Health in early adulthood and fertility: A study based on the 1958 British cohort

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Short title: Health and fertility: The 1958 British cohort

Abstract

Health is rarely used as an explanatory variable in fertility studies in high-income contexts, unlike in low-income settings. Using the 1958 National Child Development Study, we explore how self-rated health (SRH) and body mass index (BMI) at age 23 relate to achievement of fertility goals by age 46. We find that worse SRH and a BMI outside the healthy range at age 23 are strongly associated with lower fertility and underachieving fertility goals. While poor SRH is associated with lower fertility mostly among men, BMI outside the healthy range at 23 is more significant for women. Additional analyses indicate that employment and union history partly mediate the effect of health on fertility, but health retains a substantive direct effect. Our findings suggest that health in early adulthood is an important determinant, whether direct or indirect, of family life-course trajectories. This paper endorses the inclusion of health as an explanatory variable in studies of fertility in high-income contexts.

Keywords: health; fertility; fertility intentions; BMI; self-rated health; life course; Great Britain

Introduction

The link between health and fecundity (the biological ability to have children) has been an area of significant interest in the epidemiological, evolutionary, and historical literature. Poor health has been shown to affect fertility through several, possibly sex-specific, pathways. For example, certain diseases may lead to infertility issues, be incompatible with pregnancy, limit individuals' ability to participate in leisure activities and family life, including childrearing, or even undermine their chances of entering a stable union or finding a job (Fair et al. 2000; Dow and Kuhn 2004).

Demographic research on how health affects modern-day fertility (actual childbearing outcomes) is common in studies of low-income contexts. This line of enquiry may be motivated by the well-known proximate determinants of fertility framework for 'natural fertility' populations (Bongaarts 1978), which includes sterility because of disease as one component of the model. Demographic studies on the impact of health on fertility in high-income countries, however, are more limited. Instead, research connecting health and fertility together tends to take the opposite causal argument, looking at how childbearing affects health in later life (e.g. Dribe 2004; Yi and Vaupel 2004; Hank 2010; Read et al. 2011). Socio-economic factors, such as family background, educational level, employment career, and economic stability are much more common explanations and control variables for individual reproductive choices in high-income contexts (Balbo et al. 2013). This could be because of the perception that infectious diseases have little influence over people's lives in these countries. However, the Covid-19 pandemic has prompted a renewed interest in comprehending how the direct and indirect consequences of ill health can influence childbearing decision-making and behaviour (Berrington et al. 2022). The impact of geography on health and fertility is also currently gaining interest: particularly the question of

how climate change and pollution can influence health and therefore fertility (Conte Keivabu et al. 2024).

This study makes a substantive contribution to the demographic literature by revisiting the pivotal role of health in shaping fertility outcomes, taking Great Britain as a case study and adopting a life-course perspective. Our aim is to assess the relationships between two different health-related measures—namely self-rated health (SRH) and body mass index (BMI), both recorded during early adulthood—and fertility. While we acknowledge that changes in health status during adulthood are also likely to affect fertility trajectories, the scope of this paper is to shed light on the role of health in young adulthood, while also considering the fact that health as a young person operates as a proxy for health across the life course (Haas 2007). Instead of looking at how health influences single fertility transitions—which would make it impossible to account for potential recuperation effects—we focus on completed fertility. In addition, we not only investigate fertility outcomes but also explore the decision-making processes that shape them, by looking at the relationship between individuals' health and the probability of them realizing their ideal family size (as declared at age 23). This dual perspective offers a more comprehensive understanding of the dynamic interplay between health, personal aspirations, and fertility choices across the whole life course. It also highlights the instrumental role that health plays from an early age in family trajectories. Furthermore, informed by previous literature about sex differences in the causes and consequences of health and in the way in which health is self-assessed (Vlassoff 2007; Schneider et al. 2012), we conduct all the analyses separately by sex, which adds a crucial dimension to our study. Finally, we run additional analyses to test the extent to which socio-demographic factors, such as union history and employment status, mediate the link between health in early adulthood and fertility.

To this aim, we use longitudinal data from the 1958 National Child Development Study (NCDS) of individuals born in Great Britain and adopt a gender perspective by investigating the health–fertility link separately among women and men.

Background

Empirical evidence for a link between health and fertility

In general, the existing epidemiological literature highlights that those with health problems tend to desire (McGrath et al. 1999; Cvancarova et al. 2009) and have fewer children than healthy individuals (Chen et al. 2001; Langeveld et al. 2002). Differing but complementary interdisciplinary perspectives provide explanations for this relationship. At a more ultimate level of explanation, the evolutionary anthropological literature views having children as an energetically costly process. Health problems also require significant energy resources, and the competing energy needs between poor health and fertility ultimately result in fewer children (Jasienska et al. 2017). Furthermore, experience of poor health (or harsher environments) in childhood and youth is likely to have long-term 'priming' effects on reproductive decision-making and outcomes, although evidence from the evolutionary literature is not conclusive about whether reproduction and health 'traits' are always associated with each other in the same way (Coall et al. 2016; Brown and Sear 2021). At a more proximate level, the medical and epidemiological literature offers more direct explanations about why poor health is detrimental to childbearing plans and outcomes. To begin with, poor health can affect both men's and women's ability to achieve a pregnancy. Particular health conditions, both physical and mental, are known causes of infertility (Fair et al. 2000; Bhongade et al. 2015). Similarly, markers of poor health and health behaviours—such as high/low BMI, low levels of physical fitness, and smoking—have all been associated with inability to conceive among both women and men (Rich-Edwards et al. 2002; Ramlau-Hansen et al. 2007; Chambers and Anderson 2015; Harlev et al. 2015; Wootton et al. 2023).

For men, issues with fertility appear to stem from associations between high BMI or smoking and reduced sperm quality (Cheng and Ng 2007; Hammoud, Wilde et al. 2008; Harlev et al. 2015). For women, high/low BMI and smoking can affect ovulation frequency and likelihood of successful implantation (Mattison 1982; Ellison, 1990). Treatment for health conditions, such as cancer, can also result directly in infertility issues (Dow and Kuhn 2004).

Beyond the direct impact of health and health behaviours on ability to conceive, health has also been demonstrated to be a key decision-making factor for planning children. For example, those with health problems may wish to have fewer or no children because their health problems would make caring for a child difficult (McDonald 2002; Katz 2006) or because they are concerned about passing their health condition(s) on to their children (Katz 2006). Women with health conditions may also not wish to be pregnant as this may exacerbate their pre-existing condition(s) or cause complications during pregnancy/birth (Fair et al. 2000; Drew 2002; Dow and Kuhn 2004). Social science studies have also offered more proximate explanations for the effect of health on fertility plans and outcomes via indirect routes. For example, health can affect individuals' ability to be employed or maintain a job, which in turn may make having children too expensive (Currie and Madrian 1991). Health may similarly make finding a partner (which is a strong predictor of having children) and maintaining a partnership challenging (Mynarska et al. 2015; Tocchioni 2018). A Finnish and Swedish study, for example, found evidence that men in worse health were particularly likely to be childless as a result of not being in a partnership (Liu et al. 2023).

However, despite the wealth of evidence suggesting a link between health and fertility, few studies published in socio-demographic journals have explicitly tested for a link between health and fertility in high-income contexts. This is curious, given the comparatively large body of demographic literature focusing on the opposite direction of association: how fertility affects later-life health (e.g. Grundy and Tomassini 2005; Hank 2010; Read et al.

2011; Grundy and Read 2015; Hanson et al. 2015; Grundy and Foverskov 2016; O'Flaherty et al. 2016; Barclay and Kolk 2018; Keenan and Grundy 2019; Sironi 2019; Torche and Rauf 2020; Yeatman and Smith-Greenway 2021; Bonsang and Skirbekk 2022; Machů et al. 2022; Stannard et al. 2023; Barclay et al. 2024). Some studies of fertility determinants include health (typically SRH) as a control variable (e.g. Aassve et al. 2016; Beaujouan et al. 2019; Testa and Bolano 2021; Guzzo 2022; Jarosz et al. 2023; Yi and Shangguan 2024); however, socio-economic controls are included much more frequently.

Among studies that explicitly test for a connection between health and fertility, most focus on the relationship between BMI and childbearing. Three studies based in the United States found a significant relationship between obesity in early adulthood and the likelihood of remaining childless for both men and women (Jokela et al. 2008; Frisco and Weden 2013; Lee et al. 2023). A Swedish study by Barclay and Kolk (2020) analysed the link between health and fertility among men and found strong relationships between three health markers (BMI, physical fitness, and height) and completed fertility, and these persisted after controlling for education and income. Another study, using Finnish data, found a relationship between being outside the healthy weight range in early adulthood and number of children achieved 21 years later (Jokela et al. 2007).

Studies that do not focus on BMI use various different ways of capturing health. Holton et al. (2011) found that in their sample of Australian women, concerns about health conditions and ability to have children were associated with childbearing ideals and outcomes. Two other Australian studies found that worse SRH was associated with decreased desires (Gray et al. 2013) and expectations (Lazzari and Beaujouan 2025) for children among men and women. A study in Norway found that sickness absence from work was positively associated with transitions to parenthood, but long-term receipt of health-related benefits was negatively associated with fertility (Syse et al. 2022). Alderotti and Trappolini (2022)

explored this topic among Italy's migrant population by sex, duration of stay, and parity. They found that among migrant men, poor SRH negatively affected fertility intentions, especially among recent migrants who already had at least one child. The effect was stronger among women, especially among long-term migrants with chronic illnesses and mental health conditions. Another study, based on Swedish and Finnish registers, found strong associations between different health issues and the probability of childlessness, all mediated by singlehood and education (Liu et al. 2023). These health issues included mental–behavioural disorders (especially among men), congenital anomalies and endocrine–nutritional–metabolic disorders (especially among women), and inflammatory and autoimmune diseases. The importance of adolescent mental health for men's rather than women's fertility outcomes and intentions has also been observed in a Norwegian and an Australian study (Evensen and Lyngstad 2020; Lazzari and Beaujouan 2025).

A life-course perspective on health and fertility

In this paper, we explore how health in young adulthood affects fertility outcomes and the fulfilment of fertility goals. This approach is informed by frameworks and theories of reproductive decision-making. Psychosocial theories of reproductive decision-making, such as the cognitive–social model (Bachrach and Morgan 2013), outline that expectations about life outcomes (e.g. having children) are formed during early adulthood, based on normative ideals about families and childhood experiences. These expectations are revised over the life course in the presence of situational changes, competing preferences, and new information. In the context of health, these could include: diagnosis of a particular illness that changes the likelihood of an individual having children; a potential incompatibility between prioritizing health/recovery goals and family goals; or simply a change in preferences for having children in the light of information about their own health. The Traits–Desires–Intentions–Behaviour framework (TDIB; Miller et al. 2004) outlines the pathway for how fertility desires and

intentions may change over the life course as a result of these changing life circumstances: biologically based or non-conscious motivations for children (traits) are adapted in young adulthood into desires for children (e.g. number of children wanted in the presence of no obstacles). Desires are often highly informed by social norms about having children. In high-income countries, such as the UK, these social norms have typically been pro-natal (i.e. parenthood is seen as a desirable goal) in the last half of the twentieth century, with a persistent desired family size of two (Sobotka and Beaujouan 2014). These desires are then translated into more realistic childbearing intentions, which are fulfilled through instrumental behaviours to try to achieve or avoid a pregnancy. However, situational factors such as health can modify this pathway at each step, making the formation of fertility intentions and the likelihood of achieving those intentions malleable across the life course. Another study using the NCDS, by Berrington and Pattaro (2014), operationalized the TDIB framework in this way and found that educational attainment at age 23 mattered for the likelihood of women, but not men, fulfilling their reproductive goals (stated at age 23) by age 46. In a similar vein, this paper aims to investigate how reproductive goals formed at an early age, prior to the occurrence of major life milestones, manifest in actual outcomes over a span of 20 years and how this relates to health in early adulthood.

Operationalizing health in early adulthood

A challenge for this research was how to operationalize health in the analysis. Health is a multifaceted concept, with multiple intertwining components including physical, mental, and functional health, disease, and emotional well-being. In this analysis we are limited to the measures of health included in the NCDS early-adulthood wave in 1981, when participants were aged 23. Possible health measures included whether an individual had a health condition at 23, when they were diagnosed, and how long they had had the condition. However, we decided that a binary indicator of whether an individual had a health condition was too

general and introduced too many issues when comparing different types of health conditions. Another possibility was a measure of whether the individual was out of work due to poor health, but this was quite specific to more severe cases of ill health. Mental health scales were also used in the NCDS to measure mental health at age 23; however, due to insufficient variation in mental-health-related variables, we decided not to include them among the main explanatory variables in our analyses. Neither did we explore health behaviours such as smoking, as there were too few waves to provide detailed information on changing behaviour during the period of observation. We therefore decided to use a holistic health measure, SRH. The NCDS question asks *How is your health in general?*, with an ordered categorical response scale ranging from 'excellent' to 'poor'. SRH is considered a standard measure of an individual's general or global health because it encompasses their individual view on their physical, functional, and mental health (De Bruin 1996). It is known to be predictive of subsequent health outcomes, care needs, and mortality (Luppa et al. 2010; Bamia et al. 2017; Simonsson and Molarius 2020). The measure has been included in the NCDS since 1981 (age 23), the starting survey wave of our analysis. While there can be issues with comparing SRH between countries or overtime (as different countries or age groups may have different cultural constructions of what being in good health means), comparisons within the same country among a cohort born in the same year minimize this issue. Nonetheless other situational factors (e.g. their relative socio-economic position or sex) may affect individuals' perceptions of their own health (Idler and Benyamini 1997). SRH could be both directly and indirectly related to the likelihood of having children. Poor SRH is likely to indicate the presence of health conditions, which could affect individuals' ability to have children, but it may also affect their likelihood of finding and maintaining a partnership and their labour market activity.

For our second measure of health in early adulthood, we used BMI at age 23, as it was measured for the whole sample and is less variable across the life course than some other health measures (Bridger Staatz et al. 2023). BMI is measured by dividing an individual's weight in kilograms by their height in metres squared. In the NCDS age 23 survey, height and weight were self-reported by participants. In clinical practice, the scores are usually divided into underweight, healthy weight, over-weight, and obese groupings, the cut-offs of which vary slightly between ethnic groups to account for varying risks of developing diabetes (NHS Information Centre 2012). While belonging to one of the BMI groups in and of itself is not indicative of good or poor health, being outside the healthy weight range comes with associated risks of developing certain health conditions, such as hypertension and diabetes. It is a somewhat crude measure of risk, however, as BMI does not account for other important determinants of health (e.g. sex, ethnicity, and muscle mass) that can also vary with BMI. For childbearing, being outside the healthy weight range could affect outcomes both directly and indirectly. Being underweight or overweight may affect the physiology of men and women in ways that make the likelihood of experiencing infecundity or negative birth outcomes more likely (e.g. Nguyen et al. 2007; Craig et al. 2017; Calvacante et al. 2019; Boutari et al. 2020). However, being outside the healthy weight range may also have social consequences for childbearing: for example, it may affect the likelihood of finding a partner (Kallen et al. 1984; Ajslev et al. 2012) or result in discrimination by social institutions, which in turn affects family-formation trajectories (Puhl and Brownell 2001; Flint et al. 2016; Campos-Vazquez et al. 2020).

Hypotheses

Our literature review has highlighted that different types of health measures may affect fertility in different ways: some health conditions and treatments may have more direct impacts on biological ability to have children, whereas other health conditions may have a

greater impact on mediating factors, such as ability to find a partner. The first key contribution of this paper is therefore to explore the effect of two different health measures (BMI and SRH) recorded at age 23 on two different fertility outcomes at age 46 (completed fertility and realization of the ideal family size stated at age 23). Our main hypothesis (Hypothesis 1) is that those with a BMI outside the healthy weight range and with worse SRH will be more likely to report lower completed fertility and more likely to underachieve their fertility goals than those in the healthy BMI range or with better SRH.

We also use mediation analysis to understand whether the impacts of these two health measures are more likely to influence fertility through direct or indirect mechanisms. Our second hypothesis is that BMI will be more likely to have direct impacts on fertility than SRH will, because existing evidence shows that a high/low BMI can result in physiological changes that affect fecundity (e.g. Ellison 1990, Harlev et al. 2015).

The second key contribution of the paper is to model these associations separately for men and women. While we think our first hypothesis will hold for both men and women, our literature review also pointed to many possible gendered/sex-specific effects of health on fertility. Our third hypothesis is that the effect size and statistical significance of the association between high/low BMI and fertility will be greater for women than men. We expect this on the basis that there are several known mechanisms between high/low BMI and women's ability to conceive and sustain a pregnancy, compared with for men, where the primary direct mechanism is between high BMI and sperm quality. There is also evidence of a more pronounced effect of BMI on fertility among women than men in existing studies (Frisco and Weden 2013; Lee et al. 2023). Relatedly, our fourth hypothesis is that there will be less of a difference between men and women for the effect of SRH relative to the effect of BMI, as the former is likely to work through indirect and direct pathways for both sexes. However, there may be differences between men and women in the mediation analysis owing

to the typical gendered division of labour in this mid-century birth cohort. Many respondents will have been in male-breadwinner or dual-earner couples during their childbearing years. Our fifth hypothesis is therefore that there will be a greater mediating role of employment on the health–fertility link for men than women. Sixth, we also expect a greater mediating role for union status for men than women, based on some evidence of this in previous literature (Liu et al. 2023).

Data and methods

Data and sample selection

We used data from the 1958 NCDS. The study is a birth cohort following the lives of an initial 17,415 people born in England, Wales, and Scotland (Great Britain) in a single week of 1958. The study has 11 sweeps (waves) and is still active today.

We selected individuals who participated in the 1981 sweep (i.e. at age 23) and completed the follow-up until 2004 (i.e. until age 46; $n = 8{,}219$). We excluded those with missing information about their health conditions at age 23 or fertility outcomes at age 46 (about 0.3 per cent; $n = 25$). To retrieve information about individuals' employment and union histories (which we use as a control variable and to test as potential mediators of the health–fertility association), we further restricted our sample to individuals who also participated in the 1991 sweep (i.e. when they were aged 33; n = 7,819 included). Since information about health was first collected at age 23, we further restricted our analyses to individuals who were childless at that age (23.5 per cent excluded; n = 1,839). Thus, our final sample consists of 5,980 individuals: 2,815 women and 3,165 men.

While we acknowledge that excluding individuals who already have at least one child by age 23 may introduce bias into our analyses (e.g. they may be positively selected for health, as they managed to have a child earlier), we believe that this approach remains the most reliable way to observe the relationship between health in early adulthood and

completed fertility or achievement of ideal family size. Although using data from the previous survey sweep, collected in 1974 when participants were 16, could address the selectivity issue—since the proportion of individuals who already have a child by age 16 is much smaller than at age 23—it would require relying on health measures and ideal family size as reported at age 16, which could introduce additional (and potentially more serious) issues. First, as already mentioned, SRH was not available in the 1974 questionnaire and, notably, evidence suggests that self-assessments of health before age 18 are less reliable, as they can be influenced by parents and are a less stable construct (Wade and Vingilis 1999; Trappolini and Giudici 2021). Second, the ideal family size declared at age 16 was likely influenced by the social norms and family-building behaviours prevalent at that time, making it less representative of a genuine commitment to having a specific number of children (Berrington and Pattaro 2014). For these reasons, we chose to limit our analysis to individuals who were childless at age 23. We conducted separate analyses for those with at least one child by age 23; these are discussed in the Additional analyses section.

Outcome variables

We focused on two fertility outcomes. The first was the number of children an individual had at age 46, which in this paper we use as a proxy for completed fertility. To analyse the relationship between health and number of children at age 46, we created a four-category dependent variable indicating whether the respondent had no children, one child, two children, or three or more children at that age.

The second outcome was achievement of ideal family size by age 46, measured in relation to the self-assessed total number of children desired, as stated at age 23 among those who wanted children (hereafter, ideal family size). This information was derived from the question, *How many children would you like altogether?* To study this aspect, we generated a categorical variable indicating whether at age 46 the individual had: (1) exactly the number of

children that they stated when they were 23 or more than this ('ideal size achieved'); (2) one child fewer than the ideal number declared at age 23 ('underachieved by one'); or (3) two or more children fewer than the ideal number declared at age 23 ('underachieved by two or more'). We grouped those who 'overachieved' their intentions together with those who met them, because the relationship between poor health and fertility outcomes was expected to be negative, so underachievement was the outcome of interest. Furthermore, the share of those who exceeded their ideal family size stated at 23 was relatively small (12.3 per cent of the sample; n = 738), and most of these had only one child more (10.2 per cent of the sample; n= 607). Finally, individuals who declared they did not want any (more) children when they were 23 were excluded from this second set of analyses as they were not asked the question about the total number of children wanted (*n* = 1,353; 22.6 per cent of the sample). Those who answered 'don't know' or did not answer the question about the total number of children wanted at age 23 were also excluded (6.3 per cent of the sample; n = 376). After these exclusions, the final sample size for this analysis is 4,251 individuals.

Main explanatory and control variables

We considered two main explanatory variables: SRH and BMI, both measured when respondents were aged 23. There was poor correlation between the two health outcomes analysed, equal to around 0.10 both among men and women, suggesting that there was room to investigate them separately.

In the NCDS, SRH is derived from the question, *How is your health in general?* There were four possible answers: 'poor', 'fair', 'good', or 'excellent'. For our analyses, we combined the poor and fair categories due to the small number of cases of the former (*n* = 52). We computed BMI using respondents' self-reported height and weight ($BMI = \frac{weight}{height^2}$) and grouped it into four categories following the guidelines provided by the World Health Organization: 'underweight' (BMI < 18.5), 'healthy weight' (BMI 18.5–24.9),

‘overweight’ (BMI 25–30) and ‘obese’ (BMI>30). We did not adjust the boundaries for different ethnic groups because of the low proportion in the sample (<5 per cent) with two foreign-born parents or not from a White ethnic background.

In all the analyses, we incorporated a set of socio-demographic control variables. More precisely, the set of variables included educational level (‘university degree’, ‘A level and equivalent’ [qualification attained at 18], ‘O level and equivalent’ [qualification attained at 16], or ‘no qualification’) and macro-area of residence (‘North England’, ‘South England’, ‘Wales’, ‘Scotland’, or ‘unknown’) at age 23. We also used the interview when respondents were aged 16 (Sweep 4 in 1974) to collect information about their number of siblings (continuous variable) and their migration background (‘both parents were born in the UK’ or ‘at least one parent was born abroad’). Finally, because we were most interested in the direct effect of health on fertility, we further controlled for two variables that may mediate the relationship between health and fertility: namely, employment and union histories. For the former, we used the respondent’s employment status at ages 23, 33, and 46 to control for their employment history (‘not employed in all waves’, ‘employed in at least one wave’, or ‘employed in all waves’); for the latter, we used the respondent’s union status at ages 23, 33, and 46 to control for their union history (‘cohabiting or married in all waves’, ‘cohabiting or married in two out of three waves’, or ‘cohabiting or married in only one wave or in no waves’).

Methods

To investigate the health–fertility link, we used multinomial logistic regression models and conducted two separate analyses.

In the first analysis, we examined the relationship between the number of children at age 46 and the two main explanatory variables—SRH and BMI—separately. In the second analysis, we modelled the association between the realization of ideal family size among

those who wanted children at age 23 and the two health measures analysed, while controlling for a set of socio-demographic characteristics. Additionally, we ran separate models for men and women, considering that existing literature suggests that health may influence fertility along sex- and gender-specific pathways.

We started by estimating the relative risk ratios. Then, to facilitate interpretation of the results, we computed the average marginal effects (AMEs) to present our findings. AMEs express the effect on the probability of observing the outcome of interest as a categorical covariate change from one category to another or as a continuous covariate increase by one unit averaged across the values of the other covariates included in the model equations. For space reasons, we present only the results for health-related variables in the main paper. The full models are shown in the supplementary material.

We also performed a preliminary investigation into the mediating roles of employment and union status in the health–fertility relationship. While a comprehensive understanding of this would require a dedicated framework, we performed basic mediation analyses using the Karlson–Holm–Breen (KHB) method (Karlson et al. 2012) to test these two variables as potential mediators. The results are discussed in the Additional analyses section.

Results

Descriptive statistics

A full table of descriptive statistics is available in the supplementary material (Table S1). Regarding our main explanatory variables, the majority of the sample reported either excellent SRH (45 per cent for women, 49 per cent for men) or good SRH (48 and 44 per cent, respectively), and most were of healthy-weight BMI at age 23 (83 per cent of women and 79 per cent of men). Men were slightly more likely to be in the overweight BMI category

compared with women (16 per cent compared with 9 per cent) and women were more likely to be underweight in terms of BMI (7 vs 3 per cent).

Regarding our main outcomes of interest, the average number of children by age 46 was 1.6 among women and 1.5 among men, and mean ideal family size at age 23 for those who wanted children was 2.8 among both men and women. Men were more likely than women to have no children at age 46 (31 per cent vs 24 per cent), and women were more likely to have two children than men (43 vs 37 per cent). There were minimal differences between men and women in whether they achieved or underachieved their ideal family size stated at age 23. When looking at the overall sample, around two in five achieved their goal, one in five underachieved by one child, and another two in five underachieved by two or more children.

Fertility by age 46

Table 1 shows the results of multinomial logistic models examining the association between health measures and number of children at age 46, represented as AMEs for the probability of being childless, having two children, and having three or more children (the category 'one child', omitted for space reasons, is shown in the full models in Table S2, supplementary material). The results highlight significant associations between health measures and fertility outcomes, with large effect sizes, and also display some differences between men and women.

Table 1 *Multinomial logistic models of number of children at age 46: Women and men in Great Britain*

	Women						*Men*					
Number of children	None		Two		Three or more		None		Two		Three or more	
SRH (ref. = Excellent)												
Good	0.01	(0.390)	0.02	(0.271)	−0.02	(0.099)	0.05	(0.001)	−0.04	(0.020)	−0.01	(0.431)
Fair/Poor	0.03	(0.286)	−0.05	(0.231)	−0.02	(0.487)	0.07	(0.027)	−0.03	(0.381)	−0.04	(0.177)
BMI (ref. = Healthy weight)												

Underweight	0.05	(0.100)	−0.01	(0.820)	0.03	(0.367)	0.05	(0.351)	−0.04	(0.481)	−0.02	(0.618)
Overweight	0.03	(0.355)	0.01	(0.650)	−0.03	(0.139)	0.01	(0.560)	−0.01	(0.639)	0.01	(0.685)
Obese	0.20	(0.004)	−0.09	(0.208)	−0.07	(0.089)	0.03	(0.534)	−0.03	(0.614)	0.03	(0.412)

Notes: Table reports AMEs, with *p*-values in brackets. Models control for level of education, macro-area of residence, employment history, number of siblings, migration background, and union history. Ref. refers to the reference category. Results of the full model are shown in Table S2, supplementary material.

Source: Authors' elaboration based on data from the 1958 National Child Development Study (NCDS).

Among women, BMI exerts a stronger influence than SRH on completed fertility. Women with an obese BMI at 23 exhibit a 20 percentage point (pp) higher likelihood of remaining childless and a seven pp lower likelihood of having three or more children than women with a BMI in the healthy weight range at age 23. Women with an underweight BMI are also more likely to be childless by age 46 than those whose BMI is within the healthy range, by five pp (weakly significant), while no significant results are detected for overweight BMI. The effect sizes for SRH are generally smaller than for BMI, with the only borderline significant effect being a slightly lower likelihood of having three or more children at age 46 (by two pp) among women who report good (instead of excellent) SRH.

Conversely, among men, SRH emerges as a strong predictor of fertility. Men reporting good or fair/poor health at age 23 are more likely to remain childless than those reporting excellent health, by five and seven pp, respectively. Additionally, men with good SRH display a four pp lower likelihood of having two children at age 46. Men with fair/poor health at age 23 are less likely to have two children or three or more children by age 46, but estimates are not significant. Regarding the relationship between BMI and fertility by age 46, no significant results are detected for men.

In terms of other explanatory variables, there are some significant findings across all control variables, and these are consistent across both the SRH and BMI models (see Table S2, supplementary material). However, the most prominent differences are by education,

employment, and union history in both models. Among both men and women, those without a degree are more likely to have one child and less likely to have three or more children, and those who are not in a cohabiting or married union in all waves are less likely to have two or more children. Employment shows slightly different patterns between the sexes: women who are not employed in any wave are less likely to have two children and more likely to have three or more children than women who are employed in at least one wave, whereas for men, those employed in all waves are less likely to be childless and more likely to have two children than men employed in at least one wave.

Achievement of ideal family size

Table 2 presents the results of models examining the achievement of ideal family size, as reported at age 23 by those who wanted children. Among women, those reporting fair/poor SRH at age 23 demonstrate a lower likelihood of attaining their ideal number of children by age 46, with a seven pp difference compared with those reporting excellent health (weakly significant). Results also suggest that women reporting good or fair/poor SRH at age 23 are more likely to underachieve their ideal number of children by one child or two or more children, but AMEs show poor statistical precision and estimates are not significant. As regards the role of BMI, the magnitude of the effect is especially striking among women with an obese BMI. These women are less likely (by 13 pp) to achieve their ideal family size and more likely to underachieve it by two or more children (by 23 pp), compared with women with a BMI in the healthy weight range. Women with an overweight BMI also show lower chances of achieving their ideal family size, by five pp. Women with an underweight BMI are less likely than women with a healthy-weight BMI to underachieve their ideal family size by one child (by five pp).

Table 2 Multinomial logistic models of achievement of ideal family size by age 46: Women and men in Great Britain

	Women						*Men*					
Ideal family size	Ideal size achieved		Underachieved by one		Underachieved by two or more		Ideal size achieved		Underachieved by one		Underachieved by two or more	
SRH (ref. Excellent)												
Good	−0.03	(0.148)	0.02	(0.289)	0.01	(0.590)	−0.05	(0.002)	−0.02	(0.256)	0.07	(0.000)
Fair/Poor	−0.07	(0.068)	0.03	(0.407)	0.04	(0.343)	−0.07	(0.054)	0.03	(0.273)	0.03	(0.322)
BMI (ref. Healthy weight)												
Underweight	0.02	(0.684)	−0.05	(0.057)	0.04	(0.285)	−0.02	(0.680)	−0.03	(0.483)	0.05	(0.442)
Overweight	−0.05	(0.097)	0.00	(0.917)	0.05	(0.155)	−0.01	(0.552)	0.02	(0.246)	0.00	(0.873)
Obese	−0.13	(0.055)	−0.10	(0.160)	0.23	(0.001)	−0.08	(0.206)	−0.03	(0.533)	0.11	(0.091)

Notes: Table reports AMEs, with *p*-values in brackets. Models control for level of education, macro-area of residence, employment history, number of siblings, migration background, and union history. Ref. refers to the reference category. Results of the full model are shown in Table S3, supplementary material.

Source: As for Table 1.

Among men, those reporting good or fair/poor SRH at age 23 also display lower probabilities of achieving their ideal number of children than those reporting excellent SRH, with differences of 5 and 7 pp, respectively. Men reporting good (instead of excellent) health are also more likely to underachieve their ideal number of children by two or more children (by 7 pp). In relation to BMI, the only borderline significant result among men is that those with an obese BMI at age 23 are more likely to underachieve their ideal number of children by two or more children, exceeding those with a BMI in the healthy weight range by 11 pp. Men with an underweight BMI at age 23 are also less likely to achieve their ideal number of children by two or more children, albeit the AMEs are not significant.

In terms of trends in control variables, there are fewer significant findings when compared with the model for number of children by age 46 (see Table S3, supplementary material). However, education, employment, and union history trends remain consistent in both models: women with an O level (or equivalent) or no qualification are less likely to underachieve by two or more children than women with a degree; men employed in all waves are more likely to achieve their ideal and less likely to underachieve their ideal by two or

more children than men who are employed in at least one wave; and men and women who are not in a marital/cohabiting union in all waves are less likely to achieve their ideal family size and more likely to underachieve their ideal by two or more children than those who are in a union in all waves.

Additional analyses

As mentioned earlier, we conducted additional analyses to provide a better understanding of potential mechanisms for the effect of health in early adulthood on fertility.

First, to address sample selection bias (due to including only individuals who were childless at 23), we replicated the completed fertility analysis only on those originally excluded because they had had at least one child before age 23. The outcomes in this replicated analysis were number of children at age 46: 'one', 'two', or 'three or more'. Results in Table S4 confirm that health exerts a significant effect on fertility, with patterns similar to those found in the main childless-at-23 sample for women: while SRH shows no significant association with completed fertility, BMI does (e.g. underweight or overweight women at 23 are 19 pp and eight pp, respectively, more likely to have only one child by age 46 compared with those with a healthy-weight BMI). In contrast, no significant association between either health measure and fertility emerges for men. We also attempted separate models for those with two children at age 23, but the sample size was insufficient.

Regarding fertility intentions, we applied the logistic regression to individuals in this excluded sample who at age 23 intended to have at least one more child, and we modelled the probability of not achieving this intention. Results (Table S5) indicate significant associations between BMI and the likelihood of not fulfilling this intention for both men and women. Specifically, underweight women are 20 pp more likely and overweight men 11 pp less likely to fall short of their fertility goals compared with those of a healthy-weight BMI. However,

the sample size for these models is rather small (1,024 women and 408 men), which may prevent some effects from reaching statistical significance.

Second, we used the KHB model to compare the effect of health on completed fertility and the probability of achieving ideal family size, both with and without accounting for potential mediators. We replicated the models twice, testing employment status and union history as mediators. While the KHB model facilitated the exploration of mediation mechanisms, we acknowledge that more comprehensive data would be necessary for a thorough mediation analysis: specifically, for detailed tracking of employment and union histories between the time health is measured at 23 and when fertility outcomes are observed at 46 (see Data and methods section). As such, we do not claim causal validity for our findings. Nevertheless, our approach provides a valid comparison of the effects of health on fertility both with and without these mediators, estimating the magnitude and significance of the mediated effects. Results are presented in Tables S6–S11. In some cases, employment significantly mediates the negative effect of poor health on both completed fertility and the probability of achieving ideal family size among men; this may reflect the breadwinner model prevalent in this generation. However, at least half of the total health effect on fertility remains direct. Evidence of union history mediating the health–fertility relationship can also be observed for both men and women, particularly for achieving ideal family size. For example, individuals reporting fair or poor health instead of excellent health display a higher likelihood of underachieving their ideal family size by two or more children (coefficients: 0.464 for women, 0.735 for men, both significant); of this gap, one-third to one-half is mediated by union history (mediated effect: 0.150 for women, 0.373 for men). Overall, these results suggest that while employment (among men) and union status (for both men and women) can significantly mediate the health–fertility link, a substantial portion of the effect

remains direct, supporting the idea that health in young adulthood exerts a considerable direct influence on fertility during the life course.

A final robustness check concerned the role of parental background as a proxy for early-childhood experiences that may confound health, fertility intentions, and fertility outcomes. While our initial intention was to maintain a parsimonious model and avoid over-controlling for educational and employment variables, we decided to repeat all the main analyses including a control for father's social class at the time of the respondent's birth to account for any potential bias. We found that father's social class was highly correlated with the respondent's educational level, and controlling for this variable did not alter our results (available on request). Therefore, we chose to present the models without controlling for father's social class in the paper.

Discussion and conclusion

Using longitudinal data from the 1958 NCDS, this study investigated the relationship between health measured in two ways during early adulthood (at age 23) and fertility outcomes at age 46 for men and women in Great Britain. The study yielded robust findings, revealing some sizeable and significant associations between health and fertility outcomes, while accounting for an extensive set of control variables.

We found that poorer SRH and a BMI outside the healthy weight range in early adulthood were both significantly associated with having fewer children and underachieving fertility ideals for men and women in this British birth cohort. Specifically, we found that poorer SRH was associated with having fewer children and underachieving fertility goals for men and with a lower chance of reaching ideal family size for women. For BMI, we found some evidence that men with an obese BMI were less likely to achieve their fertility goals. However, we found stronger evidence that women with an obese BMI were more likely to have fewer children and to underachieve their fertility goals. An underweight BMI was

weakly associated with a greater probability of women being childless at age 46. We therefore have some evidence to support Hypothesis 1, that being in poorer health (whether measured by BMI or SRH) results in fewer children for both men and women, although there is better evidence for this from the SRH measure, in contrast to Hypothesis 2.

The findings from this study also suggest differing effects of health on fertility for men and women: while SRH at 23 was strongly associated with men's fertility outcomes (which we did not predict; see Hypothesis 4), BMI was a stronger predictor for women in this sample as we expected (Hypothesis 3). This latter finding aligns with previous studies (Frisco and Weden 2013; Lee et al. 2023) and could suggest that BMI has a greater physiological effect on women's ability to have children than men's: the epidemiological, clinical, and genetic literature has extensively described the endocrinological mechanisms through which obesity can increase or exacerbate infertility (Talmor and Dunphy 2015; Craig et al. 2017). However, the differences between men and women could also be explained by social factors including partnership behaviour and weight-based stigma (e.g. in hiring practices). Not having a stable job can also affect the likelihood of having children (Alderotti et al. 2021). We explored this in a preliminary way through a KHB mediation model and did indeed find a stronger mediating role of employment for men than women, consistent with Hypothesis 5. We did not find a stronger mediating role of union status for men as expected (Hypothesis 6) based on findings by Liu et al. (2023), with proportion of a mediation present for both men and women.

Our results are notable for several reasons. First, the effect sizes remained significant even after controlling for factors widely acknowledged as strong predictors of fertility, suggesting that early-life health may play an important role in shaping family trajectories within this cohort. For example, our result of a 20 pp higher likelihood of being childless at age 46 for obese women is especially prominent in light of the fact that the average share of

childless women in our sample was 24.2 per cent. Similarly, for men reporting fair or poor SRH, the likelihood of achieving ideal family size was seven pp lower, which translates into a 20 per cent relative change, considering that the share of men achieving ideal family size in the sample was about 37 per cent. Second, given that this analysis did not account for the likely worsening of health over the life course (which could impact childbearing), these results are particularly striking. For example, a study using the same birth cohort found that the proportion with a BMI above the healthy weight range increased over time and that individuals with the highest BMI at age 23 typically displayed the highest BMI across the whole life course (Bridger Staatz et al. 2023). Neither of these factors was fully accounted for in our analysis. However, caution should be applied when interpreting our results about BMI and fertility because BMI was computed from self-reported height and weight: self-reporting may introduce bias, and the direction of this bias is unclear. While social desirability bias may lead individuals to under-report their weight (which would result in underestimation of the effect of obesity on fertility), previous research suggests that misreporting patterns differ by sex (see e.g. Betz et al. 1994).

Although the findings were sizeable when controlling for other explanatory factors, a notable pattern in the results was that rather than a graded effect between worsening health and fertility, our effects were statistically significant only for those in the most extreme categories of poor health. For example, results tended to be significant only for obese individuals rather than overweight individuals. It should be noted, however, that the effect sizes in these extreme categories were comparable to—and at times larger than—the effects of employment and education. The fact that we found significant results only for extreme categories is perhaps not surprising, as those with worse health at age 23 were also probably more likely to sustain poor health throughout their lives, while those who were in good health at 23 but moved into poorer health categories during the period of observation may have

diluted our findings. This again points to an important impact of poor health in early adulthood on fertility across the life course.

For both models (number of children and achieved ideal family size), we found patterns in outcomes by education, employment, and union history, with stronger and more consistent trends in the number-of-children models. For number of children, the models suggested that men and women who were higher educated and in a married/cohabiting union for longer were likely to have more children at age 46 than those who were lower educated or in a union for less time. The educational pattern we found for women is surprising, given that another study using this cohort has observed the opposite association (Berrington and Pattaro 2014), which probably reflects the selectivity of our sample. However, that 2014 study also noted a sizeable proportion of women with a degree in this cohort going on to have three or more children, which drove their finding that highly educated women were more likely both to under- and overachieve their ideal family size. This distribution is also likely the reason behind the association we observed for women's education and completed family size. For achieved family size, we found some evidence that lower-educated women and men/women who were in a union for longer were more likely to achieve their goals. Both these findings were also observed in the earlier NCDS study of fertility goals (Berrington and Pattaro 2014). These comparisons between our study and Berrington and Pattaro's highlight that the addition of health as an explanatory variable does not alter the important impacts of education, employment, and union history in driving fertility patterns in this cohort. However, our KHB mediation analysis also found that the role of health in fertility was not completely mediated through the pathways of employment and union formation, highlighting the potential of an important independent effect of early-adulthood health.

While the longitudinal aspect of this work constituted an important component of our paper, it also had limitations: longitudinal data will always suffer from sample attrition over

time, and due to the non-annual data collection, our main analysis had to be limited to individuals who had not had children by age 23 (corresponding to the first wave conducted during the cohort's adult lives). We imposed this exclusion criterion to address the endogeneity between health and fertility and also the known influence of parenthood on fertility desires and expectations (Iacovou and Tavares 2011). As discussed in the Additional analyses section, we ran separate models on individuals who had already had children by age 23, and results generally confirmed our take-home message: that poor health in early adulthood affects completed fertility and the realization of fertility intentions in a gendered way. However, we acknowledge that this criterion may still have introduced the possibility of selection effects into the final analytical sample, particularly in terms of health status and fertility. For example, the average family size of women in our sample was 1.6 compared with 1.99 among all women born in England and Wales in 1958 (ONS 2024) or 1.91 according to another study using this cohort at age 46 (Berrington and Pattaro 2014).

To examine the potential selection effects of the exclusions, we explored differences in the health explanatory variables at age 23 among those who were lost to follow-up (1,255 women and 1,799 men) or who had had a child before that age (1,278 women and 561 men). We found that these excluded individuals were more likely to be of overweight or obese BMI and less likely to report a BMI inside the healthy weight range at age 23 than the remaining sample. They were also less likely to report excellent health and more likely to report their health as fair or poor at age 23. The fact that poor health, together with low socio-economic status and being male, positively relates to dropout is an enduring finding in research about attrition in longitudinal studies (see e.g. Bowling et al. 2016). In this regard, it should be noted that we took all these variables into account in our models, as we controlled for health, education, and employment and stratified by sex. Using information about selection on available covariates in the data reduced the amount of residual (i.e. unexplained) variation

due to attrition, likely reducing bias due to selection on observables (see e.g. Alderman et al. 2001; Alderotti et al. 2022). As those lost to follow-up seemed to be in poorer health than the remaining sample, this would suggest that our estimates of the effects of health on fertility may be too low. However, the finding that those who had a child before age 23 were more likely to report a high BMI may have the opposite implication. Interestingly, this was also found by Lee et al. (2023), who noted that women with an obese BMI in their American sample were not delayed in their transition to first birth and that overweight women even made a faster transition than those with a healthy-weight BMI. However, overweight and obese women were still more likely than those with a healthy-weight BMI to remain childless. Another limitation of the sample selection was that we lacked information regarding whether individuals with poorer health had already revised their plans for having children downward prior to age 23. However, this would mean that our findings potentially underestimate the true effect of health on fertility outcomes. Among the data-driven limitations, we also highlighted earlier that the data at our disposal did not allow a proper investigation of the effects of mental health issues on fertility, a topic that may be of special interest (see e.g. Alderotti and Trappolini 2022). In this regard, we encourage future research to analyse how mental disorders in young adults (e.g. depression, anxiety) may affect fertility intentions and/or realization to provide a more comprehensive understanding of the health–fertility link.

A final limitation is that the analysed sample came from a unique cohort (i.e. 1958), which means that the findings may not be applicable to other high-income contexts. However, it should be noted that the prevalence of obesity in early adulthood has increased in recent birth cohorts across many high-income countries: the escalation of obesity rates has been a prominent global concern, with estimates indicating a tripling of global obesity between 1975 and 2016 (Jaacks et al. 2019). At the same time, research has consistently

shown that being overweight or obese can negatively impact fecundity (Hammoud, Gibson et al. 2008). Similarly, the incidence and prevalence of mental illness is also spreading among young people in recent birth cohorts (see e.g. McGorry et al. 2013), and poor mental health is well known to affect SRH negatively (Lachytova et al. 2017). Thus, given such secular shifts in health among the young, we argue that our results shed light on one aspect—the relationship between health in young adulthood and fertility—that will likely become more and more relevant in subsequent generations and is relatively unlikely to be affected by external validity issues related to the single-cohort composition of our working sample.

To conclude, this study makes a significant contribution to the demographic literature by establishing a novel link between two dimensions of health in early adulthood and subsequent fertility outcomes over the life course for both men and women. By elucidating the influential role of early-life health in shaping fertility trajectories, our analysis emphasized the importance of understanding and appropriately accounting for early-adulthood health as a potential determinant of future family size and fertility goals in demographic analysis. The sex-specific differences we detected underscore the intricate interplay between health and fertility outcomes, leading us to advocate for a holistic approach when investigating the factors that influence both men's and women's reproductive decisions. This study's unique longitudinal perspective adds valuable insights to the existing body of knowledge, paving the way for further exploration of the complex mechanisms connecting health with fertility dynamics and encouraging those analysing fertility in high-income contexts to pay greater attention to the role of health.

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