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Concordance of health states in couples: Analysis of self-reported, nurse administered and blood-based biomarker data in Understanding Society

Authors: Apostolos Davillas, Stephen Pudney

Corresponding author: Stephen Pudney

ScHARR, University of Sheffield,
Regent Court, 30 Regent Street, Sheffield, S1 4DA
Tel: (+44) (0)114 222 9187
Fax: (+44) (0)114 222 0710
Email: steve.pudney@sheffeld.ac.uk

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Concordance of health states in couples:

Analysis of self-reported, nurse administered and blood-based biomarker data in Understanding Society

Apostolos Davillas

Institute for Social and Economic Research, University of Essex

Stephen Pudney

School of Health and Related Research, University of Sheffield

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Abstract: We use a range of self-reported health measures, nurse-administered health measures and blood-based biomarkers to examine the concordance between the health states of partners in marital/cohabiting relationships. A lifecourse model of cumulative health exposures is used to interpret the empirical pattern of between-partner health correlation in relation to the elapsed duration of the relationship. This allows us to distinguish non-causal homogamy correlation arising from assortative mating, from potentially causal effects of shared lifestyle factors. We find important differences between the results for different health indicators, with strongest homogamy correlations observed for adiposity, associated biomarkers like blood pressure, heart rate, blood sugar and cholesterol, and also self-assessed general health. We find no evidence of a "dose-response relationship" for marriage duration, and show theoretically that this implies – perhaps counterintuitively – that shared lifestyle factors and homogamous partner selection make roughly equal contributions to the concordance we observe in most of the health measures we examine.

Keywords: Biomarkers; Health; Homogamy; Spousal concordance; Understanding Society

JEL codes: C2, C8, I10

Contact: Steve Pudney, ScHARR, University of Sheffield, 30 Regent Street, S1 4DA, UK; tel. +44(0)114 222 9187; email: steve.pudney@sheffield.ac.uk

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1 Introduction

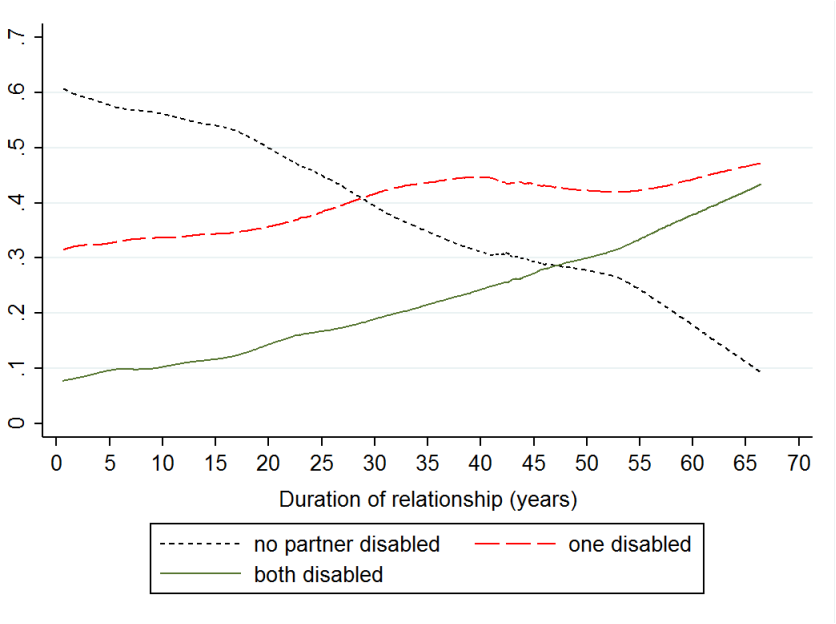
The research literature on the health of couples is relatively sparse: a systematic review by Meyler et al. (2007) identified 103 published articles (58 on mental health, 26 on physical health and 19 on health behaviours). Those numbers are tiny compared to the literature on individuals, twins and siblings. Existing evidence includes cross-section analysis of the spousal associations of incidence for a range of diagnosed diseases (Hippisley-Cox et al., 2002) and prospective studies which have found concordance in some specific health domains, including psychiatric disorders (Joutsenniemi et al., 2011), alcohol dependency (Leadley et al., 2000) and obesity (The and Gordon-Larsen, 2009; Oreffice and Quintana-Domeque, 2010; Wilson, 2012). Many studies are based on self-reported health indicators (Banks et al., 2013; Monden, 2007) or focus on specific health conditions or indicators (Meyler et al., 2007). Moreover, the body of evidence on the initial source of the observed health concordance and its evolution through time as marriages[1] proceed is small, based on preliminary findings that do not explicitly explore the concordance profiles across the duration of the union or use proxies for partnership duration, like the age of the partners (Di Castelnuovo et al., 2009).

There are two major reasons for an interest in the association of morbidities of marital partners. One relates to the consequences for public policy on disability. Any tendency for disease or disability to be concentrated within couples will affect the social cost of disease. It is estimated that the market value of informal care supplied to disabled people in the UK in 2015 amounted to £132bn, which is comparable to the total cost of the National Health Service (Buckner and Yeandle, 2015). A substantial proportion of that cost is met by the domestic partners of disabled people: around a third, in the case of care received by people aged 65 and over (Pickard et al., 2007). This is essentially a system of informal insurance through the pooling of risk by couples but, if disability affects both partners simultaneously, the couple's capacity to provide care for each other may be impaired. Health concordance within couples represents positive correlation of risks and therefore reduces the effectiveness of pooling and self-insurance, increasing dependency on external care services, and raising the costs of social care. Figure 1 illustrates the way this risk varies with marriage duration, using data from waves 2 and 3 of the UK *Understanding Society* household panel described

[1]For economy of language, we use the terms marriage and partnership interchangeably to denote any domestic partnership, whether or not it has legal marital status.

in section 3. The proportion of couples with both partners reporting any functional disability increases from a few percent in new marriages to almost a third after 50 years. Extending this analysis to biomarkers which may be precursors to future disability, gives a picture in terms of the risk of co-disability in couples, rather than current prevalence.

Figure 1: Proportions of couples with 0, 1 and 2 members reporting a limiting health problem or disability, by duration of partnership (smoothed locally-linear regression)



A second reason for interest in comorbidity within marriage is that it may tell us something about the causal processes generating health outcomes later in life. Some of the most influential work on health over the lifecourse, notably that by Barker (1991), emphasises environmental conditions in the foetal stage and infancy as dominant influences on the risk of disease much later in life (see Almond and Currie (2011) for a review). An extreme version of this view envisages little role for domestic partnership as an influence on adult health.

To understand the health of couples, it is important to distinguish two processes: *homogamy* (a tendency for people to choose partners similar to themselves) and *causal concordance* (increasing concordance of health states caused by lifestyle influences shared within the marriage). There is little research on their relative roles in determining the correlation between health outcomes in couples, but there has been a related debate on contagion *versus* homophily in health behaviours and obesity (Christakis and Fowler, 2007; Lyons, 2011). There has been experimental research aiming to distinguish the effects of homophily and

social contagion in some settings (Centola, 2011), but marriage is not amenable to randomized experimental control and the importance of homogamy as a factor underlying health outcomes in couples remains uncertain.

The key difficulty here is the absence of definitive data. The ideal would be a prospective study that samples individuals early in life and then tracks them into and through marriage with other sample members, observing health outcomes in later life. No such study exists on any representative basis. For that reason, most research on couples has been cross-sectional or short-range longitudinal, with the sample of partners selected at a point after marriage. Retrospective recall data has been used (Booker and Pudney, 2013), but there are inevitable doubts about the accuracy of recall of early life events, and the available health indicators in such surveys are often limited.

A further difficulty is the multi-dimensional nature of the concept of health and the difficulty of measuring health in general-population surveys. We use survey data from waves 2 and 3 of the *Understanding Society* household panel and consider an unusually wide range of health indicators of four types: self-reported subjective assessments; self-reported existence of diagnosed conditions; indicators derived from nurse-administered measurements; and biomarkers derived from analysis of blood samples.

We make two main contributions to the literature on spousal concordance of health. First, we show with a simple theoretical model that the variation of the intra-couple health correlation with elapsed marriage duration is informative about the relative importance of assortative mating and shared lifestyle as influences on long-term health outcomes. But we also show that the correlation-duration profile needs careful interpretation: in particular, a constant or even declining correlation does not necessarily imply that shared lifestyle exposures are unimportant. We find empirically that homogamy is an important source of concordance for the highly visible feature of adiposity and also for indicators of dimensions of health that are known to be related to adiposity, including cardiovascular health and diabetes risk. We also find that the correlation between partners' health states is essentially unrelated to the elapsed duration of marriage, which implies – perhaps counter-intuitively – that shared lifestyle factors are of approximately equal importance to homogamy as a source of health concordance. We show that these results are robust to a range of potential

difficulties, including survival bias, the effect of medication, time variation in homogamy, and other features of our research design.

Our second major contribution is to extend the literature on health concordance by using a wide range of health indicators, including self-reported assessments of health and functional difficulties, self-reported diagnoses of specific conditions, nurse-administered health measures and blood-based biomarkers. Unlike many existing studies that rely on self-reported health measures or focus on specific indicators (Banks et al., 2013; Meyler et al., 2007; Monden, 2007; Wilson, 2012), we use a large set of alternative subjective and objective health measures. Subjective indicators have been shown to be predictive of future morbidity (Idler and Benyamini, 1997), but are subject to misreporting (Bago d'Uva et al., 2008) which may result in spurious health concordance because of interactions between partners in the survey interview setting. Reports of diagnosed conditions may be similarly interdependent – for example, a woman's diagnosis of diabetes may prompt her husband or their GP to call for a test for him. Objectively-measured biomarkers are free of this type of cross-contamination but they are designed to be sensitive to specific dimensions of health, so a range of measures should be considered.

2 Theories of health concordance in couples

There are at least six plausible causal mechanisms that could lead to concordance of health outcomes in long-established partnerships. The most obvious rests on household production theory (Becker, 1965), which emphasises the cost advantages of communal production within the home of basic commodities like nutrition and some physical activity. A large body of evidence linking diet and physical exercise to health outcomes (Willett, 1994; Haskell et al., 2007) supports this theory.

Modern medicine and technology have diminished the role of infectious disease as a threat to health, especially in high-income countries, but it remains a possibility in some cases, for example in older couples where both partners may have relatively weak immune systems.

Recent research on the human biome suggests that the microbial flora in the human gut and elsewhere on the body may have an important influence on a number of disease processes (Claesson et al., 2012; Turnbaugh et al., 2006). Little is yet known about the transmission

of microbes between individuals within the home environment but, if there is a mechanism causing people in an intimate relationship to share a microbial population, this may be an important pathway for the incidence of disease.

A fourth possible mechanism involves social engagement. Social isolation has been identified empirically as a psychological stressor, with observable expression in elevated blood levels of lipids and cortisol (Grant et al., 2009). There is a persuasive body of empirical evidence linking stress to physical and mental illness (Chrousos, 1992). Marital partners share to some extent their pattern of social engagement and exposure to social stress, implying correlated health outcomes.

A fifth possibility arises from each partner's strong economic and emotional incentives to have a partner who is in good health. This may prompt each partner to attempt to exert control over the other's health-related behavior (Averett et al., 2008). Such attempts are only likely to be credible and effective if they match the potential controller's own behavior, so intra-marital social control may be mutual and create a tendency for similar health outcomes.

A sixth potential mechanism relates to the marriage market. There is evidence that body shape and possibly other physical signs of health have an influence on individuals' success in partnership formation (Oreffice and Quintana-Domeque, 2010). In relationships that are believed by the partners to be stable and sustained, there could therefore be less incentive to maintain a healthy lifestyle, and there is some corresponding empirical evidence of a tendency for body mass to increase after marriage (The and Gordon-Larsen, 2009).

These theoretical arguments suggest causal mechanisms that would generate shared exposures to a common set of health influences during the course of the marriage, and it is likely that some mixture of these mechanisms is present in the population. We do not attempt the infeasible task of distinguishing them empirically, but instead use a simple model of the accumulation of risk as a statistical description of the shared exposures. Riley (1989) set out a concept of "insult accumulation" which holds that damage from exposures ("insults") experienced during the life course accumulates and leads to a long-term deterioration of health, through a range of behavioural, environmental and biological processes.

Write the birth dates of a husband and wife as b_h and b_w and their date of marriage as m. The couple is observed in the survey at time $T = m + d$, where d is the elapsed duration

of the marriage. At the time of observation, they are aged a_h and a_w, where $a_j = T - b_j$. Their observed health states are $H_h(T)$ and $H_w(T)$. Their (unobserved) perinatal health states were $H_h(b_h)$ and $H_w(b_w)$ and, from birth to marriage, they experience unobserved personal sequences of exposures, $\{z_h(b_h+1)\dots z_h(m)\}$ and $\{z_w(b_w+1)\dots z_w(m)\}$, which may be correlated as a result of assortative mating. From marriage to the survey date T, they continue to experience environmental exposures, which are partly shared and partly personal. The personal components are $\{x_h(m_h+1)\dots x_h(m_h+d)\}$ and $\{x_w(m+1)\dots x_w(m+d)\}$, the shared exposure is $s(m+1)\dots s(m+d)$, and these three processes are assumed mutually independent. This independence is essentially definitional: the shared component is defined to cover all sources of correlation, including the common physical and social environment and standard of living, in addition to specific shared behaviours like diet and exercise.

Let $\lambda(t)$ be a cumulation factor representing the impact on current health of exposures t periods earlier (with $\lambda(0) \equiv 1$). The two partners' observed health states are the cumulative result of perinatal health and subsequent accumulation of exposures:

$$H_j(T) = A_j + \sum_{t=m+1}^{m+d} \lambda(T-t)\ [x_j(t) + s(t)] \qquad\qquad j = h, w \tag{1}$$

where A_j is the component of health arising from pre-marriage exposures:

$$A_j = \lambda(a_j)H_j(b_j) + \sum_{t=b_j+1}^{m} \lambda(T-t)z_j(t) \tag{2}$$

In the appendix, we show that the mean health state of each partner j when observed at time T is a potentially nonlinear function of age a_j so that it is appropriate to use an age-adjusted form of each health indicator as a residual after extracting a nonparametric estimate of the health-age profile.

Define the duration-specific correlation between the two partner's health states, $R(d)$ as $corr(H_h(T), H_w(T) \mid d)$. To allow for persistence and short-term serial correlation in exposures, we assume that each of the processes $\{z_h(t), z_w(t), x_h(t), x_w(t), s(t)\}$ is the sum of a time-invariant persistent effect and a moving average process. In the appendix, we use these assumptions to demonstrate two propositions about the correlation profile $R(d)$:

$$Homogamy\ correlation: \qquad R(0) = corr(A_h, A_w) \tag{3}$$

$$Long-term\ correlation: \qquad R(\infty) = \frac{V_s}{V_x + V_s} \tag{4}$$

where V_s and V_x are the variances of the dominant components – either the variances of the time-invariant components of $s(t)$ and $x_h(t), x_w(t)$ if perfectly persistent components are present, or else the variances of the moving average components if there are no perfectly-persistent components.

$R(d)$ is increasing with duration (in the large) if $R(\infty) > R(0)$, which is satisfied if the proportion of variance attributable to shared exposure is greater than the initial correlation arising from homogamy. Some authors have interpreted the absence of a rising duration profile for the intra-marriage correlation as implying the absence of shared lifestyle influences on partners' health (Wilson, 2012; Monden, 2007). But note that, even if $R(d)$ is flat or slowly declining rather than rising, there could be a substantial degree of causal concordance arising from shared influences during marriage if homogamy is such that $R(0)$ is large.

We estimate the correlation profile $R(d)$ using a 3-stage local smoothing method:

(1) Construct an age-adjusted version of the biomarker or other health indicators as the residuals $\hat{u}_h, \hat{u}_w$ from local linear regressions of $H_h(T)$ and $H_w(T)$ on the age of the respective partners when observed at interview.

(2) For each duration d in a grid of values $d_1 \dots d_K$, calculate the local correlation:

$$\hat{R}(d) = \frac{\sum_i \omega_i(d)\hat{u}_{hi}\hat{u}_{wi} - \left(\sum_i \omega_i(d)\hat{u}_{hi}\right)\left(\sum_i \omega_i(d)\hat{u}_{wi}\right)}{\left[\left(\sum_i \omega_i(d)\hat{u}_{hi}^2 - \left(\sum_i \omega_i(d)\hat{u}_{hi}\right)^2\right)\left(\sum_i \omega_i(d)\hat{u}_{wi}^2 - \left(\sum_i \omega_i(d)\hat{u}_{wi}\right)^2\right)\right]^{1/2}} \tag{5}$$

where $\omega_i(d)$ is the Epanechnikov kernel,[2] rescaled to sum to 1 over all sample observations and h is a specified bandwidth.[3]

(3) Repeat the calculations for each of a set of N bootstrap resamples to compute a confidence interval at each grid point d.

We present results derived both with and without the nonparametric adjustment for age. As we show in the appendix, there is a case for including the couple's ages at marriage in the regression model used for age-adjustment at step (1), and also in the estimation of the duration-specific correlation. In section 5.5 below, we demonstrate the robustness of our results to the inclusion of age-at-marriage variables.

[2]Defined as $0.75\left(1 - [(d_i - d)/h]^2\right)/h$ for $|d_i - d| < h$ and 0 otherwise.

[3]Note that an extension of the Fan and Yao (1998) result on the estimation of conditional variance functions establishes that there is no loss of asymptotic efficiency involved in estimating $R(d)$ from residuals $\hat{u}_h, \hat{u}_w$ rather than the ideal but unavailable true age-adjusted health variables u_h, u_w.

3 Data

The data come from the UK Household Longitudinal Survey (UKHLS), also known as Understanding Society, supplemented in some cases by data from its predecessor, the British Household Panel Survey (BHPS). The UKHLS is a large, national representative panel survey covering about 40,000 households in the UK (Knies, 2015), with a design that involves overlapping 2-year waves. Individuals have been interviewed annually since the initial wave in 2009-10. At wave 2, participants in the BHPS were absorbed into the UKHLS.

A set of health measures and a non-fasted blood sample were collected by trained nurses, 5 months on average after the wave 2 interview for non-BHPS respondents and similarly at wave 3 for the BHPS sample. Respondents were eligible for nurse visits if they took part in the main survey, were aged 16 or over, lived in Great Britain (not Northern Ireland), conducted their interview in English and were not pregnant. Blood sample collections were further restricted to those who had no clotting or bleeding disorders, were not taking anti-clotting medication, and had no history of fits. McFall et al. (2014) describe the design and organisation of the nurse visit sample in further detail.

We pool data from the wave 2 non-BHPS and the wave 3 BHPS samples, giving a potential sample of 48,328 people (wave 2: 36,963; wave 3: 11,365). Of those, 35,937 were eligible for nurse visits and 34,358 for blood sample collection. Of those, 20,700 participated in the nurse visits and blood-based biomarker data are available for 13,107 respondents. Our working sample is restricted to those who were in a cohabitating or marital relationship at the time of interview (wave 2 and 3 for the non-BHPS and BHPS samples). Information on the month and year the current relationship started was collected separately from each partner. For the non-BHPS wave 2 sample, start dates are derived from the partnership history questionnaire modules (at UKHLS waves 1 and 2). For the BHPS sample, we used the partnership history file, containing retrospective lifetime partnership histories and subsequent panel data information from 1-18 BHPS waves (Pronzato, 2011), and the partnership history modules at UKHLS waves 2 and 3.

For unmarried cohabitating respondents, we use the date (month and year) at which the couple began cohabiting as the starting point of their union. For married couples, we use the date of marriage or start of cohabitation, if earlier. The duration of the union is calculated

by subtracting these dates from the date of the interview or nurse visit at which the relevant health indicator was measured. Self-reported health measures are generated by the main survey interview and biomedical measures by the nurse visits (on average 5 months later). Partners report durations separately, so they can be compared and cross-checked. Overall, 81% of couples agreed exactly about the start of their union and disparities are less than one year for 94% of couples. Our final duration variables are averaged across partners if both partners report and duration is based on one partner only if relevant data are missing for the other. Couples with duration differences of more than one year were excluded. The analysis sample for biomedical measures (sections 3.3 and 3.4 below) contains a maximum of 4,582 couples; for self-reported health measures (sections 3.1 and 3.2), the nurse visit was not required, so the available sample expands to a maximum of 12,899 couples.

We use a wide range of health measures ranging from subjective assessments to objective anthropometric measurements and blood-based biomarkers. All health indicators are potentially 'distorted' as indicators of underlying health by the effect of medication, particularly the cholesterol and blood sugar biomarkers which are used in diagnosis. The results presented in section 4 are based on analysis of health measures in directly observed form. But those measures may be influenced directly (since measures like blood pressure and cholesterol are used as diagnostic criteria) or indirectly by medication, which may generate bias. However, in section 5.2 we show that the findings are robust to alternative ways of addressing the medication effect.

3.1 Subjective health measures

We use four subjective health assessments, collected in the main survey interview at wave 2 (non-BHPS sample) or 3 (BHPS sample). The conventional self-assessed health (SAH) measure categorises respondents on a five-point scale, ranging from 1 = excellent to 5 = poor health. Such assessments are subject to misreporting, depending on circumstances (Bago d'Uva et al., 2008). However, although crude, SAH has been found to be predictive of future morbidity (Idler and Benyamini, 1997).

SF-12 is a 12-item generic measure of health-related quality of life. Although SF-12 is self-reported and contains SAH as one of its components, it has shown to be a stronger predictor of health outcomes than SAH alone (Frick and Ziebarth, 2013). Our main focus

here is on physical health, so we use the PCS-12 sub-measure (McDowell, 2006), constructed to take values between 0 and 100 and standardized mean 50 and standard deviation 10. Higher scores indicate better physical functioning.

We also examine two self-reported functional disability measures. Respondents were asked if they experienced any long-standing physical or mental impairment, illness or disability; those who answered yes were then asked if those health problems/disabilities mean substantial difficulties with: mobility; lifting; carrying or moving objects; manual dexterity; continence; hearing; sight; communication; memory or ability to concentrate, learn or understand; recognising physical danger; recognising physical coordination; personal care; or other difficulty. Those reporting functional difficulty in at least one domain of life are classified as disabled. We also use the number of reported functional difficulties as an index of severity of disability, ranging ranging from 0 to 11.

3.2 Self-reported diagnosed conditions

Self-reported diagnoses of specific chronic health conditions are derived from questions at wave 1 or 2. Respondents were asked whether they had ever been told by a doctor or other health professional that they had any health condition from a list provided. These diagnosis indicators are restricted to the UKHLS sample since non-comparable questions were asked of the BHPS sample. Because of the low prevalence rate of some of the specified conditions, we grouped them as: arthritis; cardiovascular disease (congestive heart failure, coronary heart disease, angina, heart attack, stroke or hypertension); endocrine disease (hyperthyroidism, hypothyroidism or any type of diabetes); and respiratory disease (asthma, emphysema or chronic bronchitis). High blood pressure is also examined as separate outcome because of its relatively high prevalence rates and for comparison with relevant clinically measured indicators. The number of reported health conditions is also used as an index of comorbidities.[4]

[4]Except for the previously listed health conditions, we also include cancer, epilepsy any kind of liver condition when calculating the number of reported health conditions. These conditions cannot be grouped in any of the previous categories and their very low prevalence prevents us from considering them separately.

3.3 Nurse-measured indicators

A range of measures were collected at the nurse visits; we focus on adiposity, resting heart rate and blood pressure. In the absence of a gold standard adiposity measure (O'Neill, 2015), we use waist circumference (WC) and the body mass index (BMI). WC captures central adiposity and is considered a stronger predictor of health risks than BMI (Janssen et al., 2004). For respondents weighed as more than 130kg, their (own) estimated body weight was used to construct BMI because the scales were not designed to be accurate above this level. Waist circumference (in cm) was measured at the midpoint between the lower rib and the upper margin of the iliac crest. It was measured twice and again if there was a difference exceeding 3cm. We followed common practice (Hamer and Stamatakis, 2012) in taking the mean of the two closest measurements.

Blood pressure (BP) and resting heart rate (HR) measurements were made by the nurses in accordance with the European Society of Hypertension guidelines for blood pressure monitoring (Parati et al., 2008). Respondents were asked not to eat, smoke, drink alcohol or participate in any vigorous activity for 30 minutes before the nurse visits (non-compliers are excluded from our analysis of BP and HR). Three consecutive readings of systolic and diastolic blood pressure (in mmHg) and HR (in beats per minute) were taken at one minute intervals. We use the average of the second and third reading, to allow for the possibility that the first measurement might be higher than usual (Johnston et al., 2009).[5] Systolic blood pressure (SBP) is the maximum pressure in an artery at the moment when the heart is pumping blood and diastolic blood pressure (DBP) is the lowest pressure in an artery in the moments between beats when the heart is resting. SBP and HR are generally considered more relevant to health risks than DBP (Haider et al., 2003). We treat SBP, DBP and HR as continuous variables, but also construct a binary variable indicating hypertension as SBP > 140 or DBP > 90 (see Table 1).

3.4 Blood-based biomarkers

We study inflammatory, blood glucose and 'fat in the blood' biomarkers. The results of the next section are based on directly observed biomarkers adjusted for age but not for the effects

[5]The HR is sometimes interpreted as a measure of cardiovascular fitness rather than health – a rather subtle distinction.

Table 1: Clinically defined thresholds for biological risk factors

Indicator	Threshold	
High risk clinical thresholds		
Systolic blood pressure (SBP)	140 mmHg	Chobanian et al. (2003)
Diastolic blood pressure (DBP)	90 mmHg	Chobanian et al. (2003)
Resting heart rate (HR)	90 bpm	Seccareccia et al. (2001)
Total cholesterol (TC)	6.2 mmol/L	NCEPEP (2001)
HDL cholesterol	1 mmol/L	NCEPEP (2001)
Triglycerides (TG)	2 mmol/L	Kolovou et al. (2011)
HbA1c	48 mmol/mol	WHO (2011)
C-Reactive Protein (CRP)	3 mg/L	Pearson et al. (2003)
Fibrinogen[§]	2.7g/L (men), 2.8g/L (women)	
Categorical variables for the cardiovascular risk score index		
Normal =0	WC < 94 cm (men), WC < 80 cm (women)	
Overweight=1	94 ≤ WC < 102 (men), 80 ≤ WC < 88cm (women)	
Obese=2	WC ≥ 102 (men), WC ≥ 88cm (women)	
Normotensive = 0	SBP < 120 and DBP < 80 mmHg	
Pre-hypertensive = 1	120 ≤ SBP < 140 or 80 ≤ DBP < 90 mmHg	
Hypertensive = 2	SBP ≥140 or DBP ≥ 90 mmHg	
Non-diabetic = 0	HbA1c < 42 mmol/mol	
Pre-diabetic = 1	42 ≤ HbA1c < 48 mmol/mol	
Diabetic = 2	HbA1c ≥ 48 mmol/mol	
Inflammation: low risk = 0	CRP < 1.0 mg/L	
Inflammation: medium risk = 1	1.0 ≤ CRP < 3.0 mg/L	
Inflammation: high risk = 2	CRP ≥ 3.0 mg/L	

[§] In the absence of clinical cut-points for Fibrinogen, we use the 75th percentile of gender-specific distributions.

of any medication that respondents might be taking. Since medication is often targeted at the condition causing abnormal biomarker readings, it may prevent the measured biomarker from reflecting fully the underlying morbidity as we would wish. We investigate this issue in section 5.2 and find no evidence of significant distortions from the use of medication.

C-reactive protein (CRP) and Fibrinogen are our biomarkers for inflammation. CRP (in mg/ L) is an acute phase protein in the blood associated with general chronic or systemic inflammation which reflects disease history and may be a risk factor for future health and mortality(Emerging Risk Factors Collaboration, 2010). CRP rises as part of the immune response to infection and is elevated in the presence of chronic conditions. Participants with CRP over 10 mg/L are excluded from analysis, since this is considered evidence of current infection rather than chronic processes (Pearson et al., 2003). Fibrinogen (in g/L)

is a glycoprotein that aids the body to stop bleeding by promoting blood clotting, but it is also regarded as an inflammatory biomarker (Jain et al., 2011) and is directly related to coronary artery thrombosis. We use CRP and Fibrinogen as continuous variables.

Glycated haemoglobin (HbA1c) is used as a continuous measure (in mmol/mol) of the level of sugar in the blood over the 8-12 weeks before measurement, and is validated as a diagnostic test for diabetes (WHO, 2011).

Cholesterol and triglycerides concentrations are markers for fatty substances in the blood.[6] Total cholesterol (TC) includes both "good" high-density lipoprotein (HDL) cholesterol and "bad" varieties, mainly low-density lipoprotein (LDL) and very-low-density lipoprotein (VLDL). Since reliable measurement of LDL cholesterol requires study participants to fast, TC and HDL (both in mmol/L) are the appropriate measures to use here (Benzeval et al., 2014). Triglycerides (TG) are blood-borne fats (lipids) coming from dietary sources or the liver. High levels of TC and TG and low levels of HDL cholesterol are associated with increased risk of cardiovascular disease (Liu et al., 2013).

We also use two cumulative risk score indexes, ranging from 0 to 8, which combine dimensions of health to give an overall assessment of respondents' physiological condition and dysregulation. The first is a summary index of multi-system risk that captures the cumulative strain on organs and tissues. Such indexes are often called allostatic load and regarded as a measure of the wear and tear on the body reflecting the physiological consequences of chronic or repeated exposure to stress. Our index combines markers for inflammation (CRP and Fibrinogen), cardiovascular (SBP, DBP and HR) and metabolic (WC, TC, HDL, TG and HbA1c) functioning, by summing binary indicators defined on clinical thresholds (Table 1).[7] Following Walsemann et al. (2016), we construct a second index for cardiovascular disease risk by summing four risk factors, WC, BP, HbA1c and CRP, each divided into categories (labelled 0, 1, 2) on the basis of clinically significant thresholds (Table 1).

[6] In the early literature, triglycerides were recommended for use only in fasting samples, but recent opinion holds that triglycerides provide a good measure in non-fasting samples, as long as the appropriate clinical threshold is used (Kolovou et al., 2011).

[7] This index relates to secondary and tertiary response to stress. Some authors include cortisol as a component to capture primary responses, but cortisol was not collected because of time-of-day and other measurement difficulties in the UKHLS context. Similar constructions to ours have been used in previous studies (Vie et al., 2014).

4 Results

4.1 Homogamy measures

We estimate the homogamy correlation $R(0)$ using a bandwidth of $h = 7.5$, so only marriages starting within 7.5 years of the interview contribute to the estimate.[8] Table 2 shows the result from an analysis of the complete case sample consisting of couples with both partners surviving to the wave 2 or 3 interview where both provide an observation on the relevant health indicator. The available sample size is considerably larger for the self-reported indicators than for the biomarkers. Section 5.4 explores robustness with respect to sample selection and finds no major impact.

We find evidence for a modest degree of health-related homogamy. Generic self-assessed health measures give highly significant early-relationship correlations of 0.19-0.28 without age adjustment and 0.16-0.26 with adjustment. There is weaker evidence of a homogamy effect in self-reported diagnosed conditions. The loss of significance (perticularly in the age-adjusted case) is partly due to the lower statistical precision for estimates based on binary indicators, but may also arise because such conditions often take a long time to become symptomatic and would thus be potential rather than diagnosed at the start of the relationship when the two partners are relatively young.

We find that the homogamy correlations for the two adiposity measures are higher in magnitude (about 0.36 after adjusting for age in the case of both BMI and WC) comparing to the other health outcomes. This is unsurprising, since adiposity is a highly visible personal characteristic that may be manifest early in life and is likely to be involved in partner selection. Homogamy correlations are also observed for BP and HR. However, their magnitude is lower; for example, the homogamy correlation for SBP is 0.23 (unadjusted) or 0.17 (age-adjusted) and significant at the 1% level in both cases. For the blood-based biomarkers, there are statistically significant homogamy correlations for HDL cholesterol, HbA1c and Fibrinogen, and also for the composite measures of allostatic load and CVD risk. Again, the correlation profiles are reduced in magnitude following age adjustment but remain significant at the 5% level.

[8] Point estimates are little changed when the bandwidth is reduced to 5 years, but confidence intervals are somewhat wider.

Table 2: Estimated homogamy correlations (bandwidth h = 7.5 years)

Health measure	Unadjusted $\hat{R}(0)$	Unadjusted std.err.	Age-adjusted $\hat{R}(0)$	Age-adjusted std.err.	Number of couples
Self-assessed generic health measures					
Self-assessed health	0.281***	0.022	0.257***	0.021	12,881
Functional difficulty§	0.211***	0.032	0.177***	0.032	10,975
Number of functional difficulties	0.190***	0.036	0.156***	0.038	10,975
PCS-12	0.207***	0.031	0.170***	0.029	8,400
Self-reported diagnosed conditions					
Arthritis§	0.208***	0.051	0.104*	0.054	8,156
Respiratory§	0.016	0.028	0.013	0.027	8,156
High blood pressure§	0.066**	0.033	0.008	0.032	8,156
Endocrine§	0.054	0.034	0.039	0.044	8,156
Any cardiovascular condition§	0.127***	0.034	0.064*	0.034	8,156
Number of conditions	0.135***	0.037	0.062*	0.032	8,156
Adiposity					
BMI	0.385***	0.038	0.362***	0.040	4,308
Waist circumference (WC)	0.409***	0.038	0.365***	0.040	4,443
Blood pressure/heart rate measurements					
Systolic blood pressure (SBP)	0.233***	0.060	0.166***	0.058	3,395
Diastolic blood pressure (DBP)	0.178***	0.049	0.121**	0.049	3,395
Hypertension§	0.203***	0.066	0.138**	0.063	3,395
Resting heart rate	0.178***	0.048	0.175***	0.048	3,395
Biomarkers					
Total cholesterol (TC)	0.110*	0.065	0.129*	0.066	2,207
HDL cholesterol	0.224***	0.057	0.232***	0.061	2,197
Triglycerides (TG)	0.101	0.061	0.094	0.062	2,203
HbA1c	0.337***	0.075	0.236***	0.077	2,006
C-Reactive Protein (CRP)	0.071	0.066	0.070	0.067	2,000
Fibrinogen	0.186***	0.065	0.143**	0.073	2,189
Systemic risk scores (from categorical indicators: Table 1)					
Allostatic load	0.215**	0.088	0.193**	0.084	1,323
CVD risk score	0.373***	0.086	0.301**	0.083	1,353

§ Binary indicator. Bootstrapped standard errors (500 replications). *, **, *** =significant at 10%, 5% , 1%

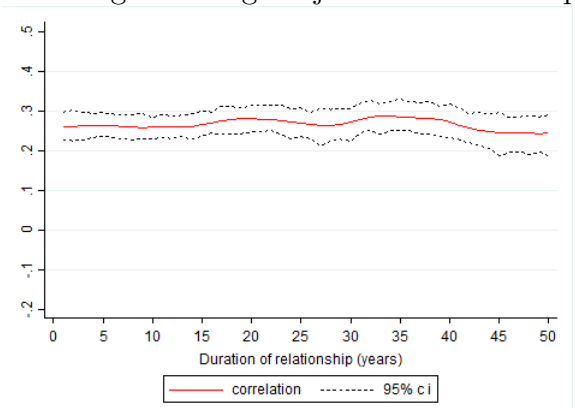
4.2 Correlation profiles

Figures 2-6 show the estimated correlation profiles $\hat{R}(d)$ and the 95% pointwise confidence interval at each duration, for subjective general health assessments, self-reported diagnoses, nurse-administered measures, blood-based biomarkers and derived imeasures of allostatic

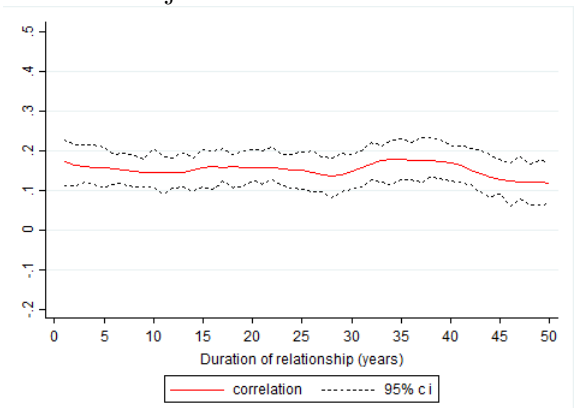
load and CVD risk respectively.[9]

All the estimated correlation profiles are essentially flat. There is no evidence of an increase in the correlation with duration of marriage (which would have implied that shared lifestyle factors account for more of the variation between couples than do individual-specific factors). But equally, there is no significant decline with duration. This finding is consistent with the proposition that assortative mating and shared lifestyle factors make approximately equal contributions to the modest between-partner correlations that we observe. There is considerable consistency: all the indicators which are able to detect correlation at all generate this same conclusion, so it appear rather robust across the various dimensions of health. This finding is also consistent with the conclusions of Booker and Pudney (2013), who used a latent variable analysis of long-term recall data.

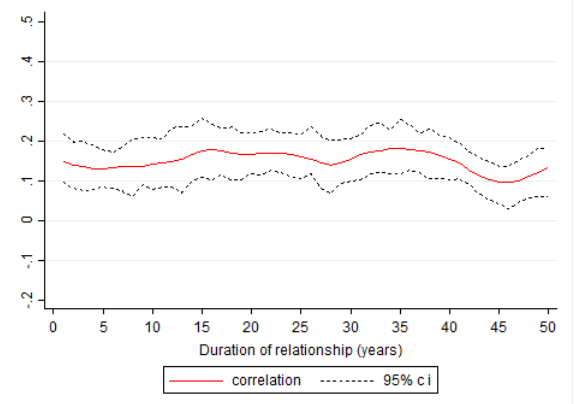
Figure 2: Age-adjusted correlation profiles for subjective health indicators



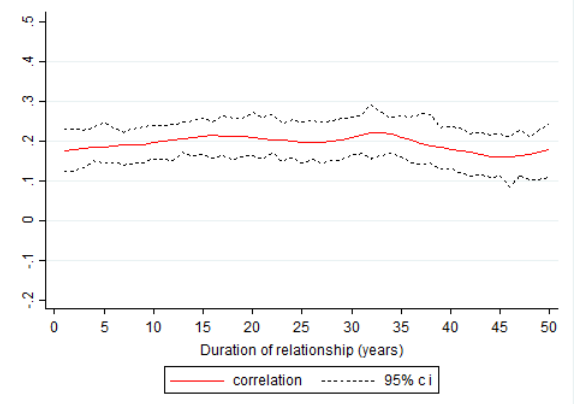
(a) Self-assessed health



(b) Functional difficulty



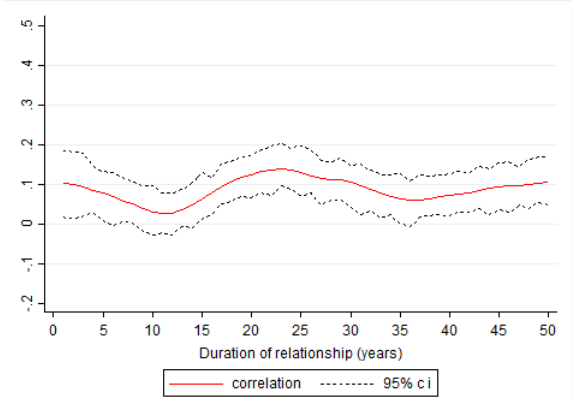
(c) Number of functional difficulties



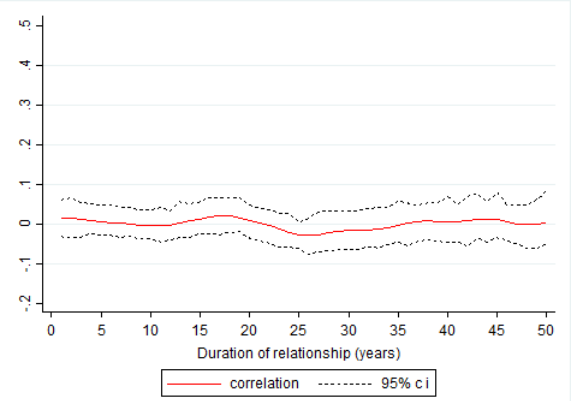
(c) PCS-12

[9]We show only age-adjusted estimates; unadjusted versions are qualitatively very similar and are available from the authors on request.

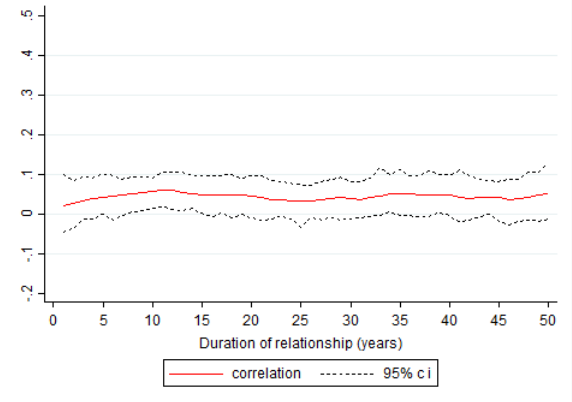
Figure 3: Age-adjusted correlation profiles for self-reported diagnosed conditions



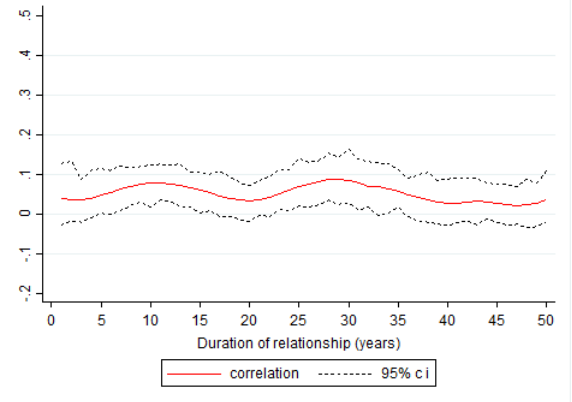
(a) Arthritis



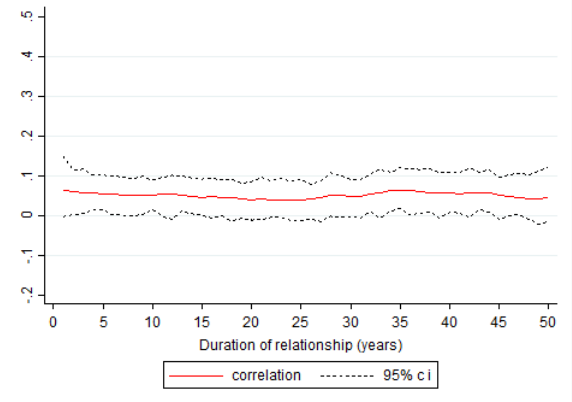
(b) Respiratory disease



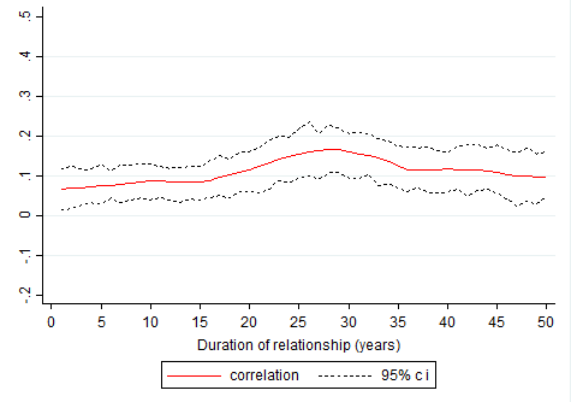
(c) High blood pressure



(d) Endocrine

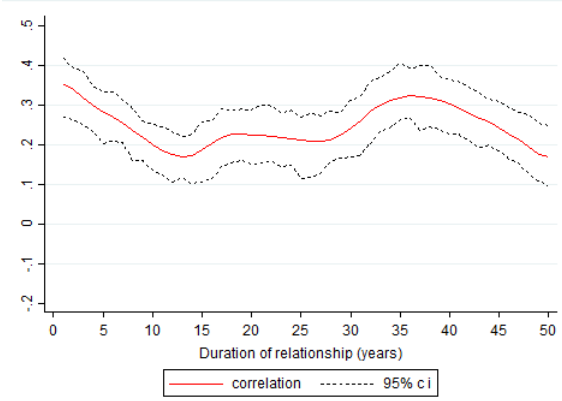


(e) Any cardiovascular disease



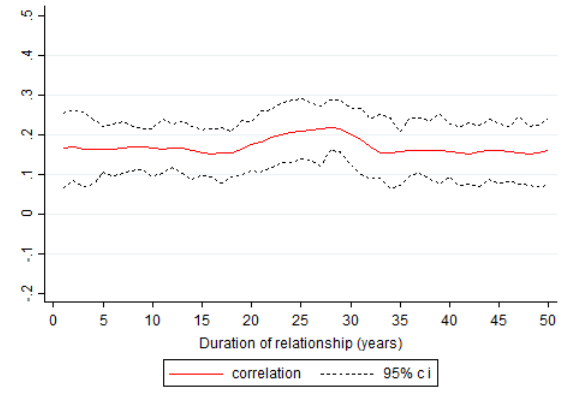
(f) Number of conditions

Figure 4: Age-adjusted correlation profiles for nurse-measured indicators

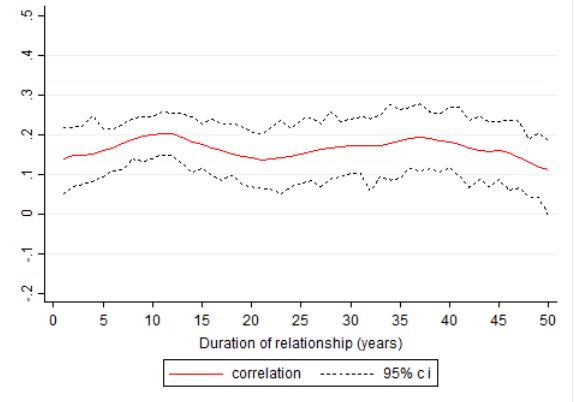


(a) BMI

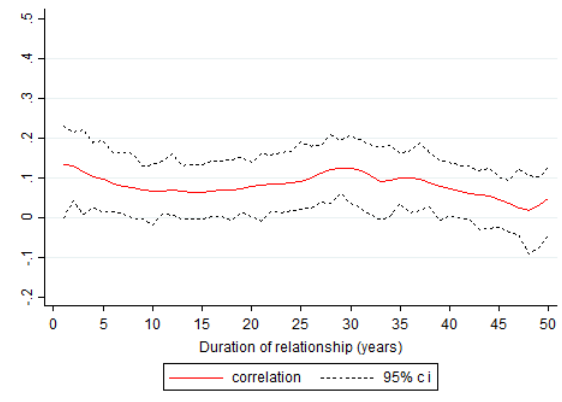
(b) Waist circumference



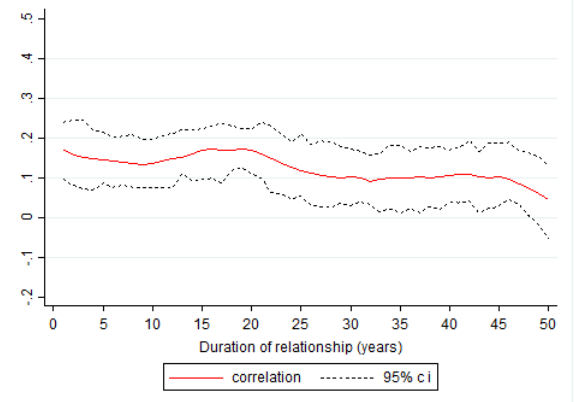
(c) Systolic BP



(d) Diastolic BP

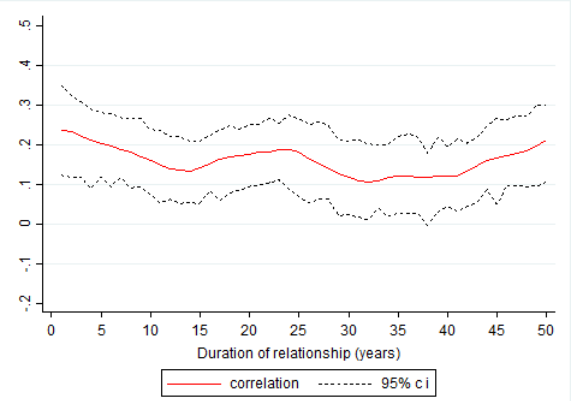


(e) Hypertension

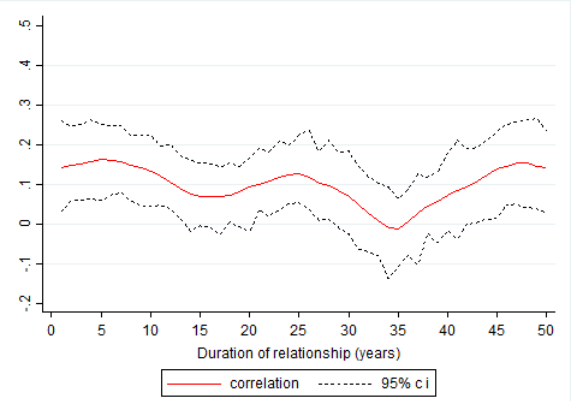


(f) Resting heart rate

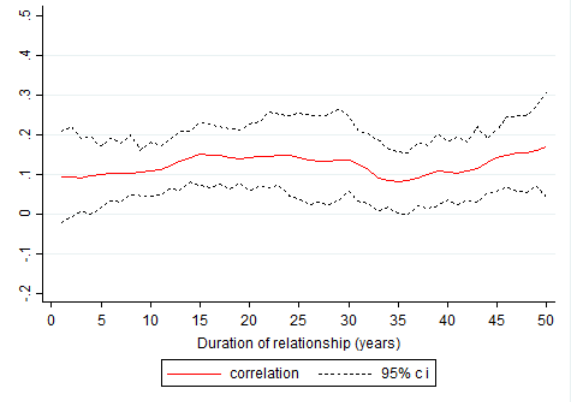
Figure 5: Age-adjusted correlation profiles for blood-based biomarkers



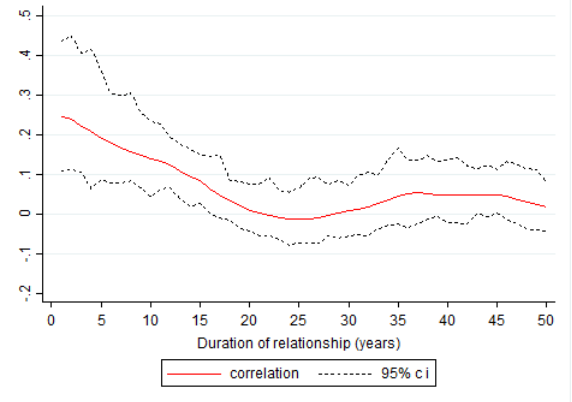
(a) HDL cholesterol



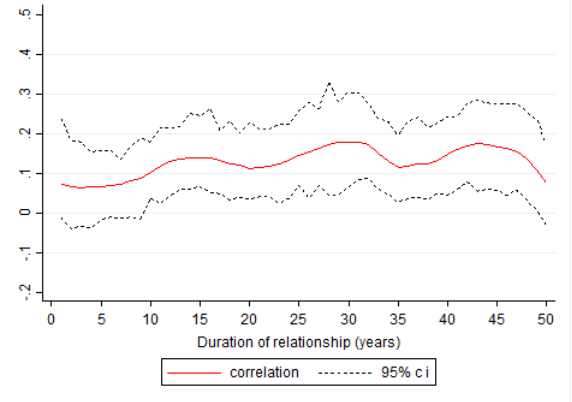
(b) Total cholesterol



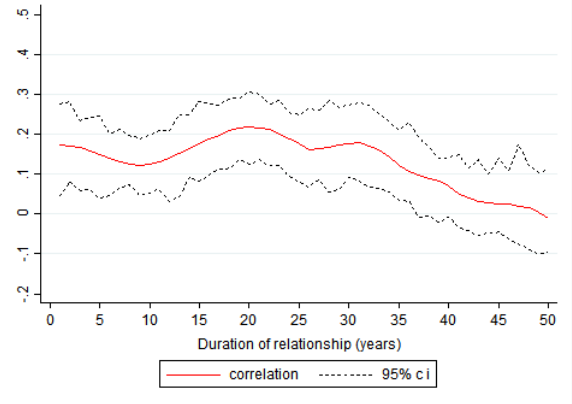
(c) Triglycerides



(d)HbA1c

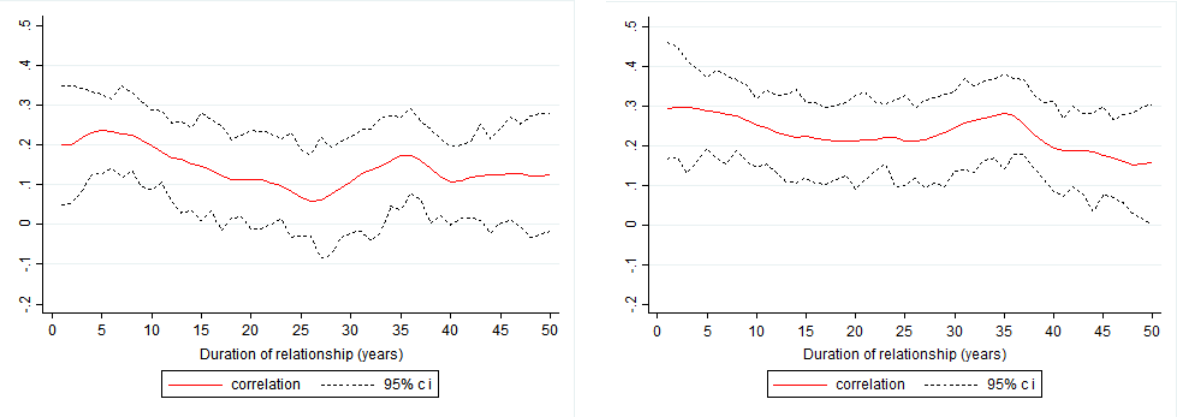


(e) C-Reactive Protein



(f) Fibrinogen

Figure 6: Age-adjusted correlation profiles for allostatic load and CVD risk



(a) Allostatic load

(b) CVD risk

5 Robustness

There are three major complications not so far addressed: survival bias, the effect of medication and the possible confounding of duration profiles by time variations in the strength of homogamy. Sections 5.1-5.3 investigate the robustness of our findings to these, and sections 5.4-5.5 examine alternatives for selection of the analysis sample and adjustment of health indicators for age at marriage in addition to age at health measurement.

5.1 Mortality and survival bias

Mortality is obviously related to health and, by sampling from the stock of couples with two surviving partners, we under-represent marriages which end early through mortality. This may or may not be a problem, depending on the purpose of the analysis. In section 1, we gave two important motivations for our analysis of between-partner health correlations. One relates to the increased care needs of couples with a high risk of both partners being or becoming disabled. The appropriate analysis in that case is a statistical description of the joint occurrence of ill-health (or risk of ill-health) in couples that are currently intact. Since the set of intact couples constitutes the population of interest, there is no problem of survival bias and no special measures need to be taken.

An alternative motivation is to shed light on the causal processes leading to ill-health in couples. In this case, the statistical population of interest is the set of all marriages which started during some specified period, and the appropriate correlation-duration profile describes the evolution of couples' health within that population. Mortality is then a complicating factor, because an analysis of couples which are intact at a given interview date excludes marriages in which one or both partners have died prior to the time of interview. Since mortality risk is related to health state, this generally leads to survival bias. We expect the bias to attenuate the spousal correlation in samples of intact marriages, more so at longer durations where the risk of widowhood is greater, leading also to underestimation of any positive gradient in the correlation-duration profile.

A complication is that bereavement may have a direct causal effect on the health of the surviving partner, additional to the influence of shared lifestyle factors that existed during the marriage. A growing body of evidence (Carey et al., 2014) finds an elevated risk of myocardial infarction, stroke and atrial fibrillation immediately after the death of a partner. The increase appears transient, with risk levels returning to background levels within one year. In the sensitivity analysis, we discard observations on widow(er)s who lost a partner less than two years before interview.

To indicate the likely size and nature of mortality bias, we extend the analysis by including widow(er)s in the sample for analysis, expanding the sample by at most 2,477 for self-reported health measures and 1,244 for measures from the nurse visit. We use three alternative imputation methods to generate a dummy observation on the deceased partner's indicator H. For these non-intact marriages, duration d is defined as the length of time between the date of marriage and the surviving partner's observation. We impute the missing (age-adjusted and gender-specific) indicator H for the deceased partner by using the value observed for another sample member of the appropriate gender, selected in one of the following ways:

(i) *Random imputation*: simple random hot-deck imputation from the sample of intact marriages.

(ii) *Matched imputation*: Mahalanobis matching to select the intact marriage which is closest to the non-intact marriage in terms of duration d and the health H of the surviving partner.

(iii) *Concordant imputation*: assign the (age and gender-adjusted) health indicator of the surviving spouse to the deceased partner.

Random imputation must give a lower spousal correlation than concordant imputation, since the latter implies perfect correlation. We would expect matched imputation to give an intermediate result, but there is no necessity for that to happen.

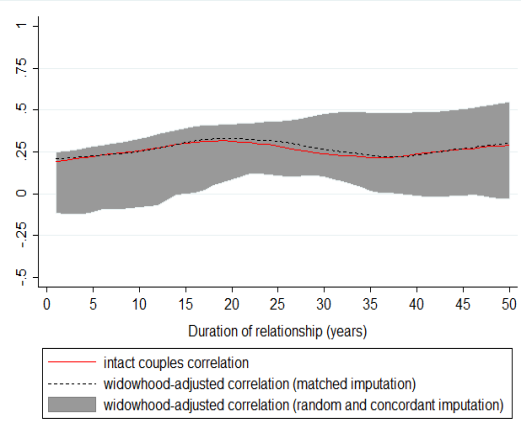
Figures 7-11 show the results for the same sets of health measures as Figures 2-6. The result is qualitatively similar for all indicators. Matched imputation changes the empirical correlation profile very little. The shaded region lying between the upper bound estimated using random imputation (assuming a randomly-allocated health outcome for the missing partner in non-intact marriages) and the lower bound from perfectly concordant imputation (where the missing partner is assigned an identical health outcome) gives an idea of the largest possible impact of mortality bias.

In every case, concordant imputation raises the profile to a modest degree and, in most cases, imparts a slight positive gradient. But the effect is not sufficient to change our earlier conclusions in a major way. The profile remains rather flat, suggesting that the proportion of total variance attributable to the shared marital factor is comparable in magnitude to the initial homogamy effect.

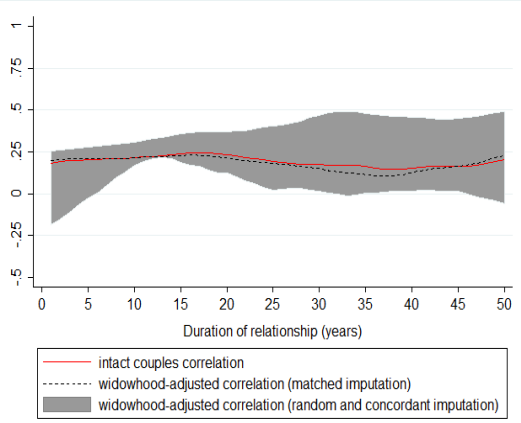
Finally, excluding observations on widow(er)s who lost a partner less than two years before the interview (roughly 10% of the widowed sample) made no perceptible difference to the estimated correlation profiles.[10]

[10] Available upon request.

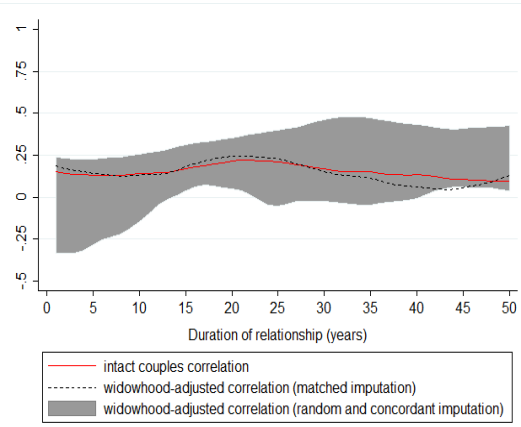
Figure 7: Correlation profiles adjusted for age and widowhood: subjective health indicators



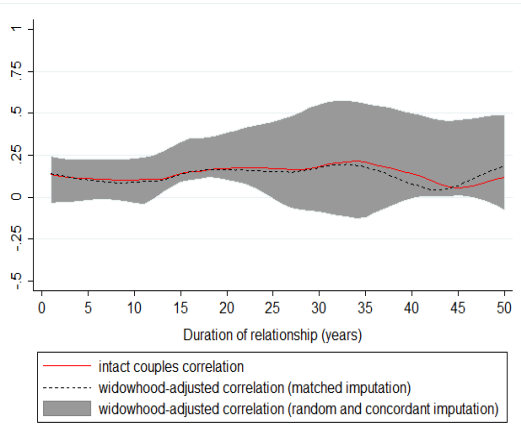
(a) Self-assessed health



(b) PCS-12

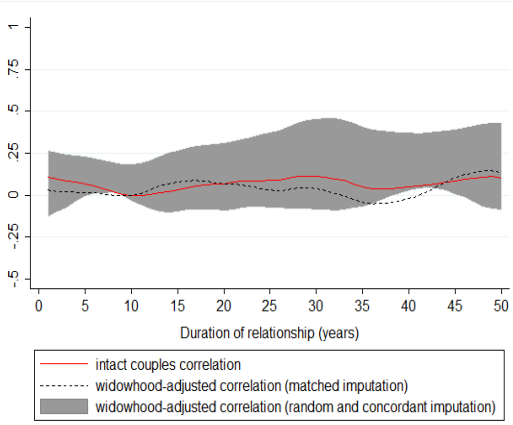


(c) Functional difficulty

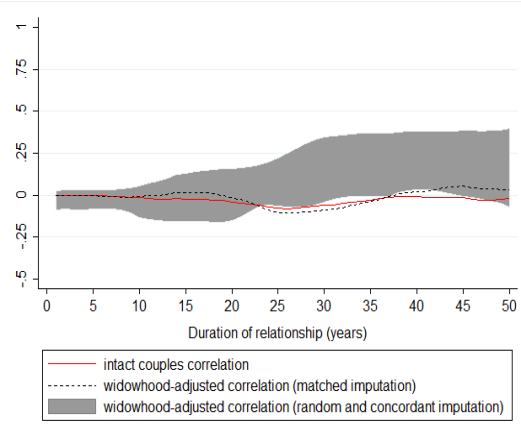


(d) Number of functional difficulties

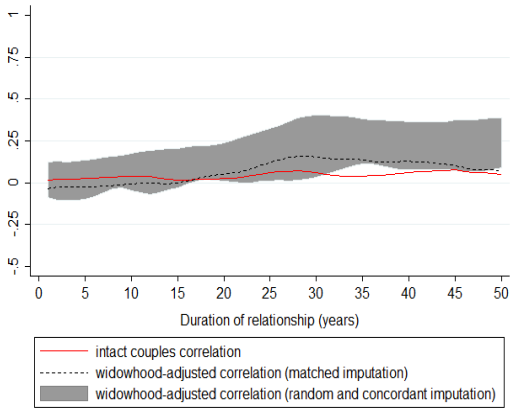
Figure 8: Correlation profiles adjusted for age and widowhood: self-reported diagnosed conditions



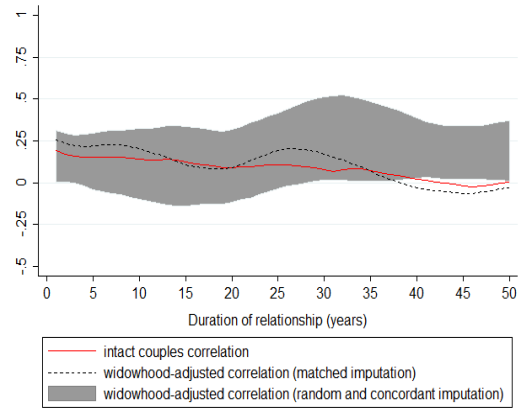
(a) Arthritis



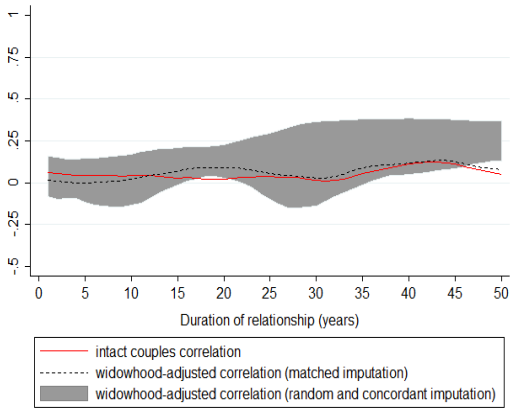
(b) Respiratory disease



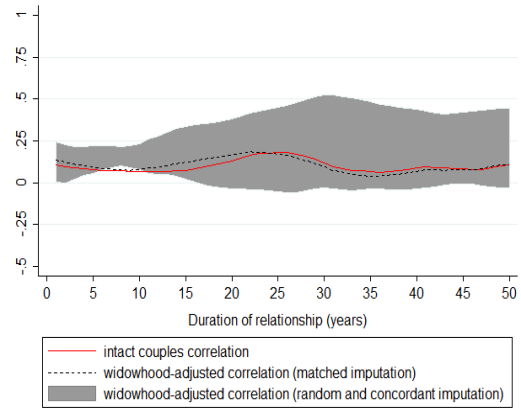
(c) High blood pressure



(d) Endocrine

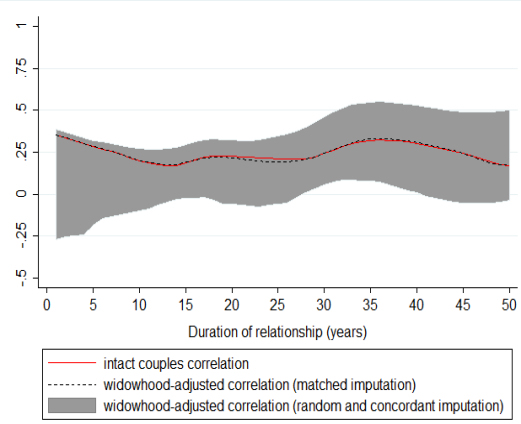


(e) Any cardiovascular disease

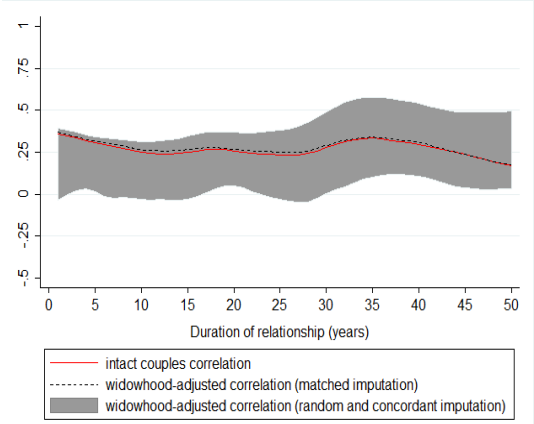


(f) Number of conditions

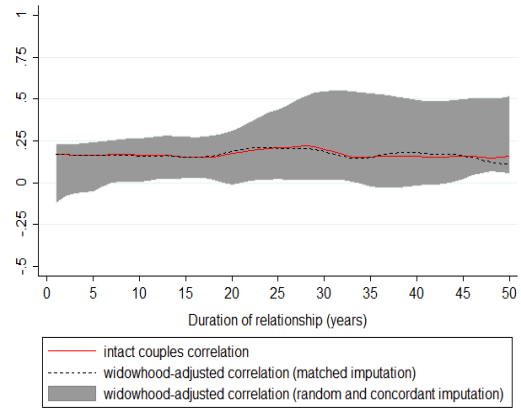
Figure 9: Correlation profiles adjusted for age and widowhood: nurse-measured indicators



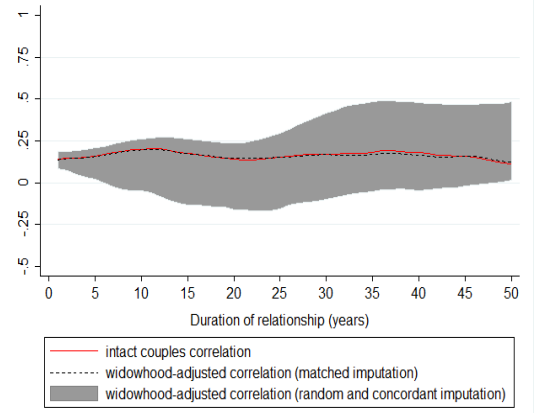
(a) BMI



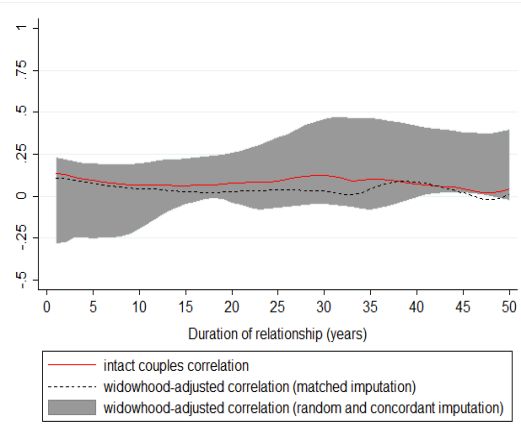
(d) Waist circumference



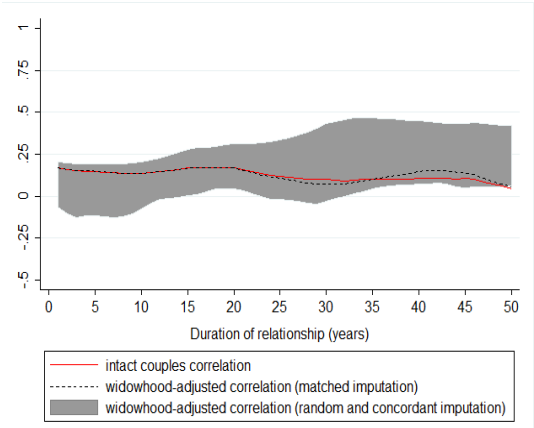
(c) Systolic BP



(d) Diastolic BP

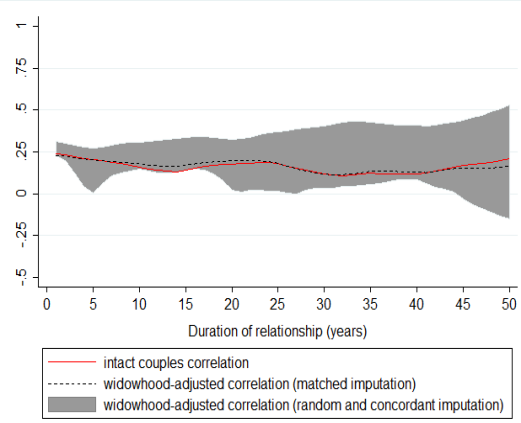


(e) Hypertension

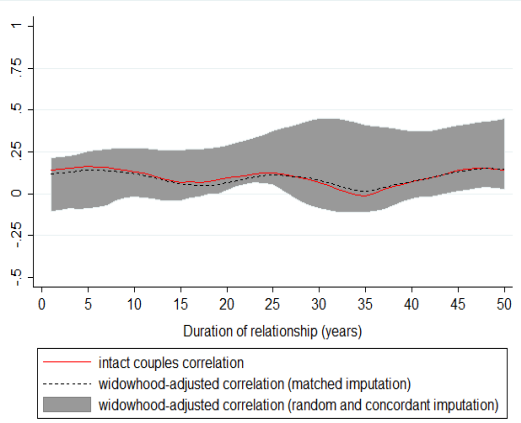


(f) Resting heart rate

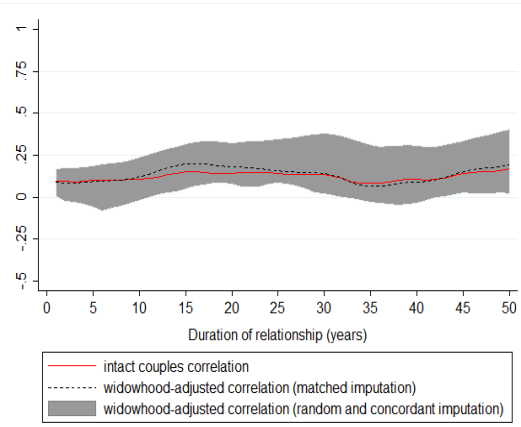
Figure 10: Correlation profiles adjusted for age and widowhood: blood-based biomarkers



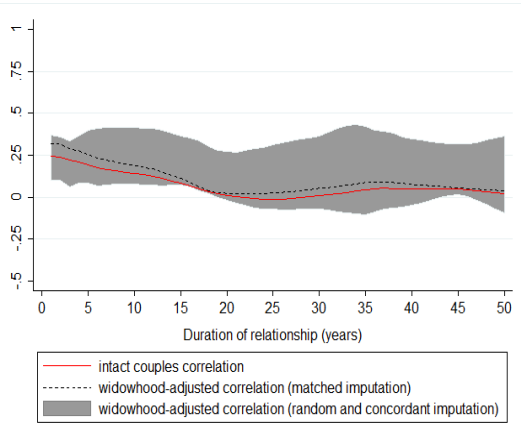
(a) HDL cholesterol



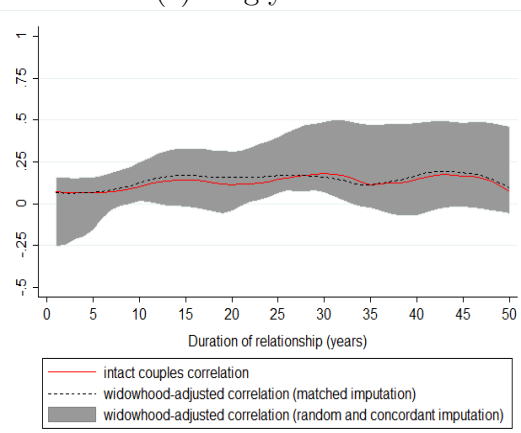
(b) Total cholesterol



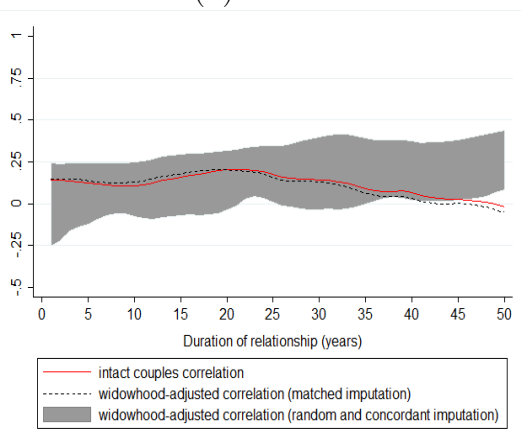
(c) Triglycerides



(d)HbA1c

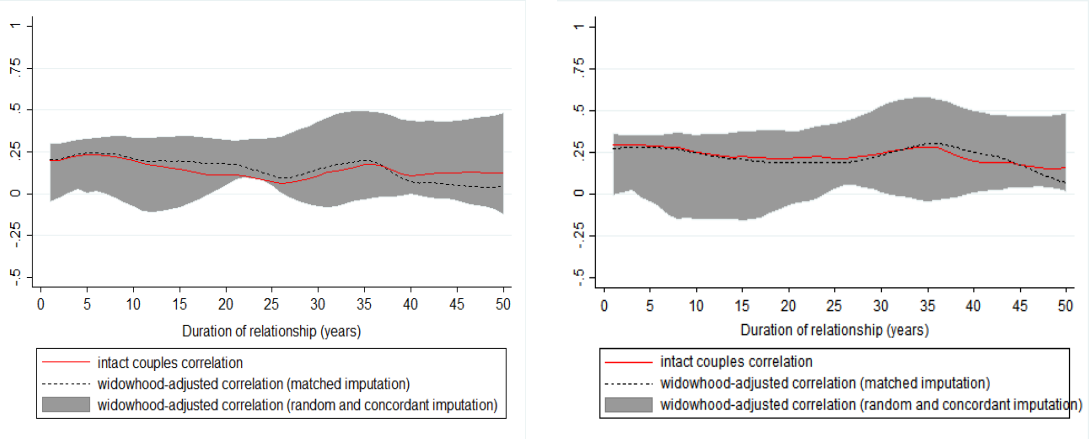


(e) C-Reactive Protein



(f) Fibrinogen

Figure 11: Correlation profiles adjusted for age and widowhood: summary risk scores



(a) Allostatic load

(b) CVD risk

5.2 Medication

In the analysis of section 4, we ignored the effect of medication as a confounding factor. There are three potential sources of bias relating to medication. First, use of medication is related to the individual's health state, so there is non-ignorable selection into treatment – indeed, medication is often calibrated to target specific 'safe' threshold levels of cholesterol or blood pressure. This means that a respondent with a biomarker maintained at a target level by medication may have an underlying health state quite different than that of a respondent with the same biomarker level, maintained naturally. Second, although the UKHLS collects information on prescribed medications, we do not observe dosage, and there may be error in the respondent's or nurse-interviewer's report of medication used. A third potential source of bias are the strong assumptions underlying the statistical method (such as imputation, sample exclusion) used to adjust the observed biomarker levels. In our view, there is no "gold-standard" method of adjusting for the effect of medication, since the problem of selection into treatment causes identification difficulties that are impossible to solve convincingly in an observational setting. Instead, we investigate the robustness of our findings by exploring the impact of three adjustment approaches that are commonly used in the research literature (Cui et al., 2003; Johnston et al., 2009). In the case of blood pressure, for each respondent observed to be on medication:

(i) fixed-increment of 10 mmHg (SBP) and 5 mmHg (DBP) (Cui et al., 2003);

(ii) exclusion from the sample (Cui et al., 2003)

(iii) modified binary hypertension measure, defining the indicator as BP > 140/90 or currently on anti-hypertensive medication.

We also adjust for the impact of statins on TC and TG and anti-diabetic medications on HbA1c, using sample exclusion and, where possible, fixed increment imputation based on clinical evidence.[11] Anti-inflammatory drugs and statins may affect CRP levels (Sheng et al., 2009; Sherifali et al., 2010) and possibly Fibrinogen (Jain et al., 2011). We restrict sensitivity analysis to sample exclusion: since the underlying causes, rather than inflammation itself, are the primary clinical target, there are no established treatment effects. Medical research has found negligible impact of statins on HDL cholesterol, which we exclude from the sensitivity analysis (Sheng et al., 2009). Sensitivity analysis for relevant components of the allostatic load and cardiovascular score indexes gives a combined sensitivity analysis for each.

Table 3 presents results for the homogamy correlations when different approaches are employed to adjust for medication. Our findings remain qualitatively unchanged compared to the unadjusted results of Table 2. The statistical significance of the homogamy correlations remains almost identical and the magnitude of the correlations is similar in the base case and the sensitivity analysis. Specifically (for age-adjusted results) statistically significant (at the 5% level) homogamy correlations are observed, irrespective of medication adjustment, for blood pressure, HbA1c and Fibrinogen. The only possible exceptions to the general insensitivity to adjustment for medication are the homogamy correlations for allostatic load and CVD risk scores, which are strengthened by adjustment to a small degree. To conserve space, we do not reproduce the duration-correlation profiles for each combination of biomarker and adjustment method, which are all qualitatively similar to those in Figures 2-6.

[11] For those on statins, we add 1.54 mmol/L and 0.31 mmol/L to observed TC and TG respectively (Sheng et al., 2009). We adjust HbA1c using the type of anti-diabetic medications prescribed as a proxy for severity (Slade, 2012): the recorded level is raised by 1.5% for those taking oral anti-diabetic medications (Sherifali et al., 2010) and is coded as 58 mmol/mol (the NICE-recommended threshold) for those on injected insulin with HbA1c < 58. No change is made if HbA1c > 58mmol/mol with injected medication.

Table 3: Sensitivity of estimated homogamy correlations to adjustment for medication (bandwidth $h = 7.5$ years)

	Unadjusted		Age-adjusted		Number
Health measure	$\hat{R}(0)$	std.err.	$\hat{R}(0)$	std.err.	of couples
Systolic blood pressure (SBP)					
No adjustment	0.233***	0.060	0.166***	0.058	3,395
Fixed increment method	0.281***	0.063	0.176***	0.053	3,395
Sample exclusion method	0.156***	0.051	0.122***	0.047	2,115
Diastolic blood pressure (SBP)					
No adjustment	0.178***	0.049	0.121**	0.049	3,395
Fixed increment method	0.205***	0.047	0.132***	0.047	3,395
Sample exclusion method	0.173***	0.045	0.127***	0.047	2,115
Binary hypertension					
No adjustment	0.203***	0.066	0.138**	0.063	3,395
Combined BP/medication criterion	0.276***	0.061	0.116***	0.058	3,395
Total cholesterol					
No adjustment	0.110*	0.065	0.129*	0.066	2,207
Fixed increment method	0.133*	0.070	0.081	0.070	2,207
Sample exclusion method	0.141**	0.062	0.097	0.064	1,537
Triglycerides (TG)					
No adjustment	0.101	0.061	0.094	0.062	2,203
Fixed increment method	0.109	0.067	0.095	0.064	2,203
Sample exclusion method	0.055	0.058	0.051	0.056	1,535
HbA1c					
No adjustment	0.337***	0.075	0.236***	0.077	2,006
Fixed increment method	0.332***	0.070	0.237***	0.082	2,006
Sample exclusion method	0.233***	0.058	0.149***	0.058	1,817
C-Reactive Protein					
No adjustment	0.071	0.066	0.070	0.067	2,000
Sample exclusion method	0.061	0.088	0.081	0.085	1,286
Fibrinogen					
No adjustment	0.186***	0.065	0.143**	0.073	2,189
Sample exclusion method	0.190***	0.061	0.162**	0.074	1,917
Allostatic load					
No adjustment	0.215**	0.088	0.193**	0.084	1,323
Adjusted	0.355***	0.075	0.233***	0.078	1,323
CVD risk score					
No adjustment	0.373***	0.086	0.301**	0.083	1,353
Adjusted	0.433***	0.085	0.331***	0.087	1,353

5.3 Time-varying homogamy

We have a single cross-section observation of the full set of health indicators, so the date of marriage, observed duration and near-fixed date of interview are related by the identity

$m + d = T$. Our analysis assumes a stationary environment in the sense that the profile is invariant to the initiation date m. In a more general setting, write the duration profile for a cohort marrying at date m as $R_m(d)$. Our cross-section estimate of the gradient $R(d) - R(0)$ identifies the following quantity:

$$R_{T-d}(d) - R_T(0) \equiv [R_{T-d}(d) - R_{T-d}(0)] \; - \; [R_T(0) - R_{T-d}(0)] \tag{6}$$

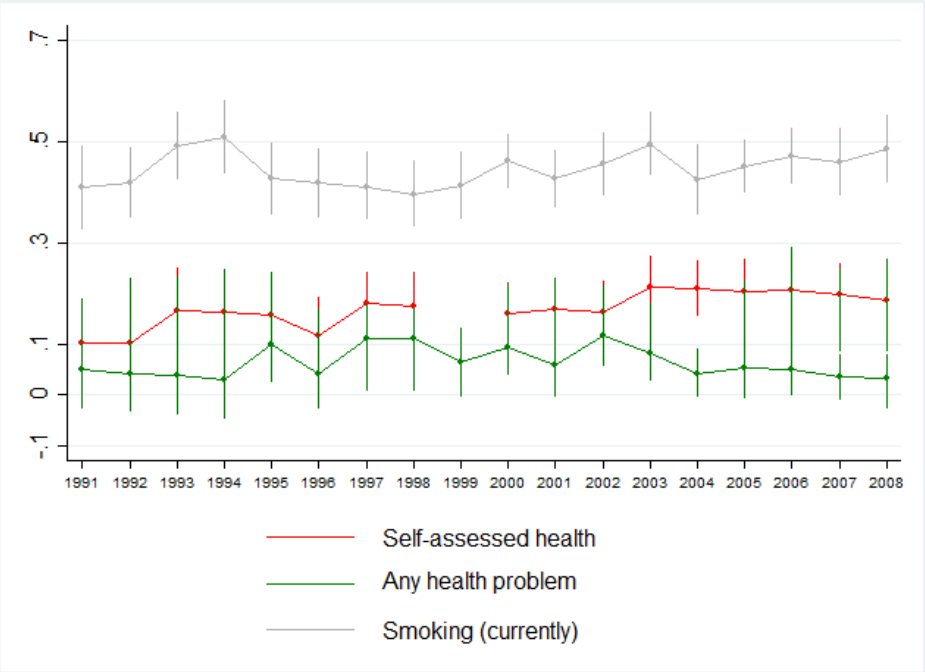
If the aim is to identify the gradient for the cohort marrying at date $m = T - d$, then (6) implies that we underestimate the gradient by an amount $R_T(0) - R_{T-d}(0)$, which is the change in the homogamy correlation over the d years up to the survey date T.

This can only be settled definitively with repeated sweeps of biomarker collection widely separated in time. No such data exist for UK surveys comparable with UKHLS, but we do have observations on some self-reported health measures from the long-run BHPS panel. Figure 12 shows homogamy correlations estimated for couples participating in the BHPS over an 18-year period, using three health measures: self-assessed health (SAH); existence of a long-standing health problem/condition; and current smoking. The last two of these show no trend over time. There is some suggestion of a very slight upward trend for SAH, but essentially only because the first two waves are outliers. This evidence gives grounds for confidence that our main results are not significantly contaminated by trend effects.

5.4 Selection of the sample

The main results for self-reported and nurse-visit health indicators were based on different samples, each using the largest available set of observations. To examine their sensitivity to alternative sample selections, estimates were computed for the nurse visit subsample to give comparability with the biomedical health measures and for the wave 2 sample to give comparability with self-reported diagnosed conditions. The results for homogamy correlations are given in Table 4. We find negligible differences compared to our base-case analysis in Table 2. The same is true of the estimated correlation-duration profiles which are not presented here (details available on request).

Figure 12: British Household Panel Survey: estimated homogamy correlations by wave, 1991-2008 (age-adjusted, Epanechnikov kernel, bandwidth=7.5)



5.5 Age at marriage

In principle, the procedure to adjust for age should also adjust for age at marriage (see appendix) and the duration-specific correlation $R(d)$ could also vary with the partners' ages at marriage. To explore this, we allowed for additional covariates in the age adjustment procedure using the semi-parametric partially-linear regression model (Robinson, 1988), which allows the age effect to be a general nonlinear function and enters age at marriage linearly as another covariate. Of the 50 estimated coefficients for age at marriage, none was significantly different from zero at the nominal 1% level and only two were significant at 5% (details available on request). Since multiple tests are involved, a correction such as the Bonferroni is appropriate. None of the age-at-marriage coefficients was anywhere near conventional statistical significance after correction for multiple testing, so we conclude that our results are robust to age-at-marriage effects (see also Banks et al. (2013) for a similar finding).

Table 4: Estimated homogamy correlations for the nurse-visit and wave 2 subsamples

Health measure	Unadjusted $\hat{R}(0)$	Unadjusted std.err.	Age-adjusted $\hat{R}(0)$	Age-adjusted std.err.	Number of couples
Self-reported indicators in the nurse-visit sample					
Self-assessed generic health measures					
Self-assessed health	0.219***	0.039	0.182***	0.040	4,578
PCS-12	0.218***	0.050	0.161***	0.047	3,762
Functional difficulty§	0.209***	0.049	0.155***	0.050	4,578
Number of functional difficulties	0.181***	0.057	0.141**	0.057	4,578
Self-reported diagnosed conditions†					
Arthritis§	0.201**	0.074	0.109	0.079	3,413
Respiratory§	-0.009	0.044	-0.007	0.044	3,413
High blood pressure§	0.098*	0.056	-0.004	0.056	3,413
Endocrine§	0.129	0.077	0.115	0.081	3,413
Any cardiovascular condition§	0.168***	0.055	0.059	0.058	3,413
Number of conditions	0.212***	0.058	0.109**	0.051	3,413
All indicators in the non-BHPS wave 2 sample					
Self-assessed generic health measures					
Self-assessed health	0.271***	0.023	0.248***	0.021	11,253
PCS-12	0.189***	0.028	0.154***	0.028	7,022
Functional difficulty§	0.208***	0.033	0.174***	0.033	9,457
Number of functional difficulties	0.168***	0.037	0.134***	0.038	9,457
Nurse-measured indicators					
BMI	0.380***	0.042	0.356***	0.044	3,560
Waist circumference	0.403***	0.042	0.356***	0.044	3,676
Systolic BP	0.241***	0.067	0.184**	0.060	2,809
Diastolic BP	0.164***	0.055	0.106**	0.053	2,809
Hypertension§	0.223***	0.073	0.164**	0.065	2,809
Resting HR	0.170***	0.050	0.166***	0.052	2,809
Blood-based biomarkers					
Total cholesterol	0.101	0.065	0.111	0.067	1,805
HDL cholesterol	0.229***	0.060	0.235***	0.066	1,795
Triglycerides	0.121	0.075	0.112	0.070	1,800
HbA1c	0.337***	0.080	0.237***	0.090	1,634
C-Reactive Protein	0.063	0.075	0.067	0.069	1,631
Fibrinogen	0.172**	0.071	0.131*	0.073	1,791
Summary measures					
Allostatic load	0.204**	0.093	0.181**	0.092	1,069
CVD risk score	0.327***	0.108	0.250***	0.092	1,096

§ Binary indicator. Bootstrapped standard errors (500 replications) *, **, *** = significant at 10%, 5% , 1%.

6 Conclusions

This paper makes several new contributions to the small research literature on the concordance of health status within marital/cohabiting partnerships. Unlike most studies in this field, we are able to use a large set of health indicators that encompasses the subjective health assessments and self-reported diagnosed conditions that underpin most of the literature, but also more objective nurse-administered meaures and blood-based biomarkers. We develop and apply flexible nonparametric methods to data from the UKHLS/BHPS household survey.

The analysis allows us to explore the relative importance of two distinct processes: initial non-causal concordance arising from assortative mating; and subsequent causal concordance generated by shared influences operating throughout marriage. We also make a new theoretical contribution to the interpretation of empirical evidence on spousal concordance. It is often assumed that the existence of shared 'lifestyle' factors causally influencing the health outcomes of marital partners must necessarily imply that health concordance increases with the duration – a 'dose-response relationship for marriage'. But we have used a simple life-course model of cumulative health exposures to show that this is not the case. We show that, if concordance turns out to be unrelated to marriage duration, the implication is not that shared within-marriage lifestyle factors are absent, but that they are equally important as initial partner selection in a precise sense.

The inter-spousal correlation observed early in marriage (the *homogamy correlation*) is a measure of the contribution of assortative mating to spousal concordance. We find important differences between health indicators. Statistically significant homogamy correlations (of around 0.2) are found for all self-reported subjective assessments of health and functional difficulty. For the indicators of diagnosed conditions (which are self-reported but in principle objective), there is very little evidence of assortative mating, presumably because most of those conditions have a long development period and are rare in early-mid life when most relationships begin. Among objective health measures, the largest and most significant homogamy correlations (around 0.36) are observed for adiposity measures. Also significant, but smaller in magnitude (around 0.2) are the correlations for measures of blood pressure, resting heart rate, HDL cholesterol and blood sugar, which all tend to accompany adiposity. We also find similar similar homogamy correlations for composite systemic measures

of health, designed to reflect allostatic load or (particularly) cardiovascular risk. There is significant evidence of a small homogamy correlation (around 0.15) in one of our biomarkers for inflammatory response (fibrinogen), but not the other (C-reactive protein).

We have found a remarkably consistent result on the relationship between the spousal correlation and union duration. For none of our twenty-four health indicators is there any evidence of a change in the correlation with increasing duration. It is tempting to interpret this to mean that lifestyle factors shared within marriage are unimportant as influences on health, but this would be a misinterpretation. Using a lifecourse framework of risk accumulation to guide interpretation, it indicates instead that such factors are approximately comparable to assortative mating as a source of the spousal concordance that we see empirically. This is an important point to consider when reading the research literature on self-assessed health, BMI and blood pressure (Monden, 2007; Di Castelnuovo et al., 2009; Wilson, 2012).

A further contribution of our study is the finding that our results are robust with respect to a number of potential sources of bias. Particularly worrying for research in this area is the impact of medication which can mask the underlying health state from observable indicators, and mortality which means that observed intact marriages are not representative of marriages in general. By exploring different approaches to observations affected by medication and using a new simulation approach to indicate the possible range of mortality bias, we have found grounds to claim that our key results are robust.

There are important policy implications. The co-existence of health homogamy and causal concordance due to the shared exposure suggests that health inequalities are larger between households than between individuals, highlighting the importance of targeting potential health policies at couples rather than individuals. This seems particularly so for adiposity and cardiovascular and diabetes risk, where spousal concordance is especially strong. From a long-term population genetic perspective, the presence of health homogamy for dimensions of health with a strong genetic component (such as adiposity, cholesterol and blood sugar levels) may indicate genetic predispositions for the next generation (Silventoinen et al., 2003) which would contribute to increasing health inequalities in the long run.

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Appendix: The accumulation model of correlated health risks

The exposure-accumulation model is:

$$H_j(T) = \lambda(T-b_j)H_j(b_j) + \sum_{t=b_j+1}^{m} \lambda(T-t)z_j(t) + \sum_{t=m+1}^{m+d} \lambda(T-t)\ [x_j(t)+s(t)] \qquad j=h,w$$

Assume that all couples are observed at the same date T, and consider the first and second moments of the observed health indicators conditional on the partners' ages at marriage, ω_h and ω_w, and the elapsed duration of marriage, d (this implies conditioning on age at interview, a_h, a_w and birth dates, b_h, b_w, since $a_j \equiv \omega_j + d \equiv T - b_j$). To allow for changes in the environment over time, we assume the conditional means of the processes $z_j(t), x_j(t), s(t)$ are functions, $\mu_z(t), \mu_x(t), \mu_s(t)$ of time. We also assume that person j's pre-marital health exposures are independent of the age of the person (s)he marries, so that the mean function of H_j can be written as a function $M_j(a_j, \omega_j)$:

$$M_j(a_j,\omega_j) = \lambda(a_j)E\left[H_j(b_j)|a_j,\omega_j\right] + \sum_{t=b_j+1}^{m} \lambda(T-t)\mu_z(t) + \sum_{t=m+1}^{m+d} \lambda(T-t)\ [\mu_x(t)+\mu_s(t)]$$

Write $t = b_j + \tau, T - t = a_j - \tau$ in the first summation and $t = m + \tau = T - a_j + \omega_j + \tau, T - t = \omega_j + \tau$ in the second:

$$\begin{aligned} M_j(a_j,\omega_j) \;\; = \;\; & \lambda(a_j)E\left[H_j(b_j)|a_j,\omega_j\right] + \sum_{\tau=1}^{\omega_j} \lambda(a_j-\tau)\mu_z(T-(a_j+\tau)) \\ & + \sum_{\tau=1}^{a_j-\omega_j} \lambda(\omega_j+\tau)\ [\mu_x(T-a_j+\omega_j+\tau)+\mu_s(T-a_j+\omega_j+\tau)] \end{aligned}$$

This is a nonlinear function of a_j and ω_j, and there is far more variation in a_j, so adjustment for age is more important than for age at marriage. Note that $M_j(a_j, \omega_j)$ can be expressed equivalently as a function $M_j^*(d, \omega_j) \equiv M_j(\omega_j + d, \omega_j)$.

For second moments, begin with the case of a couple observed at the start of marriage. For them, $d = 0$ and $a_j = \omega_j$ and their observed health states in mean deviation form are:

$$\begin{aligned} H_j(T) - M^*(0,\omega_j) \;\; = \;\; & \lambda(\omega_j)\left\{\ [H_j(b_j) \ - \ E\left[H_j(b_j)|b_j,\omega_j\right]\ \right\} \\ & + \sum_{\tau=1}^{\omega_j} \lambda(\omega_j-\tau)\ \left\{\ z(T-\omega_j+\tau) - \ \mu_z(T-\omega_j+\tau)\ \right\}\ , \quad j=h,w \end{aligned}$$

The homogamy correlation for the cohort of couples marrying at ages ω_h and ω_w is:

$$R_T(0,\omega_h,\omega_w) = corr\left(H_h(m) - M_h^*(d,\omega_h)\ ,\ H_w(m) - M_w^*(d,\omega_w)\ \middle|\ d=0,\omega_h,\omega_w\right)$$

Consider a marriage of elapsed duration $d \geq 1$ and redefine $H_j(b_j), z_j(t), x_j(t)$ and $s(t)$ to be deviations from their means $E\left[H_j(b_j)|a_j,\omega_j\right]$,$\mu_z(t), \mu_x(t)$ and $\mu_s(t)$. Assume that each mean deviation for $z_j(t), x_j(t)$ and $s(t)$ is the sum of a time-invariant persistent component to allow for persistence and an MA(1) process to allow for serial dependence:

$$\begin{aligned} z_j(t) &= u_j + \xi_j(t) + \phi\xi_j(t-1)\ , \qquad j=h,w \\ x_j(t) &= u_j + \varepsilon_j(t) + \theta\varepsilon_j(t-1)\ , \qquad j=h,w \\ s(t) &= v + \eta(t) + \psi\eta(t-1) \end{aligned}$$

where u_h, u_w, v and the white noise processes $\{\xi_h\},\{\xi_w\},\{\varepsilon_h\},\{\varepsilon_w\},\{\eta\}$ are mutually independent. The cumulated post-marital health impacts are:

$$\begin{aligned} \sum_{t=m+1}^{T} \lambda(T-t)\ \left[x_j(t)+s(t)\right] &= \left(\sum_{t=T-d+1}^{T}\lambda(T-t)\right)\left[u_j+v\right] \\ &\quad + \sum_{t=T-d+1}^{T}\lambda(T-t)\left[\varepsilon_j(t)+\theta\varepsilon_j(t-1)+\eta(t)+\psi\eta(t-1)\right] \\ &= \Lambda(d)\left[u_j+v\right] + \lambda(T-d+1)\left[\theta\varepsilon_j(T-d) + \psi\eta(T-d)\right] + \varepsilon_j(T) + \eta(T) \\ &\quad + \sum_{t=T-d+1}^{T-1}\lambda(T-t)\left[(1+\theta)\varepsilon_j(t) + (1+\psi)\eta(t)\right] \end{aligned}$$

where $\Lambda(d) = \sum_{i=0}^{d-1}\lambda(i)$ is the partially-cumulated impact sequence. The conditional variance of observed health for partner j is:

$$\begin{aligned} Var\left(H_j(T)\,|\,\omega_h,\omega_w,d\right) &= \Omega_j(\omega_h,\omega_w,d) + 2\Lambda(d)\sum_{i=0}\omega_j - 1\sigma_u^2 + \Lambda(d)^2\left[\sigma_u^2+\sigma_v^2\right] \\ &\quad + \lambda(T-d+1)^2\left[\theta^2\sigma_\varepsilon^2+\psi^2\sigma_\eta^2\right] + \sigma_\varepsilon^2+\sigma_\eta^2 + \Psi(d)\left[(1+\theta)^2\sigma_\varepsilon^2+(1+\psi)^2\sigma_\eta^2\right] \end{aligned}$$

where $\Omega_j(\omega_h,\omega_w,d) = var\left(\lambda(a_j)H_j(b_j) + \sum_{t=b_j+1}^{m}\lambda(T-t)z_j(t)\ \middle|\ \omega_h,\omega_w,d\right)$ is the variance of cumulated pre-marital exposures and $\Psi(d) = \sum_{i=1}^{d-1}\lambda(i)^2$. The covariance between the partners' observed health states is:

$$\begin{aligned} Cov\left(\left[H_h(T)-M_h\right],\ \left[H_w(T)-M_w\right]\,|\,\omega_h,\omega_w,d\right) &= \Omega_{hw}(\omega_h,\omega_w,d) + \Lambda(d)^2\sigma_v^2 \\ &\quad + \lambda(T-d+1)^2\psi^2\sigma_\eta^2 + \Psi(d)(1+\psi)^2\sigma_\eta^2 \end{aligned}$$

where $\Omega_{hw}(\omega_h, \omega_w, d)$ is the conditional covariance between the observed components of health which originate in the pre-marital period.

We follow the spirit of the lifecourse approach and assume that $\Lambda(d)$ and $\Psi(d)$ are unbounded as d increases, so that the effect of earlier exposures build up over time.[12] We make the following reasonable assumptions:

(1) The cumulated sequence of squared autoregressive coefficients is of lower order in d than the squared cumulated sequence: $\lim_{d\to\infty} \{ \Psi(d)/[\Lambda(d)^2] \} = 0$

(2) The squared autoregressive coefficient $\lambda(T-d+1)^2$ is of lower order in d than $\Lambda(d)$ and $\Psi(d)$.

(3) If the conditional pre-marriage second moments $\Omega_h, \Omega_w, \Omega_{hw}$ vary at all with subsequent marriage duration, they do so in a way that keeps them of lower order in d than the cumulated sequences $\Lambda(d)$ and $\Psi(d)$, for every combination of ω_h, ω_w.

Under these assumptions, the between-partner health correlation conditional on duration (and implicitly any combination of ages at marriage, ω_h, ω_w) displays the following asymptotic behaviour:

$$R(d) \underset{d\to\infty}{\longrightarrow} \begin{cases} \frac{\sigma_v^2}{\sigma_u^2+\sigma_v^2} & \text{if } \sigma_u^2, \sigma_v^2 \neq 0 \\ \\ \frac{(1+\psi)^2\sigma_\eta^2}{(1+\theta)^2\sigma_\varepsilon^2+(1+\psi)^2\sigma_\eta^2} & \text{if } \sigma_u^2 = \sigma_v^2 = 0 \end{cases}$$

Define the dominant variances of the partners' health indicators, V_s, V_x as:

$$(V_s, V_x) = \begin{cases} \left(\sigma_{u_s}^2, \sigma_{u_x}^2\right) & \text{if } \sigma_u^2, \sigma_v^2 \neq 0 \\ \\ \left((1+\theta_s)^2\sigma_{v_s}^2, (1+\theta_x)^2\sigma_{v_x}^2\right) & \text{if } \sigma_u^2 = \sigma_v^2 = 0 \end{cases}$$

Then:

$$R(d) \underset{d\to\infty}{\longrightarrow} \frac{V_s}{V_x + V_s}$$

[12] An example is the random walk model where exposures have a simple additive effect and $\lambda(T-t) = 1$ for all t, implying $\Lambda(d) = d; \Psi(d) = d-1$.