The University Of Sheffield.

Department Of Economics.

Sheffield Economic Research Paper Series.

Saving Behaviour and Biomarkers: A High-Dimensional Bayesian Analysis of British Panel Data

Sarah Brown, Pulak Ghosh, Daniel Gray, Bhuvanesh Pareek and Jennifer Roberts

ISSN 1749-8368

SERPS no. 2017005

February 2017

SAVING BEHAVIOUR AND BIOMARKERS: A HIGH-DIMENSIONAL BAYESIAN ANALYSIS OF BRITISH PANEL DATA

Sarah Brown[1], **Pulak Ghosh**[2], **Daniel Gray**[1]*, **Bhuvanesh Pareek**[3] **and Jennifer Roberts**[1]

Abstract: Using British panel data, we explore the relationship between saving behaviour and health, as measured by an extensive range of biomarkers, which are rarely available in large nationally representative surveys. The effects of these objective measures of health are compared with commonly used self-assessed health measures. We develop a semi-continuous high-dimensional Bayesian modelling approach, which allows different data-generating processes for the decision to save and the amount saved. We find that composite biomarker measures of health, as well as individual biomarkers, are significant determinants of saving. Our results suggest that objective biomarker measures of health have differential impacts on saving behaviour compared to self-reported health measures, suggesting that objective health measures can further our understanding of the effect of health on financial behaviours.

Key Words: Bayesian Modelling; Biomarkers; Household Finances; Saving; Two-Part Model.

JEL Classification: D12; D14.

Acknowledgements: We are grateful to the Data Archive at the University of Essex for supplying *Understanding Society* waves 1 to 4.

February 2017

[1] Department of Economics, University of Sheffield, Sheffield, UK;

[2] Indian Institute of Management (IIMB), Bangalore, India;

[3] Indian Institute of Management (IIM), Indore, India

* Corresponding Author: d.j.gray@sheffield.ac.uk, Department of Economics, University of Sheffield, Sheffield, S1 4DT, UK; Telephone: 0114 2229653.

1. Introduction

For many decades, saving behaviour has attracted extensive interest in the economics literature.[1] In particular, motivations for saving have been widely explored from both a theoretical and an empirical perspective. A commonly held view is that individuals are not saving enough. Indeed, the recent financial crisis has revealed the financial vulnerability faced by many households. For example, Garon (2012) comments that, in the US, *'it has become painfully clear that millions lack the savings to protect themselves against foreclosures, unemployment, medical emergencies, and impoverished retirements.'* The personal saving rate in the US has fallen from 8% in the 1980s to below 4% in 2011 (Donnelly *et al.*, 2012), increasing slightly to 5.2% in the third quarter of 2015 (US BEA, 2015). Similarly, in the UK, the household saving rate has halved since the middle of 2010 from 11.5% to 5.8% in the fourth quarter of 2015 (ONS, 2016).

Given the general consensus amongst policy-makers that individuals are not saving enough, it is important to further our understanding of the determinants of saving behaviour. Hence, we contribute to the existing empirical literature by exploring the relationship between saving and health. The literature has already paid some attention to this relationship, but previous work has been largely limited to representing health using self-reported measures; these are plagued by recall problems and reporting biases, and thus may not be appropriate measures of health risk. In contrast, this study uses three types of health measure and compares their role in determining saving behaviour and financial asset accumulation. Firstly, like the majority of the previous literature, we use self-reported or self-assessed health (SAH); secondly, we use a continuous index of overall health (the SF-12 index[2]), which is self-reported but more objective than SAH; and thirdly, we use a set of biomarkers, objective measurements taken by a nurse, which are commonly interpreted as important markers of future health, which are unaffected by recall issues or reporting bias. In addition, we contribute to the

[1] See Browning and Lusardi (1996) for a comprehensive review of household saving.
[2] The SF-12 is a multidimensional measure of health comprising 12 questions relating to issues such as pain, physical functioning, social functioning and mental health; a continuous preference based index of health related quality of life was created from the SF-12 by Brazier and Roberts (2004).

existing saving literature by developing a semi-continuous high-dimensional Bayesian modelling approach in order to account for the potentially different data generating processes underlying the decision to save and the decision regarding the amount saved. Bayesian modelling techniques have only been applied to household finances in a small number of papers (see, for example, Brown *et al.*, 2014, 2015, 2016), which is surprising given that the Bayesian approach allows flexible modelling in complex applications and hence seems to be ideally suited to modelling financial behaviour. We focus on modelling a measure of active saving, *i.e.*, the monthly flow into savings, as well as a stock measure, specifically the amount of financial assets held.

Our results show that a range of health measures, beyond SAH, have a significant impact on financial behaviours. Our results confirm those of the existing literature; that is, self-reported health measures are an important determinant of financial behaviours, with better SAH and SF-12 index scores being positively associated with both the decision to participate in saving and the amount saved. In addition, the composite biomarker measures of health, as measured by allostatic load, exert a significant impact on saving decisions. Specifically, worse health serves to reduce both the propensity to save and the amount saved. Furthermore, individual biomarkers including body mass index, waist circumference, triglyceride levels, markers of diabetes and markers of inflammatory load are all found to be statistically significant determinants of saving behaviour, with worse health associated with lower levels of monthly saving. Similar results are found when we explore the determinants of financial asset holding.

2. Background

The role of health has been widely discussed in the context of the precautionary saving motive, where individuals hold a contingency fund in case of adverse future events. For example, Lusardi (1998), who explores a sample of individuals aged 51 to 61 from wave one of the US Health and Retirement Survey, reports evidence in line with the theory of precautionary saving, suggesting that individuals who face higher income risk save more. She comments that, apart from income risk, health and

longevity risk can also be important and can provide useful insights to explain household wealth holdings in the US (Lusardi, 1998, p.453).

Further evidence supporting the importance of the precautionary saving motive is reported by Kennickell and Lusardi (2004), who analyse the 1995 and 1998 waves of the US Survey of Consumer Finances (SCF), which include a subjective measure of precautionary wealth accumulation. This information is elicited from responses to a question asking respondents to directly report their desired amount of precautionary wealth, that is, the level of savings a respondent believes they require to cover unanticipated emergencies. The results suggest that although a precautionary saving motive affects virtually every household, and especially older households, it does not lead to a large amount of wealth accumulated. The authors conclude that risks beside income risk should be taken into account when modelling saving behaviour; indeed, they find that, relative to other risks, health risk, measured by state level out-of-pocket health expenditure and whether respondents foresee expenses for health care in the next 5-10 years, leads to the largest amounts of precautionary savings overall. DeVaney *et al.* (2007) use data from the 2001 SCF to explore a hierarchy of savings motives derived from Maslow's hierarchy of needs (Maslow, 1943). They find that health has a significant effect on moving up the hierarchy from the lower levels of saving for basic needs and safety needs, but after that, when saving is for higher level needs, such as esteem and self-actualisation, health is no longer a significant determinant.

In their studies of the savings motives of households using the 2007 SCF, Fisher and Montalto (2010, 2011) find that those in poorer health are substantially less likely to save than those in good health. The causal pathway is unclear; however, they suggest that it could be because higher medical costs prevent savings, and/or the result of a shorter expected lifespan reducing the incentive to save.[3] Of course a further possible explanation is that, as well as effects via increased medical costs, poor

[3] Other studies that have explored longevity and health risk include Starr-McCluer (1996) and Hubbard *et al.* (1995).

health can also directly affect household income via its impact on employment and productivity (Haan and Myck, 2009).

Guariglia and Rossi (2004) use the 1996 to 2000 waves of the British Household Panel Survey (BHPS) to show that savings and health are related even in the context of a free universal health care system where everyone is in effect insured against unexpected health care expenditure. They argue that due to long waiting lists for free National Health Service treatments and indicators of poor quality treatment, a significant minority of the UK population hold private medical insurance.[4] Controlling for SAH and using instrumental variables to control for the potential endogeneity of insurance purchase, Guariglia and Rossi (2004) find that those with insurance have significantly higher savings than those without. However, there is evidence of crowding out of savings by private insurance in rural areas characterised by fewer health care providers, and also in those areas where residents feel that the quality of local health care providers is poor.[5]

The health measures used in the existing literature have been limited to SAH, largely due to data availability. One recent exception is Ricketts *et al.* (2013), who explore the relationship between health and willingness to save using the 2006 wave of the National Longitudinal Survey of Youth. They explore a variety of health measures such as health perception and physical and mental health, thus allowing comparison between subjective health measures, *i.e.* health perception and less subjective measures, such as diagnosed illnesses, and the SF-12 mental and physical health component scores. The importance of exploring different measures of health is apparent since they are found to have different effects on the willingness of individuals to save. Better health perception and higher physical and mental health scores are positively related to willingness to save, whilst diagnosed health problems and higher depression scores are associated with a lower willingness to save.

[4] Approximately 11% of the UK population had some form of private medical insurance in 2014. However, cover is rarely comprehensive; few policies offer maternity or mental health cover and none provide cover for accidents and emergency or primary care (Kings Fund, 2014).

[5] In a similar study for Italy, which also has a universal public health care system, Jappelli *et al.* (2007) show that in districts with lower quality health care, precautionary savings are higher.

To explore the precautionary saving motive, the health variables used should be measures of health risk. SAH, where respondents rate their own general health on a response scale from say *very poor* to *excellent*, has been extensively used in previous studies largely because it is commonly available in many household survey data sets. This measure is arguably a reasonable proxy for health risk since it may contain private information on health and health related behaviours that are predictive of future health and are known only to the respondent. Idler and Benyamini (1997), for example, show that SAH is predictive of mortality even after conditioning on objective measures of health. However, SAH measures are subject to well-known recall and reporting biases (Bound, 1991), and as a result they conflate health information with other potentially unobservable information on respondent characteristics much, such as labour market preferences and personality traits, which may also influence savings behaviour.

Unlike the majority of the existing literature, we do not rely solely on SAH but also include a comprehensive set of biomarkers to measure health. We compare these biomarkers to SAH, and also to the SF-12 index. Biomarkers are objective measures of health; they are directly measured traits that provide insight into the functioning of biological systems, so they can help to highlight the underlying mechanisms between health status and financial decisions. Recently, Boen and Yang (2016) explore the effects of wealth changes, as caused by the great recession, on biosocial functioning in older adults. They find that losses in wealth were associated with increases in systolic blood pressure and C-reactive protein (a marker for inflammatory load – see below). Biomarkers can involve the measurement of biological molecules from blood and urine samples, but they also include physical measures such as blood pressure, waist measurement and body mass index. Biomarkers provide important clinical information on disease status and can also detect sub-clinical disease, which may be below the threshold of individual perception; thus they can be interpreted as markers for future health (Lyons and Basu, 2012). Developments in 'field-friendly' methods for collecting biomarkers mean that they are becoming more common in household surveys. Of our three types of health measure, the biomarkers are wholly objective and SAH is wholly subjective; the SF-12 index

sits between these two, since while it is based on self-reported information it is derived from answers to twelve specific health related questions that are less prone to reporting biases than the overall health evaluation encapsulated in SAH.[6]

3. Data

We exploit data drawn from *Understanding Society*, the UK Household Longitudinal Study (UKHLS), which is the follow-up survey to the BHPS, a survey conducted by the Institute for Social and Economic Research from 1991 to 2008. The BHPS was replaced by *Understanding Society* in 2009; it is a national representative longitudinal survey of approximately 40,000 households in the UK, with face-to-face interviews carried out between January 2009 and January 2011 for wave 1.

In this study, we exploit the Health, Biomarkers and Genetics data collected from a nurse visit. This nurse health assessment was carried out after the main questionnaire of waves 2 and 3 for a sample of the General Population Sample and BHPS Sample of *Understanding Society*, respectively. The nurse visits resulted in a sample of approximately 12,000 individuals and provide information on a range of objective measures of health and health risks, which may be clinical precursors to chronic health conditions. Benzeval *et al.* (2014) provide a full description of the sample and health measures collected. In our analysis, we only consider working age individuals (18 to 65 years) in line with González and Özcan (2013), and those from the nationally representative General Population Sample.[7] Given the timing of the nurse visits and the availability of information on financial behaviours, we focus on financial behaviours contained in wave 4 of *Understanding Society*, which means we explore measures of saving behaviour post the nurse visits taking place. The biomarker health data collected after wave 2 for the General Population Sample is linked to financial behaviours reported in wave 4, and we exploit other health measures (SAH and SF-12) from wave 3 of *Understanding Society*. Using financial behaviours reported in wave 4 reduces the potential for

[6] For example, one SF12 question is: *How much of the time during the past 4 weeks have you felt clam and peaceful*?; responses are on a 6-point scale ranging from *all the time* to *none of the time.*

[7] The General Population Sample excludes both the ethnic minority boost sample and the BHPS sample. This maintains that the sample is nationally representative and avoids potential panel conditioning relating to BHPS members who have been surveyed for many waves.

reverse causality as the measurement of the biomarker and health variables pre-dates the financial outcome variables. This gives a total sample of 2,928 individuals with all relevant financial and health information, once missing values are dropped.

3.1 Financial Variables

We focus on the effects of health risk on monthly saving as measured by the responses to the following question: "*Do you save any amount of your income, for example, by putting something away now and then in a bank, building society, or Post Office account other than to meet regular bills? About how much, on average, do you manage to save a month?"* Thus, our measure of monthly saving is akin to active saving as defined as that part of wealth accumulation related to contributions to saving accounts.[8] Our approach is in line with Guariglia (2001), Guariglia and Rossi (2004), Rossi (2009) and Yoshida and Guariglia (2002) who explore household saving in the UK using the BHPS.[9] Given the skewed nature of the responses, following Gropp *et al.* (1997), we take the natural logarithm of positive values, and code responses of no saving to the first part of the question as zero. A significant proportion of the sample (approximately 53%) report that they do not save on a monthly basis. Given the high proportion of zero observations, we develop a two-part semi-continuous Bayesian model, detailed in Section 4 below, to allow for the possibility of different data generating processes for the decision to save and the amount saved, conditional on the individual deciding to save.

Information is also available on monthly private pension contributions, which can be regarded as longer-term saving. This information is derived from the following questions: "*Other than your main employer or occupational pension scheme, are you currently a member of any personal pension scheme or do you currently contribute to any personal pension scheme? Please include any Additional Voluntary Contribution scheme you may belong to."* If the individual responds yes, then

[8] In contrast, passive saving refers to wealth accumulation related to asset appreciation.

[9] A potential alternative empirical strategy would be to explore the ratio of monthly saving to household income as opposed to the absolute level of saving. For brevity and in line with the existing literature, we only present results relating to the absolute level of saving, whilst controlling for household income.

they are asked whether they *"Contribute regularly to personal pension?"* and *"How much do you usually contribute? What period does this cover?"* From the responses to this sequence of questions, a monthly amount of private pension contributions is calculated and is added to the value of amount saved. Hence, we analyse two measures of monthly saving: firstly, a measure which does not include private pension contributions and secondly a measure which includes both saving and private pension contributions in order to analyse a more comprehensive definition of saving. Summary statistics relating to the savings variables are presented in Table 1A and Figures 1 to 4 present the distributions of the two dependent variables.

As well as exploring monthly saving, which can be regarded as a flow into savings accounts, we also explore the effects of health status on the stock of financial assets held. Such analysis ties in with existing studies on health and wealth; for example, Adams *et al.* (2003) and Hurd and Kapteyn (2003) amongst other studies, generally find a positive association between better health and household wealth. Our measure of financial assets is defined from the question: *"I'd like to ask about any savings or investments you may have. Which of these savings accounts do you have, if any? Savings or deposit accounts, National Savings Accounts, ISA - cash only, ISA - stocks and shares, or PEPs, Premium Bonds or Other types of savings accounts."* Individuals are then asked the monetary amount held in each of these assets. The sum of these responses forms the dependent variable relating to the stock of financial assets held. In line with the saving variables, the natural logarithm of financial assets is taken to account for the skewed nature of the variable and, for those individuals reporting that they hold no financial assets, this variable takes the value of zero. The distributions relating to the distributions of financial assets both including and excluding zero financial asset holdings are presented in Figures 5 and 6, respectively, with summary statistics presented in Table 1A.

Finally, it is apparent that the stock of financial assets held may be related to the stock of debt held. Indeed, a small number of studies explore the relationship between debt and health. For example, Drentea and Lavrakas (2000) report that both credit card debt and stress regarding debt are inversely associated with good health and Brown *et al.* (2005) find that unsecured debt is inversely

related to psychological well-being. In addition, Keese and Schmitz (2014) report that a variety of debt measures are strongly correlated with satisfaction with health and mental health once unobserved individual heterogeneity is accounted for. Hence, we explore the robustness of our results relating to the effect of health on the stock of financial assets held by jointly modelling the stock of financial assets and the stock of debt held. The level of unsecured debt is generated from the response to the following questions: *"I would now like to ask you about any other financial commitments you may have apart from mortgages. For which, if any, of these items do you currently owe any money? Please do not include credit card and other bills being fully paid off in the current month"*; and *"About how much in total is owed?"* From the responses to these questions, in line with the other financial variables, we construct a variable which captures the natural logarithm of the level of unsecured debt. For those individuals reporting that they hold no unsecured debt, the variable takes the value of zero.

3.2 Health Measures

We explore a wide range of biomarkers collected during the nurse visit, which capture an extensive range of health outcomes. We classify the biomarkers into five groups relating to the underlying health problems and associated risk factors. Table A1 in the appendix provides more detail on all of the biomarkers used and Table 1B presents the associated summary statistics. In the subsequent analysis, following standard practice (see Table A1), a natural logarithm transformation of the biomarker measures is used in order to account for skewed distributions and to allow the results to be interpreted as the effect of a percentage change in the biomarker measure on the dependent variable.

Firstly, we consider eight biomarkers markers for general health. The first six of these are particularly associated with being overweight or obese; these are total cholesterol (CHL), high density lipoprotein (HDL), triglycerides (TRI) and glycated haemoglobin (HbA1c), as well as two anthropometric measures, body mass index (BMI) and waist measurement (WST). These markers are risk factors for a number of chronic conditions including cardiovascular disease (CVD), cancer, diabetes, disability at older age and decreased life expectancy (Musaad and Haynes, 2007). CHL (or 'bad cholesterol') is a risk factor for CVD, whereas HDL (or 'good cholesterol') is protective against

it (Gordon *et al*. 2007). Albert *et al.* (2006) found that CHL and TRI levels decreased, and HDL levels increased, with increasing levels of education, whilst, Ryff *et al.* (2006) report that well-being is positively correlated with HDL and negatively with the CHL-to-HDL ratio. We also include diastolic and systolic blood pressure (DBP and SBP) in this group. Excessive levels of either are indicative of hypertension, which is a major risk factor for stroke, myocardial infarction, heart failure, chronic kidney disease, cognitive decline and premature death (NICE, 2011). Hildrum *et al.* (2008) find that anxiety and depression are correlated with low SBP, whilst Seeman *et al.* (2008) find consistent income and education gradients in both DBP and SBP.

Secondly, we consider two markers, which are general indicators for inflammation and infection, C-Reactive protein (CRP) and fibrinogen (FIB); they are elevated due to the presence of chronic conditions such as diabetes, rheumatoid arthritis and heart disease, and have been shown to be predictive of CVD and mortality (Danesh *et al.*, 1998; Sesso *et al.*, 2003). Elevated CRP can also be the body's reaction to stress (Fuligni *et al.,* 2009) and, in addition, Jürges *et al.* (2013) find that both FIB and CRP are strongly positively correlated with education.

Thirdly, we consider seven measures of liver and kidney function: these are (for the liver); albumin (ALB), alkaline phosphatase (ALK), alanine transaminase (ALT), aspartate transaminase (AST), and gamma glutamyl transferase (GGT); and for the kidneys; creatinine (CRE) and urea (URE). All of these markers can be signs of general health and, in particular, poor liver function is associated with alcohol and drug use and obesity (van Beek, 2014). A number of these markers are also associated with chronic heart disease (Danesh *et al.*, 1998), and URE is indicative of arthritis (Kraus *et al.,* 2002). Böckerman *et al.* (2014) demonstrate a positive causal effect of CRE on employment and wages.

Fourthly, we analyse two measures which reflect the body's stores of iron, haemoglobin (HGB) and ferritin (FER). Low iron stores cause anaemia, which is indicative of poor nutrition and its subsequent health consequences (WHO, 2011). Anaemia is associated with longer hospitalization and a greater risk of mortality and CVD, especially among older people (Culleton *et al.,* 2006).

Furthermore, Basta *et al.* (1979) and Haas and Brownlie (2001) found that anaemia was significantly related to work capacity; as a result, the influence of anaemia may cause reductions in worker productivity which could influence saving behaviour.

Finally, we consider two hormones, insulin-like growth factor 1 (IGF) and dihydroepiandrosterone sulphate (DHE). IGF is associated with growth, development, muscle strength and cognition, and can be indicative of general diet as well as heart disease, diabetes, cancer (Troncoso *et al.*, 2014). DHE is implicated in cardiovascular health and all-cause mortality, especially in older men (Barrett-Connor *et al.*, 1986). As a consequence of DHE declining with age, (Šulcová *et al.*, 1997), there have been many studies which use it as a marker of the aging process and a potential indicator of longevity. Furthermore, Lennartsson *et al.* (2013) assert that DHE has been linked with psychosocial conditions and that it has a protective role during psychosocial stress. Given its relationship with a variety of health outcomes, DHE may also have a direct impact on a range of potential economic outcomes.

As well as these individual biomarkers, we also include three commonly used composite measurements which combine information from a set of biomarkers. Firstly, allostatic load is a summary measure representing the number of biomarkers falling in a high risk percentile (upper 25%) based on the sample distribution.[10] Figure 7 presents the distribution of this measure. Allostatic load represents the cumulative impact of stressors on the body's regulatory systems, with high load leading to poorer health outcomes (Seeman *et al.* 1997). Allostatic load is a comprehensive, multidimensional approach to assessing physiological function and prospective research has associated allostatic load at baseline with increased risk for all-cause mortality, CVD, and declines in cognitive and physical function (Seeman *et al.*, 2002). Secondly, an alternative health risk index is based on non-normal levels of the biomarkers (Levine and Crimmins, 2014). It is the sum of the binary variables indicating whether individuals have non-normal levels for each biomarker based on clinical guidance of cut-off

[10] For the purposes of calculating allostatic load, biomarker values that are decreasing in bad health are reversed.

points.[11] Higher values of the index are indicative of worse health, see Figure 8 for the distribution of this measure. Thirdly, a Z-score measure of allostatic load is entered in our model as a continuous explanatory variable, where an individual's Z-score represents the absolute value of the standardized distance between their level of a given biomarker and the population mean for that biomarker, see Figure 9. A score of 1 denotes whether the individual is either one standard deviation above or below the mean, and the scores are summed (Seplaki *et al.*, 2005). The Z-score differs from the simple allostatic load because it provides a continuous rather than categorical measure of physiological function and thus preserves more of the information from the individual biomarkers; the results from Seplaki *et al.* (2005) suggest that it may be a better predictor of a wider array of health outcomes.

As well as the biomarker data, we also use two alternative measures of health. Firstly, overall SAH is measured on a five-point scale classified from the question, "*In general, would you say your health is excellent/very good/good/ fair/poor"*; this variable is coded so that a higher score represents better health. Secondly, the SF-12 index (SF-12ind) is derived from answers to the twelve questions that make up the SF-12 measure of health related quality of life (Brazier and Roberts, 2004). The index is preference rated using results from a UK population sample to weight the different dimensions of health, such as pain and physical functioning. The result is a continuous index where one represents full-health and zero is equivalent to being dead.[12]

4. Methodology

As stated above, a substantial proportion of individuals in wave 4 of *Understanding Society* report that they do not save on a monthly basis. Such a finding is common in the household finance literature. Hence, many of the statistical models used in the existing literature treat components of household finances, such as savings or debt, as censored variables since they cannot have negative values. Consequently, a tobit approach has been commonly used to allow for this truncation (see, for

[11] For those biomarkers with no agreed clinical cut-off, the upper 25% based on the sample distribution is used, or expected ranges are used.

[12] Theoretically, the SF-12 index can take negative values, which are interpreted as very severe health states considered worse than being dead; however, these are rarely observed in practice.

example, Brown and Taylor, 2008). As discussed in Brown e*t al.* (2015), the problem with this approach lies in the possibility that the decision to save and the decision regarding the level of savings may be characterised by different influences.[13] Hence, we develop a Bayesian two-part model to model saving behaviour; by 'two-part', we refer to data generated from a response which is a mixture of true zeros and continuously distributed positive values (Olsen and Schafer, 2001; Tooze *et al.*, 2002). The two-part model allows for differences in the influences on participating in saving behaviour and on the amount saved on a monthly basis. Our Bayesian approach is highly flexible in the context of complex modelling behaviour and, hence, seems particularly appropriate for analysing financial decision-decision making.

4.1 A Semi-Continuous Bayesian Model

Semi-continuous data can be viewed as arising from two distinct stochastic processes; one governs the occurrence of zeros and the second determines the observed value given a non-zero response. The first process is commonly referred to as the occurrence or binary part of the variable, and the second is often termed the continuous part. Two-part mixture models are an ideal choice for such data, since they explicitly accommodate both data generating processes. A log-normal distribution is frequently chosen to model the non-zero values, giving rise to the Bernoulli-log-normal two-part model as follows:

$$f(y_i) = (1-\pi_i)1_{(y_i=0)} + [\pi_i \times \mathrm{LN}(y_i;\mu_i,\sigma^2)]1_{(y_i>0)};\ y_i \geq 0, 0 \leq \pi \leq 1 \qquad (1)$$

where y_i is the response for the dependent variable of the i^{th} individual, $\pi_i = \Pr(y_i > 0)$, and $\mathrm{LN}(y_i;\mu_i,\sigma^2)$ denotes the log-normal density evaluated at $(y_i > 0)$, μ_i and σ^2 denote the mean and variance of $\ln(y_i|y_i > 0)$, respectively.

[13] A double-hurdle model is an alternative econometric specification, which allows independent variables to have different effects on the probability of, for example, saving and on the level of saving if it is non-zero. A potential limitation of a double-hurdle model is finding suitable instruments in order to identify the model. The Bayesian approach described above does not suffer from this limitation. Furthermore, the double-hurdle model has not been extended to the multivariate case, which means that it cannot be used when jointly modelling different aspects of household finances (below we focus on jointly modelling financial assets and debt).

The key distinction between this two-part model and the standard tobit model lies in the fact that in the tobit model, the zeros arise from censoring of an underlying continuous variable y^* that falls below some threshold value, whereas, in semi-continuous models, the zeros are valid observed responses corresponding, for example, in our application, to not saving on a monthly basis. The tobit and two-part models also differ in that the former assumes a single underlying distribution for the data, whereas the latter is a mixture of two separate data generating processes, one for the zeros and one for the positive values.

Two-part models for semi-continuous data can be extended to the regression setting by incorporating predictors into each component of the model. For example, the Bernoulli-log-normal two-part model regression model is given by:

$$f(y_i) = (1-\pi_i)1_{(y_i=0)} + [\pi_i \times \text{LN}(y_i; \mu_i, \sigma^2)]1_{(y_i>0)};\ y_i \geq 0, 0 \leq \pi \leq 1 \quad (2a)$$

$$g(\pi) = g[\Pr(y_i > 0)] = \boldsymbol{x}_i\beta_1 \quad (2b)$$

$$\mu_i = E[\ln(y_i)\,|y_i > 0] = \boldsymbol{x}_i\beta_2 \quad (2c)$$

where in equation 2b, $\boldsymbol{x}_i$ represents the vector of explanatory variables for the binary part of the model and in equation 2c, $\boldsymbol{x}_i$ represents the set of explanatory variables for the continuous part of the model, and β_1 and β_2 are the associated parameters, respectively. In both parts of the model, in addition to the measures of health detailed in Section 3.2 above, we control for: gender; a quadratic in age; marital status as captured by variables indicating married, divorced, or widowed, with not in a relationship being the omitted category; highest level of education captured by variables indicating degree, other high level qualification, A-level, GCSE, or other qualification, while below GCSE level is the omitted category; the number of children present in the household; employment status indicating employed, self-employed or retired with not currently working being the omitted category; and the natural logarithm of monthly household income. We also include 11 regions of residence controls. In addition, we include two additional controls in the binary part of the model. The first additional control is a binary indicator for being in arrears in the last 12 months in at least one of; council tax,

household bills (such as electricity, gas, water rates, telephone) or housing payments (i.e. rent or mortgage). The second additional control is an eight-point index of material deprivation, which is based on responses indicating the number of areas where people state that they have difficulty finding the money for. These areas are namely: holidays; entertaining friends and family; shoes; keeping the house in a decent state of repair; contents insurance; furniture; and major electrical goods (such as a washing machine). Table 1C presents summary statistics for the explanatory variables used in the empirical analysis.

4.2 Elastic Net as Shrinkage Priors

To complete the Bayesian specification of the model, priors need to be assigned for all unknown parameters. Since the number of explanatory variables is large, the dimensionality of the vectors of regression parameters, β_1 and β_2 could also be quite large. As discussed in Belloni *et al.* (2012), this could lead to non-reliable estimation due to the high-dimensionality of the parameter space. In order to overcome this issue, shrinkage methods have been increasingly used.[14] Assume that $\beta = \{\beta_j; j = 1, 2, \dots, J\}$ is the set of high-dimensional covariates. A general hierarchical formulation of the shrinkage prior would then take the following form:

$$\beta_j|\tau_j^2 \sim N(0, \tau_j^2);\ \tau_j^2 \sim F \qquad (3)$$

where τ_j^2 is the variance parameter which has a prior F. In the existing statistics literature, different choices of *F* are made, which leads to different families of shrinkage prior. Belloni *et al.* (2012) used $F \sim \exp(\lambda^2/2)$ resulting in the well-known lasso prior, where λ is the shrinkage parameter. The lasso method is a commonly used and popular shrinkage prior that yields a high probability that an estimated parameter is near zero and also allows each coefficient to have a large effect. However, a major disadvantage of the lasso shrinkage method is that it fails to account for possible multicollinearity between the covariates. This is a serious drawback for two main reasons. Firstly, it

[14] It should be noted that in a high-dimensional setting, maximum likelihood procedures typically fail with unstable estimates with large variance. To address such problems, a number of shrinkage methods have been proposed.

is difficult to check for multicollinearity using variance inflation factors for all possible pairwise covariates and, secondly, another issue of high-dimensional covariates concerns spurious correlation, which can impose multicollinearity even if there is no theoretical basis for the presence of correlation. Thus, we need a prior which not only performs the shrinkage, but which is also robust in the presence of multicollinearity. The Bayesian elastic net as proposed by Zou and Hastie (2005) performs shrinkage even when there are unknown groups of multicollinear predictors. Thus, we use a Bayesian elastic net prior as follows:

$$\beta_j|\tau_j^2 \sim N\left(0, \tau_j^2\right);\ \tau_j^2 \sim F \tag{4a}$$

$$F = (w_j^{-2} + \lambda_2)^{-1};\ w_j^2 \sim \exp(\lambda_1^2/2) \tag{4b}$$

$$\lambda_1^2 \sim Gamma(a, b);\ \lambda_2^2 \sim Gamma(c, d). \tag{4c}$$

We use the same kind of prior for both $\beta_1$and β_2. We use this two-part approach to model our two measures of monthly savings behaviour (i.e. excluding and including private pension contributions) as well as the measure of the stock of financial assets held.

4.3 A Copula Approach for Joining the Financial Assets and Debt Two-Part Models

Interdependence is likely to exist between a range of financial behaviours, such as asset accumulation and debt holding (see, for example, Brown and Taylor, 2008, and Brown *at al.*, 2015). Hence, to explore the robustness of our findings, we jointly estimate the two-part model of financial asset holding with a two-part model of unsecured debt. In this section, we develop a copula approach for joining the two-part model of financial asset holding with a two-part model of unsecured debt. In line with monthly saving and the stock of financial assets held, the two-part model of unsecured debt allows for the semi-continuous nature of unsecured debt holding and allows explanatory variables to have different influences on the probability of holding unsecured debt and on the amount of unsecured debt (*i.e.* the continuous part of the variable).

Let $r_{i,1}$ and $r_{i,2}$ be two random binary variables indicating whether individual *i* holds financial assets and holds unsecured debt, respectively. Conditional on $r_{i,1} = 1$ and $r_{i,2} = 1$, let $y_{i,1}$ and $y_{i,2}$ be

two variables denoting the amount of financial assets held and the amount of unsecured debt, respectively. Thus, the joint distribution of the two dependent variables, financial assets and unsecured debt, can be decomposed as follows:

$$f(r_{i,1}, r_{i,2}, y_{i,1}, y_{i,2}) = f_B(r_{i,1}, r_{i,2}) \times f_A(y_{i,1}, y_{i,2} | r_{i,1}, r_{i,2}) \quad (5)$$

where f_B denotes the joint distribution of the binary random variables and f_A denotes the joint distribution of the respective amounts held. Following Frees and Sun (2010), we employ a bivariate probit regression for $f_B(r_{i,1}, r_{i,2})$ and a copula model for the joint distribution of $f_A(y_{i,1}, y_{i,2} | r_{i,1}, r_{i,2})$.

To model $f_B(r_{i,1}, r_{i,2})$, we have four possibilities:

$$r_{i,1} = 1, r_{i,2} = 1 \Rightarrow f_B(r_{i,1}, r_{i,2}) = \Phi_2(\boldsymbol{x}_i^T \zeta_1, \boldsymbol{x}_i^T \zeta_2; \rho) \quad (6a)$$

$$r_{i,1} = 1, r_{i,2} = 0 \Rightarrow f_B(r_{i,1}, r_{i,2}) = \Phi(\boldsymbol{x}_i^T \zeta_1) - \Phi_2(\boldsymbol{x}_i^T \zeta_1, \boldsymbol{x}_i^T \zeta_2; \rho) \quad (6b)$$

$$r_{i,1} = 0, r_{i,2} = 1 \Rightarrow f_B(r_{i,1}, r_{i,2}) = \Phi(\boldsymbol{x}_i^T \zeta_2) - \Phi_2(\boldsymbol{x}_i^T \zeta_1, \boldsymbol{x}_i^T \zeta_2; \rho) \quad (6c)$$

$$r_{i,1} = 0, r_{i,2} = 0 \Rightarrow f_B(r_{i,1}, r_{i,2}) = 1 - \Phi(\boldsymbol{x}_i^T \zeta_1) - \Phi(\boldsymbol{x}_i^T \zeta_2) - \Phi_2(\boldsymbol{x}_i^T \beta_1, \boldsymbol{x}_i^T \beta_2; \rho) \quad (6d)$$

where Φ_2 is the cumulative density function (*CDF*) of a standard bivariate normal distribution with correlation parameter, ρ, and (6a) is the case where the individual holds both financial assets and debt, (6b) is the case where the individual holds financial assets and no debt, (6c) is the case where the individual holds no financial assets but holds debt, and (6d) is the case where the individual holds neither financial assets nor debt.

To model the joint distribution of the amounts held, $f_A(y_{i,1}, y_{i,2})$, we use a copula method to connect the joint marginal distributions. Thus, we write,

$$f_A(y_{i,1}, y_{i,2} | r_{i,1}, r_{i,2}) = f_{FA}(y_{i,1}) \times f_D(y_{i,2}) \times c(F_{FA}(y_{i,1}), F_D(y_{i,2}) \quad (7)$$

where $F_{FA}(y_{i,1})$ is the *CDF* corresponding to $f_{FA}(y_{i,1})$ and $F_D(y_{i,2})$ is the *CDF* corresponding to $f_D(y_{i,2})$, where the subscript *FA* denotes financial assets and the subscript *D* denotes debt, respectively. We assume a log-normal distribution for $f_{FA}(y_{i,1})$ and $f_D(y_{i,2})$, as follows:

$$f_{FA}(y_{i,1}) \sim \text{LN}(y_{i,1};\ \mu_{i,1}, \sigma_1^2) \quad (8a)$$

$$\mu_{i,1} = \boldsymbol{x}_i^T \boldsymbol{\eta}_1 \quad (8b)$$

$$f_D(y_{i,2}) \sim \text{LN}(y_{i,2};\ \mu_{i,2}, \sigma_2^2) \quad (8c)$$

$$\mu_{i,2} = \boldsymbol{x}_i^T \boldsymbol{\eta}_2. \quad (8d)$$

For the covariates $\zeta_1, \zeta_2, \boldsymbol{\eta}_1, \boldsymbol{\eta}_2$, we use the Bayesian elastic net as detailed in Section 4.2 above. Finally, $c(.,.)$ denotes the probability density function of the Gaussian copula distribution, with the partial derivative of the *CDF* as follows:

$$c(u_1, u_2) = \frac{\delta^2 C(u_1, u_2)}{\delta u_1 \delta u_2}. \quad (9)$$

Here $C(u_1, u_2)$ denotes the *CDF* of the Gaussian copula, which in our application is a bivariate Gaussian copula with parameter ϱ, as defined by application of Sklar's theorem (Nelsen, 2006):

$$C(u_1, u_2) = \Phi_2\left(\Phi^{-1}(u_1), \Phi^{-1}(u_2); \varrho\right) \quad (10)$$

where Φ is the *CDF* of the standard normal distribution and $\Phi_2(\cdot,\cdot;\varrho)$ is the *CDF* of the bivariate standard normal distribution with the correlation coefficient denoted by $\varrho \in (-1,1)$. In our application, $(u_1, u_2) = (F_{FA}(y_{i1}), \text{F}_D(\text{y}_{i2}))$. The measure of concordance for the bivariate Gaussian copula is dependent on the correlation coefficient (ϱ), as follows:

$$\upsilon = \frac{2}{\pi}\arcsin(\varrho) \quad (11)$$

Thus, the copula approach detailed above allows us to explore the robustness of our results relating to the effect of health on the stock of financial assets held to jointly modelling both financial asset and debt holding.

5. Results

We initially explore the results relating to the semi-continuous model of monthly saving behaviour. Specifically, we consider the effects of health status on our two measures of monthly saving behaviour; firstly, saving and, secondly, saving plus private pension contributions. We then explore the influence of health status on the stock of financial assets held, and, finally, we estimate a joint model of financial asset and unsecured debt holding. It should be noted that the health measures are included individually, in conjunction with the additional independent variables outlined in Table 1C.

5.1 Semi-Continuous Bayesian Model of Monthly Saving

The estimated coefficients and corresponding marginal effects relating to the demographic variables and health measures from estimating the semi-continuous model of monthly saving are presented in Tables 2A and 2B, respectively.[15] Table 3 presents the results relating to the case where the dependent variable includes monthly saving and private pension contributions; for brevity, in Table 3, we only present the results relating to the health variables.

Considering the results for the demographic and socio-economic variables in Table 2A shows that, in line with the existing literature, education, gender, family composition, employment status and household income all have significant impacts on saving behaviour. Specifically, education has a significant positive impact on the amount saved, but not on the decision to save. Compared to having below GCSE level of education, individuals who hold a degree save 86.4% more. Furthermore, females save a lower amount than their male counterparts as do people with dependent children; specifically, compared to males, females save 25.8% less, whilst an increase in one child reduces the amount saved by 15.8%. In addition, as expected, income exerts a positive impact on both the decision to save and the amount saved, with a 1% increase in income increasing savings by 0.6%. Material deprivation decreases the likelihood of saving, whilst being in arrears does not have a significant impact on saving decisions.

Table 2B presents the effects of health status on saving. The results indicate that both SAH and the SF-12 index have significant impacts on saving behaviours. Better health is associated with both an increased likelihood of saving and the amount saved; for example, a 0.1 unit increase in the SF-12 score increases saving by 15.5%. Considering the impact of SAH on saving behaviour reveals that a change from *poor* health to *fair* health (the lowest two categories) has a greater impact on saving behaviour than a change from *very good* to *excellent* health (the highest two categories).

[15] For comparison to the existing literature relating to saving behaviour, Table 2A does not include any health measures. Table 2B independently includes each measure of health, in conjunction with the variables presented in Table 2A. Once the health measures are sequentially included, the estimated coefficients relating to the demographic variables remain similar, that is, they display similar magnitudes and significance levels.

Specifically, the results indicate that the change from *poor* to *fair* health increases saving by 15.1% whilst a change from *very good* to *excellent* health increases saving by 9.2%.

We now focus on our key contribution, the effects of the objective measures of health status as measured by the biomarkers. We initially explore the composite biomarker measures and then discuss the individual health markers, which are statistically significant. It is clear that the three composite measures are inversely related to both the decision to save and the amount saved. This is consistent with the effects of SAH and the SF12 index, where better health is positively associated with saving behaviours. For example, taking the measure of allostatic load based on clinical cut-offs, shows that a one-point increase in this measure (indicating worse health) reduces saving by 8.2%, whilst, a one-point increase in the index of allostatic load reduces saving by 4.6%. Considering the continuous Z-score measure indicates that a one standard deviation increase in this results in a 2.8% decrease in saving.

Focusing on the effects of the individual biomarkers reveals that both BMI and waist measurement are inversely related to saving behaviour; both influence the amount saved, where a 1% increase in BMI causes a 0.2% decrease in the amount saved. Furthermore, both the levels of TRI and HbA1c (markers for general health) are inversely related to the amount saved, and CRP, an indicator of stress and inflammation, is associated with lower savings levels. Finally, measures of kidney and liver function, potential indicators of poor lifestyles, have significant impacts on saving behaviours; specifically, GGT and CRE have a negative impact on the amount saved.

Turning our attention to Table 3, where monthly private pension contributions are incorporated into the dependent variable, reveals a similar story to Table 2B. Specifically, both SAH and the SF-12 index maintain a significant positive relationship with saving behaviours. The magnitudes of the impacts are also similar: for example, considering the effects of SAH reveals that moving from *poor* to *fair* health, and from *very good* to *excellent* health, increases saving and private pension contributions by 12.7% and 8.1%, respectively; whilst a 0.1 unit increase in the SF-12 index increases saving and private pension contributions by 13.6%. In addition, all three measures of

allostatic load remain statistically significant determinants of saving behaviour with worse health associated with failing to save and saving lower amounts. A one-point increase in allostatic load, indicating worse health, based on the clinical cut-offs reduces saving by 8.5%, a similar magnitude to when savings is considered independently of private pension contributions. In addition, increases in the two alternative measures of allostatic load suggest that poorer health is associated with reductions in saving. The results relating to GGT and CRE, in contrast to those presented in Table 2B, fail to have statistically significant impacts on saving, whilst TRI, HbAc1, BMI, WST and CRP all maintain a similar negative impact on saving behaviours. HbA1c and CRP influence the amount saved, whilst ALKP and GGT influence the decision to save. Exploring the magnitudes reveals that a 1% increase in BMI and WST measurement is associated with a 0.20% and 0.24% decrease in saving, respectively.

5.2 Semi-Continuous Bayesian Model of the Stock of Financial Assets

Table 4 presents the results for the effect of health on the stock of financial assets held, and these suggest that health plays an important role here as well. Specifically, better SAH is associated with a higher propensity to hold financial assets and a higher amount of assets held. In line with the prior results relating to saving, there is a larger impact at the bottom of the SAH distribution compared to the top. The largest increase in financial asset holding results from a change from *poor* to *fair* health (32.1%), whilst the smallest effect is from a change from *very good* to *excellent* health (28.6%). Likewise, the SF-12 index exerts a positive impact, with a 0.1 point increase in the index being associated with a 16.7% increase in financial asset holding.

Considering the biomarker measures of health once again reveals that the composite measures of allostatic load are significant determinants of financial asset holding; specifically, all three measures are inversely related to the amount of assets held, that is, poorer health is associated with a lower level of financial asset holding. Looking at the magnitudes shows that a unit increase in the allostatic load index is associated with a 14.5% decrease in financial assets, which is very similar to that associated with the measure based on clinical cut-offs (14.3%). In addition, a 1 standard deviation

increase in the Z-score reduces financial assets by 7.1%. Moreover, higher levels of HDL (TRI) are positively (inversely) associated with financial asset holding and the amount of financial assets held. Higher values of BMI and WST are associated with lower levels of financial asset holding. Interestingly, both measures of inflammation, CRP and FIB, are inversely related to the amount of financial assets held, but not related to the decision to hold financial assets. This indicates that stress and inflammation adversely influence financial asset accumulation. Furthermore, both hormone measures are positively associated with financial asset holding, that is they influence the decision to hold financial assets, but fail to have an impact on the level of financial assets held.

Overall these results suggest that the largest effects of health on saving behaviour are represented by SAH, followed by the SF12 index and then by the composite biomarker measures. These relative effects are consistent with the argument that SAH (and to a lesser extent the SF12 index), due to its subjective nature, conflates biomedical health information with other information on respondent characteristics and/or individual health perception. The effect sizes suggest that these additional effects are important influences on saving behaviour, in addition to the biomedical information that the biomarkers provide.

5.3 Joint Bayesian Model of Financial Assets and Debt

Table 5 presents the results for the health measures from the joint model of financial assets and unsecured debt holding. In line with the previous results, allostatic load is inversely related to the amount of financial assets held, while worse health is associated with an increased likelihood of holding unsecured debt. The results, however, indicate that the measures of allostatic load do not have a significant impact on the level of debt held. Considering the individual biomarkers reveals that the results from the joint modelling approach relating to the household's financial assets accord with the semi-continuous model discussed above. Specifically, CRP and FIB, BMI and WST, and TRI, HDL and HbA1c are all significant determinants of financial asset holding. Biomarkers of general health have a significant impact on debt holding. Specifically, both DBP and SBP are positively associated with the amount of unsecured debt, whilst BMI and waist values both increase the likelihood of

holding unsecured debt. In addition, there is some evidence that liver function and kidney function tests are predictive of unsecured debt holding. For example, GGT and CRE are associated with an increased probability of unsecured debt holding. This positive association between poor health and debt is in line with the existing literature; see for example, Jenkins *et al.* (2008).

The results relating to SAH reveal that better health is associated with an increased likelihood of holding financial assets, holding a higher amount of financial assets and a lower likelihood of holding unsecured debt. In contrast, the SF-12 index does not have a statistically significant impact on unsecured debt accumulation, but is found to influence the amount of financial assets held. Neither measures of SAH influence the amount of unsecured debt held, only the decision to hold this type of debt.

6. Conclusion

Individual health status has been included as an explanatory variable in existing studies which explore the determinants of saving and financial decision making. Typically, and largely due to data availability, SAH forms the basis of these health measures. This paper has contributed to the literature by exploring the effects of more objective measures of health, as measured by biomarkers, on household financial decision making. In addition, we have developed a flexible Bayesian semi-continuous framework to analyse the effects of biomarkers on saving behaviour. Such a modelling framework is applicable where there is a prior belief that there are separate decision making processes relating to the decision to save and the amount saved. Our findings have confirmed that such an approach is highly appropriate with biomarkers found to have distinct effects across the two decisions, thereby endorsing the use of such a flexible framework for analysing financial behaviour.

The results confirm that health status is an important determinant of household financial decision making. In line with the existing literature, better SAH has a positive influence on saving behaviour. In addition, we document that more objective health measures also influence saving and financial decision making. We consistently find that composite measures of health status, as measured by allostatic load, have the expected impacts. In addition, we find that individual biomarkers,

specifically those relating to general health, and particularly those reflecting chronic conditions like diabetes risk and overweight status, are strong predictors of financial behaviours. Furthermore, the joint modelling approach revealed that SAH, measures of allostatic load and individual biomarkers displayed significant relationships with both financial asset accumulation and unsecured debt holding.

Our results make a useful contribution to the very small amount of existing evidence available for the UK. We confirm that health has a significant impact on financial behaviour and we find no support for precautionary savings motives, which predict that, to the extent that poor health contributes to higher income risk, individuals with poorer health will save more; our results, both for SAH and biomarkers, show the opposite effects. One explanation is that incentives for precautionary savings are weakened in the UK institutional context of universally provided health care insurance, which is very different to the US where most of the evidence emerges from. Another explanation is that not all of the information contained in biomarkers will be known to the individuals concerned; BMI and WST for example may be known but not perceived as a problem, and further some of the blood and urine markers may not even be known, unless they have been tested and the results explained. So while these biomarkers are good clinical markers of health risk, they may not affect perceived health risk, until that time they become apparent to the individual concerned and the information contained in them is explained.

The analysis presented in this paper demonstrates that biomarkers can provide important additional information beyond self-reported health measures. Moreover, these health measures can potentially reveal important underlying pathways between health and financial decision making. From a wider perspective, the increasing availability of biomarkers in secondary data sources provides a wide array of opportunities to incorporate objective health measures into economic research. These measures potentially allow the exploration of underlying mechanisms and the effect of more specific health conditions on economic outcomes. Future research, given data availability could focus on changes of objective health measures over time so it would be possible to account for

unobserved individual heterogeneity, given that this might be important in both financial decision making and individual health status.

References

Adams, P., Hurd, M.D., McFadden, D., Merrill, A. & T. Ribeiro (2003). Healthy, Wealthy, and Wise? Tests for Direct Causal Paths between Health and Socioeconomic Status. *Journal of Econometrics*, *112*(1), 3-56.

Albert, M.A., Glynn, R.J., Buring, J. & P.M. Ridker (2006). Impact of Traditional and Novel Risk Factors on the Relationship between Socioeconomic Status and Incident Cardiovascular Events. *Circulation*, *114*(24), 2619-2626.

Barrett-Connor, E., Khaw, K.T. & S.S. Yen (1986). A prospective Study of Dehydroepiandrosterone Sulfate, Mortality, and Cardiovascular Disease. *New England Journal of Medicine*, *315*(24), 1519-1524.

Basta, S.S., Karyadi, D. & N.S. Scrimshaw (1979). Iron Deficiency Anaemia and the Productivity of Adult Males in Indonesia. *American Journal of Clinical Nutrition*, *32*(4), 916-925.

Belloni, A., Chen, D., Chernozhukov, V. & C. Hansen (2012). Sparse Models and Methods for Optimal Instruments with an Application to Eminent Domain. *Econometrica*, *80*(6), 2369-2429.

Benzeval, M., Davillas, A., Kumari, M. & P. Lynn (2014). *Understanding Society:* UK Household Longitudinal Study: Biomarker User Guide and Glossary. Colchester: University of Essex.

Bockerman, P., Bryson, A., Hakulinen, C., Pehkonen, J., Pulkki-Raback, L., Raitakari, O. & J. Viinikainen, (2014). Biomarkers and Long-term Market Outcomes: The Case of Creatine, No. dp1279. *Centre for Economic Performance, LSE.*

Boen, C. & Y.C. Yang (2016). The Physiological Impacts of Wealth Shocks in Late Life: Evidence from the Great Recession. *Social Science and Medicine, 150*, 221-230.

Bound, J. (1991). Self-reported versus Objective Measures of Health in Retirement Models. *Journal of Human Resources*, 26, 106–138.

Brazier, J.E. & J. Roberts (2004). Estimation of a Preference-based Index Measure of Health for the SF-12. *Medical Care*, 42(9), 851-859

Brown, S. & K. Taylor (2008). Household Debt and Financial Assets: Evidence from Germany, Great Britain and the USA. *Journal of the Royal Statistical Society: Series A (Statistics in Society)*, *171*(3), 615-643.

Brown, S., Ghosh, P. & K. Taylor (2014). The Existence and Persistence of Household Financial Hardship. *Journal of Banking and Finance*, 46, 285-298.

Brown, S., Ghosh, P. & K. Taylor (2016). Household Finances and Social Interaction: Bayesian Analysis of Household Panel Data. *Review of Income and Wealth*, 62(3), 467-488.

Brown, S., Ghosh, P., Su, L. & K. Taylor (2015). Modelling Household Finances: A Bayesian Approach to a Multivariate Two-Part Model. *Journal of Empirical Finance, 33*, 190-207.

Brown, S., Taylor, K. & S.W. Price (2005). Debt and Distress: Evaluating the Psychological Cost of Credit. *Journal of Economic Psychology*, *26*(5), 642-663.

Browning, M. & A. Lusardi (1996). Household Saving: Micro Theories and Micro Facts. *Journal of Economic Literature*, 24, 1797-1855.

Bureau of Economic Analysis, U.S. Department of Commerce (2015). Personal Income and Outlays: November 2015, http://www.bea.gov/newsreleases/national/pi/2015/pi1115.htm.

Culleton, B.F., Manns, B.J., Zhang, J., Tonelli, M., Klarenbach, S. & B.R. Hemmelgarn (2006). Impact of Anaemia on Hospitalization and Mortality in Older Adults. *Blood*, *107*(10), 3841-3846.

Danesh, J., Collins, R., Appleby, P. & R. Peto (1998). Association of Fibrinogen, C-reactive Protein, Albumin, or Leukocyte count with Coronary Heart Disease: Meta-Analyses of Prospective Studies. *Journal of the American Medical Association*, 279, 1477–1482.

Devaney, S.A., Anong, S.T. & S.E. Whirl, (2007). Household Savings Motives. *Journal of Consumer Affairs*, 41, 174–186.

Donnelly, G., Iyer, R. & R.T. Howell (2012). The Big Five Personality Traits, Material Values, and Financial Well-being of Self-Described Money Managers. *Journal of Economic Psychology*, *33*(6), 1129-1142.

Drentea, P. & P.J. Lavrakas (2000). Over the Limit: The Association among Health, Race and Debt. *Social Science and Medicine*, *50*(4), 517-529.

Fisher, P.J. & C.P. Montalto (2010). Effect of Saving Motives and Horizon on Saving Behaviors. *Journal of Economic Psychology*, 31, 92–105.

Fisher, P.J. & C.P. Montalto (2011). Loss Aversion and Saving Behavior: Evidence from the 2007 U.S. Survey of Consumer Finances. *Journal of Family Economic Issues* 32, 4–14.

Frees, E.W. & Y. Sun (2010). Household Life Insurance Demand: A Multivariate Two-Part Model. *North American Actuarial Journal*, *14*(3), 338-354.

Fuligni, A.J., Telzer, E.H., Bower, J., Irwin, M.R., Kiang, L. & S.W. Cole (2009). Daily Family Assistance and Inflammation among Adolescents from Latin American and European Backgrounds. *Brain, Behavior, and Immunity*, *23*(6), 803-809.

Garon, S. (2012). *Beyond our Means: Why America Spends while the World Saves*. Princeton University Press.

González, L. & B. Özcan, (2013). The Risk of Divorce and Household Saving Behavior. *Journal of Human Resources*, *48*(2), 404-434.

Gordon, T., Castelli, W.P., Hjortland, M.C., Kannel, W.B. & T.R. Dawber (2007). High Density Lipoprotein as a Protective Factor against Coronary Heart Disease: The Framingham Study. *American Journal of Medicine*, 62,707-714.

Gropp, R., Scholz, J.K. & M.J. White (1997). Personal Bankruptcy and Credit Supply and Demand. *The Quarterly Journal of Economics*, 217-251.

Guariglia, A. & M. Rossi (2004). Private Medical Insurance and Saving: Evidence from the British Household Panel Survey. *Journal of Health Economics*, *23*(4), 761-783.

Guariglia, A. (2001). Saving Behaviour and Earnings Uncertainty: Evidence from the British Household Panel Survey. *Journal of Population Economics*, *14*(4), 619-634.

Haan, P. & M. Myck (2009). Dynamics of Health and Labor market risks. *Journal of Health Economics*, 28(6), 1116-1125.

Haas, J.D. & T. Brownlie (2001). Iron Deficiency and Reduced Work Capacity: A Critical Review of the Research to Determine a Causal Relationship. *The Journal of nutrition* 131, no. 2 (2001), 676S-690S.

Hildrum, B., Mykletun, A., Holmen, J. & A.A. Dahl (2008). Effect of Anxiety and Depression on Blood Pressure: 11-year Longitudinal Population Study. *The British Journal of Psychiatry, 193*(2), 108-113.

Hubbard, G., Skinner, J. & S. Zeldes (1995). Precautionary Saving and Social Insurance. *Journal of Political Economy*, 103, 360-399.

Hurd, M. & A. Kapteyn (2003). Health, Wealth, and the Role of Institutions. *Journal of Human Resources, 38*(2), 386-415.

Idler E.L. & Y. Benyamini (1997). Self-rated Health and Mortality: A review of Twenty-Seven Community Studies. *Journal of Health and Social Behaviour* 38(1), 21–37

Jappelli, T., Pistaferri, L. & G. Weber (2007). Health Care Quality, Economic Inequality, and Precautionary Saving. *Health Economics,* 16, 327–346.

Jenkins, R., Bhugra, D., Bebbington, P., Brugha, T., Farrell, M., Coid, J. & H. Meltzer (2008). Debt, Income and Mental Disorder in the General Population. *Psychological Medicine, 38*(10), 1485-1493.

Jürges, H., Kruk, E. & S. Reinhold (2013). The Effect of Compulsory Schooling on Health—Evidence from Biomarkers. *Journal of Population Economics*, *26*(2), 645-672.

Keese, M. & H. Schmitz (2014). Broke, Ill, and Obese: Is There an Effect of Household Debt on Health? *Review of Income and Wealth, 60*(3), 525-541.

Kennickell, A. & A. Lusardi (2004). Disentangling the Importance of the Precautionary Saving Motive. *Working Paper 10888, Cambridge MA, National Bureau of Economic Research.*

Kings Fund (2014). *The UK Private Health Market*. The UK Private Health Market. Commission on the Future of Health and Social Care in England.

Kraus, V.B., Huebner, J.L., Fink, C., King, J.B., Brown, S., Vail, T.P. & F. Guilak (2002). Urea as a Passive Transport Marker for Arthritis Biomarker Studies. *Arthritis and Rheumatism*, *46*(2), 420-427.

Lennartsson, A.K., Theorell, T., Kushnir, M.M., Bergquist, J. & I.H. Jonsdottir (2013). Perceived Stress at Work is Associated with Attenuated DHEA-S Response during Acute Psychosocial Stress. *Psychoneuroendocrinology*, *38*(9), 1650-1657.

Levine, M.E. & E.M. Crimmins (2014). A Comparison of Methods for Assessing Mortality Risk. *American Journal of Human Biology*, *26*(6), 768-776.

Lusardi, A. (1998). On the Importance of the Precautionary Saving Motive. *American Economic Review*, 449-453.

Lyons, T.J. & A. Basu (2012). Biomarkers in Diabetes: Hemoglobin A1c, Vascular and Tissue Markers. *Translational Research: The Journal of Laboratory and Clinical Medicine*, 159(4), 303–312.

Maslow, A.H. (1943). A Theory of Human Motivation. *Psychological Review*, 50(4), 370–396.

Musaad, S. & E.N. Haynes (2007). Biomarkers of Obesity and Subsequent Cardiovascular Events. *Epidemiological Review*, 29, 98-114.

Nelsen, R.B. (2006). *An Introduction to Copulas, 2nd ed.* New York: Springer-Verlag, *2006*.

NICE – National Institute for Health and Social Care (2011). Hypertension in Adults: Diagnosis and Management. www.nice.org.uk/guidance/cg127

Office of National Statistics (ONS) (2016). Quarterly National Accounts: Quarter 1 (Jan to Mar) 2016, Statistical Bulletin.

Olsen, M.K. & J.L. Schafer (2001). A Two-part Random-Effects Model for Semicontinuous Longitudinal Data. *Journal of the American Statistical Association, 96*(454), 730-745.

Ricketts, C.F., Rezek, J.P. & R.C. Campbell (2013). The Influence of Individual Health Outcomes on Individual Savings Behavior. *The Social Science Journal*, *50*(4), 471-481.

Rossi, M. (2009). Examining the Interaction between Saving and Contributions to Personal Pension Plans: Evidence from the BHPS. *Oxford Bulletin of Economics and Statistics, 71*(2), 253-271.

Ryff, C.D., Dienberg Love, G., Urry, H.L., Muller, D., Rosenkranz, M.A., Friedman, E.M., Davidson, R.J. & B. Singer (2006). Psychological Well-being and Ill-being: Do they have Distinct or Mirrored Biological Correlates? *Psychotherapy and Psychosomatics*, *75*(2), 85-95.

Seeman T.E., Singer, B.H., Ryff, C.D., Love, G.D. & L. Levy-Storms (2002). Social Relationships, Gender, and Allostatic Load across Two Age Cohorts. *Psychosocial Medicine*, 64, 395–406.

Seeman, T.E., Singer, B. H., Rowe, J.W., Horwitz, R.I. & B. McEwen (1997). Price of Adaptation - Allostatic Load and its Health Consequences. *Archives of Internal Medicine,* 157, 2259-2268.

Seeman, T.E., Merkin, S.S., Crimmins, E., Koretz, B., Charette, S. & A. Karlamangla (2008). Education, Income and Ethnic Differences in Cumulative Biological Risk profiles in a National Sample of US Adults: NHANES III (1988–1994). *Social Science and Medicine*, *66*(1), 72-87.

Seplaki, C.L., Goldman, N., Glei, D. & M. Weinstein (2005). A Comparative Analysis of Measurement Approaches for Physiological Dysregulation in an Older Population. *Experimental Gerontology* 40, 438–449.

Sesso, H.D., Buring, J.E., Rifai, N., Blake, G.J., Gaziano, J.M. & P.M. Ridker (2003). C-reactive Protein and the Risk of Developing Hypertension. *Journal of the American Medical Association*, 290, 2945–2951.

Starr-McCluer, M. (1996). Health Insurance and Precautionary Savings. *American Economic Review*, 86, 285-295.

Šulcová, J., Hill, M., Hampl, R. & L. Starka (1997). Age and Sex related Differences in Serum Levels of Unconjugated Dehydroepiandrosterone and its Sulphate in Normal Subjects. *Journal of Endocrinology*, *154*(1), 57-62.

Tooze, J.A., Grunwald, G.K. & R.H. Jones, (2002). Analysis of Repeated Measures Data with Clumping at Zero. *Statistical Methods in Medical Research*, *11*(4), 341-355.

Troncoso, R., Ibarra, C., Vicencio, J.M., Jaimovich, E. & S. Lavandero (2014). New Insights into IGF-1 Signaling in the Heart. *Trends in Endocrinology & Metabolism*, *25*(3), 128-137.

van Beek, J.H., de Moor, M.H., Geels, L.M., Sinke, M.R., de Geus, E.J., Lubke, G.H., Kluft, C., Neuteboom, J., Vink, J.M., Willemsen, G. & D.I. Boomsma (2014). The Association of Alcohol Intake with Gamma-glutamyl Transferase (GGT) levels: Evidence for Correlated Genetic Effects. *Drug and Alcohol Dependence*, *134*, 99-105.

World Health Organization (2011). Haemoglobin Concentrations for the Diagnosis of Anaemia and Assessment of Severity. Available at http://www.who.int/vmnis/indicators/haemoglobin.pdf

Yoshida, A. & A. Guariglia (2002). Estimating Saving Functions in the Presence of Excessive-Zeros Problems. *The Econometrics Journal*, *5*(2), 435-456.

Zou, H. & T. Hastie (2005). Regularization and Variable Selection via the Elastic Net. *Journal of the Royal Statistical Society: Series B (Statistical Methodology)*, *67*(2), 301-320.

Appendix: Figures and Tables

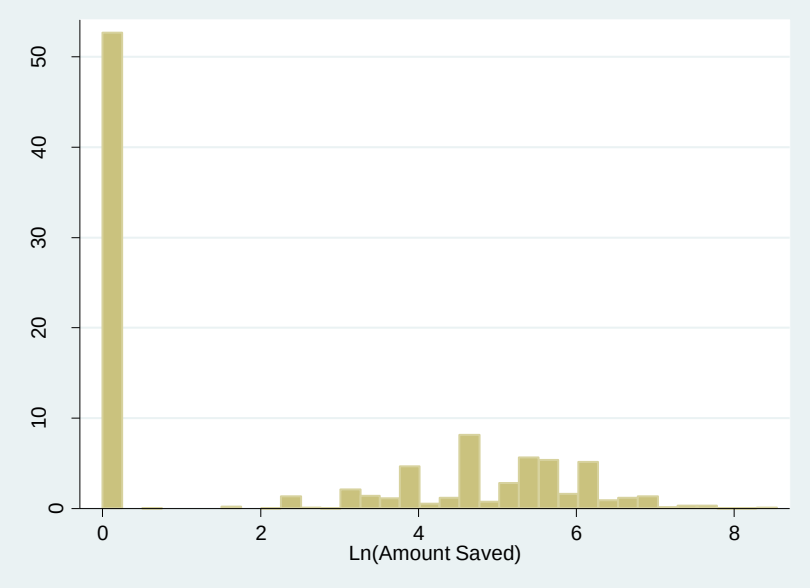

Figure 1: Distribution of the Amount Saved

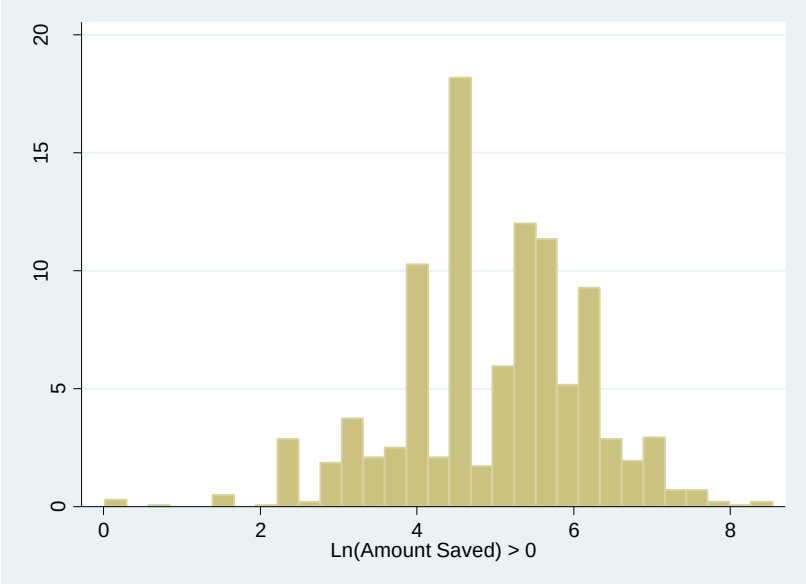

Figure 2: Distribution of the Amount Saved where Monthly Saving is Greater than Zero

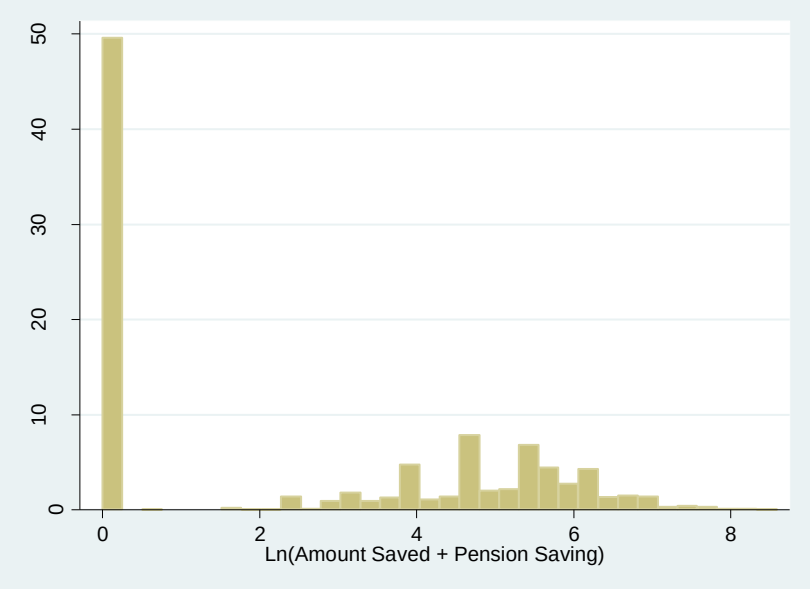

Figure 3: Distribution of the Amount Saved and Pension Contributions

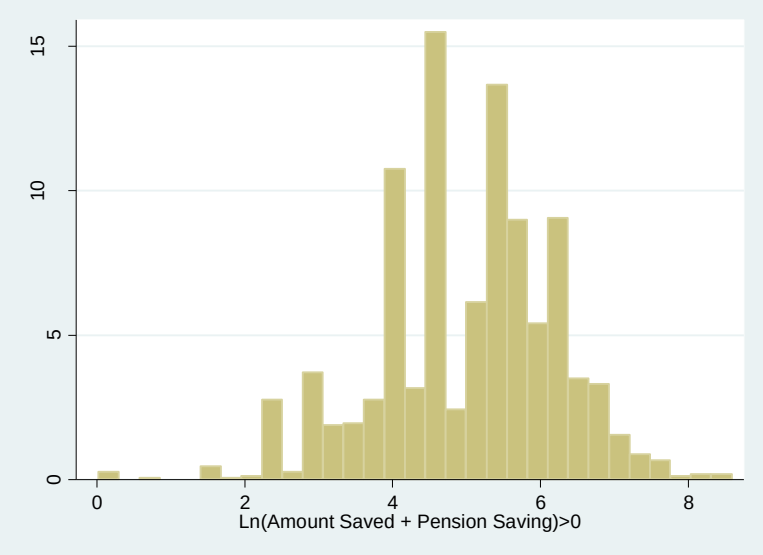

Figure 4: Distribution of the Amount Saved and Pension Contributions where Monthly Saving and Pension Contributions are Greater than Zero

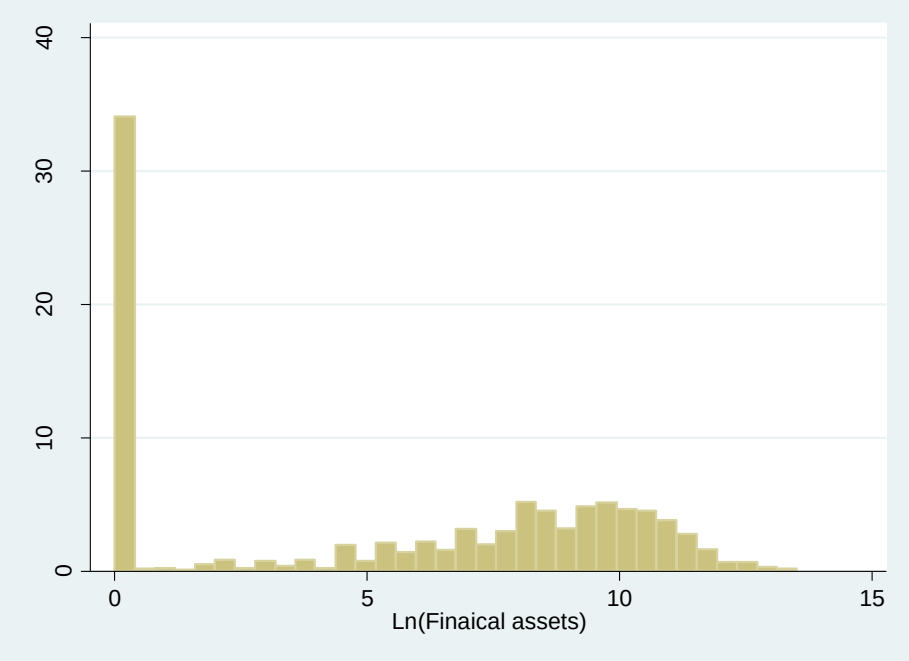

Figure 5: Distribution of the Financial Assets

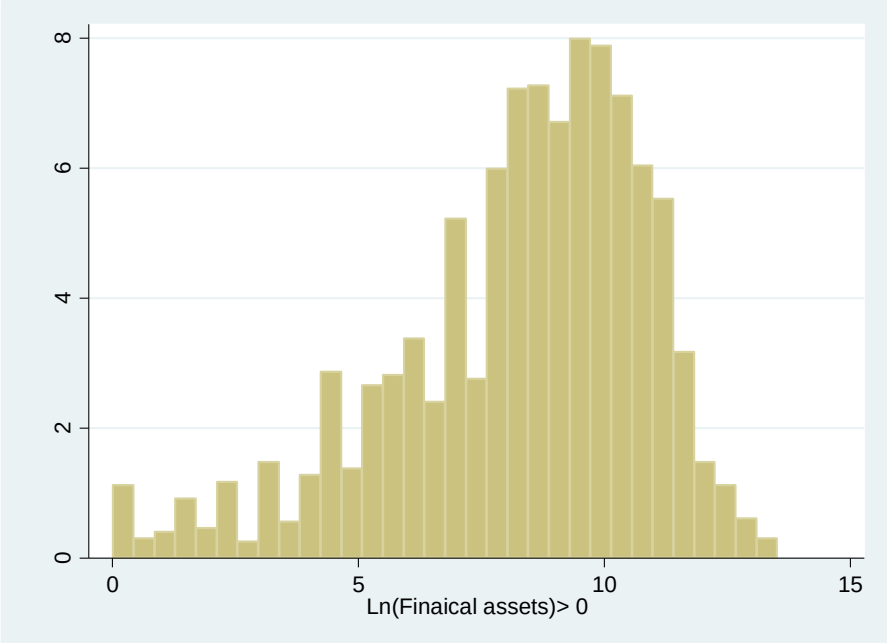

Figure 6: Distribution of the Financial Assets where Financial Assets are Greater than Zero

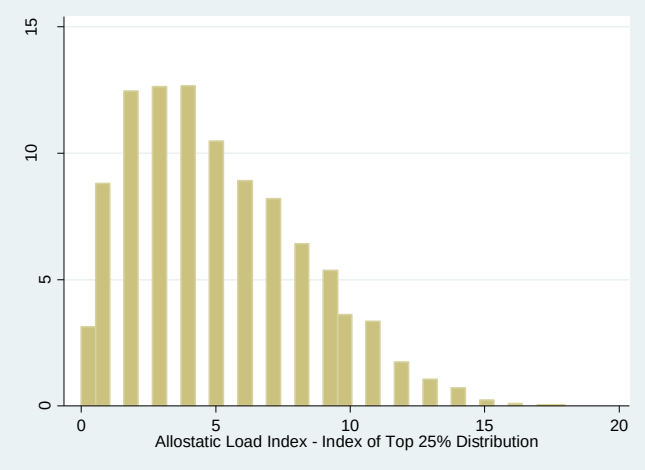

Figure 7: Allostatic Load Index

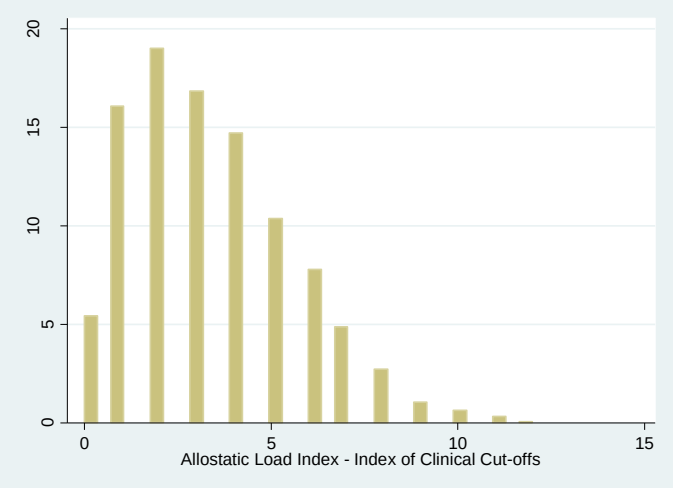

Figure 8: Allostatic Load Index: Clinical Cut-offs

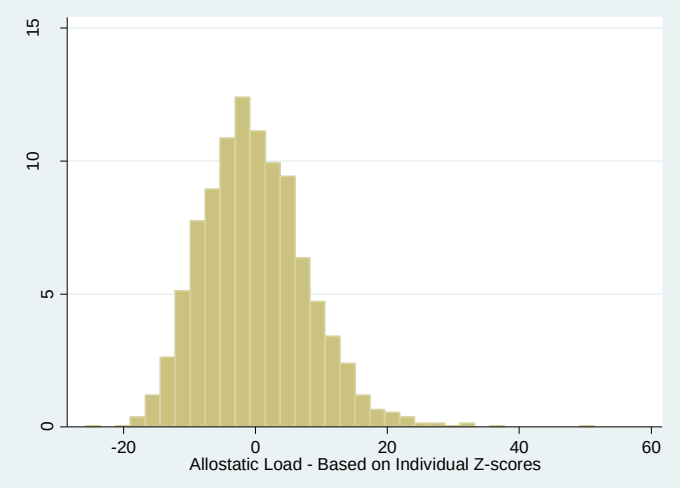

Figure 9: Allostatic Load Index: Z-Scores

Table 1A – Summary Statistics: Dependent Variables

Variable	Mean (Std. Dev.)
Save (Binary)	0.475 (0.500)
Ln(Amount Saved) (All Individuals)	2.371 (2.625)
Ln(Amount Saved) (Individuals who save)	4.990 (1.198)
Save and/or Contribute to Pension (Binary)	0.505 (0.500)
Ln(Amount Saved plus Pension Contribution) (All Individuals)	2.528 (2.646)
Ln(Amount Saved plus Pension Contribution) (All Individuals who save/make contributions)	5.004 (1.210)
Financial Investments (Binary)	0.667 (0.471)
Ln(Financial Investments) (All individuals)	5.522 (4.450)
Ln(Financial Investments) (Individuals who hold financial investments)	8.283 (2.614)
Unsecured Debt (Binary)	0.341 (0.474)
Ln(Unsecured Debt) (All individuals)	2.694 (3.861)
Ln(Unsecured Debt) (Individuals who hold unsecured debt)	7.897 (1.611)
N	**2,928**

Table 1B – Summary Statistics: Health Measures

Health Measure	Mean (Standard Deviation)
Composite Biomarkers	
Allostatic Load - Index	5.094 (3.239)
Allostatic Load – Clinical Cut	3.388 (2.208)
Allostatic Load - Z-Score	-0.088 (7.834)
Individual Biomarkers	
General Health	
Ln(CHL)	1.680 (0.208)
Ln(HDL)	0.397 (0.303)
Ln(TRI)	0.411 (0.557)
Ln(HbA1c)	3.569 (0.154)
Ln(DBP)	4.299 (0.144)
Ln(SBP)	4.812 (0.122)
Ln(BMI)	3.317 (0.184)
Ln(WST)	4.525 (0.150)
Inflammatory markers	
Ln(CRP)	0.252 (0.940)
Ln(FIB)	0.973 (0.189)
Markers of Anaemia	
Ln(HGB)	4.915 (0.104)
Ln(FER)	4.473 (0.927)
Liver and Kidney Function Tests	
Ln(ALB)	3.855 (0.059)
Ln(ALKP)	4.196 (0.281)
Ln(ALT)	3.247 (0.467)
Ln(AST)	3.379 (0.263)
Ln(GGT)	3.209 (0.676)
Ln(CRE)	4.285 (0.203)
Ln(URE)	1.752 (0.238)
Hormones	
Ln(DHE)	1.402 (0.685)
Ln(IGF)	2.897 (0.341)
Self-Assessed Health Status	
SF-12ind	0.788 (0.134)
SAH	2.564 (1.062)
N	**2,928**

Table 1C - Summary Statistics: Independent Variables

Variables	Mean	Standard Deviation
Age	47.874	(11.826)
Age Squared	2431.764	(1077.134)
Female	57.3	
Education (Omitted Category: Below GCSE)		
Degree	29.2	
Other Higher Qualification	15.6	
A-Level	20.8	
GCSE	19.4	
Other Qualification	8.50	
Relationship Status (Omitted Category: Single/never married)		
Married	69.5	
Divorced	12.2	
Widow	2.10	
Number of Children	0.591	(0.940)
Employment status (Omitted Category: Unemployed/Not in the labour force)		
Employed	62.3	
Self-Employed	9.90	
Retired	12.2	
Ln(Household Monthly Income)	8.145	(0.694)
Region (Omitted Category: Residing in London)		
North East	6.8	
North West	11.6	
Yorkshire	7.3	
East Midlands	9.5	
West Midlands	9.26	
East	11.0	
South East	17.3	
South West	12.0	
Wales	2.6	
Scotland	5.0	
Arrears	11.9	
Material Deprivation	1.096	(1.865)
N	**2,928**	

Note: Mean and standard deviation are reported for continuous variables and proportions are reported for binary independent variables.

Table 2A – A Semi-Continuous Model of Monthly Saving

	Binary Part			Continuous Part			Marginal Effects†
Independent Variables	Mean	2.50%	97.50%	Mean	2.50%	97.50%	
Age	-0.796	-1.502	-0.085	-0.103	-0.590	0.380	-0.770
Age Squared	0.647*	-0.075	1.378	0.264	-0.233	0.769	0.806
Female	-0.034	-0.196	0.133	-0.229*	-0.346	-0.114	-0.258
Degree	0.209	-0.185	0.591	0.703*	0.396	1.000	0.864
Other Higher Qualification	0.179	-0.228	0.581	0.592*	0.269	0.899	0.732
A-Level	0.035	-0.370	0.428	0.465*	0.148	0.771	0.494
GCSE	-0.048	-0.443	0.347	0.245	-0.066	0.552	0.204
Other Qualification	0.017	-0.437	0.465	0.153	-0.198	0.493	0.167
Married	-0.361*	-0.655	-0.057	-0.017	-0.222	0.190	-0.363
Divorced	-0.038	-0.401	0.327	-0.022	-0.280	0.240	-0.054
Widow	-0.001	-0.620	0.622	0.049	-0.395	0.498	0.048
Number of Children	-0.034	-0.143	0.077	-0.129*	-0.205	-0.051	-0.158
Employed	0.909*	0.634	1.189	0.435*	0.204	0.665	0.958
Self-Employed	0.529*	0.171	0.891	0.454*	0.158	0.743	0.812
Retired	0.472*	0.096	0.846	0.217	-0.094	0.526	0.544
Ln(Household Monthly Income)	0.301*	0.195	0.409	0.383*	0.305	0.459	0.635
North East	0.329	-0.105	0.762	-0.092	-0.411	0.227	0.150
North West	0.333	-0.043	0.714	-0.197	-0.477	0.076	0.048
Yorkshire	0.523*	0.105	0.946	-0.018	-0.319	0.286	0.337
East Midlands	0.501*	0.104	0.898	0.120	-0.168	0.410	0.463
West Midlands	0.314	-0.083	0.709	0.074	-0.214	0.360	0.306
East	0.374	-0.006	0.755	-0.108	-0.386	0.175	0.162
South East	0.310	-0.041	0.664	0.064	-0.197	0.320	0.294
South West	0.360	-0.027	0.741	-0.004	-0.283	0.270	0.258
Wales	-0.078	-0.681	0.52	-0.042	-0.503	0.421	-0.109
Scotland	0.529*	0.050	1.013	-0.210	-0.547	0.126	0.148
Arrear	-0.228	-0.517	0.065				-0.208
Material Deprivation	-0.446*	-0.513	-0.379				-0.446

* - denotes statistical significant at the 5% level. Mean indicates the estimated coefficient whilst, 2.50% and 97.50% denotes 95% credible interval.

† - Marginal effects are calculated using the following way:

Continuous independent variables $\frac{\Delta E(y|x)}{E(y|x)} = \beta.\frac{\varphi(\Phi^{-1}(\pi))}{\pi} + \gamma$

Binary independent variables $\frac{\Delta E(y|x)}{E(y|x)} = \ln\left(\frac{\Phi\left(\beta+\Phi^{-1}(\pi)\right)}{\pi}\right) + \gamma$

Where φ and $\Phi$are the p.d.f and the c.d.f of the standard normal distribution, β is the coefficient in the binary part of the model, γ is the coefficient in the continuous variable, whilst π is the probability of that the saving is positive. In this case we take π to equal the sample proportion, that is, 0.4751.

Table 2B – Summary of Health Measures in the Semi-Continuous Model of Monthly Saving[16]

	Binary Part			Continuous Part			Marginal
Health Measures	Mean	2.50%	97.50%	Mean	2.50%	97.50%	Effects†
Composite Biomarkers							
Allostatic Load - Index	-0.020	-0.053	0.005	-0.026*	-0.047	-0.006	-0.046
Allostatic Load – Clinical Cut	-0.050*	-0.089	-0.012	-0.04*	-0.068	-0.012	-0.082
Allostatic Load - Z-Score	-0.020*	-0.033	-0.007	-0.011*	-0.020	-0.002	-0.028
Individual Biomarkers							
General Health							
Ln(CHL)	-0.036	-0.122	0.049	0.053	-0.008	0.116	0.023
Ln(HDL)	0.084	-0.004	0.174	0.052	-0.012	0.117	0.122
Ln(TRI)	-0.124	-0.211	-0.037	-0.070*	-0.134	-0.006	-0.174
Ln(HbA1c)	-0.058	-0.15	0.032	-0.090*	-0.160	-0.019	-0.139
Ln(DBP)	0.007	-0.078	0.091	0.000	-0.060	0.060	0.006
Ln(SBP)	0.025	-0.067	0.119	0.034	-0.032	0.101	0.055
Ln(BMI)	-0.117	-0.205	-0.031	-0.101*	-0.166	-0.035	-0.199
Ln(WST)	-0.150	-0.246	-0.055	-0.121*	-0.191	-0.050	-0.247
Inflammatory markers							
Ln(CRP)	-0.045	-0.127	0.039	-0.07*	-0.129	-0.009	-0.108
Ln(FIB)	0.031	-0.054	0.115	-0.035	-0.096	0.027	-0.009
Markers of Anaemia							
Ln(HGB)	0.027	-0.080	0.136	0.033	-0.045	0.113	0.056
Ln(FER)	-0.017	-0.113	0.078	0.064	-0.005	0.134	0.050
Liver and Kidney Function							
Ln(ALB)	-0.038	-0.124	0.050	0.032	-0.028	0.092	0.000
Ln(ALKP)	-0.090	-0.174	-0.005	-0.037	-0.095	0.023	-0.112
Ln(ALT)	-0.067	-0.156	0.024	-0.028	-0.094	0.039	-0.084
Ln(AST)	-0.055	-0.140	0.030	-0.021	-0.086	0.044	-0.067
Ln(GGT)	-0.091	-0.181	0.000	-0.068*	-0.135	-0.001	-0.144
Ln(CRE)	-0.041	-0.140	0.060	-0.005*	-0.080	0.071	-0.039
Ln(URE)	0.017	-0.070	0.105	0.019	-0.045	0.084	0.033
Hormones							
Ln(DHE)	-0.032	-0.136	0.070	0.036	-0.035	0.107	0.009
Ln(IGF)	0.037	-0.059	0.137	0.029	-0.041	0.100	0.060
Self-Assessed Health							
SAH¶	0.140*	0.055	0.224	0.152*	0.089	0.214	0.151
SF-12ind	1.106*	0.449	1.769	0.619*	0.132	1.094	1.546

* - denotes statistical significant at the 5% level. Mean indicates the estimated coefficient whilst, 2.50% and 97.50% denotes 95% credible interval.

† - Marginal effects are calculated using the following way:

Continuous independent variables $\frac{\Delta E(y|x)}{E(y|x)} = \beta.\frac{\varphi(\Phi^{-1}(\pi))}{\pi} + \gamma$

Binary independent variables $\frac{\Delta E(y|x)}{E(y|x)} = \ln\left(\frac{\Phi\left(\beta+\Phi^{-1}(\pi)\right)}{\pi}\right) + \gamma$

Where φ and $\Phi$are the p.d.f and the c.d.f of the standard normal distribution, β is the coefficient in the binary part of the model, γ is the coefficient in the continuous variable, whilst π is the probability of that the saving is positive. In this case we take π to equal the sample proportion, that is, 0.4751.

¶ - Marginal effect presented is for the change from 0-1, corresponding to a change from poor to fair health. The marginal effect is calculated in the following way:

$$\frac{\Delta E(y|x)}{E(y|x)} = \frac{1}{(b-a)}\ln\left(\frac{\Phi\left(\beta b+\Phi^{-1}(\pi)\right)}{\Phi\left(\beta a+\Phi^{-1}(\pi)\right)}\right) + \gamma.$$

The marginal effects relating to changes from *fair* to *good* health, *good* to *very good* health, and *very good* to *excellent* health are 0.131, 0.112 and 0.092, respectively.

[16] All health measures are included independently along with the independent variables included in Table 2A and outlined in Section 4.1.

Table 3 – Summary of Health Measures in aSemi-Continuous Model of Monthly Saving and Pension Contributions[17]

	Binary Part			Continuous Part			Marginal
Health Measures	Mean	2.50%	97.50%	Mean	2.50%	97.50%	Effects†
Composite Biomarkers							
Allostatic Load - Index	-0.029*	-0.058	0.000	-0.026*	-0.047	-0.006	-0.049
Allostatic Load – Clinical Cut	-0.06*	-0.099	-0.022	-0.038*	-0.066	-0.011	-0.085
Allostatic Load - Z-Score	-0.021*	-0.034	-0.009	-0.011*	-0.02	-0.002	-0.028
Individual Biomarkers							
General Health							
Ln(CHL)	-0.041	-0.126	0.047	0.048	-0.013	0.109	0.016
Ln(HDL)	0.089	-0.001	0.180	0.048	-0.015	0.111	0.118
Ln(TRI)	-0.125*	-0.214	-0.037	-0.070*	-0.132	-0.007	-0.169
Ln(HbA1c)	-0.063	-0.155	0.027	-0.073*	-0.142	-0.003	-0.123
Ln(DBP)	0.018	-0.068	0.102	-0.008	-0.067	0.052	0.006
Ln(SBP)	0.020	-0.074	0.112	0.035	-0.031	0.101	0.051
Ln(BMI)	-0.119*	-0.206	-0.034	-0.110*	-0.174	-0.045	-0.204
Ln(WST)	-0.150*	-0.245	-0.055	-0.126*	-0.195	-0.056	-0.244
Inflammatory markers							
Ln(CRP)	-0.047	-0.131	0.036	-0.061*	-0.119	0.000	-0.098
Ln(FIB)	0.008	-0.078	0.092	-0.021	-0.080	0.039	-0.015
Markers of Anaemia							
Ln(HGB)	0.037	-0.069	0.142	0.045	-0.032	0.124	0.074
Ln(FER)	-0.046	-0.146	0.049	0.068	0.000	0.137	0.032
Liver and Kidney Function							
Ln(ALB)	-0.020	-0.108	0.065	0.033	-0.026	0.092	0.017
Ln(ALKP)	-0.107*	-0.194	-0.021	-0.042	-0.099	0.018	-0.127
Ln(ALT)	-0.081	-0.172	0.013	-0.018	-0.083	0.048	-0.082
Ln(AST)	-0.059	-0.143	0.027	-0.016	-0.079	0.048	-0.063
Ln(GGT)	-0.109*	-0.200	-0.016	-0.048	-0.113	0.018	-0.134
Ln(CRE)	-0.011	-0.113	0.090	-0.004	-0.078	0.07	-0.013
Ln(URE)	0.020	-0.067	0.107	0.013	-0.050	0.076	0.029
Hormones							
Ln(DHE)	-0.014	-0.115	0.087	0.029	-0.040	0.100	0.018
Ln(IGF)	0.015	-0.085	0.114	0.054	-0.015	0.124	0.066
Self-Assessed Health							
SAH¶	0.124*	0.039	0.207	0.136*	0.074	0.196	0.127
SF-12ind	0.908*	0.249	1.570	0.638*	0.163	1.100	1.355

* - denotes statistical significant at the 5% level. Mean indicates the estimated coefficient whilst, 2.50% and 97.50% denotes 95% credible interval.

† - Marginal effects are calculated using the following way:

Continuous independent variables $\frac{\Delta E(y|x)}{E(y|x)} = \beta.\frac{\varphi(\Phi^{-1}(\pi))}{\pi} + \gamma$

Binary independent variables $\frac{\Delta E(y|x)}{E(y|x)} = \ln\left(\frac{\Phi\left(\beta+\Phi^{-1}(\pi)\right)}{\pi}\right) + \gamma$

Where φ and $\Phi$are the p.d.f and the c.d.f of the standard normal distribution, β is the coefficient in the binary part of the model, γ is the coefficient in the continuous variable, whilst π is the probability of that the saving is positive. In this case we take π to equal the sample proportion, that is, 0.505.

¶ - Marginal effect presented is for the change from 0-1, corresponding to a change from poor to fair health. The marginal effect is calculated in the following way:

$$\frac{\Delta E(y|x)}{E(y|x)} = \frac{1}{(b-a)}\ln\left(\frac{\Phi\left(\beta b+\Phi^{-1}(\pi)\right)}{\Phi\left(\beta a+\Phi^{-1}(\pi)\right)}\right) + \gamma.$$

The marginal effects relating to changes from *fair* to *good* health, *good* to *very good* health, and *very good* to *excellent* health are 0.111, 0.096 and 0.081, respectively.

[17] All health measures are included independently along with the independent variables included in Table 2A and outlined in Section 4.1.

Table 4 – Summary of Health Measures in Semi-Continuous Model of Financial Assets Holding[18]

	Binary Part			Continuous Part			Marginal Effects†
Health Measures	Mean	2.50%	97.50%	Mean	2.50%	97.50%	
Composite Biomarkers							
Allostatic Load - Index	-0.032*	-0.062	-0.001	-0.128*	-0.164	-0.091	-0.145
Allostatic Load – Clinical Cut	-0.016	-0.057	0.025	-0.134*	-0.184	-0.085	-0.143
Allostatic Load - Z-Score	-0.013	-0.026	0.001	-0.064*	-0.080	-0.048	-0.071
Individual Biomarkers							
General Health							
Ln(CHL)	0.024	-0.070	0.120	0.045	-0.071	0.159	0.058
Ln(HDL)	0.114*	0.023	0.208	0.337*	0.223	0.451	0.399
Ln(TRI)	-0.096*	-0.191	-0.002	-0.262*	-0.379	-0.144	-0.314
Ln(HbA1c)	-0.048	-0.140	0.046	-0.298*	-0.422	-0.174	-0.324
Ln(DBP)	-0.003	-0.093	0.088	-0.056	-0.167	0.056	-0.058
Ln(SBP)	0.005	-0.095	0.104	-0.063	-0.184	0.058	-0.060
Ln(BMI)	-0.042	-0.131	0.048	-0.496*	-0.607	-0.383	-0.519
Ln(WST)	-0.033	-0.132	0.063	-0.538*	-0.661	-0.415	-0.556
Inflammatory markers							
Ln(CRP)	-0.068	-0.159	0.021	-0.256*	-0.364	-0.146	-0.293
Ln(FIB)	-0.062	-0.152	0.028	-0.216*	-0.326	-0.105	-0.250
Markers of Anaemia							
Ln(HGB)	-0.019	-0.129	0.089	-0.054	-0.193	0.086	-0.064
Ln(FER)	-0.124*	-0.226	-0.016	-0.038	-0.163	0.089	-0.106
Liver and Kidney Function							
Ln(ALB)	0.072	-0.020	0.163	0.126	0.014	0.238	0.165
Ln(ALKP)	-0.097*	-0.189	-0.008	-0.247*	-0.354	-0.138	-0.300
Ln(ALT)	0.000	-0.097	0.094	-0.146*	-0.269	-0.022	-0.146
Ln(AST)	-0.022	-0.112	0.07	-0.067	-0.182	0.050	-0.079
Ln(GGT)	-0.063	-0.159	0.035	-0.185*	-0.305	-0.064	-0.219
Ln(CRE)	-0.087	-0.195	0.018	0.003	-0.130	0.137	-0.044
Ln(URE)	0.017	-0.076	0.110	-0.033	-0.148	0.082	-0.024
Hormones							
Ln(DHE)	0.001	-0.108	0.110	0.160*	0.031	0.291	0.161
Ln(IGF)	0.031	-0.073	0.135	0.211*	0.084	0.340	0.228
Self-Assessed Health							
SAH¶	0.108*	0.020	0.196	0.373*	0.262	0.481	0.321
SF-12ind	-0.267	-0.967	0.421	1.813*	0.951	2.659	1.668

* - denotes statistical significant at the 5% level. Mean indicates the estimated coefficient whilst, 2.50% and 97.50% denotes 95% credible interval.

† - Marginal effects are calculated using the following way:

Continuous independent variables $\frac{\Delta E(y|x)}{E(y|x)} = \beta.\frac{\varphi(\Phi^{-1}(\pi))}{\pi} + \gamma$

Binary independent variables $\frac{\Delta E(y|x)}{E(y|x)} = \ln\left(\frac{\Phi\left(\beta+\Phi^{-1}(\pi)\right)}{\pi}\right) + \gamma$

Where φ and $\Phi$are the p.d.f and the c.d.f of the standard normal distribution, β is the coefficient in the binary part of the model, γ is the coefficient in the continuous variable, whilst π is the probability of that the saving is positive. In this case we take π to equal the sample proportion, that is, 0.667.

¶ - Marginal effect presented is for the change from 0-1, corresponding to a change from poor to fair health. The marginal effect is calculated in the following way:

$$\frac{\Delta E(y|x)}{E(y|x)} = \frac{1}{(b-a)}\ln\left(\frac{\Phi\left(\beta b+\Phi^{-1}(\pi)\right)}{\Phi\left(\beta a+\Phi^{-1}(\pi)\right)}\right) + \gamma.$$

The marginal effects relating to changes from *fair* to *good* health, *good* to *very good* health, and *very good* to *excellent* health are 0.309, 0.297 and 0.286, respectively.

[18] All health measures are included independently along with the independent variables included in Table 2A and outlined in Section 4.1.

Table 5 - Joint Modelling Semi-Continuous Model of Financial Assets and Unsecured Debt Holding[19]

	Financial Assets						Total Unsecured Debt					
	Binary Part			Continuous Part			Binary Part			Continuous Part		
Health Measures	Mean	2.5%	97.5%	Mean	2.5%	97.5%	Mean	2.5%	97.5%	Mean	2.5%	97.5%
Composite Biomarkers												
Allostatic Load - Index	-0.018	-0.036	0.000	-0.127*	-0.164	-0.088	0.028*	0.009	0.047	0.012	-0.023	0.047
Allostatic Load – Clinical Cut	-0.009	-0.032	0.016	-0.134*	-0.183	-0.083	0.036*	0.012	0.059	0.006	-0.042	0.053
Allostatic Load - Z-Score	-0.007	-0.016	0.001	-0.064*	-0.080	-0.048	0.013*	0.005	0.020	0.002	-0.012	0.015
Individual Biomarkers												
General Health												
Ln(CHL)	0.070	-0.183	0.335	0.232	-0.293	0.765	0.040	-0.221	0.283	-0.010	-0.516	0.494
Ln(HDL)	0.215*	0.029	0.402	1.101*	0.729	1.462	-0.095	-0.270	0.088	0.152	-0.215	0.510
Ln(TRI)	-0.099*	-0.191	-0.008	-0.468*	-0.679	-0.263	0.079	-0.017	0.176	-0.034	-0.211	0.143
Ln(HbA1c)	-0.170	-0.518	0.184	-1.635*	-2.407	-0.88	0.331	-0.013	0.670	0.016	-0.626	0.636
Ln(DBP)	-0.015	-0.378	0.340	-0.195	-0.921	0.594	0.071	-0.283	0.432	0.858*	0.190	1.560
Ln(SBP)	0.021	-0.435	0.467	-0.144	-1.017	0.723	-0.010	-0.466	0.466	0.946*	0.166	1.789
Ln(BMI)	-0.120	-0.397	0.153	-2.497*	-3.074	-1.893	0.588*	0.315	0.855	0.110	-0.413	0.610
Ln(WST)	-0.106	-0.486	0.277	-2.938*	-3.679	-2.182	0.869*	0.497	1.240	0.162	-0.498	0.814
Inflammatory markers												
Ln(CRP)	-0.041	-0.096	0.014	-0.275*	-0.384	-0.161	0.054	-0.001	0.108	0.044	-0.060	0.152
Ln(FIB)	-0.187	-0.467	0.113	-1.103*	-1.662	-0.564	0.302*	0.012	0.571	0.204	-0.321	0.741
Markers of Anaemia												
Ln(HGB)	-0.081	-0.669	0.541	0.449	-0.133	1.020	0.034	-1.051	1.094	-0.217	-1.21	0.765
Ln(FER)	-0.078*	-0.145	-0.012	-0.036	-0.173	0.100	0.054	-0.007	0.115	-0.049	-0.178	0.070
Liver and Kidney Function Tests												
Ln(ALB)	0.665	-0.216	1.486	1.947*	0.492	3.459	-0.182	-1.043	0.685	0.287	-1.101	1.729
Ln(ALKP)	-0.205*	-0.389	-0.010	-0.823*	-1.210	-0.439	-0.023	-0.204	0.158	-0.087	-0.435	0.269
Ln(ALT)	-0.001	-0.123	0.118	-0.295*	-0.546	-0.035	0.113	-0.007	0.233	-0.028	-0.252	0.211
Ln(AST)	-0.041	-0.24	0.165	-0.205	-0.600	0.230	0.145	-0.056	0.331	-0.199	-0.567	0.175
Ln(GGT)	-0.048	-0.137	0.033	-0.266*	-0.435	-0.090	0.107*	0.022	0.187	0.144	-0.009	0.302
Ln(CRE)	-0.255	-0.553	0.054	0.141	-0.465	0.756	0.329*	0.047	0.625	-0.265	-0.824	0.310
Ln(URE)	0.046	-0.182	0.251	-0.109	-0.601	0.359	0.145	-0.095	0.367	-0.107	-0.567	0.316
Hormones												
Ln(DHE)	-0.001	-0.092	0.093	0.238*	0.049	0.439	0.016	-0.076	0.110	0.162	-0.028	0.339
Ln(IGF)	0.051	-0.124	0.233	0.639*	0.282	1.005	-0.115	-0.288	0.064	0.161	-0.164	0.481
Self-Assessed Health												
SAH	0.061*	0.01	0.114	0.374*	0.269	0.477	-0.062*	-0.113	-0.010	0.038	-0.055	0.131
SF-12ind	-0.165	-0.549	0.231	1.841*	0.994	2.629	-0.349	-0.740	0.040	0.280	-0.481	1.027

* - denotes statistical significant at the 5% level. Mean indicates the estimated coefficient whilst, 2.50% and 97.50% denotes 95% credible interval.

[19] All health measures are included independently along with the independent variables included in Table 2A and outlined in Section 4.1.

Online Appendix - Table A1: Details of Biomarkers

Biomarker	Description	Clinical Cut-off	Log Transform (Example Recent References)
General Health			
Total Cholesterol *(CHL)*	Total cholesterol is a risk factor associated CVD. Cholesterol is a steroid which is insoluble in blood and is transported around the body in lipoprotein particles. Apolipoprotein A is important for the delivery of cholesterol to the liver; Apolipoprotein B carries low density lipoproteins which cause narrowing of arteries.	Normal should be 5mmol/L	Restrepo and Rieger (2016), Koda *et al.* (2016),
High Density Lipoprotein *(HDL)*	Apolipoprotein A contains HDL cholesterol and is important for the delivery of cholesterol to the liver for break down. HDL helps remove cholesterol from arteries.	HDL-cholesterol should be > 1mmol/L	Koda *et al.*(2016), Glei *et al.*(2014)
Triglycerides *(TRI)*	Triglycerides are predictive of CVD and high levels are associated with low HDL cholesterol. Triglycerides are the most common types of fats in the body. They are contained in the blood and can be used as energy in cells or can be stored as fat.	Desirable non-fasting triglyceride level is <2mmol/l	Restrepo and Rieger (2016), Koda *et al.* (2016), Bockerman *et al.* (2014), Glei *et al.* (2014)
Glycated Haemoglobin *(HbA1c)*	HbA1c is an indicator of diabetes risk and is a measurement of the level of sugar in the blood over the previous 8-12 weeks prior to measurement.	> 48 mmol/mol indicates diagnosis of diabetes	Koda *et al.*(2016)
Diastolic Blood Pressure *(DBP)*	Diastolic blood pressure being the lowest pressure. High blood pressure is associated with increased CVD and all-cause mortality.	>90 mmHg	Robbins *et al.* (2003), Glei *et al.*(2014)
Systolic Blood Pressure *(SBP)*	Systolic blood pressure is the peak pressure. High blood pressure is associated with increased CVD and all-cause mortality.	>140mmHg	Restrepo and Rieger (2016) Glei *et al.* (2014)
Body Mass Index *(BMI)*	BMI is the weight in KG divided by height in metres squared. BMI is a common measure of body fat and is a measure of health status and is indicative of a range of health outcomes, including CVD and all-cause mortality.	Underweight (<18.5), Normal (18.5 – 24.9), overweight (25 – 29.9) and obese (30+)	Kahn and Cheng (2008), Glei *et al.* (2014)
Waist Measurement *(WST)*	WST is a marker of abdominal fat. Abdominal fat is a predictor of heart disease, type 2 diabetes, insulin resistance and cancers.	>102cm for males and >88cm for females	Kahn and Cheng (2008), Connelly *et al.* (2003)
Inflammatory markers			
C-Reactive Protein *(CRP)*	CRP is a marker of inflammatory load; high values are associated with increased risk of CVD and mortality. CRP is produced in the liver which increases in response to acute inflammation. CRP forms part of the body's defence mechanism against harmful stimulus.	>3 mg/L considered a risk factor for CVD, >10mg/L reflective of recent infection and these data are removed prior to analyses	Jürges *et al.* (2013), Glei *et al.* (2014), Blanchflower *et al.* (2011)
Fibrinogen *(FIB)*	Fibrinogen is a marker of inflammation and it helps the body to stop bleeding by helping blood clots to form. Higher levels of fibrinogen are associated with the development of CVD. Fibrinogen levels reflect inflammatory processes.	Data are continuous and there are no established clinical cut-points	Su *et al.* (2008), Gravholt *et al.* (2012)
Markers of Anaemia			
Haemoglobin *(HGB)*	Low levels of HGB is suggestive of anaemia, a lack of iron in the blood. It is associated with longer hospitalization and greater risk of mortality and CVD. HGB is the iron-containing molecule responsible for carrying oxygen around the body.	Anaemia defined (WHO guidelines) as Hb levels <13 g/dL for men and <12 g/dL for women	Rasmussen *et al.* (2005), Kelly *et al.*(1993)

Ferritin *(FER).*	Levels of ferritin reflect the body's iron stores and it is symptomatic of anaemia. Low ferritin level is predictive of uncomplicated iron deficiency anaemia. However, high ferritin levels suggest excess body iron, which is also indicative of poor health.	≤ 20 ug/L indicate depletion of iron and Ferritin levels >300 (>200) ug/L may indicate iron overload in men and post- (pre-) menopausal women	Rasmussen *et al.* (2005), Mei *et al.* (2014)
Liver and Kidney Function Tests			
Albumin *(ALB)*	Measures the main protein made by the liver and low levels may be indicative of a loss of liver function. ALB helps maintain the osmotic pressure of the blood. Low levels may be a sign of liver or kidney disease or reflect poor nutrition.	<35 U/L, >50 U/L	Roy *et al.* (2006), Robbins *et al.* (2006)
Alkaline Phosphatase *(ALK)*	An enzyme related to the bile ducts which often increased when bile ducts are blocked, either inside or outside the liver.	Aged 20-70: <30 U/L, >130 U/L Aged >70: <30 U/L, >150 U/L	Kelly *et al.*(1993), Carbone *et al.* (2013)
Alanine Transaminase *(ALT)*	An enzyme mainly found in the liver and is used for detecting hepatitis. In addition, raised levels indicate liver damage.	>40 U/L	Schwertner *et al.* (1994), Carbone *et al.* (2013)
Aspartate Transaminase *(AST)*	An enzyme found in the liver, the heart and other muscles. Increased levels of AST indicate potential liver damage.	>40 U/L	Schwertner *et al.* (1994), Carbone *et al.* (2013)
Gamma Glutamyl Transferase *(GGT)*	An enzyme which is involved in the transfer of amino acids around the blood. Raised levels of GGT are associated with liver disease.	Males >70 g/L, Females>45 g/L	Gravholt *et al.* (2012), van Beek *et al.* (2014)
Creatinine *(CRE)*	Creatinine is a chemical waste product of muscle function, which is passed through the kidneys and excreted in urine. CRE levels indicate how effectively the kidneys are 'cleaning' the blood.	Normal: men 60 to 110 micromol/L (0.7 to 1.2 mg/dL), women 45 to 90 micromol/L (0.5 to 1.0 mg/dL)	Bockerman *et al.* (2014), Robbins *et al.* (2003)
Urea *(URE)*	Urea is a waste product of the breakdown of proteins. High levels indicate that the kidneys are not functioning effectively.	Normal 2.5-7.8 mmol/L	Shiju *et al.* (2015)
Hormones			
Dihydroepiandrosterone Sulphate *(DHE)*	DHE has been associated with CVD. Low levels are associated with CVD and all-cause mortality whilst higher levels are related to better health outcomes. DHE levels decline with age.	Age and Gender Specific cut points	Coutinho *et al.* (2007), Fischer *et al.* (2004)
Insulin-like Growth Factor 1 *(IGF)*	Low IGF-1 levels have been shown to be associated with heart disease and high levels have been shown to be predictive of some cancers. IGF-1 is a hormone, which influences growth and development in childhood and continues to influence anabolic processes in adults.	Age and Gender Specific cut-points	Fischer *et al.* (2004), Ye *et al.* (2012)

Table A1 References:

Blanchflower, D. G., Christakis, N. A. & A. J. Oswald (2011). *An Introduction to the Structure of Biomarker Equations*. Mimeo.

Bockerman, P., Bryson, A., Hakulinen, C., Pehkonen, J., Pulkki-Raback, L., Raitakari, O. & J. Viinikainen (2014). *Biomarkers and Long-term Market Outcomes: The Case of Creatine* (No. dp1279). Centre for Economic Performance, LSE.

Carbone, M., Mells, G. F., Pells, G., Dawwas, M. F., Newton, J. L., Heneghan, M. A. & G. J. Alexander (2013). Sex and age are determinants of the clinical phenotype of primary biliary cirrhosis and response to ursodeoxycholic acid. *Gastroenterology*, *144*(3), 560-569.

Connelly, P. W., Hanley, A. J., Harris, S. B., Hegele, R. A. & B. Zinman, (2003). Relation of waist circumference and glycemic status to C-reactive protein in the Sandy Lake Oji-Cree. *International Journal of Obesity*, *27*(3), 347-354.

Coutinho, H. M., Leenstra, T., Acosta, L. P., Olveda, R. M., McGarvey, S. T., Friedman, J. F. & J. D. Kurtis (2007). Higher serum concentrations of DHEAS predict improved nutritional status in helminth-infected children, adolescents, and young adults in Leyte, the Philippines. *The Journal of Nutrition*, *137*(2), 433-439.

Fischer, F., Schulte, H., Mohan, S., Tataru, M. C., Köhler, E., Assmann, G. & A. Von Eckardstein (2004). Associations of insulin-like growth factors, insulin-like growth factor binding proteins and acid-labile subunit with coronary heart disease. *Clinical Endocrinology*, *61*(5), 595-602.

Glei, D.A., Goldman, N., Rodríguez, G. & M. Weinstein (2014). Beyond self-reports: changes in biomarkers as predictors of mortality. *Population and Development Review*, *40*(2), 331-360.

Gravholt, C. H., Mortensen, K. H., Andersen, N. H., Ibsen, L., Ingerslev, J. & B. E. Hjerrild (2012). Coagulation and fibrinolytic disturbances are related to carotid intima thickness and arterial blood pressure in Turner syndrome. *Clinical Endocrinology*, *76*(5), 649-656.

Jürges, H., Kruk, E. & S. Reinhold (2013). The effect of compulsory schooling on health—evidence from biomarkers. *Journal of population economics*, *26*(2), 645-672.

Kahn, H. S. & Y. J. Cheng (2008). Longitudinal changes in BMI and in an index estimating excess lipids among white and black adults in the United States. *International Journal of Obesity*, *32*(1), 136-143.

Kelly, W. K., Scher, H. I., Mazumdar, M., Vlamis, V., Schwartz, M. & S. D. Fossa (1993). Prostate-specific antigen as a measure of disease outcome in metastatic hormone-refractory prostate cancer. *Journal of Clinical Oncology*, *11*(4), 607-615.

Koda, M., Kitamura, I., Okura, T., Otsuka, R., Ando, F. & H. Shimokata (2016). The associations between smoking habits and serum triglyceride or hemoglobin A1c levels differ according to visceral fat accumulation. *Journal of Epidemiology*, *26*(4), 208.

Mei, Z., Serdula, M. K., Liu, J. M., Flores-Ayala, R. C., Wang, L., Ye, R., & L. M. Grummer-Strawn (2014). Iron-containing micronutrient supplementation of Chinese women with no or mild anemia during pregnancy improved iron status but did not affect perinatal anemia. *The Journal of nutrition*, *144*(6), 943-948.

Rasmussen, S., Bergsjø, P., Jacobsen, G., Haram, K. & L. S. Bakketeig (2005). Haemoglobin and serum ferritin in pregnancy—correlation with smoking and body mass index. *European Journal of Obstetrics & Gynecology and Reproductive Biology*, *123*(1), 27-34.

Restrepo, B. J. & M. Rieger (2016). Denmark's Policy on Artificial Trans Fat and Cardiovascular Disease. *American Journal of Preventive Medicine*, *50*(1), 69-76.

Robbins, J., Nelson, J. C., Rautaharju, P. M. & J. S. Gottdiener (2003). The association between the length of the QT interval and mortality in the Cardiovascular Health Study. *The American Journal of Medicine*, *115*(9), 689-694.

Roy, D., Quiles, J., Avanzas, P., Arroyo-Espliguero, R., Sinha, M. & J. C. Kaski (2006). A comparative study of markers of inflammation for the assessment of cardiovascular risk in patients presenting to the emergency department with acute chest pain suggestive of acute coronary syndrome. *International Journal of Cardiology*, *109*(3), 317-321.

Schwertner, H. A., Jackson, W. G. & G. Tolan (1994). Association of low serum concentration of bilirubin with increased risk of coronary artery disease. *Clinical chemistry*, *40*(1), 18-23.

Shiju, T. M., Mohan, V., Balasubramanyam, M. & P. Viswanathan, (2015). Soluble CD36 in plasma and urine: a plausible prognostic marker for diabetic nephropathy. *Journal of Diabetes and its Complications*, *29*(3), 400-406.

Su, S., Snieder, H., Miller, A. H., Ritchie, J., Bremner, J. D., Goldberg, J. & V. Vaccarino (2008). Genetic and environmental influences on systemic markers of inflammation in middle-aged male twins. *Atherosclerosis*, *200*(1), 213-220.

van Beek, J. H., de Moor, M. H., Geels, L. M., Sinke, M. R., de Geus, E. J., Lubke, G. H. & D. I. Boomsma (2014). The association of alcohol intake with gamma-glutamyl transferase (GGT) levels: Evidence for correlated genetic effects. *Drug and Alcohol Dependence*, *134*, 99-105.

Ye, M., Yu, H., Yu, W., Zhang, G., Xiao, L., Zheng, X. & J. Wu (2012). Evaluation of the significance of circulating insulin-like growth factor-1 and C-reactive protein in patients with chronic obstructive pulmonary disease. *Journal of International Medical Research*, *40*(3), 1025-1035.