69%	8.69%	8.90%
Individual's Education	7.07%	13.69%	24.98%	38.35%	39.31%
Systolic blood pressure	**Q25**	**Q50**	**Q75**	**Q90**	**Q95**
MLD index	0.0020***	0.0022***	0.0020***	0.0020***	0.0020***
		% contribution to IOp			
Age	41.87%	57.46%	73.00%	77.04%	77.38%
Gender	45.11%	29.58%	9.42%	3.12%	0.65%
Childhood language	4.19%	2.11%	1.66%	1.11%	0.80%
Parental occupation	2.79%	3.45%	5.49%	7.79%	7.32%
Parental education	1.74%	2.60%	3.17%	3.32%	2.79%
Individual's Education	4.29%	4.80%	7.25%	7.64%	11.11%
Cholesterol ratio	**Q25**	**Q50**	**Q75**	**Q90**	**Q95**
MLD index	0.0039***	0.0075***	0.0090***	0.0101***	0.0076***
		% contribution to IOp			
Age	34.28%	31.99%	29.98%	22.16%	25.27%
Gender	55.15%	59.30%	63.68%	70.73%	64.48%
Childhood language	0.03%	0.09%	0.12%	0.16%	0.01%
Parental occupation	3.71%	3.67%	2.30%	1.92%	4.01%
Parental education	3.38%	1.88%	0.84%	0.43%	0.89%
Individual's Education	3.43%	3.07%	3.05%	4.61%	5.34%
HbA1c	**Q25**	**Q50**	**Q75**	**Q90**	**Q95**
MLD index	0.0015***	0.0022***	0.0029***	0.0064***	0.0177***
		% contribution to IOp			
Age	81.01%	78.23%	71.19%	64.85%	55.94%
Gender	0.65%	0.68%	1.48%	3.88%	12.64%
Childhood language	1.04%	0.59%	1.14%	2.00%	3.43%
Parental occupation	6.63%	6.80%	7.07%	8.09%	7.63%
Parental education	5.20%	4.15%	4.28%	4.43%	3.14%
Individual's Education	5.40%	9.55%	14.91%	16.74%	17.21%
CRP	**Q25**	**Q50**	**Q75**	**Q90**	**Q95**
MLD index	0.0356***	0.0313***	0.0239***	0.0138***	0.0073***
		% contribution to IOP			
Age	46.10%	33.57%	20.79%	19.11%	14.43%
Gender	1.00%	7.70%	15.25%	12.72%	4.49%
Childhood language	0.27%	0.23%	0.72%	0.71%	0.70%
Parental occupation	13.11%	13.60%	23.99%	21.77%	34.50%
Parental education	14.43%	14.33%	11.65%	11.59%	15.21%
Individual's Education	25.09%	30.57%	27.59%	34.09%	30.67%
Fibrinogen	**Q25**	**Q50**	**Q75**	**Q90**	**Q95**
MLD index	0.0041***	0.0026***	0.0026***	0.0021***	0.0017***
		% contribution to IOp			
Age	66.80%	57.36%	52.76%	46.08%	42.19%
Gender	8.27%	6.73%	6.90%	7.65%	4.66%
Childhood language	0.63%	0.36%	0.94%	0.51%	0.23%
Parental occupation	8.23%	10.47%	10.20%	10.61%	12.01%
Parental education	4.49%	7.00%	5.55%	6.88%	9.41%
Individual's Education	11.60%	18.09%	23.65%	28.27%	31.50%
Allostatic load	**Q25**	**Q50**	**Q75**	**Q90**	**Q95**
MLD index	0.0028***	0.0019***	0.0011***	0.0010***	0.0009***
		% contribution to IOP			
Age	66.00%	58.76%	53.08%	47.61%	41.44%
Gender	12.50%	11.15%	7.84%	3.03%	5.53%
Childhood language	0.27%	0.24%	0.38%	0.28%	1.11%
Parental occupation	7.25%	6.97%	10.02%	15.85%	17.39%
Parental education	5.18%	6.12%	5.49%	8.24%	7.52%
Individual's Education	8.79%	16.77%	23.19%	25.00%	27.02%

Bootstrapped standard errors in parenthesis.
***P<0.01

These results highlight that the pronounced role of socioeconomic circumstances (parental occupation, parental education and own education) in shaping IOp at higher quantiles of the allostatic load distribution, would have been masked if the focus was solely on analysis at the mean (see subsection 4.1). Our "beyond the mean" analysis indicates that ill health is not simply a matter of gender and age inequalities, with our set of socioeconomic circumstances become much more relevant towards the right tails of biomarkers distribution, where clinical concerns are focused. These results are broadly consistent with previous "beyond the mean" studies that mainly focus on the role particular socio-economic factors, extending the existing research to the IOp concept, where inequalities in health are based on a broader set of circumstance. In their analyses, Carrieri and Jones (2017) and Costa-Font et al. (2009) found that the observed educational gradients become steeper towards the right tails of a selected set of blood-based biomarkers and the body mass index, respectively.

Decomposition by gender and age

Figure 5 presents gender differentials in the contribution of circumstances for allostatic load, based on our split sample analysis by gender implemented at various quantiles using the RIF method. The observed gender differentials at the lower quantiles of the distribution favour men; this echoes the analysis that uses estimates at the mean (Figure 4). However, we find that these gender differentials decrease in magnitude towards the higher quantiles of the distribution of allostatic load, along with the absolute magnitude of the variation in outcomes across circumstances. These results indicate that moving to higher quantiles of the allostatic load distribution, gender differences in the role of circumstances on shaping health are less pronounced.

Figure 6 presents the MLD indexes estimated separately for each age group using the fitted RIF values for each quantile (equation 7). We find that moving to the right tails of the allostatic load distribution, the inverted U-shaped pattern of IOp by age (observed for our "mean-based" IOp analysis; Figure 4) becomes less evident, with IOp gradually increasing with age. The cumulative advantage hypothesis (e.g., Kim and Durden, 2007), rather than the age-as-leveller, seems to exert the dominant role when the focus is on the right tail of the distribution, suggesting that adverse circumstances and health disadvantages accumulate as individuals age, suggesting a more pronounced role of circumstances in health as people age. The later results further highlight that our set of circumstances other than age play a greater role in shaping inequalities where elevated health risks are prominent; this result would be masked if the focus was solely on IOp analysis at the mean (Figure 4)

Figure 5. Gender differentials in IOp across the distribution of allostatic load.

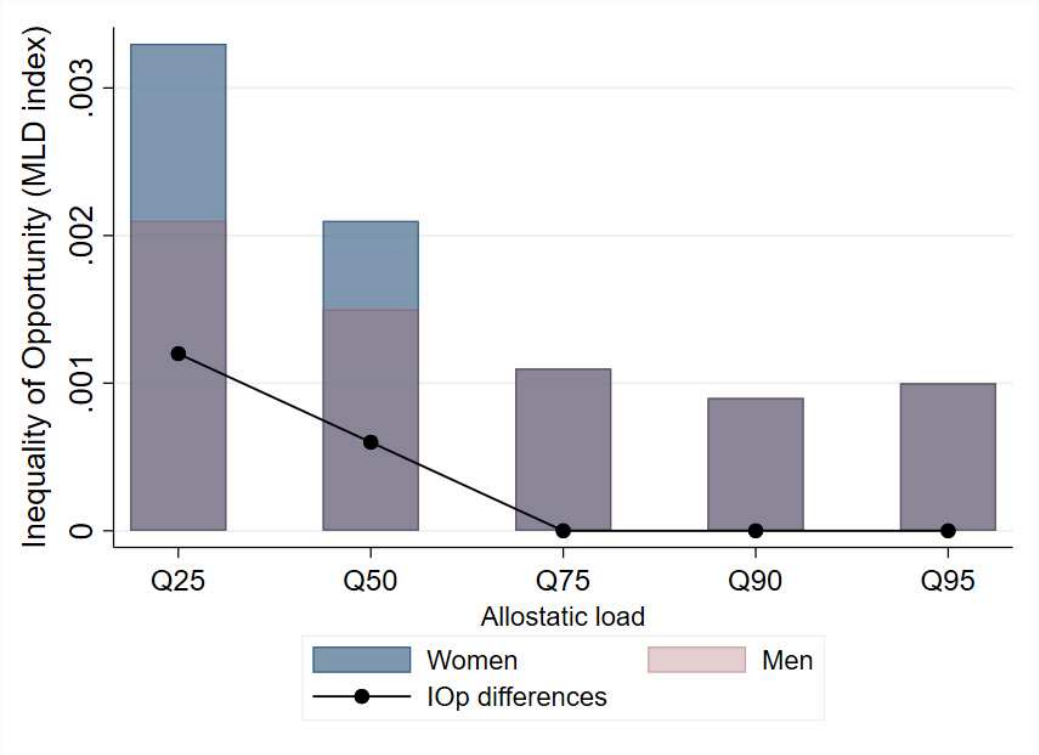
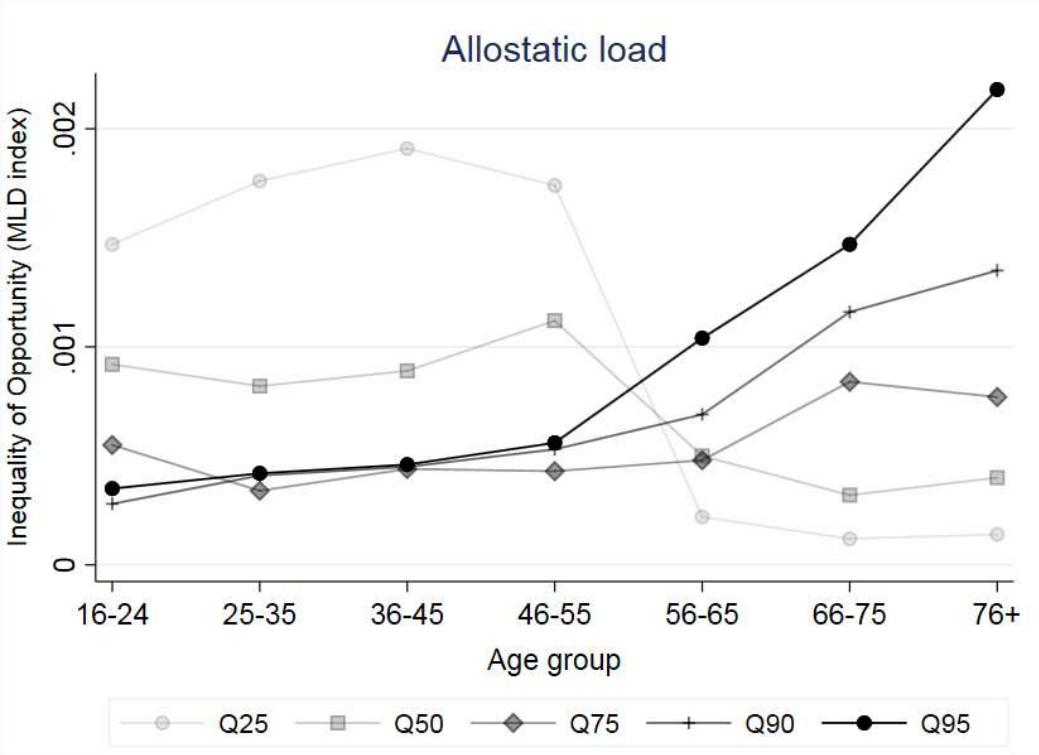


Figure 6. IOp by age groups at different quantiles of allostatic load.



4.3. Sensitivity analysis: the role of mortality

Mortality is obviously related to health and our analysis so far ignores the potential role of mortality bias by focusing on individuals who survived to UKHLS Wave 2. One may implicitly assume that the deceased would have achieved the average level of health associated with their circumstances and, thus, mortality bias may be disregarded in the ex-ante analysis of IOp in health. Specifically, in our analysis of IOp, we have adopted the ex ante approach - in the sense of comparing opportunity sets before effort has been exerted and the outcomes are realised. With the mean-based IOp all individuals are allocated the mean outcome given their circumstances (i.e. $E(y|C)$). This would also be true of any individuals who are missing due to death - as we are implicitly assigning them the mean outcome, consistent with the ex ante perspective. This makes the case that treating deaths as ignorable may be compatible with the ex ante mean-based IOp approach.

However, by focusing on individuals who are survivors at Wave 2, we may under-represent those individuals with lower socio-economic status, given that these people are more likely to die younger. This may or may not be a problem, depending on the scope of the analysis. If the focus of our paper is a description of the gradients in biomarkers across circumstances for the surviving population, there is no problem of survival bias and no additional analysis need to be taken. However, this is not the case if we are interested in accounting for the role of mortality in our ex ante IOp analysis.

As one way to indicate the likely size and nature of mortality bias in our analysis, we extend the analysis by including those individuals who were alive at Wave 1 and are deceased at Wave 2, expanding our analysis sample for allostatic load by 179 individuals. UKHLS is representative of the UK population at Wave 1 and we can identify those individuals who are deceased at Wave 2. For those individuals deceased at UKHLS Wave 2, all circumstance variables, except the childhood language variable, are collected at Wave 1; the fact that our set of circumstances are time invariant (date of birth has been used to calculate what the deceased's age would have been at Wave 2), this allows us to pool the deceased sample with our main analysis sample and conduct sensitivity analysis with respect to mortality bias. We use three alternative imputation methods to generate an imputed observation for the deceased individuals' allostatic load indicator:

a) Random imputation (drawn from all non-deceased): hot-deck imputation from the gender and age-specific sample of non-deceased individuals with valid allostatic load measures at UKHLS wave 2;
b) Random imputation (drawn from those deceased at subsequent waves): hot-deck imputation from the gender and age-specific sample of individuals who are alive at wave 2, but are deceased at subsequent UKHLS waves[17];

[17] Drawing from the sample of individuals who are deceased at subsequent waves allows us to impute allostatic load for the deceased based on a pool of valid allostatic load values of individuals who have excess risk of fragile health. The allostatic load distribution of those deceased at

c) Imputation of allostatic load values for the deceased individuals using a constant high allostatic load value (i.e., three s.d. above the mean).

Table 3 presents inequality results for allostatic load and the Shapley decomposition of circumstances to IOp when accounting for mortality using the three alternative imputation methods. To recap here our sensitivity analysis, as opposed to the base case results, does not account for foreign language as a circumstance; however, this should not prevent us from making meaningful comparisons regarding the robustness of the base case results given the negligible contribution of the foreign language variable. We find virtually no differences (in terms of the total inequality, absolute and relative IOp) between the results based on the two random imputation procedures (Table 3) and our base case results for allostatic load (Table 1). Imputation using a constant high allostatic load value results into slightly higher total inequalities and IOp compared to our base case results, reflecting the fact that adding a constant high allostatic load to the deceased individuals increases the dispersion of the allostatic load distribution[18]. However, in any case, the Shapley decomposition on the percentage contribution of circumstance variables to IOp are similar between all imputation methods (Table 3) and our base case results (Figure 3).

Table 3. IOp and Shapley decomposition accounting for mortality: Allostatic load

Inequality index	Random imputation (all non-deceased at wave 2)			Random imputation (deceased at subsequent waves)			Imputation: 3 s.d. above mean		
	Total inequality	Absolute IOp	% of total inequality†	Total inequality	Absolute IOp	% of total inequality†	Total inequality	Absolute IOp	% of total inequality†
MLD index	0.0073***	0.0016***	21.70	0.0075***	0.0017***	22.40	0.0087***	0.0023***	26.10
Circumstances	% contribution of circumstances			% contribution of circumstances			% contribution of circumstances		
Age	60.15%			59.41%			55.03%		
Gender	10.40%			10.61%			8.45%		
Parental occupation	8.31%			8.25%			7.47%		
Parental education	6.16%			6.46%			12.75%		
Individual's Education	15.00%			15.18%			16.30%		
Total	100%			100%			100%		
Sample size	6,345								

Notes: Unlike the corresponding base case results (Table 1 and Figure 3), English language at home during childhood is missing in our set of circumstance variables here because of data availability for the deceased individuals at UKHLS Wave 2.
†Relative IOp (column 3) stands for the (%) share of absolute IOp (column 2) to the total inequality (column 1) and reflect the contribution of observed circumstances to the total inequality in allostatic load.
***P<0.01

Table 4 presents the contribution of circumstances at different quantiles of the allostatic load distribution after accounting for mortality (using the alternative imputation procedures). Overall, Table 4 shows that the contribution of circumstances is similar to our base case results across the allostatic load quantiles (Table 2), with the role of socio-

subsequent waves is less peaked and is skewed to the right compared to the corresponding allostatic load distribution for all individuals observed at Wave 2.

[18] One may argue that there is no practical or theoretical limitation for using a higher allostatic load values to impute the deceased's missing allostatic load. Our evidence show that this will result in even higher inequalities, suggesting our current sensitivity analysis results as potential low bounds, which does not alter qualitatively the conclusions of our paper.

economic circumstances, as opposed to age and gender, increasing in magnitude as moving towards the higher quantiles of the allostatic load distribution.[19]

Table 4. Contribution of circumstances at different allostatic quantiles and Shapley decomposition accounting for mortality bias.

Panel A. Random imputation (all non-missing at wave 2)					
Allostatic load	**Q25**	**Q50**	**Q75**	**Q90**	**Q95**
MLD index	0.0029***	0.0020***	0.0011***	0.0008***	0.0007***
			% contribution to IOP		
Age	66.67%	58.37%	54.03%	47.87%	42.55%
Gender	11.89%	10.84%	8.27%	3.07%	5.58%
Parental occupation	7.49%	7.03%	9.21%	16.18%	17.54%
Parental education	5.27%	6.64%	6.01%	8.47%	7.53%
Individual's Education	8.68%	17.12%	22.48%	24.41%	26.80%
Panel B. Random imputation (deceased at subsequent waves)					
Allostatic load	**Q25**	**Q50**	**Q75**	**Q90**	**Q95**
MLD index	0.0029***	0.0020***	0.0012***	0.0010***	0.0009***
			% contribution to IOP		
Age	66.49%	57.39%	53.40%	47.21%	39.46%
Gender	11.97%	11.30%	8.41%	3.25%	5.59%
Parental occupation	7.60%	7.18%	9.31%	14.29%	14.69%
Parental education	5.32%	6.86%	6.81%	10.49%	13.50%
Individual's Education	8.63%	17.27%	22.08%	24.75%	26.76%
Panel C. Imputation: 3 s.d. above mean					
Allostatic load	**Q25**	**Q50**	**Q75**	**Q90**	**Q95**
MLD index	0.0030***	0.0023***	0.0019***	0.0038***	0.0053***
			% contribution to IOP		
Age	66.15%	56.93%	51.98%	45.18%	44.50%
Gender	11.61%	10.85%	5.32%	3.40%	3.04%
Parental occupation	7.70%	6.90%	7.98%	9.45%	11.79%
Parental education	5.52%	8.01%	13.06%	12.97%	13.13%
Individual's Education	9.01%	17.30%	21.65%	29.00%	27.55%

Notes: Unlike the corresponding base case results (Table 2), English language at home during childhood is missing in our set of circumstance variables here because of data availability for the deceased individuals at UKHLS Wave 2.
***P<0.01

Finally, to explore the robustness of our split sample IOp analysis by age across quantiles of the allostatic load distribution (Figure 6), we repeated our analysis using the three different imputation methods to account for the potential role of mortality (Figure A1, appendix). Overall, we find similar IOp patterns to the base case results, with IOp increasing with age as moving to higher quantiles of the allostatic load distribution (q90 and q95). It should be noted that imputation using a high fixed value results in a much steeper increase in IOp by age when focusing at the higher quantiles of the allostatic load distribution (Panel C, Figure A1, Appendix); this echoes the higher IOp that we observe in the case of imputation based on a high constant value, further reinforcing our results with IOp being even higher at older ages when mortality bias is accounted for.

[19] An exception here is the higher MLD index for the 90th and 95th allostatic load quantiles in the case of the imputation using a constant value (Table 4; Panel C) as opposed to the corresponding base case results (Table 2; allostatic load panel); this reflects the increased dispersion at the far right tails of the allostatic load distribution due to the use of a constant value to impute allostatic load values for the deceased. However, the corresponding results on the percentage contribution of circumstances (Shapley decomposition results for the 90th, 95th quantiles, Table 4, Panel C) reveal limited differences to our base case results.

5 Conclusions

Using UK nationally representative data we explore *ex ante* IOp in health and its underlying sources using objective nurse-collected and blood-based biomarkers that are intended to reflect the risk of preventable chronic conditions, such as obesity, diabetes and cardiovascular health. We find that IOp accounts for a non-trivial part of the total variation in our health measures. For example, in the case of allostatic load, our composite health indicator regarded as a measure of cumulative wear and tear in a number of physiological systems, we find that about 20% of the total inequality is attributed to the role of circumstances (IOp). Shapley-decomposition techniques show that apart from age and gender, parental education, parental occupational status and own educational attainment are important sources of IOp.

We propose an extension to the decomposition of *ex ante* IOp using the RIF method. This analysis allows us to decompose IOp and its sources across quantiles of the biomarker distribution. We find the presence of systematic contributions of circumstances for all biomarkers considered in our analysis across the whole distribution. In most cases, the contribution of age and sex declines relative to socioeconomic circumstances in the right tails, the part of the distribution where health risks are more pronounced. Focusing on allostatic load, the contribution of all socioeconomic circumstances increases from 21% at the lowest quantiles to more than 50% at the higher quantiles of the allostatic load distribution. Analysis focusing solely "at the mean" masks the important role of our set of socioeconomic circumstances (i.e., parental occupation, parental education and own education) on shaping health inequalities towards the right tails of biomarkers distribution, where clinical concerns are focused. Our "beyond the mean" analysis indicate that ill health is not simply a matter of gender and age inequalities, with our results confirming and extending existing research on the long-lasting role of childhood socioeconomic status (e.g., Case et al., 2005; Cohen et al., 2010) in the context of the IOp in health.

We also estimated IOp in our composite biomarker measure (allostatic load) by gender and across age groups. Splitting our sample by gender we find pronounced IOp in allostatic load (attributed to all measured circumstances apart from gender), with the gender differences in IOp in allostatic load being less relevant at the right tails of the allostatic load distribution. Our split sample analysis by age reinforces our finding that socio-economic circumstances play a greater role on shaping inequalities as people age towards the tails of the distribution where the health risks are much more prominent.

Given that IOp has been of interest to the health policy agenda (e.g., NHS England, 2017, NHS, 2019) and it has been placed at the top of the 'inequality of what' debate by relevant institutions (e.g. World Bank, 2005), more insight on the measurement of IOp is of particular importance. Our proposed methodology, over and above the specific application to health, may contribute and motivate "beyond the mean" analysis in the context of measuring and analysing IOp in several other dimensions of individuals' well-being, where focusing on the tails of the distribution is of particular importance.

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Appendix

Table A1. Circumstance variables used in the analysis.

	Mean
Age groups	
16-25	0.075
26-35	0.126
36-45	0.183
46-55	0.188
56-65	0.190
66-75	0.147
76+	0.091
Gender	
Male	0.432
Female	0.568
Language at home during childhood	
English at childhood	0.930
Other language at childhood	0.070
Mother occupation	
1(low-skilled)	0.144
2	0.258
3	0.084
4(high-skilled)	0.084
Missing	0.030
Not working (reference)	0.400
Father occupation	
1(low-skilled)	0.087
2	0.238
3	0.386
4(high-skilled)	0.146
Missing	0.089
Not working (reference)	0.054
Highest parental education	
No/some qualification (reference)	0.501
Post-school qualification/certificate	0.247
Degree	0.100
Missing	0.151
Educational attainment	
No/basic qualification (reference)	0.150
O-level	0.315
A-level/post-secondary	0.310
Degree	0.224

Table A2. Descriptive statistics for biomarkers for the full and restricted samples.

	Full sample			**Excluding missing parental data**		
	Mean	Std. err.	Sample size	Mean	Std. err.	Sample size
Waist-to-height ratio	0.561	0.001	14,068	0.560	0.001	11,119
Systolic blood pressure (mmhg)	126.17	0.155	11,865	126.00	0.161	9,450
Cholesterol ratio	3.741	0.014	9,005	3.732	0.016	7,228
HbA1c (mmol/mol)	37.240	0.082	8,468	37.111	0.090	6,803
CRP (mg/L)	2.092	0.022	8,311	2.044	0.024	6,672
Fibrinogen (g/L)	2.789	0.006	8,964	2.782	0.007	7,199
Allostatic load	27.475	0.043	6,166	27.380	0.047	4,976

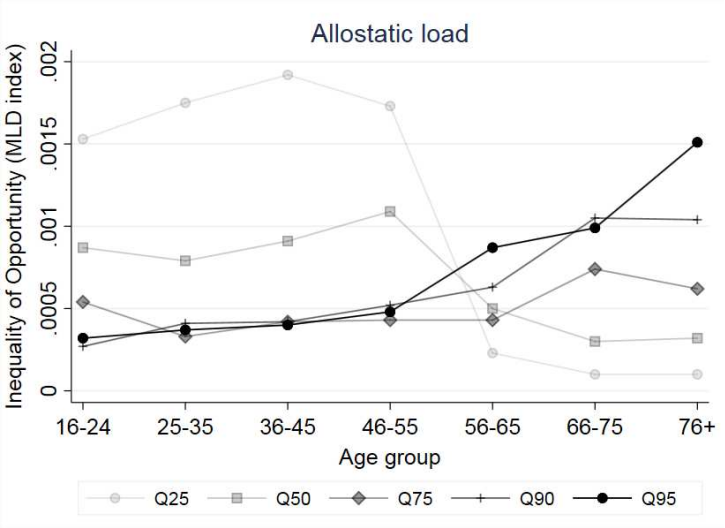
Table A3. IOp measures and Shapley decomposition using different inequality measures.

	Measures of IOp		Shapley decomposition: % contribution of circumstances to IOp						
	Absolute IOp	Relative IOp	Age	Gender	Childhood language	Parental occupation	Parental education	Individual's education	Total contribution
MLD index‡									
Waist to height ratio	0.0020***	0.169***	64.68%	5.48%	0.36%	8.01%	6.83%	14.63%	100%
Systolic blood pressure	0.0017***	0.198***	61.96%	21.94%	2.56%	4.34%	2.82%	6.38%	100%
Cholesterol ratio	0.0064***	0.110***	29.46%	63.40%	0.03%	2.27%	1.20%	3.64%	100%
HbA1c	0.0030***	0.195***	70.73%	2.95%	1.84%	7.09%	4.54%	12.85%	100%
C-reactive protein	0.0161***	0.039***	23.41%	10.15%	0.42%	20.24%	13.26%	32.52%	100%
Fibrinogen	0.0023***	0.107***	58.61%	8.18%	0.48%	9.50%	4.98%	18.26%	100%
Allostatic load	0.0016***	0.218***	59.68%	10.57%	0.24%	8.34%	5.90%	15.28%	100%
Variance share†									
Waist to height ratio	-	0.161***	63.80%	5.60%	0.40%	8.10%	6.80%	15.40%	100%
Systolic blood pressure	-	0.191***	61.90%	22.00%	2.50%	4.30%	2.80%	6.50%	100%
Cholesterol ratio	-	0.095***	28.51%	64.44%	0.03%	2.23%	1.18%	3.61%	100%
HbA1c	-	0.146***	70.20%	3.00%	1.80%	7.00%	4.40%	13.60%	100%
C-reactive protein	-	0.033***	24.10%	10.00%	0.40%	19.10%	11.0%	35.40%	100%
Fibrinogen	-	0.105***	58.30%	8.20%	0.50%	9.30%	4.80%	18.90%	100%
Allostatic load	-	0.210***	59.12%	10.70%	0.24%	8.28%	5.83%	15.83%	100%

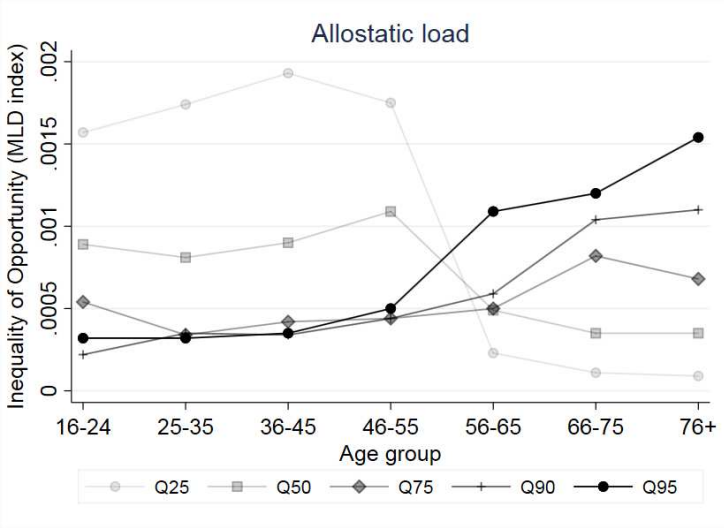
Notes:
‡The results using the MLD index are the same to those presented in Table 1 and Figure 3 (main text). As in Table 1, relative IOp stands for the proportion of the total inequality in biomarkers that is attributed to IOp but, unlike Table 1, here is not expressed in percentage terms.
†The variance share is by definition a relative measure of IOp, capturing the share of the total variation in each biomarker that is attributed to the observed circumstances.
***P<0.01

Figure A1. IOp by age groups at different quantiles of allostatic load distribution accounting for mortality bias

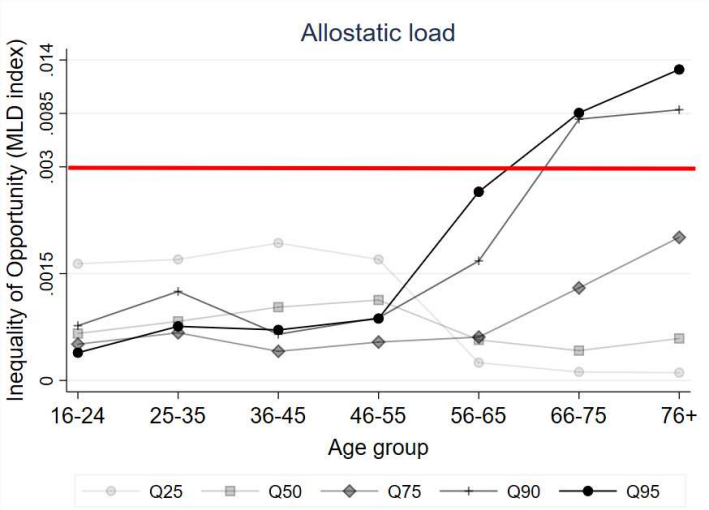
Panel A: Random imputation (all non-missing at wave 2)



Panel B: Random imputation (deceased at subsequent waves)



Panel C: Imputation: 3 s.d. above mean



Notes; Red line indicates a scale break (y axis) for the case of the last graph above.