



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



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## ABSTRACT

We use self-reported health measures, nurse-administered measurements and blood-based biomarkers to examine the concordance between health states of partners in marital/cohabiting relationships in the UK. A model of cumulative health exposures is used to interpret the empirical pattern of between-partner health correlation in relation to elapsed relationship duration, allowing us to distinguish non-causal correlation due to assortative mating from potentially causal effects of shared lifestyle and environmental factors. We find important differences between the results for different health indicators, with strongest homogamy correlations observed for adiposity, followed by blood pressure, heart rate, inflammatory markers and cholesterol, and also self-assessed general health and functional difficulties. We find no evidence of a “dose–response relationship” for marriage duration, and show that this suggests – 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.

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## 1. Introduction

Research on the health of couples is sparse relative to research on individuals, twins and siblings (Meyler et al., 2007). Existing evidence includes cross-section analysis of the spousal associations of incidence for a range of diagnosed diseases (Hippisley-Cox et al., 2002; Banks et al., 2013) 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), obesity (The NS and Gordon-Larsen, 2009; Wilson, 2012) and smoking behavior (Banks et al., 2013). Many studies are based exclusively on self-reported health indicators or focus on specific health conditions or indicators; few studies are able to separate initial selection effects from subsequent duration effects (Di Castelnuovo et al., 2009).

To understand the health of couples, it is important to distinguish homogamy (a tendency for people to choose partners similar to themselves) and causal concordance (correlation of health states caused by lifestyle and environmental influences shared within the marriage).<sup>1</sup> The economic theory of the marriage market (Becker, 1973, 1974) predicts that complementarity of partners' traits in the marriage production function leads to homogamy in the form of positive assortative mating. Empirical evidence suggests that matching processes are multidimensional and not driven by a single individual characteristic (Chiappori et al., 2012). Although health (more particularly, observable health dimensions) may be a matching criterion itself, it is more likely that health selection arises from indirect selections through other characteristics, such as behaviours, age, education and socio-economic position (Chiappori et al., 2012; Clark and Etilé, 2006).

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<sup>1</sup> 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.

Homogamy and shared lifestyle/environmental influences are not necessarily unrelated. For example, if initial attraction rests partly on a shared love of overeating, then that preference may contribute to a shared diet that damages health. In this example, homogamy both generates a correlation in the pre-marital health states of the couple and contributes to establishing the subsequent shared lifestyle and home environment, so homogamy and marital lifestyle need not be statistically or causally independent. Nevertheless, it is the actual diet that damages health, not the love of food *per se*. In this paper we aim to distinguish between homogamy in the specific sense of correlation between health states of partners at the start of their union and shared lifestyle/environment in the sense of common factors that influence health through marriage, however those factors arise.

In the wider context of the debate on contagion *versus* homophily in health behaviors and obesity (Christakis and Fowler, 2007), experimental research has achieved some results (Centola, 2011); however, marriage is not amenable to randomized experimental control and the importance of homogamy as a factor in couples' health outcomes remains uncertain.

There are two main reasons for an interest in the association of morbidities of marital partners. One is that this analysis may tell us something about the causal processes generating health outcomes in adult life. If homogamy is found not to be a significant source of spousal health concordance, this focuses attention on a wide range of possible theoretical mechanisms including household production, peer effects, marriage market effects and various kinds of contagion (see Section 2). On the other hand, if shared adult environment and lifestyle effects are unimportant, the well-established positive association between marriage and health (Rendall et al., 2011) may not be causal, and the argument for environmental exposures in the foetal and infancy stages as the dominant influences on the risk of disease much later in life (Almond and Currie, 2011; Barker, 1991) is strengthened.

The second motivation relates to public policy and the capacity of couples to absorb adverse shocks. Even if health selection occurs at the time of partnership formation, a causal effect of the shared environment is necessary for health concordance to persist or increase through time. This matters for policy because persistent concordance may result in wider health inequalities across couples and any tendency for disability and morbidity to become more concentrated within couples also affects the social cost of disease. For example, the market value of informal care supplied to disabled people in the UK in 2015 is estimated at £132bn, comparable to the total cost of the National Health Service (Buckner and Yeandle, 2015), with much of that cost met by the domestic partners of disabled people (Pickard et al., 2007). This is a system of informal insurance through the pooling of risk within couples but, if disability affects both partners simultaneously, their capacity to provide care for each other may be impaired – reducing the effectiveness of pooling and self-insurance, increasing dependency on external care services, and raising the cost of social care. Separating homogamy from causal concordance may be also relevant for public health prevention programs. Although homogamous health selection is largely immune to policy, evidence on causal effects of shared environment and lifestyle may provide a basis for screening programmes and other interventions that exploit information on the health of one partner to identify elevated risks for the other partner (Di Castelnuovo et al., 2009).

A key difficulty is the absence of definitive data. The ideal would be a prospective study that samples individuals early in life, tracking them through marriages with other sample members, observing health outcomes in later life. No such study exists on a representative basis. Consequently, 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 have been used (Booker and Pudney, 2013), but there are doubts about recall accuracy and the limited health indicators available.

A further difficulty is the multi-dimensional nature of the concept of health and the difficulty of measuring health in general-population surveys. We exploit the availability of an unusually wide range of health indicators in the UK *Understanding Society* household panel.<sup>2</sup> They are 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 use a statistical model, which captures formally the theory of cumulative exposures that is highly influential in lifecourse epidemiology (Riley, 1989; Power et al., 2013) to show that the variation of the intra-couple health correlation with elapsed marriage duration is informative about the relative importance of homogamy and shared environment and 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 exposures are unimportant. We find empirically that homogamy is an important source of concordance in certain dimensions of health, particularly adiposity and also cardiovascular health and inflammation which are known to be related to adiposity. We also find that the correlation between partners' health states is essentially unrelated to the elapsed duration of marriage, which, under reasonable assumptions, implies – perhaps counter-intuitively – that shared 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, age at marriage effects, time variation in homogamy, the effect of medication, and other features of our research design.

A second contribution is to extend the literature on health concordance by using a wide range of health indicators. Unlike many 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 complementary subjective and objective health measures. Subjective indicators (such as self-assessed general health or functional disability) 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. Objective biomarkers are free of this type of cross-contamination but 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 causal health concordance in long-established partnerships. The most obvious rests on household production theory (Becker, 1965), which emphasizes 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. Second, despite advances in public

<sup>2</sup> Our analysis excludes Northern Ireland, where the full range of health measures was not collected.

health, biological contagion remains a possible source of concordance, for example in older couples where both partners may have relatively weak immune systems. Third, research on the human biome suggests that the microbial flora in the human gut and elsewhere on the body may have an important role in some disease processes (Claesson et al., 2012). If there is a mechanism causing people in an intimate relationship to share a microbial population, this may be an important pathway for the co-occurrence of disease. A fourth possible mechanism involves social engagement, which has been linked empirically to stress patterns, with observable expression in blood levels of lipids and cortisol (Grant et al., 2009). Marital partners have shared patterns of social engagement and exposure to social stress, implying correlated health outcomes. A fifth possibility is social contagion within marriage stemming from each partner's economic and emotional incentives to have a healthy partner, prompting each to influence the other's health-related behaviour (Cutler and Glaeser, 2010; De Giorgi et al., 2013). A sixth potential mechanism relates to the marriage market. Body shape and possibly other physical signs of health have an influence on individuals' success in partnership formation (Oreffice and Quintana-Domeque, 2010) so, in relationships believed by the partners to be secure, there may be less incentive to maintain a healthy lifestyle. The evidence of a tendency for body mass to increase after marriage (The NS and Gordon-Larsen, 2009) is consistent with this.

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. For example, 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 behavioral, 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 at time  $T = m + d$ , where  $d$  is the elapsed duration of the marriage. When observed, 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 homogamy. From marriage to the survey date, they continue to experience health exposures which are partly shared and partly personal. The personal components are  $\{x_h(m + 1) \dots x_h(m + d)\}$  and  $\{x_w(m + 1) \dots x_w(m + d)\}$  and the shared exposure is  $s(m + 1) \dots s(m + d)$ . These processes are mutually independent by definitional: the shared component is defined to cover all sources of correlation, including the common physical and social environment and standard of living and specific shared behaviors like diet and exercise. We do not assume that the causal factors at work pre-marriage ( $z_h(t)$ ,  $z_w(t)$ ) are independent of exposures post-marriage ( $x_h(t)$ ,  $x_w(t)$ ,  $s(t)$ ) so, for example, the shared lifestyle within marriage can be inherited to some extent from behaviours established before marriage.

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:

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

where  $A_j$  is the component of health arising from pre-marriage exposures<sup>3</sup>:

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

In the appendix, we show that the mean health state of each partner  $j$  when observed at time  $T$  is a potentially non-linear 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  $corr(H_h(T), H_w(T)|d)$ . To allow for persistence and serial correlation in exposures, we assume that each of the processes  $\{z_h(t), z_w(t), x_h(t), x_w(t), s(t)\}$  may involve personal and shared random effects and moving average components:

$$z_j(t) = \gamma u_j + \kappa v + \xi_j(t) + \phi \xi_j(t-1), \quad j = h, w \quad (3)$$

$$x_j(t) = u_j + \varepsilon_j(t) + \theta \varepsilon_j(t-1), \quad j = h, w \quad (4)$$

$$s(t) = v + \eta(t) + \psi \eta(t-1) \quad (5)$$

where  $u_h, u_w, v$  and the white noise processes  $\{\xi_h\}, \{\xi_w\}, \{\varepsilon_h\}, \{\varepsilon_w\}, \{\eta\}$  are mutually independent. The persistent shared effect,  $v$  is included in the process  $z_j(t)$  for pre-marital exposures to allow for preferences or personal capitals that may be involved in partner matching as well as later lifestyle outcomes. The parameters  $\gamma$  and  $\kappa$  are used to allow the persistent factors to have different impacts in early than in later life, for instance because parents limit their children's freedom to exercise choice (in which case  $0 \leq \gamma, \kappa < 1$ ).

In the appendix, we use these assumptions to demonstrate that  $R(0)$  gives a natural measure of health-related homogamy and that, for increasing duration,  $R(d)$  generally converges to a limiting value:

$$R(\infty) = \frac{\sigma_v^2}{\sigma_u^2 + \sigma_v^2} \quad (6)$$

where  $\sigma_u^2$  is the variance of the persistent personal effects  $u_h$  and  $u_w$  and  $\sigma_v^2$  is the variance of the shared effect  $v$ . However, if persistent effects are absent ( $\sigma_u^2 = \sigma_v^2 = 0$ ), the limit is instead  $R(\infty) = \sigma_\eta^2 / [\sigma_\varepsilon^2 + \sigma_\eta^2]$  provided the shared and personal non-persistent shocks have the same autocorrelation ( $\theta = \psi$ ). In each case, the limit  $R(\infty)$  is the proportion of the variance of the most persistent part of health shocks (either  $u_j + v$  or  $\varepsilon_j(t) + \eta(t)$ ) which is attributable to the shared component ( $v$  or  $\eta(t)$ , respectively). These variance proportions can be interpreted as long-run measures of the importance of shared marital exposures relative to homogamy as a source of health concordance.  $R(d)$  is increasing with duration (in the large) if  $R(\infty) > R(0)$ , which occurs 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 our theoretical result suggests 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.

Of course, the limiting results do not necessarily apply over a finite range and the form for  $R(d)$  derived in the appendix is complicated and not necessarily monotonic, its shape depending on a

<sup>3</sup> Note that, under reasonable assumptions, the functions  $\lambda(\cdot)$  governing the impact of exposures can differ with gender without affecting our theoretical results (see appendix). Note also that the effects any previous marriages are subsumed within the term  $A_j$ .

number of parameters. However, illustrative simulations for four plausible structures with very different time series properties are reported in the appendix (Fig. A1), all displaying smooth monotonic convergence towards  $R(\infty)$ , and thus supporting the way we interpret correlation profiles. Moreover, if (as we find empirically) correlation profiles are essentially flat over a wide range, the implication is that – unless there is some exotic property of health shock sequences that would cause a substantial change in the correlation profile only at very long durations –  $R(d)$  must coincide with  $R(\infty)$ . It is hard to see a plausible source of such strange behaviour, so it seems reasonable to interpret the absence of a gradient in the correlation profile as evidence that homogamy and shared exposures are equally important in the specific sense that  $R(0) = \text{var}(s)/(\text{var}(x) + \text{var}(s))$ .

We investigate the correlation profile  $R(d)$  using a 4-stage local smoothing method:

- (1) Construct age-adjusted versions of the biomarker or other health indicator 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.<sup>4,5</sup>
- (2) For each duration  $d$  in a grid of values  $d_1 \dots d_K$ , calculate the local correlation:

$$\hat{R}(d) = \frac{\sum_i \omega_i(d) \hat{u}_{hi} \hat{u}_{wi} - (\sum_i \omega_i(d) \hat{u}_{hi})(\sum_i \omega_i(d) \hat{u}_{wi})}{\left[ (\sum_i \omega_i(d) \hat{u}_{hi}^2 - (\sum_i \omega_i(d) \hat{u}_{hi})^2)(\sum_i \omega_i(d) \hat{u}_{wi}^2 - (\sum_i \omega_i(d) \hat{u}_{wi})^2) \right]^{1/2}} \quad (7)$$

where  $h$  is a specified bandwidth.  $\omega_i(d)$  is the Epanechnikov kernel modified to allow for the boundary at  $d=0$  using a linear boundary adjustment (Jones and Foster, 1993).<sup>6</sup>

- (3) Calculate a summary,  $\hat{\beta}$ , of the overall slope of the correlation profile by regressing  $\hat{R}(d)$  on  $d$  over the duration grid.
- (4) Repeat the calculations for each of a set of  $N$  bootstrap resamples to compute confidence intervals for  $\hat{R}(d)$  at each grid point  $d$  and for  $\hat{\beta}$  overall.

We plot the estimates  $\hat{R}(d)$  together with pointwise bootstrap confidence intervals, but it is not possible to draw inferences about global behaviour of the profile directly from these, since estimates  $\hat{R}(d)$  at different points  $d$  are not statistically independent. Steps (3)–(4) give a test of the hypothesis of overall flatness for the whole duration range.

### 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, with a design that involves overlapping 2-year waves. Individuals have been interviewed annually since the initial wave in 2009–2010. At wave 2, participants in the BHPS were absorbed into the UKHLS.

<sup>4</sup> Some of our health indicators are ordinal rather than continuous and their age-adjusted forms could be constructed instead as generalised residuals from (ordinal) probit models. However, this introduces additional assumptions of linearity and normality unless complex semi-parametric models are used. Experiments with linear probit models suggested that results are very similar to those presented here, so we did not pursue this option further.

<sup>5</sup> As we show in the appendix, there is a case for including the couple's ages at marriage in the regression model used at step (1). However, our results are robust to the inclusion of age-at-marriage variables, which proved insignificant (Supplement Section S3).

<sup>6</sup> In practice, boundary adjustment makes very little difference to our estimates.

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 (McFall et al., 2014).<sup>7</sup> We pool data from the wave 2 non-BHPS and the wave 3 BHPS samples, giving a potential sample of 35,937 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 cohabiting or marital relationship at the time of interview (wave 2 and 3 for the non-BHPS and BHPS samples). The analysis sample for biomedical measures (Sections 3.3 and 3.4) contains a maximum of 4443 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 12,881 couples.

Information on the month and year the current relationship started was collected separately from each partner. For married couples, we use the date of marriage or start of cohabitation, if earlier. Start dates are derived from the UKHLS and BHPS partnership history updated by subsequent panel data information. The duration of each union is calculated by subtracting the start date from the date of the interview or nurse visit at which the relevant health indicator was measured; 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 variable is the average across partners, or is based on one partner if relevant data are missing for the other; couples with duration differences of more than one year were excluded. In 34% (larger full sample) or 27% (smaller biomarker sample) of couples, one or both partners had a previous union that ended in divorce or separation. Excluding those couples from the analysis does not change the results reported below in any substantive way.

We use health measures ranging from subjective assessments to objective anthropometric measurements and blood-based biomarkers; their empirical distributions are summarised in Table 1. Any health indicator is potentially distorted as a measure of underlying health by the effect of medication, particularly the cholesterol and blood sugar biomarkers which are used in practical diagnosis. The results presented in Sections 4 and 5 are based on analysis of health measures in directly observed form but, in Section 6.3, 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 categorizes respondents on a five-point scale, ranging from 1 = excellent to 5 = poor health. SF-12 is a 12-item generic measure of health-related quality of life. Our main focus here is on physical health, so we use the PCS-12 sub-measure, normalized to vary between 0 and 100 with mean 50 and standard deviation 10. Higher scores indicate better physical functioning. We also examine two self-reported functional disability measures. Respondents were asked about any long-standing physical or mental impairment that they might have, and the consequent functional difficulties.<sup>8</sup> We use the

<sup>7</sup> 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.

<sup>8</sup> From a list covering: mobility; lifting; carrying or moving objects; manual dexterity; continence; hearing; sight; communication; memory or ability to con-



**Table 1**  
Summary statistics for health measures and demographic characteristics.

	# Couples	Husband		Wife	
		Mean	Std. dev.	Mean	Std. dev.
Self-assessed health	12,881	2.614	1.108	2.575	1.097
Functional difficulty	10,975	0.215	0.411	0.219	0.414
# Functional difficulties	10,975	0.549	1.356	0.546	1.329
PCS-12	8400	49.97	10.68	50.02	10.93
Diagnosed arthritis <sup>a</sup>	8156	0.143	0.350	0.187	0.390
Diagnosed hypertension <sup>a</sup>	8156	0.237	0.425	0.209	0.406
Diagnosed endocrine <sup>a</sup>	8156	0.105	0.316	0.131	0.369
Diagnosed cardiovascular <sup>a</sup>	8156	0.288	0.453	0.230	0.421
BMI (kg/m <sup>2</sup> )	4308	28.527	4.697	28.131	5.868
Waist circumference (cm)	4443	101.128	12.633	89.953	13.703
Systolic blood pressure (mmHg)	3218	131.18	14.76	125.094	16.18
Diastolic blood pressure (mmHg)	3218	74.79	10.81	73.27	9.99
Hypertension <sup>a</sup>	3218	0.261	0.439	0.196	0.397
Heart rate (bpm)	3395	67.032	11.079	69.428	10.096
TC/HDL cholesterol	2196	4.173	1.462	3.460	1.146
Triglycerides (mmol/L)	2203	2.098	1.241	1.585	0.913
HbA1c (mmol/mol)	2006	38.010	8.584	36.600	6.987
C-reactive protein (mg/L)	2000	1.822	1.873	2.037	2.009
Fibrinogen (g/L)	2189	2.736	0.570	2.825	0.590
Allostatic load	1274	2.079	1.549	1.799	1.536
CVD risk score	1287	3.133	1.758	2.960	1.792
Age	12,881	51.192	15.270	48.702	15.157
Ethnicity					
White <sup>a</sup>	12,881	0.811	0.391	0.844	0.363
Mixed/multiple ethnic groups <sup>a</sup>	12,881	0.009	0.094	0.011	0.104
Asian/Asian British <sup>a</sup>	12,881	0.087	0.282	0.095	0.294
Black/African/Caribbean <sup>a</sup>	12,881	0.033	0.178	0.032	0.177
Ethnicity missing <sup>a</sup>	12,881	0.060	0.238	0.018	0.132
Number of marriages/cohabitations (including current)					
1 <sup>a</sup>	12,881	0.752	0.432	0.804	0.397
2 <sup>a</sup>	12,881	0.122	0.327	0.134	0.341
3 <sup>a</sup>	12,881	0.023	0.150	0.022	0.148
4+ <sup>a</sup>	12,881	0.006	0.079	0.005	0.072
Past marriage details missing	12,881	0.096	0.295	0.035	0.184
Marriage duration (yrs)	12,881	22.633	16.043	22.633	16.043

<sup>a</sup> Binary indicator.

report of any functional difficulty as a binary indicator of incidence and also their number as a severity index, ranging from 0 to 11.

### 3.2. Self-reported diagnosed conditions

Self-reported ever-diagnosed specific chronic health conditions are derived from questions at wave 1 or 2 (UKHLS respondents only). 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). High blood pressure is also examined as separate outcome because of its relatively high prevalence rates and for comparison with relevant clinically measured indicators.

### 3.3. Nurse-measured indicators

We use waist circumference (WC) and body mass index (BMI) as measures of central and overall adiposity. Resting heart rate (HR) and blood pressure were measured as described by McFall et al.

(2014). Three readings were taken at one minute intervals. We use the average of the second and third reading, to allow for the possibility that the first might be higher than usual (Johnston et al., 2009). 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); HR is sometimes interpreted as a measure of cardiovascular fitness rather than health. We treat SBP, DBP and HR as continuous variables but also construct a binary hypertension indicator as SBP > 140 or DBP > 90.

### 3.4. Blood-based biomarkers

We study inflammatory, blood glucose and 'fat in the blood' biomarkers. The results of Sections 4 and 5 are based on directly observed biomarkers adjusted for age but not for the effects of any medication that respondents might be taking. We investigate this in Section 6.3 and find no evidence of significant distortions from the use of medication.

C-reactive protein (CRP) and fibrinogen are our biomarkers for inflammation. CRP 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 mor-

**Table 2**  
Estimated homogamy correlations.

Health measure	Unadjusted		Age-adjusted		Number of couples
	$\hat{R}(0)$	Std. err.	$\hat{R}(0)$	Std. err.	
<i>Self-assessed generic health measures</i>					
Self-assessed health	0.294***	0.035	0.266***	0.035	12,881
Functional difficulty <sup>a</sup>	0.236***	0.049	0.202***	0.052	10,975
Number of functional difficulties	0.210***	0.057	0.172***	0.059	10,975
PCS-12	0.245***	0.041	0.208***	0.041	8400
<i>Self-reported diagnosed conditions</i>					
Arthritis <sup>a</sup>	0.201**	0.084	0.065	0.093	8156
High blood pressure <sup>a</sup>	0.041	0.048	-0.029	0.045	8156
Endocrine <sup>a</sup>	0.053	0.055	0.013	0.055	8156
Any cardiovascular condition <sup>a</sup>	0.139***	0.054	0.077 <sup>†</sup>	0.046	8156
<i>Adiposity</i>					
BMI	0.407***	0.071	0.382***	0.075	4308
Waist circumference (WC)	0.438***	0.071	0.398***	0.070	4443
<i>Blood pressure/heart rate measurements</i>					
Systolic blood pressure (SBP)	0.364***	0.125	0.284***	0.100	3218
Diastolic blood pressure (DBP)	0.133	0.100	0.080	0.094	3218
Hypertension <sup>a</sup>	0.322***	0.120	0.235**	0.109	3218
Resting heart rate	0.199***	0.072	0.181**	0.085	3395
<i>Biomarkers</i>					
TC/HDL cholesterol	0.185**	0.083	0.232***	0.081	2196
Triglycerides (TG)	0.195 <sup>†</sup>	0.109	0.180 <sup>†</sup>	0.105	2203
HbA1c	0.317***	0.108	0.071	0.115	2006
C-reactive protein (CRP)	0.249**	0.114	0.237**	0.117	2000
Fibrinogen	0.173**	0.082	0.132**	0.065	2189
<i>Systemic risk scores (from categorical indicators: Supplementary Table S1)</i>					
Allostatic load	0.421***	0.151	0.386***	0.146	1274
CVD risk score	0.534***	0.135	0.402***	0.142	1287

Bandwidth  $h = 7.5$  years.

<sup>a</sup> Binary indicator. Bootstrapped standard errors (500 replications).

<sup>†</sup> Significant at 10% level.

\*\* Significant at 5% level.

\*\*\* Significant at 1% level.

tality (Pearson et al., 2003).<sup>9</sup> Fibrinogen 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 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). We use two markers for fatty substances in the blood: the ratio of total cholesterol (TC) to high-density lipoprotein (HDL) cholesterol; and triglycerides (TG). High levels of the cholesterol ratio and TG 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 an index of multi-system risk; similar indexes are often called allostatic load and regarded as measures of 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 (Supplementary Table S1).<sup>10</sup> Following Walsemann et al. (2016), we also

<sup>9</sup> 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).

<sup>10</sup> 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

construct an index of cardiovascular disease risk by summing four risk factors, WC, BP, HbA1c and CRP, each categorized as (0, 1, 2) on the basis of clinical thresholds (Supplementary Table S1).

#### 4. Results: homogamy measures

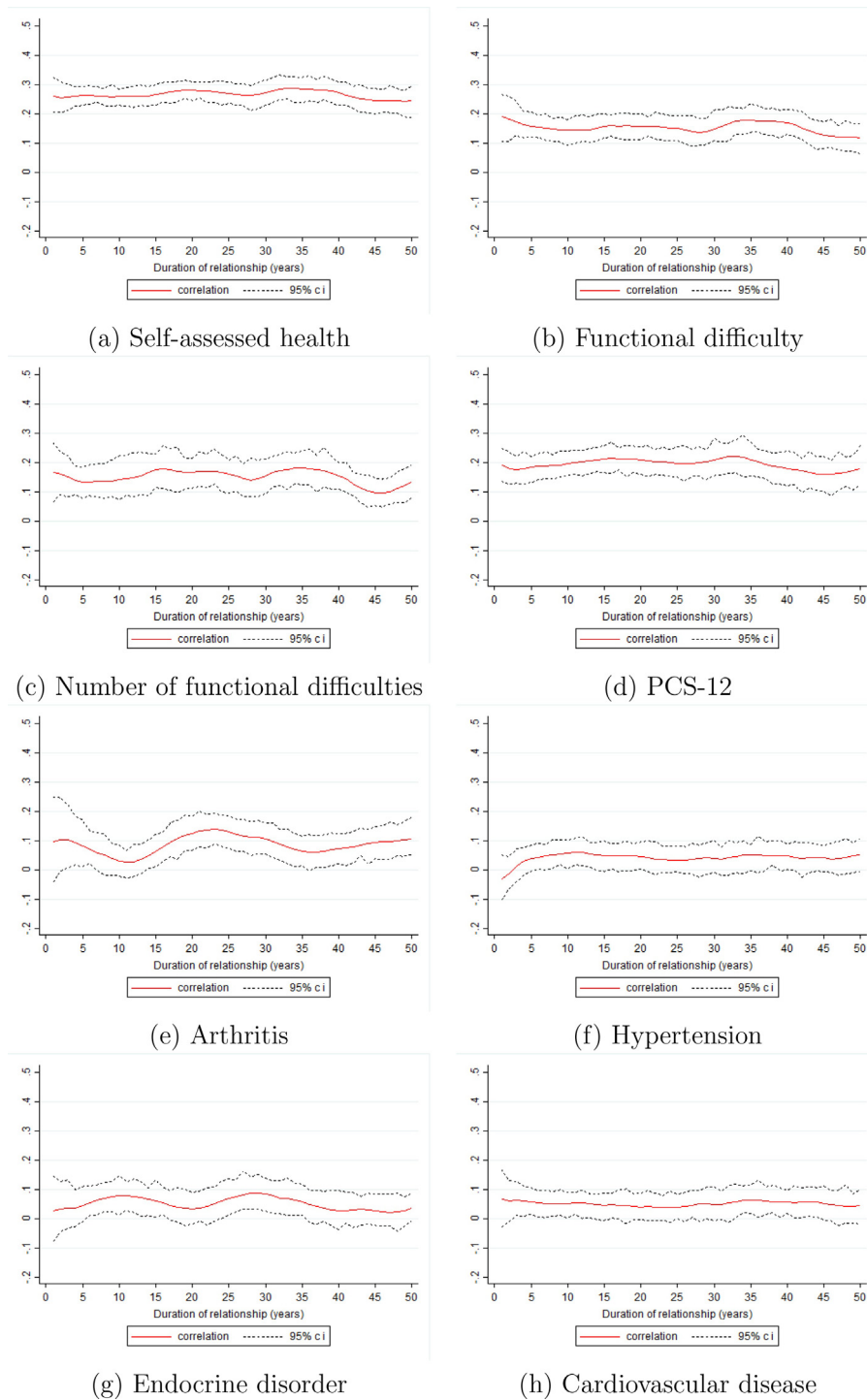
We estimate the homogamy correlation  $R(0)$  using a bandwidth of  $h = 7.5$ , so only marriages starting within 7.5 years of the interview contribute to the estimate.<sup>11</sup> Table 2 shows the result from an analysis of the complete case sample consisting of couples with both partners surviving 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.<sup>12</sup>

We find evidence of health-related homogamy. Generic self-assessed health measures give highly significant early-relationship correlations of 0.21–0.29 without age adjustment and 0.17–0.27 with adjustment. There is weaker evidence of a homogamy effect in self-reported diagnosed conditions. The loss of significance (particularly in the age-adjusted case) is partly due to the lower statistical precision for estimates based on binary indicators, but may also

context. Similar constructions to ours have been used in previous studies (Vie et al., 2014).

<sup>11</sup> We observe a substantial number of couples with duration  $\leq 7.5$  years, comprising 21% of the total sample and 13% of the biomarker sample. Point estimates are little changed when the bandwidth is reduced, e.g. to 5 years, but confidence intervals are somewhat wider.

<sup>12</sup> Our results are robust to the choice of alternative samples (Supplement, Section S2).



**Fig. 1.** Age-adjusted correlation profiles for subjective health indicators and self-reported diagnosed conditions.

arise because such conditions often take a long time to become symptomatic and would thus be undiagnosed at the start of the relationship when the two partners are relatively young.

Homogamy correlations for the adiposity measures are larger (about 0.39 after adjusting for age in the case of both BMI and WC) than for 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 0.36 (unadjusted) or 0.28 (age-adjusted) and

significant at the 1% level in both cases. The contrast between insignificant correlation for self-reported hypertension and significant correlation for nurse-assessed hypertension could arise from three sources: the existence of undiagnosed cases; timing differences (nurses measured current BP while diagnosis refers to any time in the past); and respondents' reporting error and BP measurement noise.

Among the blood-based biomarkers, there are statistically significant homogamy correlations for the cholesterol ratio, triglycerides, inflammatory markers (CRP and fibrinogen), and also for the composite measures of allostatic load and CVD risk. Again, the

**Table 3**  
Summary gradient tests (age adjusted measures).

Health measure	Gradient $\hat{\beta}$	Std. err.	P-value
SAH	0.000	0.001	0.876
Functional difficulty	-0.001	0.001	0.543
# Functional difficulties	-0.001	0.001	0.556
PCS-12	0.000	0.001	0.601
Diagnosed arthritis	0.000	0.001	0.717
Diagnosed hypertension	0.000	0.001	0.722
Diagnosed endocrine	-0.001	0.001	0.582
Diagnosed cardiovascular	0.000	0.000	0.963
BMI	0.000	0.001	0.782
Waist circumference	-0.001	0.001	0.216
Systolic blood pressure	0.000	0.001	0.781
Diastolic blood pressure	0.000	0.001	0.861
Hypertension	-0.001	0.001	0.366
Heart rate	-0.002	0.001	0.122
TC/HDL cholesterol	0.001	0.002	0.449
Triglycerides	0.000	0.001	0.866
HbA1c	-0.003	0.002	0.060
C-reactive protein	0.001	0.002	0.445
Fibrinogen	0.132	0.083	0.113
Allostatic load	-0.003	0.002	0.132
CVD risk score	-0.002	0.002	0.180

correlation profiles are reduced in magnitude following age adjustment but remain significant at the 5% level (10% for triglycerides).

## 5. Results: correlation profiles

Figs. 1–3 show the estimated correlation profiles  $\hat{R}(d)$  for subjective general health assessments, self-reported diagnoses, nurse-administered measures, blood-based biomarkers and composite measures.<sup>13</sup> The estimated correlation profiles are essentially flat, as confirmed by Table 3 which gives the results of the overall gradient test, for which the bootstrap  $P$ -value is above 5% in every case and above 10% in every case except HbA1c, where the  $P$ -value of 6% would be far above conventional significance levels if we were to make a correction (such as the Bonferroni) for multiple testing.

It is interesting that correlation profiles appear to be flat for health measures where there is no significant evidence of homogamy, just as they are for measures displaying significant homogamy. This predominantly affects the self-reported diagnoses, which we think are indicators of questionable quality, because of the relatively weak relationship between the gradual development of the underlying disease state and the process of formal diagnosis and the vagaries of reporting behaviour. Again, the contrast with the small but significantly positive correlation profile for measured hypertension tends to confirm the weakness of self-reported diagnosis as a health measure.

Overall, there is no compelling evidence of an increase in the correlation with duration of marriage, nor is there any significant decline. Under reasonable assumptions, this finding is consistent with the proposition that homogamy and shared environmental or lifestyle factors make approximately equal contributions to the between-partner correlations that we observe. It is also consistent with the conclusions of Booker and Pudney (2013), who used a latent variable analysis of long-term recall data.

## 6. Robustness

We have investigated the robustness of our findings in a number of respects. In our view, the three most important potential complications are: survival bias; the confounding of duration profiles

by time variations in the strength of homogamy; and the impact of medication. Those analyses are reported in Sections 6.1–6.3. Details of robustness in other respects are given in the Supplement. They demonstrate robustness with respect to choice of sample used for analysis (Supplement, Section S2) and to the incorporation of age-at-marriage effects (Section S3). The Supplement also reports a placebo test based on smoking behaviour in the BHPS (Section A5).

### 6.1. 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 interest in 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 risk of joint occurrence 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 relates to the role of shared exposures in the causal processes leading to ill-health. Here, the statistical population of interest is the set of all marriages (starting during some reference period) and the correlation-duration profile should describe the evolution of couples' health in that population. Mortality is a confounding factor because a sample of couples which are intact at a given interview date under-represents those where one or both partners have died prior to the time of interview (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 to underestimation of any positive gradient in the correlation-duration profile. Much of the published literature ignores this issue, despite the potential for bias.

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 2477 for self-reported health measures and 1244 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 the length of time between the start of the partnership and observation of the surviving partner. 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 hot-deck imputation from the sample of intact marriages.
- (ii) *Matched imputation*: Mahalanobis matching to select the intact marriage closest in terms of duration  $d$  and the health  $H$  of the surviving partner.<sup>14</sup>
- (iii) *Concordant imputation*: assign the (age and gender-adjusted) health indicator of the surviving spouse to the deceased partner.

<sup>13</sup> We show only age-adjusted estimates; unadjusted versions are qualitatively very similar and are available from the authors on request.

<sup>14</sup> For our purposes, it is important to achieve a very good match on both health and duration and, for that reason, we match on those two variables alone. It would be possible to include a very large number of other personal and household characteristics in the Mahalanobis criterion, but at the expense of some weakening of the health/duration match.



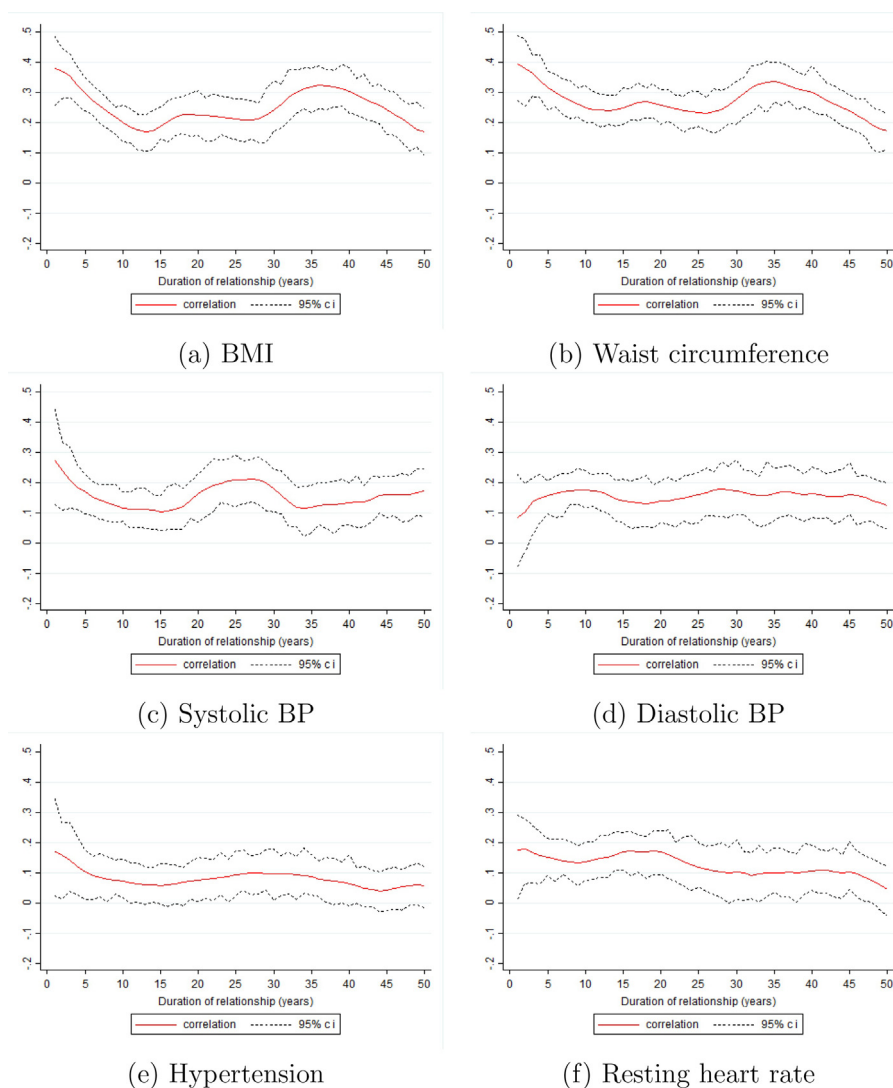


Fig. 2. Age-adjusted correlation profiles for nurse-measured indicators.

Random imputation provides a lower bound under the maintained assumption that any spousal correlation that exists is non-negative. This is not the lowest bound possible – it could be reduced by using a worst case procedure which assigns the missing partner a health state as far from the surviving partners as possible. But negative spousal correlation would conflict with most empirical evidence on marriage and it is reasonable to rule it out *a priori*. Random imputation must give a lower spousal correlation than concordant imputation, since the latter implies perfect correlation. We expect matched imputation to give an intermediate result but there is no strict necessity for that.

Fig. 4 show the results for the indices of allostatic load and CVD risk; other health measures give similar results and are relegated to the Supplement. Matched imputation changes the empirical correlation profile very little. The shaded region lying between the lower bound estimated using random imputation and the upper bound from perfectly concordant imputation gives an idea of the largest possible impact of mortality bias. Concordant imputation raises the profile to a modest degree and imparts a slight positive gradient. The increase in width of the bounds with duration also gives scope for a positive gradient, since any curve lying within the bounds is possible in principle. But if there is indeed a positive gradient masked by mortality bias, it is evidently not large enough to change our 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.<sup>15</sup>

## 6.2. Time-varying homogamy and repeated observation

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:

$$R_{T-d}(d) - R_T(0) \equiv [R_{T-d}(d) - R_{T-d}(0)] - [R_T(0) - R_{T-d}(0)] \quad (8)$$

<sup>15</sup> A complication is that bereavement may have a direct causal effect on the health of the surviving partner, additional to the influence of shared exposures that existed during the marriage. Evidence (Carey et al., 2014) finds transient elevated risks of myocardial infarction, stroke and atrial fibrillation immediately after the death of a partner, with risk levels returning to background levels within a year. 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.

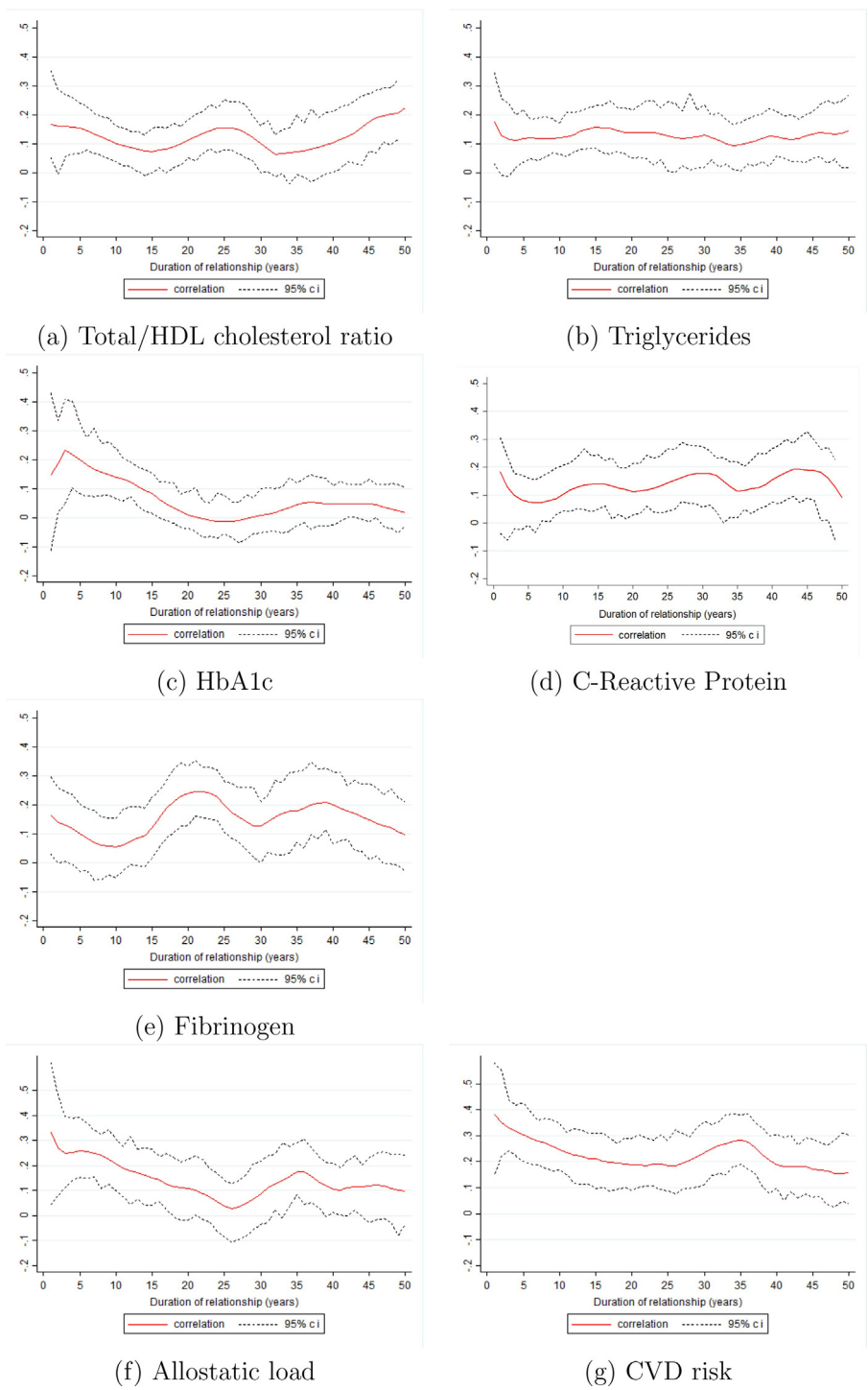


Fig. 3. Age-adjusted correlation profiles for blood-based biomarkers and composite indicators.

If the aim is to identify the gradient for the cohort marrying at date  $m = T - d$ , then (8) 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 two self-reported health measures from the long-run BHPS panel. Fig. 5 shows homogamy correlations estimated for couples participating in the BHPS over the 1991–2008 period, using SAH and existence of a long-standing health problem/condition. There is no signifi-

cant trend over time, which suggests that our main results are not significantly contaminated by homogamy trend effects.

Another check on the limitations imposed by cross-sectional observation of health markers is to rerun the analysis using the same two self-reported markers that were observed repeatedly in the BHPS.<sup>16</sup> Part (a) of Fig. 6 relates to BHPS couples with marriage duration under 7.5 years at wave 1 and follows them through a maximum of 18 waves to 2008. As a robustness check, Fig. 6(b)

<sup>16</sup> We are grateful to an anonymous referee for this suggestion.

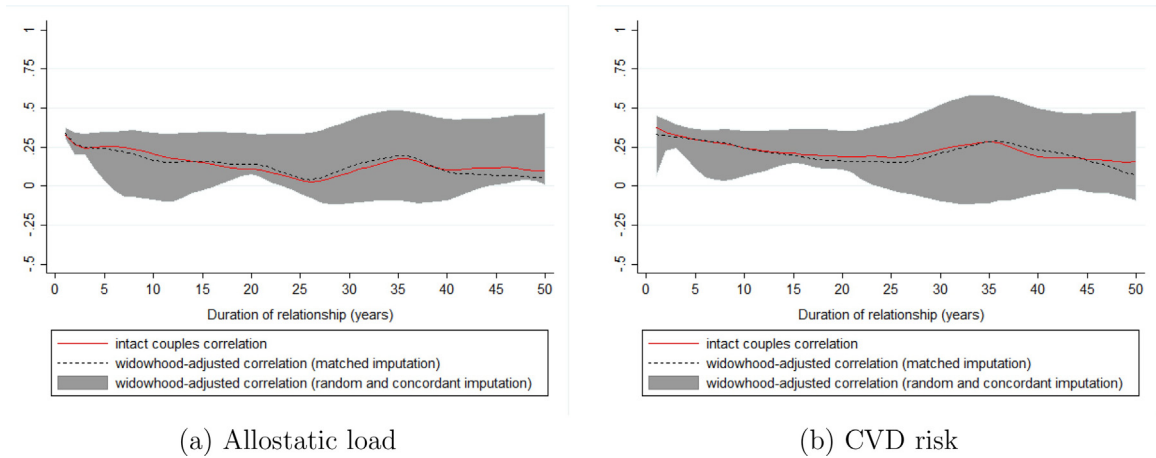


Fig. 4. Correlation profiles adjusted for age and widowhood: composite indices.

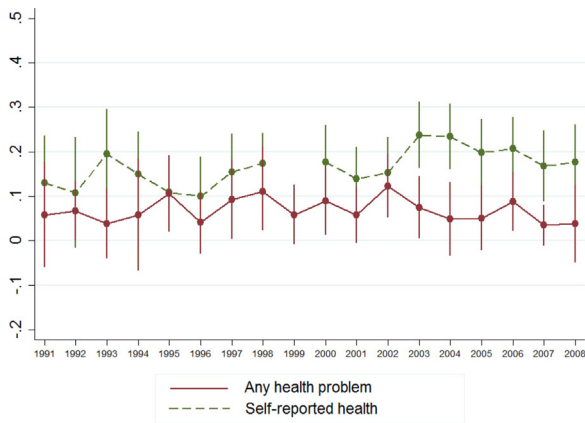


Fig. 5. BHPS: estimated homogamy correlations by wave, 1991–2008 (age-adjusted, Epanechnikov kernel, bandwidth = 7.5).

does the same for couples with an elapsed marriage duration of  $15 \pm 7.5$  years at wave 1. Both plots confirm our core finding of a flat duration profile for SAH and the existence of a long-standing health problem.

### 6.3. Medication

In the main analysis, we made no adjustment for the effect of medication as a confounding factor. The use of medication is

related to the individual's underlying health state, so there may be endogenous 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. 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 the adjustment approaches that are commonly used in the research literature (Cui et al., 2003; Johnston et al., 2009).

In the case of blood pressure, we compare three adjustment procedures: (i) a fixed-increment of 10 mmHg (SBP) and 5 mmHg (DBP); (ii) exclusion from the sample and (iii) a modified binary hypertension measure, defined as  $BP > 140/90$  or currently on anti-hypertensive medication (Cui et al., 2003). We compare two adjustments for the potential impact of statins on the cholesterol ratio and TG: adding fixed increments (0.54 mmol/L and 0.31 mmol/L to the observed TC and TG concentrations (Sheng et al., 2009); or exclusion of those on statins from the sample. For those on anti-diabetic medications, we adjust the analysis of HbA1c by using increments depending on the type of anti-diabetic medication (Slade, 2012) or by sample exclusion.

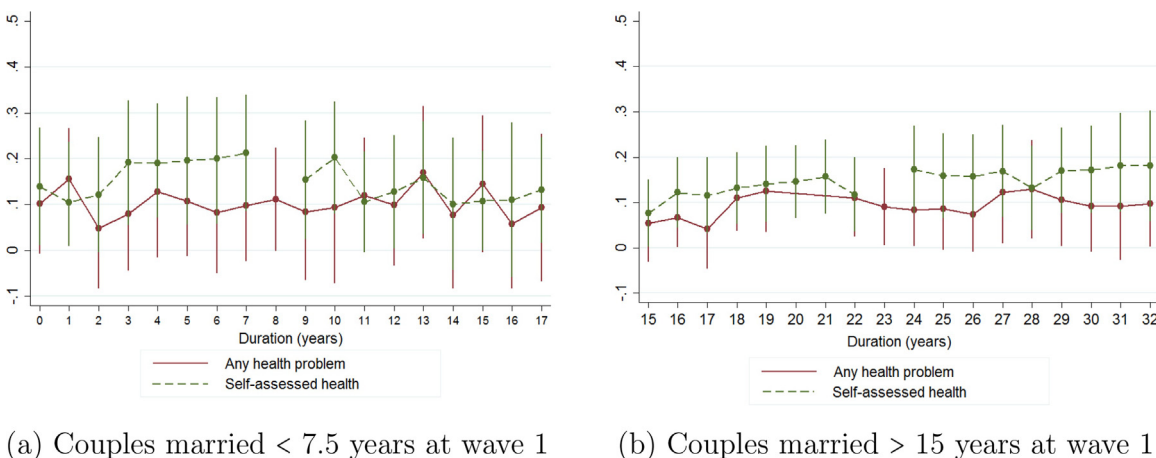


Fig. 6. British Household Panel Survey: estimated homogamy correlations by wave, 1991–2008 (age-adjusted, Epanechnikov kernel, bandwidth = 7.5).

**Table 4**  
Sensitivity of estimated homogamy correlations to adjustment for medication (bandwidth  $h = 7.5$  years).

Health measure	Unadjusted		Age-adjusted		Number of couples
	$\hat{R}(0)$	Std. err.	$\hat{R}(0)$	Std. err.	
<i>Systolic blood pressure (SBP)</i>					
No adjustment	0.364***	0.125	0.284***	0.100	3218
Fixed increment method	0.425***	0.119	0.290***	0.105	3218
Sample exclusion method	0.312**	0.152	0.256***	0.101	2115
<i>Diastolic blood pressure (SBP)</i>					
No adjustment	0.133	0.100	0.080	0.094	3218
Fixed increment method	0.165	0.105	0.089	0.087	3218
Sample exclusion method	0.094	0.094	0.057	0.082	2115
<i>Binary hypertension</i>					
No adjustment	0.322***	0.120	0.235**	0.109	3218
Combined BP/medication criterion	0.380***	0.119	0.162*	0.085	3218
<i>TC/HDL cholesterol</i>					
No adjustment	0.185**	0.083	0.232***	0.081	2196
Fixed increment method	0.217**	0.089	0.223***	0.084	2196
Sample exclusion method	0.216**	0.095	0.237***	0.085	1532
<i>Triglycerides (TG)</i>					
No adjustment	0.195*	0.109	0.180*	0.105	2203
Fixed increment method	0.201*	0.111	0.169*	0.090	2203
Sample exclusion method	0.224**	0.110	0.208*	0.108	1535
<i>HbA1c</i>					
No adjustment	0.317***	0.108	0.071	0.115	2006
Fixed increment method	0.322***	0.108	0.074	0.116	2006
Sample exclusion method	0.207**	0.100	0.068	0.108	1817
<i>C-reactive protein</i>					
No adjustment	0.249**	0.114	0.237**	0.117	2000
Sample exclusion method	0.258**	0.131	0.261**	0.123	1286
<i>Fibrinogen</i>					
No adjustment	0.173**	0.082	0.132**	0.065	2189
Sample exclusion method	0.170**	0.080	0.127**	0.067	1917
<i>Allostatic load</i>					
No adjustment	0.421***	0.151	0.386***	0.146	1274
Adjusted	0.554***	0.105	0.402***	0.124	1274
<i>CVD risk score</i>					
No adjustment	0.534***	0.135	0.402***	0.142	1287
Adjusted	0.553***	0.144	0.383***	0.147	1287

\* Statistical significance = 10%.

\*\* Statistical significance = 5%.

\*\*\* Statistical significance = 1%.

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. Sensitivity analysis for relevant components of the allostatic load and cardiovascular score indexes gives a combined sensitivity analysis for each.

Table 4 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. 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 Figs. 1–3.

## 7. 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 measures 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 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 or environmental factors are absent, but that they are equally important as initial partner selection, in a specific 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 0.38) are observed for adiposity measures. Also significant, but smaller in magnitude (around 0.18–0.28) are the correlations for measures of blood pressure, resting heart rate, the cholesterol ratio triglycerides and inflammatory markers, which all tend to accompany adiposity. We also find rather larger homogamy correlations for composite systemic measures of health, designed to reflect allostatic load or (particularly) cardiovascular risk.

We find 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 and environment 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 SAH, BMI and blood pressure (Monden, 2007; Di Castelnuovo et al., 2009; Wilson, 2012).

A further contribution of our study is the finding of robustness with respect to a number of potential sources of bias. Particularly worrying for research in this area are: the impact of mortality, which means that observed intact marriages are not representative of marriages in general; time-variations in homogamy that might confound the empirical duration profile; and medication which can mask the underlying health state. We have used simulation to indicate the possible range of mortality bias, demonstrated the empirical stability of health-specific homogamy, and compared different approaches to observations affected by medication. These robustness checks have found no grounds to suggest that our key results are non-robust. Moreover, our results are not sensitive to age-at-marriage effects, and the key finding of flat concordance profiles also holds for subject health assessments reported by the same couples followed over time.

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.

Finally, there are potential limitations that should be borne in mind. The analysis rests on the ability of our statistical model and estimation techniques to capture the complex interactions that underlie the joint evolution of partners health. Until we have longer observation periods with multiple repetitions of biomarker measurement for the same individuals, there will be inevitably be some questions about our evidence, despite its apparent robustness. A particular concern in this area is that the cross-sectional nature of most of our health measures do not allow us to explore the

potentially role of cohort effects. Although we have been able to demonstrate robustness for repeated self-reported measures in the BHPS, it is not possible to show that this holds also for biomarkers. The second, major limitation is that our empirical separation of the roles of homogamy and subsequent causal effects of shared lifestyle, rests on a reduced form approach that cannot distinguish the many possible behavioural and biological mechanisms through which shared exposures may affect couples health. The identification of those processes is a formidable challenge that will face health economists and epidemiologists far into the future.

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## Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <http://dx.doi.org/10.1016/j.jhealeco.2017.09.010>.

## Appendix B. The accumulation model of correlated health risks

The exposure-accumulation model is:

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

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,  $\omega_h$  and  $\omega_w$ , 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  $M_j(a_j, \omega_j)$ :

$$M_j(a_j, \omega_j) = \lambda(a_j)E [H_j(b_j)|a_j, \omega_j] + \sum_{t=b_j+1}^m \lambda(T-t)\mu_z(t) + \sum_{t=m+1}^{m+d} \lambda(T-t)[\mu_x(t) + \mu_s(t)] \tag{B.2}$$

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:

$$M_j(a_j, \omega_j) = \lambda(a_j)E [H_j(b_j)|a_j, \omega_j] + \sum_{\tau=1}^{\omega_j} \lambda(a_j - \tau)\mu_z(T - a_j + \tau) + \sum_{\tau=1}^{a_j - \omega_j} \lambda(a_j - \omega_j - \tau) [\mu_x(T - a_j + \omega_j + \tau) + \mu_s(T - a_j + \omega_j + \tau)] \tag{B.3}$$

This is a nonlinear function of  $a_j$  and  $\omega_j$ , 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:

$$H_j(T) - M^*(0, \omega_j) = \lambda(\omega_j) \{ [H_j(b_j) - E [H_j(b_j)|b_j, \omega_j]] + \sum_{\tau=1}^{\omega_j} \lambda(\omega_j - \tau) \{ z(T - \omega_j + \tau) - \mu_z(T - \omega_j + \tau) \}, j = h, w \tag{B.4}$$

The homogamy correlation for the cohort of couples marrying at ages  $\omega_h$  and  $\omega_w$  is:

$$R_T(0, \omega_h, \omega_w) = \text{corr} (H_h(m) - M_h^*(d, \omega_h), H_w(m) - M_w^*(d, \omega_w)) | d = 0, \omega_h, \omega_w \tag{B.5}$$

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  $E [H_j(b_j)|a_j, \omega_j]$ ,  $\mu_z(t)$ ,  $\mu_x(t)$  and  $\mu_s(t)$ .

Under the assumed structures (3)–(5) for the sequences of health shocks, the cumulated post-marital health impacts are:

$$\sum_{t=m+1}^T \lambda(T-t) [x_j(t) + s(t)] = \left( \sum_{t=T-d+1}^T \lambda(T-t) \right) [u_j + v] + \sum_{t=T-d+1}^T \lambda(T-t) [\varepsilon_j(t) + \theta\varepsilon_j(t-1) + \eta(t) + \psi\eta(t-1)] = \Lambda(d)[u_j + v] + \lambda(T-d+1) [\theta\varepsilon_j(T-d) + \psi\eta(T-d)] + \varepsilon_j(T) + \eta(T) + \sum_{t=T-d+1}^{T-1} \lambda(T-t) [(1+\theta)\varepsilon_j(t) + (1+\psi)\eta(t)] \tag{B.6}$$

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

$$\text{Var} (H_j(T)|\omega_h, \omega_w, d) = \Omega_j(\omega_h, \omega_w, d) + 2\Lambda(d)\Lambda(\omega_j) [\gamma\sigma_u^2 + \kappa\sigma_v^2] + \Lambda(d)^2[\sigma_u^2 + \sigma_v^2] + \lambda(T-d+1)^2 [\theta^2\sigma_\varepsilon^2 + \psi^2\sigma_\eta^2] + \sigma_\varepsilon^2 + \sigma_\eta^2 + \Psi(d) [(1+\theta)^2\sigma_\varepsilon^2 + (1+\psi)^2\sigma_\eta^2] \tag{B.7}$$

where  $\Omega_j(\omega_h, \omega_w, d)$  is the variance of cumulated pre-marital exposures given by (B.4) and  $\Psi(d) = \sum_{i=1}^{d-1} \lambda(i)^2$ . The second term of (B.7) arises from the presence of the persistent factors  $u_j$  and  $v$  in both the pre- and post-marital exposure sequences  $z_j(t)$  and  $x_j(t)$ ; the other variance terms come from the last four terms of (B.6). The covariance between the partners' observed health states is:

$$\text{Cov} ([H_h(T) - M_h], [H_w(T) - M_w]|\omega_h, \omega_w, d) = \Omega_{hw}(\omega_h, \omega_w, d) + \Lambda(d)^2\sigma_v^2 + \lambda(T-d+1)^2\psi^2\sigma_\eta^2 + \Psi(d)(1+\psi)^2\sigma_\eta^2 \tag{B.8}$$

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.

The correlation  $R(d)$  is the ratio of (B.8) to the square root of the product of the variances given by (B.7).

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.<sup>17</sup> We make the following reasonable assumptions:

- (i) The cumulated sequence of squared autoregressive coefficients is of lower order in  $d$  than the squared cumulated sequence:  $\lim_{d \rightarrow \infty} \{ \Psi(d) / [\Lambda(d)^2] \} = 0$ .
- (ii) The squared autoregressive coefficient  $\lambda(T-d+1)^2$  is of lower order in  $d$  than  $\Lambda(d)$  and  $\Psi(d)$ .
- (iii) 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  $\omega_h, \omega_w$ .

The correlation profiles implied by (B.7) and (B.8) are complicated and not necessarily monotonic in  $d$ . However, under assumptions (i)–(iii) asymptotic behaviour of  $R(d)$  is clear. If fully persistent effects are present ( $\sigma_u, \sigma_v \neq 0$ ), the terms in  $\Lambda(d)^2$  in (B.7) and (B.8) are dominant as  $d \rightarrow \infty$ . If  $u_h, u_w$  and  $v$  are absent, the terms in  $\Psi(d)$  dominate (B.7) and (B.8) at long durations. It follows immediately that the between-partner health correlation conditional on duration (and implicitly any combination of ages at marriage,  $\omega_h, \omega_w$ ) converges as follows:

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

With both persistent effects present ( $\sigma_u^2, \sigma_v^2 > 0$ ), the spousal correlation converges to the variance share of the joint component  $v$ . In the special case where the personal health shocks are persistent but the shared component is not ( $\sigma_u^2 > 0, \sigma_v^2 = 0$ ),  $R(d)$  eventually

converges to 0; in the reverse case ( $\sigma_u^2 = 0, \sigma_v^2 > 0$ ),  $R(d)$  converges to 1.

If persistent effects are absent ( $\sigma_u = \sigma_v = 0$ ), under the MA(1) specification the variances of personal and shared exposures are  $\text{var}(x_h(t)) = \text{var}(x_w(t)) = (1+\theta^2)\sigma_\varepsilon^2$  and  $\text{var}(s(t)) = (1+\psi^2)\sigma_\eta^2$ . In that case, the asymptote  $R(\infty)$  is equal to the variance share  $\text{var}(s(t))/[\text{var}(x(t)) + \text{var}(s(t))]$  if the sequences of shared and personal health shocks are serially uncorrelated ( $\theta = \psi = 0$ ), or have the same non-zero autocorrelation ( $\theta = \psi \neq 0$ ). If  $\theta$  and  $\psi$  differ,  $R(d)$  converges to a limit  $[\text{var}(s(t)) + 2\psi]/[\text{var}(s(t)) + \text{var}(x(t)) + 2(\psi + \theta)]$ , which exceeds the

<sup>17</sup> An example is the random walk model where exposures have a simple additive effect and  $\lambda(T-t) = 1$  for all  $t$ , implying  $\Lambda(d) = d; \Psi(d) = d - 1$ .

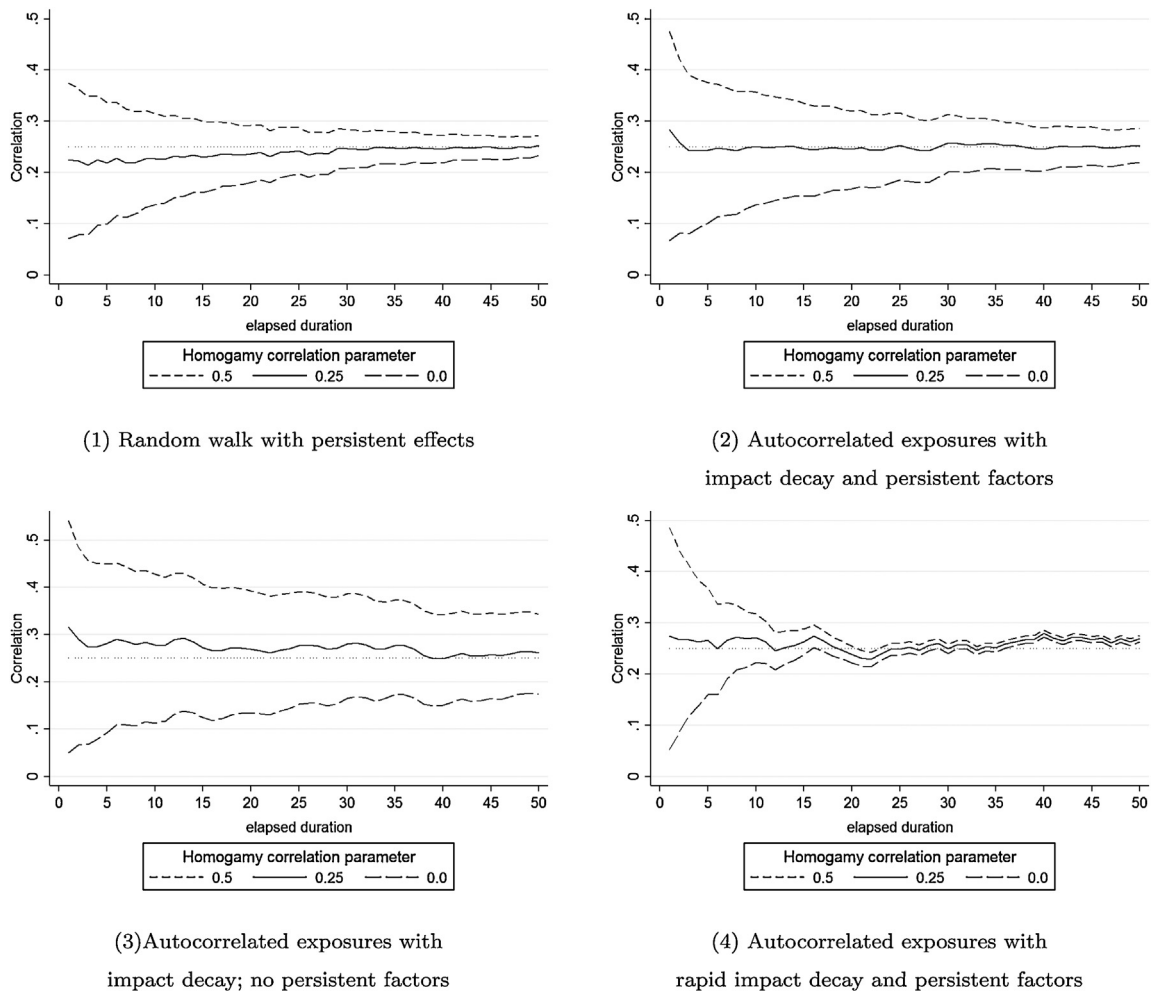


Fig. A1. Intra-couple correlation profiles for three simulated health processes.

variance share if  $\psi/\theta > \text{var}(s(t))/[\text{var}(s(t)) + \text{var}(x(t))]$  and vice versa.<sup>18</sup>

For finite durations, the spousal correlation profile has a complicated structure and its shape depends on the parameters  $\{\Omega_{hw}, \Omega_h, \Omega_w, \sigma_u, \sigma_v, \sigma_\varepsilon, \sigma_\eta, \theta, \psi\}$ . To illustrate the range of possibilities, Fig. A1 shows the correlation profiles generated from four hypothetical health processes by Monte Carlo simulation, with  $\xi_h(t), \xi_w(t), \varepsilon_h(t), \varepsilon_w(t), \eta(t)$  generated as pseudo-random draws from zero-mean normal distributions.<sup>19</sup> In all four examples, personal exposures  $\varepsilon_h(t)$  and  $\varepsilon_w(t)$  have standard deviation 1.0. The variances of  $\xi_h(t), \xi_w(t)$  are set so that the sequences of pre-marital exposures  $z(t)$  and post-marital exposures  $x_j(t) + s(t)$  have identical variances. In each example involving persistent factors, their pre-marital influence is set as  $\gamma = \kappa = 0.5$ , implying that half of shared later lifestyle is acquired from preferences or habits originating before marriage. We allow for other forms of homogamy by setting  $\text{corr}(H_h(b_h), H_w(b_w)) = \text{corr}(\xi_h(t), \xi_w(t))$  at three alternative values: 0.5, 0.25 and 0.0.<sup>20</sup>

Example 1 is a random walk process with  $\phi = \theta = \psi = 0$ , and  $\lambda_0 = 1, \lambda_1 = 0$ ; shared exposures  $\eta$  are generated with  $\sigma_\eta = 0.5$ , and the persistent effects with standard deviations  $\sigma_u = 0.2, \sigma_v = 0.06667$  so that the long-run correlation  $R(\infty) = 0.25$ . Example 2 allows for MA(1) dependence with  $\phi = \theta = \psi = 0.75$ , implying a 1st-order autocorrelation of 0.48; there is some decay in dependence on past exposures with  $\lambda_0 = \lambda_1 = 0.5$  and persistent factors are more important than in example 1, with  $\sigma_u = 0.3$  and  $\sigma_v = 0.1$ , again implying  $R(\infty) = 0.25$ . Example 3 is identical to example 2 except that the persistent effects are removed ( $\sigma_u = \sigma_v = 0$ ) and  $\sigma_\eta$  is increased slightly to 0.577 so that  $R(\infty)$  is again 0.25. Example 4 is the same as example 3 except that we remove the MA(1) components ( $\phi = \theta = \psi = 0$ ) and allow much more decay in the impact of past exposures, by setting  $\lambda_0 = 0.2, \lambda_1 = 0.8$ .

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<sup>18</sup> Note that these results remain true if there are gender differences in the impact of exposures on health outcomes ( $\lambda_h(\cdot) \neq \lambda_w(\cdot)$ ), provided our assumptions on  $\lambda(\cdot)$  apply equally to men and women. In that case, the dominant term  $\Lambda(d)^2$  is replaced by  $\Lambda_h(d)\Lambda_w(d)$  but still cancels out in the ratio defining the limiting correlation.

<sup>19</sup> We used 5000 Monte Carlo replications, realising the processes over 75-year lifespans with a 4-year age difference between partners.

<sup>20</sup> Note that, for the first two cases,  $R(0)$  is not exactly 0.5, 0.25 since the exposures experienced by the man in his first four years of life contribute to his health variance but not to the homogamy covariance. However, the difference is small.

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