

Stockholm Research Reports in Demography | no 2020:19



How does the age of the child affect parental survival?

Sven Drefahl, Eleonora Mussino

ISSN 2002-617X | Department of Sociology



Stockholm
University

Demography Unit

How does the age of the child affect parental survival?

Sven Drefahl

Stockholm University Demography Unit

Eleonora Mussino

Stockholm University Demography Unit

Abstract

Previous research investigated several different aspects of the relationship between having a child and parental mortality. An aspect of research that was neglected until now is the age of the child. If children have an effect on parental mortality it is likely to change as they grow up. We extend previous research by applying a longitudinal approach using Swedish Register data and hazard regression models for men and women separately. Adjusting for age and a variety of control variables we found that risk of dying increases by age of the youngest child for both sexes. 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*

Stockholm Research Reports in Demography 2020:19

ISSN 2002-617X

© Sven Drefahl and Eleonora Mussino



This work is licensed under a [Creative Commons Attribution 4.0 International License](https://creativecommons.org/licenses/by/4.0/).

Introduction

It was always true in human history that giving birth and raising children posed a risk for the health of the mother. It was only in the course of the last century that these medical risks have been greatly reduced. Today, children may pose a risk in other aspects, for example for the labour market career and through the double-burden of managing both employment and parenthood. Having the responsibility for raising a child also comes with substantial changes for the lifestyle of the parents. Parents change 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 whether having a child of different ages affects parental mortality in different ways, which causes of death are affected most, and what factors may be responsible for this.

Background

Previous research 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 the advancements in the medical system, it 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 in the postpartum period that lasts from about 12 weeks in Nepal (Pradhan et al. 2002) to up to three years in Bangladesh (Menken et al. 2003; Lane 2004). 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 in the postpartum period for up to one year (World Health Organization 1992).

A second aspect that was investigated by earlier studies involves post-reproductive aging from an evolutionary perspective. These theories propose a trade-off between childbearing history and survival (Kirkwood and Rose 1991; Ellison 2009). A prominent theory that attempts to identify these patterns in humans is the cost-of-reproduction hypothesis. It 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. Partridge et al. 1987; Golet et al. 2004; Lester et al. 2004). 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 of 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. The cost-of-reproduction may also be economic and indirectly affect the health of the parents. This is particularly true for mothers, who are the most penalized by their new status in the labour market (Aisenbrey et al. 2009). Despite these indications, the question of whether reproduction negatively affects human longevity remains unresolved (Doblhammer and 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 and Kravdal 2010). Consistently with this mechanism, a study of cause-specific mortality found lower risk of breast, uterine and ovarian cancer among high parity women (Barclay et al. 2016).

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; Ronsmans et al. 2001; Hyppönen et al. 2005). 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 related to the results found by Perls et al. (1997) and McArdle et al. (2006).

Doblhammer (2000), Smith et al. (2002), 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).

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 are not well understood, but it is assumed that direct social support from children in case 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 for increasing longevity is particularly strong in old ages (Modig et al. 2017). Having a large number of children is not just associated with greater access to social support; a large family size 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 that 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, is mainly focused on mothers. Investigations of men are few and limited, such as the study by Dekker and Schouten (1993), who examined the effect of lifetime reproduction of men on mortality from coronary heart disease, rather than all-cause mortality. Although their results were not statistically significant, the authors found higher mortality among men with four or more children. This effect was somewhat lower than that seen for women (Dekker and Schouten 1993). A study by Hyppönen et al. (2005) also indicates that the effects among men might be different from the ones 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 socio-economic 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 particular from injury, addiction, and ischaemic heart disease (Weitof et al. 2004). Previous research also investigated behavioural changes among men after the birth of child. Blackburn et al. (2005) studied the smoking behaviour 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 and Mielck, 1993). Blackburn et al. (2005) found that the vast majority of men changed their smoking behaviour in one or several aspects. It is assumed that economic and social pressure to quit smoking are the most important reasons behind these behavioural 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 encourages also a better integration into the community and this has a particular protective effect for men (Knoester and Eggebeen 2006). However, parents have less time for exercise (Bellows-Riecken and Rhodes 2008) and obesity seems associated to the number of children for both middle-aged women and men (Weng et al. 2004).

Until now, scientific interest was not 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 the adolescence of their children, were neglected. But this relationship in particular might have gained importance with the ongoing modernization of societies, the changes in the role of women, 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 became increasingly involved in parenting. These considerations about the combining of marital, parental, and work roles are incorporated in several sometimes conflicting hypotheses. The multiple-role hypothesis concludes that the triple burden of being a mother, wife, and 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). In contrast, the role accumulation hypothesis suggests that the benefits of having several roles will outweigh harmful effects, mainly due to a higher income and additional social relationships outside of the home and marriage (Martikainen 1995).

A further aspect of research that was 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 changes 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 mother's mortality 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 1981–85. 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 who are 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 mother's mortality is changing 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.

Research questions and hypotheses

We indicated in the previous section that there has been virtually no study that has focused 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 a 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. Following this reasoning, having an older child could even be a protective factor and thus increase parental survival as compared to those with very young children.

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 life style. Parents may feel obliged to reduce or give up some hazardous health behaviours, such as smoking, or they feel less inclined or having less time for higher-risk leisure activities. However, both of the aspects might be more significant for parents with younger children and less for those with older children and lifestyle changes might be more pronounced in women than in men. 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 select subgroup that is healthier on average than couples who were 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 of these forms of selection would lead to reduced mortality for parents of young children, but its effect may vanish when the child gets older and lead to a decrease of the mortality advantage with age. Following the cost-of-reproduction hypothesis, which stipulates possible long-term detrimental effects of pregnancy and parenthood, this reduction could potentially accelerate with age.

It is likely that the impact of children on parental mortality differs 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 the society and family policies aim to involve both sexes equally in parenting. Nevertheless, we assume that the change in mortality by the age of child is less pronounced in fathers, but it is likely that the gender differences are smaller than they would probably be in other countries.

We also plan to explore whether the effect of the age of the youngest child varies according to different causes of death. Survival models for deaths from neoplasm, diseases of the circulatory system, external causes, and suicide were conducted. Regarding our two conflicting hypotheses, it is assumed that, if behavioural changes are important, a decreased risk of dying among parents with young children would be observed for external causes of death and suicides. If stress is an important factor, circulatory diseases may show an elevated risk for parents of young children.

Method and data

Data

The data that is analysed here includes 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. Parity was limited from zero to three children because of computational limitations. 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 to 50 years old on 1 January 1991 *and* having three or fewer children, of whom the youngest child is less than 21 years of age
- reaching age 26 between 2 January 1991 and 31 December 2012 *and* having three or fewer children;

To exit the study, the following seven ways are possible: by

- dying between 2 January 1991 and 31 December 2012;
- reaching age 50 between 2 January 1991 and 31 December 2012;
- becoming the parent of a fourth child between 2 January 1991 and 31 December 2012;
- experiencing the death of a child between 2 January 1991 and 31 December 2012;
- celebrating the 21th birthday of the youngest child between 2 January 1991 and 31 December 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.

Table 1. Distribution of time at risk for men and women

Covariate	Women			Men		
	Person years in Thousands	%	Deaths	Person years in Thousands	%	Deaths
Age of the youngest child						
0	1,337.9	5.54	183	1,461.4	5.66	448
1	1,370.7	5.67	247	1,456.9	5.64	629
2 to under 4	3,176.6	13.14	929	3,197.1	12.38	1,955
5 to under 9	4,093.3	16.94	2,047	3,871.1	14.99	3,611
10 to under 17	5,238.4	21.68	4,663	4,384.2	16.98	6,387
18 to under 21	2,672.5	11.06	4,357	1,488.2	5.76	4,137
Childless	6,277.0	25.97	6,400	9,958.9	38.57	16,762
Calendar period						
1991 – 1994	4,645.6	19.22	4,475	4,927.2	19.08	8,148
1995 – 1999	5,631.6	23.30	4,658	5,991.5	23.21	8,407
2000 – 2004	5,473.9	22.65	4,065	5,855.0	22.68	7,264
2005 – 2009	5,303.7	21.95	3,605	5,697.6	22.07	6,474
2010 – 2011	3,111.6	12.88	2,023	3,346.4	12.96	3,636
Highest achieved education (ISCED)						
1 & 2	2,772.1	11.47	4,623	4,254.5	16.48	10,571
3	7,833.5	32.41	7,059	9,004.0	34.88	13,185
4	4,274.1	17.69	2,253	4,470.4	17.32	4,149
5	4,096.6	16.95	2,232	3,795.5	14.70	2,853
6 & 7	5,133.8	21.24	2,287	4,193.7	16.24	2,529
Unknown / Missing	56.3	0.23	372	99.6	0.39	642
Income quintile						
1	5,820	24.08	4,132	4,351	16.86	7,417
2	5,538	22.92	4,775	4,302	16.66	7,131
3	4,700	19.45	4,173	4,706	18.23	6,635
4	4,330	17.92	3,239	5,459	21.14	6,205
5	3,747	15.51	2,507	6,960	26.96	6,539
Unknown/Missing	30	0.13	0	36	0.14	2
Parity						
0	6,277	25.97	6,400	9,959	38.57	16,762
1	4,298	17.78	3,555	4,492	17.40	5,917
2	9,594	39.70	6,076	8,137	31.52	7,833
3	3,998	16.54	2,795	3,230	12.51	3,417
Marital status						
Never married	11,199	46.34	7,575	9,947	38.53	8,042
Married	10,716	44.34	8,062	14,137	54.76	21,127
Dissolved union	2,251	9.31	3,189	1,732	6.71	4,760
Total of each variable ^a	24,116	100.00	18,826	25,817	100.00	33,929

^a Total may not always sum up to these values due to rounding

Source: Own calculations based on Swedish register data

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, thus we 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. 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.

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 of individuals over their life course. The general proportional hazards regression model is expressed by;

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

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

We use the Cox proportional Hazard model proposed by Cox (1972), which is a semi-parametric transition rate model. 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 the 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 choose 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 18–21 would exist in the data, at older ages, no or extremely few parents with children 0–1 would exist in the data. In respect to the hazard regression, this choice should facilitate that the proportional-hazards assumption is 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. Model M1 and M2 are the main models; Models M3–M5 provide robustness checks. In the second part of our analysis we will estimate models for four different causes of deaths groups separately (Kleinbaum and Klein, 2005). If one specific cause of death is the event of interest, all other deaths 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 and Moeschberger, 2003). Estimating all causes of deaths simultaneously is therefore statistically equivalent to the estimation for each cause of death separately (Prentice, 1978; Kalbfleisch and Prentice, 2002).

Results

The results for mortality from all causes by the age of the youngest child for the whole observation period from 1 January 1991 until 31 December 2012 are shown in Figure 1 (women) and Figure 2 (men). Both figures consist of five separate curves showing the relative risk of dying by age of the youngest child. The reference category comprises all index persons who have a newborn child. The relative mortality risk for persons who do not have any children are given by the dashed lines. In Model 1 and 3 the mortality risk for childless persons is compared to individuals who have newborn children. When parity is included in Model 2 and 5, 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 those two models, the point estimates for the childless dummy is presented together with the results of the additional covariates in Table 2. Model 4 shows the relative mortality risks by the age of the youngest child only for parents (without including childless) and Model 5 includes only

individuals aged 34–36. The latter model is the most important robustness check. It only includes men and women of a narrow age range at which children of all ages are common or at least are possible to observe. 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.

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 the survival chances of a subject, 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 we study. 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 more than 5 times higher mortality risk. Similarly, mothers of the same age whose youngest child is 5–10 years old experience a two times higher mortality risk; those who are mothers of a 18–21 year old child show a 3.5 times elevated risk. In fathers we find a comparable pattern; 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 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 based on the literature, which means that mortality decreases considerably with rising income. For women, the decrease is less pronounced than for men. Similarly, the chance of survival is shown to increase the higher the educational degree. 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 or have one child. Results are consistent also for women (Table 2).

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 4), strengthen the impression that the mortality is much lower when the kid 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.

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

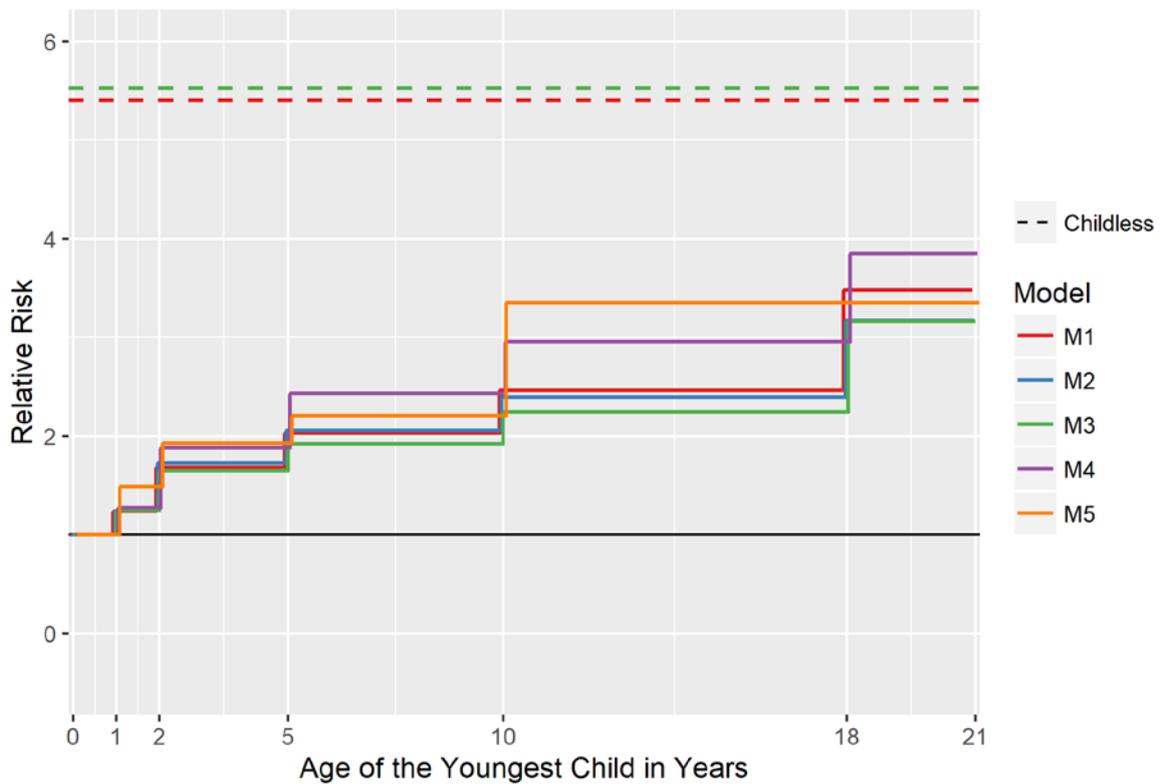


Figure 2. Model 1–5 (men): relative risk of dying by the age of the youngest child

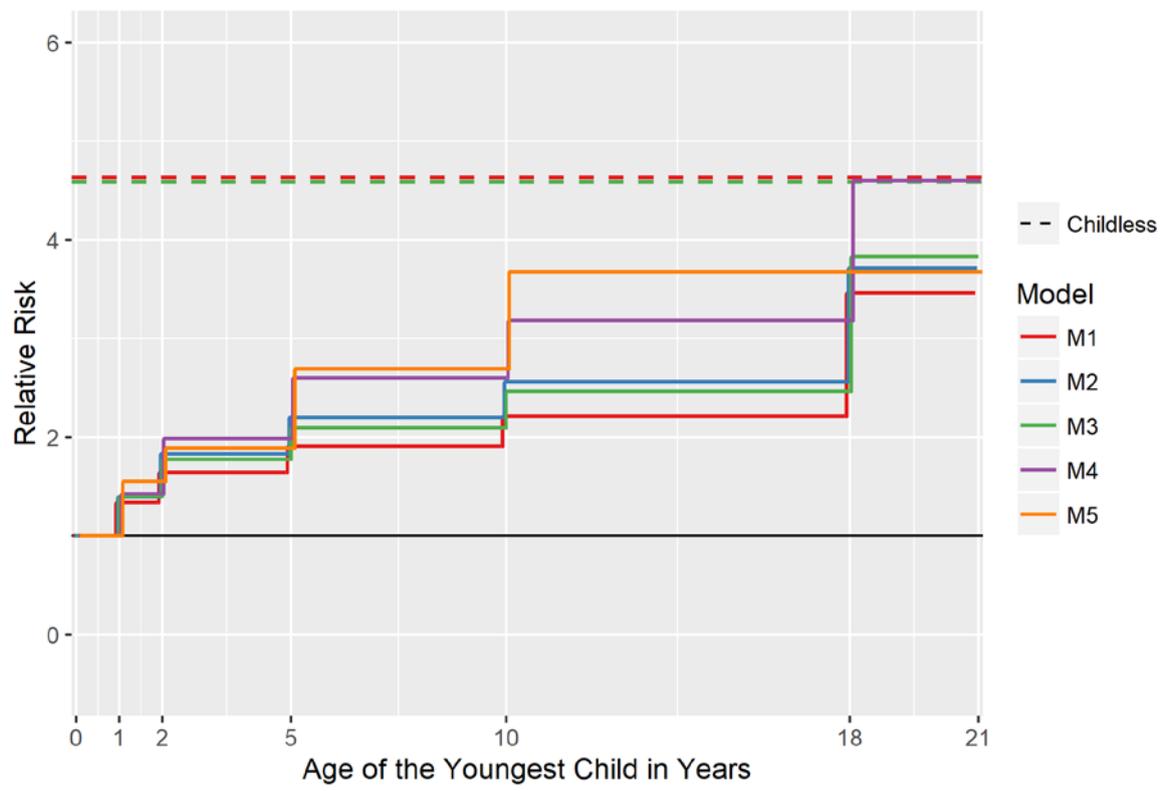


Table 2. Model 2–5: effect of the remaining covariates on the hazard of mortality

	M2	M3	M4	M5
Highest achieved education (ISCED)				
1 & 2	1	1	1	1
3	0.663***	0.658***	0.723***	0.542***
4	0.514***	0.512***	0.582***	0.376***
5	0.462***	0.459***	0.554***	0.339***
6 & 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	1.56e-21	1.64e-21	9.70e-21	
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***			9.151***
1	1.580***		1.582***	1.770***
2	1		1	1
3	0.976		0.992	0.879
Observations	22968029	22968029	18082596	2849706
Failures	18826	18826	12426	918
Person days	8826777892	8826777892	6534117682	733021569

Source: Own calculations based on Swedish register data

Table 3. Model 2–5(men): effect of the remaining covariates on the hazard of mortality

	M2	M3	M4	M5
Highest achieved education (ISCED)				
1 & 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 & 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***			6.239***
1	1.483***		1.446***	1.648***
2	1		1	1
3	0.992		1.022	1.039
Observations	23747765	23747765	16667989	3064132
Failures	33929	33929	17167	1878
Person days	9429957202	9429957202	5792461634	793061669

Source: Own calculations based on Swedish register data

Summary

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 and increases slowly with the age of the child for both sexes. Introducing covariates did not substantively alter this pattern.

Mortality by Causes of Death

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

Women: the risk of dying from cancer in women is exceptionally low among mothers of newborns. For mothers of the same age, who have a child that is 1 to under 2 years old, mortality is elevated by 80%, for those who have children 2 to under 5 years of age 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 less pronounced for mothers of children older than 2 years of age.

For circulatory diseases we also observe a distinct pattern. While for all other causes of death, mortality was found to be lowest among parents of newborns, the risk of dying from circulatory diseases decreases in the first years of motherhood. Compared to the reference group comprised of mothers of newborn, the lowest mortality rates were found for mothers of a 2 to under 10 year-old children. 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 10. The observed suicide risk are very low for mothers of young children as well, particularly when they are below 5 years. Having children is less protective for suicide for mothers of older children and increase further the older the youngest child.

Men: the cause-specific mortality patterns show a similar general pattern as 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 just 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.

Summary

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 circular diseases were lower than for all causes, while for external causes of death, these differentials were much more elevated.

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

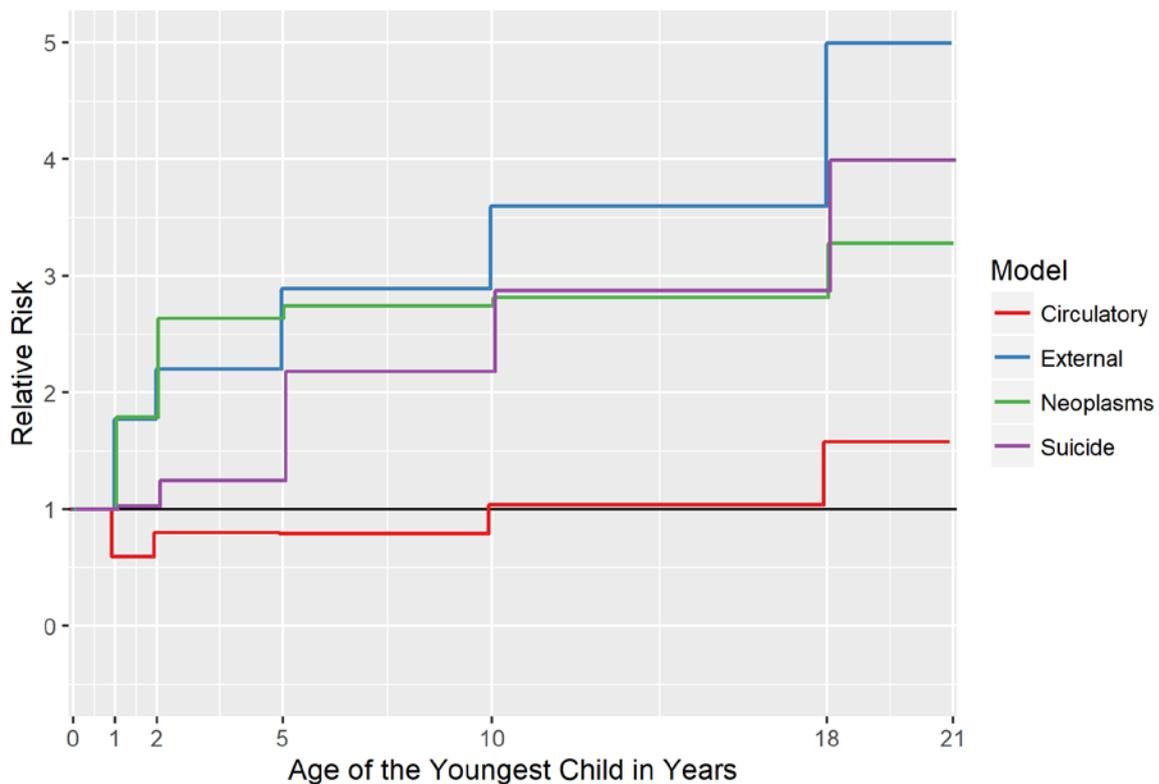
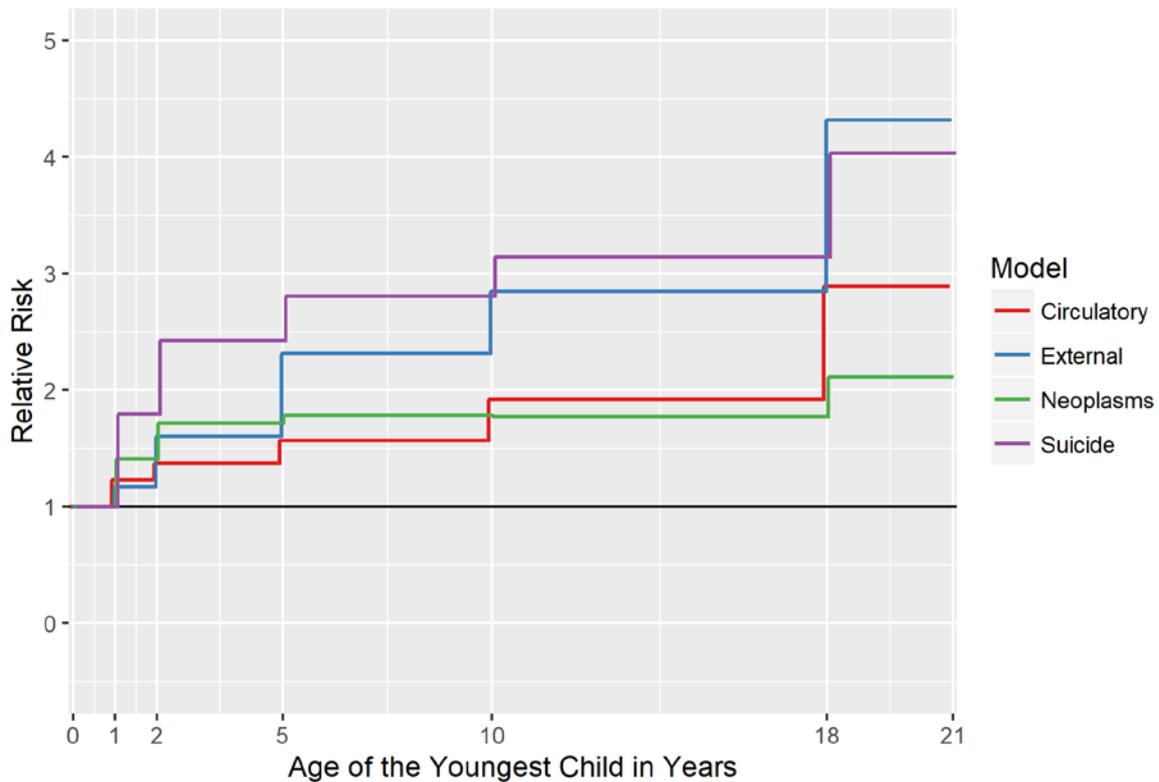


Figure 4. Model 6a–6d (men): relative risk of dying by the age of the youngest child for selected causes of death



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 caring for a child may lead to increased mortality levels when a child is small, in particular for the 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. With age of the youngest child, mortality increases. This pattern supports our second hypothesis, which suggested that health selection and behavioural changes are the most potent mechanisms for explaining the observed mortality pattern. Our results also confirm the findings in Martikainen (1995) and additionally investigate how the effect of the youngest children’s age is mitigated by other factors. This additional step forward, plus the focus on specific cause of death, increases our understanding on how parent mortality is affected by different aspects of parenthood.

To determine the most important of the two potential mechanisms, 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 behavioural changes may explain the survival advantage among parents of younger children, although it was not possible to address health selection properly here because the data did not include information on health status. This reasoning is mainly due to the assumed effects of behavioural and life style factors. External causes and suicide are two causes of deaths that are almost exclusively triggered by behavioural and lifestyle factors. For both causes of death, we found a survival advantage similar to those seen for the other causes of deaths when the child is small. This suggests that having a small child probably leads to a substantial decrease in risky behaviour and risky lifestyles. However, it seems implausible that behavioural changes affect cancer mortality in the short term, although they may be more relevant for long-term effects. Therefore, it is supposed that the strong reductions for these causes for men and women of very young children can be attributed for the most part to selection effects. It would be reasonable to assume that women with a long-term illness such as cancer 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 here to ascertain directly their relative importance on the overall mortality pattern. Thus, the missing health information on the individual level remains the most important drawback of this investigation, and it should be addressed in future research.

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

The only study that we found that has also studied the effect of the age of the youngest child on parental mortality was by Martikainen (1995). As his focus was not on the effects of the age of the youngest child, they did not control their 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. The study by Martikainen (1995) was also 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 novelty aspect of this work is that the investigation was also done 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 and 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 therefore it is likely that positive and negative effects associated with parenting are also experienced by Swedish men.

Acknowledgements

This research was supported by the Swedish Initiative for Research on Microdata in the Social and Medical Sciences (SIMSAM), grant 340-2013-5164, as well as VR, grant 2018-01825. We are grateful for the useful comments on an earlier version of the manuscript by Hainer Maier, James W. Vaupel, and Gunnar Andersson, as well as participants at the European Population Conference 2018.

References

- Aisenbrey, Silke, Marie Evertsson, and Daniela Grunow. 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, Abraham. 2004. Telomeres and human aging: facts and fibs, *Science of aging knowledge environment: SAGE KE*, 2004(51): pe43.
- Barclay, Kieron, Katherine Keenan, Emily Grundy, Martin Kolk, and Mikko Myrskylä. 2016. Reproductive history and post-reproductive mortality: A sibling comparison analysis using Swedish register data. *Social Science & Medicine*, 155: 82–92.
- Barha, Cindy K., Courtney W. Hanna, Katarina G. Salvante, Samantha L. Wilson, Wendy P. Robinson, Rachel M. Altman, and Pablo A. Nepomnaschy. 2016. Number of children and telomere length in women: a prospective, longitudinal evaluation. *PloS one*, 11(1): e0146424.
- Beeton, Michael L., George U. Yule, and Karl Pearson. 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, Kai H. and Ryan E. Rhodes. 2008. A birth of inactivity? A review of physical activity and parenthood. *Preventive medicine*, 46(2): 99–110.
- Blackburn, Clare, Sheila Bonas, Nick Spencer, Alan Dolan, Christine Coe, and Robert Moy. 2005. Smoking behaviour change among fathers of new infants. *Social Science & Medicine*, 61(3): 517–526.
- Brenner, Hermann and Andreas Mielck. 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, Jacqueline M. and Evert G. Schouten. 1993. Number of pregnancies and risk of cardiovascular disease. *The New England Journal of Medicine*, 329(25): 1893–1894.

Doblhammer, Gabriele, Rogers, Joseph Lee and Roland Rau. 2000. Seasonality of birth in nineteenth and twentieth century Austria: Steps toward a unified theory of human reproductive seasonality. *Social Biology*, 47: 201–217.

Doblhammer, Gabriele and Jim Oeppen. 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.

Ellison, Peter T. 2009. *On fertile ground: A natural history of human reproduction*. Harvard University Press.

Golet, Gregory H., Joel A. Schmutz, David B. Irons, and James A. Estes. 2004. Determinants of reproductive costs in the long-lived black-legged kittiwake: A multiyear experiment. *Ecological Monographs*, 74(2): 353–372.

Gornick, Janet C. and Marcia K. Meyers. 2008. Creating gender egalitarian societies: An agenda for reform. *Politics & Society*, 36(3): 3.

Grundy, Emily and Øystein Kravdal. 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, Michael, Jonathan Stieglitz, Paul L. Hooper, Cristina Gomes, and Hilard Kaplan. 2012. From the womb to the tomb: the role of transfers in shaping the evolved human life history. *Experimental gerontology*, 47(10): 807–813.

Hyppönen, Elina, George Davey Smith, Peter Shepherd, and Chris Power. 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.

Kalbfleisch, John D. and Ross L. Prentice. 2002. *The Statistical Analysis of Failure Time Data*. Wiley Series in Probability and Statistics. Wiley-Interscience, New York, 2 edition.

Kirkwood, T. B. L. and M. R. Rose. 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, John P. and Melvin L. Moeschberger. 2003. *Survival Analysis: Techniques for Censored and Truncated Data*. Statistics for Biology and Health. Springer, New York, 2 edition.

Kleinbaum, David G. and Mitchel Klein. 2005. *Survival Analysis - A Self Learning Text*. Statistics for Biology and Health. Springer, 2 edition.

Knoester, Chris and David J. Eggebeen. 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, Karen L. 2005. Children's help and the pace of reproduction: cooperative breeding in humans. *Evolutionary Anthropology: Issues, News, and Reviews: Issues, News, and Reviews*, 14(6): 224–237.

Kramer, Karen L. and Peter T. Ellison. 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., B. J. Shuter, and P. A. Abrams. 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, Pekka. 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.

McArdle, Patrick F., Toni I. Pollin, Jeffrey R. O'Connell, John D. Sorkin, Richa Agarwala, Alejandro A. Schäffer, Elizabeth A. Streeten, Terri M. King, Alan R. Shuldiner, and Braxton D. Mitchell. 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, Courtney L. Robert Quinlan, and Courtney D. Malcom. 2013. Cooperative breeding and maternal energy expenditure among aka foragers. *American Journal of Human Biology*, 25(1): 42–57.

Menken, Jane, Linda Duffy, and Randall Kuhn. 2003. Childbearing and women's survival: New evidence from rural Bangladesh. *Population and Development Review*, 29(3): 405–426.

Modig, K., M. Talbäck, J. Torssander, and A. Ahlbom. 2017. Payback time? Influence of having children on mortality in old age. *J Epidemiol Community Health*, 71(5): 424–430.

Mueller, Ulrich. 2004. Does late reproduction enhance life span? Findings from European royalty. *Population and Development Review*, 30(3): 449–466.

Obeso, José Ramón. 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, Linda, Alison Green, and Kevin Fowler. 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, Thomas T., Laura Alpert, and Ruth C. Fretts. 1997. Middle-aged mothers live longer. *Nature*, 389(6647): 133.

Plantenga, Janneke, Chantal Remery, Hugo Figueiredo, and Mark Smith. 2009. Towards a European union gender equality index. *Journal of European Social Policy*, 19(1): 19–33.

Pradhan, Elizabeth Kimbrough, Keith P. West Jr, Joanne Katz, Parul Christian, Subarna K. Khatri, Steven C. LeClerq, Sanu Maiya Dali, and Sharada Ram Shrestha. 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., Peterson, Jr., A. V., Flournoy, N., Farewell, V. T. and N. E. Breslow 1978. The analysis of failure times in the presence of competing risks. *Biometrics*, 34(4): 541–554.

Ronsmans, C., M. Khlat, B. Kodio, M. Ba, L. De Bernis, and J. F. Etard. 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 and C. E. Finch (eds), *Between Zeus and the salmon: the biodemography of longevity*. Washington D.C.: National Academy Press, pp. 96–107.

Smith, Ken R., Geraldine P. Mineau, and Lee L. Bean. 2002. Fertility and post-reproductive survival. *Social Biology*, 49(3–4): 185–205.

Weitof, Gunilla Ringbäck, Bo Burström, and Måns Rosén. 2004. Premature mortality among lone fathers and childless men. *Social science & medicine*, 59(7): 1449–1459.

Weng, Haoling H., Lori A. Bastian, Donald H. Taylor Jr, Barry K. Moser, and Truls Ostbye. 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*. Geneva: World Health Organization, pp. 721–764.

Stockholm Research Reports in Demography

Stockholm University, 106 91
Stockholm, Sweden www.su.se |
info@su.se | ISSN 2002-617X



Stockholm
University

Demography Unit