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In sickness and in health? Comorbidity in older couples

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Non-technical summary

In this paper we examine the tendency for both members of a marital couple to experience similar degrees of good or ill-health – a phenomenon which we refer to as marital comorbidity. Comorbidity within couples is important for policy purposes. For example if both members of a couple suffer disability, they may not be able to care for each other and rely on outside sources of care, increasing costs to themselves and to national and local benefit schemes. It is also potentially important for understanding the development of health over the lifecourse. By analysing longitudinal data giving information about childhood health and also health during marriage, we are able to better understand the relative importance of early and late influences on health in later life. The aim of this paper is to examine the extent and origins of marital comorbidity and the role of the early-life environment and the later shared home environment, as influences on health in later life.

We use data from the Survey of Health and Retirement in Europe (SHARE) study, which is a multi-country longitudinal survey of people aged 50 and over and their partners. The third wave of SHARE, conducted in 2008-2009, provided a retrospective life history for each participant. Survey respondents were asked about their health during childhood and also their fertility, partnership, work and health histories from the age of 15 to the current date. This provided detailed data on almost 7,000 partnerships. We develop a dynamic statistical model involving latent health measures for each marital partner in the childhood, pre-marriage and post-marriage periods. Health emerges as a cumulative process, with strongly significant evidence that past health states influence later health.

However, the results suggest that, for the SHARE cohort relatively late in life, only about 15% of the total variation in health across individuals could be attributed to influences at work prior to marriage. Within the marital period, a high proportion of the variation in health (52% for women and 73% for men) is attributable to factors we are not able to observe or measure. We also found that the health states of partners before they married were almost as strongly correlated as the health changes experienced during the marital period. Thus assortative mating (the tendency for people to marry others with similar health states) is as strong in correlation terms as the effect of the shared marital environment.

Our analysis is necessarily speculative and is based on long-range recall data which is of questionable reliability. We discuss some of the options for finding more reliable data, but the prospects are not very good at present. Almost all longitudinal health surveys are individual-based and cannot provide information on both members of a couple in both the pre- and post-marital phases of their lives, as required. But the issue of comorbidity within couples is important and has received very little research attention, so further work to develop usable datasets would be very valuable.

In sickness and in health?

Comorbidity in older couples

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Abstract

We investigate the nature and origin of comorbidity, defined as the tendency of members of marital couples to display correlated patterns of ill-health in later life. In the absence of long-term prospective data on couples, we use long-range recall data from the pan-European SHARELife survey and estimate a latent variable model of the health states of marital partners in childhood, early adulthood and late adulthood. We find strong persistence in health states and a strong comorbidity correlation, attributable almost equally to homogamy in relation to early health and common factors operating within marriage.

Keywords: Health, disability, lifecourse, homogamy, SHARE

JEL codes: I12, J13

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1 Introduction

The term comorbidity usually refers to multiple diseases experienced simultaneously by a single individual. Here instead, we consider comorbidity from the viewpoint of married or cohabiting couples[1] to describe any situation in which both members of the couple are significantly affected by disease or disability. Comorbidity is important for two main reasons.

First, any tendency for disease or disability to be concentrated within couples will change the nature and distribution of the social cost of disease. For example, it has been estimated that the implicit annual cost of informal care supplied to disabled people in the UK amounts to £119bn, which easily exceeds the total cost of the National Health Service (Buckner and Yeadle 2011). 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, Table 2). This can be interpreted as a large-scale system of informal insurance, based on the principle of risk pooling by couples. But, if disability affects both partners simultaneously, it reduces their capacity to provide care for each other. Marital comorbidity represents positive correlation between risks within couples, and therefore reduces the effectiveness of pooling and the capacity of couples to self-insure. Dependency on external care services increases as a consequence of comorbidity, with corresponding budgetary impacts on the government through increased financial costs of social care and higher rates of dependence on welfare benefits.

A second reason to be interested in the phenomenon of marital comorbidity is that it may tell us something about disease processes, and particularly the relative importance of early and late environmental influences on health in later life. For example, if we were to find a strong tendency for comorbidity even in couples where the two partners have very different early histories, that would constitute evidence in favor of shared lifestyle factors as a cause, and against the view that environmental influences very early in life are the dominant cause of poor health in later life. This would in turn have policy implications for the optimal balance between health interventions directed at young families and interventions aimed more directly at the older population where disease and disability are most prevalent.

[1]For economy of language, we use the term marriage from this point on to denote any domestic partnership, whether or not it has legal marital status (marriages comprise 96% of such relationships in our dataset). We do distinguish between the two types of relationship in our empirical analysis.

The research literature on comorbidity in couples is 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 behaviors). These numbers are tiny compared to the literature on individuals, twins and siblings. Work on marital comorbidity includes cross-sectional analysis of the associations between spousal incidence of a range of diseases (Hippisley-Cox *et al* 2002) and more persuasive longitudinal studies which have found evidence of comorbidity in various health domains, including psychiatric disorders (Joutsenniemi *et al* 2011), alcohol dependency (Grant *et al* 2007) and obesity (The and Gordon-Larsen 2009). However, there is little epidemiological research based on long-term data that covers the long durations of many marital relationships and the preceding histories of the marital partners.

Economic and social research has looked at the way that disease and disability affects households. For example, economic research on retirement decisions suggests that the state of health of the domestic partner is an important influence on the timing of withdrawal from the labor market and consequently post-retirement income (see Disney *et al* 2006 for the UK, Bound *et al* 1999 and McGarry 2004 for the US and Cavapozzi 2008 for various European countries). Work on disability in the social policy literature has examined policy responses in terms of the design of care services and financial support for households affected by disability but has not focused specifically on the issue of marital comorbidity as an important dimension of household need. Virtually none of this work has examined the origins of comorbidity and its relationship with socio-economic disadvantage earlier in life.

Our aim in this paper is to describe the extent of comorbidity and investigate its sources, using data from a large, longitudinal, multi-country survey (SHARELife) which takes a whole-life perspective. An important issue in the use of this observational approach is the role of homogamy as a potential confounding factor. It has been clearly established that there is a tendency for people to associate with others who are similar to themselves: a phenomenon first termed homophily by Lazarsfeld and Merton (1954) and usually referred to as homogamy or assortative mating in the context of partnership formation. Homogamy, and homophily more generally, has been observed in relation to a wide range of characteristics, including age, ethnicity, gender, religion and occupation (Fong and Isajiw 2000, Moody 2001, McPherson *et al* 2001). Although there is less evidence on homophily in health states, it is highly likely that some part of any comorbidity evident in the data is attributable to

early-life individual factors which happen to be brought together in couples by assortative mating, rather than to processes operating as a consequence of the relationship itself.

We attempt to answer a number of specific research questions. First, is there *prima facie* evidence of comorbidity? Is there also evidence of homogamy in terms of childhood and pre-partnership adult health outcomes? Is the estimated degree of comorbidity reduced after allowing for homogamy with respect to childhood and pre-partnership adult health? Does comorbidity increase with partnership duration, as we would expect if it is the outcome of processes operating during the course of the partnership? We begin by outlining some alternative theories of comorbidity.

2 Theories of comorbidity

It is important to distinguish "true" comorbidity (meaning that factors originating in the marriage itself have a direct causal influence on the partners' health outcomes) from concordance of health outcomes arising from similarity of individual-specific causal factors predating the marriage. Rather little research has focused specifically on the origins of comorbidity, but there are at least five plausible causal mechanisms that could lead to comorbidity in long-established partnerships.

Social contact and stress Social isolation has been identified empirically as a psychological stressor, with observable expression in terms of low levels of subjectively-reported wellbeing (Diener et al 1999) and 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 (see Chrousos 1992 for an overview). Partnership itself has a direct impact on the two partners' social engagement and risk of isolation, and the nature of the partnership and its links to wider society can be expected to have some impact on the body's stress system. Marital partners engage with each other and have access to a wider combined network of family and friends. Consequently, they share to some extent the same pattern of social engagement and exposure to social stress, with the possibility of similar health consequences.

Social control and influence Each partner in a long-term relationship has strong private incentives, both economic and emotional, to have a partner who is in a good state of health. This may prompt each partner to attempt to exert control over the other's health-related

behavior. 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 thus create a tendency for similar health outcomes. Social influence on behavior may also operate through unconscious processes of demonstration and imitation, within the shared social networks of the marriage itself and the couple's wider social circle.

Direct contagion Contagious physical disease is a much less important threat to health in Europe than it once was, so the argument for direct communication of health shocks between spouses is relatively weak. However, smoke inhalation by the partners of cigarette smokers, may be a significant channel for physical disease. In the case of mental illness or disability, there is also a credible argument for a direct causal impact of one partner's health state on that of the other, since the symptoms of mental disorder and the burden of caring responsibilities may be very stressful for those exposed to them. If stress is an important risk factor, this may lead to mental or physical illness in the partner.

Household production The theory of household production (Becker 1965) emphasizes the cost advantages of household-level production of basic commodities like nutrition and entertainment. Meals are prepared and eaten in the home and the quality of this shared element of the diet may be related to disease processes. The home is also the setting for production of common entertainment outputs, ranging from health-promoting leisure activities like family walks in the country to health-impairing sedentary activities like television-watching. There is a compelling body of evidence linking diet and physical exercise to health outcomes (see Blanchflower et al. 2012, Haskell et al. 2007, Kannel and Sorlie 1979, Penedo and Dahn 2005, Warburton et al 2006, Willett 1994, WHO 1990).

The marriage market There is strong evidence that body shape and possibly other physical signs of health have an influence on individuals' success in partnership formation (Sobal 2005). It follows that, once a stable marital relationship has been established, there is some reduction in the incentive to maintain a healthy lifestyle. Empirical evidence of a tendency for body mass to increase after marriage is consistent with this idea (The and Gordon-Larsen 2009). On this argument, participants in a marriage perceived to be unstable will retain a stronger incentive to maintain a high value on the external marriage market by staying in shape. Thus, even without commonality through direct contagion, social influences or household production, we might observe similarity in the trajectories of morbidity in the

two marital partners by virtue of their simultaneous transition from single to married status, with the size of this effect being related to shared perceptions of the stability of the marriage.

In contrast to these theoretical explanations, some of the most influential work on health over the lifecourse emphasizes processes which leave little room for true causal comorbidity. An example is the large literature, notably work by Barker (1991), which suggests that environmental conditions during the fetal stage and infancy have a dominant influence on the risk of disease much later in life. In addition to fetal environment, patterns of early growth during infancy are also linked to long-term health outcomes (Hales and Ozanne, 2003). For example, studies have found that rapid growth among low birth weight infants is associated with higher risk of coronary heart disease (Eriksson et al. 2001), obesity (Stettler and Iotova 2010), insulin resistance (Hales et al. 1991; Ong and Dunger 2002) and cancer (McCormack et al. 2003; Okasha et al. 2002). Recent research in economics also suggests an impact on many non-health outcomes such as as labor market success (Black et al. 2007, Currie and Moretti 2007, Oreopoulos et al. 2008, Royer 2009) and marital status (Almond et al. 2010; Brandt et al. 2008). Almond and Currie (2011) conclude that the evidence for the fetal origins hypothesis is abundant, but there remain areas of uncertainty, including gender effects, since much of the early research was conducted with male-only samples. The causal dominance of the fetal/infancy environment as a determinant of health outcomes is still challenged. Research by Marmot and colleagues suggests that socioeconomic inequalities in childhood and adulthood are also associated with differential risks of disease in later life (Marmot et al 2001, Marmot 2005, Marmot and Wilkinson 1999, Marmot et al. 1987, Wilkinson 1992). These findings are closely related to cumulative disadvantage theory (Ferraro and Kelley-Moore 2003), which expands on the fetal origins hypothesis by asserting that relative advantage or disadvantage in early life influences life trajectories, resulting in accumulation of risk or protective factors leading to better or poorer short- and long-term outcomes. Thus, individuals who experience adverse *in utero* events also tend to be born into lower socioeconomic circumstances, do worse in school and get lower income jobs; these combined circumstances may also play a large part in the development of disease.

A second line of research which casts doubt on the extent of true comorbidity deals with homophily. Although there is limited evidence on homophily in health states, there is a large medical literature on the diffusion of health-related behaviors, where it has been mooted as

an important confounding factor in the debate over the contagion hypothesis for obesity and other phenomena (Christakis and Fowler 2007, Lyons 2011). Ingenious experiments have been used to distinguish the effects of homophily and social contagion in some settings (see 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.

It is very difficult to discriminate among these theories of comorbidity since they involve factors like social interactions, household production and perceptions of marital stability, which are generally not observed and are hard to measure in the survey context. Instead, we adopt the more modest aim of distinguishing "causal" comorbidity from the effects of homogamy. This is a very challenging task, because the requirement for long-term observation of individuals who become linked through marriage is difficult to fulfill. In the next section we consider the advantages and disadvantages of alternative data sources and describe the SHARELife survey data that we use for our analysis.

3 Data

3.1 Alternative data sources

We are interested in long-term health processes, operating over a large part of the lifecourse. There are very few individual-level sources of long-term data that can be used for this work, and all have potentially serious drawbacks. There are three classes of observational data that can be considered:

Prospective longitudinal surveys would be the preferred type of data. Long-running examples include the English Whitehall I and II studies (Marmot et al 1978, Marmot et al 1991) and the 1946 and 1958 UK birth cohort studies (Cooper et al 2012). Some of these prospective surveys cover specific groups which may be unrepresentative of the wider population, and they often suffer from significant attrition problems. Most seriously for our purposes, they are generally individual-based and do not give adequate coverage of the health history of the marital partner of the main respondent. A possible solution to this shortcoming is to use a survey confined to a small geographical area, which can observe large

numbers of marriages within the sample. An example of this type of survey is the *Children of the 1950s* study (Leon et al 2006), which follows a cohort of people who were in primary school (age 6-12 years) in Aberdeen in 1962. Of the roughly 12,000 children in the study, the 2001 interview sweep generated 844 respondents (from the roughly 7,000 respondents to that sweep) who reported being in a domestic partnership with another study participant and over a thousand who were co-resident with another participant. This suggests around 400-500 couples whose data may be analysed. The small size of this sample and the selected nature of the sample of people who choose to marry locally illustrate the drawbacks of this approach.[2]

Register data are a possible source of data, since it is in principle possible to match basic socio-economic and demographic data to health service records from the fetal stage onwards, using unique individual identifiers. The most complete datasets of this kind are from the Nordic countries, where universal personal identifiers provide a sound basis for linking registers. A drawback of this approach is that the available health outcome measures would only be those visible to the health service, which might introduce selection bias into health measurement. More seriously, the relatively short length of the available complete matched social and medical histories prevents analysis of long-term health consequences. For example, in Norway the birth register with indicators like birthweight and the Apgar score begins in 1967, and this can in principle be linked to registers for cause of death, cancer, medium-term sick leave and disability pension receipt, which are relevant to later life health status. However, there there is not a complete register of early immunization and child health so, even for this cohort, the early part of health histories is not well documented.

Retrospective survey data One way of overcoming these problems is to use data from retrospective general-population surveys that ask respondents to use long-term recall to give information on circumstances and health outcomes earlier in life. These surveys have two serious limitations. First, the impossibility of interviewing the deceased means that the health history of the deceased partners of widow(er)s is unobservable, so the sample of fully-observed couples is potentially distorted by mortality censoring which may cause selection bias. As far as we are aware, nothing is known about the nature and magnitude of this bias

[2]We are grateful to Heather Clark (University of Aberdeen) for providing these sample numbers.

for our purposes. Second, the reliability of long-term recall is questionable, although there is some empirical evidence on this, which we summarize below.

3.2 The SHARELife survey

In the absence of better alternatives, our analysis is based on the Survey of Health and Retirement in Europe (SHARE), which is a harmonized longitudinal survey of people aged 50 and over together with their domestic partners. SHARE was launched in 2004 in eleven countries: Denmark, Sweden, the Netherlands, Belgium, Germany, Austria, Switzerland, France, Italy, Spain and Greece and has since been expanded to include the Czech Republic and Poland. We exclude Greece from our analysis because of concerns about data quality arising from sample anomalies which are currently unexplained, and combine Austria, Germany and Switzerland to avoid small sample sizes that make it difficult to identify country effects. Fieldwork is conducted biennially and the third wave, implemented in October 2008-September 2009 and known as SHARELife, was devoted to a retrospective assessment of the whole life history of the respondent, covering the details of respondents' relationships, fertility, health, employment, migration and much else.

The SHARELife questionnaire and interview method is designed to achieve the most accurate recall possible. Respondents construct their personal histories using a computer-assisted life grid version of the Life Calendar Method (LCM). The advantage of LCM is the use of major events such as marriage or birth of child as anchors to explore other less salient events that may have occurred around the same time and this has been shown to achieve better recall than simple retrospective question designs (Börsch-Supan and Schröder 2008, Belli et al 2004). The accuracy of this type of dat has been assessed in several studies. Berney and Blane (1998) found recall of paternal occupation and residential information to be between 80-100% accurate after 50 years. Krall *et al* (1988) used child health records to validate respondent's reports, at age 30, 40 and 50, of childhood illnesses. Overall, respondents recalled childhood illnesses with good accuracy; however there were cases of under and over-reporting of illnesses. The accuracy of recall has been examined for the SHARELife data by Havari and Mazzonna (2011), who concluded that overall recall of childhood health had good internal and external consistency. In particular, they found no evidence that cognitive function or educational level influenced recall of childhood illnesses once country effects and

socioeconomic status during childhood were allowed for. However, they identified a possible problem of contamination (or *coloring*) of the remembered past by present conditions. They compared estimates of the impact of self-assessed good childhood health on similar measures of adult health collected concurrently in SHARElife and earlier in SHARE wave 2: the concurrent adult health outcome gave a logit coefficient significantly higher (by a fifth for men and a third for women) for the concurrent adult health measure, suggesting a modest contamination effect. We use this same approach, by including repeated general health measures from SHARE waves 1 and 2 and from SHARElife as indicators of within-marriage health outcomes.[3]

3.3 Identifying partnership histories

We use data from individuals who are in a cohabiting or marital relationship at the time of interview for SHARELife. Information on the current relationship (such as its year of initiation) is collected separately from each of the partners, so that there is potential for conflict in their responses, which provides a useful indicator of recall accuracy. The status of the current relationship was defined as the most recent marriage or cohabitation where the respondent indicated that they were still living with their partner. For each relationship respondents were asked for information about the year the relationship started, the year they moved into together and the year they got married, if applicable. For the purposes of this study we use the year the respondent moved in with their partner as the point of origin of the relationship, since it marks the start of the shared environment. In the majority of cases it also coincides with the reported year the relationship started and the year of marriage. The duration of the relationship was calculated by subtracting the year of the start of the relationship from the year of interview. Durations were calculated for both partners and then compared for consistency. The average duration was 39.20 and 39.19 years for men and women respectively, with standard deviations of 11.12 and 11.13 years. Overall, 92% of couples agreed on the start year of their relationship (see Appendix Figures A1 and A2 for the distribution of mismatches of marriage dates). For the purposes of this analysis couples with duration differences of 2 years or less were included, resulting in retention of 95% of

[3]For evidence of this type of recall error in a different context see Pudney (2011) who found evidence of contamination by present circumstances in recall of past financial wellbeing

originally sampled couples. Sample mean durations are 9.76 and 12.74 years for women and men respectively.

3.4 Three-phase health measures

In longitudinal data, the members of a couple are linked by two common dates: the time (T_M) at which their marital relationship began and the time (T_I) at which they are both interviewed as survey respondents. At the time of interview, their relationship has lasted for $\delta_M = T_I - T_M$, which is a measure of their 'exposure' to a shared lifestyle. Before the start of their relationship, they have independent personal histories, which we divide into two phases: childhood, defined in conformity with the SHARELife questionnaire as lasting $\delta_C = 16$ years and pre-marital adulthood, which lasts for $\delta_A^H = T_M - T_1^H$ and $\delta_A^W = T_M - T_1^W$ for the husband and wife respectively. This chronology is illustrated in Figure 2.

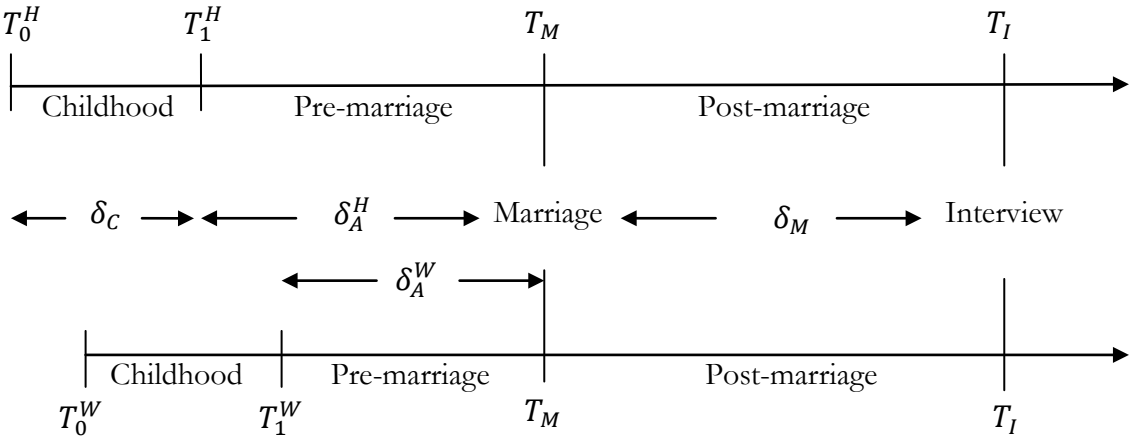

Figure 2 Lifecourse chronology

SHARElife respondents receive three sets of questions about their personal health history. The first is a questionnaire module covering childhood illnesses in the first 16 years of life; the second asks for recall of all significant episodes of ill-health from age 16 to the date of interview; and the third asks about the respondent's current state of health. From these, we construct the lifestage-specific indicators summarized in Table 1. Note that events like

marriage are central to the LCM recall procedure, so we can be fairly confident of the recalled timing of major health outcomes relative to marriage.

Table 1 Period-specific indicators of health outcomes

Variable		Men		Women	
		N	%	*N*	%
Childhood					
General assessment of childhood health[1]:	Excellent	2517	36	2103	30
	Very good	2341	34	2373	34
	Good	1590	23	1877	27
	Fair	387	6	436	6
	Poor	117	2	166	2
Missed school for a month or more through illness		664	10	772	11
Hospitalized a month or more or 3 times any year		465	7	451	6
Number of illnesses at ages 0-5[2]:	0	4267	61	4010	57
	1	2481	35	2708	39
	2	213	3	244	3
	3	30	1	29	1
Number of illnesses at ages 6-10[2]:	0	4072	58	3849	55
	1	2521	36	2760	39
	2	372	5	340	5
	3	26	1	42	1
Number of illnesses at ages 11-15[2]:	0	5937	85	5788	83
	1	920	13	1030	15
	2	122	2	144	2
	3	12	1	29	1
Any long-standing childhood illness[3]		154	2	293	4
Pre-partnership					
Number of illnesses starting before the marriage[4]:	0	6841	98	6793	97
	1	95	1	119	2
	2	43	1	59	1
Any pre-marital ill-health episode limiting activity		627	9	597	10
Notable period of ill-health prior to marriage[5]		281	4	289	4
Post-marital period					
General assessment of current health[1]:	Excellent	525	8	462	7
	Very good	1077	15	1040	15
	Good	2621	38	2703	39
	Fair	1832	26	1932	28
	Poor	927	13	848	12
Number of illnesses starting after the marriage:	0	6231	89	6305	90
	1	532	8	501	7
(same indicator observed at	2	172	2	134	2
SHARE waves 1 & 2 also used)	3	51	1	46	1
Any post-marital ill-health episode limiting activity		1686	24	1537	22
Notable period of ill-health after marriage[5]		2687	38	2782	40
		Mean	SD	Mean	SD
Dominant hand grip strength (z-score)[4]		0.24	1.15	0.11	1.4

Notes: [1] Corresponding factor loading normalized to +1. [2] Respondents were asked about 19 specific illnesses in childhood and any other serious condition for a total of 20. For each illness respondents were asked for the age of onset, 0-5, 6-10, or 11-15 years. A count of the number of illnesses in each age group was created, using only the 9 illnesses reported by 5% or more of the sample. [3] Respondents were asked if each illness lasted over a year. Any positive response was used to indicate a long-standing illness. [4] Corresponding factor loading normalized to -1. [5] Respondents were asked if there was a single distinct period of poor health relative the rest of his/her life; if so, the indicators record occurrence pre- or post-marriage. [4] Relative to height.

4 The statistical model

4.1 Specification

Partner p's health trajectory is a sequence of unobserved health states $h_t^p, t = 1...(T_I - T_0^p)$, for $p = H, W$. Our analysis views the current health state as a (weighted) accretion of influences over time:

$$h_t^p = \sum_{s=0}^{t} \phi_{st} \lambda_s^p \tag{1}$$

where λ_0^p is the pre-natal initial health endowment, λ_s^p is the effect of all influences operating in his or her sth year of life and ϕ_{st} is a weighting factor that captures any tendency for impacts to be greatest in particular sensitive or critical periods and subsequently to diminish or increase over time. We assume that the latter effect is uniform in the sense that ϕ_{rt}/ϕ_{st} is independent of t for any r, s. This implies, for example, that the appropriate overall measure of health during childhood does not change structure when we change the time period t from which we view it.

The form (1) is consistent with a wide range of dynamic structures, including stationary and non-stationary autoregressive processes[4], so it can accommodate Cunha et al's (2006) concept of self-productivity in health terms. We can separate the process (1) into components arising in childhood, pre-marital adulthood and the post-marital period, denoted periods 1, 2 and 3 respectively:

$$h_t^p = \left[\lambda_0^p + \sum_{s=0}^{15} \phi_{st} \lambda_s^p\right] + \left[\sum_{s=16}^{T_M - T_0^p} \phi_{st} \lambda_s^p\right] + \left[\sum_{s=15+\delta_A^p+1}^{T_I - T_0^p} \phi_{st} \lambda_s^p\right] \tag{2}$$

$$= \Lambda_1^p + \Lambda_2^p + \Lambda_3^p \tag{3}$$

Our most parsimonious model specification assumes that the appropriate summary measures of incremental health during each life phase is related to a set of observed covariates describing the characteristics of the individual and socioeconomic conditions during that period:

$$\Lambda_1^p = \boldsymbol{X}_1^p \boldsymbol{\beta}_1^p + U_1^p \tag{4}$$

[4]For the 1st-order autoregressive case $\phi_{st} = \rho^{t-s}$, where ρ is the Markov coefficient; if ρ is time dependent, this becomes $\prod_{j=s}^{t} \rho_j$.

$$\Lambda_2^p = \boldsymbol{X}_2^p \boldsymbol{\beta}_2^p + \Lambda_1^p \theta_{12}^p + U_2^p \tag{5}$$

$$\Lambda_3^p = \boldsymbol{X}_3^p \boldsymbol{\beta}_3^p + \Lambda_2^p \theta_{23}^p + \Lambda_1^p \theta_{13}^p + U_3^p \tag{6}$$

We allow unrestricted correlations between the two partners' unobserved health components (U_τ^H, U_τ^W) for each phase τ, to allow for common neighbourhood and other environmental factors during each life phase.

Except for the continuous grip strength measure, all the health indicators are ordinal so the measurement model for the jth observable indicator of health during life stage τ is:

$$Pr\left(H_{\tau j}^p = r\right) = Pr\left(\Gamma_{\tau jr}^p < \Lambda_\tau^p a_{\tau j}^p + e_{\tau j}^p \leq \Gamma_{\tau jr+1}^p\right) \tag{7}$$

where the six parameters $a_{\tau j}^p$ are factor loadings; one loading for each j, τ is normalised to +1 or −1 as required for the interpretation of Λ_τ^p as latent health rather than illness. The unobservable $e_{\tau j}^p$ is an indicator-specific residual or response error component. The $\Gamma_{\tau jr}^p$ are threshold parameters specific to the jth indicator for phase τ and partner p. We do not assume that the measurement errors $e_{\tau j}^p$ are all mutually independent, and instead allow for a set of specific correlations, which are summarized in the next section. The resulting model in our preferred specification has 243 parameters, which are estimated jointly using the robust weighted least squares method of Muthén (1984) and Muthén et al (1997), as implemented in the MPlus 6.1 software package.

4.2 The measurement model

The estimated factor loadings $(a_{\tau j}^p)$ and threshold parameters $(\Gamma_{\tau jr}^p)$ are presented in Appendix Table A2. As expected, indicators of health have statistically significant loadings which are positive for indicators of good health and negative for indicators of illness. The only exception to this is the 'objective' grip strength measure, whose loading is small and negative for women and grossly insignificant for men (the results are virtually unchanged if the grip strength measure is discarded).

We do not make the standard assumption that these health indicators are independent conditional on latent health. Instead, we allow for a specific pattern of stochastic dependence between the measurement errors $e_{\tau j}^p$, arising from three different sources.

Timing uncertainty The retrospective nature of SHARElife data introduces a degree of timing uncertainty, so we allow for correlations between the errors in an individual's recall of illnesses occurring within the three age ranges of childhood (0-5, 6-10 and 11-15). We expect these correlations to be negative since, if an illness is wrongly classified in (say) the 0-5 age range rather than the correct 6-10 range, early health will be under-reported but later health over-reported.

Inherent correlation Some indicators are inherently linked. For example, if a respondent was hospitalized frequently or at length during childhood, it is very likely that he or she will have missed a month of schooling. Similarly if, when invited to nominate a single period of serious ill-health, the respondent reports it as starting during the pre-marital period, it cannot be reported in the post-marital period. Conditional on health, these inherent relationships imply strong positive and negative correlations respectively, the former contemporaneous and the latter over time.

Influence on reporting behavior Contemporaneously correlated reporting errors may arise as a result of mutual influence between the two partners when reporting their state of health at interview. We believe this to be much more likely for subjective assessments than for recall of specific events so we allow for correlations among the general assessments, of health during childhood and at the time of interview, given by the two respondents.[5]

The estimated pattern of correlation between measurement errors is set out in Table 2. The results are largely as expected: there are negative correlations for all cases of timing uncertainty; positive correlations between the two partners' contemporaneous responses to subjective health questions; and inherent correlations with the anticipated signs. Unsurprisingly, the largest of these estimated correlations are for the timing of illnesses in early childhood and the association of hospitalization and school absence. Despite the use of the LCM aid to recall, there is also evidence of substantial timing uncertainty in adult health conditions.

Qualitatively, the results of our analysis are remarkably insensitive to the existence of this pattern of correlations between response errors. If the model is re-estimated with all

[5]This form of contamination has been found in survey responses to subjective wellbeing questions, for instance Conti and Pudney (2011) reported within-household cross-contamination of individual responses to questions on job satisfaction.

the correlations set out in Table 2 constrained to zero, the dynamic structure and estimated impact of the explanatory covariates remain essentially unchanged.

Table 2 Residual correlations between specific health indicators

Survey indicators	Women	Men
Timing uncertainty		
Childhood: illnesses 0-5 & 6-10	-0.623	-0.614
Childhood: illnesses 0-5 & 11-15	-0.168	-0.119
Childhood: illnesses 6-10 & 11-15	-0.170	-0.151
Inherent correlation		
Childhood: hospitalization & missed school	0.486	0.468
Pre- & post-marital occurrence of worst illness	-0.162	-0.248
Social influence		
Childhood: male & female general health	0.197	
Post-marital: male & female general health (wave 1)	0.097	
Post-marital: male & female general health (wave 2)	0.054	
Post-marital: male & female general health (wave 3)	0.093	

All correlation parameters are statistically significant at 1%level.

4.3 The estimated latent dynamic structure

The observed covariates are defined and summarized in Table A1 of the appendix. All covariate vectors include a complete set of dummy variables for country of residence. In addition, the childhood covariates X_1^W, X_1^H cover family socioeconomic position, rural residence, and indicators of specific source of childhood hardship, including: parental alcohol abuse, frequency of family moves, an absent father, experience of World War II hostilities; experience of famine; and dispossession of the family during childhood. The estimated coefficients $\boldsymbol{\beta}_1^p$ are presented in Table 3; they indicate significant health disadvantage for children in most countries relative to the reference case of Denmark; this is specifically so for Austria/Germany/Switzerland, the Netherlands, Spain, France, Belgium and the Czech Republic. We find highly significant adverse effects of certain types of exceptional family circumstances on child health. Heavy parental drinking has a significant negative effect for both men and women. Frequent moves of the family home, location in a rural area and an absentee father all have a significant negative impact, while high paternal social class has a significant positive impact, for women. Spending part of childhood in a belligerent country during WWII, being born at a time and place affected by wartime or post-war famine and

dispossession of the family have a significant negative impact for men. We have found no strongly significant evidence of an effect for the country's GDP per head during childhood.

Table 3 Latent variable model: partners' childhood health

	Female partner		Male partner	
	Parameter	Std.err.	Parameter	Std.err.
Austria/Germany/Switzerland	-0.442***	(0.061)	-0.406***	(0.065)
Sweden	-0.007	(0.066)	-0.145**	(0.067)
Netherlands	-0.458***	(0.068)	-0.420***	(0.071)
Spain	-0.228**	(0.093)	-0.189*	(0.098)
Italy	-0.041	(0.076)	0.100	(0.079)
France	-0.386***	(0.067)	-0.318***	(0.070)
Belgium	-0.230***	(0.065)	-0.183***	(0.067)
Czech Republic	-0.274***	(0.083)	-0.369***	(0.086)
Poland	-0.004	(0.094)	0.082	(0.099)
# home moves	-0.061***	(0.013)	-0.014	(0.012)
Rural area	-0.089***	(0.031)	0.002	(0.031)
Parental drinking	-0.186***	(0.048)	-0.220***	(0.050)
WWII: non-belligerent	0.002	(0.073)	0.059	(0.073)
WWII: belligerent	0.070	(0.041)	-0.086*	(0.049)
High parental SES	0.061*	(0.033)	0.038	(0.042)
Absent father	-0.149***	(0.046)	-0.060	(0.045)
Famine	-0.107	(0.078)	-0.246***	(0.087)
Dispossessed	-0.109	(0.067)	-0.119*	(0.072)
GDP per capita	-0.004	(0.018)	0.021	(0.019)
$Var(U_C)$	0.464***	(0.026)	0.423***	(0.031)
$Cov(U_C^W, U_C^H)$		0.104***	(0.013)	

Sample size: $n = 5,964$. Significance: * = 10%; ** = 5%; *** = 1%

Table 4 shows the estimated impact of the characteristics and circumstances represented by the observed covariates, and also the latent autoregressive structure of the two partner's latent health during the two stages of adulthood. Note that interpretation of these parameters is not straightforward since different health indicators are observable for each life phase and the normalisations of the factor loadings at each phase are necessarily different. The parameters θ govern the cumulative nature of latent health. All these parameters are strongly significant with the expected positive sign, except for the insignificant negative estimated impact of childhood latent health on post-marital health for men.

Dynamic latent variable models have been used extensively in research on the economics of cognitive and non-cognitive human capital formation and in many studies of child development in other disciplines. In those contexts, it is generally safe to assume that indicators like test scores and measures of non-cognitive skills are sensitive to differences in the underlying

levels of latent skills current at the time of measurement. This is less clearly so for health. It is quite possible that a young person may have poor latent health which increases the risk of disease in later life, but is symptom-free during youth. If this is typical, indicators of health disorders in the early stages of life may be insensitive to the latent health states that will give rise to chronic disorders later in life, acting instead only as measures of vulnerability to transient childhood illness. We call this the *early invisibility* problem. If early invisibility is indeed a problem, it implies the existence of two dimensions of latent childhood health: (i) the factor Λ_1^p given by (4) which feeds forward to drive later health outcomes, but which has little or no impact on observable childhood indicators; (ii) a factor ξ_1^p, representing vulnerability to observable transient childhood illness, influenced by the childhood environment $\boldsymbol{X}_1^p$ but not important as an influence on adult health outcomes. The latent health state underlying observed health outcomes during marriage would then be:

$$\Lambda_3^p = \boldsymbol{X}_3^p \boldsymbol{\beta}_3^p + \Lambda_2^p \theta_{23}^p + \Lambda_1^p \theta_{13}^p + \xi_1^p \psi^p + U_3^p \tag{8}$$

where the long-term impact of transient childhood vulnerability is negligible: $\psi^p \approx 0$. Since Λ_1^p is not reflected in any observable indicator, θ_{13}^p is not identifiable. However, using (4) and (8), the partial reduced form equation is:

$$\Lambda_3^p = \boldsymbol{X}_3^p \boldsymbol{\beta}_3^p + \Lambda_2^p \theta_{23}^p + \boldsymbol{X}_1^p \beta_1^p \theta_{13}^p + \xi_1^p \psi^p + \left(U_3^p + U_1^p \theta_{13}^p\right) \tag{9}$$

Thus, if we estimate an extended model including both latent child health ξ_C^p and the childhood covariates $\boldsymbol{X}_1^p$ in the equations for latent adult health, the invisibility problem implies we should find little or no effect for the former ($\psi^p \approx 0$) but non-zero coefficients for the latter ($\boldsymbol{\beta}_1^p \theta_{13}^p \neq 0$). Table 2 shows the results of Wald variable-exclusion tests of the null hypothesis $\boldsymbol{\beta}_1^p \theta_{13}^p = 0$ and the analogous hypothesis for pre-marital adulthood. They are statistically insignificant for both men and women, while the autoregressive coefficients $\theta_{12}^p, \theta_{13}^p$ in the extended model[6] are almost unchanged in magnitude and statistical significance after including the additional covariates $\boldsymbol{X}_1^p$. Thus the observable childhood health measures available to us appear to be sensitive indicators of the relevant concept of latent health.

For pre- and post-marital adult phases, the covariates include: the length of the period, the length of any post-15 education, experience of physical injury, childbearing, smoking behavior, and experience of financial hardship or hunger during the period. During the

[6]Not reproduced here; available on request.

pre-marriage adult stage we find fewer significant country effects. Experience of a longer duration of hunger, a longer pre-marriage duration, having ever smoked and experiencing a physical injury have a significant negative effect on both men and women. Post-15 education has a significant positive effect for both men and women. Child-bearing prior to marriage has a significant negative effect for women, the magnitude of which declines with the age at childbirth. For men, smoking for a longer period of time had a significant positive effect.

Country differences are more evident during the post-marriage period for both men and women. Those who live in Austria/Germany/Switzerland, Spain, Italy, France, the Czech Republic and Poland have significant health disadvantages compared to those who live in Denmark. Time spent in financial hardship and the occurrence of physical injury both have a significant negative impact for both men and women. For women, there is again a significant negative impact of child-bearing, the effect declining in magnitude with age and the number of births. At least part of these fertility effects are likely to be reverse causation, since a woman's health state may affect her fertility. There is a significant negative effect of smoking which, for men, appears to decline with the duration of smoking. The latter perverse effect may be a consequence of smoking-related mortality.

Table 4 Latent variable model: partners' adult health

	Pre-marriage		Post-marriage	
	Women	Men	Women	Men
Childhood health (parameters $\theta^p_{12}, \theta^p_{13}$)	0.532***	0.653***	–0.020	0.079**
	(0.056)	(0.071)	(0.063)	(0.038)
Pre-marital adult health (parameter θ^p_{23})	-	-	0.737***	0.306***
			(0.119)	(0.044)
Austria/Germany/Switzerland	0.205	0.144	-0.336***	-0.241***
	(0.127)	(0.223)	(0.114)	(0.088)
Sweden	0.048	-0.005	-0.126	0.013
	(0.132)	(0.251)	(0.116)	(0.095)
Netherlands	0.183	0.189	-0.138	-0.150
	(0.139)	(0.234)	(0.121)	(0.093)
Spain	0.154	1.085***	-0.410**	-0.498***
	(0.223)	(0.417)	(0.187)	(0.155)
Italy	0.253	0.437	-0.442***	-0.317***
	(0.166)	(0.292)	(0.144)	(0.111)
France	-0.045	-0.019	-0.209*	-0.197**
	(0.132)	(0.239)	(0.116)	(0.096)
Belgium	0.054	0.160	-0.165	-0.081
	(0.130)	(0.067)	(0.113)	(0.093)
Czech Republic	0.353	0.296	-0.551***	-0.323***
	(0.243)	(0.339)	(0.187)	(0.113)
Poland	0.172	0.260	-0.737***	-0.574***
	(0.244)	(0.396)	(0.184)	(0.129)
Age finished education (if post-15)	0.025***	0.041***	-	-
	(0.006)	(0.010)		
Any births during period	-0.813***	-	-0.259**	-
	(0.270)		(0.129)	
Number of births during period	0.044	-	0.020*	-
	(0.044)		(0.011)	
Age at first birth (if during period)	0.028***	-	0.009**	-
	(0.011)		(0.004)	
Duration of financial hardship during period	0.005	0.010	-0.010***	-0.010***
	(0.004)	(0.008)	(0.002)	(0.002)
Duration of hunger during period	-0.022*	-0.047**	-0.003	-0.004
	(0.012)	(0.020)	(0.013)	(0.018)
Physical injury during period	-1.403***	-1.403***	-0.813***	-0.777***
	(0.197)	(0.197)	(0.042)	(0.036)
Duration of period	-0.036***	-0.038***	-0.022	0.009
	(0.008)	(0.012)	(0.031)	(0.031)
Ever smoked during period	-0.100**	-0.235***	-0.201	-0.403***
	(0.041)	(0.074)	(0.128)	(0.113)
Years smoked during period	0.006	0.018*	0.000	0.009***
	(0.005)	(0.009)	(0.004)	(0.003)
$Var(U)$	0.133***	0.511***	0.480***	0.497***
	(0.032)	(0.095)	(0.020)	(0.015)
$Cov(U^W_A, U^H_A)$	-0.016		0.146***	
	(0.019)		(0.011)	
Wald χ^2_{20} for exclusion of childhood covariates	18.36		22.02	
[P-value]	[0.563]		[0.339]	

Significance: * = 10%; ** = 5%; *** = 1%

5 Comorbidity or homogamy?

In our observational framework, 'true' causal comorbidity can only be distinguished from the effects of homogamy by examining the evolving relationship between the two partners' latent health through the sequence of life phases. The dynamic structure (4)-(6) implies that the final health state observed in the post-marital period can be decomposed into components arising from childhood, the pre-marital adult life phase and the post-marital phase. The variance matrix of the final health state of the two marital partners can be decomposed into two corresponding terms: one originating in the childhood and pre-marital adulthood phases, the other originating in the marital period. Appendix 1 gives details of this decomposition. The variance matrix of post-marital health can in turn be decomposed into variance arising from observable factors $\boldsymbol{X}_3^p$ and unobservable factors U_3^p.

The variance decomposition suggests that, for both men and women, pre-marital variation accounts for no more than 15% of the total. This finding is consistent with the view that the shared environment in adult life is at least as important as the childhood environment as a determinant of health outcomes, but there are, of course, large caveats related to data quality, health measurement and dynamic specification to be borne in mind.

Of the variation attributable to the marital period, 52% (women) to 73% (men) stems from unobservable variation. This unobservable heterogeneity displays a husband-wife correlation of around 0.3, which is slightly larger than the correlation (0.25) between the two partners' pre-marital latent health. Thus homogamy appears to be almost as strong in correlation terms as the shared marital environment.

Table 5 Sources of variance and correlation in latent health during marriage

	Intra-marital sources of variation		Pre-marital	All sources
	Unobservable	Observable		
	Variance decomposition			
Wife	0.480	0.311	0.126	0.917
Husband	0.497	0.081	0.100	0.678
	Between-partner correlation in latent health			
	0.299	-0.074	0.247	0.205

6 Conclusions

There is a large research literature on individual health over the lifecourse, but much less attention has been paid to the source of the strong association between health states of marital partners who may have spent most of their lives together in a shared family environment. This 'marital comorbidity' is important because it may affect the ability of couples to cope with care needs late in life and because it is testament to the role of shared adult family environment in determining later-life health.

But research on marital comorbidity is very challenging. It requires very long-term data on paired individuals, both pre- and post marriage, a set of health indicators which are adequate to reveal underlying health states, and statistical modelling methods powerful enough to distinguish homogamous association and true causal association of health health states.

In this paper, we have considered a number of theoretical arguments leading us to expect some causal link between health states of marital partners; set out the types of data source that might provide the data required to analyse the origins of marital comorbidity; and presented a simple illustrative analysis of long-term recall data from the pan-European SHARElife survey. That analysis suggested a large role for the shared marital environment as opposed to the early life conditions emphasised by much of the research literature.

However, it must be admitted that analysis of this kind rests on heroic assumptions about data quality and the ability of simple dynamic structures to capture the complex interactions that underlie the joint evolution of partners' health. It may be that this research area is simply too difficult ever to be addressed satisfactorily with available data and analysis methods; nevertheless, its potential importance makes it worth the effort.

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Appendix 1: Variance decomposition

The final health state (6) can be split into three components arising from periods $t = 1...3$:

$$\boldsymbol{\Lambda}_3 = \{\boldsymbol{X}_3\boldsymbol{B}_3 + \boldsymbol{U}_3\} + \{[\boldsymbol{X}_2\boldsymbol{B}_2 + \boldsymbol{U}_2]\,\boldsymbol{\Theta}_{23}\} + \{[\boldsymbol{X}_1\boldsymbol{B}_1 + \boldsymbol{U}_2]\,[\boldsymbol{\Theta}_{13} + \boldsymbol{\Theta}_{12}\boldsymbol{\Theta}_{23}]\} \tag{10}$$

where: $\boldsymbol{\Lambda}_3 = (\Lambda_3^H, \Lambda_3^W)$; $\boldsymbol{X}_t = \left(\boldsymbol{X}_t^H, \boldsymbol{X}_t^W\right)$; $\boldsymbol{B}_3$ is block-diagonal, with $\boldsymbol{\beta}_t^H$ and $\boldsymbol{\beta}_t^W$ on the diagonal; and $\boldsymbol{\Theta}_{st}$ is the diagonal matrix with $\theta_{st}^H, \theta_{st}^W$ on the diagonal (for each $ts = \{21\}, \{32\}, \{31\}$). The data covariance matrices are $\boldsymbol{M}_{st} = cov(\boldsymbol{X}_s, \boldsymbol{X}_t)$. The covariance matrix, $\boldsymbol{\Sigma}$, of $\boldsymbol{\Lambda}_3$ has six components:

$$\boldsymbol{\Sigma} = \boldsymbol{V}_{33} + \boldsymbol{V}_{22} + \boldsymbol{V}_{11} + [\boldsymbol{V}_{32} + \boldsymbol{V}_{32}'] + [\boldsymbol{V}_{31} + \boldsymbol{V}_{31}'] + [\boldsymbol{V}_{21} + \boldsymbol{V}_{21}'] \tag{11}$$

where:

$$\begin{aligned}
\boldsymbol{V}_{33} &= \boldsymbol{B}_3'\boldsymbol{M}_{33}\boldsymbol{B}_3 + \boldsymbol{\Omega}_{33} \\
\boldsymbol{V}_{22} &= \boldsymbol{\Theta}_{23}'\,[\boldsymbol{B}_2'\boldsymbol{M}_{22}\boldsymbol{B}_2 + \boldsymbol{\Omega}_{22}]\,\boldsymbol{\Theta}_{23} \\
\boldsymbol{V}_{11} &= [\boldsymbol{\Theta}_{13} + \boldsymbol{\Theta}_{12}\boldsymbol{\Theta}_{23}]'\,[\boldsymbol{B}_1'\boldsymbol{M}_{11}\boldsymbol{B}_1 + \boldsymbol{\Omega}_{11}]\,[\boldsymbol{\Theta}_{13} + \boldsymbol{\Theta}_{12}\boldsymbol{\Theta}_{23}] \\
\boldsymbol{V}_{32} &= \boldsymbol{B}_3'\boldsymbol{M}_{32}\boldsymbol{B}_2\boldsymbol{\Theta}_{23} \\
\boldsymbol{V}_{31} &= \boldsymbol{B}_3'\boldsymbol{M}_{31}\boldsymbol{B}_1\,[\boldsymbol{\Theta}_{13} + \boldsymbol{\Theta}_{12}\boldsymbol{\Theta}_{23}] \\
\boldsymbol{V}_{21} &= \boldsymbol{\Theta}_{23}'\boldsymbol{B}_2'\boldsymbol{W}_{21}\boldsymbol{B}_1\,[\boldsymbol{\Theta}_{13} + \boldsymbol{\Theta}_{12}\boldsymbol{\Theta}_{23}]
\end{aligned} \tag{12}$$

In Table 5 above, the variation arising from unobservable intra-marital factors is summarised by $\boldsymbol{\Omega}_{33}$; observable intra-marital variation is $\boldsymbol{B}_3'\boldsymbol{M}_{33}\boldsymbol{B}_3$ and variation driven by pre-marital factors is the remainder $\boldsymbol{\Sigma} - \boldsymbol{V}_{33}$, giving an additive decomposition of latent health variation.

Appendix 2: Additional Figures and Tables

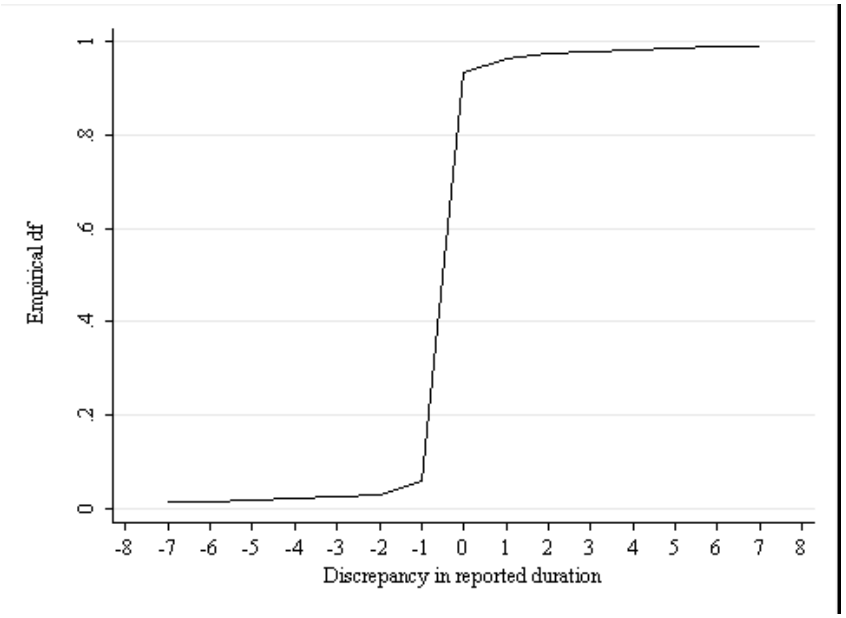

Figure A1 The distribution of mismatches between partners' reported relationship durations

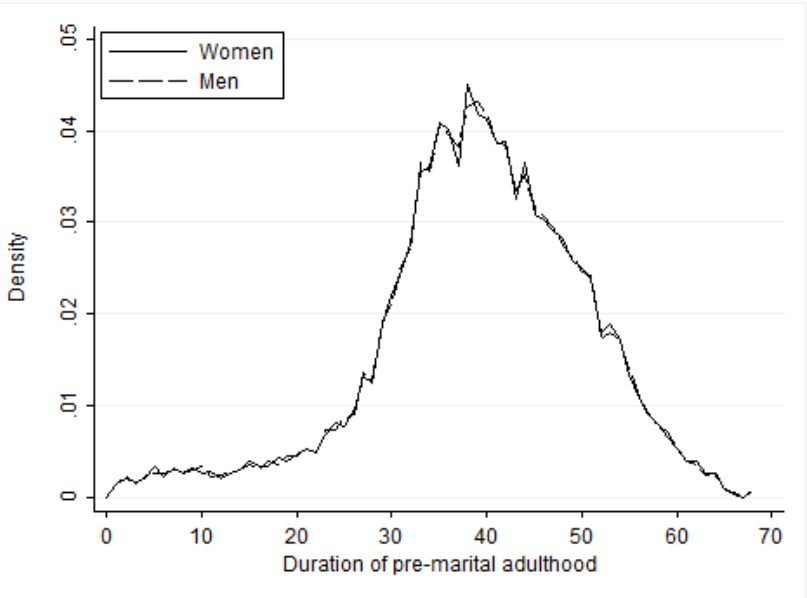

Figure A2 The distribution of reported marriage durations

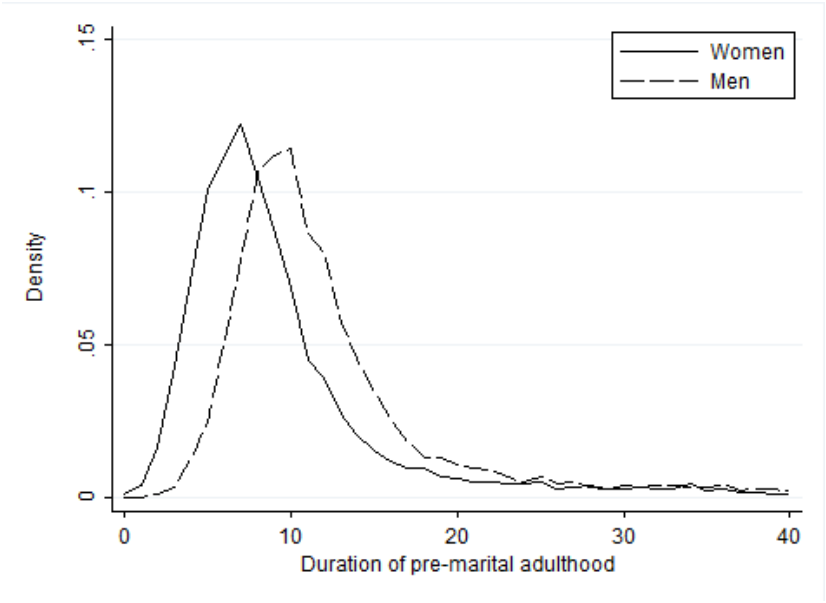

Figure A3 The distributions of durations of pre-marital adulthood

Table A1 Means of covariates[1]

	Men	Women
Childhood		
Rural Residence during childhood	0.555	0.547
Parents heavy drinking	0.079	0.082
Childhood in neutral country during WWII	0.091	0.080
Childhood in belligerent country during WWII	0.264	0.220
Professional/managerial parent	0.124	0.122
Famine exposure at birth[2]	0.037	0.037
Biological father absent at age 10	0.092	0.088
Family dispossessed at age 10	0.038	0.036
More than 1 move during childhood	0.672	0.660
Year of birth - 1900	41.798	44.783
Per capita GDP (US$) at age 10	5,324	5,882
Pre-partnership adulthood		
Length of pre-partnership adult period	12.740	9.760
Age finished full-time education (if after 16)	17.165	16.420
Physical injury during period	0.035	0.018
Any births during period	-	0.106
Number of births during period	-	0.196
Age at birth of first child (if during period)	-	2.251
Duration of financial hardship during period	0.722	0.678
Duration of hunger during period	0.436	0.293
Ever smoked during period	0.574	0.297
Number of years smoked during period	1.651	0.869
Partnership		
Length of marriage/partnership	39.196	39.187
Legally married	0.964	0.964
Age finished full-time education (if after 16)	17.333	16.666
Physical injury during marriage	0.123	0.093
Any births during marriage	-	0.865
Number of births during marriage	-	2.176
Age at birth of first child (if during marriage)	-	21.558
Duration of financial hardship during marriage	2.522	2.700
Duration of hunger during marriage	0.032	0.061
Ever smoked during marriage	0.197	0.136
Number of years smoked during marriage	6.045	3.969
Country		
Austria	0.030	0.030
Germany	0.080	0.080
Sweden	0.065	0.065
Netherlands	0.091	0.091
Spain	0.096	0.096
Italy	0.125	0.125
France	0.094	0.094
Swizterland	0.045	0.045
Belgium	0.118	0.118
Czech Republic	0.078	0.078
Poland	0.097	0.097
Denmark [3]	0.080	0.080

[1] $n = 6,999$. [2] Born in Netherlands in 1945 or Poland in 1941-6 or Austria or Germany in 1945-8. [3] Reference category

Table A2(*i*) Measurement model: childhood

Health indicator						
H_1	H_2	H_3	H_4	H_5	H_6	H_7
Factor loadings: women						
1	-.803	-.768	-.267	-.357	-.563	-.758
	(.046)	(.051)	(.030)	(.031)	(.040)	(.054)
Threshold parameters: women						
-2.047	.836	.865	-.707	.256	2.138	2.371
(.281)	(.494)	(.588)	(.320)	(.311)	(.393)	(.750)
-1.423			.970	1.760	3.171	
(.279)			(.320)	(.312)	(.394)	
-0.392			1.893	2.710	3.850	
(.279)			(.331)	(.316)	(.399)	
0.521						
(.279)						
Factor loadings: men						
1	-.934	-.831	-.230	-.324	-.480	-.708
	(.066)	(.065)	(.035)	(.034)	(.045)	(.071)
Threshold parameters: men						
-2.395	1.489	1.951	-.468	.334	1.797	2.903
(.294)	(.521)	(.541)	(.323)	(.311)	(.406)	(.925)
-1.712			1.150	1.756	2.875	
(.279)			(.322)	(.312)	(.408)	
-0.745			2.010	2.863	3.757	
(.293)			(.326)	(.324)	(.429)	
0.147						
(.293)						

Health indicators are ordered as in Table 1

Table A2(*ii*) Measurement model: pre-marital adulthood

Health indicator			
	H_1	H_2	H_3
Factor loadings: women	-1	-1.083	-1.165
		(0.131)	(0.131)
Threshold parameters: women	2.259	1.653	1.724
	(3.215)	(2.107)	(0.709)
	3.136		
	(3.330)		
Factor loadings: men	-1	-0.609	-0.658
		(0.071)	(0.073)
Threshold parameters: men	3.328	3.089	1.871
	(2.304)	(1.525)	(0.654)
	4.023		
	(2.387)		

Health indicators are ordered as in Table 1

Table A2(*iii*) Measurement model: post-marital adulthood

Health indicator						
H_1	H_2	H_3	H_4	H_5	H_6	H_7
Factor loadings: women						
1	.845	.916	-.644	-.641	-.759	-.029
	(.018)	(.019)	(.026)	(.024)	(.021)	(.014)
Threshold parameters: women						
-1.955	-2.335	-2.615	2.583	-.666	1.152	Intercept
(.276)	(.383)	(.292)	(.524)	(.435)	(.338)	1.566
-0.862	-1.194	-1.501	3.290			(1.660)
(.275)	(.382)	(.290)	(.528)			Variance
.351	.070	-.303	3.881			2.797
(.276)	(.382)	(.290)	(.530)			(.032)
1.151	.960	.576				
(.277)	(.383)	(.291)				
Factor loadings: men						
1	.899	.967	-.747	-.715	-.880	.003
	(.018)	(.018)	(.026)	(.024)	(.020)	(.008)
Threshold parameters: men						
-2.134	-1.765	-2.504	2.117	-.092	.996	Intercept
(.283)	(.385)	(.300)	(.524)	(.405)	(.338)	.590
-1.144	-0.705	-1.461	2.786			(2.994)
(.282)	(.385)	(.299)	(.527)			Variance
.020	.571	-.281	3.405			3.013
(.282)	(.385)	(.299)	(.530)			(.044)
0.811	1.357	.549				
(.283)	(.385)	(.300)				

Health indicators are ordered as in Table 1