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Do socioeconomic health gradients persist over time and beyond income? A distributional analysis using UK biomarker data

Kompal Sinha[†], Apostolos Davillas[‡], Andrew M. Jones[§], Anurag Sharma[¶]

Abstract

This paper analyses the relationship between health and socioeconomic disadvantage by adopting a dynamic approach accounting for spatial and temporal changes across ten domains including social isolation, environment, financial hardship and security. As a first step we develop a measure of overall multidimensional deprivation and undertake a decomposition analysis to explore the role of breadth and duration of deprivation on shaping the deprivation gradient in health. Subsequently, we employ unconditional quantile regression to conduct a distributional analysis of the gradient to understand how the gradient evolves for people with vulnerability in health. In contrast to the majority of existing studies, we capture health status using a range of nurse measured biomarkers, rather than self reported health measures, taken from the UKHLS and BHPS databases. The first main finding is that the socioeconomic gradient in most of our health measures is not solely attributed to income as it accounts for only 3.8% of total deprivation and thus it is important to account for other domains through a multidimensional deprivation measure in health gradient analysis. Our second finding is the existence of a systematic deprivation gradient for BMI, waist circumference, heart

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rate, C-reactive protein and HbA1c where evolution over time is an important factor particularly for individuals with greater burden of illness lying at the right tail of the biomarker distribution. Thus cost effective health policy would need to adopt targeted interventions prioritising people experiencing persistent deprivation in dimensions such as housing conditions and social isolation.

Keywords: Biomarkers, Multidimensional deprivation, Shapley decomposition, Unconditional quantile regression.

JEL classifications: C1, D63, I12, I14, I31, I32.

1 Introduction

The association between socioeconomic status and health has been long established in the literature (Frijters et al., 2015; Jürges et al., 2013; Loucks et al., 2009; Jones and Wildman, 2008; Deaton and Paxson, 1998). Despite the broad understanding that disadvantage reduces the wellbeing of individuals in the society, deprivation remains a leading cause of morbidity and mortality in most countries (Dickerson and Popli, 2016) resulting in growing economic burden on the health system along with rising inequalities. This makes it imperative for effective health policies to adopt a holistic approach to overall disadvantage that provides policymakers with insights on the structure of disadvantage, enabling them to not only identify specific dimensions that need targeting, but also to provide insights on whether to prioritise people experiencing disadvantage in multiple domains, people experiencing disadvantage for longer periods of time, or people experiencing consecutive periods of persistent disadvantage, issues relevant to reducing health inequalities. To this avail, this paper proposes a holistic measure of multidimensional deprivation that dynamically captures the breadth, duration and persistence aspects of deprivation over a 10 year period, and subsequently analyse the influence of these components on health outcomes. We define breadth of deprivation to account for the disadvantage experienced by an individual in multiple domains, capturing how an individual's health is influenced by deprivation in multiple domains. Duration of deprivation is defined as the number of years an individual is deprived in each domain, capturing how length of deprivation influences health. While this component allows us to account for the dynamic aspect of deprivation, it does not account for the fact that being deprived has negative effects that accumulate over time, and experiencing multi-period spells of deprivation is much harder to endure than multiple single-period spells interrupted by one (or more) period(s) out of deprivation (Bossert et al., 2019). Thus, we define persistence of deprivation as uninterrupted spells of disadvantage experienced by an individual over a given period, capturing the fact that experiencing persistent deprivation can have a larger influence on health outcomes. Here the distinction between duration and persistence being that persistence refers to the number of consecutive spells of deprivation experienced by an individual over a period, duration refers to the years

¹For example, experiencing three consecutive years of deprivation could be harder to endure than three single year deprivations spaced by periods of no deprivation.

of disadvantage experienced across the time period (Bossert et al., 2019). Moreover, going beyond the standard practice of using just income as a proxy for socioeconomic status, our measure of multidimensional deprivation uses ten additional domains - education, economic activity, housing conditions, affordability of basic consumer durables, car ownership, affordable lifestyle, financial hardships, social engagement, environment and security to capture social disadvantage. This allows us to provide robust policy relevant evidence on the relationship between health and dynamic multidimensional disadvantage.

The context of this study is the UK. This is particularly important from the policy perspective since the UK suffers from one of the highest level of deprivation among developed countries (Marmot, 2020). Even though UK was the first country to adopt a systematic policy to reduce socioeconomic inequalities in health in 1997 (Mackenbach, 2011), absolute inequalities in the country remain a matter of policy concern. The National Health Service (NHS) is continually seeking new evidence on how socioeconomic conditions and disadvantage shape disparities in health and well-being. The Office of National Statistics reported 7.7 million people in the UK lived in persistent poverty in 2017, and the resulting disparities in health are a concern for policy makers (Caul, 2020). Notwithstanding this, policy has refrained from accounting for the dynamics of deprivation and has adopted a static approach to understanding the influence of multiple deprivation on health outcomes. The Marmot review of health equity in England (Marmot, 2020) only briefly mentions the role of dynamic deprivation on health and wellbeing in the UK. With the Office of National Statistics recording 128.3 deaths per 100,000 population in the most deprived areas and 57.5 deaths per 100,000 population in less deprived areas during the coronovirus pandemic of 2019-20, the association between multidimensional deprivation and health has been reinforced in recent times (Caul, 2020), making it particularly relevant to understand the dynamics of association between the two for future health policy.

This paper presents a new approach to analyse the association between deprivation in multiple domains and health outcomes and makes several contributions to the literature. First, we adopt a broad concept of multidimensional deprivation, improving the existing literature which has largely used either unidimensional² socioeconomic measures (e.g., Braveman et al.,

²Such as income (Carrieri and Jones, 2017, Bilger et al., 2017, Jolliffe, 2011) education (Cohen et al., 2013), and relative social status (Bilger and Carrieri, 2013).

2005, Gruenewald et al., 2009, Johnston et al., 2009) or aggregate cumulative measures of SES (e.g., Kim and Durden, 2007, Loucks et al., 2009). In context of the association of socioeconomic status (SES) with health, these measures does not establish how these different dimensions of deprivation may be distributed across individuals and over time or how they are related to health outcomes (Blázquez et al., 2014). Consequently, the inequalities in health (Allanson and Petrie, 2013; Allanson et al., 2010) vary with the measures of socioeconomic status and are difficult to recoincile (Lindelow, 2006). There is thus a need for "... more careful research on how different dimensions of SES are related, and on the pathways by which the respective dimensions impact on health related variables" (Costa-Font and Hernández-Quevedo, 2012).

For the measurement and understanding of the health inequalities ensuing from disadvantage it is important to disentangling the contribution of each of the components of disadvantage (Costa-Font and Hernández-Quevedo, 2012). A challenge in doing so is that the literature measuring multidimensional disadvantage (Alkire and Foster, 2011) remains mostly static (or cross-sectional) (Foster, 2009). The few papers incorporating dynamic disadvantage (Gradín et al., 2018, Dutta et al., 2013, Bossert and D'Ambrosio, 2019, D'Ambrosio, 2016) either only accounts for breadth (i.e., number of domains) (Foster, 2009) or accounts for duration (i.e., number of years) (D'Ambrosio et al., 2012) of deprivation, albeit mostly in unidimensional context (Dutta et al., 2013). Studies that account for both breadth and duration of deprivation (Alkire et al., 2017) do not account for persistence (i.e., the approach proposed by Nicholas et al. (2019)), and the ones that do account for persistence of deprivation do not distinguish between breadth and duration aspects of deprivation (i.e., the approach proposed by Nicholas and Ray (2012)). Thus, there has not been any study that accounts for persistence while simultaneously differentiating between breadth and duration of deprivation in multiple domains. Due to these gaps in literature the role of persistence of dynamic socioeconomic disadvantage across multiple domains in shaping health outcomes remains to be studied. To fill this gap, we develop a measure based on two recent developments in the development economics literature where we use the Nicholas et. al. (2019) framework as a benchmark and incorporate uninterrupted spells of deprivation in multiple domains using the approach proposed by Nicholas and Ray (2012) and Gradin et al (2012), to propose a persistence augmented measure of multidimensional deprivation that simultaneously distinguishes between the breadth and dynamic components of disadvantage. Specifically, we combine the approach of Nicholas and Ray (2012) that allows persistence (but did not differentiate between the dimensionality and duration aspects of deprivation) and Nicholas, Ray and Sinha (2019) that allows differentiating between the dimensionality and duration aspects of deprivation (but did not allow persistence). Our holistic measure of multidimensional deprivation allows us to identify those who experience deprivation across a wide variety of dimensions (in a given period), and those who experience deprivation for the most periods (in any given dimension), along with identifying those experiencing chronic deprivation (i.e., uninterrupted spells of deprivation). Adopting this framework is particularly useful since it is sensitive to the length of deprivation allowing us to account not only for whether the same individuals are getting more deprived over time, but also whether they are doing so in the same dimensions. Extending the framework in this way allows us to, not only study the differentiated role of components of deprivation on health outcomes, but also distinguish how experiencing uninterrupted spells of deprivation (persistence) alongside breadth and duration of deprivation might influence health outcomes. Capitalising on the feature of decomposability of this dynamic multidimensional deprivation measure, we are able to compute the contribution of breadth and dynamic component to overall deprivation, and undertake a distributional analysis of how these components influence health outcomes. An issue of particular interest when the objective is to compare across groups that have experienced deprivation over a period of time with those who have experienced chronic deprivation due to uninterrupted spells.

Second, unlike most of the previous literature on the SES – health gradient, we employ a set of nurse-collected and blood-based biomarkers most relevant to non-communicable disease risk: adiposity measures, blood pressure, resting heart rate, inflammatory biomarkers, blood glucose and cholesterol ratio. While subjective self reported measures of health have been criticised as partial measures of health plagued by reporting bias and individual subjectivity (Jürges, 2007, 2013; Bago d'Uva et al., 2008; Dowd and Zajacova, 2010; Giordano and Lindstrom, 2010), there are several advantages of using biomarkers. They are objective measures of health compared to conventional self-reported health measures; they provide direct information on pre-disease mechanisms that are below the individual's perception or clinical diagnosis thresholds and, thus, allow for a better understanding of the deprivation-

health gradient before diseases become evident; they act as a "secondary" physiological responses to stress (Acabchuk et al., 2017; Davillas et al., 2017), and are closer to the process through which social and economic stressors get "under the skin" (Glei et al., 2013). The advantages of biomarkers are being acknowledged by a growing literature in economics (Davillas and Pudney, 2020; Böckerman et al., 2017; Carrieri and Jones, 2017; Jürges et al., 2013).

Third, we account for the potential variation in the relationship between health and SES across the distribution of nurse-collected and blood-based biomarkers. Our analysis estimates the deprivation gradient at the mean and across quantiles of the distribution of biomarkers using unconditional quantile regression (UQR) techniques to evaluate the heterogeneity of the deprivation gradients. While existing studies typically explore the effect of SES on the conditional mean of the health outcome of interest (for instance, Jürges et al., 2013), analyses based solely on the mean can mask important information in other parts of the distribution (Carrieri and Jones, 2017). This is particularly important for our analysis given the greater burden of illness and possibly higher costs for the healthcare system at the extreme tails of the biomarkers distribution.

Fourth, an empirical concern in the analysis of the determinants of health is the issue of causality. These concerns have been addressed in the literature by employing longitudinal datasets that track individuals across time, making it is possible to observe changes in SES that allow identification of causal link between SES and health (Pickett and Wilkinson, 2015; Frijters et al., 2005; Adams et al., 2003). We are using the longitudinal dataset Understanding Society, the UK Household Longitudinal Survey (UKHLS). An advantage of this dataset is that we use longitudinal information on SES indicators collected in the years before the time of measuring health outcome which allows us to partially alleviate concerns about contemporaneous effect and causality issues of health on SES. Here, similar to Hauck and Rice (2004), we assume SES and health outcomes are not simultaneously determined in our estimation because the measures of deprivation in multiple domains we use are from past-years, while our health variables measure recent health. While yearly SES may impact health outcomes, it would be implausible for recent biomarkers to influence SES in past-years (Hauck and Rice, 2004).

The rest of the paper is organised as follows. The next section, discusses the methods

and framework for estimating the measurement of dynamic multidimensional and empirical model. Section 3 outlines the UKHLS and BHPS datasets used for our analysis. Section 4 presents and discusses the estimation results. Section 5 concludes with discussion on policy implications.

2 Methods

2.1 Dynamic multidimensional deprivation measure

$$d_{ijt}^{\alpha} = \begin{cases} (1 - \frac{x_{ijt}}{F_j})^{\alpha} & \text{if } x_{ijt} < F_j \forall j, t \\ 0 & \text{otherwise} \end{cases}$$
 (1)

The sensitivity parameter $\alpha \geq 0$ allows for the weight given to a particular indicator to increase with depth of deprivation in that dimension.⁴ It is common practice in the literature to set binary thresholds to determine an individual's deprivation, i.e., restrict $\alpha = 0$ such that $d_{ijt}^{\alpha} \in \{0,1\} \forall j,t$. Specifically, $d_{ijt}^{0} = 1$ when individual is deprived in dimension j at time t, and $d_{ijt}^{0} = 0$ otherwise. Accordingly, each individual i has an overall deprivation

³This was first suggested by Atkinson (2003) and used by Alkire and Foster (2011).

⁴This is similar to the poverty measure proposed by Foster et al. (1984).

profile A_i :

$$\mathbf{A_i} = \begin{pmatrix} d_{i11} & \cdots & d_{i1t} \\ d_{i2,1} & \cdots & d_{i2t} \\ \vdots & \ddots & \ddots \\ d_{iJ1} & \cdots & d_{iJT} \end{pmatrix}$$

where $d_{ijt} \in \{0,1\} \forall j \in \{1,...J\}, t \in \{1,2,...T\}$. The individual deprivation score μ_i is a function $f: D_i \to R$ where \mathbf{R} is the set of real numbers.⁵ The population achievement profile is a vector $\rho = (\mu_1, ..., \mu_N)$ of individual scores in non-decreasing order. Aggregating the individual deprivation scores, the multidimensional deprivation index Ω is then a function $g: \rho \to \mathbf{R}$ and can be represented by, following Nicholas et al. (2019):

$$\Omega = \frac{1}{N} \sum_{i=1}^{N} \left(\delta \frac{1}{T} \sum_{t=1}^{T} \left(\frac{1}{J} \sum_{j=1}^{J} d_{ijt} \right)^{\beta} + (1 - \delta) \frac{1}{J} \sum_{j=1}^{J} \left(\frac{1}{T} \sum_{t=1}^{T} d_{ijt} \right)^{\beta} \right) \times c_{i}$$
 (2)

where $0 \le \delta \le 1$ and $\beta > 0$. This deprivation measure sets the parameter $\beta > 0$ to allow for the score to be sensitive to the distribution of disadvantage across individuals. While equation 2 incorporates duration of deprivation, it does not explicitly account for persistence. We generalise equation 2 to incorporate the effect of persistence using Gradín et al. (2012)'s unidimensional generalisation of persistence weights. We posit each observation of d_{ijt} belongs to a deprivation spell, s_{ijt} , defined as length of uninterrupted deprivation spells in a particular dimension to give us the persistence augmented measure:

$$\Omega = \frac{1}{N} \sum_{i=1}^{N} \left(\delta \frac{1}{T} \sum_{t=1}^{T} \left(\frac{1}{J} \sum_{j=1}^{J} d_{ijt} s_{ijt} \right)^{\beta} + (1 - \delta) \frac{1}{J} \sum_{j=1}^{J} \left(\frac{1}{T} \sum_{t=1}^{T} d_{ijt} s_{ijt} \right)^{\beta} \right) \times c_{i}$$
 (3)

where $s_{ijt} \in [0,1]$ is a non-negative increasing function of v_{ijt} which is the length of the deprivation spell associated with a particular d_{ijt} . Defining a functional form for s allows explicitly incorporating a trade-off between an additional indicator of deprivation or deprivation for an additional consecutive period. Following Gradín et al. (2012) we define $s_{ijt} = (v_{ijt}/T)^{\theta}$ where T is the total period considered. s_{ijt} takes the value of 1 when the disadvantage (d_{ijt}) is a part of a $v_{ijt} = T$ year spell, i.e., individual i is deprived in dimension

⁵Since μ_i takes the (TXK) matrix D_i as its input, in principle there can be a maximum of $2^{(TXK)}$ different types of deprivation scores, one for each possible permutation of the deprivation profile.

j in all years. $\theta \geq 0$ is a parameter that determines the sensitivity of the index to the length of individual deprivation spells. This allows the multidimensional index to satisfy the property of durational persistence monotonicity (i.e., the requirement that for any individual i, j and t, Ω increases as s_{ijt} increases). The indicator function c_i takes the form:

$$c_i = \begin{cases} 1 & \text{if } \sum_{t=1}^{T} \sum_{j=1}^{J} d_{ijt}^0 \ge z \\ 0 & otherwise \end{cases}$$

where $(J \times T) \geq z \geq 1$. Following the dual cut-off measure of Alkire and Foster (2011) class of poverty measures c_i is dependent on z. The value of z = 1 would result in the equivalent of union method⁶ of identification and at z = (JXT) would result in the intersection method⁷. However, in the present case deprivation is counted both across dimension and time which allows the possibility of identifying the poor using an additional cut-off.

This measure of dynamic multidimensional disadvantage satisfies the properties of subgroup decomposability, normalisation, dimension monotonicity, durational monotonicity, dimensional transfer principal and durational transfer principal.⁸ The proposed measure also meets the requirement of dimensional convexity and durational convexity⁹, giving a convex combination of deprivation due to dimensions and persistence - the dimension measure and the dynamic measure - shown in the right hand side of Equation 3. The first component (dimension measure, $\Omega^{dimension}$) measures the prevalence of overall deprivation, and is calculated for each year separately and then averaged over all years:

$$\Omega^{dimension} = \frac{1}{N} \sum_{i=1}^{N} \left(\delta \frac{1}{T} \sum_{t=1}^{T} \left(\frac{1}{J} \sum_{i=1}^{J} d_{ijt} s_{ijt} \right)^{\beta} \right) \times c_i$$
 (4)

The second component (dynamic measure, $\Omega^{dynamic}$) forms the duration measure of depri-

⁶A person is considered poor if deprived in at least one dimension (Bourguignon and Chakravarty, 2003).

⁷A person is considered poor if deprived in all dimensions (Ray and Sinha, 2015)

⁸In the interest of space, we have provided a brief description of the measure. For details and proofs of the properties the reader is referred to Gradin (2012) and Nicholas, Ray and Sinha (2019).

⁹Dimensional (durational) convexity suggests the effect of an increase in any of an individual's deprivation on the aggregate deprivation score is a strictly positive function of the deprivations in other dimensions (periods) that share the same period (dimension) as the deprivation in question.

vation:

$$\Omega^{dynamic} = \frac{1}{N} \sum_{i=1}^{N} \left((1 - \delta) \frac{1}{J} \sum_{j=1}^{J} \left(\frac{1}{T} \sum_{t=1}^{T} d_{ijt} s_{ijt} \right)^{\beta} \right) \times c_i$$
 (5)

and is calculated for each dimension and then averaged over dimensions. Here both dimensions and time each counts equally. The two parameters β and δ account for dimensional convexity (i.e., giving more weight to individuals experiencing deprivation across multiple dimensions within the same period) and duration convexity (i.e., individuals experiencing deprivation across multiple years within the same dimension) respectively. The parameter θ accounts for persistence of deprivation which takes the value $\theta = 0$ when we do not account for persistence giving us two deprivation components: $\Omega^{dimension}$ and $\Omega^{duration}$. When we account for persistence the parameter takes the value $\theta = 1$ giving us the components: $\Omega^{dimension}$ and $\Omega^{persistence}$. We assume equal weight for dimensions and duration of deprivation (i.e., $\delta = 0.5$) and each individual's deprivation profile is squared to allow for sensitivity to the across-individual distribution (i.e., $\beta = 2$) following Nicholas et al. (2019). We consider the persistence parameter θ for two values for no-persistence (i.e., $\theta = 0$) and persistence (i.e., $\theta = 1$) where the former accounts for duration of deprivation and the latter additionally accounts for persistence of deprivation. An important aspect of our deprivation measure (equation 3), is that the contribution of each dimension to overall deprivation is a non-linear function of other dimensions, which does not allow direct decomposition of our deprivation measure into dimensions. However, using the Shapley decomposition method proposed by Shorrocks (2013) we decompose the contribution of each dimension to overall deprivation, and then decompose the dimensional contribution into: a) a part of deprivation due to distribution of breadth within individuals; and b) a part of deprivation due to the distribution of length of deprivation across time for an individual (Nicholas et al., 2019). Accordingly, equation 3 can be rearranged to yield three additive components as:

$$\Omega = \bar{\Omega} + \delta(\Omega^{dimension} - \bar{\Omega}) + (1 - \delta)(\Omega^{dynamic} - \bar{\Omega}) = \Omega_A + \Omega_B + \Omega_C$$
 (6)

where $\bar{\Omega} = \frac{1}{N} \sum_{i=1}^{N} (\frac{\sum_{t=1}^{T} \sum_{j=1}^{J} d_{ijt}}{j*T})^{\beta}$. The first component, Ω_A , is the sum of count of deprivations averaged over individuals and is the distribution insensitive component, i.e., it is not influenced by how deprivation is distributed across dimensions and across time. This

indicates that a change in the pattern of deprivations for any individual has no impact on this component. The second component, Ω_B , measures the distribution of breadth component across dimensions or prevalence of deprivation component. This takes the value of zero if the breadth of deprivation is the same for each year for all individuals. The third component, Ω_C , is distribution of the length component across dimensions or persistence of deprivation. This component will take the value of zero if the length of deprivation is the same across each dimension for all individuals. The decomposition of overall deprivation into the contribution from each dimensions is the sum $\Omega_A + \Omega_B$ (i.e., $\Omega^{dimension}$), and the dynamic contribution is the sum $\Omega_A + \Omega_C$ (i.e., $\Omega^{duration}$). It should be explicitly noted that Ω_A , Ω_B and Ω_C , facilitate comparability across the $\Omega^{duration}$, $\Omega^{dimension}$ for the scenario with no persistence, and $\Omega^{persistence}$, $\Omega^{dimension}$ for the case with persistence (Nicholas et al., 2019).

2.2 OLS and Quantile Regressions

The nurse-collected and blood-based biomarkers are initially modelled using linear regression model estimated by OLS. Distributional regression techniques are also applied to consider the entire distribution of each biomarker (H_i) . We employ unconditional quantile regression (UQR) models, which allow us to estimate unconditional quantile partial effects (Firpo et al., 2009). UQR models are based on the recentered influence function (RIF) that can be estimated by computing sample quantiles of the health measure (q_{τ}) and then estimating the density of the distribution of health measures at the quantiles using kernel density methods. That is,

$$RIF(H_i; q_\tau) = q_\tau + \frac{\tau - 1[H_i \le q_\tau]}{f_H(q_\tau)}$$

where q_{τ} is the observed sample quantile, $1[H_i \leq q]$ is an indicator function taking the value of one if the observed value of health measure of interest is less than or equal to the observed quantile q_{τ} and zero otherwise; $f_H(q_{\tau})$ is the estimated kernel density of the particular health measures at the τ^{th} quantile. The RIF is then regressed on our set of covariates using OLS. Bootstrap methods with 500 replications is used to obtain unbiased estimates of the variance covariance matrix of the parameter estimates (Buchinsky, 1998).

2.3 Health model specification

We specify our health model separately for each of the nine biomarkers, where each biomarker is a function of the measure of multidimensional deprivation. The sequence of model specifications is as follows. We start by exploring how multidimensional material deprivation is associated with health beyond income by specifying health as a function of material deprivation¹⁰ along with income and other covariates. Each health outcome i.e., biomarker (H_i) is regressed on our long term income and dynamic multidimensional material deprivation measure along with other covariates. This is done at the mean using OLS and across quantiles (with 0.05 increments) using RIF regressions defined as follows:

$$RIF(H_i; q_\tau) = \beta_{0\tau} + \beta_{1\tau} ln(Inc_{iLT}) + \beta_{2\tau} \Omega_i + \beta'_{3\tau} \mathbf{x_i} + \epsilon_{i\tau}$$
 (7)

where Inc_{LT} is the long term income (calculated as an average income over BHPS wave 9 to wave 18) and $\beta_{1\tau}$ is the coefficient for income; Ω , is our dynamic multidimensional measure and $\beta_{2\tau}$ is the corresponding coefficient at τ^{th} quantile. The vector \mathbf{x} is the set of covariates, $\beta'_{3\tau}$ are the relevant coefficients and $\epsilon_{i\tau}$ is the error term at each quantile.

Next, we make use of the unique feature of our measure of multidimensional deprivation to decompose it into a component due to breadth and a component due to dynamic deprivation. We exploit this feature to re-estimate equation 7 incorporating components to investigate how the gradient changes with breadth of deprivation relative to length of deprivation. For this analysis we use our holistic multidimensional measures which includes income. The specification is as follows:

$$RIF(H_i; q_\tau) = \theta_{0\tau} + \theta_{2\tau}\Omega_{iA} + \theta_{3\tau}\Omega_{iB} + \theta_{4\tau}\Omega_{iC} + \theta'_{5\tau}x_i + \epsilon_{i\tau}$$
(8)

where Ω_{iA} , Ω_{iB} and Ω_{iC} are the three components of overall multidimensional deprivation (Ω_i) as discussed in equation 6.11

A simple way to explore the relative contribution of the breadth component $(\Omega^{dimension})$ and the dynamic component $(\Omega^{dynamic})$ to each biomarker is to estimate, for each quantile

¹⁰This measure of material deprivation is based on 10 domains.

¹¹It should be explicitly noted that Equation 5 assumes that the effect of each of the three deprivation sub-components can be effectively separated by estimating $\theta_{2\tau}$, $\theta_{3\tau}$ and $\theta_{4\tau}$.

 τ , a counterfactual as follows:

$$\widetilde{H}_{i}^{t}(q_{\tau}) = \hat{\theta}_{0\tau} + \hat{\theta}_{2\tau}\Omega_{iA} + \hat{\theta}_{3\tau}\Omega_{iB} + \hat{\theta}_{4\tau}\Omega_{iC} + \hat{\theta}_{5\tau}'x_{i}$$

$$\tag{9}$$

where $\hat{\theta}$ coefficients represent the estimated coefficients in Equation 8. As the RIF equations are additive and linear, fitted values for each biomarker can be estimated using the RIF method at each quantile (\tilde{H}_i^t) , while the contribution of the $\Omega^{dimension}$ and $\Omega^{dynamic}$ is calculated as $\hat{\theta}_{2\tau}\Omega_{iA} + \hat{\theta}_{3\tau}\Omega_{iB}$ and $\hat{\theta}_{2\tau}\Omega_{iA} + \hat{\theta}_{4\tau}\Omega_{iC}$, respectively. This dynamic component for the case when $\theta = 0$ is $\Omega^{duration}$ and is $\Omega^{persistence}$ when $\theta = 1$. The ratio of each of the latter to the total prediction (\tilde{H}_i^t) shows the percentage contribution to each of the components to the fitted biomarker values at quantile τ .

3 Data

We use the British Household Panel Survey (BHPS) sub-sample of the UK Household Longitudinal Survey (UKHLS), also known as Understanding Society. The BHPS is a widely used representative longitudinal UK study that covered the period between 1991 and 2009 (18 waves) up to the time it was absorbed into the UKHLS. A distinguishing feature of this database is that for the BHPS respondents followed up in the UKHLS, a set of nurse-measured health indicators and non-fasted blood samples were collected after the UKHLS wave 3 main survey (Benzeval et al., 2014). Data collection for Wave 2 and Wave 3 was conducted over 2010-2012. These objective measures of health along with the detailed longitudinal socio-demographic information from BHPS makes this an ideal database for our study. We use the longitudinal data on socio-economic indicators to construct our dynamic multidimensional measure of deprivation based on the domains listed in Table A2. This measure of deprivation along with contemporaneous information on individuals demographic characteristics from UKHLS wave 3 main survey are used as explanatory variables to model health outcomes. We restrict our analysis to individuals with non-missing information across all these dimensions to construct a balanced panel of BHPS waves over 1999-2008

¹²These variables were self-reported by the survey respondent and are prone to potential reporting bias however the BHPS is a prospective survey wherein individuals are surveyed every year. These surveys are has been reported to be less prone to reporting bias (Longhi and Nandi, 2014).

(wave 9 to wave 18). The final samples are created by merging these balanced panel datasets with the UKHLS wave 3 (2010) nurse visits and main survey data for the BHPS sample. The resulting long term sample has 57,070 observations across the 10 waves (waves 9 -18, 1999-2008) of BHPS (5,707 unique individuals).

3.1 Nurse-collected health measures

We use measures of adiposity, heart rate (HR) and blood pressure in our analysis. To capture central adiposity we use waist circumference (WC) as well as the Body Mass Index (BMI). BMI is calculated as body weight (in kilograms) over the square of height (in metres). Three repeated measurements of heart rate (HR), systolic and diastolic blood pressure (SBP, DBP) were taken at intervals of one minute. We skip the first reading, believed to impose upward biases, and computed HR, SBP and DBP as the average of the second and third readings. Values of SBP (DBP) above 140 (90) mmHg are considered as hypertensive.

3.2 Blood-based biomarkers

We use measures of inflammation, blood glucose and "fat in the blood" biomarkers. Two biomarkers of inflammation are examined: CRP and fibrinogen. CRP is an acute phase protein that reflects chronic inflammation. CRP values over 5 mg/L are considered to be of high risk, while CRP above 10 mg/L is suggestive for severe acute infections (Ishii et al., 2012). Fibrinogen (in g/L) is a glycoprotein that stops bleeding by helping blood clots to form, also considered as an inflammatory biomarker. Glycated haemoglobin (HbA1c) is a validated diagnostic test for diabetes. HbA1c \geq 48 mmol/mol is suggestive for diabetes (> 42 for predictable risk), with higher levels capturing the severity of the condition (WHO, 2011). Cholesterol ratio, calculated as the ratio of total cholesterol over high density lipoprotein cholesterol, is our "fat in the blood" biomarker. A cholesterol ratio greater than 4 is suggestive for elevated atherosclerotic risk (Millán et al., 2009). Descriptive statistics of all health outcomes are presented in Appendix Table A1. Some studies in the literature have constructed a single health index such as the allostatic load (Makdissi et al., 2013). However, following Clarke and Erreygers (2020), we consider each biomarker separately as they are indicators for different diseases and could be influenced by different drivers. For targeted

policy interventions it is informative to consider how deprivation and its components are associated with each biomarker separately.

3.3 Measures of socio-economic status

Deprivation in non-monetary domains, such as social isolation, is of great concern to policy makers in the UK. For effective government support it is important to target policy intervention at the most vulnerable segments of society which requires an understanding of the differential influences of monetary and non-monetary aspects of deprivation on health outcomes. To understand the partial association between multidimensional deprivation and health beyond income we need to have a measure of multidimensional deprivation: a measures of multidimensional deprivation: a measures of multidimensional deprivation based on non-income domains only - multidimensional non-monetary deprivation (MND); and another measure of multidimensional deprivation based on deprivation in both income and non-income domains - multidimensional deprivation (MDD). The MND measure allows us to analyse the relative importance of disadvantage in multiple domains in explaining the health gradient beyond income. The MDD allows us to evaluate the relative importance of overall disadvantage in monetary and non-monetary domains on health outcomes.

3.3.1 Multidimensional deprivation

An important issue in constructing our dynamic multidimensional deprivation measure is the selection of dimensions (Alkire, 2002). For our analysis, the choice of these dimensions is motivated by the recommendations in the existing literature (Bossert et al., 2013, Stiglitz et al., 2010). Stiglitz et al. (2010) identified the following domains as shaping individual well-being: material living standard (income, consumption and wealth); education; personal activities; political voice and governance; social connection and relationships; and insecurity (economic and physical). For example, social isolation, as an integral part of disadvantage and is of grave concern in the UK with the literature, such as Holt-Lunstad et al. (2010), arguing that the adverse health impacts of loneliness are equivalent to detrimental effects of smoking and obesity. Following these recommendations and data availability, our measure

of multidimensional deprivation considers domains for both non-monetary and monetary dimensions. The MND is based on 10 domains and the MDD is based on 11 domains, the only difference between the two being that MDD includes the ten dimensions of the MND along with income. The complete set of 10 domains used in the construction of MND include: education, economic activity, housing conditions, affordibility of basic consumer durables, car ownership, affordable lifestyle, financial hardships, social engagement and environment and security. For MDD this list includes income to make the complete set of 11 domains: income, education, economic activity, housing conditions, afford consumer durables, car ownership, affordable lifestyle, financial hardships, social engagement and environment and security. This is also in accordance with the recommendations by policy makers such as the European Union which require indices of material deprivation with income based poverty and employment indicators (Bossert et al., 2013).

Each of these domains comprise of a set of dimensions relevant to the domain. The deprivation threshold in each domain is defined as deprivation in one or more dimensions. This is known as the *union approach* to classifying overall deprivation that is inclusive of all dimensions and is sensitive to the inequality in distribution of deprivation (Datt, 2019). This approach allows accounting for the extra burden of multidimensional deprivation, thus acknowledging that disadvantage in multiple dimensions can result in making the transition out of deprivation difficult (Banerjee et al., 2015). The domain on education, economic activity, security, car ownership are based on single dimension. The domain on education takes the value 1 if the respondent is either uneducated or highest level of education is less than high school (Level A). The domain economic activity (an indicator of the individual's employment status) takes the value 1 if the individual is not employed and a value 0 if they are employment. The domain on housing condition comprises of the response regarding shortage of space, not enough light, lack of adequate heating, damp walls/floors, separate bathroom and central heating in house. If the household reports a lack in access for one or more of these dimensions it is coded as 1. The domain on affordability of consumer durables comprises of dimensions on household's ability to afford basic modern day durables including video recorder, fridge freezer, washing machine, drier, dishwasher, home computer, satellite dish or cable television. An individual is considered to be deprived if they do not have access to one or more of the consumer durables. The domain car ownership records whether there is a car available in the household or not. The domain affordable lifestyle comprises of three dimensions on can not afford to replace furniture, feed visitors once a month, keep house well decorated. The domain financial hardship includes dimensions on whether the household was late in paying rent, housing requirement required cutback, can not afford to pay for annual holiday. The domain on social engagement includes two dimensions, the frequency of talking to neighbours and frequency of meeting people, with both dimensions taking a value of 1 if response is twice a month or less and 0 if response is once a week or most days. The domain on environment includes two dimensions on pollution/environmental problems and noise from neighbours, both taking the value 1 if respondent attest. The domain on security includes one dimension on vandalism or crime in neighbourhood that takes the value 1 if the household reports of these problems. The last domain on income deprivation takes the value of 1 if the income is less than 60% of median household income and 0 otherwise. These domains are described in Appendix Table A2 and their summary statistics are presented in Table A5. Correlation between domains is presented in Table A6 suggest statistically significant and low correlation between deprivation in most domains.

The analysis of deprivation across multiple dimensions over time requires a balanced panel that covers maximum dimensions across the longest possible time period (Nicholas et al., 2019), but without compromising on the dimensions, sample size or the number of years in the panel. Within this context, experimenting with different time frames we found a ten year time interval over 1999-2008 (BHPS waves 9 to 18) to give us the largest sample and hence most suitable for constructing our long-run deprivation measure. We created our working sample by merging the balanced panel of BHPS waves 9 to 18 with the UKHLS wave 3 followed up by nurse visits for biomarker data.

3.3.2 Income data

We use the household income data available in the BHPS. To facilitate comparison over time and between households, household income is deflated using the Retail Price Index, to express income in January 2010 prices, and equivalised using the modified OECD scale. For consistency with our longitudinal multidimensional deprivation measure, we measure income as the within individual average income measured over BHPS waves 9 to wave 18 (i.e., 1999 - 2008). We treat income as two separate sub analyses: as an independent measure

of SES and as a domain in our MDD measure of deprivation. The former is used for the analysis assessing the importance of non-monetary deprivation on health, beyond income, we controlling for income and multidimensional deprivation separately in using MND and income as two separate variable in the regression analysis. Here income is transformed to natural logarithms to allow for the concavity of the health income association and skewness of income distribution. In case of the latter where income is included as a domain in the MDD, it takes the value 1 if income falls below 60% of average income and 0 otherwise.

3.4 Other covariates

The covariates used to model our health outcomes over and above deprivation and income were collected during the UKHLS wave 3 and are presented with summary statistics in Appendix Table A6. We use a similar set of covariates as by Contoyannis et al. (2004) and Carrieri and Jones (2017). Specifically, our estimation models include 15 age dummies (age group dummies for five years intervals between 15 and 84 and a dummy for those over 84), gender (male vs female) and ethnicity (white vs non-white). We include marital status since it may affect household production of health and demand for health. A set of household characteristics (household size and number of children in the household) and household composition dummies are also included. Finally, dummies for regions are added to capture regional variations.

4 Results

4.1 Shapley decomposition of overall deprivation into its components

Table 1 presents the Shapley decomposition of multidimensional deprivation (MDD) into the contribution of the breadth and dynamic component for each domain. Panel A of this table presents the results for MDD without accounting for persistence and panel B presents the results for MDD accounting for persistence of multidimensional deprivation.

Comparing the results in panel A and panel B of this table suggests the following. First, the monetary domain (income) has a proportionally smaller contribution to overall deprivation than the non-monetary domains (accounting for 5.19% of overall deprivation in panel A

(column [2]) and 3.38% in the panel B (column [2])). This is an important results as it highlights the importance of embracing a broader concept of deprivation beyond income. Thus indicating that income is not important perse and is a mere indicator of autonomy over economic resources (Bossert et al., 2013). Second, accounting for persistence of deprivation results in an increase in the contribution of non-monetary dimensions and a lower contribution of income to overall deprivation (contribution of non-monetary domains to overall deprivation increases from 94.81% to 96.62% panel B (column[2])). In other words, accounting for persistence of deprivation reduces the proportional contribution of income in overall deprivation (the contribution of income in panel B (3.38%) compared to panel A (5.19%)). This result corresponds with the existing wellbeing literature which suggests that with time individual's aspirations change which results in any change in income to result in a little increase in overall wellbeing across time (OECD, 2013, Graham and Pettinato, 2002). Furthermore, the decline in the proportional contribution of income to overall deprivation with persistence also suggests that it is relatively easier to get out of income deprivation across time than out of material deprivation as suggested in the literature (Bossert et al., 2013, Fahey, 2007).

Comparing the prevalence (column[7]) and dynamic components (column[8]) suggests overall MDD is dominated by the prevalence component rather than by the dynamic component for all domains (economic activity, housing conditions, financial hardship, social engagement environment, security and income), except two domains (affordability of consumer durables and education). Thus, breadth of deprivation has a greater role to play and overall deprivation is dominated by the prevalence of deprivation in both panels. The rank ordering of dimensions for MDD with persistence is similar to those for MDD without persistence, in line with the previous literature on multidimensional deprivation with no persistence (see Nicholas et al. (2019) for China and Whelan and Maître (2012) for Europe). The size of the contribution to overall deprivation is much larger in panel B than in panel A for affordability of consumer durables, education, and no car. Here, it is interesting to note that the contribution of income increasing from 19.9% to 25.43% highlights the role of persistent deprivation in this domain in overall deprivation. The large contribution of affordability of consumer durables is attributable to ultradeprivation in this domain due to higher rates of deprivation in the dimensions associated with this domain (Table A5). Thus multifaceted

interventions targeting deprivation could help alleviate extra burden of extreme deprivation in domains with higher contribution to overall deprivation (Banerjee et al., 2015).

4.2 Income and deprivation gradient in health

A comparative study of the gradient for deprivation in multiple non-monetary domains and the income gradient would establish the relative importance of the two measures of SES. Accordingly, we consider income and MND deprivation¹³ gradient in the biomarker distribution (equation 7) and estimate it across quantiles (with 0.05 increments). These results for each biomarker are presented in Figure 1, Figure 2 and Appendix Table A4.

Overall, the deprivation and income gradients are more pronounced and larger in magnitude towards the right tails of the biomarker distributions, where elevated biomarkers are a sign of a greater burden of illness for individuals and higher costs for the healthcare system. Specifically, for our two adiposity measures, although no systematic associations at the mean (see Table A4) are observed, we find a steep increase in deprivation gradients after the 75^{th} percentile of the BMI (i.e., BMI > $31.7 \text{ kg/}m^2$) and waist circumference (i.e., > 106 cm) distribution (Figure 1); these correspond to BMI and waist circumference values close to the clinical threshold for elevated health risks, indicating stronger positive associations with greater deprivation. For income, our UQR results also show that the OLS estimator masks notable differences in the income-adiposity gradient across the BMI and waist circumference distributions. For example, we find that the negative income gradient peaks at around the 95^{th} percentile of the BMI distribution, which is about 5 times higher than the corresponding OLS coefficient. The evidence on the gradient due to material deprivation over and above the effect of income suggests that income alone is not sufficient to account for the socioeconomic gradient in adiposity measures.

For blood pressure measurements, results show no systematic deprivation gradients both at the mean and across quantiles of their distribution. On the other hand, the deprivation gradient is much more pronounced, independent of income, for our cardiovascular fitness measure (heart rate) towards the right tail of its distribution (Figure 1). For example, analysis "beyond the mean" reveals that although there is a flat pattern in the deprivation gradient in heart rate across most of its distribution, there is a steep increase at the far

 $^{^{13}}$ Here we use the measure of material deprivation (based on 10 domains) as explained in Section 3.

right tails of the distribution; the deprivation gradient at the 95^{th} percentile is about 2.5 times higher than the OLS coefficient. A gradually increasing negative income gradient is also evident at higher quantiles of the heart rate distribution.

For inflammatory biomarkers (CRP and fibrinogen), our analysis at the mean (Table A4) suggests a systematic income gradients with the corresponding results for deprivation less pronounced. However, UQR estimates (Figure 2) show a different result, with gradually increasing and statistically significant (at least the 5% level) deprivation gradients beyond the normal range of CRP (i.e., for CRP > 3). There are no systematic associations for very high CRP values, mostly reflecting non-systematic but recent infections (CRP > 10; Ishii et al., 2012). Similarly, we find increasing income gradients towards the highest quantiles of the CRP distribution. A generally flat income and deprivation gradient is evident across the distribution of fibrinogen, in line with previous evidence (Carrieri and Jones, 2017).

For our "blood sugar" biomarker (HbA1c), we find a sharp increase in the positive deprivation gradient towards the right tail of the distribution (Figure 2). Specifically, we find a "saddle" point at around the 90^{th} percentile of the distribution (corresponding to the clinical threshold of diabetes), with the relevant UQR coefficient being statistically significant at the 10% level. For cholesterol ratio, a predictor of several heart diseases, we find no systematic associations with deprivation over and above the role of income. However, the income gradients in cholesterol ratio remain fairly stable up to the 75^{th} percentile of the cholesterol ratio distribution, which is very close to the high-risk threshold of 4 (Millán et al., 2009), and then gradually increases toward the far right tails of the distribution.

4.3 Decomposing the multidimensional deprivation in health gradient into its sources

Table 2 presents the estimation results for equation (9) which decomposes the multidimensional deprivation in health gradient into its sources allowing us to conduct a counterfactual analysis of the relative contribution of breadth (prevalence) and length (dynamic) of deprivation to each biomarker using our holistic MDD measure (based on deprivation across 11 domains). The percentage contribution of each of the two components to the predicted counterfactual outcome for each biomarker is estimated at the 10th, 25th, 50th, 75th, 90th

and 95^{th} quantile of the distribution. Capitalising on the ability of our proposed MDD measure to account for persistence, we estimate this equation for MDD without persistence (i.e., for $\theta = 0$ in panel A) and MDD with persistence (i.e., for $\theta = 1$ in panel B). For any given biomarker, if the sign of the percentage contribution is positive (negative) means that the component increases (decreases) our health measures, indicating a positive (negative) overall association with ill health, since our biomarkers measure ill health.

4.3.1 Distributional analysis of the role of breadth and dynamic components of MDD in explaining biomarkers

Our results highlight the distinctive role of the breadth (prevalence) and dynamic components of deprivation in shaping the gradient in biomarkers. Heterogeneity in the association between biomarkers and two MDD components suggests the size of the gradient varies across the distribution of biomarkers. For most biomarkers, the percentage contribution of the prevalence component for the no persistence MDD model (panel A) had the largest increase across quantiles of 18 times for cholesterol ratio (-0.50\% to 8.80\%) followed by 13 times each for BMI (-0.37% to 4.61%) and HbA1c (1.25% to 16.60%). The gradient of the dynamic MDD is steeper than the prevalence MDD at the higher quantiles of the biomarker distribution due to a substantial increase in the contribution for the dynamic component across the distribution. Considering cholesterol ratio, the prevalence component increased by 18 times and the dynamic component increased by 28 times across the distribution, emphasising the increasing role of these components at higher quantiles. Even though the dynamic MDD component dominated the dimension MDD component across quantiles for most biomarkers, for biomarkers of adiposity (BMI, WC), inflammation (fibringen) and blood sugar (HbA1c), dimension deprivation contributed more than duration of deprivation at the highest quantile (q95). The contribution of two MDD components with the strong distributional gradient in health highlights the distinctive role of prevalence and duration of multidimensional deprivation in explaining health outcomes.

Our model with persistence (panel B) shows similar results of a clear gradient in the percentage contribution of both components of MDD to biomarkers. Deprivation in prevalence and persistence exerts a positive influence across the distribution for most biomarkers, with some exceptions - higher quantiles for HbA1c and bottom quantiles for BMI, diastolic BP

and cholesterol ratio. In both panels the percentage contribution of breadth component is increasing in magnitude towards the right tails of the biomarker distribution. The percentage contribution of dimension deprivation increased across quantiles by up to 18 times for HbA1c (0.98% to 17.59%), 16 times for BMI (-0.21% to 3.29%), and 12 times for cholesterol ratio (-0.50% to 5.62%) with the exception of systolic blood pressure. For the dynamic MDD component, the percentage contribution increased across quantiles by up to 8 times for BMI (0.29% to 2.28%), 9 times for diastolic blood pressure (-0.42% to 3.28%) and five times cholesterol ratio (2.45% to 12.24%), and decreasing only slightly for fibrinogen, systolic BP with the exception of HbA1c which declined by 28 times. Considering the biomarkers with the steepest change for both components, i.e., HbA1c, BMI and cholesterol ratio, the sharp increase in the prevalence component was offset by a sharper decline in the persistence component for HbA1c resulting in the former component dominating the latter in the percentage contribution to this biomarker. For BMI and cholesterol ratio, while both components have a steep gradient, the size for the dimension components was larger than that for the dynamic component.

4.3.2 Analysis of the role of persistence in the contribution of prevalence and persistence of MDD to biomarker

Contrasting the results for MDD with no persistence (Table 2, panel A) with those with persistence (Table 2, panel B) suggests that the dynamic component dominates the dimension component for most biomarkers at lower quantiles, indicating greater health damage caused by persistence of deprivation. Accounting for persistence of deprivation increases the percentage contribution of the dynamic component by up to three times larger at lower quantiles for most biomarkers (except for HR, fibrinogen and cholesterol ratio where it decreased). At highest quantiles however, where risk of ill health is higher, the role of the two MDD components in explaining predicted biomarkers was specific to the relevant biomarker. At higher quantiles, the size increases up to 60% (diastolic BP, CRP and fibrinogen) and decreased up to 55% (BMI, WC, HR, HbA1c and cholesterol ratio). This is also accompanied by a shrinking in the size of the duration component below the median of up to 55% (heart rate, BMI, WC, systolic, HbA1c and cholesterol ratio). It is interesting to note that while there was an overall decline in the percentage contribution of prevalence component,

as we move from panel A to B, the overall percentage contribution of the dynamic component increased, taking over the relative domination of the prevalence component at more quantiles.

While the overall picture in the model without persistence suggested that the percentage contribution of prevalence component exerted a positive and large association with biomarkers at higher quantiles, the model with persistence suggests that the size of this association shrinks for most biomarkers with dynamic component dominating for BMI, WC, CRP and HbA1c and dynamic component dominating for diastolic BP, systolic BP, HR, fibrinogen and cholesterol ratio. For example, for adiposity measures (BMI, WC), HR, blood sugar (HbA1c) and cholesterol ratio¹⁴, the dynamic component accounts for a larger contribution to the gradient in health at the lower quantiles (below q(50)) of the biomaker distribution in the model with persistence than in the model without persistence. Overall, at most quantiles below q(75), the contribution of the dimension component is smaller than the duration component in both panels suggesting the domination of dynamic MDD over dimension MDD in their contribution to the biomarkers.

The most striking observation is that at quantiles beyond the median, while there is no clear domination of either of the two components of MDD, there is an overall increase in the number of biomarkers showing a domination of the dynamic component over the duration component. The dynamic component continued to dominate prevalence components at higher quantiles of HR, cholesterol ratio, diastolic BP, systolic BP and fibrinogen although the size of the domination shrinked in moving from panel A to panel B. For measures of adiposity (BMI and WC), CRP and blood sugar (HbA1C), on the other hand, the prevalence component continues to dominate the contribution at highest quantiles (q(95)). Specifically, at quantiles beyond q(75), the size of prevalence component increased for diastolic BP, fibrinogen (q(95)), and HbA1c (q(95)) as we account for persistence (panel B). The results for CRP are an outlier in our analysis as in the existing literature (Davillas and Jones, 2020), the contribution of the dimension component increased from 17.67% at the bottom to 55.40% at the top of the distribution for deprivation without persistence and from 6.36% at q(10) to

¹⁴For cholesterol ratio the proportional contribution of dimension component was negative (-0.50%) at the bottom and positive (8.80%) at the top quantile for no persistence and negative (-0.50%) at the bottom and positive (12.24%) at the top of the distribution for model with persistence, an increase by approximately 4 percentage points.

37.65% at q(95) for deprivation with persistence. Results at highest quantile q(95) suggests that dynamic component deprivation exerts a larger contribution to the predicted cholesterol ratio at the higher quantiles, indicating prevalence of MDD exerts a greater influence on these measures than the dynamic component. For fibrinogen (beyond q(50)), while the prevalence MDD component dominated the dynamic component in panel A, accounting for persistence resulted in the dynamic MDD component exerting a greater influence than the prevalence component at higher quantiles. These results suggest the persistence of MDD exerted a greater influence than prevalence of MDD for these measures notwithstanding an overall increase in the influence of dynamic component and decrease in the prevalence component once we account for persistence.

5 Discussion and conclusions

The measurement and understanding of the dynamic relationship between multidimensional deprivation and health is important for design and evaluation of policies targeting health of the disadvantaged population. ing. This paper has developed a new approach to measure deprivation in multiple domains that incorporates the breadth, duration and persistence of deprivation, allowing us to analyse how longitudinal histories of deprivation and spells of deprivation shape the deprivation gradient in health. Capitalising on the axiomatic property of decomposability of multidimensional deprivation into its components, we disintegrate the contribution of prevalence and persistence of deprivation in explaining the gradient in health across the distribution of biomarkers for the UK.

The first finding relates to the importance of a broader approach to measure deprivation that extends beyond income (Bilger and Carrieri, 2013, Blázquez et al., 2014). Adopting a holistic approach to measure multidimensional disadvantage, our analysis highlights the importance of accounting for duration and persistence of deprivation in monetary and non-monetary domains of deprivation and its gradient in biomarkers. The unique feature of our multidimensional deprivation measure that decomposes deprivation into its static and dynamic components (i.e., breadth, duration and persistence of deprivation) using the Shapley decomposition method allows us to compute the contribution of prevalence and persistence of deprivation in each domain to overall deprivation. Specifically, our analysis suggests that

income, a traditional measure of disadvantage, contributes less to overall deprivation in the UK and is ranked low (7^{th}) in the set of 11 domains considered in our MDD measure. Overall persistent deprivation in the UK is dominated by deprivation in education (25.43%), basic durables (43.23%), unemployment (9.50%), housing conditions (4.36%) and social isolation (3.95%), with income deprivation accounting for a smaller proportion (3.38%). Thus, policy targeting disadvantage should prioritise non-monetary domains rather than just income for policy to have the desired effect (Bossert et al., 2013). This is in line with the Europe 2020 growth strategy which set out five targets to be achieved by 2020 which specifically included non-monetary domains only (Bárcena-Martín et al., 2014). Incorporating this multidimensional approach to measuring deprivation suggest that the socioeconomic gradient in most of our health measures is not solely attributed to income and it is important to account for deprivation in multiple domains of socioeconomic wellbeing. The existence of a systematic deprivation gradient, beyond income, across the distribution of most of the biomarkers in our analysis (i.e., BMI, waist circumference, heart rate, CRP and haemoglobin) and the gradient becomes larger in magnitude at higher quantiles of the distribution of biomarkers, where higher health risks are evident.

Another contribution of this paper relates to the distributional heterogeneity in the contribution of the prevalence and persistence components of deprivation to biomarkers. Using unconditional quantile regression to conduct a distributional analysis of biomarkers helps in understanding how the gradient evolves for people with varying vulnerability in health. We find a strong association between health (BMI, HbA1c, C-reactive protein and BP) and persistence of deprivation across the distribution, indicative of the grave consequences persistence of deprivation has on health. Considering cholesterol ratio, persistence of deprivation accounted for around 20 percentage point more contribution at highest quantile than at the lowest quantile, highlighting the greater role of socioeconomic status for biomarkers at higher quantiles that are beyond the clinical threshold (Bilger et al., 2017). Our analysis suggests deprivation in multiple domains and its components will have strong implications for population health outcomes, and effective policy interventions designed to reduce health risk should account for persistence in critical domains.

Capitalising on the theoretical framework, this paper for the first time, analyses the role of dynamic deprivation with particular emphasis on accounting for persistence and suggests that ignoring these aspects of deprivation would provide a misleading picture of the gradient in health. For most biomarkers the dynamic component of deprivation is more relevant in shaping the observed deprivation gradients than the dimension gradient with some exception (for BMI, WC, CRP, fibrinogen and HbA1c) at higher quantiles. The associations between deprivation in multiple domains, persistence of deprivation and health identified in this paper has important implications for the equitable and efficient allocation of resources. Our analysis highlights the need to specifically target persistent disadvantage in domains such as education, durables, unemployment, housing conditions and social isolation, beyond income. Higher rates of multidimensional deprivation and welfare reforms that do not help groups vulnerable to economic crisis are likely to result in poor health outcomes.

Finally, the importance of understanding the role of multidimensional deprivation and its components in influencing on health (Marmot, 2020; Finn and Goodship, 2014; Bloomer et al., 2012) is important for future health policy (Iacobucci, 2020). Our proposed measure is also relevant to analysis in the context of measuring and analysing the role multidimensional deprivation and the distinctive role of breadth and persistence in several other dimensions of individuals' well-being where focusing on the health of multidimensionally disadvantaged people is of particular concern.

Table 1: Shapley Decomposition of Dynamic Multidimensional Deprivation (MDD)

	Panel A			
${\bf Multidimensional}$	Deprivation	with	no	persistence

		Percentage			Total		
Domain	Dynamic deprivation	contribution		column	Prevalence	Duration	
		of o	ompone	$_{ m ents}$	(3)- (5)	of deprivation	of deprivation
	(Ω)	Ω_A	Ω_B	Ω_C		$(\Omega^{dimension})$	$(\Omega^{duration})$
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Education	19.90	55.64	0.11	44.24	100	17.31	20.27
Economic activity	9.00	65.73	1.67	32.60	100	9.47	9.03
Housing conditions	7.61	72.84	4.46	22.70	100	9.17	7.41
Afford consumer durables	33.95	48.51	0.04	51.45	100	25.72	34.60
No car	4.74	73.35	1.51	25.13	100	5.54	4.76
Affordable lifestyle	3.56	86.02	5.66	8.33	100	5.10	3.43
Financial hardship	0.86	101.74	10.48	-12.22	100	1.50	0.78
Social engagement	7.64	68.57	4.00	27.43	100	8.65	7.48
Environment	3.61	83.71	6.68	9.61	100	5.09	3.43
Security	3.95	91.48	6.86	1.66	100	6.06	3.75
${\rm Income}$	5.19	74.89	4.01	21.10	100	6.38	5.07
Overall	100	62.17	1.92	35.92	100	100	100

Panel B

Multidimensional Deprivation with persistence

	Percentage			Total	persisteries		
Domain	Dynamic deprivation	cor	ntributi	on	column	Prevalence	Persistence
		of co	ompone	$_{ m nts}$	(3)- (5)	of deprivation	of deprivation
	(Ω)	$\overline{\Omega_A}$	Ω_B	Ω_C		$(\Omega^{dimension})$	$(\Omega^{persistence})$
Education	25.43	46.25	0.03	53.72	100	23.70	25.50
Economic activity	9.50	57.42	0.41	42.17	100	11.06	9.49
Housing conditions	4.36	66.95	1.18	31.87	100	5.98	4.32
Afford consumer durables	43.23	38.96	0.02	61.02	100	33.93	43.35
No car	5.04	63.38	0.37	36.24	100	6.47	5.04
Affordable lifestyle	1.70	81.54	1.40	17.06	100	2.84	1.68
Financial hardship	0.15	113.77	3.73	-17.53	100	0.36	0.15
Social engagement	3.95	62.38	1.14	36.48	100	5.05	3.91
Environment	1.55	79.58	1.80	18.63	100	2.55	1.53
Security	1.70	90.99	1.82	7.19	100	3.17	1.67
${\rm Income}$	3.38	70.68	1.03	28.29	100	4.88	3.36
Overall	100	49.37	0.29	50.34	100	100	100

Note: Refer to Equation 3 and 3 in Section 2.1. The multidimensional deprivation measure is based on 11 dimensions across 10 years.

 $[\]Omega_A$: Sum of count of deprivation averaged over individuals. This is the distribution sensitive component.

 $[\]Omega_B$: Component of multidimensional deprivation due to distribution of breadth of deprivation.

 $[\]Omega_C$: Component of multidimensional deprivation due to the distribution of length of deprivation.

N=57,070 observations for 5,707 individuals over 10 years (BHPS wave 9 to wave 18, 1999-2008).

Figure 1: Income and deprivation (MND) gradient in nurse-collected health measures: unconditional quantile regression

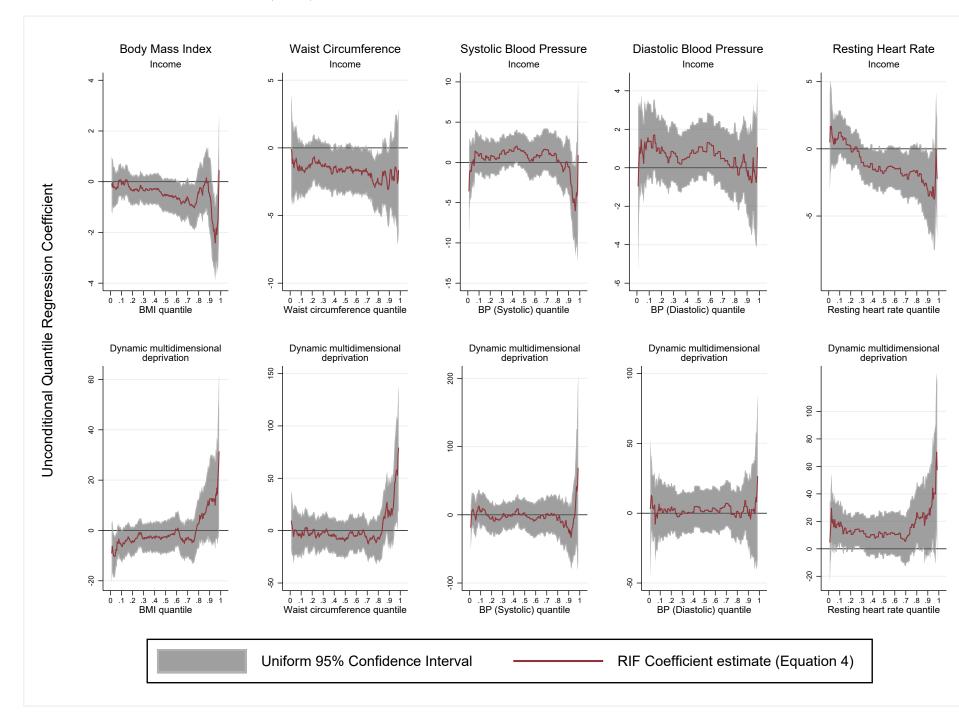


Figure 2: Income and deprivation (MND) gradients in blood-based biomarkers: unconditional quantile regression estimates

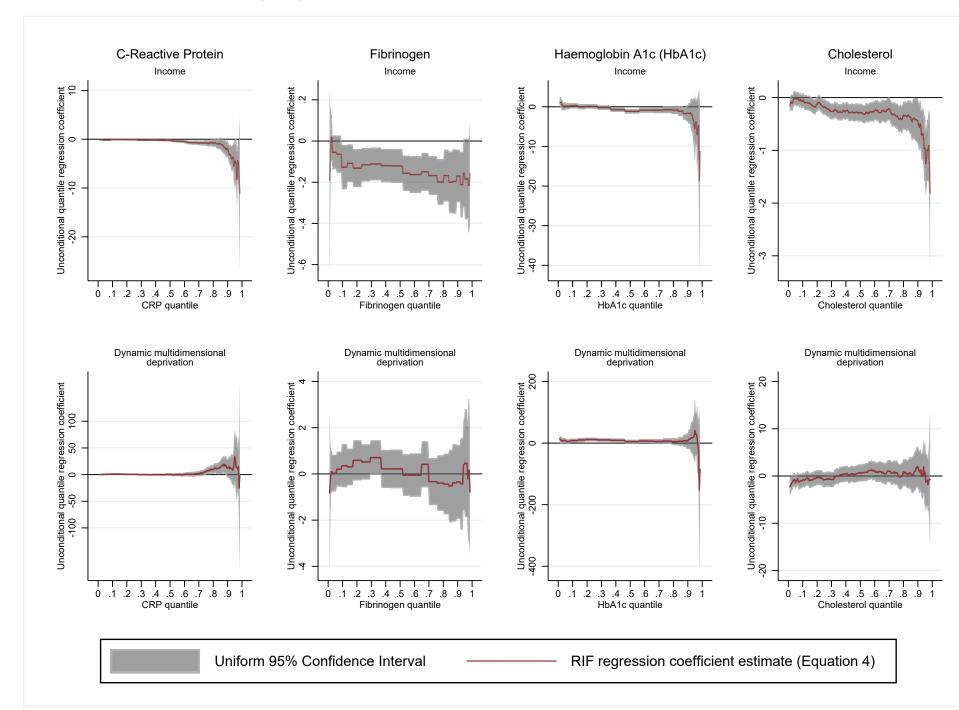


Table 2: Percentage contribution of components of multidimensional deprivation to predicted biomarkers *

Biomarker Component $q(10)$ $q(50)$ $q(90)$ $q(90)$ $q(90)$ BMI Prevalence (Ω dimension) 0.37 0.84 1.22 1.67 3.59 4.61 Waist Prevalence (Ω dimension) 0.21 3.39 3.02 1.46 5.29 3.86 Waist Prevalence (Ω dimension) 0.73 0.89 0.89 1.56 5.26 3.19 Gircumference Duration (Ω duration) 0.18 0.49 0.22 0.24 0.02 -0.34 Blood Pressure Duration (Ω duration) 0.55 1.45 0.95 1.17 0.88 1.21 Systolic Prevalence (Ω dimension) 1.23 0.15 -0.27 -0.19 0.29 1.27 Resting Prevalence (Ω dimension) 1.70 1.94 1.98 1.89 2.88 2.70 heart rate Duration (Ω duration) 2.78 1.02 2.32 1.28 9.4 1.54 Protein Duration (Ω duration) 3.34	Pan	el A - Multidimensiona	l depriv	vation (I	MDD) v	without	persisten	ıce
Prevalence (Ω ^{dimension}) 0.37 0.84 1.22 1.67 3.59 3.66		Deprivation		$\mathbf{Q}\iota$	ıantile d	of healtl	n measure	es
Duration (Ω ^{duration})	Biomarker	${f Component}$	$\mathbf{q}(10)$	$\mathbf{q}(25)$	$\mathbf{q}(50)$	q(75)	$\mathbf{q}(90)$	${f q(95)}$
Duration (Ω ^{duration})	BMI	Prevalence ($\Omega^{dimension}$)	-0.37	0.84	1.22	1.67	3.59	4.61
Circumference Duration $(Ω^{duration})$ 4.88 4.45 0.73 0.30 2.12 1.55 Diastolic Prevalence $(Ω^{dimension})$ 0.18 0.49 -0.22 -0.24 0.02 -0.34 Blood Pressure Duration $(Ω^{duration})$ 0.55 1.45 0.95 1.17 0.88 1.21 Systolic Prevalence $(Ω^{dimension})$ 1.23 0.15 -0.27 -0.19 0.29 1.27 Resting Prevalence $(Ω^{dimension})$ 1.70 1.94 1.98 1.89 2.88 2.70 C-Reactive Prevalence $(Ω^{dimension})$ 2.78 1.02 4.83 4.00 5.24 5.04 C-Reactive Prevalence $(Ω^{dimension})$ 24.10 0.37 40.09 32.80 20.41 -7.21 Fibrinogen Prevalence $(Ω^{dimension})$ 3.30 2.99 2.42 1.86 2.68 2.50 Portein Duration $(Ω^{duration})$ 3.44 6.01 6.69 -0.90 -3.73 1.97		Duration $(\Omega^{duration})$	0.21	3.39	3.02	1.46	5.29	3.86
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	Waist	Prevalence $(\Omega^{dimension})$	0.73	0.89	0.89	1.56	2.62	3.19
Blood Pressure Duration (Ω ^{duration}) 0.55 1.45 0.95 1.17 0.88 1.21 Systolic Prevalence (Ω ^{dimension}) 0.62 -0.05 -0.43 -0.37 0.00 0.46 Blood Pressure Duration (Ω ^{duration}) 1.23 0.15 -0.27 -0.19 0.29 1.27 Resting Prevalence (Ω ^{dimension}) 1.70 1.94 1.98 1.89 2.88 2.70 heart rate Duration (Ω ^{duration}) 2.78 1.02 4.83 4.00 5.24 5.04 C-Reactive Prevalence (Ω ^{dimension}) 24.10 0.37 40.09 32.80 20.41 -7.21 Fibrinogen Prevalence (Ω ^{dimension}) 3.30 2.99 2.42 1.86 2.68 2.50 Duration (Ω ^{duration}) 3.44 6.01 6.69 -0.90 -3.73 1.97 HbAre Prevalence (Ω ^{dimension}) 1.25 1.45 1.99 1.68 5.36 16.60 Duration (Ω ^{duration}) 0.41	Circumference	Duration $(\Omega^{duration})$	4.88	4.45	0.73	0.30	2.12	1.55
Blood Pressure Duration (Ω ^{duration}) 0.55 1.45 0.95 1.17 0.88 1.21 Systolic Prevalence (Ω ^{dimension}) 0.62 -0.05 -0.43 -0.37 0.00 0.46 Blood Pressure Duration (Ω ^{duration}) 1.23 0.15 -0.27 -0.19 0.29 1.27 Resting Prevalence (Ω ^{dimension}) 1.70 1.94 1.98 1.89 2.88 2.70 heart rate Duration (Ω ^{duration}) 2.78 1.02 4.83 4.00 5.24 5.04 C-Reactive Prevalence (Ω ^{dimension}) 24.10 0.37 40.09 32.80 20.41 -7.21 Fibrinogen Prevalence (Ω ^{dimension}) 3.30 2.99 2.42 1.86 2.68 2.50 Duration (Ω ^{duration}) 3.44 6.01 6.69 -0.90 -3.73 1.97 HbAre Prevalence (Ω ^{dimension}) 1.25 1.45 1.99 1.68 5.36 16.60 Duration (Ω ^{duration}) 0.41	Diastolic	Prevalence $(\Omega^{dimension})$	-0.18	0.49	-0.22	-0.24	0.02	-0.34
Blood Pressure Duration (Ω ^{duration}) 1.23 0.15 -0.27 -0.19 0.29 1.27 Resting Prevalence (Ω ^{dimension}) 1.70 1.94 1.98 1.89 2.88 2.70 heart rate Duration (Ω ^{duration}) 2.78 1.02 4.83 4.00 5.24 5.04 C-Reactive Prevalence (Ω ^{dimension}) 17.67 -1.98 12.02 23.21 28.94 55.40 Protein Duration (Ω ^{duration}) 3.30 2.99 2.42 1.86 2.68 2.50 Duration (Ω ^{duration}) 3.44 6.01 6.69 -0.90 -3.73 1.97 HbA1c Prevalence (Ω ^{dimension}) 1.25 1.45 1.99 1.68 5.36 16.60 Duration (Ω ^{duration}) 0.41 2.93 1.32 -0.18 -4.22 -10.60 Cholesterol Prevalence (Ω ^{dimension}) -0.50 1.07 4.45 4.62 6.63 8.80 Ratio Deprivation Quantile Quanti	Blood Pressure	Duration $(\Omega^{duration})$	0.55	1.45	0.95	1.17	0.88	1.21
Blood Pressure Duration (Ω ^{duration}) 1.23 0.15 -0.27 -0.19 0.29 1.27 Resting Prevalence (Ω ^{dimension}) 1.70 1.94 1.98 1.89 2.88 2.70 heart rate Duration (Ω ^{duration}) 2.78 1.02 4.83 4.00 5.24 5.04 C-Reactive Prevalence (Ω ^{dimension}) 17.67 -1.98 12.02 23.21 28.94 55.40 Protein Duration (Ω ^{duration}) 3.30 2.99 2.42 1.86 2.68 2.50 Duration (Ω ^{duration}) 3.44 6.01 6.69 -0.90 -3.73 1.97 HbA1c Prevalence (Ω ^{dimension}) 1.25 1.45 1.99 1.68 5.36 16.60 Duration (Ω ^{duration}) 0.41 2.93 1.32 -0.18 -4.22 -10.60 Cholesterol Prevalence (Ω ^{dimension}) -0.50 1.07 4.45 4.62 6.63 8.80 Ratio Deprivation Quantile Quanti	Systolic	Prevalence ($\Omega^{dimension}$)	0.62	-0.05	-0.43	-0.37	0.00	0.46
heart rate Duration (Ω ^{diration}) 2.78 1.02 4.83 4.00 5.24 5.04 C-Reactive Prevalence (Ω ^{dimension}) 17.67 -1.98 12.02 23.21 28.94 55.40 Protein Duration (Ω ^{duration}) 24.10 0.37 40.09 32.80 20.41 -7.21 Fibrinogen Prevalence (Ω ^{dimension}) 3.30 2.99 2.42 1.86 2.68 2.50 Duration (Ω ^{duration}) 3.44 6.01 6.69 -0.90 -3.73 1.97 HbA1c Prevalence (Ω ^{dimension}) 1.25 1.45 1.99 1.68 5.36 16.60 Duration (Ω ^{duration}) 0.41 2.93 1.32 -0.18 -4.22 -10.60 Cholesterol Prevalence (Ω ^{dimension}) -0.50 1.07 4.45 4.62 6.63 8.80 Ratio Q ^{duration} 0.71 1.43 8.73 8.77 18.54 20.91 Panel B - Multidimensional deprivation (MDD) vith persistence	Blood Pressure	Duration $(\Omega^{duration})$	1.23	0.15	-0.27	-0.19	0.29	1.27
heart rate Duration (Ω ^{diration}) 2.78 1.02 4.83 4.00 5.24 5.04 C-Reactive Prevalence (Ω ^{dimension}) 17.67 -1.98 12.02 23.21 28.94 55.40 Protein Duration (Ω ^{duration}) 24.10 0.37 40.09 32.80 20.41 -7.21 Fibrinogen Prevalence (Ω ^{dimension}) 3.30 2.99 2.42 1.86 2.68 2.50 Duration (Ω ^{duration}) 3.44 6.01 6.69 -0.90 -3.73 1.97 HbA1c Prevalence (Ω ^{dimension}) 1.25 1.45 1.99 1.68 5.36 16.60 Duration (Ω ^{duration}) 0.41 2.93 1.32 -0.18 -4.22 -10.60 Cholesterol Prevalence (Ω ^{dimension}) -0.50 1.07 4.45 4.62 6.63 8.80 Ratio Q ^{duration} 0.71 1.43 8.73 8.77 18.54 20.91 Panel B - Multidimensional deprivation (MDD) vith persistence	Resting	Prevalence ($\Omega^{dimension}$)	1.70	1.94	1.98	1.89	2.88	2.70
C-Reactive Prevalence $(\Omega^{dimension})$ 17.67 -1.98 12.02 23.21 28.94 55.40 Protein Duration $(\Omega^{duration})$ 24.10 0.37 40.09 32.80 20.41 -7.21 Fibrinogen Prevalence $(\Omega^{dimension})$ 3.30 2.99 2.42 1.86 2.68 2.50 Duration $(\Omega^{duration})$ 3.44 6.01 6.69 -0.90 -3.73 1.97 HbA1c Prevalence $(\Omega^{dimension})$ 1.25 1.45 1.99 1.68 5.36 16.60 Duration $(\Omega^{duration})$ 0.41 2.93 1.32 -0.18 -4.22 -10.60 Cholesterol Prevalence $(\Omega^{dimension})$ -0.50 1.07 4.45 4.62 6.63 8.80 Ratio $\Omega^{duration}$ 0.71 1.43 8.73 8.77 18.54 20.91 Panel B - Multidimensional deprivation (MDD) with persistence Deprivation Quantile of health measures Biomarker Component q(10) <td< td=""><td>heart rate</td><td>Duration $(\Omega^{duration})$</td><td>2.78</td><td>1.02</td><td>4.83</td><td>4.00</td><td>5.24</td><td>5.04</td></td<>	heart rate	Duration $(\Omega^{duration})$	2.78	1.02	4.83	4.00	5.24	5.04
Fibrinogen Prevalence (Ωdimension) 3.30 2.99 2.42 1.86 2.68 2.50 Duration (Ωduration) 3.44 6.01 6.69 -0.90 -3.73 1.97 HbA1c Prevalence (Ωdimension) 1.25 1.45 1.99 1.68 5.36 16.60 Duration (Ωduration) 0.41 2.93 1.32 -0.18 -4.22 -10.60 Cholesterol Prevalence (Ωdimension) -0.50 1.07 4.45 4.62 6.63 8.80 Ratio Ωduration 0.71 1.43 8.73 8.77 18.54 20.91 Panel B - Multidimensional deprivation (MDD) with persistence Deprivation Quantile of health measures Biomarker Component Q(10) q(25) q(50) q(75) q(90) q(95) BMI Prevalence (Ωdimension) -0.21 0.16 0.44 0.81 2.69 3.29 Waist Prevalence (Ωdimension) 0.32 -0.29 0.48 0.48<	C-Reactive	Prevalence ($\Omega^{dimension}$)	17.67	-1.98	12.02	23.21	28.94	55.40
Fibrinogen Prevalence (Ωdimension) 3.30 2.99 2.42 1.86 2.68 2.50 Duration (Ωduration) 3.44 6.01 6.69 -0.90 -3.73 1.97 HbA1c Prevalence (Ωdimension) 1.25 1.45 1.99 1.68 5.36 16.60 Duration (Ωduration) 0.41 2.93 1.32 -0.18 -4.22 -10.60 Cholesterol Prevalence (Ωdimension) -0.50 1.07 4.45 4.62 6.63 8.80 Ratio Ωduration 0.71 1.43 8.73 8.77 18.54 20.91 Panel B - Multidimensional deprivation (MDD) with persistence Deprivation Quantile of health measures Biomarker Component Q(10) q(25) q(50) q(75) q(90) q(95) BMI Prevalence (Ωdimension) -0.21 0.16 0.44 0.81 2.69 3.29 Waist Prevalence (Ωdimension) 0.32 -0.29 0.48 0.48<	Protein	Duration $(\Omega^{duration})$	24.10	0.37	40.09	32.80	20.41	-7.21
Duration (Ω ^{duration}) 3.44 6.01 6.69 -0.90 -3.73 1.97 HbA1c Prevalence (Ω ^{dimension}) 1.25 1.45 1.99 1.68 5.36 16.60 Duration (Ω ^{duration}) 0.41 2.93 1.32 -0.18 -4.22 -10.60 Cholesterol Prevalence (Ω ^{dimension}) -0.50 1.07 4.45 4.62 6.63 8.80 Ratio Ω ^{duration} 0.71 1.43 8.73 8.77 18.54 20.91	Fibrinogen	Prevalence ($\Omega^{dimension}$)	3.30	2.99	2.42	1.86	2.68	2.50
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$		Duration $(\Omega^{duration})$	3.44	6.01	6.69	-0.90	-3.73	1.97
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	HbA1c	Prevalence ($\Omega^{dimension}$)	1.25	1.45	1.99	1.68	5.36	16.60
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$		Duration $(\Omega^{duration})$	0.41	2.93	1.32	-0.18	-4.22	-10.60
Ratio Ω ^{duration} 0.71 1.43 8.73 8.77 18.54 20.91 Panel B - Multidimensional deprivation (MDD) with persistence Deprivation Quantile of health measures Biomarker Component q(10) q(25) q(50) q(75) q(90) q(95) BMI Prevalence (Ω ^{dimension}) -0.21 0.16 0.44 0.81 2.69 3.29 Persistence (Ω ^{persistence}) 0.29 3.48 3.05 1.24 3.30 2.28 Waist Prevalence (Ω ^{dimension}) 0.32 -0.29 0.08 0.48 1.47 2.48 Circumference Persistence (Ω ^{persistence}) 4.89 5.21 1.85 -0.11 1.77 1.45 Diastolic Prevalence (Ω ^{dimension}) -0.16 0.25 0.08 -0.46 0.09 -0.01 Blood Pressure Persistence (Ω ^{persistence}) -0.42 1.14 1.12 1.98 1.89 3.28 Systolic Pe	Cholesterol	Prevalence ($\Omega^{dimension}$)	-0.50	1.07	4.45	4.62	6.63	8.80
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	Ratio	$\Omega^{duration}$	0.71	1.43	8.73	8.77	18.54	20.91
$\begin{array}{ c c c c c c c c c c c c c c c c c c c$	Pa	anel ${f B}$ - Multidimension	ıal depr	ivation	(MDD)	with p	ersistence	e
BMI Prevalence (Ω ^{dimension}) -0.21 0.16 0.44 0.81 2.69 3.29 Persistence (Ω ^{persistence}) 0.29 3.48 3.05 1.24 3.30 2.28 Waist Prevalence (Ω ^{dimension}) 0.32 -0.29 0.08 0.48 1.47 2.48 Circumference Persistence (Ω ^{persistence}) 4.89 5.21 1.85 -0.11 1.77 1.45 Diastolic Prevalence (Ω ^{dimension}) -0.16 0.25 0.08 -0.46 0.09 -0.01 Blood Pressure Persistence (Ω ^{persistence}) -0.42 1.14 1.12 1.98 1.89 3.28 Systolic Prevalence (Ω ^{dimension}) 0.48 -0.16 -0.16 0.00 0.45 0.28 Blood Pressure Persistence (Ω ^{persistence}) 1.18 0.30 -0.15 0.18 1.02 1.05 Resting Prevalence (Ω ^{dimension}) 0.75 1.19 0.05 0.64 1.59 2.70 heart rate Persiste		Deprivation		•		of healtl		es
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	Biomarker		$\mathbf{q}(10)$	$\mathbf{q}(25)$	$\mathbf{q}(50)$	$\mathbf{q}(75)$	$\mathbf{q}(90)$	${f q(95)}$
Waist Prevalence (Ω ^{dimension}) 0.32 -0.29 0.08 0.48 1.47 2.48 Circumference Persistence (Ω ^{persistence}) 4.89 5.21 1.85 -0.11 1.77 1.45 Diastolic Prevalence (Ω ^{dimension}) -0.16 0.25 0.08 -0.46 0.09 -0.01 Blood Pressure Persistence (Ω ^{persistence}) -0.42 1.14 1.12 1.98 1.89 3.28 Systolic Prevalence (Ω ^{dimension}) 0.48 -0.16 -0.16 0.00 0.45 0.28 Blood Pressure Persistence (Ω ^{persistence}) 1.18 0.30 -0.15 0.18 1.02 1.05 Resting Prevalence (Ω ^{dimension}) 0.75 1.19 0.05 0.64 1.59 2.70 heart rate Persistence (Ω ^{persistence}) 2.31 0.84 4.63 3.95 4.30 2.78 C-Reactive Prevalence (Ω ^{dimension}) 6.36 12.09 3.89 11.60 19.49 37.65 <t< td=""><td>BMI</td><td>Prevalence $(\Omega^{dimension})$</td><td>-0.21</td><td>0.16</td><td>0.44</td><td>0.81</td><td>2.69</td><td>3.29</td></t<>	BMI	Prevalence $(\Omega^{dimension})$	-0.21	0.16	0.44	0.81	2.69	3.29
Circumference Persistence (Ωpersistence) 4.89 5.21 1.85 -0.11 1.77 1.45 Diastolic Prevalence (Ωdimension) -0.16 0.25 0.08 -0.46 0.09 -0.01 Blood Pressure Persistence (Ωpersistence) -0.42 1.14 1.12 1.98 1.89 3.28 Systolic Prevalence (Ωdimension) 0.48 -0.16 -0.16 0.00 0.45 0.28 Blood Pressure Persistence (Ωpersistence) 1.18 0.30 -0.15 0.18 1.02 1.05 Resting Prevalence (Ωdimension) 0.75 1.19 0.05 0.64 1.59 2.70 heart rate Persistence (Ωpersistence) 2.31 0.84 4.63 3.95 4.30 2.78 C-Reactive Prevalence (Ωdimension) 6.36 12.09 3.89 11.60 19.49 37.65 Protein Persistence (Ωpersistence) 10.60 47.04 37.56 34.46 28.57 27.93 Fibrin		Persistence ($\Omega^{persistence}$)	0.29	3.48	3.05	1.24	3.30	2.28
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	Waist	Prevalence $(\Omega^{dimension})$	0.32	-0.29	0.08	0.48	1.47	2.48
Blood Pressure Persistence (Ω ^{persistence}) -0.42 1.14 1.12 1.98 1.89 3.28 Systolic Prevalence (Ω ^{dimension}) 0.48 -0.16 -0.16 0.00 0.45 0.28 Blood Pressure Persistence (Ω ^{persistence}) 1.18 0.30 -0.15 0.18 1.02 1.05 Resting Prevalence (Ω ^{dimension}) 0.75 1.19 0.05 0.64 1.59 2.70 heart rate Persistence (Ω ^{persistence}) 2.31 0.84 4.63 3.95 4.30 2.78 C-Reactive Prevalence (Ω ^{dimension}) 6.36 12.09 3.89 11.60 19.49 37.65 Protein Persistence (Ω ^{persistence}) 10.60 47.04 37.56 34.46 28.57 27.93 Fibrinogen Prevalence (Ω ^{dimension}) 2.04 2.03 1.33 0.83 1.89 2.70 AbAlt Prevalence (Ω ^{dimension}) 0.98 0.97 1.24 1.58 4.60 17.59 P	Circumference	Persistence ($\Omega^{persistence}$)	4.89	5.21	1.85	-0.11	1.77	1.45
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	Diastolic	Prevalence $(\Omega^{dimension})$	-0.16	0.25	0.08	-0.46	0.09	-0.01
Blood Pressure Persistence (Ω ^{persistence}) 1.18 0.30 -0.15 0.18 1.02 1.05 Resting Prevalence (Ω ^{dimension}) 0.75 1.19 0.05 0.64 1.59 2.70 heart rate Persistence (Ω ^{persistence}) 2.31 0.84 4.63 3.95 4.30 2.78 C-Reactive Prevalence (Ω ^{dimension}) 6.36 12.09 3.89 11.60 19.49 37.65 Protein Persistence (Ω ^{persistence}) 10.60 47.04 37.56 34.46 28.57 27.93 Fibrinogen Prevalence (Ω ^{dimension}) 2.04 2.03 1.33 0.83 1.89 2.70 Persistence (Ω ^{persistence}) 4.05 4.74 5.86 0.43 -1.28 3.13 HbA1c Prevalence (Ω ^{dimension}) 0.98 0.97 1.24 1.58 4.60 17.59 Persistence (Ω ^{dimension}) 0.57 3.10 1.79 0.56 -4.47 -15.34 Cholesterol Prevalence (Ω ^{dimension}) <td>Blood Pressure</td> <td></td> <td>-0.42</td> <td>1.14</td> <td>1.12</td> <td>1.98</td> <td>1.89</td> <td>3.28</td>	Blood Pressure		-0.42	1.14	1.12	1.98	1.89	3.28
Resting heart rate Prevalence (Ω ^{dimension}) 0.75 1.19 0.05 0.64 1.59 2.70 heart rate Persistence (Ω ^{persistence}) 2.31 0.84 4.63 3.95 4.30 2.78 C-Reactive Prevalence (Ω ^{dimension}) 6.36 12.09 3.89 11.60 19.49 37.65 Protein Persistence (Ω ^{persistence}) 10.60 47.04 37.56 34.46 28.57 27.93 Fibrinogen Prevalence (Ω ^{dimension}) 2.04 2.03 1.33 0.83 1.89 2.70 Persistence (Ω ^{persistence}) 4.05 4.74 5.86 0.43 -1.28 3.13 HbA1c Prevalence (Ω ^{dimension}) 0.98 0.97 1.24 1.58 4.60 17.59 Persistence (Ω ^{dimension}) 0.57 3.10 1.79 0.56 -4.47 -15.34 Cholesterol Prevalence (Ω ^{dimension}) -0.50 1.90 2.93 2.40 3.99 5.62	Systolic	Prevalence $(\Omega^{dimension})$	0.48	-0.16	-0.16	0.00	0.45	0.28
heart rate Persistence (Ω ^{persistence}) 2.31 0.84 4.63 3.95 4.30 2.78 C-Reactive Prevalence (Ω ^{dimension}) 6.36 12.09 3.89 11.60 19.49 37.65 Protein Persistence (Ω ^{persistence}) 10.60 47.04 37.56 34.46 28.57 27.93 Fibrinogen Prevalence (Ω ^{dimension}) 2.04 2.03 1.33 0.83 1.89 2.70 Persistence (Ω ^{persistence}) 4.05 4.74 5.86 0.43 -1.28 3.13 HbA1c Prevalence (Ω ^{dimension}) 0.98 0.97 1.24 1.58 4.60 17.59 Persistence (Ω ^{persistence}) 0.57 3.10 1.79 0.56 -4.47 -15.34 Cholesterol Prevalence (Ω ^{dimension}) -0.50 1.90 2.93 2.40 3.99 5.62	Blood Pressure	Persistence ($\Omega^{persistence}$)	1.18	0.30	-0.15	0.18	1.02	1.05
heart rate Persistence (Ω ^{persistence}) 2.31 0.84 4.63 3.95 4.30 2.78 C-Reactive Prevalence (Ω ^{dimension}) 6.36 12.09 3.89 11.60 19.49 37.65 Protein Persistence (Ω ^{persistence}) 10.60 47.04 37.56 34.46 28.57 27.93 Fibrinogen Prevalence (Ω ^{dimension}) 2.04 2.03 1.33 0.83 1.89 2.70 Persistence (Ω ^{persistence}) 4.05 4.74 5.86 0.43 -1.28 3.13 HbA1c Prevalence (Ω ^{dimension}) 0.98 0.97 1.24 1.58 4.60 17.59 Persistence (Ω ^{persistence}) 0.57 3.10 1.79 0.56 -4.47 -15.34 Cholesterol Prevalence (Ω ^{dimension}) -0.50 1.90 2.93 2.40 3.99 5.62	Resting	Prevalence $(\Omega^{dimension})$	0.75	1.19	0.05	0.64	1.59	2.70
Protein Persistence (Ω ^{persistence}) 10.60 47.04 37.56 34.46 28.57 27.93 Fibrinogen Prevalence (Ω ^{dimension}) 2.04 2.03 1.33 0.83 1.89 2.70 Persistence (Ω ^{persistence}) 4.05 4.74 5.86 0.43 -1.28 3.13 HbA1c Prevalence (Ω ^{dimension}) 0.98 0.97 1.24 1.58 4.60 17.59 Persistence (Ω ^{persistence}) 0.57 3.10 1.79 0.56 -4.47 -15.34 Cholesterol Prevalence (Ω ^{dimension}) -0.50 1.90 2.93 2.40 3.99 5.62	heart rate	Persistence ($\Omega^{persistence}$)	2.31	0.84	4.63	3.95	4.30	2.78
Protein Persistence (Ω ^{persistence}) 10.60 47.04 37.56 34.46 28.57 27.93 Fibrinogen Prevalence (Ω ^{dimension}) 2.04 2.03 1.33 0.83 1.89 2.70 Persistence (Ω ^{persistence}) 4.05 4.74 5.86 0.43 -1.28 3.13 HbA1c Prevalence (Ω ^{dimension}) 0.98 0.97 1.24 1.58 4.60 17.59 Persistence (Ω ^{persistence}) 0.57 3.10 1.79 0.56 -4.47 -15.34 Cholesterol Prevalence (Ω ^{dimension}) -0.50 1.90 2.93 2.40 3.99 5.62	C-Reactive	Prevalence ($\Omega^{dimension}$)	6.36	12.09	3.89	11.60	19.49	37.65
Persistence (Ω ^{persistence}) 4.05 4.74 5.86 0.43 -1.28 3.13 HbA1c Prevalence (Ω ^{dimension}) 0.98 0.97 1.24 1.58 4.60 17.59 Persistence (Ω ^{persistence}) 0.57 3.10 1.79 0.56 -4.47 -15.34 Cholesterol Prevalence (Ω ^{dimension}) -0.50 1.90 2.93 2.40 3.99 5.62	Protein	Persistence ($\Omega^{persistence}$)	10.60	47.04	37.56	34.46	28.57	27.93
HbA1c Prevalence ($\Omega^{dimension}$) 0.98 0.97 1.24 1.58 4.60 17.59 Persistence ($\Omega^{persistence}$) 0.57 3.10 1.79 0.56 -4.47 -15.34 Cholesterol Prevalence ($\Omega^{dimension}$) -0.50 1.90 2.93 2.40 3.99 5.62	Fibrinogen	Prevalence ($\Omega^{dimension}$)	2.04	2.03	1.33	0.83	1.89	2.70
HbA1c Prevalence ($\Omega^{dimension}$) 0.98 0.97 1.24 1.58 4.60 17.59 Persistence ($\Omega^{persistence}$) 0.57 3.10 1.79 0.56 -4.47 -15.34 Cholesterol Prevalence ($\Omega^{dimension}$) -0.50 1.90 2.93 2.40 3.99 5.62		Persistence $(\Omega^{persistence})$	4.05	4.74	5.86	0.43	-1.28	3.13
Cholesterol Prevalence $(\Omega^{dimension})$ -0.50 1.90 2.93 2.40 3.99 5.62	HbA1c	Prevalence ($\Omega^{dimension}$)	0.98	0.97	1.24	1.58	4.60	17.59
Cholesterol Prevalence ($\Omega^{dimension}$) -0.50 1.90 2.93 2.40 3.99 5.62 Ratio Persistence ($\Omega^{persistence}$) 2.45 1.02 8.01 8.59 14.18 12.24		Persistence ($\Omega^{persistence}$)	0.57	3.10	1.79	0.56	-4.47	-15.34
Ratio Persistence ($\Omega^{persistence}$) 2.45 1.02 8.01 8.59 14.18 12.24	Cholesterol	Prevalence $(\Omega^{dimension})$	-0.50	1.90	2.93	2.40	3.99	5.62
, ,	Ratio	Persistence $(\Omega^{persistence})$	2.45	1.02	8.01	8.59	14.18	12.24

Author's calculations using UKLHS dataset (1999-2008). N=5,707. Calculations based on Equation 6. The measure of multidimensional deprivation used for this table includes income deprivation as an additional dimension, a total of 11 domains over 10 years.

^{*}All numbers are in percentage points.

References

- Acabchuk, R. L., J. Kamath, J. D. Salamone, and B. T. Johnson (2017). Stress and chronic illness: The inflammatory pathway. Social Science & Medicine 185, 166.
- Adams, P., M. D. Hurd, D. McFadden, A. Merrill, and T. Ribeiro (2003). Healthy, wealthy, and wise? tests for direct causal paths between health and socioeconomic status. <u>Journal</u> of Econometrics 112(1), 3–56.
- Alkire, S. (2002). Dimensions of human development. World Development 30(2), 181–205.
- Alkire, S., M. Apablaza, S. Chakravarty, and G. Yalonetzky (2017). Measuring chronic multidimensional poverty. Journal of Policy Modeling 39(6), 983–1006.
- Alkire, S. and J. Foster (2011). Counting and multidimensional poverty measurement.

 Journal of Public Economics 95(7), 476–487.
- Allanson, P., U.-G. Gerdtham, and D. Petrie (2010). Longitudinal analysis of income-related health inequality. Journal of Health Economics 29(1), 78–86.
- Allanson, P. and D. Petrie (2013). On the choice of health inequality measure for the longitudinal analysis of income-related health inequalities. <u>Health economics</u> <u>22</u>(3), 353–365.
- Atkinson, A. B. (2003). Multidimensional deprivation: contrasting social welfare and counting approaches. The Journal of Economic Inequality 1(1), 51–65.
- Bago d'Uva, T., O. O'Donnell, and E. Van Doorslaer (2008). Differential health reporting by education level and its impact on the measurement of health inequalities among older Europeans. International Journal of Epidemiology 37(6), 1375–1383.
- Banerjee, A., E. Duflo, N. Goldberg, D. Karlan, R. Osei, W. Parienté, J. Shapiro, B. Thuysbaert, and C. Udry (2015). A multifaceted program causes lasting progress for the very poor: Evidence from six countries. Science 348(6236).
- Bárcena-Martín, E., B. Lacomba, A. I. Moro-Egido, and S. Pérez-Moreno (2014). Country differences in material deprivation in e urope. Review of Income and Wealth 60(4), 802–820.

- Benzeval, M., A. Davillas, M. Kumari, and P. Lynn (2014). Understanding society: The UK Household Longitudinal Study Biomarker User Guide and Glossary. <u>Institute for Social</u> and Economic Research, University of Essex.
- Bilger, M. and V. Carrieri (2013). Health in the cities: When the neighborhood matters more than income. Journal of Health Economics 32(1), 1–11.
- Bilger, M., E. J. Kruger, and E. A. Finkelstein (2017). Measuring socioeconomic inequality in obesity: looking beyond the obesity threshold. Health Economics 26(8), 1052–1066.
- Blázquez, M., E. Cottini, and A. Herrarte (2014). The socioeconomic gradient in health: how important is material deprivation? The Journal of Economic Inequality 12(2), 239–264.
- Bloomer, E., J. Allen, A. Donkin, G. Findlay, and M. Gamsu (2012). The impact of the economic downturn and policy changes on health inequalities in London. <u>London: UCL</u> Institute of Health Equity.
- Böckerman, P., A. Bryson, J. Viinikainen, C. Hakulinen, L. Pulkki-Råback, O. Raitakari, and J. Pehkonen (2017). Biomarkers and long-term labour market outcomes: The case of creatine. Journal of Economic Behavior & Organization 142, 259–274.
- Bossert, W., S. R. Chakravarty, and C. D'Ambrosio (2013). Multidimensional poverty and material deprivation with discrete data. Review of Income and Wealth 59(1), 29–43.
- Bossert, W., S. R. Chakravarty, and C. d'Ambrosio (2019). Poverty and time. In <u>Poverty</u>, Social Exclusion and Stochastic Dominance, pp. 63–82. Springer.
- Bossert, W. and C. D'Ambrosio (2019). Intertemporal material deprivation: A proposal and an application to eu countries. In <u>Deprivation</u>, <u>Inequality and Polarization</u>, pp. 15–35. Springer.
- Bourguignon, F. and S. R. Chakravarty (2003). The measurement of multidimensional poverty. Volume 1, pp. 25–49. Springer.
- Braveman, P. A., C. Cubbin, S. Egerter, S. Chideya, K. S. Marchi, M. Metzler, and S. Posner (2005). Socioeconomic status in health research: one size does not fit all. <u>JAMA</u> <u>294</u>(22), 2879–2888.

- Buchinsky, M. (1998). Recent advances in quantile regression models: a practical guideline for empirical research. Journal of Human Resources 33(1), 88–126.
- Carrieri, V. and A. M. Jones (2017). The income-health relationship 'beyond the mean': New evidence from biomarkers. Health Economics 26(7), 937–956.
- Caul, S. (2020). Deaths involving covid-19 by local area and socioeconomic deprivation: deaths occurring between 1 march and 31 may 2020. Office of National Statistics, United Kingdom (UK).
- Clarke, P. and G. Erreygers (2020). Defining and measuring health poverty. <u>Social Science</u> & Medicine 244, 112633.
- Cohen, A. K., M. Rai, D. H. Rehkopf, and B. Abrams (2013). Educational attainment and obesity: a systematic review. Obesity Reviews 14(12), 989–1005.
- Costa-Font, J. and C. Hernández-Quevedo (2012). Measuring inequalities in health: what do we know? what do we need to know? Health Policy 106(2), 195–206.
- D'Ambrosio, C. (2016). An overview of intertemporal measures of individual well-being: Can they explain life satisfaction better? In <u>Advances in Happiness Research</u>, pp. 39–54. Springer.
- D'Ambrosio, C., W. Bossert, and S. Chakravarty (2012). Poverty and time. <u>Journal of</u> Economic Inequality 10, 145–162.
- Datt, G. (2019). Distribution-sensitive multidimensional poverty measures. <u>The World Bank</u> <u>Economic Review</u> 33(3), 551–572.
- Davillas, A., M. Benzeval, and M. Kumari (2017). Socio-economic inequalities in c-reactive protein and fibrinogen across the adult age span: Findings from understanding society. Scientific Reports 7(1), 1–13.
- Davillas, A. and A. M. Jones (2020). Ex ante inequality of opportunity in health, decomposition and distributional analysis of biomarkers. Journal of Health Economics 69, 102251.
- Davillas, A. and S. Pudney (2020). Using biomarkers to predict healthcare costs: Evidence from a uk household panel. <u>Journal of Health Economics</u> 73, 102356.

- Deaton, A. S. and C. H. Paxson (1998). Aging and inequality in income and health. <u>The</u> American Economic Review 88(2), 248–253.
- Dickerson, A. and G. K. Popli (2016). Persistent poverty and children's cognitive development: evidence from the UK millennium cohort study. <u>Journal of the Royal Statistical</u> Society: Series A (Statistics in Society) 179(2), 535–558.
- Dowd, J. B. and A. Zajacova (2010). Does self-rated health mean the same thing across socioeconomic groups? evidence from biomarker data. Annals of Epidemiology 20(10), 743–749.
- Dutta, I., L. Roope, and H. Zank (2013). On intertemporal poverty measures: the role of affluence and want. Social Choice and Welfare 41(4), 741–762.
- Fahey, T. (2007). The case for an eu-wide measure of poverty. <u>European Sociological</u> Review 23(1), 35–47.
- Finn, D. and J. Goodship (2014). Benefits take-up and poverty (chapter) in reducing poverty in the UK: a collection of evidence reviews. Joseph Rowntree Foundation.
- Firpo, S., N. M. Fortin, and T. Lemieux (2009). Unconditional quantile regressions. Econometrica 77(3), 953–973.
- Foster, J., J. Greer, and E. Thorbecke (1984). A class of decomposable poverty measures. Econometrica: Journal of the Econometric Society, 761–766.
- Foster, J. E. (2009). A class of chronic poverty measures. <u>Poverty dynamics: interdisciplinary</u> perspectives, 59–76.
- Frijters, P., J. P. Haisken-DeNew, and M. A. Shields (2005). The causal effect of income on health: Evidence from German reunification. <u>Journal of Health Economics</u> <u>24</u>(5), 997–1017.
- Frijters, P., D. W. Johnston, M. A. Shields, and K. Sinha (2015). A lifecycle perspective of stock market performance and wellbeing. <u>Journal of Economic Behavior & Organization 112</u>, 237–250.

- Giordano, G. N. and M. Lindstrom (2010). The impact of changes in different aspects of social capital and material conditions on self-rated health over time: a longitudinal cohort study. Social science & medicine 70(5), 700–710.
- Glei, D. A., N. Goldman, V. M. Shkolnikov, D. Jdanov, S. Shalnova, M. Shkolnikova, and M. Weinstein (2013). To what extent do biomarkers account for the large social disparities in health in Moscow? Social Science & Medicine 77, 164–172.
- Gradín, C., O. Cantó, and C. del Río (2018). Poverty over time: empirical findings. In Handbook of Research on Economic and Social Well-Being. Edward Elgar Publishing.
- Gradín, C., C. Del Río, and O. Cantó (2012). Measuring poverty accounting for time. Review of Income and Wealth 58(2), 330–354.
- Graham, C. and S. Pettinato (2002). Frustrated achievers: Winners, losers and subjective well-being in new market economies. Journal of Development Studies 38(4), 100–140.
- Gruenewald, T. L., S. Cohen, K. A. Matthews, R. Tracy, and T. E. Seeman (2009). Association of socioeconomic status with inflammation markers in black and white men and women in the coronary artery risk development in young adults (CARDIA) study. <u>Social</u> Science & Medicine 69(3), 451–459.
- Hauck, K. and N. Rice (2004). A longitudinal analysis of mental health mobility in Britain. Health Economics 13(10), 981–1001.
- Holt-Lunstad, J., T. B. Smith, and J. B. Layton (2010). Social relationships and mortality risk: a meta-analytic review. PLoS Medicine 7(7), e1000316.
- Iacobucci, G. (2020). Covid-19: Deprived areas have the highest death rates in England and Wales.
- Ishii, S., A. S. Karlamangla, M. Bote, M. R. Irwin, D. R. Jacobs Jr, H. J. Cho, and T. E. Seeman (2012). Gender, obesity and repeated elevation of c-reactive protein: data from the CARDIA cohort. PloS One 7(4), e36062.

- Johnston, D. W., C. Propper, and M. A. Shields (2009). Comparing subjective and objective measures of health: Evidence from hypertension for the income/health gradient. <u>Journal</u> of Health Economics 28(3), 540–552.
- Jolliffe, D. (2011). Overweight and poor? on the relationship between income and the body mass index. Economics & Human Biology 9(4), 342–355.
- Jones, A. M. and J. Wildman (2008). Health, income and relative deprivation: Evidence from the BHPS. Journal of Health Economics 27(2), 308–324.
- Jürges, H. (2007). True health vs response styles: exploring cross-country differences in self-reported health. Health Economics 16(2), 163–178.
- Jürges, H., E. Kruk, and S. Reinhold (2013). The effect of compulsory schooling on health—evidence from biomarkers. Journal of Population Economics 26(2), 645–672.
- Kim, J. and E. Durden (2007). Socioeconomic status and age trajectories of health. <u>Social</u> Science & Medicine 65(12), 2489–2502.
- Lindelow, M. (2006). Sometimes more equal than others: how health inequalities depend on the choice of welfare indicator. Health economics 15(3), 263–279.
- Longhi, S. and A. Nandi (2014). A practical guide to using panel data. Sage.
- Loucks, E. B., J. W. Lynch, L. Pilote, R. Fuhrer, N. D. Almeida, H. Richard, G. Agha, J. M. Murabito, and E. J. Benjamin (2009). Life-course socioeconomic position and incidence of coronary heart disease: the Framingham Offspring Study. <u>American Journal of Epidemiology</u> 169(7), 829–836.
- Mackenbach, J. P. (2011). The english strategy to reduce health inequalities. <u>The Lancet 377(9782)</u>, 1986–1988.
- Makdissi, P., D. Sylla, and M. Yazbeck (2013). Decomposing health achievement and socioeconomic health inequalities in presence of multiple categorical information. <u>Economic Modelling</u> 35, 964–968.
- Marmot, M. (2020). Health equity in England: the Marmot review 10 years on. BMJ 368

- Millán, J., X. Pintó, A. Muñoz, M. Zúñiga, J. Rubiés-Prat, L. F. Pallardo, L. Masana, A. Mangas, A. Hernández-Mijares, P. González-Santos, et al. (2009). Lipoprotein ratios: physiological significance and clinical usefulness in cardiovascular prevention. <u>Vascular</u> Health and Risk Management 5, 757.
- Nicholas, A. and R. Ray (2012). Duration and persistence in multidimensional deprivation: Methodology and australian application. Economic Record 88(280), 106–126.
- Nicholas, A., R. Ray, and K. Sinha (2019). Differentiating between dimensionality and duration in multidimensional measures of poverty: Methodology with an application to China. Review of Income and Wealth 65(1), 48–74.
- OECD (2013). Oecd framework for statistics on the distribution of household income, consumption and wealth.
- Pickett, K. E. and R. G. Wilkinson (2015). Income inequality and health: a causal review. Social Science & Medicine 128, 316–326.
- Ray, R. and K. Sinha (2015). Multidimensional deprivation in China, India and Vietnam: a comparative study on micro data. <u>Journal of Human Development and Capabilities</u> <u>16</u>(1), 69–93.
- Shorrocks, A. F. (2013). Decomposition procedures for distributional analysis: a unified framework based on the shapley value. <u>The Journal of Economic Inequality</u> <u>11(1)</u>, 99–126.
- Stiglitz, J. E., A. Sen, J.-P. Fitoussi, et al. (2010). Report by the commission on the measurement of economic performance and social progress. Technical report, Paris: Commission on the Measurement of Economic Performance and Social Progress.
- Whelan, C. T. and B. Maître (2012). Understanding material deprivation: A comparative european analysis. Research in Social Stratification and Mobility 30(4), 489–503.
- WHO (2011). Use of glycated haemoglobin (HbA1c) in diagnosis of diabetes mellitus: abbreviated report of a WHO consultation. Technical report, World Health Organisation.

6 Appendix

Table A1: Descriptive statistics for health measures (UKHLS, Wave 3 (2010-12))

Health variables	Mean	q(10)	q(25)	q(50)	q(75)	q(95)
Nurse-measured biomarkers						
BMI (Kg/m ²)	28.75	22.50	25.00	27.95	31.68	39.07
Waist Circumference (cm)	96.50	78.25	86.30	95.75	105.70	121.95
Systolic blood pressure (mmhg)	128.54	107.50	116.75	127.00	139.00	159.00
Diastolic blood pressure (mmhg)	73.54	60.00	66.00	73.00	81.00	92.50
Resting heart rate (bpm)	68.80	55.50	61.00	68.00	75.50	89.00
Blood based biomakers						
C-Reactive Protein (mg/l)	3.31	0.40	0.70	1.50	3.20	11.50
Fibrinogen (g/l)	2.88	2.20	2.50	2.80	3.20	4.00
${ m HbA1C} \; ({ m mmol/mol})$	38.30	32.00	34.00	37.00	40.00	52.00
Cholesterol Ratio (TC:HDL)	3.84	2.38	2.87	3.57	4.53	6.45

Author's calculations based on UKHLS dataset. N=5,707 observations.

Table A2: Description of dimensions of multidimensional deprivation in the UK^*

Domain	Dimensions for each domain	Description
Education	Low level of formal education	1 if respondent is uneducated;
	of respondent or household head	or highest level is less than high school;
		0 higher than high school (A-level).
Economic	Employment status	1 if individual is unemployed / retired/
Activity	of individual	${ m carer/student/longtime\ sick\ and}$
		no other household member working.
		0 if individual is employed/self employed,
		or at least one member working.
Housing	Shortage of space	1 if yes, 0 otherwise
Conditions	Not enough light	1 if yes, 0 otherwise
	Lack of adequate heating	1 if yes, 0 otherwise
	Damp walls, floors	1 if yes, 0 otherwise
	Does not have separate bathroom	1 if yes, 0 otherwise
	No central heating	1 if yes, 0 otherwise
Afford Consumer	Lack: video recorder/dvd player	1 if yes, 0 otherwise
durables	Lack: deep freeze or fridge freezer	1 if yes, 0 otherwise
	Lack: washing machine	1 if yes, 0 otherwise
	Lack: tumble drier	1 if yes, 0 otherwise
	Lack: dishwasher	1 if yes, 0 otherwise
	Lack: home computer/pc	1 if yes, 0 otherwise
	Lack: satellite dish/ sky television	1 if yes, 0 otherwise
	Lack: cable television	1 if yes, 0 otherwise
Car ownership	No car available in the household	1 if yes, 0 otherwise
Affordable	Can not afford to replace furniture	1 if yes, 0 otherwise
Lifestyle	Can not afford feed visitors once a month	1 if yes, 0 otherwise
	Can not afford keep house well decorated	1 if yes, 0 otherwise
Financial	Been over two months late with rent	1 if yes, 0 otherwise
hardship	Housing payment required cutback	1 if yes, 0 otherwise
_	Cannot afford to pay for annual holiday	1 if yes, 0 otherwise
Social	Frequency of talking to neighbours	1 if twice a month or less.
engagement		0 if once a week or most days.
	Frequency of meeting people	1 if twice a month or less.
		0 if once a week or most days.
Environment	Pollution/environmental problems	1 if yes, 0 otherwise
	Noise from neighbours	1 if yes, 0 otherwise
Security	Vandalism or crime in neighbourhood	1 if yes, 0 otherwise
Income	Income below threshold	1 if income is less than 60% of median
		household income,
		0 otherwise

^{*} Based on British Household Panel Survey Wave 9-18.

 $\begin{tabular}{ll} Table A3: Description and summary statistics for the covariates used in the health regression models \\ \end{tabular}$

Variable	Description	Mean	Standard Deviation
Age (years)	Age (15-19)	0.01	0.08
	Age (20-24)	0.03	0.16
	Age (25-29)	0.05	0.22
	Age (30-34)	0.08	0.26
	Age $(35-39)$	0.10	0.30
	Age (40-44)	0.12	0.32
	Age (45-49)	0.11	0.31
	Age (50-54)	0.10	0.30
	Age (55-59)	0.10	0.30
	Age (60-64)	0.09	0.28
	Age (65-69)	0.08	0.26
	Age (70-74)	0.06	0.24
	Age (75-79)	0.05	0.21
	Age (80-84)	0.03	0.16
	Age (85+)	0.01	0.11
Gender	Male	0.46	0.50
Race	White	0.98	0.15
Marital Status	Single	0.10	0.30
	Married	0.75	0.43
	Separated/Divorced	0.08	0.26
	Widowed	0.07	0.26
Region	North East	0.03	0.18
	North West	0.09	0.29
	Yorkshire and Humber	0.07	0.26
	East Midlands	0.07	0.25
	West Midlands	0.06	0.24
	East of England	0.07	0.26
	London	0.05	0.21
	South East	0.10	0.30
	South West	0.07	0.25
	Wales	0.19	0.39
	Scotland	0.20	0.40
Household characteristics	Household size	2.71	1.28
	Number of kids	0.54	0.92
Household type	Lone parent	0.03	0.18
irodsenora type	Couple: with children	0.28	0.45
	Couple: without children	0.48	0.50
	Single: non elderly	$0.40 \\ 0.09$	$0.30 \\ 0.28$
	Single: elderly	0.09	$0.28 \\ 0.29$
	Other: group households	$0.09 \\ 0.02$	$0.29 \\ 0.12$
	Multiple family households	0.02 0.01	$0.12 \\ 0.11$
Observations		707	0.11

Author's calculations based on UKHLS wave 3 dataset.

Table A4: Income and deprivation gradient in biomarkers at mean and quantiles in the UK

Dependent	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Variable	Variables	OLS	q(10)	q(25)	q(50)	q(75)	q(95)
BMI	ln(income)	-0.478*	-0.113	-0.381	-0.501	-0.979**	-2.408***
		(0.278)	(0.327)	(0.293)	(0.335)	(0.452)	(0.744)
	Deprivation (Ω)	-0.211	-5.827*	-1.954	-2.466	-4.075	10.08
	(MND)	(2.779)	(3.266)	(2.889)	(3.349)	(4.437)	(8.918)
	,	,	N = 2626	,	,	,	,
Waist	ln(income)	-1.704**	-1.657	-1.187	-1.661*	-2.452**	-1.580
Circumference	,	(0.678)	(1.046)	(0.887)	(0.897)	(0.963)	(1.758)
	Deprivation (Ω)	$2.564^{'}$	-6.869	-3.956	-8.934	-6.087	45.61**
	(MND)	(6.702)	(10.27)	(8.721)	(8.915)	(9.502)	(20.89)
	()	()	N=2548	()	()	()	(====)
Systolic BP	ln(income)	0.310	0.761	0.361	1.464	0.847	-4.506*
		(0.913)	(1.240)	(1.075)	(1.129)	(1.399)	(2.475)
	Deprivation (Ω)	-2.358	6.595	-6.357	2.798	-0.783	-4.046
	(MND)	(9.257)	(12.95)	(11.09)	(11.69)	(14.48)	(28.94)
	,	,	N=2141	,	,	,	,
Diasotic BP	ln(income)	0.581	1.360	0.565	0.821	0.173	0.517
	,	(0.598)	(1.025)	(0.833)	(0.765)	(0.869)	(1.432)
	Deprivation (Ω)	2.070	1.758	-0.543	1.082	-0.892	3.128
	(MND)	(6.065)	(11.45)	(8.359)	(7.832)	(8.750)	(17.66)
	(111112)	(0.000)	N=2141	(0.000)	(1.002)	(0.100)	(11.00)
Resting	ln(income)	-1.114*	1.211	0.133	-1.836**	-1.690*	-2.901
heart rate	,	(0.628)	(0.839)	(0.685)	(0.785)	(0.973)	(1.845)
	Deprivation (Ω)	16.35**	16.20**	14.13**	$8.299^{'}$	15.59	36.33*
	(MND)	(6.366)	(7.897)	(6.339)	(7.953)	(10.29)	(19.91)
	,	,	N=2145	,	,	,	,
C-Reactive	ln(income)	-1.043**	-0.0785	-0.103	-0.246*	-0.804***	-4.433*
Protein	,	(0.446)	(0.0719)	(0.0726)	(0.127)	(0.304)	(2.277)
	Deprivation (Ω)	4.851	1.061*	0.488	$0.280^{'}$	5.481*	$33.82^{'}$
	(MND)	(4.526)	(0.614)	(0.719)	(1.270)	(3.280)	(24.89)
	()	(=)	N=1777	()	(=:=:-)	()	(==:::)
Fibrinogen	ln(income)	-0.142***	-0.127**	-0.115***	-0.121***	-0.168***	-0.185*
		(0.0354)	(0.0549)	(0.0414)	(0.0419)	(0.0480)	(0.0999)
	Deprivation (Ω)	0.0850	0.358	0.517	0.218	-0.333	0.466
	(MND)	(0.359)	(0.478)	(0.390)	(0.427)	(0.519)	(1.191)
	,	,	N = 1767	,	, ,	,	,
HbA1c	ln(income)	-1.280**	0.259	0.0960	-1.150***	-0.733*	-5.520
	,	(0.540)	(0.345)	(0.296)	(0.297)	(0.442)	(4.337)
	Deprivation (Ω)	$8.072^{'}$	8.418**	11.68***	$\dot{5}.157^{st}$	6.802	$41.02^{'}$
	(MND)	(5.534)	(3.357)	(3.034)	(2.983)	(4.917)	(52.67)
	()	,	N=1683	()	()	()	,
Cholesterol	ln(income)	-0.304***	-0.0566	-0.176**	-0.287***	-0.356***	-0.962***
Ratio	()	(0.0801)	(0.0707)	(0.0747)	(0.0859)	(0.122)	(0.323)
	Deprivation (Ω)	0.296	-0.839	-0.796	0.753	0.633	0.653
	(MND)	(0.812)	(0.787)	(0.814)	(0.923)	(1.308)	(3.396)
	(-1,11,17)	(0.012)	(0.101)	(0.014)	(0.020)	(1.000)	(0.000)

Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1

Source: UKHLS Wave 3.

Table A5: Summary statistics for the deprivation dimensions

Deprivation domain	Mean	Min	Max					
Income	0.18	0	1					
Education	0.15	0	1					
Economic activity	0.27	0	1					
Housing conditions	0.28	0	1					
Afford consumer durables	0.99	0	1					
Car ownership	0.13	0	1					
Affordable lifestyle	0.12	0	1					
Financial hardship	0.04	0	1					
Social engagement	0.30	0	1					
Environment	0.14	0	1					
Security	0.15	0	1					
Observations		57,070)					
Source: British Household Panel Survey Wave 9-18.								

Table A6: Correlation coefficients across deprivation domains (All years)

Deprivation	Income	Education	Economic	Housing	Afford	Car	Affordable	Financial	Social	Env.	Security
Dimension			activity	conditions	cons. dur.	owner	lifestyle	hardship	eng.		
Income	1.0000										
Education	0.1915**	1.0000									
Ladouton	(0.0000)	1.0000									
Econ. activity	0.4336*	0.1839^{\star}	1.0000								
Boom, accining	(0.0000)	(0.0000)	1.000								
Housing cond.	0.0145*	0.0132*	-0.0869*	1.0000							
110 3.2.11.6	(0.0005)	(0.0016)	(0.0000)	<u> </u>							
Afford cons. dur.	0.0204*	0.0076	0.0405*	0.0135^{\star}	1.0000						
	(0.0000)	(0.0692)	(0.0000)	(0.0013)							
Car ownership	0.2784*	0.1490*	0.3173*	0.0381*	0.0251^{\star}	1.0000					
=	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0000)						
Aff. lifestyle	0.1822^{\star}	0.0833*	0.0596^{\star}	0.1779*	0.0201*	0.1743^{\star}	1.0000				
-	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0000)					
Fin. hardship	0.0509^{\star}	0.0281*	-0.0381*	0.1069*	0.0090**	0.0452^{\star}	0.2449^{\star}	1.0000			
	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0307)	(0.0000)	(0.0000)				
Social eng.	-0.0712*	-0.0853*	-0.0890*	0.0214^{\star}	-0.0077	-0.0323*	0.0135^{\star}	0.0098**	1.0000		
	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0645)	(0.0000)	(0.0012)	(0.0197)			
Environment	0.0188*	0.0215^{\star}	-0.0211*	0.2110*	0.0118*	0.0507^{\star}	0.1205^{\star}	0.0752^{\star}	0.0372^{\star}	1.0000	
	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0047)	(0.0000)	(0.0000)	(0.0000)	(0.0000)		
Security	0.0553^{\star}	0.0435^{\star}	0.0311*	0.1661*	0.0115^{\star}	0.0855^{\star}	0.1156^{\star}	0.0847^{\star}	$0.0042^{'}$	0.2755^{\star}	1.0000
-	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0059)	(0.0000)	(0.0000)	(0.0000)	(0.3190)	(0.0000)	
p-value in parenthe	reses. *p<0	$0.01 \stackrel{\star\star}{p} < 0.05$)	-							
Source: British Ho	ougobold D	and Survey	$W_{2MO} = 0.18$								

Source: British Household Panel Survey Wave 9-18.