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How does the age of the youngest child affect parental survival?

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Abstract

Previous research has investigated several different aspects of the relationship between having a child and parental mortality. One aspect of research that has been neglected until now is the age of the child. If children have an effect on parental mortality, this is likely to change as they grow up. We apply hazard regression models to longitudinal Swedish register data of the total population for men and women separately. Adjusting for a variety of control variables, we find that parents with younger children experience a substantive mortality advantage compared to parents—of the same age—who have older children. The mortality advantage decreases gradually as the age of the youngest child increases. Robustness checks confirm that this result cannot be explained by differences in the parent's age and parental age at first birth. Childless women and men of the same age experience the highest mortality. Additional models for different causes of death suggest that selection, behavioral changes, and unobserved protective effects contribute to this pattern.

Keywords: Mortality, Age, Parental survival, Family, Parity, Selection, Survival analysis

Introduction

Throughout human history, it has always been true that giving birth and raising children poses a risk for the health of the mother. It is only during the course of the last century that these medical risks have been greatly reduced. Today, children may pose a risk in other respects, for example for the parent's labor market prospects and through the double burden of managing both employment and parenthood. Having responsibility for raising a child also involves substantial changes in the parents' lifestyles. Parents change their behaviors to meet their new obligations, but they also have less time to perform their usual and perhaps more risky activities. We argue that while all these factors could have an effect on the health of the parents, the magnitude of their impact may depend on whether a child is a baby, a teenager, or an adult. This paper investigates the association (and not the causal effect) between how a child of different ages affects parental mortality in different ways, which causes of death are affected the most, and what factors may be responsible for this.

Background

Previous research has investigated several different aspects of the relationship between having a child and parental mortality. The first aspect refers to maternal mortality connected to the birth of a child. In the past, maternal mortality was one of the most prominent causes of death among women of reproductive ages, but due to medical advances this risk has nearly disappeared in most industrialized countries. Nevertheless, maternal mortality is still observable today, especially in developing countries where women experience an elevated risk of death during the postpartum period, lasting from about 12 weeks in Nepal (Pradhan et al., 2002) to up to three years in Bangladesh (Lane, 2004; Menken et al., 2003). These results might not be transferable to more developed countries because the conditions surrounding childbirth are completely different, but they are an indication that giving birth potentially has an effect on maternal mortality that lasts longer than is usually studied. Although empirical evidence remains inconsistent, the WHO assumes that women remain vulnerable during the postpartum period for up to one year (World Health Organization, 1992).

A second aspect that has been investigated by earlier studies involves post-reproductive aging from an evolutionary perspective on the link between childbearing history and survival (Ellison, 2009; Kirkwood & Rose, 1991). A prominent theory that attempts to identify these patterns in humans is the cost-of-reproduction hypothesis. This states that the post-reproductive survival of mothers decreases with an increasing number of births, because using resources for reproduction reduces the available resources for maintenance, which consequently leads to a decrease in the length of life. In animals, evidence for the cost-of-reproduction hypothesis can be found in various species (e.g., Golet et al., 2004; Lester et al., 2004; Partridge et al., 1987). In plants, a review by Obeso (2002) concluded that most case studies generally support the predictions of the cost-of-reproduction hypothesis. In humans, there is some evidence in Amish people that increasing parity is correlated with a number of adverse health outcomes for women, such as obesity, diabetes, and cardiovascular disease (McArdle et al., 2006), while it probably lowers the risk of breast and ovarian cancer (Smith et al., 2002). A study conducted by Doblhammer (2000) also supports the proposition that reproduction comes at a cost. Using contemporary English and Austrian data, the author shows that women of higher parities experienced a higher mortality risk later in life than women with only one or two children. Similarly, excess mortality was found for childless women. By using historical data on aristocratic British families, Doblhammer and Oeppen (2003) also found a strong positive correlation between parity and mortality later in life. Even after controlling for health differences and mortality selection, the results remained significant. Looking at historical and modern register data in Utah and Quebec, Gagnon et al. (2009) also found a negative influence of parity on post-reproductive survival. The cost of reproduction may also be economic and indirectly affect the health of the parents. This is particularly true for mothers, who are penalized the most by their new status in the labor market (Aisenbrey et al., 2009). Despite these indications, the question of whether reproduction negatively affects human longevity remains unresolved (Barclay & Kolk, 2019; Doblhammer & Oeppen, 2003). For example, recent studies have found a protective effect of having more children on biological aging (Barha et al., 2016) and that experiencing more pregnancies exposes women to higher levels of hormones (e.g.,

estradiol), which indirectly protects cells from aging (Aviv, 2004) and also decreases the risk of certain health problems such as breast, ovarian, and uterine cancer (Grundy & Kravdal, 2010). Consistent with this mechanism, a study of cause-specific mortality found a lower risk of breast, uterine, and ovarian cancer among high parity women (Barclay et al., 2016). In two recent meta-analyses, Zeng et al. (2016) and Högnäs et al. (2017) concluded that there is a nonlinear association between parity and maternal mortality, but that the nonlinear association is moderated both by parents' sex and by cohort.

A third aspect that connects the mortality of women to their fertility history is related to heterogeneity, represented by differences in the health status among female populations. Women who are able to achieve pregnancy are generally recognized to be healthier on average than other women (Beeton et al., 1900; Hyppönen et al., 2005; Ronsmans et al., 2001). On the other hand, underlying health problems at younger ages could affect both fertility and health in later life, causing a spurious relationship between number of children and mortality (Meyer & Modig, 2021). If it is assumed that coital inability is associated with a serious disease, there is a reduction in longevity for women with few children (Smith et al., 2002). This healthy-pregnant-woman effect is also supported by the results of Perls et al. (1997) and McArdle et al. (2006). Doblhammer (2000), Smith et al. (2002), Mirowsky (2005), and McArdle et al. (2006) found that women who give birth to a child at later ages will experience higher post-reproductive survival. These results are also supported by historical data (Mueller, 2004), and are consistent with evolutionary theories proposed by Rose et al. (1997). In addition, early childbearing is associated with worse health and mortality, because it indicates negative social selection as well as interruptions in educational and labor market carriers, and higher risks of single parenthood and partnership disruptions (Berkman et al., 2015; Hobcraft & Kiernan, 2001; Huisman et al., 2003; Kane et al., 2013). However, Gagnon (2009) found little evidence that early fertility affects post-reproductive survival.

Social characteristics are a further aspect that also could have an influence on parental mortality. Individuals with greater access to social support are thought to be healthier and to experience lower mortality (Smith et al., 2002). Children are known to be one of the most important components of social support, and thus might have an effect on the longevity of their parents. Until now, the possible underlying mechanisms have not been well understood, but it is assumed that direct social support from children in the event of illness or an age-related need for help are the main reasons why parents live longer (Smith et al., 2002). A recent study in Sweden found that the positive effect of children's social support on increasing longevity is particularly strong in old ages (Modig et al., 2017). Having a large number of children is not only associated with greater access to social support; a large family may also be indicative of happier marriages, which may in turn be associated with an extended life span (McArdle et al., 2006). Having children might also increase social support indirectly because having more children might increase material support from the mother's network (Barha et al., 2016). Evolutionary anthropologist theories speak about 'allomaternal care', such as grandparents or relatives who support mothers in child-rearing, and more children may lead to greater support, reducing the energy spent and slowing the process of cellular aging (Kramer and Ellison, 2010; Gurven et al., 2012; Kramer, 2005; Meehan et al., 2013).

Most of the studies cited above, and indeed most of the existing research regarding the effect of children on parental mortality, are mainly focused on mothers. Investigations on men are few and limited (see Barclay et al., 2023 for an overview), such as the study by Hyppönen et al. (2005), which indicates that the effects among men might be different from those seen in women, and that later reproduction was associated with improved longevity in women but not in men. A Swedish study, however, found that men who have children had lower mortality on average than men without children. Even after controlling for socioeconomic factors, lone non-custodial fathers and lone childless men were found to have the greatest risks of premature mortality from all-cause mortality, but in particularly from injury, addiction, and ischemic heart disease (Weitoft et al., 2004). Similar results have been found more recently by Barclay and Kolk (2019). Jensen et al. (2009) also found that semen quality may be a fundamental biomarker of overall male health and mortality, supporting the idea that health selection into parenthood might be associated with (old-age) mortality for men as well. Previous research has also investigated behavioral changes among men after the birth of a child. Blackburn et al. (2005) studied the smoking behavior among fathers of newborn children. Like smoking by mothers, smoking by fathers has been linked to adverse health outcomes, including sudden death, asthma, and other respiratory diseases. In an earlier study, it was found that smoking cessation at childbirth was much less likely among men than among women (Brenner & Mielck, 1993). Blackburn et al. (2005) found that the vast majority of men changed their smoking behavior in one or more respects. It is assumed that economic and social pressure to quit smoking are the most important reasons behind these behavioral changes. The same mechanism could probably lead to sample problems and false reporting, because smoking in the presence of infants is seen as irresponsible by the public (Blackburn et al., 2005). Parenthood also encourages better integration into the community, and this has a particularly protective effect for men (Knoester & Eggebeen, 2006). However, parents have less time for exercise (Bellows-Riecken & Rhodes, 2008), and obesity seems to be associated with the number of children for both middle-aged women and men (Weng et al., 2004).

Until now, scientific interest has not been focused on the entire period of parenthood, but mainly on the mortality of the mother, either shortly after giving birth or past the reproductive phase. Other possible aspects, like the effect of parental stress on survival during their children's adolescence, have been neglected. However, this relationship in particular may have gained importance with the ongoing modernization of societies, the changes women's roles, and the increase in women's employment. As women try to combine work and motherhood, having children has become much more demanding—not only for women, but also for men, who have become increasingly involved in parenting. These considerations about combining marital, parental, and work roles are incorporated into several—sometimes conflicting—hypotheses. The multiple-role hypothesis concludes that the triple burden of being a mother, a wife, and a paid employee has adverse health effects, mainly because of a role conflict (conflicting expectations and demands) and a role overload (having too much to do). By contrast, the role accumulation hypothesis suggests that the benefits of having several roles will outweigh the harmful effects, mainly due to a higher income and additional social relationships outside the home and the marriage (Martikainen, 1995).

A further aspect of research that has been neglected until now is the age of the child. If there is an effect of children on parental mortality, it is likely that this effect will change with the age of the child because the demands placed on parents by children change as they grow up. To our knowledge, only one study analyzing mothers' mortality has controlled for the age of the child. In order to calculate age-standardized relative mortality rates, Martikainen (1995) applied Poisson regression on the Finnish census records of 1980, which were linked with all deaths during the period 1981–1985. Regarding the effect of the age of the youngest child, the authors found that all-cause mortality is lowest for mothers who have a child younger than two years of age. For this age group, age-standardized mortality is only 66% of the mortality of women with children aged 16 or older. Starting from this low level, mortality increases with the age of the youngest child, but even mothers with a child aged 16 years or older have decreased mortality relative to women who do not have any children living in the household. As this aspect was not the main focus of his paper, Martikainen (1995) does not make any assumptions about possible explanations for this finding. In addition, the study is limited, as it only includes children living in the household. The study is also not able to investigate how the effect of children's ages is influenced by other variables, such as parental characteristics like education, socioeconomic status, and marital status. This study indicates that the impact of children on mothers' mortality changes substantially with age. However, virtually nothing is known about the causes of this effect, how it is connected to other aspects of parenthood, and whether we find similar effects in fathers.

Hypotheses

We indicated in the previous section that there have been virtually no studies focusing on the association between the age of the youngest child and parental mortality. Therefore, we will examine two conflicting hypotheses for this association for all-cause mortality.

According to the first hypothesis, we argue that the mortality of parents with younger children is probably elevated, particularly for women. This is attributable first to possible adverse short-term effects of pregnancy, and second to the considerable demands of caring for young children. The stress associated with child care may lead to worse parental health, and thus to higher mortality levels. This could also be reflected in an analysis of specific cause of death, with an elevated risk for parents of young children from deaths attributable to circulatory diseases. Following this reasoning, having an older child could even be a protective factor and thus increase parental survival compared to those with very young children, also resulting in lower risk of death from circulatory and neoplasm diseases.

However, there are also a number of arguments that support the idea of lower mortality for those with young and especially very young children. For example, previous research has indicated that parenthood is associated with healthier lifestyle. Parents may feel obliged to reduce or give up some hazardous health behaviors, such as smoking, or they may feel less inclined to participate in—or have less time for—higher-risk leisure activities. However, both these aspects might be more significant for parents with younger children and less significant for those with older children, and lifestyle changes might be more pronounced in women than in men. This could result in a decrease in

deaths attributable to external causes such as accidents and suicide, as well as from circulatory diseases. A second argument that supports the idea of lower mortality for those with younger children is health selection. Previous studies have shown that couples who are able to have a child are a selected subgroup that is healthier on average than couples who are not able to have a child. This could be because individuals who are less healthy are also less likely to be able to find a partner, and consequentially less likely to have a child. It is also likely that individuals with severe illnesses are postponing childbirth. Both these forms of selection would lead to reduced mortality for parents of young children, but their effect may vanish when the child gets older and lead to a decrease in the mortality advantage with age. Following the cost-of-reproduction hypothesis, which describes possible long-term detrimental effects of pregnancy and parenthood, this reduction could potentially accelerate with age, resulting in higher risk of neoplasm and deaths from circulatory diseases with the increasing age of the child.

It is likely that the impact of children on parental mortality will differ for fathers and mothers. Giving birth to a child has long-lasting effects on the female body, and fathers are generally less involved in parenting and child-rearing than mothers. In contrast to most other countries, Scandinavian countries like Sweden are usually seen as egalitarian welfare states, which means that social and family policies aim to involve both sexes equally in parenting. Nevertheless, we assume that the change in mortality with the age of the child is less pronounced in fathers, but it is likely that the gender differences will be smaller than they would probably be in other countries. Also, if an increase in deaths from circulatory diseases is found when the child is young, this will be particularly true for women. On the contrary, a decrease in deaths from external causes such as accidents and suicide when the child is young will be more evident among men.

Data and methods

Data

The data that are analyzed here include a total of 4,491,289 Swedish-born individuals aged 26 to 50 who were living in Sweden between January 1, 1991 and December 31, 2012, and who had no more than three children. 2,315,678 are male and 2,175,611 are female. Having four or more children is relatively rare in contemporary Sweden, and those families are likely to be selected based on a number of unobserved characteristics. To limit the influence of these high-order births and families on the results of the analysis, we decided not to include them. An explanation for the chosen age range is given in the methods section.

Thus, there are two ways for individuals to enter the study by:

- being 26–50 years old on January 1, 1991 *and* being childless or having three or fewer children, of whom the youngest child is less than 21 years of age; or
- reaching age 26 between January 2, 1991 and December 31, 2012 *and* being childless or having three or fewer children.

There are seven ways to exit the study by:

- dying between January 2, 1991 and December 31, 2012;

- reaching age 50 between January 2, 1991 and December 31, 2012;
- becoming the parent of a fourth child between January 2, 1991 and December 31, 2012;
- experiencing the death of a child between January 2, 1991 and December 31, 2012;
- celebrating the 21st birthday of the youngest child between January 2, 1991 and December 31, 2012;
- being alive on December 31, 2012; or
- emigrating from Sweden between January 2, 1991 and December 31, 2012.

On average, each individual was observed for 11.1 years, which adds up to a total of 49.98 million person years. A total of 33,929 men and 18,826 women from the study population died between 1991 and 2012. An overview of the distribution of time at risk measured in days for men and women for all covariates is shown in Table 1. In the appendix, we also present a flow chart (Fig. 5) outlining all the steps to identify our study population.

The proportion of missing values in this data set is very low. Missing information is most common for *highest achieved educational degree* (0.31%) and *income* (0.13%). In most instances, information on income is systematically missing in the year of death and in the year of emigration, and we thus decided to impute income information from the year before both events.

The variables *date of migration*, *date of birth*, and *date of death* are measured with monthly precision, and are used to define an individual's time periods at risk. The variable *personal identification number of the youngest child* is used to establish the linkage between parent and child. The variables *income*, *education*, *civil status*, *parity*, *age of the youngest child*, and *period* were coded as time-varying covariates. Parity and age of the youngest child refer to own biological children, regardless of whether or not they live in the household. The same applies for the variables that indicate whether the individual is childless. *Sex* is treated as a time-constant covariate. To investigate the mortality pattern for different causes of death, a variable indicating the underlying *cause of death* was used to recode the failure/censoring indicator.

For the analyses of causes of deaths, we identified the underlying cause of death for each observed deaths and categorized it according to three broad chapters following the 9th and 10th revisions of the International Classification of Diseases and Related Health Problems (ICD): (1) neoplasms—ICD-9 chapter II and ICD-10 chapter II, (2) diseases of the circulatory system—ICD-9 chapter VII and ICD-10 chapter IX, and (3) external causes of mortality—ICD-9 chapter XVII and ICD-10 chapter XX. We excluded suicide and intentional self-harm from (3), and included those deaths as a separate category: (4) suicides—ICD-9 codes 950–959 and ICD-10 codes X60–X84. Among the Swedish adult population aged 25–49 between 1997 and 2012, these four cause of death groups comprise about 80% of all observed deaths among both men and women. Among men, roughly 40% of all deaths are from external causes of morbidity and mortality (vs. about 25% among women). Neoplasms account for roughly 20% of all deaths among men and 45% of all deaths among women, while circulatory diseases account for about 20% of all deaths among men and 10% among women (Socialstyrelsen, 2023).

Table 1 Deaths and exposure time to the risk of death for men and women (Jan 1, 1991–Dec 31, 2012)

Covariate	Women			Men		
	Person years		Deaths	Person years		Deaths
	In thousands	%		In thousands	%	
Age of the youngest child						
0	1337.9	5.54	183	1461.4	5.66	448
1	1370.7	5.67	247	1456.9	5.64	629
2 to under 4	3176.6	13.14	929	3197.1	12.38	1955
5 to under 9	4093.3	16.94	2047	3871.1	14.99	3611
10 to under 17	5238.4	21.68	4663	4384.2	16.98	6387
18 to under 21	2672.5	11.06	4357	1488.2	5.76	4137
Childless	6277.0	25.97	6400	9958.9	38.57	16,762
Calendar period						
1991–1994	4645.6	19.22	4475	4927.2	19.08	8148
1995–1999	5631.6	23.30	4658	5991.5	23.21	8407
2000–2004	5473.9	22.65	4065	5855.0	22.68	7264
2005–2009	5303.7	21.95	3605	5697.6	22.07	6474
2010–2011	3111.6	12.88	2023	3346.4	12.96	3636
Highest achieved education						
ISCED 1 and 2	2772.1	11.47	4623	4254.5	16.48	10,571
ISCED 3	7833.5	32.41	7059	9004.0	34.88	13,185
ISCED 4	4274.1	17.69	2253	4470.4	17.32	4149
ISCED 5	4096.6	16.95	2232	3795.5	14.70	2853
ISCED 6 and 7	5133.8	21.24	2287	4193.7	16.24	2529
Unknown/missing	56.3	0.23	372	99.6	0.39	642
Income quintile						
1	5820	24.08	4132	4351	16.86	7417
2	5538	22.92	4775	4302	16.66	7131
3	4700	19.45	4173	4706	18.23	6635
4	4330	17.92	3239	5459	21.14	6205
5	3747	15.51	2507	6960	26.96	6539
Unknown/missing	30	0.13	0	36	0.14	2
Parity						
0	6277	25.97	6400	9959	38.57	16,762
1	4298	17.78	3555	4492	17.40	5917
2	9594	39.70	6076	8137	31.52	7833
3	3998	16.54	2795	3230	12.51	3417
Marital status						
Never married	11,199	46.34	7575	9947	38.53	8042
Married	10,716	44.34	8062	14,137	54.76	21,127
Dissolved union	2251	9.31	3189	1732	6.71	4760
Total of each variable[a]	24,116	100.00	18,826	25,817	100.00	33,929

Source: Own calculations based on Swedish register data

[a] Total may not always add up to these values due to rounding

We consider seven levels of highest educational level: 1 = not completed compulsory education (< 9 years), 2 = completed compulsory education (9 years), 3 = upper secondary (2 years), 4 = upper secondary (3 years), 5 = college/university < 3 years, 6 = college/university ≥ 3 years, 7 = research education

Methods

We apply hazard regression models to examine the influence of the age of the youngest child on the individual's mortality. Hazard regression, also called event-history analysis or survival analysis, represents the most suitable analytical framework for studying the time-to-failure distribution of events for individuals over their life course. The general proportional hazards regression model is expressed by:

$$h(t|X_1,\ldots,X_k) = h_0(t)\exp\left(\sum_{j=1}^{k}\beta_j X_j(t)\right), \tag{1}$$

where $h(t|X_1, \ldots, X_k)$ is the hazard rate for individuals with characteristics $X_1, \ldots, X_k$ at time t, $h_0(t)$ is the baseline hazard at time t, and β_j, $j=1, \ldots, k$ are the estimated coefficients of the model.

We use the Cox proportional hazards model proposed by Cox (1972), which is a semi-parametric transition rate model. The underlying time process of the Cox model is biological age of the studied individuals. Its main property is that it makes no specific assumption about the shape of the baseline hazard $h_0(t)$. As we are only interested in the direction and magnitude of the effects of observed covariates, and not in the interpretation of a baseline hazard, the Cox model is a reasonable choice here. All regression models were calculated for men and women separately. All analyses are carried out using the *st* family of commands in Stata 15.1.

Our study population consists of all individuals aged 26–50. We chose this age range to keep structural zeros at a minimum, e.g., to avoid including combinations of parental age and child age that are implausible or impossible. At younger parental ages, no parents with children aged 18–21 would exist in the data, while at older ages, no or extremely few parents with children aged 0–1 would exist in the data. In respect of the hazard regression, this choice should facilitate the proportional hazards assumption being fulfilled and thus minimize bias in our estimates. For each model, we carry out standard tests for the proportional hazards assumption.

In the first part of our analysis, we estimate five separate regression models for all-cause mortality, controlled for age of the individual. Models M1 and M2 are the main models that include all individuals; Model M1 controls only for *age of the individual* and *age of the youngest child*, and Model M2 additionally controls for *income*, *education*, *civil status*, *parity*, and *period*. Models M3–M5 provide robustness checks for our estimates. In Model M3, we do not control for *parity* to assess whether there is an association between *parity* and our main variable of interest—*age of the youngest child*. In Model M4, we only include parents (because childless individuals are likely to differ regarding a number of unobserved characteristics that may potentially influence our earlier estimates). Model M5 includes only individuals aged 34–36. This model is the most important of our robustness checks. It only includes men and women of a narrow age range at which children of all ages can be observed. This model tests whether parental age has been properly accounted for and can be separated from the effect of the age of the youngest child in the previous models. Additionally, age at childbearing should have no or only a very minor effect at these ages. For example, a 35-year-old parent of a 1-year-old should not differ from a 35-year-old parent of a 0-year-old based on the age at which they had

these children (age 35 vs age 34). We argue that mortality differences between these two individuals can instead be attributed to currently having children of different ages.

In the models without parity (M1 and M3), the mortality risk for childless individuals is compared to individuals who have children below 1 year of age. In the models that include *parity* in addition (M2 and M5), the reference category is necessarily different. Here, childless individuals are compared to those men and women who have two children *and* whose youngest child is below 1 year of age.

We also explore whether the effect of the age of the youngest child varies according to different causes of death. Overall, previous studies have found that reproductive history and post-reproductive mortality may have different associations for different causes of death (e.g., Barclay et al., 2016). In the second part of our analysis, we will estimate survival models separately for deaths from neoplasm, diseases of the circulatory system, external causes, and suicide (Kleinbaum & Klein, 2005). If one specific cause of death is the event of interest, all other death events from other causes would be treated as censored, in addition to the censored observations that were already observed in the model for all-cause mortality. Thus, for every cause of death of interest, a separate cause-specific hazard function is calculated. Given the assumption that all competing risks are independent of each other, these cause-specific hazard rates are identical to the marginal hazard rates (Klein & Moeschberger, 2003). Estimating all causes of deaths simultaneously is therefore statistically equivalent to the estimation for each cause of death separately (Kalbfleisch & Prentice, 2002; Prentice, 1978).

Results

Mortality from all causes of death

The results for mortality from all causes by the age of the youngest child for the whole observation period from January 1, 1991 until December 31, 2012 are shown in Fig. 1 (women) and Fig. 2 (men). Both figures consist of five separate curves, showing the

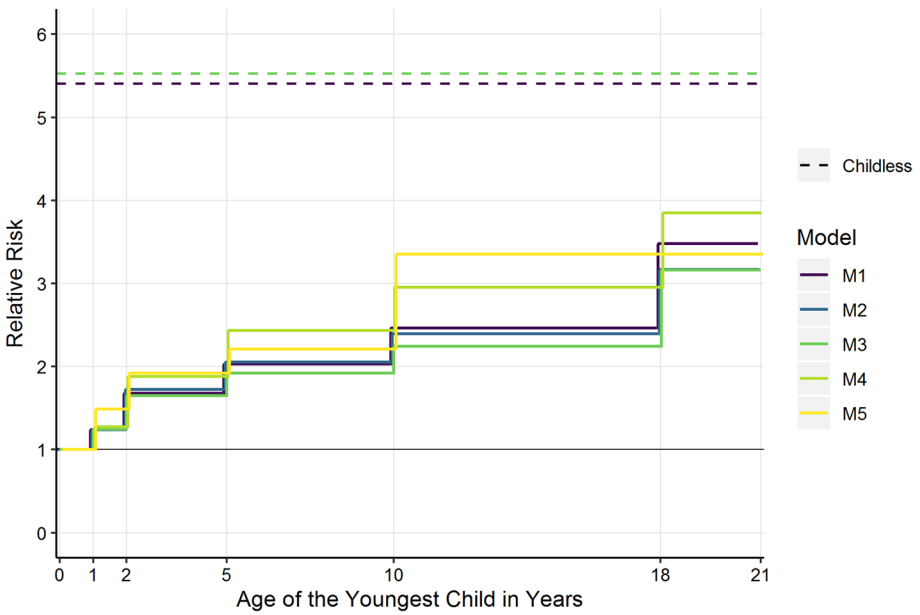

Fig. 1 Models 1–5 (women): relative risk of dying by the age of the youngest child

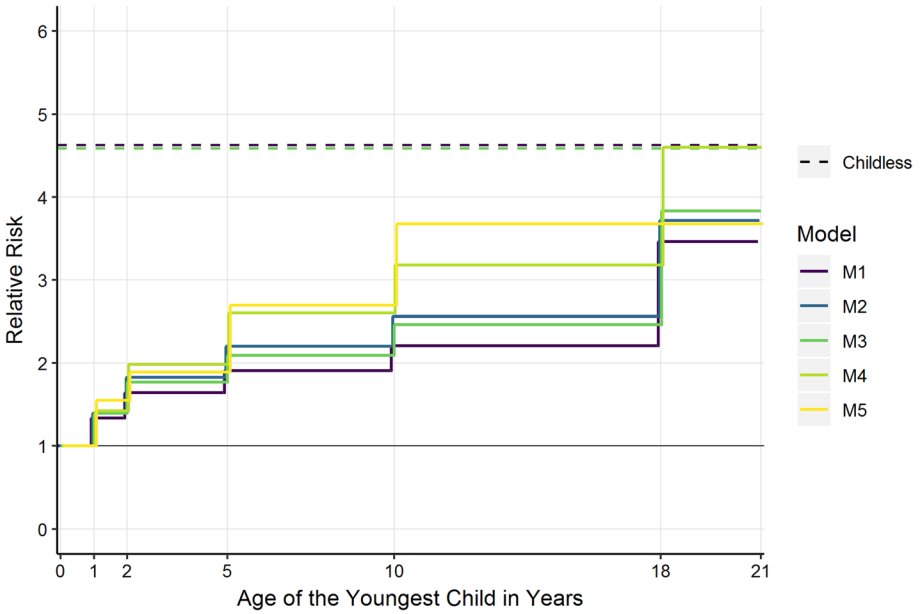

Fig. 2 Models 1–5 (men): relative risk of dying by the age of the youngest child

relative risk of dying by age of the youngest child. The reference category comprises all index individuals who have a newborn child. The relative mortality risks for individuals who do not have any children are given by the dashed lines. In Models M1 and M3, the mortality risk for childless individuals is compared to individuals who have newborn children. When parity is included in Models M2 and M5, the reference category for childless individuals changes, and is then represented by all individuals who have two children *and* whose youngest child is newborn. For these two models, the point estimates for the childless dummy are presented together with the results of the additional covariates in Table 2.

The first model (M1) includes the *age of the youngest child* and the indicator for being *childless* as sole variables. It gives the effect of having one or more children on a subject's survival chances, and how this effect changes by the age of the youngest child. Thus, this model is only adjusted for these two variables and for the age of the parent being studied. It shows that mortality from all causes is lowest for mothers and fathers whose child is less than one year old. As the age of the child increases, parental mortality also rises. Compared to mothers of a newborn, childless women of the same age experience a mortality risk that is more than five times higher. Similarly, mothers of the same age whose youngest child is 5–10 years old experience a two times higher mortality risk, while those who are mothers of an 18- to 21-year-old child show a 3.5 times elevated risk. We find a comparable pattern in fathers, with the lowest mortality in the reference group of fathers of a child less than 1 year old. Childless men of the same age experience a mortality risk 4.6 times higher. Fathers of the same age who have a child aged 18–21 show a mortality risk about 3.5 times higher than the risk faced by the reference group.

In the second model, all additional control variables are introduced. For both sexes, the mortality pattern by the age of the youngest child changes only slightly, but differently for men and women. Controlling for these additional factors increases the mortality

Table 2 Models 2–5 (women): hazard ratios of all-cause mortality for all other covariates

	M2	M3	M4	M5
Highest achieved education				
ISCED 1 and 2	1	1	1	1
ISCED 3	0.663***	0.658***	0.723***	0.542***
ISCED 4	0.514***	0.512***	0.582***	0.376***
ISCED 5	0.462***	0.459***	0.554***	0.339***
ISCED 6 and 7	0.424***	0.422***	0.523***	0.354***
Unknown/missing	2.958***	2.949***	2.526***	
Marital status				
Never married	1	1	1	1
Married	1.173***	1.272***	1.102***	0.905
Dissolved union	1.567***	1.611***	1.539***	1.731***
Income quintile				
1	1	1	1	1
2	0.978	0.996	0.879***	0.952
3	0.838***	0.866***	0.789***	0.824
4	0.598***	0.618***	0.638***	0.669***
5	0.482***	0.504***	0.537***	0.415***
Unknown/missing	0	0	0	
Calendar period				
1991–1994	1	1	1	1
1995–1999	0.880***	0.877***	0.878***	0.921
2000–2004	0.811***	0.806***	0.818***	0.857
2005–2009	0.737***	0.731***	0.722***	0.779*
2010–2011	0.711***	0.704***	0.681***	0.877
Parity				
Childless	6.683***	5.526***		9.151***
1	1.580***		1.582***	1.770***
2	1		1	1
3	0.976		0.992	0.879
Observations	22,968,029	22,968,029	18,082,596	2,849,706
Failures	18,826	18,826	12,426	918
Person days	8,826,777,892	8,826,777,892	6,534,117,682	733,021,569

Source: Own calculations based on Swedish register data

*$p < .05$

**$p < .01$

***$p < .001$

advantage of having a young child for men. All control variables behave as expected (see Table 3). The effects of income and highest achieved educational degree are roughly in line with the literature, which means that mortality decreases considerably with rising income. The decrease is less pronounced for women than for men. Similarly, the chance of survival is shown to increase with the degree of education. All in all, this model suggests that socioeconomic variables play a minor role in explaining the effects by the age of the youngest child. Survival rates increase with time, especially for men. Regarding marital status, the analysis showed that—compared to married people—mortality is higher for never married and highest for dissolved union. The parity of the subject also explains some of the mortality differentials in the studied population. Compared to men with two children, we observed an increase in mortality among men who are childless

Table 3 Models 2–5 (men): hazard ratios of all-cause mortality for all other covariates

	M2	M3	M4	M5
Highest achieved education (ISCED)				
1 and 2	1	1	1	1
3	0.777***	0.775***	0.808***	0.660***
4	0.592***	0.590***	0.630***	0.499***
5	0.488***	0.485***	0.546***	0.389***
6 and 7	0.437***	0.434***	0.530***	0.320***
Unknown/missing	1.955***	1.961***	1.590***	
Marital status				
Never married	1	1	1	1
Married	1.930***	2.074***	1.873***	1.825***
Dissolved union	2.834***	2.917***	2.798***	3.256***
Income quintile				
1	1	1	1	1
2	0.943***	0.947**	0.848***	0.909
3	0.760***	0.770***	0.719***	0.791**
4	0.541***	0.549***	0.578***	0.550***
5	0.395***	0.402***	0.463***	0.416***
Unknown/missing	0.0210***	0.0214***	0.0568**	
Calendar period				
1991–1994	1	1	1	1
1995–1999	0.828***	0.825***	0.822***	0.839**
2000–2004	0.728***	0.725***	0.727***	0.725***
2005–2009	0.683***	0.680***	0.674***	0.701***
2010–2011	0.650***	0.647***	0.642***	0.746**
Parity				
Childless	5.489***	4.590***		6.239***
1	1.483***		1.446***	1.648***
2	1		1	1
3	0.992		1.022	1.039
Observations	23,747,765	23,747,765	16,667,989	3,064,132
Failures	33,929	33,929	17,167	1878
Person days	9,429,957,202	9,429,957,202	5,792,461,634	793,061,669

Source: Own calculations based on Swedish register data

*$p < .05$

**$p < .01$

***$p < .001$

or have one child. The results are also consistent for women (Table 2). Model M3 shows similar patterns, underlining that—for both men and women—our results are not biased by an association between age of the youngest child and parity.

When we do not include childless individuals, the mortality pattern by the age of the youngest child is attenuated considerably for both men and women (Model M4), strengthening the impression that mortality is much lower when the child is younger. The effects of the remaining covariates are rather stable. To check the robustness of our results, we only consider men and women aged 34–36 in Model 5. Due to the lower number of failures, we decided to aggregate all parents of children aged 10 and older in this model. However, the results support the conclusions drawn from our main Model 2. In addition, this model also shows that the effect of the age of the youngest child cannot

be explained by parental age and parental age at birth. Compared to fathers and mothers of a newborn, parents of 1-year-old children experience about 50% higher mortality, and parents of children aged 2–5 almost twice as high mortality. All these children were born to mothers and fathers aged 29–36, ages at which negative selection into early parenthood and positive selection into late parenthood should play no role or only a very minor role, especially for men.

Visual and numerical inspections of the proportionality assumption show no problems for our main independent variable *age of the youngest child* across all presented models, but do reveal some interaction between the baseline and socioeconomic categories. To account for the potential impact, additional models were conducted that stratify by education and income, allowing the baseline hazard to freely vary by educational and income level. We found no changes in the point estimates for our main independent variable of interest. Further, in Model 5, we found no violations of the proportional hazards assumption, either graphically or numerically.

In sum, in this study we found a distinct mortality pattern by the age of the youngest child. Fathers and mothers of a newborn experience by far the lowest mortality risk, which increases slowly with the age of the child for both sexes. Introducing covariates did not substantively alter this pattern. Robustness checks confirm that this result cannot be explained by differences in the parental age at first birth.

Mortality by causes of death

We will now examine the pattern of the main variables of interest for different causes of death. We will study mortality variations for neoplasms, diseases of the circulatory system, external causes, and suicide. The results are shown in Figs. 3 and 4.

Women: The risk of women dying from cancer is exceptionally low among mothers of newborns. For mothers of the same age who have a child aged 1 to under 2,

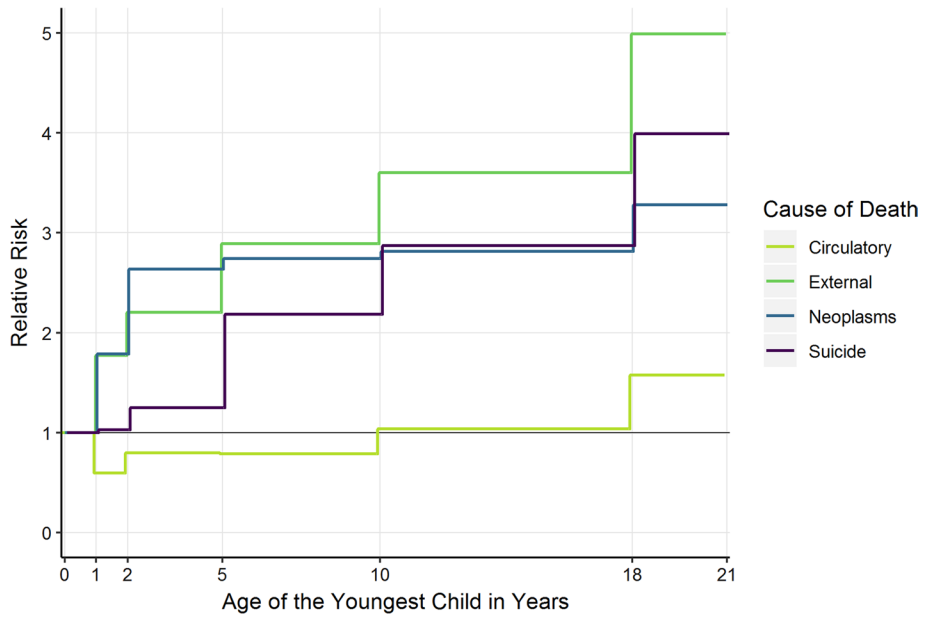

Fig. 3 Models 6a–6d (women): relative risk of dying by the age of the youngest child for selected causes of death

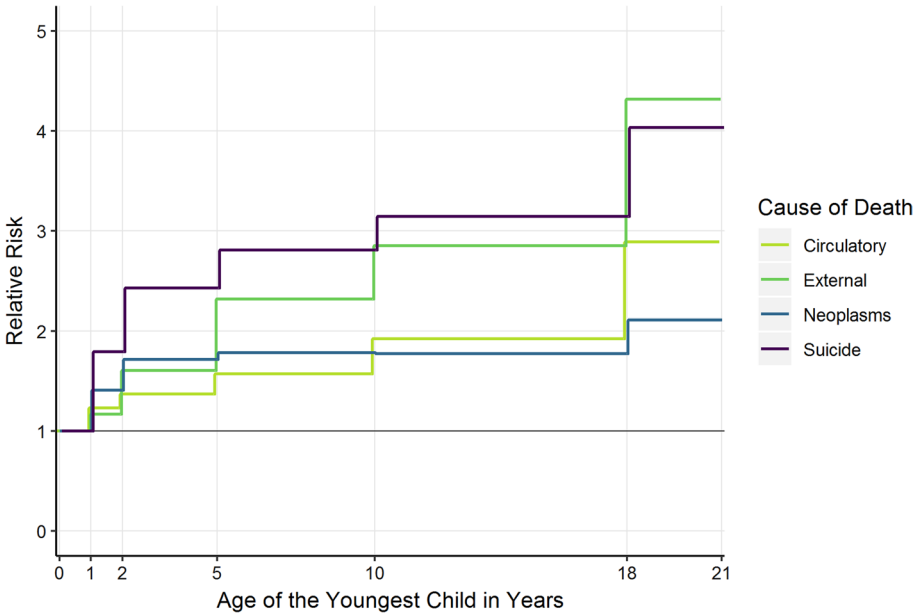

Fig. 4 Models 6a–6d (men): relative risk of dying by the age of the youngest child for selected causes of death

mortality is elevated by 80%, while for those who have children aged 2 to under 5, mortality increases 2.6-fold. The increase becomes less steep thereafter, but peaks for women of adult children, whose mortality is about 3.3 times higher than for women in the reference group. The mortality advantage for cancers for mothers of very young children is more pronounced than for all-cause mortality, but is less pronounced for mothers of children older than 2 years of age.

For circulatory diseases, we also observe a distinct pattern. While mortality was found to be lowest among parents of newborns for all other causes of death, the risk of dying from circulatory diseases decreases in the first years of motherhood. Compared to the reference group comprising mothers of newborns, the lowest mortality rates were found for mothers of children aged 2 to under 10. For them, the risk of dying is about 20% lower than for the reference group. However, the differences are not statistically significant.

For external causes, the risk of dying is again exceptionally low among mothers of newborns and increases again as the age of the youngest child rises. Compared to all-cause mortality, excess mortality is higher for women with children above the age of 10. The observed suicide risk is also very low for mothers of young children, particularly when they are below 5 years of age. Having children is less protective against suicide for mothers of older children, and the risk increases further with the age of the youngest child.

Men: The cause-specific mortality patterns show a similar general pattern to all-cause mortality, with some variations in the size of the excess mortality for fathers with older children. For all causes, we found that mortality is lowest for fathers of newborns. In the model that only includes cancer deaths or circulatory disease, variation by the age of the youngest child is lower than for all causes. For circulatory diseases, the male pattern diverges substantially from the female pattern.

In sum, the analysis of different causes of death showed that parental mortality differentials by the age of the youngest child are found for all causes of death, while the magnitudes and patterns of these differentials vary. In both sexes, mortality differentials for cancers and circulatory diseases were lower than for all causes, while for external causes of death, these differentials were much more elevated.

Discussion

In this investigation, we examined the parental mortality pattern by the age of the youngest child. Using the whole Swedish-born population aged 26–50 living in Sweden between 1991 and 2012, we applied hazard regression methods. In the theory section, we proposed two conflicting hypotheses. The first hypothesis stated that the stress of caring for a child may lead to increased mortality levels when the child is young, in particular for mothers, while having an older child may even represent a protective factor, as caring for a child becomes less demanding. No evidence to support this hypothesis was found. The analysis showed that the risk of dying among parents is lowest when their child is a newborn. Mortality increases with the age of the youngest child. This pattern supports our second hypothesis, which suggested that health selection and behavioral changes are the most potent mechanisms for explaining the observed mortality pattern. Our robustness checks confirm that the results are almost identical if we limit our study population to ages at which negative selection into earlier parenthood should play no role or only a very minor role. This strongly suggests that our results cannot be explained by differences in parental age or parental age at birth, but are largely independent of these factors, even if separating the effects mathematically is not straightforward. Our results also confirm the findings of Martikainen (1995), and additionally investigate how the effect of the youngest child's age is mitigated by other factors. This additional step, plus the focus on specific cause of death, increases our understanding of how parent mortality is affected by different aspects of parenthood.

To determine which is the most important of the two potential hypotheses, mortality was investigated for different causes of deaths. We found that parents of newborns experience a survival advantage for all causes of death, while the extent of this advantage varies. The advantage is more elevated for *external causes of death* and *suicide*, and less pronounced for mortality from *neoplasm and circulatory disease*, in particular for women. Regarding our hypothesis, the results by different causes of death suggest that both health selection and behavioral changes may explain the survival advantage among parents of younger children, although it was not possible to address health selection properly because the data did not include information on health status. This reasoning is mainly due to the assumed effects of behavioral and lifestyle factors. *External causes* and *suicide* are two causes of deaths that are almost exclusively triggered by behavioral and lifestyle factors. For both causes of death, we found a survival advantage similar to those seen for other causes of death when the child is young. This suggests that having a young child likely leads to a substantial decrease in risky behavior and risky lifestyles. The strong reduction in mortality from these causes when the child is young also largely rules out alternative explanations associated with effects of parental age at birth. These effects should lead to a mortality disadvantage for parents with older children, as these

parents were negatively selected into early parenthood. However, they cannot explain the strong mortality reductions for shorter periods of time that we have observed here.

In terms of cancer mortality, it seems implausible that behavioral changes and lifestyle factors can explain the mortality reductions in the short term, although they may be more relevant for long-term effects. Therefore, we suppose that the strong reductions in these causes for men and women of very young children can, for the most part, be attributed to selection effects. It would be reasonable to assume that parents with a long-term illness such as cancer will postpone childbearing.

Although the analysis of different causes of death provided us with a partial answer to the question of whether selection or behavioral effects shape the mortality advantage for parents of younger children, it was not possible to ascertain their relative importance for the overall mortality pattern. Thus, the missing health information at individual level remains the most important drawback of this investigation, and this should be addressed in future research.

Another drawback in the data are related to lifestyle and behavioral changes, which offers a second possible explanation for the observed effects. Again, it was not possible to address these issues directly because the Swedish registers do not include these types of predictors.

The only study we found that has also studied the effect of the age of the youngest child on parental mortality was conducted by Martikainen (1995). As his focus was not on the effects of the age of the youngest child, he did not control his models for the effects of possible confounding factors. Here, it was shown for the first time that the parental mortality pattern by the age of the youngest child persists even when a variety of parental and family characteristics are controlled for. Martikainen's (1995) study was extended here by dividing the age of the youngest child into small one- and two-year age groups. Due to the large amount of data, it was still possible to obtain statistically significant results.

Another novel aspect of this work is that the investigation was also carried out for fathers. We hypothesized that a possible effect of the age of the youngest child on parents would be less pronounced for fathers, because previous literature has suggested that giving birth to a child probably has long-lasting effects on the female body, and because men are generally less involved in parenting than women. Our results confirm that men also experience a survival advantage when their child is young. However, it is possible that the similarities between Swedish men and women are partly due to the Swedish social context. Sweden, like the other Scandinavian countries, is widely considered to be an example of a gender-egalitarian country (Gornick & Meyers, 2008; Plantenga et al., 2009). If parental leave use is considered, Sweden is even considered to be the leading country in Europe (OECD, 2016). This suggests that Swedish men are heavily involved in raising their children, and it is therefore likely that positive and negative effects associated with parenting are also experienced by Swedish men. This means that in other less gender equal contexts, the differences between men and women could be greater. Overall, this paper confirms the popular belief that the progression of parental burden and challenges increases with the age of the child: "Little children, little trouble, big children, big trouble".

Appendix

See Fig. 5.

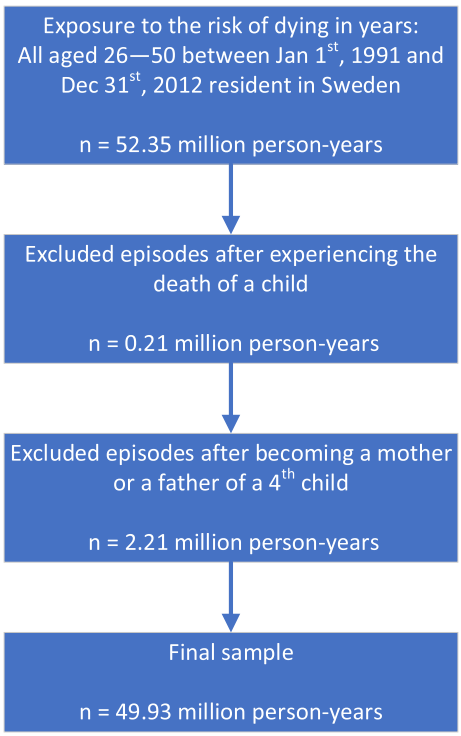

Fig. 5 Flow diagram showing the inclusion and exclusion criteria

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Author contributions
All authors read and approved the final manuscript.

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Availability of data and materials
This study is produced under the Swedish Statistics Act, where privacy concerns restrict the availability of register data for research. Aggregated data can be made available by the authors, conditional on ethical vetting. The authors access the individual-level data through Statistics Sweden's micro-online access system MONA.

Declarations

Ethics approval and consent to participate
The article is based on human demographic data, derived from administrative Swedish register data of the total population of Sweden. Research on this data falls under the act on ethical review and the personal data act and associated rules and guidelines. This research has been approved by a Swedish national ethical review board, and data have been made available by Statistics Sweden. All the information in the Statistics Sweden data source has been anonymized and contains no direct identifying information.

Competing interests
The authors declare that they have no conflict of interest.

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References

Aisenbrey, S., Evertsson, M., & Grunow, D. (2009). Is there a career penalty for mothers' time out? A Comparison of Germany, Sweden and the United States. *Social Forces, 88*(2), 573–605.
Aviv, A. (2004). Telomeres and human aging: facts and fibs, *Science of Aging Knowledge Environment: SAGE KE*, 2004 (51): pe43.
Barclay, K., Keenan, K., Grundy, E., Kolk, M., & Myrskyla, M. (2016). Reproductive history and post-reproductive mortality: A sibling comparison analysis using Swedish register data. *Social Science & Medicine, 155*, 82–92.
Barclay, K., & Kolk, M. (2019). Parity and mortality: An examination of different explanatory mechanisms using data on biological and adoptive parents. *European Journal of Population, 35*, 63–85. https://doi.org/10.1007/s10680-018-9469-1
Barclay, K., Kolk, M., Kravdal, Ø. (2023). Birth spacing and the health of mothers and fathers: an analysis of physical and mental health using individual- and sibling-fixed effects. SRRD 01/2023 Stockholm University.
Barha, C. K., Hanna, C. W., Salvante, K. G., Wilson, S. L., Robinson, W. P., Altman, R. M., & Nepomnaschy, P. A. (2016). Number of children and telomere length in women: A prospective, longitudinal evaluation. *PLoS ONE, 11*(1), e0146424.
Beeton, M. L., Yule, G. U., & Pearson, K. (1900). Data for the problem of evolution in man. V. On the correlation between duration of life and the number of offspring. *Proceedings of the Royal Society of London, 67*, 159–179.
Bellows-Riecken, K. H., & Rhodes, R. E. (2008). A birth of inactivity? A review of physical activity and parenthood. *Preventive Medicine, 46*(2), 99–110.
Berkman, L. F., Zheng, Y., Glymour, M. M., Avendano, M., Borsch-Supan, A., & Sabbath, E. L. (2015). Mothering alone: cross-national comparisons of later-life disability and health among women who were single mothers. *Journal of Epidemiology Community Health, 69*, 865–872.
Blackburn, C., Bonas, S., Spencer, N., Dolan, A., Coe, C., & Moy, R. (2005). Smoking behaviour change among fathers of new infants. *Social Science & Medicine, 61*(3), 517–526.
Brenner, H., & Mielck, A. (1993). The role of childbirth in smoking cessation. *Preventive Medicine, 22*, 225–236.
Cox, D. R. (1972). Regression models and life-tables. *Journal of the Royal Statistical Society, Series B: Statistical Methodology, 34*(2), 187–220.
Dekker, J. M., & Schouten, E. G. (1993). Number of pregnancies and risk of cardiovascular disease. *The New England Journal of Medicine, 329*(25), 1893–1894.
Doblhammer, G., & Oeppen, J. (2003). Reproduction and longevity among the British peerage: The effect of frailty and health selection. *Proceedings of the Royal Society of London, Series B: Biological Sciences, 270*, 1541–1547.
Doblhammer, G., Rogers, J. L., & Rau, R. (2000). Seasonality of birth in nineteenth and twentieth century Austria: Steps toward a unified theory of human reproductive seasonality. *Social Biology, 47*, 201–217.
Ellison, P. T. (2009). *On fertile ground: A natural history of human reproduction*. Harvard University Press.
Gagnon, A., Smith, K. R., Tremblay, M., Vézina, H., Paré, P. P., & Desjardins, B. (2009). Is there a trade-off between fertility and longevity? A comparative study of women from three large historical databases accounting for mortality selection. *American Journal of Human Biology, 21*(4), 533–540.
Golet, G. H., Schmutz, J. A., Irons, D. B., & Estes, J. A. (2004). Determinants of reproductive costs in the long-lived black-legged kittiwake: A multiyear experiment. *Ecological Monographs, 74*(2), 353–372.
Gornick, J. C., & Meyers, M. K. (2008). Creating gender egalitarian societies: An agenda for reform. *Politics & Society, 36*(3), 3.
Grundy, E., & Kravdal, Ø. (2010). Fertility history and cause-specific mortality: A register-based analysis of complete cohorts of Norwegian women and men. *Social Science & Medicine, 70*(11), 1847–1857.
Gurven, M., Stieglitz, J., Hooper, P. L., Gomes, C., & Kaplan, H. (2012). From the womb to the tomb: The role of transfers in shaping the evolved human life history. *Experimental Gerontology, 47*(10), 807–813.
Hobcraft, J., & Kiernan, K. (2001). Childhood poverty, early motherhood and adult social exclusion. *British Journal of Sociology, 52*(3), 495–517.
Högnäs, R. D., Roelfs, E., Shor, C. M., & Reece, T. (2017). J-Curve? A meta-analysis and meta-regression of parity and parental mortality. *Population Research & Policy Review, 36*(2), 273–308.
Huisman, M., Kunst, A. E., & Mackenbach, J. P. (2003). Socioeconomic inequalities in morbidity among the elderly; a European overview. *Social Science & Medicine, 57*(5), 861–873.
Hyppönen, E., Smith, G. D., Shepherd, P., & Power, C. (2005). An intergenerational and lifecourse study of health and mortality risk in parents of the 1958 birth cohort: (ii) mortality rates and study representativeness. *Public Health, 119*(7), 608–615.
Jensen, T. K., Jacobsen, R., Christensen, K., Nielsen, N. C., & Bostofte, E. (2009). Good semen quality and life expectancy: a cohort study of 43,277 men. *American Journal of Epidemiology., 170*(5), 559–565. https://doi.org/10.1093/aje/kwp168
Kalbfleisch, J. D., & Prentice, R. L. (2002). *The statistical analysis of failure time data* (2nd ed.). Wiley-Interscience.
Kane, J. B., Morgan, P., Mullan Harris, K., & Guilkey, D. (2013). The educational consequences of teen childbearing. *Demography, 50*(6), 2129–2150.
Kirkwood, T. B. L., & Rose, M. R. (1991). Evolution of senescence: Late survival sacrificed for reproduction. *Philosophical Transactions of the Royal Society of London, Series b: Biological Sciences, 332*, 15–24.
Klein, J. P., & Moeschberger, M. L. (2003). *Survival analysis: Techniques for censored and truncated data* (2nd ed.). Springer.
Kleinbaum, D. G., & Klein, M. (2005). *Survival analysis—A self learning text* (2nd ed.). Springer.
Knoester, C., & Eggebeen, D. J. (2006). The effects of the transition to parenthood and subsequent children on men's well-being and social participation. *Journal of Family Issues, 27*(11), 1532–1560.

Kramer, K. L. (2005). Children's help and the pace of reproduction: Cooperative breeding in humans. *Evolutionary Anthropology: Issues, News, and Reviews, 14*(6), 224–237.
Kramer, K. L., & Ellison, P. T. (2010). Pooled energy budgets: Resituating human energy-allocation trade-offs. *Evolutionary Anthropology: Issues, News, and Reviews, 19*(4), 136–147.
Lane, T. (2004). In Bangladesh, a woman's risk of death is elevated for two to three years after the birth of each child. *International Family Planning Perspectives, 30*(1), 45.
Lester, N. P., Shuter, B. J., & Abrams, P. A. (2004). Interpreting the von Bertalanffy model of somatic growth in fishes: The cost of reproduction. *Proceedings of the Royal Society of London, Series b: Biological Sciences, 271*(1548), 1625–1631.
Martikainen, P. (1995). Women's employment, marriage, motherhood and mortality: A test of the multiple role and role accumulation hypotheses. *Social Science & Medicine, 40*(2), 199–212.
Mayer, A., & Modig, K. (2021). The role of having children for the incidence of and survival after hip fracture—A nationwide cohort study. *Bone, 145*, 115873. https://doi.org/10.1016/j.bone.2021.115873
McArdle, P. F., Pollin, T. I., O'Connell, J. R., Sorkin, J. D., Agarwala, R., Schäffer, A. A., Streeten, E. A., King, T. M., Shuldiner, A. R., & Mitchell, B. D. (2006). Does having children extend life span? A genealogical study of parity and longevity in the Amish. *Journal of Gerontology Series a: Biological Sciences and Medical Sciences, 61A*(2), 190–195.
Meehan, C. L., Quinlan, R., & Malcom, C. D. (2013). Cooperative breeding and maternal energy expenditure among Aka foragers. *American Journal of Human Biology, 25*(1), 42–57.
Menken, J., Duffy, L., & Kuhn, R. (2003). Childbearing and women's survival: New evidence from rural Bangladesh. *Population and Development Review, 29*(3), 405–426.
Mirowsky, J. (2005). Age at First Birth, Health, and Mortality. *Journal of Health and Social Behavior, 46*(1), 32–50.
Modig, K., Talbäck, M., Torssander, J., & Ahlbom, A. (2017). Payback time? Influence of having children on mortality in old age. *Journal of Epidemiology and Community Health, 71*(5), 424–430.
Mueller, U. (2004). Does late reproduction enhance life span? Findings from European royalty. *Population and Development Review, 30*(3), 449–466.
Obeso, J. R. (2002). The cost of reproduction in plants. *New Phytologist, 155*(3), 321–348.
OECD. (2016). *Background brief on fathers' leave and its use*. OECD Publishing.
Partridge, L., Green, A., & Fowler, K. (1987). Effects of egg-production and of exposure to males on female survival in drosophila melanogaster. *Journal of Insect Physiology, 33*(10), 745–749.
Perls, T. T., Alpert, L., & Fretts, R. C. (1997). Middle-aged mothers live longer. *Nature, 389*(6647), 133.
Plantenga, J., Remery, C., Figueiredo, H., & Smith, M. (2009). Towards a European Union gender equality index. *Journal of European Social Policy, 19*(1), 19–33.
Pradhan, E. K., West, K. P., Jr., Katz, J., Christian, P., Khatry, S. K., LeClerq, S. C., Dali, S. M., & Shrestha, S. R. (2002). Risk of death following pregnancy in rural Nepal. *Bulletin of the World Health Organization, 80*(11), 887–891.
Prentice, R. L., Kalbfleisch, J. D., Jr., Peterson, A. V., Flournoy, N., Farewell, V. T., & Breslow, N. E. (1978). The analysis of failure times in the presence of competing risks. *Biometrics, 34*(4), 541–554.
Ronsmans, C., Khlat, M., Kodio, B., Ba, M., De Bernis, L., & Etard, J. F. (2001). Evidence for a 'healthy pregnant woman effect' in Niakhar, Senegal? *International Journal of Epidemiology, 30*(3), 467–473.
Rose, M. R. (1997). Toward an evolutionary demography. In K. W. Wachter & C. E. Finch (Eds.), *Between Zeus and the salmon: the biodemography of longevity* (pp. 96–107). National Academy Press.
Smith, K. R., Mineau, G. P., & Bean, L. L. (2002). Fertility and post-reproductive survival. *Social Biology, 49*(3–4), 185–205.
Socialstyrelsen. (2023). The statistical database. Assessed on March 3, 2023. https://www.socialstyrelsen.se/en/statistics-and-data/statistics/statistical-databases/
Weitoft, G. R., Burström, B., & Rosén, M. (2004). Premature mortality among lone fathers and childless men. *Social Science & Medicine, 59*(7), 1449–1459.
Weng, H. H., Bastian, L. A., Taylor, D. H., Jr., Moser, B. K., & Ostbye, T. (2004). Number of children associated with obesity in middle-aged women and men: results from the health and retirement study. *Journal of Women's Health, 13*(1), 85–91.
World Health Organization. (1992). *Pregnancy, Childbirth and the Puerperium* (pp. 721–764). World Health Organization.
Zeng, Y., Ni, Z., Liu, S. J., Gu, X., Huang, Q., Liu, J. A., & Wang, Q. (2016). Parity and all-cause mortality in women and men: A dose-response meta-analysis of cohort studies. *Scientific Report, 6*, 19351.

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