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Research Article

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Research Article

Pathways from fertility history to later life health: Results from analyses of the English Longitudinal Study of Ageing

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Abstract

BACKGROUND
Previous research shows associations between fertility histories and later life health. The childless, those with large families, and those with a young age at entry to parenthood generally have higher mortality and worse health than parents of two or three children. These associations are hypothesised to reflect a range of biosocial influences, but underlying mechanisms are poorly understood.

OBJECTIVES
To identify pathways from fertility histories to later life health by examining mediation through health-related behaviours, social support and strain, and wealth. Additionally to examine mediation through allostatic load – an indicator of multisystem physical dysregulation, hypothesised to be an outcome of chronic stress.

METHODS
Associations between fertility histories, mediators, and outcomes were analysed using path models. Data were drawn from the English Longitudinal Study of Ageing. Outcomes studied were a measure of allostatic load based on 9 biomarkers and self-reported long-term illness which limited activities.

RESULTS
Early parenthood (<20 for women, <23 for men) was positively associated with higher (worse) allostatic load and long-term illness. These associations were partly mediated through wealth, smoking, and physical activity. Wealth, smoking, physical activity, and social strain also mediated associations between larger family size, itself associated with early parenthood, and health outcomes. We found no significant associations between childlessness and allostatic load or long-term illness, except for an association

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between childlessness and long-term illness among women in models adjusted only for age.

CONCLUSION
In England early parenthood and larger family size are associated with less wealth and poorer health behaviours and this accounts for much of the association with health. At least part of this operates through stress-related physiological dysfunction (allostatic load).

1. Introduction

Recognition of the importance of life-course influences on health and mortality at older ages has led to growing interest in the long-term consequences of reproductive pathways, particularly in the context of changing fertility patterns. Apart from the short- and long-term physiological influences of pregnancy, child birth, and lactation on women's health, broader biosocial mechanisms may link parenting trajectories with the later life health of both women and men. These include salutogenic influences of parenthood such as incentives to avoid health-damaging behaviours, role enhancement, greater community participation, and social support from children. However, parenthood also involves stresses of various kinds, including considerable economic costs, which may have health-damaging implications. In addition to the difficulty involved in unravelling effects of these countervailing influences, reproductive careers may be influenced by earlier health status and are associated with characteristics such as educational level and marital status, known to be related to health. Patterns of association observed and their interpretation may also depend on outcome measures used and age groups studied. Outcomes such as disability or death are usually sequelae of stresses, behaviours, and physiological changes accumulated over a long preceding period. Use of indicators of earlier pre-clinical changes in health may be helpful in elucidating the processes leading to these more distal outcomes. Allostatic load, conceptualised as multisystem physiological dysregulation arising from accumulated responses to multiple stressors, has been proposed as one such measure. In this paper we use path models within a structural equation-modelling framework to formally examine pathways from fertility histories to allostatic load and later life health, using longitudinal data from the English Longitudinal Survey of Ageing (ELSA). We investigate the extent to which associations are mediated by wealth, health-related behaviours, and social support and social strain in models controlling for socio-demographic characteristics and an indicator of respondents' health in childhood.

1.1 Previous research

1.1.1 Number of children

Most studies of contemporary populations have found a U- or J-shaped association between number of children born (parity) and later life mortality and health among women, and, in a few studies, among men, with both nulliparity and high parity (four, five, or more children) being associated with increased risks relative to having two children (Doblhammer 2000; Alonzo 2002; Grundy and Tomassini 2005; Hurt et al. 2006; Jaffe et al. 2009). However, in Scandinavian populations, which have generous social supports for parents, there seems to be no, or a reduced, mortality 'penalty' for high parity (Hinkula et al. 2006; Grundy and Kravdal 2008, 2010), indicating the importance of contextual factors.

Results from studies including outcomes other than or in addition to mortality show some variations that may reflect differences in measures, methodologies, and statistical power as well as contextual influences. Analyses of German survey data, for example, found that in Western Germany mothers and fathers of four or more children reported better health, but had no elevation of mortality risk, while in Eastern Germany high parity was associated with increased mortality risks among women, but not with poorer health (Hank 2010). UK studies of associations between parity and various indicators of health, like studies of mortality, generally show disadvantages associated with nulliparity, high parity, or both (Grundy and Holt 2000; Guralnik et al. 2009; Grundy and Tomassini 2005). A more recent British study including repeated health and disability measures found that childlessness was associated with faster accumulation of health limitation in women and that high parity was associated with poorer health outcomes for both women and men (Read, Grundy, and Wolf 2011). Results from studies of US populations are less consistent, possibly because of greater variation in the demographic composition of groups studied. Kington, Lillard, and Rogowski (1997), in a study of US women aged 50 and over, found that women with six or more completed pregnancies had worse health than women who were nulliparous or had had one or two children; similar results were reported in a recent study of Mexican American women (Aiken et al. 2012). However, Spence and Eberstein (2009), in analyses of a nationally representative US longitudinal study which included deaths up to age 80, reported protective effects of high parity, especially among Black women, once adult health and social characteristics were controlled for. More consistently with other studies, they also found higher mortality risks among nulliparous white women. Other studies of North American women have found no associations between parity and limitations in functioning (Moen, Dempster-McClain, and Williams 1992; Spence 2008) or, in one study which took account of childhood circumstances, mortality at ages 53−71 (Henretta 2007).

1.1.2 Age at parenthood

Studies of age at childbearing are consistent in showing poorer later health and higher mortality among women with an early entry to motherhood (Kalil and Kunz 2002; Grundy and Tomassini 2005; Mirowsky 2005; Henretta 2007; Henretta et al. 2008; Patel and Sen 2012; Pirkle et al. 2014), although some studies indicate a different association among Black women in the USA (Spence and Eberstein 2009). Later health disadvantages of early fatherhood have also been reported (Grundy and Tomassini 2006; Grundy and Kravdal 2008; Pudrovska and Carr 2009). Results on possible effects of having children at relatively old ages are mixed. Some studies suggest that late maternity or paternity is related to better later health (Snowdon et al. 1989; Yi and Vaupel 2004; Grundy and Tomassini 2005). However, other studies of US women have found no effect (Spence 2008) or have indicated poorer health among those having children at relatively mature ages (Alonzo 2002; Mirowsky 2002). Anlyses of mortality using Norwegian register data show an inverse association between later age at last birth and later mortality for all causes and for most specific causes of death, other than breast cancer in women (Grundy and Kravdal 2010).

1.2 Hypothesised underlying mechanisms

The hypothesised causal pathways that underlie associations between reproductive histories and later health are complex. Physiological changes associated with pregnancy, childbirth, and lactation have a number of effects on women's risks of developing various specific conditions. Nulliparity and late childbearing are associated with higher risks of breast cancer (Kvale et al. 1994; Madigan et al. 1995; Grundy and Kravdal 2010). Higher parity is also protective against some other hormonally related cancers but appears to increase risks of diabetes, obesity, degenerative changes in arterial walls, and ischemic heart disease, although reasons for this are not fully understood and may include lifestyle changes associated with parenthood (Friedlander 1996; Lawlor et al. 2003; Bastian et al. 2005).

The broader biosocial effects of parenthood include both positive and negative effects that apply to men as well as women. A beneficial influence of parenthood may be the greater incentive to adopt a healthier life style and the social control of unhealthy behaviours provided by children (Umberson, Crosnoe, and Reczek 2010). A review of survey evidence undertaken by Kendig et al. (2007) suggested that older parents generally had healthier life styles than childless older people. Further evidence of a beneficial effect of parenthood on health-related behaviours comes from studies of cause-specific mortality. Analyses of Norwegian data, for example, show an inverse

association between number of children and risks of death from alcohol-related causes for women and men (Grundy and Kravdal 2010).

Other suggested health benefits of parenthood are through role enhancement and increased social participation and social support. Parenthood may enhance community participation (Furstenberg 2005; Knoster and Eggebeen 2006; Offer and Schneider 2007) and in later life parents may be able to draw on social support from children, with its known health benefits (Zunzunegui et al. 2001; Antonucci, Ajrouch, and Janevic 2003; Barefoot et al. 2005; Krause 2007). Older childless people, particularly childless men, appear to have less social interaction overall than older parents and there is evidence that a larger number of children, and having a daughter, is associated with an increased chance of regular social contact and with receiving help if needed (Tomassini et al. 2004; Dykstra and Hagestad 2007; Grundy and Read 2012).

Potentially adverse effects of parenthood include the stresses of rearing children, the narrowing of opportunities for fulfilment in other roles and potential role overload, substantial economic costs, and, in the case of those becoming parents at young ages, possible disruption of educational and career trajectories (Joshi 2002; Moffit et al. 2002; Evenson and Simon 2005; Pudrovska and Carr 2009; Dariotis et al. 2011; Wolf et al. 2011). The extent and effect of these stresses may vary according to parenting trajectories and other circumstances, for example partnership status and financial resources. Additionally, not all life-style changes associated with parenthood are beneficial and parenthood and higher parity are associated with weight gain and obesity in American and some other populations (Umberson et al. 2011).

1.2.1 Methodological issues

Inferences drawn about possible causal associations between parenting histories and later life health clearly need to take account of possible confounding factors. Childhood circumstances and early adult health status and health-related behaviours may themselves influence partnership, fecundity, and decisions about fertility. On the one hand, serious health limitations or disability in early life may restrict opportunities for partnership and parenthood (Kiernan 1989) and lifestyle-related factors such as heavy alcohol consumption and obesity may also influence partnership trajectories and are known to reduce fecundity, as well as being associated with health (Sallmen et al. 2006). On the other hand, numerous studies have shown that childhood disadvantages of various kinds, including poorer physical or mental health, are strongly associated with early parenthood (Hobcraft and Kiernan 2001; Henretta et al. 2008; Hobcraft 2008; Mollborn and Morningstar 2009), itself associated with higher overall parity. Educational status is associated both with fertility trajectories and with health and

mortality, as are marital biographies (Kravdal et al. 2012). In both cases these variables may theoretically operate as confounders, mediators, or moderators of associations between fertility histories and later life health - or indeed may be spuriously related because of both being associated with some other factor, such as childhood circumstances. Directions of associations may also be hard to disentangle; for example, educational level and timing of parenthood may be associated because in some cases early parenthood may disrupt education or because low educational attainment may lead to behavioural choices resulting in early parenthood. Similarly, pregnancy may either prompt or be consequent on marriage, and both timing and level of fertility may influence risks of subsequent marital breakdown (Koropeckyj-Cox, Pienta, and Brown 2007; Pudrovska and Carr 2009). Associations between late parenthood and subsequent health may also be influenced by selection, as those with poor health in mid-life may be less able or inclined to have a(nother) child (Snowdon et al. 1989; Yi and Vaupel 2004). The direction and magnitude of associations observed may also vary according to the outcome considered and the age at which it is observed. For example, earlier fertility and higher parity reduce women's risks of breast cancer but may increase risks from cardiovascular disease. The former constitutes a greater proportion of all-cause mortality in late middle age than in the oldest age groups, so associations between parity and female mortality could plausibly vary by age group studied. Additionally, those in the oldest age groups represent a smaller and more selected proportion of relevant birth cohorts and survival to these more advanced ages may indicate greater resilience to any health-damaging effects of particular fertility histories, as well as to other risks.

2. Research aims

The principal aim of this paper is to examine the extent to which associations between parenting histories and health in later life are mediated by three domains hypothesised to be influenced by parenting pathways and known to be associated with health. These are health-related behaviours (smoking and physical activity); social support and social strain; and wealth. A second objective is to see whether associations between fertility histories and reported health limitation are mediated by allostatic load – an indicator of cumulative physiological response to stress based on composite biomarker measures (see section 2.1). As rearing children is costly in economic terms, we expected that wealth would be an important mediator of associations between larger family size and health outcomes. We also expected that a higher number of children would be positively associated with both social support and social strain, with the former having beneficial and the latter adverse associations with health. On the basis of the previous

literature, we hypothesised that health-related behaviours would be most important in mediating associations between childlessness and later health and also, among parents, between early entry to parenthood and health. We hypothesised that all effects of fertility histories on health would be evident in associations with allostatic load and that these would mediate associations with later self-reported health limitation.

2.1 Allostatic load

Allostatic load has been conceptualised as multisystem physical dysregulation resulting from cumulative effects of responding to multiple stressors and operating through the functioning of several regulatory systems (McEwen and Stellar 1993; Todorova et al. 2013). In early life stress-responsive systems are initiated that produce hormones that maintain the soma through continual allostatic responses. Later in life, systems designed to mitigate stressors may weaken, leading to harmful dysregulation. Consequent wear and tear across multiple physiological systems is a significant contributor to overall health risk (Seeman et al. 1997, 2001). Such wear and tear is hypothesized to ensue, at least in part, from repeated exposure to life challenges, which may include social relational conflict or adversity (Seeman et al. 2002). A recent review of 2005−10 publications which examined relationships between allostatic load and risk factors, chronic diseases, morbidity, and mortality in samples of older people concluded that allostatic load captured aspects of physiological dysregulation and somatic decline and was predictive of health deterioration (Leahy and Crews 2012). Goldman and colleagues (2006), for example, found that allostatic load provided an early indicator of later poor health in a three-year study of the Taiwanese older population. Similarly, in a study of a US cohort, allostatic load level predicted frailty in a three-year follow-up among older people (Gruenewald et al. 2009).

2.1.1 Measurement of allostatic load

Allostatic load is measured using batteries of biomarkers that tap into the major mediators of allostasis including key primary mediators such as cortisol, epinephrine, and norepinephrine, and secondary outcomes such as body mass index or waist–hip ratio and glycosylated haemoglobin (McEwen and Wingfield 2003; Seplaki et al. 2005). Use of a summary measure provides a broad-based indicator of the dysregulation of these adaptive systems and has been found to be a better predictor of later health than any individual biomarker (Seeman et al. 2001; Karlamanga et al. 2002). The number and composition of biomarkers used to derive allostatic load varies between studies, but

has generally been in the range of 9−14 (Seplaki et al. 2005; Juster, McEwen, and Lupien 2010). Comparisons of different composite measures of allostatic load have shown relatively small differences in how powerful they are for predicting health outcomes (Seplaki et al. 2005). Composite allostatic load scores are usually based on a simple count of the number of biomarkers for which the individual falls above (or below) relevant cut points. Thus, each point on the allostatic load measure indicates that the respondent's value for that measure was not within the healthy range. Cut points can be based on clinical thresholds but, as there is no clear agreement about what these should be in different populations, it is more usual to base them on the distribution of the measure in the sample studied (Juster et al. 2010).

3. Data and methods

3.1 Data

We use data from waves 1-3 of the English Longitudinal Study of Ageing (ELSA), a nationally representative longitudinal study of the older population of England. The first wave of ELSA, conducted in 2002−2003, included men and women then aged 50 years or more from households which had participated in any one of the 1998, 1999, or 2001 rounds of the cross-sectional Health Survey for England (HSE). Response rates for the HSE were 69% in 1998, 70% in 1999, and 67% in 2001. This process led to the recruitment of 11,392 core members to the first wave of ELSA (response rate 67%). Comparisons with other sources, including the census, showed that the baseline ELSA survey was nationally representative (Marmot et al. 2003). The sample has been followed up at two-yearly intervals. 70% of those interviewed in wave 1 also participated in waves 2 (2004−5) and 3 (2006−7) (Banks et al. 2008). Certain core information has been collected in every ELSA wave. In alternate waves the survey includes a nurse visit when more detailed data, including physical measurements and blood samples, are gathered (de Oliviera et al. 2010). Wave 3 included a retrospective life course interview which collected information about childhood and adult life prior to joining the study.

A pre-requisite of inclusion in ELSA for those in relevant birth cohorts (born 1952 or earlier) was obviously survival to 2002/3 and, as already noted, this means that those in different age groups represent different fractions of their birth cohorts. Additionally, the fertility patterns of cohorts born in England and Wales in the first half of the 20th century varied quite considerably, with early parenthood and two child families being more common for later born cohorts whereas childlessness and one child families, but also large family sizes, were more common in earlier born cohorts. These differences in

survivorship and in female fertility for relevant birth cohorts for the whole population of England and Wales are illustrated in Table 1.

Table 1: Cohort indicators of survival to older ages (males and females) and of fertility (females), England and Wales, cohorts born 1917−1952 (aged 50−85 in 2002)

Cohort survival to age groups represented in ELSA				Completed fertility and proportions with births at ages <20, female cohorts represented in ELSA						
Year of birth	% surviving to age:	Men	Women	Year of birth	% with birth <20	Distribution (%) by completed family size at age 45				
						0	1	2	3	4+
1917	85	15	30	1920	7	21	21	27	16	15
1922	80	30	47	1925	7	17	22	28	17	16
1927	75	47	61	1930	9	13	18	30	19	20
1932	70	62	73	1935	9	12	15	32	21	20
1937	65	74	81	1940	13	11	13	36	22	18
1942	60	82	87	1945	17	10	14	43	21	12
1952	50	91	94	1950	20	14	13	44	20	10

Sources: Office for National Statistics 2010-based National Population Projections lifetable template, Demographic Analysis Unit, 2011. (Crown Copyright)
National Statistics Cohort fertility England and Wales , Tables 2 and 3, 2011; (Crown Copyright) released 07 March 2013. http://www.ons.gov.uk/ons/publications/re-reference-tables.html?edition=tcm%3A77-263133

3.1.1 Analysis sample

Our starting sample comprised the 11,233 core members who participated in wave 1 of ELSA and provided personal interviews (excluding those with proxy responses). All of these respondents provided information on age (and on number of children) and, as shown in Table 2, 90% had complete data for other wave 1 items used in the analysis. Measures used include those collected in waves 2 and 3 of the study and for these there was a more serious problem of missing data due to attrition from the study and failure to provide complete information or, more particularly, the blood samples and physical measurements required to derive the measure of allostatic load. Only 154 (1%) of the initial sample of respondents had died by wave 2; a further 350 (3%) died between waves 2 and 3. However, as shown in Table 2, interview items at wave 2 were available for only 8,651 of the initial sample (77%). Of these, 6,171 (71%) participated in the measurements needed to calculate allostatic load. By wave 3, 7,360 (66%) of the initial sample provided interview data but only 6,187 also completed the separate life history schedule. Strategies and methods used to deal with the problem of missing data are explained in section 3.3.

Table 2: Completeness of data for variables used in the analysis, ELSA waves 1−3

Wave/variables	N	%
Wave 1 starting sample[a]	11233	100
All interview items available at Wave 1	10133	90.2
Present at Wave 2 (all had all interview items)	8652	77.0
Present at Wave 3 (all had all interview items)	7360	65.5
Complete interview data[b] from Waves 1, 2 and 3	6714	59.8
Allostatic load available at Wave 2	6171	54.9
Life history data available at Wave 3 (childhood health)	6187	55.1
All data available except allostatic load	5694	50.7
All data available except childhood health	5060	45.0
All data available	4377	39.0

Note: [a] In-person interviewed sample core members.
[b] Complete data from main interview schedule, i.e., not necessarily complete for the life history module.

3.2 Measures

3.2.1 Health measures

Two health measures were used, long-term illness that limited activities reported at wave 3 (0 = no limiting long-term illness; 1= has limiting long-term illness) and an indicator of allostatic load derived from biomarker data collected in the nurse visit at wave 2. Nine biomarkers were used to create the allostatic load score. These comprised waist to hip ratio, systolic and diastolic blood pressure, and lung function, all measured by nurses, and five indicators derived from blood samples. Details of how these were measured are outlined below and have also been reported elsewhere (Read and Grundy 2014).

Waist to hip ratio. Waist circumference was measured at the mid-point between the lower rib and the upper margin of the iliac crest and hip circumference was measured at maximal buttocks. The measures were taken twice, using the same tape, and recorded to the nearest even millimetre. The mean of the two valid measures was used. 74 individuals refused waist and hip measurement.

Blood pressure was measured using three readings collected at one-minute intervals. The mean of valid readings was used. Respondents were asked not to eat,

smoke, drink alcohol, or take vigorous exercise 30 minutes before the blood pressure measure was taken. Eleven individuals refused blood pressure measurement.

Lung function was measured using the Vitalograph Micro Spirometer. Three measures were taken of peak expiratory flow (the fastest rate of exhalation in litres per minute) with the highest used as the valid one. Seventy respondents declined to participate and, in accordance with study protocols, the lung function of a further 169 respondents was not measured because the respondent had had abdominal, tracheotomy, or chest surgery in the preceding three weeks, had been admitted to hospital with a heart complaint in the preceding six weeks, had had eye surgery in the preceding four weeks, or the room temperature was less than 15 or more than 35 degrees Celsius.

The remaining five biomarkers were derived from blood samples collected at wave 2: HDL/total cholesterol ratio (mg/dL) (index of risk for cardiovascular disease); triglycerides (index of lipid metabolism); glycosylated haemoglobin (HbA1c, %) (index of glucose metabolism over the previous 30−90 days); fibrinogen (index of inflammation and cardiovascular disease, mg/dL); and C-reactive protein (index of inflammation and cardiovascular disease, mg/dL). We were not able to include a measure of cortisol, often included in derivation of allostatic load, as although this was collected, data are not available for analysis. Blood samples were not taken from 596 people who failed to give written consent and a further 472 respondents who had a clotting or bleeding disorder, had ever had a fit, or were on anti-coagulant medication. Fasting blood samples were taken whenever possible, but those over age 80 and those who had medical conditions were not asked to fast. About 45% of the sample had fasted for at least 5 hours before the blood sample was taken. We controlled for possible variation in the measure due to variable fasting by deriving three binary variables (no fasting, respondent had fasted between 5 and 8 hours, and respondent has fasted for more than 8 hours before the blood sample was taken) which were included as co-variates in the models.

For all nine measures, individuals belonging to the highest 25 percentile indicating the health risk were identified from the sample distribution. A similar method of identifying risk quartiles has frequently been used in creating composite allostatic load scores (Juster, McEwen, and Lupien 2010; Seeman, Singer, Rowe, Horwitz, and McEwen 1997).

This was done separately for men and women in age groups 50−64 and 65+. This procedure does not control for age completely, but makes it possible to present cut-offs by gender and age group. Account was taken of use of medication, which could change biochemical values, so that individuals were coded as 1 (indicating health risk) for diastolic and systolic blood pressure if they used blood pressure-lowering medication; for fibrinogen if they used anticoagulants; for triglycerides and HDL cholesterol ratio if they used cholesterol medication; for glycosylated haemoglobin if they used diabetes

medication; and for peak expiratory flow if they used lung medication. The literature suggests that diabetic, cholesterol, and blood pressure-lowering medications reduce the values of C-reactive protein by 25%-30%, so those with values in the second highest 25 percentile were given the value of 1 on this indicator (Prasad 2006). Finally, use of an inhaler or puffer 24 hours prior to the nurse visit was also used as a covariate in the analysis, as this could influence peak expiratory flow.

A mean score for each of five subsystems (inflammation, cardiovascular, metabolic, body fat, and respiratory) was calculated on the basis of the number of biomarkers where the individual belonged to the risk group. This was done to weight the score by the number of biomarkers in the five systems. The weighted score was rounded to the nearest tenth so it could be used as an ordered categorical variable. Because of a very low number of values over 0.8, the highest category included all values between 0.8 and 1.0. Information on at least 5 out of 9 biomarkers had to be available to calculate the score. The high-risk cut-offs (shown in the Appendix Table) were similar to those found in previous studies (Juster, McEwen, and Lupien 2010; Seeman et al. 1997).

3.2.2 Fertility and parenthood history variables

We derived five binary variables indicating whether respondents had had 0, 1, 2, 3, or 4+ natural living children and further dichotomous variables indicating whether they had any adopted or step-children. For parents, additional dichotomous variables were derived indicating whether or not respondents had ever experienced the death of a natural child, had had a biological child before the age of 20 (women) or 23 (men), or after age 34 (women) or 39 (men). These cut points were chosen on the basis of the relevant previous literature and the distribution observed in the sample (Hobcraft 2008; Read, Grundy, and Wolf 2011). All measures were derived from wave 1, except the item on experience of death of a child, which was obtained from the wave 3 life-history interview.

3.2.3 Intermediate variables

The hypothesised mediators investigated were wealth, social support and social strain, and health-related behaviours, all derived from information collected in wave 1.

Wealth. Wealth represents the lifetime accumulation of resources and has been shown to be a better indicator of economic status in older age groups than income (Banks et al. 2008). Respondents were asked to report all financial wealth (savings and

investments) and estimate the value of other assets including housing, cars, and valuables such as jewellery and antiques (Banks et al. 2008). The summed value of these, net of debts, was divided into quintiles and treated as a continuous variable in the analysis**.**

Health-related behaviour. We included measures of smoking and physical activity, both of which are strongly associated with health outcomes and are also hypothesised to be influenced by parenthood history. As these variables are used as mediators, we needed to be sure that they related to behaviours which followed rather than preceded fertility careers, and measures used related to current behaviours (at wave 1) rather than over the life course. The questions on smoking allowed identification of former as well as current smokers; however, in the modelling this was dichotomised (current smoker versus current non-smoker) because associations with health outcomes were similar for current and ex-smokers. Self-reported physical activity was used to create four categories: sedentary (no physical activity and, if working, in a sedentary job); low (mild physical activity at least once a week or if working in a job that was mostly standing); moderate (moderate physical activity at least once a week or if working in a job that involved physical work); and high (vigorous physical activity at least once a week or if working in a job that involved heavy manual labour). As the distribution of physical activity was approximately normal and the association with outcomes linear, this measure was treated as continuous in the analysis.

Social support and social strain. Mean scores of perceived social support and social strain from partner, children, relatives, and friends were derived from three questions asked about each. The social support questions were: 'How much do they [partner, children, relatives, friends] really understand the way you feel about things?' 'How much can you rely on them if you have a serious problem?' 'How much can you open up to them if you need to talk about your worries?' The indicator of social strain was based on responses to the questions: 'How much do they criticise you?' 'How much do they let you down when you are counting on them?' 'How much do they get on your nerves?' The mean was calculated using the items that were available. The internal consistency of the scales for both social support and strain was good (Cronbach's alpha=0.87). People reporting having no partner, children, relatives, or friends (n = 10 in wave 1) were given a missing value.

3.2.4 Other co-variates

We included in the analysis age (in single years) and co-variates known to be associated with fertility and with health, namely educational and marital status, co-residence with children, and childhood health status. In preliminary analysis (not shown) we also

included a measure of age squared to test for a non-linear association between age and the health outcomes. However, this was not associated with either outcome and so was not included in the models presented here.

In the cohorts included in this analysis most people left school at the statutory minimum age (14 or 15 from 1947 to 1972) and proportions with advanced secondary or tertiary education were very low. We therefore dichotomised the education variable distinguishing respondents with any formal qualification from those with no qualifications; as there were minimal changes in this over time we used education as reported in wave 1. Marital status was dichotomised (married versus non-married) due to small numbers in some sub-groups. In addition, a variable indicating if the respondent had experienced marital termination by wave 1 (at that point remarried, separated, divorced, or widowed) was included. For parents we included a variable indicating whether or not they were co-resident with a child, as co-residence is associated with both fertility characteristics and with reported stress (Read and Grundy 2010). Both marital status and co-residence with children were included as time-varying co-variates.

We included an indicator of health in childhood, as previous studies have shown this to be associated with fertility histories and with later life health. This was derived from retrospective information collected in the wave 3 interview. Those reporting missing school, being in hospital or confined to bed for more than a month due to illness, or that poor health had restricted physical activities for more than three months in childhood were coded as having had a childhood health problem.

3.3 Analysis

The analysis was undertaken using path models within the structural equation-modelling framework using Mplus version 5.21 (Muthén and Muthén 2007). In preliminary analysis we established that predictor, outcome, and intermediate variables were associated with each other, which is a pre-requirement of modelling designed to assess mediation (Muthén and Muthén 2007). We firstly examined direct associations between fertility and parenting variables and health outcomes by fitting logistic (limiting long-term illness) or ordered logistic regression (allostatic load) models as appropriate for the outcome variable. We then examined associations between fertility history variables and the intermediate variables by fitting logistic (smoking) or linear regression (wealth, physical activity, social support, social strain) models. This showed that having an adopted or stepchild or having experienced the death of a child were not associated with mediators and outcomes so they were not included in the final models, although shown in preliminary analyses. Finally, we used path models to examine

whether associations between fertility history variables and outcomes were mediated by the intermediate variables, and whether allostatic load at wave 2 mediated associations with long-term illness at wave 3. In this step all intermediate factors and fertility history items were entered into the model simultaneously so results shown are fully adjusted. Full mediation was evident if the direct path between fertility history and health outcome ceased to be significant when the path through the mediator was added. All paths remaining significant suggested partial mediation. All models were fitted using maximum likelihood estimation with robust standard errors (MLR) in Mplus to take into account any non-normality in the sample.

3.3.1 Handling of missing data

Complete case or available case (list deletion) analysis is a common way of dealing with missing data but may lead to biased estimates, especially if more than 10% of data are missing (Pigott 2001; He 2010; White and Carlin 2010). This is because complete case analysis involves the assumption that data are missing completely at random (MCAR) and that missingness is not related to observed or unobserved measurements. This might arise if, for example, a proportion of survey or laboratory results were lost due to some random staff or equipment failure. More usually missingness is related to some characteristic of the study sample, such as age or health status, and the MCAR assumption is invalid (Pigott 2001; He 2010). In these circumstances principled model-based methods are now recognised as the appropriate way of addressing missing data issues (Enders 2010; van Buuren 2012; Acock 2005; Enders and Bandalos 2001; Graham 2009, 2012; Pigott 2001). The two widely used model-based approaches, Multiple Imputation (MI) and Full Information Maximum Likelihood (FIML), share a common assumption that data are Missing at Random (MAR), rather than MCAR; that is, that data are missing for reasons related to variables that are completely observed. This is equivalent to saying that the behaviour of two units which share observed values has the same statistical behaviour on other observations, whether observed or not; i.e., it is assumed that systematic missingness is due to variables included in the model and that other missingness is at random (Little and Rubin 2002). When correlates of reasons for missingness are measured and included in the analysis or imputation model, FIML and MI yield unbiased estimates of parameters and their standard errors (Little et al. 2013). The two approaches differ somewhat in that MI produces results for individual missing variables whereas FIML produces estimates for means and the variance-covariance matrix and uses these to obtain model parameters; however, results are generally very similar (Acock 2005; Little et al. 2013). Here we use the FIML approach, which is particularly suitable when using complex datasets because it fits the

hypothesized model and takes into account missingness in a single run. Maximum likelihood estimation in Mplus is available for a range of different types of variable, including continuous, binary, ordered categorical, and counts. As noted above, the assumption is that if all the variables that are responsible for the missing data-generating mechanism are included in the model, then this can be ignored and parameter estimates robustly computed for participants with missing data.

Full maximum likelihood Mplus can model covariate, mediator, and outcome missingness if the variables that are correlated with missingness and/or include missingness are brought into the model (i.e., they are dependent variables). To bring into the model all covariates, age (a variable with complete data) was used as a predictor for all variables. In order to compare the full maximum likelihood strategy with case deletion we also estimated models for the complete case sample (shown in Figures) and for samples restricted to those with complete information on allostatic load or complete information on childhood health (not shown).

4. Results

4.1 Descriptive results

Tables 3 and 4 show the distribution of the sample by demographic and fertility history variables used in the analysis. We show distributions and base numbers for sample members with information available on the variable in question, although, as explained in the preceding section, those with missing data are also included in the model estimation. Table 3 shows that 37% of men and 48% of women had no educational qualifications and about a third of men and nearly half of women had experienced marital disruption through divorce or widowhood by the first wave of the study. As would be expected, the proportions currently married fell over time and were higher among men than women. Co-residence with children also became less usual in successive survey waves as parents (and children) became older. 30% of respondents met the criteria of having had poor health in childhood.

Table 3: Distribution of the sample by demographic and life history variables (% or mean and SD)

Variable	Men	*N*	Women	*N*
Age, wave 1	64.8 (10.04)	5110	65.5 (10.62)	6123
No qualification, wave 1	36.6	5099	47.6	6115
Married, wave 1	75.5	5109	59.1	6122
Married, wave 2	75.3	3884	57.2	4767
Married, wave 3	74.3	3276	55.4	4084
Marital termination (by wave 1)	31.0	5109	45.2	6122
Co-resident with child, wave 1[a]	25.9	4256	21.7	5219
Co-resident with child, wave 2[a]	21.8	3259	18.4	4092
Co-resident with child, wave 3[a]	19.8	2749	16.4	3497
Poor health in childhood[b]	29.6	2750	29.4	3437

Note: [a] Only among parents; [b] Data collected in wave 3 life history interview.

Table 4 presents similar descriptive information on the distribution of the sample by parenthood history. There were quite large differences between age groups in the distribution by parity and in proportions with early or late births, reflecting the changing patterns of fertility over the 20th century discussed earlier. Comparison with the national data (Table 1) suggests a slight under-representation in ELSA of teenage and high-parity mothers: however, the national data relate to characteristics at age 45. Information on the intermediate variables and health outcomes used in the analysis is presented in Table 5, which shows that 18% of sample members were current smokers and that over a third reported a long-term illness that limited activities at wave 3; this proportion was slightly higher among women than men.

Table 4: Distribution of the fertility history variables by age group (% or mean and SD)

	Men aged						Women aged					
Fertility history	50-64	*N*	65-79	*N*	80+	*N*	50-64	*N*	65-79	*N*	80+	*N*
Number of natural children		2691		1949		470		3111		2275		737
0	17.7		15.3		17.7		12.5		16.0		20.8	
1	14.0		15.2		23.8		14.4		14.9		25.1	
2	41.9		36.3		31.5		42.4		33.9		30.4	
3	17.1		18.6		15.0		20.5		20.0		13.4	
4+	9.3		14.6		12.1		10.2		15.2		10.3	
Early parenthood [a]	19.3	2120	11.7	1536	5.6	357	15.1	2623	7.1	1831	5.3	550
Late parenthood [a]	10.9	2121	12.1	1534	20.2	357	10.4	2624	16.4	1831	23.8	551
Adopted child	2.0	2691	3.0	1949	3.0	470	1.4	3111	2.7	2275	2.2	737
Stepchild	11.3	2691	7.6	1949	6.0	470	9.8	3111	4.6	2275	2.3	737
Child died [a,b]	3.8	1303	7.7	888	8.2	122	4.7	1670	9.1	1082	18.9	201

Note: [a] Only among biological parents, early: <20 for women, <23 for men; late <34 for women, <39 for men.
[b] Data collected in wave 3 life history interview. *N* represents denominator of the percentage shown.

Complete data for every variable used in the analysis were available for 1,996 men and 2,381 women. The complete case sample had lower proportions with no educational qualifications or with four or more children and higher proportions that were married or belonged to a higher wealth quintile. Completeness of data was also associated with lower (better) allostatic load scores, less limiting long-term illness, higher physical activity, and less current smoking. These differences in the characteristics of the sample sub-set with complete data are consistent with well-known higher rates of survey attrition and non-response among those of lower socio-economic status and those in poorer health, which also applied to ELSA (Scholes et al. 2008). As already discussed, estimation was carried out under missing-data theory using all available information.

Table 5: Distribution of the sample by intermediate variables and health outcomes (% or mean and SD)

Variables	Men	*N*	Women	*N*
Intermediate variables				
Wealth, wave 1	3.1 (1.43)	5076	3.0 (1.40)	6058
Physical activity, wave 1	2.0 (0.87)	5104	1.9 (0.88)	5583
Smoking, wave 1		5104		6115
Never smoked	26.0		43.6	
Ex-smoker	56.6		38.3	
Current smoker	17.5		18.1	
Perceived social support, wave 1	4.3 (0.53)	4661	4.3 (0.51)	5583
Perceived social strain, wave 1	2.7 (0.45)	4655	2.6 (0.46)	5570
Health outcomes				
Allostatic load weighted mean score, wave 2		2835		3336
<0.1	17.1		16.9	
0.1	14.5		15.6	
0.2	19.2		18.9	
0.3	15.0		15.5	
0.4	12.5		11.7	
0.5	10.3		9.4	
0.6	5.0		5.7	
0.7	3.6		4.3	
0.8-1.0	2.7		1.9	
Limiting long-term illness, wave 3	33.6	3274	37.9	4080

4.2 Fertility history, allostatic load, and long-term illness: direct associations

Direct associations between fertility and parenting history and allostatic load and long-term illness are shown for all men and all women and for parous men and women (those with any natural children) in Tables 6a and 6b. Two models are presented, one showing coefficients adjusted for age and parenthood history, the other adjusted for all the socio-demographic and life history variables (but not for intermediate variables). A positive sign for the coefficients shown indicates that the probability of the ordered categorical (allostatic load) or binary (limiting long-term illness) outcome increases when the predictor value increases. A larger magnitude indicates a greater increase in this probability. Negative values indicate an inverse association between the predictor and dependent variables.

Table 6a: Coefficents from maximum likelihood regression models of associations between fertility history and health outcomes in men (n = 5110)

	Allostatic load, Wave 2				Limiting long-term illness, Wave 3			
	Model 1		Model 2		Model 1		Model 2	
	All men	Fathers	All men	Fathers	All men	Fathers	All men	Fathers
Number of children (ref = 2)								
0	0.16	-	0.05	-	0.11	-	0.09	-
1	0.14	0.16	0.09	0.10	0.28*	0.29*	0.26*	0.27*
3	-0.03	-0.06	-0.05	-0.07	0.07	0.03	0.05	0.01
4	0.46***	0.33*	0.37**	0.26*	0.37**	0.22	0.30*	0.18
1st birth at <23 [a]	-	0.48***	-	0.36**	-	0.29*	-	0.19
Last birth at age >39 [a]	-	0.16	-	0.11	-	0.38**	-	0.36**
Has adopted child	0.12	-0.03	0.16	-0.01	-0.22	-0.31	-0.21	-0.29
Has stepchild	-0.11	0.01	-0.19	-0.06	0.29*	0.27	0.18	0.20
Had child who died [a]	-	0.21	-	0.20	-	0.31	-	0.28
Age	0.02***	0.02***	0.01*	0.01*	0.03***	0.03***	0.02***	0.03***
Poor childhood health	-	-	0.07	0.00	-	-	0.28***	0.33***
No educational qualification	-	-	0.58***	0.56***	-	-	0.46***	0.45***
Currently married	-	-	-0.26**	-0.32**	-	-	0.03	-0.03
Marital termination	-	-	0.16	0.10	-	-	0.21*	0.14
Co-resident with child[a]	-	-	-	0.10	-	-	-	0.00

Note: [a] Among fathers (with biological children). *P<.05; **P<.01; ***P<.001.
Source: Analysis of data from ELSA waves 1−3.

Table 6b: Coefficents from maximum likelihood regression models of associations between fertility history and health outcomes in women (n = 6123)

	Allostatic load, Wave 2				Limiting long-term illness, Wave 3			
	Model 1		Model 2		Model 1		Model 2	
Number of children (ref = 2)	All women	Mothers	All women	Mothers	All women	Mothers	All women	Mothers
0	0.06	-	0.01	-	0.25*	-	0.17	-
1	-0.00	0.00	-0.09	-0.07	0.17	0.17	0.08	0.09
3	0.17*	0.14	0.11	0.09	0.09	0.06	0.04	0.03
4	0.40***	0.32**	0.32**	0.26*	0.40***	0.31**	0.31**	0.24*
1st birth at <20 [a]	-	0.51***	-	0.42***	-	0.59***	-	0.49***
Last birth at age >34 [a]	-	-0.13	-	-0.13	-	-0.28*	-	-0.25*
Has adopted child	-0.21	0.08	0.23	0.12	0.06	0.52	0.08	0.50
Has stepchild	0.01	-0.08	-0.04	-0.15	-0.05	-0.05	-0.01	-0.03
Had child who died [a]	-	0.17	-	0.14	-	0.22	-	0.17
Age	0.02***	0.03***	0.01***	0.02***	0.04***	0.04***	0.03***	0.03***
Poor childhood health	-	-	0.01	0.14	-	-	0.41***	0.37***
No educational qualification	-	-	0.44***	0.39***	-	-	0.34***	0.30***
Currently married	-	-	-0.16	-0.11	-	-	-0.33***	-0.30**
Marital termination	-	-	0.17*	0.18	-	-	0.08	0.10
Co-resident with child [a]	-	-	-	0.09	-	-	-	0.04

Note: [a] Among mothers (with biological children). *P<.05; **P<.01; ***P<.001.
Source: Analysis of data from ELSA, waves 1−3.

Results for all men showed that, relative to men with two children, having had four or more children was associated with worse (higher) allostatic load and with having a long-term illness; having had only one child was also associated with limiting long-term illness. In both cases coefficients were attenuated after control for socio-demographic and life history variables (Model 2). Lacking an educational qualification was positively associated with allostatic load and with long-term illness. Being currently married was negatively associated with allostatic load but not with long-term illness. Results restricted to fathers showed similar associations between lack of an educational qualification and both outcomes and between poor childhood health and long-term illness. Among fathers there was a positive association between early fatherhood (<23) and higher allostatic load; having had a child at age 40 or over was positively associated with long-term illness. Larger family size (which is associated with early entry to parenthood) was associated with allostatic load but not with long-term illness. In models for fathers and all men alike, poor childhood health was associated with long-term illness but not with allostatic load.

Results for women (Table 6b) were broadly similar, although being currently married was associated with limiting long-term illness, and not with allostatic load. Further differences between results for women and men were that the association between late motherhood and long-term illness was negative rather than positive. Childlessness was associated with long-term illness in Model 1, but this ceased to be significant when socio-demographic and life history factors were controlled. Early motherhood was positively associated with both allostatic load and long-term illness in all models.

4.3 Associations between fertility history and intermediate variables

Results from models adjusted for age, education, being currently married, experience of marital disruption, and co-residence with a child (Tables 7a and 7b) showed that fertility histories were associated with the hypothesised mediating variables. Men with four or more children had less wealth, were less physically active, and were more likely to be smokers and report more social strain than fathers of two children; childless men also reported less physical activity than the reference group. Having become a father by age 23 was associated with lower wealth and poorer health-related behaviours. Men experiencing fatherhood after age 39 had less wealth and reported lower physical activity and more social strain. Having had a child who had died was negatively associated with wealth.

High parity women had less wealth, reported less physical activity, were more likely to be smokers, and perceived less social support and more social strain than mothers of two children. Women who had three children rather than two also had lower wealth and were more likely to be smokers. Childless women had higher wealth than mothers of two children. Women who had been teenage mothers had lower wealth, poorer health behaviours, and perceived less social support. Mothers who had one child compared to two children reported less physical activity and were more likely to smoke. All these intermediate variables were significantly associated with both allostatic load and with long-term illness (data not shown).

Table 7a: Coefficents from maximum likelihood regression models of associations between fertility history and wealth, health-related behaviours and social support in men (n = 5110)

	Wealth		Physical activity		Current smoking		Perceived social support		Perceived social strain	
	All men	Fathers	All men	Fathers	All men	Fathers	All men	Fathers	All men	Fathers
N of children (ref = 2)										
0	0.03	-	-0.13***	-	0.17	-	-0.05	-	-0.01	-
1	-0.03	-0.05	-0.05	-0.05	0.09	0.10	-0.02	-0.02	0.01	0.01
3	-0.16**	-0.12*	-0.01	0.01	0.23*	0.22	-0.03	-0.03	0.02	0.01
4	-0.60***	-0.47***	-0.14**	-0.08	0.55***	0.51***	-0.01	-0.01	0.05*	0.03
1st birth at <23	-	-0.24***	-	-0.13**	-	0.32**	-	0.01	-	0.01
Last birth at age >39	-	-0.30***	-	-0.11*	-	0.20	-	-0.03	-	0.06*
Has adopted child	-0.00	0.09	0.16*	0.10	0.07	-0.26	0.03	-0.02	-0.01	-0.03
Has stepchild	-0.11	-0.11	0.07	0.08	0.22	0.27	-0.05	-0.03	-0.01	0.01
Had child who died	-	-0.28*	-	-0.10	-	0.39	-	-0.06	-	0.01

Note: Models adjusted for age, education, being married, marital termination, and co-residence with child (for fathers). *P<.05; **P<.01; ***P<.001.
Source: Analysis of data from ELSA wave 1.

Table 7b: Coefficents from maximum likelihood regression models of associations between fertility history and wealth, health-related behaviours and social support in women (n = 6123) at wave 1

	Wealth		Physical activity		Current smoking		Perceived social support		Perceived social strain	
	All women	Mothers	All women	Mothers	All women	Mothers	All women	Mothers	All women	Mothers
N of children (ref = 2)										
0	0.22***	-	-0.07*	-	0.20	-	-0.02	-	0.04	-
1	0.01	-0.01	-0.07*	-0.08*	0.20	0.22*	0.02	0.02	0.02	0.02
3	-0.13**	-0.09*	0.01	0.02	0.28**	0.27**	-0.03	-0.02	0.01	-0.00
4	-0.55***	-0.43***	-0.16***	-0.11**	0.38**	0.34***	-0.07***	-0.04	0.10***	0.09***
1st birth at <20	-	-0.48***	-	-0.23***	-	0.33**	-	-0.07*	-	0.03
Last birth at age >34	-	-0.03	-	0.05	-	-0.19	-	-0.03	-	0.04
Has adopted child	-0.18	-0.02	0.07	0.08	0.31	-0.12	-0.03	-0.05	0.03	0.04
Has stepchild	0.06	0.04	-0.03	-0.04	0.04	0.06	0.02	-0.01	0.02	0.05
Had child who died	-	-0.06	-	-0.04	-	0.17	-	-0.04	-	0.06

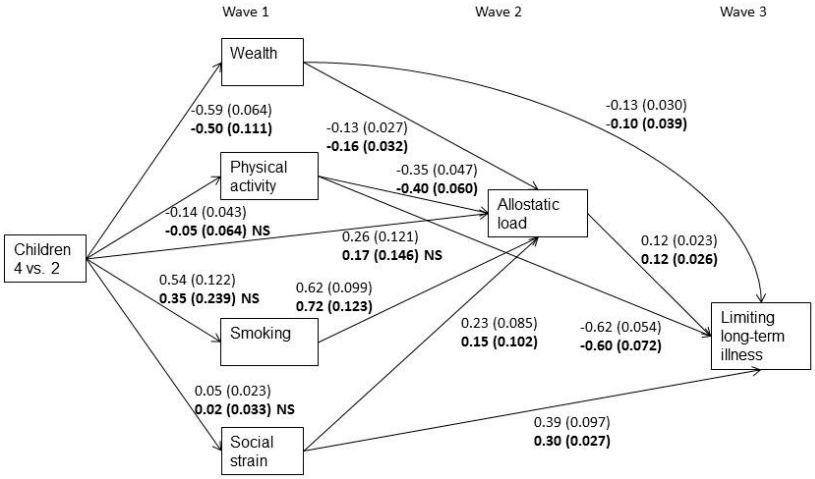
Note: Models adjusted for age, education, being married, marital termination, and co-residence with child (for mothers). *P<.05; **P<.01; ***P<.001.
Source: Analysis of data from ELSA wave 1.

4.4 Results from path models

In the final stage of analysis we estimated path models to examine mediation from fertility history through the intermediate variables to outcomes and from allostatic load to long-term illness. The models presented include results for all fertility and parenting variables which were significantly associated, directly or indirectly, with either or both outcomes (allostatic load and long-term illness). For comparison, we also show in the figures estimates from models restricted to the complete case sample. Compared to using all available information, the models restricted to complete cases provided very similar path estimates for the health outcomes, but underestimated some of the associations between fertility history and some intermediate variables.

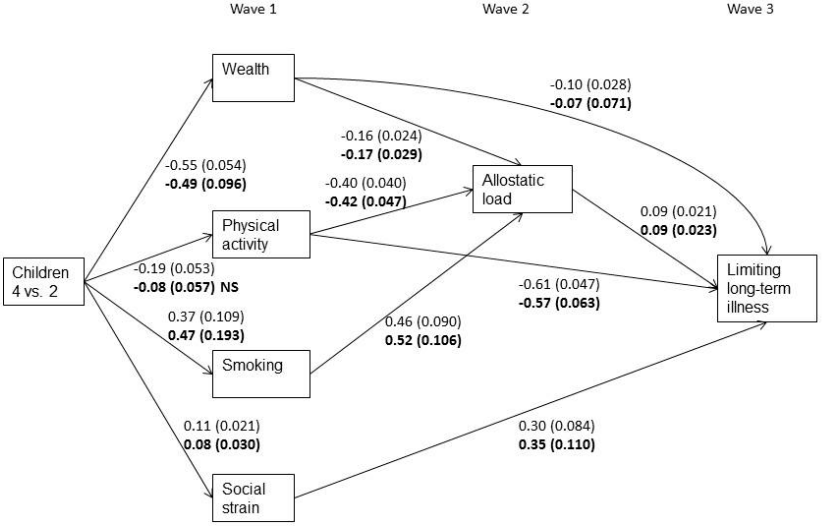
Results for all men and women (Figures 1 and 2) showed that when all intermediate factors were entered in the model, the direct (unmediated) associations between large family size and limiting long-term illness disappeared. In the model for all women the direct association between high parity and allostatic load also disappeared. For both men and women, associations were mediated through wealth, smoking, physical activity, and social strain. For men, effects of smoking on health limitation were mediated through allostatic load; this is not surprising as all of the indicators used in the derivation of allostatic load are known to be influenced by smoking. Wealth, physical activity, and social strain had direct effects on health limitation, as well as those mediated by allostatic load. For women, allostatic load mediated effects of smoking, wealth, and physical activity on long-term illness, although physical activity and wealth also had direct effects on long-term illness, and only direct effects of social strain on limiting long-term illness were significant.

Figure 1: Path model for all men in ELSA (n=5110)



In bold estimates for complete cases (n=1996). Model adjusted for age, education, being married, marital disruption, and childhood health. Significant paths are shown (unstandardized estimate and standard error)

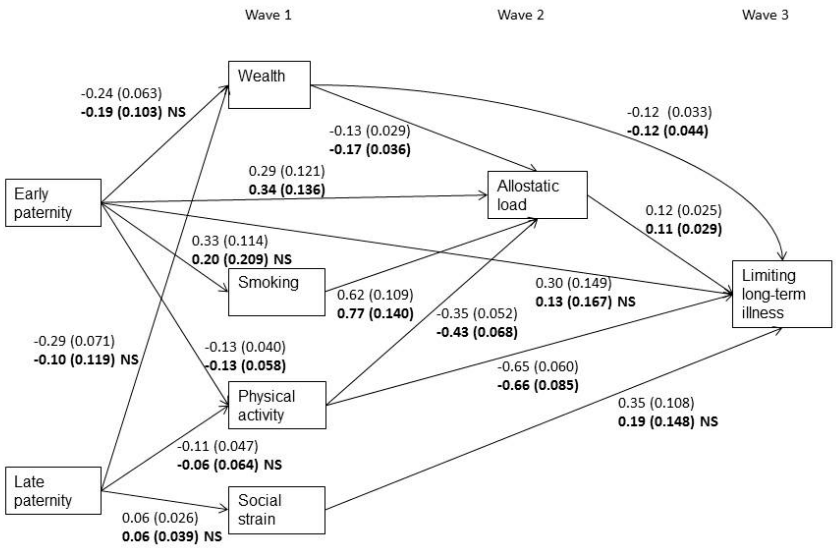
Figure 2: Path model for all women in ELSA (n=6123)



In bold estimates for complete cases (n=2381). Model adjusted for age, education, being married, marital disruption and childhood health. Significant paths are shown (unstandardized estimate and standard error)

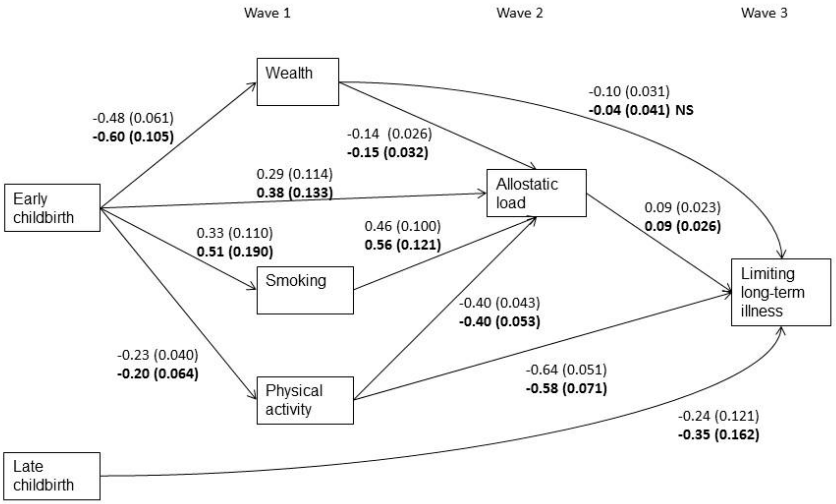
Results from models restricted to biological parents are shown in Figures 3 and 4. Associations between early fatherhood and both allostatic load and health limitation were mediated through smoking, wealth, and physical activity, although significant direct associations with both allostatic load and long-term illness remained. Allostatic load mediated influences on long-term illness, although there were also direct effects of physical activity and wealth on this outcome. Associations between late fatherhood and long-term illness were mediated via wealth, physical activity, and social strain. In mothers, as for fathers, there was a direct association between early parenthood and allostatic load, but otherwise associations were mediated through wealth, physical activity, and smoking (Figure 4). Physical activity and wealth had direct associations with health limitation, as well as associations mediated by allostatic load. Late motherhood had a direct negative association with limiting long-term illness. All associations were in the expected direction.

Figure 3: Path model for fathers in ELSA (n=4256)



In bold estimates for complete data (n=1589). Model adjusted for age, education, being married, marital disruption and childhood health. Significant paths are shown (unstandardized estimate and standard error)

Figure 4: Path model for fathers in ELSA (n=5219)



In bold estimates for complete data (n=1919). Model adjusted for age, education, being married, marital disruption and childhood health. Significant paths are shown (unstandardized estimate and standard error)

5. Discussion

Our aim in this paper was to elucidate the mechanisms whereby fertility and parenthood histories influence health in later life, specifically by examining the extent to which they were mediated by (i.e., operate through) wealth, health-related behaviours, and social support and strain. All of these have been suggested as potentially important intermediate factors in previous studies. A further aim was to develop an indicator of allostatic load and use this as a secondary mediator. We used data from a nationally representative longitudinal study of the older population of England which included retrospective information on fertility trajectories and indicators of childhood health. Results supported some of our hypotheses, but not others. We found no associations between childlessness and either the mediators or the outcomes considered, other than a positive association between childlessness and wealth among women and an association between childlessness and long-term illness in a model adjusted only for age and not any socio-demographic variables. As expected, we found that associations between large family size and poorer health outcomes were mediated by wealth and to some extent by social strain. Health-related behaviours also mediated associations between

larger family size and health outcomes in models including the childless. However, analyses restricted to those with biological children showed that early parenthood – but not number of children – was associated with poorer health behaviours (as hypothesised). These findings together indicate that in the population studied it is early parenthood (associated with larger eventual family size) which seems most important in terms of later health: however this merits further investigation.

Although we found evidence of negative effects of social strain – which was associated with early parenthood and with large family size – we failed to find beneficial effects of perceived social support. This may be related to the measure we used and the fact that the data on intermediate factors was drawn from wave 1 of the study, and we have no information on social support, strain, and health-related behaviours at earlier stages of the life course, including the main childrearing years.

We controlled to some extent for health-related selection into particular parenting pathways by including a variable indicating childhood health problems. Although this represents an advance over some previous studies which have not been able to consider any childhood factors, these data were collected retrospectively and reporting may have been influenced by current health status. This possibility is suggested by the fact that reported childhood illness was significantly associated with health limitation in wave 3 (when the retrospective data was collected) but not with allostatic load, which was based on data collected at wave 2 not subject to reporting bias.

We derived and used a measure of allostatic load in our analysis, as data on biological indicators are less susceptible to subjective reporting factors and may elucidate the biological pathways through which life-course events and circumstances are manifested in later-life health advantage and disadvantage. Negative effects of some parenting trajectories on health are hypothesised to partly reflect the effects of cumulated stress, making allostatic load a particularly relevant indicator for our analysis. Allostatic load also allows identification of differentials relevant to health at an earlier stage of the life course than measures of chronic illness of disability, important in this study as half (52%) the study members were aged less than 65 at wave 1. We found, as hypothesised, that allostatic load mediated associations between other variables, including intermediate ones, and long-term illness, although some direct effects remained, and consider that this outcome has a number of theoretical advantages over indicators based on self-reports. However, there is an obvious cost in terms of increases in missing data. We used Mplus in our analysis, which estimates models using all data available, under the assumption that missingness not predicated by variables in the models may be regarded as missing at random. In line with previous work on incomplete data (Enders and Bandalos 2001), the comparison between the full-information maximum-likelihood estimation using all available data and estimation restricted to complete cases showed that deletion of incomplete cases (i.e., ignoring

some of the observed data) may lead to underestimation of associations. When restricting analysis to compete cases only, the variance tends to get smaller due to selection, which can cause bias and less robust estimation.

It should be recognised that characteristics of the sample we consider may reflect prior differential mortality related to fertility history. The apparent slight under-representation of those who had become parents at young ages and had large family sizes, for example, might reflect reduced chances of survival to later ages for these groups. This would mean that the influence of these factors on health estimated here might be lower than would be observed in a cohort which was followed throughout its whole post-reproductive period.

Despite these limitations, this paper adds to our understanding of the processes linking parenthood trajectories, biological processes, and health in later life. Further comparative work using data from other populations and time periods is merited, as effects may be different in other populations where social selection into early parenthood and larger family size, as well as stresses experienced by parents of larger families, may be different.

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Appendix

Table 1: Allostatic load 25th percentile high-risk cut-off points for all men and women in ELSA in wave 2 (2002)

	Men		Women	
	Aged 51-65	Aged 65+	Aged 51-65	Aged 65+
Inflammation	(*N* = 1008-1017)	(*N* = 982-986)	(*N* = 1219-1232)	(*N* = 1190-1196)
C-reactive protein	>2.9	>3.4	>3.4	>3.9
Fibrinogen	>3.4	>3.7	>3.5	>3.8
Cardiovascular	(*N* = 1074)	(*N* = 1106)	(*N* = 1319)	(*N* = 1398)
Systolic blood pressure	>143	>149	>140	>151
Diastolic blood pressure	>85	>80	>83	>79
Lipid metabolism	(*N* = 1001-1017)	(*N* = 965-983)	(*N* = 1219-1233)	(*N* = 1187-1196)
HDL/Total cholesterol ratio	>5.0	>4.6	>4.4	>4.5
Triglycerides	>2.5	>2.2	>2.1	>2.1
Glycosylated haemoglobin	>5.7	>5.9	>5.6	>5.8
Body fat	(*N* = 1216)	(*N* = 1231)	(*N* = 1486)	(*N* = 1527)
Waist/hip ratio	>1.00	>1.00	>0.88	>0.89
Respiratory	(*N* = 1197)	(*N* = 1190)	(*N* = 1415)	(*N* = 1437)
Peak expiratory flow	<506	<406	<344	<265