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THE INCOME–HEALTH RELATIONSHIP 'BEYOND THE MEAN': NEW EVIDENCE FROM BIOMARKERS

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ABSTRACT

The relationship between income and health is one of the most explored topics in health economics but less is known about this relationship at different points of the health distribution. Analysis based solely on the mean may miss important information in other parts of the distribution. This is especially relevant when clinical concern is focused on the tail of the distribution and when evaluating the income gradient at different points of the distribution and decomposing income-related inequalities in health is of interest. We use the unconditional quantile regression approach to analyse the income gradient across the entire distribution of objectively measured blood-based biomarkers. We apply an Oaxaca–Blinder decomposition at various quantiles of the biomarker distributions to analyse gender differentials in biomarkers and to measure the contribution of income (and other covariates) to these differentials. Using data from the Health Survey for England, we find a non-linear relationship between income and health and a strong gradient with respect to income at the highest quantiles of the biomarker distributions. We find that there is heterogeneity in the association of health to income across genders, which accounts for a substantial percentage of the gender differentials in observed health. Copyright © 2016 John Wiley & Sons, Ltd.

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1. INTRODUCTION

The positive association between income and health is a well-established finding in the health economics literature. This relationship has been found across age groups, in many countries analysed and for a variety of health measures, including self-rated health (Mackenbach *et al*., 2005; Ettner, 1996), functional limitations (Ettner, 1996), anthropometric measures (Wagstaff *et al*., 2003) and mortality (Cutler *et al*., 2006). The association between economic conditions and health is also the basis of empirical research on 'income-related health inequalities', one of the most prominent fields in the health economics literature (e.g. Kakwani *et al*., 1997; van Doorslaer and Jones, 2003; van Doorslaer and Koolman, 2004). Studies of the influence of economic conditions on health typically use regression models of the conditional mean of the health status variable. Unfortunately, analysis based solely on the mean misses potentially important information in other parts of the distribution (Bitler *et al*., 2006). This is especially relevant to the income–health relationship, when clinical concern is focused on the tail of the distributions and when evaluating the income gradient at different points of

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the distribution of health status and decomposing income-related inequalities in health could be beneficial (Jones and Lopéz Nicolás, 2006).

This lack of evidence is likely due to two factors. On one hand, health information is often unavailable on a continuous scale in standard health or social surveys. For instance, self-assessed health and functional limitations are collected on an ordinal scale while mortality is a dichotomous indicator (by nature). 'Beyond the mean analysis' is obviously less attractive in these cases. On the other hand, the literature in econometrics has developed techniques going 'beyond the mean' only recently (see Fortin *et al*., 2011 for a review). This is because, unlike the mean-based estimation framework, estimates on the entire distribution of the dependent variables, that is, quantile regressions, cannot be easily used to estimate the impact of a covariate on the corresponding unconditional quantile of the dependent variable (Firpo *et al*., 2009). Ordinary least squares (OLS) regressions provide consistent estimates of the coefficient β of an explanatory variable, X, for the population unconditional mean of an outcome variable, Y, because the conditional mean, $E[Y|X]$, averages up to the unconditional mean, $E[Y]$, because of the law of iterated expectations. As a consequence, a linear model for conditional means $E[Y|X] = X\beta$ implies that $E[Y] = E[X]\beta$ and OLS estimates of β also indicate what is the impact of X on the population average of Y (Firpo *et al*., 2009). When the attention shifts towards the entire distribution, the situation is more complicated because conditional quantiles do not average up to their population counterparts, that is, $q_y(\tau) \neq E[q_{y|X}(\tau)]$. The analysis 'beyond the mean' of the unconditional distribution of the dependent variable is then more challenging.

In this paper, we use a distributional method proposed in the recent literature, the recentered influence function (RIF) approach of Firpo *et al*. (2009) to estimate the income gradient for a continuous measure of objective health status: blood-based biomarkers. Biomarkers are characteristics that are 'objectively measured and evaluated as indicators of normal biological processes, pathogenic processes, or pharmacologic responses to a therapeutic intervention'.[1] They are measured on a continuous scale associated with an increasing or decreasing risk (depending on the biomarker) of a disease state, and they are often highly correlated with mortality (Rosero-Bixby and Dow, 2012; Sattar *et al*., 2009; Gruenewald *et al*., 2006). We consider four blood-based biomarkers available in 10 waves of the Health Survey for England (2003–2012) associated with some of the most prevalent diseases in all Western countries: cholesterol, glycated haemoglobin, fibrinogen and ferritin. Cholesterol measures 'fat in the blood', and it is associated with a higher risk of heart disease; glycated haemoglobin (HbA1c) is a biomarker for diabetes; fibrinogen is a haemostatic marker associated with many inflammatory diseases including cardiovascular and liver diseases; ferritin is a biomarker for poor nutrition and is associated with other important diseases such as liver diseases. Our data are cross sectional and do not lend themselves to an instrumental variable analysis that might permit causal inference. In keeping with the normative literature on the measurement of health equity, we focus on the association between the biomarkers and gender, income and other socio-economic factors.

Biomarker data have also been recently used to analyse the effect of socio-economic position on the conditional mean of the biomarker score. Using biomarker data for diabetes, hypertension and cardiovascular diseases, Banks *et al*. (2006) found that English residents have on average better health than US residents. Juerges *et al*. (2013) found a positive relationship between schooling and biomarkers of cardiovascular diseases (fibrinogen and C-reactive protein). Muennig *et al*. (2007) look at differences between socio-economic groups in C-reactive protein and cholesterol homocysteine, associated with cardiovascular diseases. They found a positive effect of income and education on 'good cholesterol' and a slightly significant effect on fibrinogen. Ploubidis *et al*. (2014) found a negative impact of early life socio-economic position on fibrinogen levels later in life. Dowd and Goldman (2006) tested the influence of stress biomarkers on the relationship between socio-economic status and health. They found that chronic stress is actually not very different across socio-economic groups. A key advantage from using biomarker

[1]This definition is given by the National Institute of Health Biomarkers Definitions Working Group, see Atkinson *et al*. (2001).

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data in all these analyses is having a measure of health, which is free of reporting bias. This is particularly important given the intense debate around the extent of socio-economic-related reporting bias.[2]

All these studies corroborate the idea that social position and economic conditions contribute to shape objective health. Our paper goes beyond this literature in two ways. Firstly, we use the unconditional quantile regression approach developed by Firpo *et al*. (2009) to analyse the income gradient across the entire distribution of biomarkers. For a given quantile of the distribution, the method consists of applying the (recentered) influence function transformation to convert the dependent variable into a binary variable and then running a linear probability model regression of the transformed variable on the explanatory variables. The method works by providing a linear approximation to a non-linear functional of the distribution. This allows the law of iterated expectations to be applied to the distributional statistics of interest (i.e. the quantiles) and thus to compute approximate partial effects of the covariates. In our setting, this method allows us to properly assess the income gradient at different points of the unconditional distribution of the biomarkers. This is desirable in order to check for non-linearities in the relationship between income and health and to assess the role of income at extreme biomarker levels, which often indicate the presence or the risk of severe diseases that are associated with high costs for the health system.

Secondly, we apply an Oaxaca–Blinder (OB) decomposition at various quantiles of the biomarker distributions to analyse gender differentials in biomarkers and to measure the contribution of income (and other covariates) to these differentials. OB decomposition assesses to what extent gender differentials in biomarkers are explained by compositional differences, because of differences in observed covariates, or by differences in the elasticity of health with respect to income and other factors. More generally, decomposition analysis is useful for drawing policy implications because it helps to delineate the role of health policies, that operate on the health-gradient, from the role of fiscal policy, that operates on compositional differences (Van Doorslaer and Koolman (2004) for a discussion of the policy implications of decomposition analysis). Jones and Lopéz Nicolás (2006) found that there is considerable heterogeneity in the association of health with explanatory variables across genders, and they have shown that this has important consequences on the measurement of income-related inequalities in health. Our paper contributes to this literature by performing the decomposition analysis of gender differentials in health along the entire distribution of health status.

We find a non-linear relationship between income and health and a strong gradient with respect to income at the highest quantiles of the biomarker distributions. In some cases, that is, cholesterol, the income gradient is found only at high quantiles, while analysis 'at the mean' leads to misleading conclusions (i.e. a positive relationship between income and cholesterol). This makes the analysis on the entire distribution especially relevant in such cases. Secondly, we find that there is important heterogeneity in the association of health to income across genders, which varies significantly along the biomarker distribution and accounts for a substantial percentage of the gender differentials in observed health.

The rest of the paper is organized as follows. The next section presents the data and descriptive statistics. Section 3 discusses the empirical methodology. Section 4 presents the results. The final section summarizes and concludes.

2. DATA

Our data come from the Health Survey for England (HSE). HSE is a cross-sectional health interview survey of around 15 000 to 20 000 respondents in England conducted by the National Centre for Social Research (separate surveys are available for Scotland and Wales). The survey started in 1991 and has been carried out

[2]Empirical literature investigating the extent of reporting heterogeneity in health between socio-economic groups has found contrasting results. Some papers (i.e. Sen (2002) for India; Cawley and Choi (2015) for the educational gradient in the USA) show the existence of a large reporting heterogeneity. Some others, (i.e. Hernández-Quevedo *et al*. (2004) for the United Kingdom) do not find any heterogeneity while some others (i.e Bago d'Uva *et al*. (2008) for China, Indonesia and India) found only a small reporting heterogeneity and not in all countries analysed.

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Table I. Descriptive statistics – biomarkers

Variables	Mean	Std Dev.	Waves[1]	Observations
Cholesterol	5.57	1.15	2,3,4,5,6,7,8,9,10,11	30 770
Glycated haemoglobin	5.58	0.75	2,3,4,5,6,7,8,9,10,11	34 831
Fibrinogen	3.01	0.74	2,3,4,5,8	15 530
Ferritin	99.29	109.3	1,3,4,5,8	13 849

2002 = wave 1; 2012 = wave11.

annually since then. HSE includes a representative sample of adults aged 16 and over and since 1995 has also included children aged 2–15. From 2001 onwards, the survey covers all ages, but certain age groups are only asked questions on selected topics. An interview with each eligible person in the household is followed by a nurse visit for those who agree to take part. The interview includes a set of core questions, asked each year, on general health and psycho-social indicators, smoking, alcohol, demographic and socio-economic indicators, questions about use of health services and prescribed medicines. Biomarkers are collected during nurse visits and include not only blood samples but also anthropometric measurements, blood pressure measurements and saliva samples. During the nurse visits, the nurse asks the respondent for permission to carry out various types of measurement. Respondents are informed about the purpose and value of each test for the monitoring of various diseases. For instance, for the cholesterol test, the nurse informs participants that 'high levels are associated with blood clots, heart attack and stroke.' The delivery of information is useful in order to increase compliance and establish a good working relationship.

The most popular blood-based biomarkers, which are analysed in this paper, have been collected since 2002 in HSE. More precisely, cholesterol and HbA1c were collected from 2003 to 2012 every year. Fibrinogen was collected from 2003 to 2006 and in 2009. Ferritin was collected in 2002, from 2004 to 2006 and in 2009. Other potentially relevant biomarkers (i.e. tryglicerides, C-reactive protein) are collected sporadically, and they are not included in this analysis. We do not make any statistical transformation of the blood-based biomarkers sample and use the valid measurements in each wave, where the blood sample collected and processed successfully. We have 30 770 non-missing observations for the analysis of cholesterol spanning over the period 2003–2012, 34 831 for the analysis of HbA1c over the period 2003–2012, 15 530 for the analysis of fibrinogen from 2003 to 2006 and in 2009 and 13 849 observations for ferritin available in 2002, 2004, 2005, 2006 and 2009. An implicit age stratification comes from the age restriction used by HSE for the blood sample collection. For almost all the waves, blood samples are collected from individuals aged 16+. In a few waves a different age restriction is employed. In the 2002, only individuals aged 24 or less are included; in the 2004, individuals aged 11+ are included, while in 2005 only individuals aged 65+ are analysed. A careful control for demographics is employed in all our analysis, and sample weights are used in all regressions to take account of the survey design and age structure of the different waves.[3]

2.1. Variables and descriptive statistics

In what follows, we provide a description of the variables used in our analysis. Firstly, we describe the blood-based biomarker variables giving some detail on their unit of measurement, the clinical cutpoints (when available) and the use of biomarker values for diagnosis of a disease state. Later, we describe the income variable and other controls employed in the regressions. A complete list of the variables, along with descriptive statistics, is presented in Tables I and II.

We examine the four blood-based biomarkers for which regularly collected data are available in the HSE: total cholesterol, glycated haemoglobin, fibrinogen and ferritin. As discussed in the introduction, these markers

[3]HSE data includes three main datasets (households, individuals and individuals who give blood) and releases three set of weights. The aim of each set of weights is that each of the main datasets can be treated as broadly representative of the general household population. Our paper is based on the blood sample data; thus, 'blood sample weights' are used in all analyses.

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Table II. Descriptive statistics – independent variables

Variables	Mean	Std Dev.
Log (household equivalised income)	10.04	0.82
M (11–18) – Omitted Category	0.01	0.13
M (18–34)	0.09	0.29
M (35–44)	0.10	0.30
M (45–64)	0.16	0.36
M (65–74)	0.06	0.23
M (75+)	0.03	0.17
F (11–18)	0.02	0.13
F (18–34)	0.11	0.31
F (35–44)	0.12	0.33
F (45–64)	0.20	0.40
F (65–74)	0.07	0.25
F (75+)	0.04	0.20
No Qualification – omitted category	0.21	0.41
Degree – NVQ 4,5	0.23	0.42
Higher education	0.12	0.33
NVQ 3 – GCE A	0.14	0.35
NVQ 2 – GCE 0	0.23	0.42
NVQ1 – CSE	0.05	0.21
Other qualifications	0.02	0.15

NVQ, National Vocation Qualification; GCE, General Certificate of Education; CSE, Certificate of Secondary Education.

are highly predictive of some of the most prevalent non-communicable diseases. Total cholesterol is measured in units called millimoles per litre of blood (mmol/L). The English government recommends that total cholesterol should be equal or less than 4 mmol/L among individuals at high risk of cardiovascular disease (CVD) (i.e. obese and with a history of CVD) and equal or less than 5 mmol/L or less for healthy individuals. Values above these thresholds indicate a higher risk of CVD.

Glycated haemoglobin is a measure of the level of sugar in the blood over the previous 8 to 12 weeks before measurement. It is the proportion of haemoglobin proteins that have been bound by glucose. HbA1c can be expressed as a percentage or as a value in mmol/mol. HbA1c is measured as a percentage in all waves of the HSE. HbA1c values $\geq$6.5% indicates diagnosis of diabetes, while values between 5.7% and 6.4% indicate pre-diabetes risk (ADA, 2010; WHO, 2011a).

Fibrinogen is a marker of inflammation, and it aids the body to stop bleeding by helping blood clots to form. It is measured in grammes per litre (g/L). The measure is continuous and there are no established clinical cutpoints, but normal levels generally range between 1.5–3 g/L. Higher levels of fibrinogen are implicated in the development of CVD and many inflammatory diseases, such as liver diseases.

Levels of ferritin reflect the size of the body's stock of iron, and therefore, they are indicative of anaemia. A low ferritin level is predictive of uncomplicated iron deficiency anaemia, caused, for instance, by poor nutrition. However, high ferritin levels suggest excess body iron, which is also problematic for health because it is generally associated with important diseases such as liver diseases. WHO (2011b) suggests some cut-points: ferritin levels below $\leq$20 ug/L indicate depletion of iron, while levels $\leq$12 indicate complete absence of stored iron. Ferritin levels $>$300 ug/L may indicate iron overload in men, and post-menopausal women and $>$200 may indicate iron overload in pre-menopausal women.

Our main independent variable is household equivalised income. It includes total income of a household from all sources, after tax and other deductions, divided by the number of household members converted into equivalised adults. In order to take into account the fact that income changes are often multiplicative in the real world (i.e. a 5% raise in wages), we take the logarithms of equivalised income in all regressions; this is a common practice and allows a clearer interpretation of the income–health relationship.

As control variables, we include six age group variables (11–18, 18–34, 35–44, 45–64, 65–74, 75+) for each gender and seven dummies for educational status. Education is measured according to the following categories:

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Degree or National Vocation Qualification (NVQ) 4 or 5; Higher education below degree; NVQ 3 or General Certificate of Education (GCE) Advanced Level; NVQ 2 or GCE ordinary level; NVQ1 or Certificate of Secondary Education (CSE); Other qualifications from outside England; No qualification. Omitted categories in our analysis are men, aged 11–18 and with no qualification.

Table I shows descriptive statistics for the biomarkers. We find that average biomarker values in our sample fall largely within normal ranges but with some exceptions. In particular, average cholesterol values are around 10% higher than the cutpoint of 5 while fibrinogen average scores are a bit higher than the normal cutpoint of 3. Moreover, Table I depicts a higher dispersion around the average cholesterol and ferritin values, while other biomarkers values are less dispersed around the mean.

Table II shows descriptive statistics for the independent variables. We observe a high share of individuals aged 45–64 and a higher share of elderly women (over 65), consistent with the well-known gender differences in life expectancy. With respect to education, we observe that around 23% of individuals in our sample have a degree, and a similar share of them have an NVQ 2 or GCE. Also the share of individuals without formal education is substantial (around 21%). This includes mostly individuals belonging to older cohorts without any formal education (around 20% and an average age of 60) and students receiving compulsory education or attending a school at the time in which the interview was conducted (approximately 1% of our sample and an average age of 15).

3. EMPIRICAL METHODOLOGY

Our empirical analysis is based on the RIF method of Firpo *et al*. (2009). We use the method to estimate the relationship between income and biomarkers and we use RIF regression as a basis for OB decomposition of gender differentials in England.

As discussed in the introduction, the key advantage of the RIF approach is that it allows us to analyse the relationship between income and the *unconditional* distribution of biomarkers and to analyse and decompose differences in the *unconditional* distribution of biomarkers across genders. This possibility is essentially given by the fact that RIF method works by providing a linear approximation of the unconditional quantiles of the dependent variable.[4] The law of iterated expectations can be applied to the quantile being approximated and used to estimate the marginal effect of a covariate through a simple regression of a function of the outcome variable, the RIF, on the covariates X.

In our setting, the RIF of the biomarkers is estimated directly from the data by first computing the sample quantile q and then estimating the density of the distribution of biomarkers at that quantile using kernel density methods. Then, for a given observed quantile q_τ, a RIF is generated that can take one of two values depending upon whether or not the observation's value of the outcome variable is less than or equal to the observed quantile:

$$RIF\ (Bio; q_\tau) = q_\tau + \frac{\tau - 1[Bio \leq q_\tau]}{f_{Bio}(q_\tau)} \tag{1}$$

where q_τ is the observed sample quantile, $1[Bio \leq q_\tau]$ is an indicator variable equal to one if the observation's value of the biomarker is less than or equal to the observed quantile and zero otherwise. $f_{Bio}(q_\tau)$ is the estimated kernel density of the biomarker at the τ_{th} quantile.

The RIF defined in Equation 1 is then used as a dependent variable in a OLS regression on the covariates X, as defined in Section 2.1. In practice, this amounts to estimating a rescaled linear probability model (Jones *et al*., 2015). Indeed, the unconditional quantile of the biomarker, q_τ, may be obtained as follows:

[4]It is worth noting that the RIF approach is not confined to estimating quantile functions. For example, a recent paper by Heckley *et al*. (2016) derive the RIF for the family of bivariate rank dependent indices of socio-economic inequality in health, which includes the concentration index and other commonly used indices. Having the RIF allows a linear regression based decomposition of these indices.

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$$q_\tau = E_x\left[E[\widehat{RIF}(Bio;q_\tau)|X]\right] \tag{2}$$

where $\widehat{RIF}(Bio;q_\tau)|X$ is the estimate of RIF as defined in Equation 1 conditional on covariates X. Thanks to this linear approximation, it is now possible to apply the the law of iterated expectations. Thus, q_τ can be written as:

$$q_\tau = E[X]\,\widehat{\delta_\tau}$$

where $\hat{\delta_\tau}$ is the coefficient of the unconditional quantile regression. This linearization allows estimation of the marginal effect of a change in distribution of covariates X (including income) on the *unconditional* quantile of biomarkers, measured by the parameters $\hat{\delta_\tau}$. In our model, as well as the covariates X presented in Table II, we also include year fixed effects, to pick up time variation in biomarker levels and any survey-specific effects.

To analyse gender differentials in biomarkers, we use the OB decomposition method using the RIF regression in Equation 2 as a basis for the decomposition (Blinder, 1973; Oaxaca, 1973). A similar logic to the OB decomposition at the mean applies here (see Fortin *et al.* (2011) for a review). Formally, differences in estimated biomarker levels between men (M) and women (F) at each quantile can be decomposed as follows:

$$\Delta^{\tau}_{Bio} = \left[\widehat{RIF}(Bio_M,\ q_{M\tau}) - \left[\widehat{RIF}(Bio_F,\ q_{F\tau})\right]\right.$$

$$\Delta^{\tau}_{\mathrm{Bio}} = \left(\overline{\mathrm{X}}_{\mathrm{M}}\text{-}\overline{\mathrm{X}}_{\mathrm{F}}\right)\delta_{\mathrm{F}} + \overline{\mathrm{X}}_{\mathrm{F}}(\delta_{\mathrm{M}} - \delta_{\mathrm{F}}) \tag{3}$$

where $\overline{X}_M$ and $\overline{X}_F$ represent the sample means of covariates X for the subsample of men and women and δ_M and δ_F represent the coefficients of the unconditional quantile regression as in Equation 2 for the subsample of men and women, respectively.

The first term in Equation 3 is the part of the differential in biomarkers that is 'explained' by differences in observed covariates between the subsample of men and women. This is often called the 'composition effect'. Differences in covariates across genders are weighted by the coefficients of the unconditional quantile regression from a model estimated on the subsample of women (δ_F). The decomposition is thus formulated from the viewpoint of women as in the original work by Oaxaca (1973). In our application, the choice of the discriminated group is complicated by the fact that women might have some health advantages over men (i.e. they have a higher longevity than men, for instance) but they often earn less than men because of gender discrimination in the labour market. We therefore consider women as the discriminated group. This puts our OB decomposition in line with the traditional discrimination literature, that is, analysis of the gender wage gap (see the discussion in Neumark (1988) and Jann (2008) for more details).

The second term in Equation 3 measures the 'unexplained' part of the differential in biomarkers. This is often called the 'structural' part, and it accounts for differences in biomarkers across genders that are due to differences in the impact of the covariates and it also captures all potential effects of differences in unobserved variables.

The explained and unexplained part can be further decomposed into contributions of each covariate at each quantile. In our case, it is particularly useful to derive both the total contribution and the detailed contribution of income to the gender differentials in biomarkers. This allows us to understand to what extent differences in biomarkers are driven by differences in income between men and women ('composition effect') and/or by differences in the association of health to income across genders (also known as the 'elasticity effect'). Thanks to the additivity assumption of the OB decomposition, this is possible because the 'explained' and 'unexplained' part in Equation 3 are simply given by the sum of the contribution of individual covariates.

Thus, it is possible to derive the detailed contribution of all covariates (including income) to the 'explained part', as follows:

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$$\left(\overline{X}_M - \overline{X}_F\right)\delta_F = \left(\overline{X}_{1M} - \overline{X}_{1F}\right)\delta_{1F} + \left(\overline{X}_{2M} - \overline{X}2_F\right)\delta_{2F} + \ldots \quad (4)$$

where $\overline{X_1}$ and $\overline{X_2}\ldots$ are the means of the single covariates and δ_F are the associated coefficients of the unconditional quantile regression estimated on the subsample of women. Similarly, the contributions of each covariate to the 'unexplained part' can be obtained as follows:

$$\overline{X}_F(\delta_M - \delta_F) = \overline{X}_{1F}(\delta_{1M} - \delta_{1F}) + \overline{X}_{2F}(\delta_{2M} - \delta_{2F}) + \ldots \quad (5)$$

To draw inferences on the contributions of each covariate to the explained and unexplained part, standard errors are computed using the delta method (see Jann (2008) for more details). The explained part ($\left(\overline{X}_M - \overline{X}_F\right)\delta_F$) in Equation 3 captures all the compositional differences, in income and in the other factors, between men and women. Moreover, thanks to the additivity assumption, the detailed OB decomposition presented in Equations 4 and 5 allow us to separate the elasticity from the compositional effect of each covariate on the total differences in biomarkers between men and women.[5]

4. RESULTS

4.1. Income–health relationship

Tables III–VI show the results of RIF regressions described in Equation 2 for cholesterol, glycated haemoglobin, fibrinogen and ferritin, respectively. Column 1 of each table includes the results for OLS regression at the mean, for comparison, while columns 2–5 include results of the RIF regressions at the 25th, 50th, 75th, 90th and 95th percentile of each biomarker, respectively. To make the interpretation of our coefficients of interest easier, we plot the income coefficients for OLS and RIF regressions at points of the biomarker distribution (every 5 percentiles), along with their 95% confidence intervals, in Figures 1–4.[6]

Table III shows that the relationship between income and cholesterol is somewhat complex and that analysis at the mean misses important information. Indeed, the OLS estimate shows a positive income gradient (column 1), while RIF regressions indicate that income–cholesterol relationship varies at different points of the cholesterol distribution. At the lowest quantiles of the distribution, the income–cholesterol association is positive, while from the 75th percentile of cholesterol distribution, the relationship is negative, albeit not statistically significant. More precisely, Figure 1 shows that the 'saddle' point is located around the 80th percentile, corresponding to a cholesterol score of 6.5, (30%) higher than the clinical cutpoint of 5. After this threshold, the income gradient increases in magnitude and is much higher at the extreme points of the cholesterol distribution (i.e. around the 95th percentile). Interestingly, this pattern

[5]The unexplained part ($\overline{X}_F(\delta_M - \delta_F)$) in Equation 3 might be conceived as the treatment effect of gender on health, or, more precisely, as the population treatment effect on the treated (Fortin *et al*., 2011). However, it is important to note that the interpretation of OB decomposition in causal terms is valid under the assumptions of *ignorability and common support*. These assumptions are also the identifying assumptions of all the estimators mentioned earlier, belonging to the strand of treatment effect literature, which relies on *selection on observables*. When the OB decomposition is performed on the entire outcome distribution, as in our case, these assumptions guarantee the *invariance of conditional distribution*, namely, that the conditional distribution of the biomarker given the control variables X remains invariant under manipulations of the marginal distribution of the X. Despite that, we prefer to not give a causal interpretation of our results essentially because of the nature of our treatment. In general, gender is not a choice or a manipulable action, as we cannot obviously conceive individuals choosing which group to belong to (see for instance the discussion in Fortin *et al*., 2011 and in Holland, 1986).

[6]RIF regression results are based on a Gaussian kernel function and a bandwidth of 0.06, which is also chosen by Firpo *et al*. (2009). In a sensitivity analysis, we also experimented using 'optimal' bandwidth that would minimise the mean integrated squared error if the true distribution of the data was Gaussian. This leads to bandwidths that are a little larger than 0.06 and, when the data are skewed as they are with our biomarkers, may over-smooth the density. In practice, we find that there is very little difference in the RIF regression results with the alternative bandwidths and all our conclusions are unchanged. Similarly, we verified that all our results are unchanged using a different kernel function, the Epanechnikov kernel (results available upon request).

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Table III. Cholesterol Results - OLS and RIF regressions

		RIF Regressions				
Variables	OLS	*Q25*	*Q50*	*Q75*	*Q90*	*Q95*
Log Income	0.035***	0.048***	0.039***	0.015	-0.014	-0.015
	0.008	*0.013*	*0.011*	*0.013*	*0.017*	*0.023*
M (18-34)	0.695***	1.018***	0.572***	0.388***	0.288***	0.263***
	0.038	*0.087*	*0.044*	*0.039*	*0.040*	*0.054*
M (35-44)	1.353***	1.739***	1.268***	1.058***	0.880***	0.815***
	0.039	*0.085*	*0.045*	*0.046*	*0.057*	*0.074*
M (45-64)	1.559***	1.949***	1.525***	1.276***	1.051***	0.964***
	0.037	*0.082*	*0.041*	*0.041*	*0.050*	*0.069*
M (65-74)	1.395***	1.857***	1.364***	1.081***	0.824***	0.593***
	0.045	*0.086*	*0.052*	*0.057*	*0.077*	*0.102*
M (75+)	1.082***	1.552***	1.013***	0.737***	0.424***	0.248**
	0.050	*0.094*	*0.059*	*0.064*	*0.083*	*0.103*
F (11-18)	0.104**	0.270**	0.022	0.008	0.024	0.067
	0.042	*0.105*	*0.057*	*0.043*	*0.051*	*0.075*
F (18-34)	0.514***	0.842***	0.348***	0.197***	0.159***	0.147***
	0.036	*0.085*	*0.039*	*0.033*	*0.034*	*0.047*
F (35-44)	0.940***	1.451***	0.773***	0.476***	0.392***	0.358***
	0.038	*0.085*	*0.042*	*0.037*	*0.043*	*0.058*
F (45-64)	1.661***	1.988***	1.586***	1.449***	1.240***	1.238***
	0.037	*0.082*	*0.040*	*0.039*	*0.050*	*0.071*
F (65-74)	1.985***	2.098***	1.923***	2.026***	1.848***	1.840***
	0.043	*0.083*	*0.045*	*0.056*	*0.086*	*0.120*
F (75+)	1.787***	2.013***	1.749***	1.646***	1.549***	1.389***
	0.045	*0.087*	*0.052*	*0.066*	*0.102*	*0.140*
Degree- NVQ 4,5	-0.080***	-0.016	-0.087***	-0.090***	-0.186***	-0.225***
	0.021	*0.030*	*0.028*	*0.035*	*0.049*	*0.066*
Higher Education	-0.016	0.016	-0.017	0.016	-0.080	-0.192**
	0.023	*0.031*	*0.031*	*0.040*	*0.058*	*0.076*
NVQ 3- GCE A	-0.094***	-0.088**	-0.059**	-0.091***	-0.154***	-0.223***
	0.021	*0.035*	*0.030*	*0.035*	*0.050*	*0.066*
NVQ 2 – GCE 0	-0.048**	-0.051*	-0.029	0.010	-0.089*	-0.170***
	0.019	*0.028*	*0.026*	*0.033*	*0.048*	*0.064*
NVQ1 – CSE	-0.089***	-0.048	-0.089**	-0.049	-0.223***	-0.317***
	0.030	*0.046*	*0.041*	*0.051*	*0.068*	*0.086*
Other Qualifications	0.009	-0.012	-0.035	-0.013	-0.031	0.058
	0.045	*0.048*	*0.057*	*0.080*	*0.127*	*0.182*
Constant	4.274***	2.851***	4.142***	5.580***	7.178***	7.976***
	0.088	*0.151*	*0.117*	*0.142*	*0.202*	*0.274*
Years Fixed Effects	YES	YES	YES	YES	YES	YES
Observations	30770	30770	30770	30770	30770	30770

Standard Errors in Italics; ***, **, * indicate significance at 1%, 5% and 10%, respectively. Sample weights applied.

indicates that the income gradient is larger in the range where cholesterol levels exceed the normal range and are indicative of a disease state.

With respect to the other covariates, we find higher cholesterol levels among the elderly, (especially women) and a strong association with education at all levels of the cholesterol distribution. However, while the age gradient appears to be marginally decreasing along the cholesterol distribution, the education gradient is marginally increasing: the cholesterol gradient between educated (at any level) and individuals without formal education (reference category) increases along the cholesterol distribution and reach its peak around the 95th percentile of the cholesterol distribution.

The association between income and HbA1c is negative at all quantiles of the distribution but it varies highly in magnitude along the distribution. In this case, the OLS estimate provides a poor reflection of this pattern (column 1 of Table IV), while RIF estimates (columns 2–5 of Table IV) show that the income coefficient at 95th percentile of HbA1c distribution is 10 times higher than the income coefficient at the 25th percentile

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Table IV. Glycated Haemoglobin Results - OLS and RIF regressions

		RIF Regressions				
Variables	OLS	*Q25*	*Q50*	*Q75*	*Q90*	*Q95*
Log Income	-0.065***	-0.025***	-0.037***	-0.048***	-0.094***	-0.204***
	0.005	*0.004*	*0.004*	*0.005*	*0.010*	*0.026*
M (18-34)	0.068***	0.034	0.040*	0.044**	0.117***	0.267***
	0.025	*0.032*	*0.021*	*0.021*	*0.025*	*0.056*
M (35-44)	0.278***	0.221***	0.221***	0.245***	0.289***	0.581***
	0.026	*0.032*	*0.021*	*0.022*	*0.029*	*0.069*
M (45-64)	0.513***	0.338***	0.379***	0.465***	0.649***	1.337***
	0.025	*0.031*	*0.020*	*0.021*	*0.031*	*0.078*
M (65-74)	0.679***	0.381***	0.475***	0.639***	1.116***	2.307***
	0.027	*0.031*	*0.021*	*0.025*	*0.048*	*0.133*
M (75+)	0.697***	0.397***	0.526***	0.742***	1.187***	2.137***
	0.030	*0.031*	*0.022*	*0.027*	*0.059*	*0.166*
F (11-18)	-0.032	-0.027	-0.009	-0.041*	-0.064***	-0.134***
	0.028	*0.036*	*0.023*	*0.021*	*0.017*	*0.036*
F (18-34)	0.034	-0.019	-0.004	0.022	0.106***	0.262***
	0.024	*0.031*	*0.020*	*0.020*	*0.022*	*0.051*
F (35-44)	0.158***	0.109***	0.099***	0.116***	0.177***	0.449***
	0.025	*0.032*	*0.020*	*0.021*	*0.025*	*0.060*
F (45-64)	0.411***	0.310***	0.333***	0.396***	0.475***	0.839***
	0.024	*0.031*	*0.020*	*0.021*	*0.027*	*0.065*
F (65-74)	0.610***	0.398***	0.497***	0.661***	0.915***	1.638***
	0.027	*0.031*	*0.021*	*0.024*	*0.044*	*0.118*
F (75+)	0.628***	0.407***	0.537***	0.738***	1.100***	1.618***
	0.028	*0.032*	*0.022*	*0.026*	*0.053*	*0.137*
Degree- NVQ 4,5	-0.154***	-0.079***	-0.083***	-0.133***	-0.243***	-0.558***
	0.013	*0.010*	*0.009*	*0.012*	*0.026*	*0.073*
Higher Education	-0.119***	-0.038***	-0.047***	-0.089***	-0.186***	-0.452***
	0.014	*0.010*	*0.009*	*0.013*	*0.030*	*0.083*
NVQ 3- GCE A	-0.154***	-0.071***	-0.077***	-0.143***	-0.238***	-0.553***
	0.013	*0.011*	*0.010*	*0.012*	*0.026*	*0.073*
NVQ 2 – GCE 0	-0.107***	-0.042***	-0.044***	-0.088***	-0.195***	-0.480***
	0.012	*0.009*	*0.008*	*0.011*	*0.025*	*0.070*
NVQ1 – CSE	-0.094***	-0.018	-0.023*	-0.071***	-0.167***	-0.365***
	0.019	*0.014*	*0.013*	*0.018*	*0.040*	*0.120*
Other Qualifications	-0.116***	-0.018	-0.045***	-0.092***	-0.257***	-0.533***
	0.027	*0.018*	*0.017*	*0.024*	*0.058*	*0.162*
Constant	6.043***	5.407***	5.657***	6.126***	6.827***	8.110***
	0.055	*0.052*	*0.042*	*0.051*	*0.101*	*0.277*
Years Fixed Effects	YES	YES	YES	YES	YES	YES
Observations	34831	34831	34831	34831	34831	34831

Standard Errors in Italics; ***, **, * indicate significance at 1%, 5% and 10%, respectively. Sample weights applied.

(−0.204 vs −0.025). Figure 2 shows that the income gradient reaches its peak around the 95th percentile of the HbA1c distribution, corresponding to the clinical diagnosis of diabetes (HbA1c >6.5). A similar pattern is observed also for the other covariates: both education and age gradients increase at the highest quantiles of HbA1c distribution, and they are particularly high around the clinical threshold.

The fibrinogen–income relationship is shown in Table V (and Figure 3). Similarly to HbA1c, we find a negative association at all quantiles of the fibrinogen distribution and an higher income gradient at the top quantiles. However, in the case of fibrinogen, we observe two large jumps at the 85th and at the 90th percentile of the distribution. These are associated with fibrinogen values (3.7 and 4) which are 23% and 33% higher than the upper bound of the normal range (equal to 3), respectively. These very high fibrinogen levels are generally indicative of many important inflammatory diseases and negative acute shocks in health status. This might explain the jumps in the income–fibrinogen association, and it is somewhat different from the other biomarkers analysed, which are more related to chronic conditions. However, with the exception of these two jumps, the

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Table V. Fibrinogen Results - OLS and RIF regressions

		RIF Regressions				
Variables	OLS	Q25	Q50	Q75	Q90	Q95
Log Income	-0.039***	-0.028***	-0.050***	-0.057***	-0.041***	-0.067***
	0.008	*0.010*	*0.011*	*0.014*	*0.016*	*0.025*
M (18-34)	0.053	0.026	-0.010	0.035	0.119*	0.113
	0.039	*0.070*	*0.058*	*0.060*	*0.070*	*0.102*
M (35-44)	0.247***	0.266***	0.202***	0.163***	0.183***	0.168*
	0.040	*0.069*	*0.058*	*0.060*	*0.070*	*0.101*
M (45-64)	0.447***	0.470***	0.438***	0.353***	0.358***	0.392***
	0.039	*0.067*	*0.057*	*0.060*	*0.072*	*0.106*
M (65-74)	0.635***	0.586***	0.625***	0.589***	0.622***	0.618***
	0.045	*0.069*	*0.062*	*0.074*	*0.093*	*0.147*
M (75+)	0.839***	0.672***	0.795***	0.846***	0.887***	1.316***
	0.049	*0.069*	*0.065*	*0.084*	*0.117*	*0.208*
F (11-18)	0.143***	0.144**	0.103	0.046	0.148	0.107
	0.043	*0.073*	*0.074*	*0.080*	*0.108*	*0.150*
F (18-34)	0.385***	0.428***	0.359***	0.329***	0.358***	0.316***
	0.038	*0.066*	*0.057*	*0.060*	*0.073*	*0.104*
F (35-44)	0.402***	0.407***	0.406***	0.385***	0.346***	0.312***
	0.039	*0.068*	*0.058*	*0.062*	*0.072*	*0.103*
F (45-64)	0.581***	0.571***	0.584***	0.552***	0.488***	0.482***
	0.039	*0.067*	*0.056*	*0.061*	*0.073*	*0.106*
F (65-74)	0.798***	0.701***	0.784***	0.852***	0.772***	0.861***
	0.044	*0.067*	*0.061*	*0.073*	*0.095*	*0.150*
F (75+)	0.880***	0.689***	0.882***	1.026***	0.944***	1.120***
	0.046	*0.068*	*0.062*	*0.082*	*0.113*	*0.178*
Degree- NVQ 4,5	-0.193***	-0.157***	-0.193***	-0.262***	-0.279***	-0.222***
	0.020	*0.024*	*0.026*	*0.035*	*0.042*	*0.067*
Higher Education	-0.127***	-0.082***	-0.119***	-0.178***	-0.223***	-0.194**
	0.021	*0.025*	*0.029*	*0.039*	*0.048*	*0.077*
NVQ 3- GCE A	-0.127***	-0.088***	-0.128***	-0.187***	-0.239***	-0.215***
	0.021	*0.026*	*0.029*	*0.038*	*0.044*	*0.067*
NVQ 2 – GCE 0	-0.096***	-0.076***	-0.112***	-0.148***	-0.162***	-0.146**
	0.018	*0.021*	*0.024*	*0.034*	*0.043*	*0.066*
NVQ1 – CSE	-0.032	-0.039	-0.032	-0.038	-0.103	-0.056
	0.028	*0.033*	*0.037*	*0.051*	*0.065*	*0.104*
Other Qualifications	-0.118***	-0.042	-0.098**	-0.194***	-0.195**	-0.203
	0.039	*0.036*	*0.050*	*0.070*	*0.092*	*0.143*
Constant	3.135***	2.640***	3.190***	3.738***	3.954***	4.448***
	0.083	*0.118*	*0.119*	*0.149*	*0.169*	*0.259*
Years Fixed Effects	YES	YES	YES	YES	YES	YES
Observations	15530	15530	15530	15530	15530	15530

Standard Errors in Italics; ***, **, * indicate significance at 1%, 5% and 10%, respectively. Sample weights applied.

income gradient is smoother than the other biomarkers: the income coefficient at the 95th percentile is 'only' twice as much as the income coefficient at the 25th percentile (−0.067 vs −0.028). In this case, OLS regression (column 1 of Table V) provides a closer approximation of the average relationship between income and fibrinogen (the OLS coefficient is −0.039). As far as the other variables are concerned, Table V shows that both age and education gradients increase along the distribution of fibrinogen, in a manner consistent with the other biomarkers analysed.

With respect to ferritin, we find a positive income gradient at all points of the distribution (Table VI).[7] This is consistent with the fact that higher ferritin values generally indicate better health, with the exception

[7]To take into account the differences in the measurement scale of ferritin with respect to other biomarkers (for instance, Table I), we divided ferritin values by 100 in all regressions shown in Table VI and Figure 4. This allows us to use the same bandwidth in the kernel density estimator and makes the ferritin results more comparable with the other biomarkers.

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Table VI. Ferritin Results- OLS and RIF regressions

		RIF Regressions				
Variables	OLS	Q25	Q50	Q75	Q90	Q95
Log Income	0.028***	0.028***	0.045***	0.071***	0.097**	0.129**
	0.011	*0.006*	*0.009*	*0.017*	*0.039*	*0.061*
M (18-34)	0.678***	0.315***	0.884***	1.050***	0.971***	0.860***
	0.041	*0.024*	*0.038*	*0.061*	*0.109*	*0.155*
M (35-44)	0.956***	0.300***	0.933***	1.334***	1.956***	2.374***
	0.047	*0.026*	*0.042*	*0.074*	*0.166*	*0.272*
M (45-64)	1.015***	0.284***	0.887***	1.345***	2.193***	2.453***
	0.043	*0.027*	*0.041*	*0.064*	*0.135*	*0.200*
M (65-74)	1.041***	0.286***	0.923***	1.450***	2.505***	2.740***
	0.049	*0.028*	*0.044*	*0.075*	*0.179*	*0.300*
M (75+)	0.789***	0.237***	0.747***	1.098***	1.697***	2.173***
	0.055	*0.03*	*0.049*	*0.088*	*0.182*	*0.290*
F (11-18)	-0.117***	-0.198***	-0.162***	-0.060**	-0.001	0.003
	0.038	*0.026*	*0.026*	*0.023*	*0.021*	*0.016*
F (18-34)	-0.094**	-0.131***	-0.055	-0.124***	-0.113*	0.031
	0.04	*0.026*	*0.035*	*0.042*	*0.062*	*0.082*
F (35-44)	-0.049	-0.101***	-0.004	-0.110**	-0.048	0.061
	0.046	*0.03*	*0.042*	*0.048*	*0.075*	*0.097*
F (45-64)	0.241***	0.101***	0.383***	0.291***	0.267***	0.313***
	0.043	*0.028*	*0.041*	*0.052*	*0.076*	*0.095*
F (65-74)	0.513***	0.211***	0.656***	0.680***	0.878***	0.942***
	0.049	*0.028*	*0.044*	*0.068*	*0.128*	*0.187*
F (75+)	0.358***	0.177***	0.555***	0.493***	0.477***	0.560***
	0.050	*0.030*	*0.048*	*0.075*	*0.122*	*0.172*
Degree- NVQ 4,5	0.048*	0.029**	0.038	0.071	0.201**	0.053
	0.029	*0.013*	*0.023*	*0.044*	*0.102*	*0.155*
Higher Education	0.064**	0.015	0.057**	0.084*	0.187	0.144
	0.031	*0.014*	*0.025*	*0.05*	*0.12*	*0.189*
NVQ 3- GCE A	-0.038	0.004	0.028	-0.041	-0.167*	-0.201
	0.030	*0.014*	*0.025*	*0.045*	*0.093*	*0.142*
NVQ 2 – GCE 0	-0.027	0.044***	0.039*	-0.058	-0.178**	-0.185
	0.025	*0.012*	*0.021*	*0.036*	*0.075*	*0.113*
NVQ1 – CSE	-0.035	0.021	0.048	-0.014	-0.036	0.021
	0.042	*0.019*	*0.033*	*0.061*	*0.141*	*0.206*
Other Qualifications	-0.010	0.019	0.050	0.081	0.034	-0.392*
	0.067	*0.03*	*0.055*	*0.096*	*0.182*	*0.202*
Constant	0.181*	-0.049	-0.327***	-0.251	0.042	0.407
	0.109	*0.057*	*0.093*	*0.164*	*0.374*	*0.592*
Years Fixed Effects	YES	YES	YES	YES	YES	YES
Observations	13849	13849	13849	13849	13849	13849

Standard Errors in Italics; ***, **, * indicate significance at 1%, 5% and 10%, respectively. Sample weights applied.

of very extreme values which may also indicate the presence of health problems. The income-ferritin relationship reflects this pattern. Indeed, we find that income gradient increases almost linearly up to the 75th percentile and less than proportionally after this threshold and the estimates are less statistically significant at the 95th percentile of ferritin distribution. This pattern is depicted more clearly in Figure 4. The income coefficient increases almost linearly up to the 75th percentile (the income coefficient at the 75th percentile is about two and a half times higher than the coefficient at the 25th percentile). After this threshold (corresponding to ferritin level of 100), the income coefficient remains stable at the 80th and 85th percentile and increases at the 90th and 95th percentile but with less precision in the estimates. The other covariates exhibit a similar pattern: both age and education gradients reduce in magnitude at the top quantiles of the ferritin distribution and an education gradient is not apparent at the 95th percentile of the distribution.

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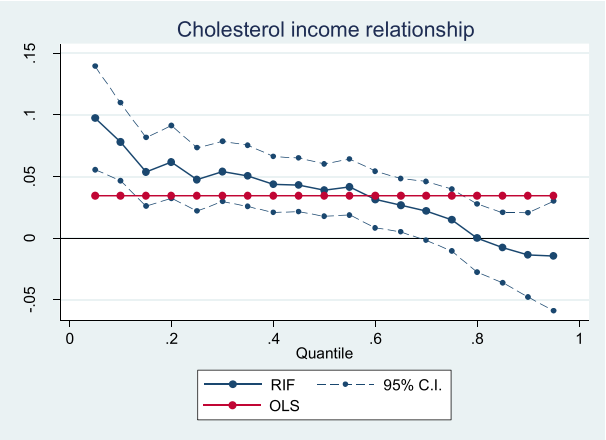

Figure 1. Income coefficient of ordinary least squares (OLS) and recentered influence function (RIF) regression. Cholesterol. [Colour figure can be viewed at wileyonlinelibrary.com]

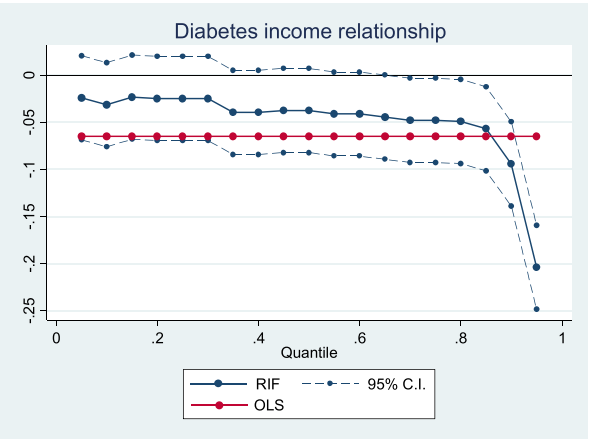

Figure 2. Income coefficient of ordinary least squares (OLS) and recentered influence function (RIF) regression. Glycated haemoglobin. [Colour figure can be viewed at wileyonlinelibrary.com]

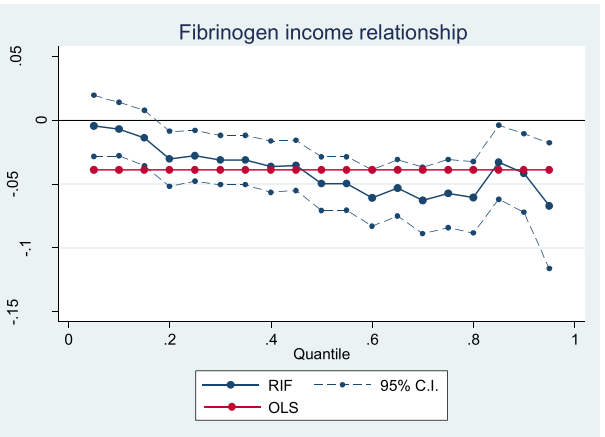

Figure 3. Income coefficient of ordinary least squares (OLS) and recentered influence function (RIF) regression. Fibrinogen. [Colour figure can be viewed at wileyonlinelibrary.com]

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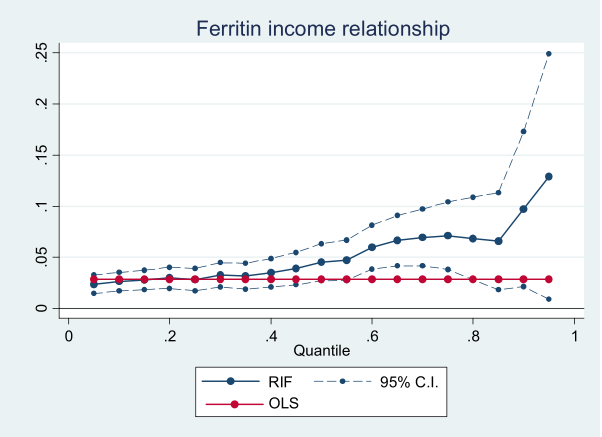

Figure 4. Income coefficient of ordinary least squares (OLS) and recentered influence function (RIF) regression. Ferritin. [Colour figure can be viewed at wileyonlinelibrary.com]

4.2. Oaxaca–Blinder decomposition of gender differentials in biomarkers

The results of OB decomposition at the 25th, 50th, 75th, 90th and 95th percentile distribution of cholesterol, HbA1c, fibrinogen and ferritin are shown in Tables VII–X, respectively. Decomposition is expressed always as a difference between levels for men 'minus' levels for men. Thus, a positive (negative) difference means that a given biomarker value is higher (lower) among men. Tables VII–X include total differences, the explained and the unexplained part and their respective standard errors. The detailed decomposition is shown in Figures 5–8, where we highlight the contribution of main factors (income, education, demographics and year fixed effects) to the explained and unexplained part at 25th, 50th, 75th, 90th and 95th percentile distribution of each biomarker.

We illustrate and explain the mechanics of the decomposition using cholesterol results as an example and discuss the main findings for all other biomarkers. Table VII shows that women have generally higher values of cholesterol at most quantiles of the distribution. This is not surprising and this is partly explained by the female sex hormone oestrogen, which tends to raise high-density lipoprotein cholesterol ('good cholesterol'). However, according to our results, gender differentials are not the same along the entire distribution. They are negligible and not statistically significant at the 25th and 50th percentiles, while they are high and statistically significant at the 90th (0.23 points is the total gender differential) and 95th percentile (around 0.28 points). The second and third rows of Table VII shows that this is explained both by a difference in the impact of covariates on cholesterol ('unexplained part') and by compositional differences in covariates ('explained part'). At high levels of cholesterol (at the 75th and 90th percentile), compositional differences are predominant, while at extreme levels of cholesterol (at the 95th percentile), the unexplained part is more important to explain gender differentials.

The detailed contribution of income and the other covariates is presented in Figure 5. The decomposition exercise shows a large contribution of demographics (red bar) at the lowest levels of cholesterol. This contribution is mainly due to a difference in the association of health to demographics while compositional differences are less important. The contribution of education is important especially from the 75th percentile, and it is also due to differences in the association of health to education across genders. The contribution of income is predominant from the 25th percentile of the cholesterol distribution, and it is particularly high at the extreme levels of cholesterol (95th percentile). Also in the case of income, gender differentials in the income–health association ('elasticity effect') are much more important than compositional differences ('compositional effect') to explain total differentials. The interpretation of the elasticity effect deserves more attention. The sign of the elasticity effect of any covariates in our OB decomposition comes from the second term in Equation 5, and it depends on two factors: the sign of

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Table VII. Oaxaca-Blinder Decomposition of gender differentials in Cholesterol

	Q25	%	Q50	%	Q75	%	Q90	%	Q95	%
Δ_{M-F}	-0.010		0.008		-0.068**		-0.225***		-0.276***	
	0.019		*0.017*		*0.021*		*0.026*		*0.032*	
Compositional	-0.028	280	-0.047***	-588	-0.055***	81	-0.056***	25	-0.056***	20
	0.006		*0.008*		*0.009*		*0.009*		*0.001*	
Elasticity	0.018	-180	0.055**	688	-0.013	19	-0.168***	75	-0.219***	80
	0.018		*0.165*		*0.019*		*0.025*		*0.031*	

Standard Errors in Italics; ***, **, * indicate significance at 1%, 5% and 10%, respectively**.**

Table VIII. Oaxaca-Blinder Decomposition of gender differentials in Glycated Haemoglobin

	Q25	%	Q50	%	Q75	%	Q90	%	Q95	%
Δ_{M-F}	0.033***		0.032***		0.024**		0.073**		0.336***	
	0.006		*0.005*		*0.007*		*0.014*		*0.041*	
Compositional	-0.009***	-27	-0.015***	-47	-0.022***	-91	-0.030***	-41	-0.060***	-18
	0.002		*0.003*		*0.004*		*0.005*		*0.009*	
Elasticity	0.042***	127	0.047***	147	0.046***	191	0.103***	141	0.397***	118
	0.006		*0.005*		*0.007*		*0.013*		*0.040*	

Standard Errors in Italics; ***, **, * indicate significance at 1%, 5% and 10%, respectively.

Table IX. Oaxaca-Blinder Decomposition of gender differentials in Fibrinogen

	Q25	%	Q50	%	Q75	%	Q90	%	Q95	%
Δ_{M-F}	-0.330***		-0.25***		-0.381***		-0.244***		-0.103**	
	0.014		*0.014*		*0.019*		*0.029*		*0.04*	
Compositional	-0.022***	7	-0.027***	11	-0.033***	9	-0.034***	14	-0.033***	32
	0.004		*0.004*		*0.006*		*0.007*		*0.009*	
Elasticity	-0.307***	93	-0.223***	89	-0.348***	91	-0.209***	86	-0.069*	68
	0.014		*0.014*		*0.018*		*0.028*		*0.039*	

Standard Errors in Italics; ***, **, * indicate significance at 1%, 5% and 10%, respectively.

the coefficients (δ_F and δ_M) and the differences in coefficients between the regression on the subsample of men and the subsample of women ($\delta_F - \delta_M$). When the coefficients are negative, a positive (negative) elasticity effect arises when the coefficient of female regression is larger (smaller) in magnitude than the coefficient of male regression. On the contrary, when the coefficients are positive, a positive (negative) elasticity effect arises when the coefficient in the female regression is smaller (larger) than the coefficient in the male regression.[8] In the case of cholesterol, as shown in the previous section, the income coefficient in the pooled regression is positive at the lowest quantiles of cholesterol and negative from the 80th percentile of cholesterol. Thus, Figure 5 shows that there is an important heterogeneity in the association of cholesterol to income which varies significantly along the cholesterol distribution. From the 25th to the 75th percentile of the distribution, the positive contribution of income means a higher (positive) association of income to cholesterol among men, while at the 90th percentile, the positive contribution means that there is a higher income gradient among women. However, it is interesting to observe that at very high and dangerous levels of cholesterol (at the 95th percentile), we see a negative contribution that indicates a higher income gradient among men.

[8]The elasticity terms in Equation 5 is $\overline{X_F}(\delta_M - \delta_F)$. Thus, with negative (positive) coefficients (δ_F and $\delta_M < 0$), a positive elasticity effect arises if $\delta_F > \delta_M$ ($\delta_M > \delta_F$) being the sample mean $\left(\overline{X_F}\right)$ always positive in our case.

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Table X. Oaxaca-Blinder Decomposition of gender differentials in Ferritin

	Q25	%	Q50	%	Q75	%	Q90	%	Q95	%
Δ_{M-F}	0.288***		0.539***		0.847***		1.250***		1.591***	
	0.010		*0.015*		*0.023*		*0.050*		*0.069*	
Compositional	0.000	0.00	-0.003	-0.55	-0.009	-1.06	-0.018	-1.44	-0.018	-1.13
	0.001		*0.003*		*0.006*		*0.011*		*0.016*	
Elasticity	0.288***	100	0.542***	100.55	0.856***	101.06	1.269***	101.44	1.609***	101.13
	0.100		*0.014*		*0.022*		*0.049*		*0.069*	

Standard Errors in Italics ; ***, **, * indicate significance at 1%, 5% and 10%, respectively.

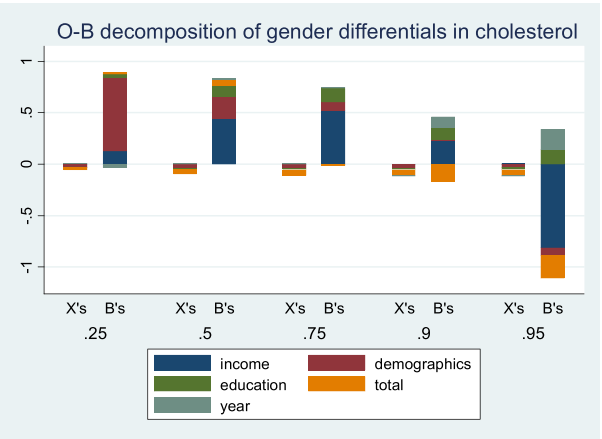

Figure 5. Detailed decomposition of gender differentials – Cholesterol. [Colour figure can be viewed at wileyonlinelibrary.com]

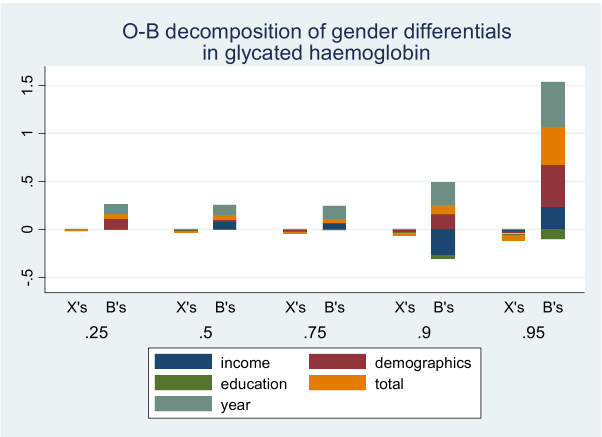

Figure 6. Detailed decomposition of gender differentials – Glycated haemoglobin. [Colour figure can be viewed at wileyonlinelibrary.com]

The OB decomposition of HbA1c is reported in Table VIII. Results indicate higher values of HbA1c among men along the entire distribution and especially at the extreme levels (at the 95th percentile). The higher prevalence of diabetes among men is a recent finding of the medical literature, and it is mainly attributed to a higher abdominal visceral fat, which represents one of the main risk factors for diabetes (see for instance Perreault *et al*., 2008). Contrary to cholesterol, gender differentials in HbA1c are largely

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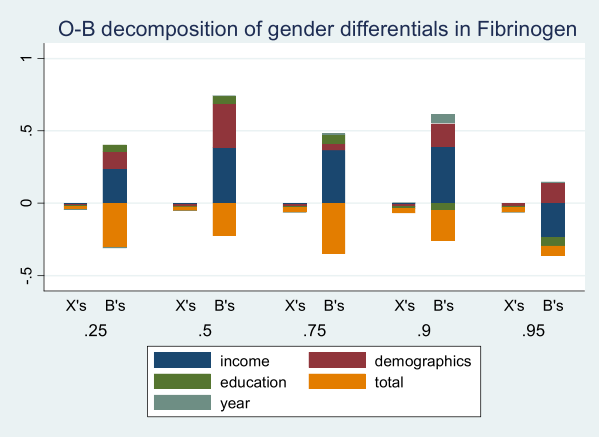

Figure 7. Detailed decomposition of gender differentials – Fibrinogen. [Colour figure can be viewed at wileyonlinelibrary.com]

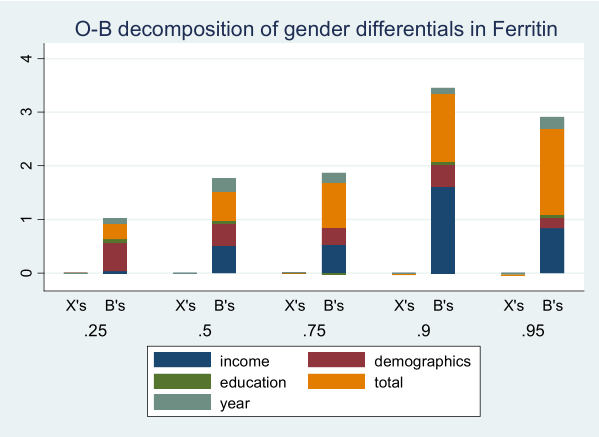

Figure 8. Detailed decomposition of gender differentials – Ferritin. [Colour figure can be viewed at wileyonlinelibrary.com]

due to a different association of HbA1c to the covariates (row 3 of Table VIII), while compositional differences are much less important to explain total gender differentials (row 2). Detailed contribution analysis in Figure 6 suggests a marginal role of demographics and a strong contribution of education (mainly due to an elasticity effect) to gender differentials. With respect to income, Figure 6 indicates that its contribution becomes predominant after the 75th percentile of the distribution. Also in the case of HbA1c, the contribution of income is largely due to gender-related differences in the association of the biomarker to income, which vary significantly along the distribution. For instance, while at the 90th percentile of the distribution, we found a higher income gradient among men, and we found the opposite (but less important in magnitude) at the 95th percentile of the distribution.

Table IX indicates higher levels of fibrinogen among women especially in the lower part of the distribution where levels are not indicative of a pathological state. Differences are largely explained by gender differentials in the association of fibrinogen with the covariates, while compositional differences are much less important. The detailed decomposition presented in Figure 7 shows that gender differentials in fibrinogen are largely explained by income, and by demographics (to a less extent), while education plays a less important role. Figure 7 also shows that the contribution of income is largely explained by an

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elasticity effect, a pattern observed also for the other biomarkers. Moreover, similarly to the other biomarkers, the elasticity is heterogeneous along the distribution. Up to the 90th percentile of the distribution, the income gradient is higher among women, while at very extreme levels of fibrinogen, the income gradient is higher among men.

Lastly, we report the OB decomposition of gender differentials in ferritin in Table X.[9] Results indicate the existence of higher ferritin levels among men along the entire distribution and especially at the 95th percentile of the distribution. These differences are partly physiological and partly associated to menstrual blood loss for women. However, decomposition analysis also suggests that differences are largely explained by an elasticity effect, while compositional differences are less important. Similar to the other biomarkers, we find that income is the largest contributing factor to gender differentials, and this happens along the entire distribution (Figure 8). Again, we find that the income contribution is mostly due to an elasticity effect. This indicates a higher income gradient among men at all levels. In substantive terms, these results suggest that the income gradient is higher among men for low ferritin problems, while we do not detect meaningful gender differentials at the highest levels of ferritin, and the income–ferritin association is less statistically significant at those levels (as discussed in Section 4.1).

5. CONCLUSIONS

The relationship between income and health is probably one of the most explored topics in health economics. A large literature documents the existence of a positive income gradient, which is found in several countries, across different age groups and according to several measures of health status. Despite this large interest, less is known about the income–health relationship at different points of the health distribution. Indeed, studies of the influence of economic conditions on health typically measure the effect of the former on the conditional mean of the health status variable through regression analysis. Analysis based solely on the mean while offering useful information, misses potentially important information in other parts of the distribution. For instance, it does not check for non-linearities in the relationship between income and health across the full conditional distribution. Moreover, it does not permit analysis of the role of economic conditions at the tails of the health distribution, which are often associated with large welfare losses for individuals and high costs for the health care system.

This paper fills this gap by offering new evidence on the income–health relationship 'beyond the mean' of the health distribution. We use a distributional method proposed in the recent literature, the RIF approach of Firpo *et al*. (2009), to estimate income gradients across the full distribution for continuous measures of objective health status: blood-based biomarkers. Moreover, we apply Oaxaca–Blinder decompositions at various quantiles of the biomarker distributions to explain gender differentials in biomarkers in England. We use repeated cross-sectional data from the Health Survey for England spanning from the 2003 to 2012, and we concentrate on four biomarkers that are predictive of some of the most prevalent non-communicable diseases: total cholesterol, HbA1c, fibrinogen and ferritin.

The principal scope of our paper is to perform a distributional analysis of the income gradient over the health status distribution. The endogeneity of income is not addressed in our analysis, and our results cannot be considered as causal. However, we use a measure of health that is free of subjective reporting bias. Indeed, biomarkers are health measures collected during a professional nurse visit and measured on a continuous scale. This rules out one important source of endogeneity in the income–health relationship.

With these features in mind, our analysis makes two important contributions to the existing literature. Firstly, analysis beyond the mean allows us to highlight aspects of the income–health relationship, which are overlooked by standard regression methods. In particular, we find that the income–health relationship is

[9]To take into account the differences in the measurement scale of ferritin with respect to others, we divided ferritin values by 100 in the decomposition analysis shown in Table X and Figure 8.

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non-linear across the health distribution and that the income gradients appear to be higher at the top quantiles of the biomarker distributions, close to the clinical cutpoints that indicate the presence of disease. For instance, we find that the income gradient at the 95th percentile is 10 times higher than income gradient at the 25th percentile of HbA1c, a marker for diabetes. At the same time, the income gradient at the 95th percentile is twice as much as the gradient at the 25th percentile of the distribution of fibrinogen, a marker of many inflammatory diseases, including cardiovascular illness. The income gradient increases almost linearly up to the 75th percentile of ferritin (a marker of anaemia and other important diseases), and it increases less than proportionally after this threshold. In these cases, the analysis at the mean provides a partial view of the income–health relationship. In the case of cholesterol, analysis at the mean leads to misleading conclusions. For instance, we find that OLS regression suggests a positive association between income and cholesterol. Instead, RIF regression suggests that at the lowest quantiles of the distribution, the income–cholesterol association is positive, while from the 75th percentile of the distribution, the relationship turns negative. Also in the case of cholesterol, the 'saddle' point is very close to the clinical threshold denoting pathologic cholesterol levels.

A second contribution of our paper is the measurement of the gender differentials in biomarkers and the assessment of the contribution of income (and other covariates) to these differentials. We find that, besides some physiological reasons, gender differentials in biomarkers are largely explained by a different association of health to covariates across genders. Importantly, detailed decomposition analysis suggests that the heterogeneity in the effect of income on health across genders accounts for a substantial percentage of the total gender differentials in observed health. Moreover, we find that income–health relationship across genders varies significantly along the biomarker distribution and that this depends on the nature of the biomarker considered. At extreme levels of biomarkers, indicating pathological cardiovascular diseases (for cholesterol and fibrinogen), we find a higher income gradient among men, while we find that the income gradient is higher among women at lowest quantiles of the distribution (at the 25th and 50th percentiles of the distribution). Similarly, at the lowest levels of ferritin, indicating iron deficiency anaemia, we find a higher income gradient among men. On the contrary, at extreme levels of HbA1c indicating severe diabetes, we find a higher income gradient among women.

These results might have important policy implications. If we follow the argument of Van Doorslaer and Koolman (2004), the importance of the gender-related differences in the association of income to health would suggest primarily health policy interventions, which operate on the health-gradient. At the same time, it seems that fiscal policy interventions are less relevant as compositional differences are not very important to explain the total gender differentials. Moreover, our analysis suggests that health policy interventions should be differently focused across genders and across the distribution of health. For instance, for the purpose of eliminating socio-economic inequalities in health, it seems important to focus on men in poor economic conditions when considering severe cardiovascular diseases or anaemia due to nutritional deficiency, for instance. On the contrary, more attention should be paid to women in poor economic conditions when considering severe diabetes.

Future research might concentrate on the reasons behind the heterogeneity of income–health relationship along the distribution of health status and across genders. Our results indicate that these aspects should be carefully considered when investigating the health gradient with respect to income, gender and other covariates.

REFERENCES

American Diabetes Association. 2010. Executive summary: standards of medical care in diabetes – 2010. *Diabetes Care* **33** (Suppl 1): S4–S10.

Atkinson A, Colburn WA, Degruttola VG, et al. 2001. Biomarkers and surrogate endpoints: preferred definitions and conceptual framework. Biomarkers Definition Working Group. *Clinical Pharmacology & Therapeutics* **69**: 89–95.

Bago d'Uva T, Van Doorslaer E, Lindeboom M, O'Donnell O. 2008. Does reporting heterogeneity bias the measurement of health disparities? *Health Economics* **17**(3): 351–375.

Banks J, Marmot M, Oldfield Z, Smith JP. 2006. Disease and disadvantage in the United States and in England. *Journal of American Medical Association* **295**(17): 2037–2045.
Bitler MP, Gelbach JB, Hoynes HW. 2006. What mean impacts miss: distributional effects of welfare reform experiments. *The American Economic Review* **96**: 988–1012.
Blinder AS. 1973. Wage discrimination: reduced form and structural estimates. *The Journal of Human Resources* **8**: 436–455.
Cawley J, Choi A. 2015. Health disparities across education: the role of differential reporting error, *NBER Working Paper*, 21317.
Cutler D, Deaton A, Lleras-Muney A. 2006. The determinants of mortality. *Journal of Economics Perspectives* **20**(3): 97–120.
Dowd JB, Goldman N. 2006. Do biomarkers of stress mediate the relation between socioeconomic status and health? *Journal of Epidemiology and Community Health* **60**: 633–639.
Ettner SL. 1996. New evidence on the relationship between income and health. *Journal of Health Economics* **15**(1): 67–85.
Firpo S, Fortin N, Lemieux T. 2009. Unconditional quantile regressions. *Econometrica* **77**(3): 953–973.
Fortin N, Lemieux T, Firpo S. 2011. Decomposition methods in economics. *Handbook of Labor Economics* **4**: 1–102.
Gruenewald TL, Seeman TE, Ryff CD *et al.* 2006. Combinations of biomarkers predictive of later life mortality, Proceedings of the National Academy of Sciences, 103:14158-14163.
Heckley G., Gerdtham, U.G., Kjellsson, G., (2016) A general method for decomposing the causes of socioeconomic inequality in health, *Journal of Health Economics*, in press (doi: j.jhealeco.2016.03.006).
Hernández-Quevedo C, Jones AM, Rice N. 2004. Reporting bias and heterogeneity in self-assessed health. Evidence from the British Household Panel Survey, Discussion Papers 18/2004, University of York.
Holland PW. 1986. Statistics and causal inference. *Journal of the American Statistical Association* **81**(396): 945–960.
Jann B. 2008. The Blinder–Oaxaca decomposition for linear regression models. *The Stata Journal* **8**(4): 453–479.
Jones AM, Lopéz Nicolás A. 2006. Allowing for heterogeneity in the decomposition of measures of inequality in health. *Journal of Economic Inequality* **4**: 347–365.
Jones AM, Lomas J, Rice N. 2015. going beyond the mean in healthcare cost regressions: a comparison of methods for estimating the full conditional distribution. *Health Economics* **24**: 1192–1212.
Juerges H, Kruk E, Reinhold S. 2013. The effect of compulsory schooling on health-evidence from biomarkers. *Journal of Population Economics* **26**: 645–672.
Kakwani NC, Wagstaff A, Van Doorslaer E. 1997. Socioeconomic inequalities in health: measurement, computation and statistical inference. *Journal of Econometrics* **77**(1): 87–104.
Mackenbach JP, Martikainen P, Looman, et al. 2005. The shape of the relationship between income and self-assessed health: an international study. *International Journal of Epidemology* **34**(2): 286–293.
Muennig P, Sohler N, Mahato B. 2007. Socioeconomic status as an independent predictor of physiological biomarkers of cardiovascular disease: evidence from NHANES. *Preventive Medicine* **45**: 35–40.
Neumark D. 1988. Employers' discriminatory behavior and the estimation of wage discrimination. *The Journal of Human Resources* **23**: 279–295.
Oaxaca R. 1973. Male-Female wage differentials in urban labor markets. *International Economic Review* **14**: 693–709.
Perreault L, Ma Y, Dagogo-Jack S, et al. 2008. Sex differences in diabetes risk and the effect of intensive lifestyle modification in the diabetes prevention program. *Diabetes Care* **31**: 1416–1421.
Ploubidis GB, Benova L, Grundy E, et al. 2014. Lifelong socio economic position and biomarkers of later life health: testing the contribution of competing hypotheses. *Social Science and Medicine* **119**: 258–265.
Rosero-Bixby LS, Dow WH. 2012. Predicting mortality with biomarkers: a population-based prospective cohort study for elderly Costa Ricans. *Population Health Metrics* **10**: 11.
Sattar N, Murra HM, Welsh P, et al. 2009. Are markers of inflammation more strongly associated with risk for fatal than for nonfatal vascular events? *Plos Medicine* **6**: 1–10.
Sen A. 2002. Health: perception vs observation. *British Medical Journal* **13**(324): 860–861.
Van Doorslaer E, Jones AM. 2003. Inequalities in self-reported health: validation of a new approach to measurement. *Journal of Health Economics* **22**: 61–87.
Van Doorslaer E, Koolman X. 2004. Exploring the differences in income-related health inequalities across European countries. *Health Economics* **13**: 609–628.
Wagstaff A, Van Doorslaer E, Watanabe N. 2003. On decomposing the causes of health sector inequalities with an application to malnutrition inequalities in Vietnam. *Journal of Econometrics* **112**(1): 207–223.
World Health Organisation. 2011a. Use of glycated haemoglobin (HbA1c) in the diagnosis of diabetes mellitus, World Health Organisation: Geneva.
World Health Organisation. 2011b. Serum ferritin concentrations for the assessment of iron status and iron deficiency in populations. Vitamin and mineral nutrition information system, World Health Organization: Geneva.

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