Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

HHS Public Access

Author manuscript

Soc Sci Med. Author manuscript; available in PMC 2015 October 01.

Published in final edited form as:

Soc Sci Med. 2014 October ; 119: 215–223. doi:10.1016/j.socscimed.2014.06.008.

The association between advanced maternal and paternal ages and increased adult mortality is explained by early parental loss

Mikko Myrskylä[a], **Irma T. Elo**[b], **Iliana Kohler**[c], and **Pekka Martikainen**[d]

Mikko Myrskylä: myrskyla@demogr.mpg.de; Irma T. Elo: popelo@pop.upenn.edu; Iliana Kohler: iliana@pop.upenn.edu; Pekka Martikainen: pekka.martikainen@helsinki.fi

[a]Max Planck Institute for Demographic Research, Konrad-Zuse-Str. 1, 18057 Rostock, Germany, Phone: +49 (0)381 2081-118, Fax: +49 (0)381 2081-418

[b]Population Studies Center and Department of Sociology, University of Pennsylvania, 3718 Locust Walk, McNeil Building, Ste. 113 Philadelphia, PA 19104-6299., Phone +1-215-898-9162, Fax: +1-215-573-2081

[c]Population Studies Center and Population Aging Research Center (PARC), University of Pennsylvania, 3718 Locust Walk, McNeil Building, Ste. 113 Philadelphia, PA 19104-6299., Phone +1-215-898-7990, Fax: +1-215-573-2081

[d]Population Research Unit, Department of Social Research, University of Helsinki, P.O. Box 18, 00014 Finland., Tel 358-9-191 23889, Fax 358-9-191 23967

Abstract

The association between advanced maternal and paternal ages at birth and increased mortality among adult offspring is often attributed to parental reproductive ageing, e.g., declining oocyte or sperm quality. Less attention has been paid to alternative mechanisms, including parental socio-demographic characteristics or the timing of parental death. Moreover, it is not known if the parental age-adult mortality association is mediated by socioeconomic attainment of the children, or if it varies over the lifecourse of the adult children. We used register-based data drawn from the Finnish 1950 census (sample size 89,737; mortality follow-up 1971–2008) and discrete-time survival regression with logit link to analyze these alternative mechanisms in the parental age-offspring mortality association when the children were aged 35–49 and 50–72. Consistent with prior literature, we found that adult children of older parents had increased mortality relative to adults whose parents were aged 25–29 at the time of birth. For example, maternal and paternal ages 40–49 were associated with mortality odds ratios (ORs)of 1.31 ($p<.001$) and 1.22 ($p<.01$), respectively, for offspring mortality at ages 35–49. At ages 50–72 advanced parental age also predicted higher mortality, though not as strongly. Adjustment for parental socio-demographic characteristics (education, occupation, family size, household crowding, language) weakened the associations only slightly. Adjustment for parental survival, measured by whether the parents were

© 2014 Elsevier Ltd. All rights reserved.

Correspondence to: Mikko Myrskylä, myrskyla@demogr.mpg.de.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

alive when the child reached age 35, reduced the advanced parental age coefficients substantially and to statistically insignificant levels. These results indicate that the mechanism behind the advanced parental age-adult offspring mortality association is mainly social, reflecting early parental loss and parental characteristics, rather than physiological mechanisms reflecting reproductive ageing.

Keywords

Maternal age; paternal age; adult mortality; parental death; reproductive ageing; Finland

INTRODUCTION

Advanced maternal and paternal ages at birth are associated with a range of negative offspring health outcomes. For example, the risk of developing Down syndrome, childhood cancer, and autism has been found to increase with maternal and/or paternal age (Durkin et al. 2008; Yip, Pawitan and Czene 2006), and a recent review stated that "[p]arental age has been shown to be a major factor, if not the most important factor, in producing variability in offspring" (Liu, Zhi and Li 2011). Less is known about the relationship between parental age and the outcomes of *adult* offspring. However, the existing literature suggests that being born to an older mother or father has severe long-term health consequences, including an increased risk of developing cancer (Yip et al. 2006), Alzheimer's disease (Rocca et al. 1991), and diabetes (Gale 2010).

This study focuses on the effects of parental age on the mortality of adult children, as prior research has shown a strong association between advanced maternal and paternal ages and adult offspring mortality. Gavrilov and Gavrilova (2012) compared 198 U.S.-born centenarians to their siblings, and found that, relative to being born to a mother aged above 25, being born to a mother younger than age 25 doubled the odds of living to 100. Kemkes-Grottenthaler (2004) analyzed 17th–19th century German data and found that, compared to being born to a mother aged 20–29, being born to a mother aged 40–49 was associated with a 8.9 years shorter lifespan for daughters and 5.2 years shorter lifespan for sons. An analysis of a 1966 birth cohort in Finland found that 14% of deaths up to age 39 were attributable to advanced paternal age (Miller et al. 2010).

These associations between parental ages and offspring mortality and the continued postponement of fertility to older ages have prompted some scholars to question how old is too old to have children, and whether fertility at older ages should be discouraged (Bray, Gunnell and Davey Smith 2006; Heffner 2004). It has been suggested that women and men should be better informed about the risks associated with bearing children at older ages (Benzies 2008). Before policy recommendations are made, however, more research is needed that examines the robustness of and the mechanism behind the advanced parental age-offspring health association.

First, much of the evidence linking advanced parental age to increased adult mortality comes from small samples and historical populations that are not representative of national populations (Gavrilov and Gavrilova 2012; Kemkes-Grottenthaler 2004; Smith et al. 2009).

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Two recent studies using representative U.S. (Myrskylä and Fenelon 2012) and Canadian (Hubbard, Andrew and Rockwood 2009) survey data found little evidence that advanced maternal age is associated with the health or mortality of adult children, and a study of 320 French centenarians found no association between parental age and offspring longevity (Robine et al. 2003). These studies were, however, limited, as they analyzed maternal age only (Myrskylä and Fenelon 2012), used small samples (Robine et al. 2003), or focused only on the oldest adults (ages 100+ in Robine et al. 2003; ages 65+ in Hubbard et al. 2009).

Second, much of the discussion on the advanced parental age-offspring health association stresses physiobiological interpretations that focus on parental reproductive ageing (Armstrong 2001; Durkin et al. 2008; Gale 2010; Kemkes-Grottenthaler 2004; Kong et al. 2012; Schmid et al. 2007). On the maternal side, explanatory models are based on declining fecundity and the increased probability of obstetric and perinatal complications (Heffner 2004; Tarín, Brines and Cano 1998). The decline in fecundity starts in the late twenties and early thirties (American Society for Reproductive Medicine 2003), and is related to the accumulation of DNA damage in germ cells (Kaytor et al. 1997) and decreasing oocyte quality (Armstrong 2001). These processes have been central to the discussion of the links between advanced maternal age and offspring health. On the paternal side, reproductive ageing refers to a decrease in the quality and quantity of the male sperm. Fathers are responsible for most of the new mutations in the human gene pool, and both DNA damage and the number of mutations increase with age in male sperm (Schmid et al. 2007). For example, Gavrilov and Gavrilova (2000) speculate that their finding that daughters, but not sons, born to older fathers have a decreased lifespan could be attributed to the fact that only daughters inherit the paternal X chromosome, which, among older fathers, may carry mutations that decrease longevity.

We propose that in addition to reproductive ageing, selection by parental socioeconomic status (SES), differences in the age at which the child loses the parent(s), and the child's own socioeconomic attainment may help to explain the advanced parental age-offspring mortality association. These explanations are occasionally touched upon in the existing literature, but their contributions to the parental age-offspring mortality association have rarely been the subject of direct analysis. These alternative mechanisms may, however, be integral to the parental age–offspring mortality association.

First, parental SES is associated with adult health (Hayward and Gorman 2004). If late parental ages correlate with low childhood SES, social selection may explain some of the relationship between advanced parental age and decreased offspring health. Today, older parents have more resources than younger parents (Bray et al. 2006). There is, however, no evidence that advanced parental ages were positively associated with SES in the 17th to early 20th centuries, the periods studied in most previous analyses. Thus, ignoring parental SES may bias the results.

Second, the age at which a child loses the parent(s) to death is systematically related to parental age: ceteris paribus, a child born to a 40-year-old mother can expect to lose the parent at an age that is 20 years younger than that of a child born to a 20-year-old mother. Parental loss at a young age influences a range of later-life outcomes, from education and

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

own socioeconomic status to health and longevity (Case, Paxson and Ableidinger 2004), with the likely mechanism being the truncated intergenerational transfer of social and economic resources. The age at which a child loses a parent may also be a proxy for shared family longevity, or frailty.

Third, the parental age-offspring mortality association may be indirect, being mediated by own socioeconomic attainment. Advanced parental ages are linked to decreased cognitive ability (Malaspina et al. 2005; Saha et al. 2009). This may predict decreased adult socioeconomic attainment, which would have negative health consequences (Cutler, Deaton and Lleras-Muney 2006).

We use a large Finnish sample that is representative of the national population to analyze how maternal and paternal ages are associated with offspring mortality at young adult ages (35–49 years) and older adult ages (50–72 years). Our contribution is threefold. First, we use a large, nationally representative sample to study the association between both maternal and paternal age and adult offspring mortality. Second, we analyze the contribution of alternative social mechanisms associated with parental socio-demographic characteristics: the age at which the child loses the parent(s), and the child's own socioeconomic attainment in the parental age-offspring mortality association. Third, we stratify the analysis by the age of the offspring, which helps to further shed light on the mechanisms involved. At ages 35–49, external causes and alcohol-related causes are comparatively important, whereas in the age group 50–72, cardiovascular diseases and cancers play a bigger role. We analyze maternal and paternal ages separately and simultaneously because they are highly correlated (Ní Bhrolcháin 2001) and may confound each other.

We hypothesize that parental survival is potentially important variable linking parental age to offspring mortality and may capture differences in intergenerational transfers or familial health. We are not able to measure these mechanisms directly because of a lack of data, but we can test whether parental survival helps to explain the parental age-adult offspring mortality association. If this is found to be the case, then the association may be less related to parental reproductive ageing, and more related to factors that operate after birth. Moreover, we study the importance of the intergenerational transfers mechanism by analyzing whether the parental survival coefficients are sensitive to controls for the child's attained socioeconomic status.

DATA AND METHODS

Sample description

Our analyses are based on a 10% sample of households drawn from the 1950 Finnish Census of Population (Statistics Finland 1997). All of the individuals in this original sample have been linked by Statistics Finland to census and death records beginning in 1970 using unique person identifiers. Individuals who died or moved out of the country (mainly to Sweden) between 1950 and 1970 cannot be linked to the 1970 census and are not included in the analysis. After 1970, about 99% of the records are linked to both census and death records. The prospective mortality follow-up begins at the end of 1970 and ends on December 31, 2007.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

The data for 1950 contain family identifiers which allow us to reconstruct families. We infer who are the children and who are the parents within a family by using information on the within-family age structure and the relationship of family members to the household head. We include in our analysis individuals aged 0–14 at the time of the 1950 census (sample size N=116,622). We exclude children who were known to have died before age 35 (N=1,001), and those who were not present in the 1970 or subsequent censuses (N=15,065, mostly people who had out-migrated). We exclude cases for which the maternal age was below 14 or above 50, or the paternal age was below 14 or above 65 (N=364); those living in single-parent families, because the age of the other parent is unknown (N=7,729); and those with missing information on independent variables (N=2,726). Our final sample consists of 89,737 persons aged 0–14 in 1950.

Variables

The dependent variables are time to death observed at ages 35–49 (middle-aged adults) and 50–72 (older adults). Mortality is followed up to to 31 December 2007, at which point the observations are censored. The key independent variables are maternal and paternal ages, which are defined as the ages of the mother and father (in years) at the time of the child's birth. These are calculated from the child's date of birth and the parents' dates of birth. The categories for maternal age are 14–19, 20–24, 25–29 (reference group), 30–34, 35–39, and 40–49. We use the same categories for paternal age, but with an extra 50–65 age group.

The other independent variables are parental socio-demographic characteristics, parental survival, and the child's socio-demographic characteristics. From the 1950 census, we obtain the parents' dates of birth and gender, as well as a rich set of socioeconomic characteristics, including social class, education, household crowdedness, family size, and language spoken at home. We measure the family's social class by the father's occupation, assigning it to one of the following categories: professional/administrative occupations, agricultural and other workers, large and small farmers (threshold of 10 hectares), and others. As an additional measure of SES, we include parental education coded as the highest level of schooling of the father or the mother (less than primary, primary, beyond primary). We control for household crowdedness, measured by the number of persons per heated room in the dwelling, the language spoken at home (Finnish versus Swedish), and the number of children below age 18 in the household in 1950 (1, 2–3, and 4+).

Information on parental survival is calculated based on the 1970 and subsequent censuses and death records. Specifically, we use an indicator of whether the parent was alive at the time when the child was age 35, the age at which our mortality follow-up begins. This indicator, which is constructed separately for mothers and fathers, is crude, but the data do not allow for more refined measures because information on mortality between the years 1950–1970 is not available. Nevertheless, this measure captures important variations in parental survival (see results section).

The child's birthday and gender also come from the 1950 census, whereas his/her socioeconomic and other characteristics are obtained from the 1970 census and later censuses when the child was 25–34 years of age (98% of these characteristics are measured at ages 30–34). These attributes include educational attainment (years), marital status (never

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

married, married, separated/divorced/widowed), home ownership (yes/no), and whether there were any children in the household.

Statistical models

We use discrete time survival models with one-year time intervals and logit link to estimate the associations between parental age and offspring mortality at ages 35–49 and 50–72. We made this model choice over alternatives such as Cox proportional hazards regression because the Cox model does not easily lend itself to mediation analysis, whereas the discrete time survival models with logit link does. We estimate four models for each age group separately for maternal and paternal ages, and one model that includes the ages of both parents. We use the method by Karlson, Holm and Breen (KHB method; Karlson, Holm and Breen 2012, Breen, Karlson, Holm 2013) and the Stata command *khb* (Kohler, Karlson, and Holm 2011) to estimate the amount of maternal and paternal age coefficients is mediated by own socioeconomic attainment and parental death.

Model 1 estimates the association between parental ages (separate models for maternal and paternal ages) and mortality, and adjusts only for the child's year of birth, sex, and annual period effects. We do not control for age, as everybody enters the risk group at the same age. Model 2 adds parental socioeconomic characteristics to Model 1 to investigate the confounding influence of parental SES. Model 3 adds offspring's own SES to Model 2 to examine whether the parental age-offspring mortality association is mediated by own socioeconomic attainment. Model 4 adds parental survival to Model 3. Model 5 adds controls for the other parent's age at birth to Model 4, and allows us to test whether the parental age coefficients are confounded by the other parent's age.

We tested the proportional odds assumption for parental age by testing the significance of the interaction between parental age and time in models 2 and 4, which are the two key models. The tests did not indicate any deviations from proportionality. We further tested whether the parental age coefficients would change if we added time interactions for all other variables in Models 2 and 4. The parental age coefficients changed only marginally. All of the models account for the clustering of siblings within families by using a robust variance-covariance estimator.

RESULTS

Descriptive analyses

Of the total sample of 89,737 persons, 23% were born to mothers under age 25, 52% to mothers aged 25–34, and 25% to mothers aged 35 or above (Table 1). The children were, on average, born in 1944, and there was little variation in birth year by maternal age. Close to half of the sample (48%) were female, and this proportion varied very little by maternal age. Ten percent of the sample were born to fathers under age 25, 50% to fathers aged 25–34, and 40% to fathers aged 35 and above. Maternal and paternal ages were strongly correlated with a linear correlation coefficient 0.72 ($p<.001$).

During the follow-up, 11,582 persons (12.9%) in the sample died. Mortality has a U-shaped association with parental age, as the proportion dead was lowest for those with maternal

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

ages of 20–34, and highest for those with maternal ages of 35 and above. Among those with paternal ages below 20 or above 40, 15 % and 14%, respectively, died during the follow-up; of those with paternal ages of 20–39, only 13% died during the follow-up (not shown).

In 15% of the families, the parents had less than primary school education, 74% had primary school education, and 11% had education beyond primary school. Parental education correlated negatively with maternal age. For example, in the maternal age group 25–29, 11% had less than primary school education, but in the maternal age group 40–49, 35% had less than primary school education. The data on other parental characteristics also suggest that advanced parental age was associated with socioeconomic deprivation. In 1950, those individuals whose mothers were above age 35 at birth lived in larger and more crowded households and in lower-SES families than those individuals born to mothers aged 20–34. The differences were similar for paternal age (not shown). Some of these associations between family socioeconomic characteristics and parental age may reflect cohort differences; to account for this, we control for the year of the child's birth.

At age 35, about 82% of the children had a living mother, 60% had a living father, and 51% had two living parents. These proportions declined sharply as the age of the mother at birth increased: among children born to mothers aged 25–29, about 59% had living parents at age 35, but only 19% of children born to mothers aged 40–49 had living parents at this age.

Individuals born to mothers aged 20–34 had the highest level of schooling at ages 30–34, which suggests an inverse relationship between maternal age and child's socioeconomic attainment. The proportions of offspring who married and had children decreased as maternal age at birth increased. The proportion who owned their homes was, however, stable over maternal ages 25–49. These associations were largely similar for paternal age (not shown).

Regression analyses

Table 2 presents the results from the regression analyses for the offspring age group 35–49, and Table 3 presents the results for the age group 50–74.

Mortality at ages 35–49—Model 1 shows that both young and old maternal ages at birth are associated with excess offspring mortality. Compared to maternal ages 25–29 (reference category), the mortality odds ratios (ORs) for maternal ages 14–19, 35–39, and 40–49 are 1.26 ($p<.05$), 1.17 ($p<.01$), and 1.31 ($p<.001$), respectively. The associations are similar for paternal ages: paternal ages 14–19, 40–49, and 50–64 have ORs of 1.29 ($p<.05$), 1.21 ($p<.001$), and 1.28 ($p<.05$), respectively.

Model 2 adds parental SES to Model 1. The mortality odds ratios for parental characteristics are in the expected direction: children with more educated parents have lower mortality than those with less educated parents. The impact of these controls on the advanced parental age coefficients is, however, modest. For example, maternal ages 35–39 and 40–49 continue to be significantly associated with mortality (ORs 1.14, $p<.05$ and 1.25, $p<.001$, respectively), and paternal ages above 40 continue to be associated with excess mortality.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Model 3 adds own socioeconomic attainment to Model 2. The mortality odds ratios show that higher educational attainment, home ownership, being married, and having children in the household are associated with lower mortality. These controls partially explain the advanced parental age odds ratios: for maternal ages 35–39 and 40–49 they are now 1.11 ($p<.05$) and 1.18 ($p<.05$), respectively; and for paternal ages 40–49 and 50–72 they are now 1.10 ($p<.05$) and 1.11 ($p<.10$), respectively. The ORs for young parental age are also weakened. A fraction of the advanced parental age-mortality association appears to be explained by the child's attained SES and family formation.

Model 4 provides a test of whether parental survival explains the parental age-offspring mortality association. Mortality odds at ages 35–49 is 20–25% lower among those who had a mother or a father alive at the start of the follow-up relative to those who did not. The magnitude of these differences is comparable to the mortality difference between the highest and lowest parental education levels. The impact of controlling for parental survival on the advanced parental age coefficients is strong: the ORs drop in magnitude and lose statistical significance for both maternal and paternal ages. For example, the ORs for maternal ages 35–39 and 40–49 are now 1.04 and 1.06 ($p>.10$ for both), respectively; and for paternal ages 40–49 and 50–65, they are 0.99 and 0.95 ($p>.10$ for both), respectively.

Model 5 adds the other parent's age to Model 4. The results are similar to those obtained with Model 4. The other parent's age does not appear to confound the parental age-offspring mortality association.

We used the KHB method to estimate formally what fraction of the statistically significant advanced maternal and paternal age coefficients obtained with Model 2 is mediated through own SES and through parental survival. The KHB method estimates these fractions from the Model 4 that includes own SES and parental survival as covariates; for details of the method, see Karlson, Holm and Breen (2012). The results showed that although own SES matters, the majority of the mediation is via parental survival. For example, of the odds ratio 1.14 ($p<.01$) for maternal age 35–39 in Model 2, in total 70% was mediated, with 19% going through own SES (all variables referring to own SES combined) and 51% through parental survival (sum of the mediation through maternal survival and paternal survival). Of the odds ratio 1.25 ($p<.001$) for maternal age 40–49 in Model 2, 19% was mediated through own SES and 55% through parental survival, with total mediation fraction being 73%. For paternal ages 40–49 and 50–64 the odds ratios 1.18 ($p<.01$) and 1.22 ($p<.05$) were fully mediated, with approximately a third of the mediation going through own SES and two thirds through parental survival.

We also estimated additional models that included parental survival variables but not own socioeconomic status (not shown). Comparison if the parental survival coefficients in this model to those obtained when own SES is controlled (Model 4) sheds light on whether parental survival influences the parental age-offspring mortality association via intergenerational transfers that might be reflected in own socioeconomic attainment, or via other mechanisms. The difference in parental survival coefficients across these models was approximately 20–25%, suggesting that intergenerational transfers may be only a part of the mechanism that ties offspring survival to the age of the parents when the child was born.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Mortality at ages 50–72—Table 3 shows the results for offspring mortality at ages 50–72. The results are qualitatively similar to those obtained for mortality at ages 35–49, with the main difference being that the unadjusted associations for parental age are weaker. For both age groups, advanced parental age is associated with higher offspring mortality (Model 1); this association is partially explained by parental SES (Model 2; the coefficients become marginally significant); and the associations vanish completely when own SES (Model 3) and parental survival (Model 4) are controlled for. Controlling for the other parent's age (Model 5) does not change these results. None of the parental age coefficients were statistically significant ($p<.05$) in Model 2 so we did not conduct formal mediation analysis for these effects.

Sensitivity analyses

We tested our key result—i.e., that net of parental characteristics and parental survival, advanced parental age is not associated with increased offspring mortality—against several robustness checks. First, the result was robust to changes in the categorization of parental ages, or to using quadratic form for parental ages. Second, we detected no significant sex interaction with parental age. Third, we conducted our analyses separately for one-child and for larger families. This approach indirectly controls for fecundity as large families were the norm when our sample was born. The results were similar for one-child and larger families, suggesting that infecundity is not an important confounder.

We also considered alternative methods for estimating the amount of parental age effects that is mediated by own SES and parental survival. In particular, we estimated accelerated failure time models with Gompertz distribution using the R package eha (Broström 2014) and the procedure described by Broström and Edvinsson (2013) for the mediation analysis. The results were similar to the ones reported here: advanced maternal and paternal ages are associated with increased mortality in particular at age 35–50; a small fraction of this is mediated by own SES and a large fraction by parental survival.

DISCUSSION

We used a large register-based sample drawn from the Finnish census of 1950 to analyze the contributions of social mechanisms such as parental survival up to offspring's age 35, parental socioeconomic characteristics, and offspring's own SES in the association between maternal and paternal ages and offspring mortality at ages 35–49 and 50–72. We found that children born to older parents (mother or father aged 35 or above) had excess mortality when compared to those born to parents aged 25–29. The association was particularly strong for offspring mortality at ages 35–49, but it was also present at ages 50–72. Controls for parental socioeconomic characteristics explained 15–30% of the association. Adding controls for parental survival until the child reached age 35 rendered these associations insignificant for both advanced maternal and paternal ages. A formal mediation analysis confirmed that although both own socioeconomic attainment and parental survival mediate the impact of advanced parental age on mortality, the main mechanism is through parental survival. These results suggest that selection by parental socioeconomic characteristics and factors related to the age at which the child loses his/her parents contribute significantly to

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

the parental age-offspring mortality association, and do not support the idea that biological aging of the reproductive system is the main explanation for this association.

The results were similar for both maternal and paternal ages. Given the large sex differences in reproductive ageing—for example, the average age of the last menstrual period for women is around 50 (Kato et al. 1998), while for men there is no biological limit for becoming a father—differences in the results by maternal age versus paternal age might have been expected if reproductive ageing had been the driving force. The similarities between the maternal and the paternal age associations also suggest that other factors than biological aging, in particular social mechanisms, are more important.

The key difference in the results for mortality at ages 35–49 and 50–72 was that the unadjusted association between advanced parental age and offspring mortality was weaker at older ages. Thus, the controls that explained the advanced parental age-offspring mortality association—parental SES and parental survival—were more important for mortality at ages 35–49 than at ages 50–72. This was also reflected in the mortality odds ratios associated with parental SES and parental survival. For example, the protective effects of parental education and parental survival were found to be approximately 50% smaller at ages 50–72 than at ages 35–49. The finding that parental SES explains some of the advanced parental age-offspring mortality association can be attributed to the patterning of parental SES with parental age. Although older parents today tend to have above-average socioeconomic status and resources (Bray et al. 2006), in Finland in the early 20th century this pattern was reversed, with older parents having the lowest level of schooling, as was shown by our descriptive statistics.

The survival of the parents until the child reached age 35 was found to be more important than parental socioeconomic characteristics in explaining the association between advanced parental age and offspring mortality. Although our measure of parental survival was crude, it nevertheless captured important variations in parental survival associated with the parents' ages at the birth of the child. The mechanisms through which parental survival beyond the birth of the child may influence the parental age-offspring health association are twofold, and may have two opposing effects.

First, parental survival may be a proxy for shared frailty within the family. The lifespans of parents and their children are correlated. It is also possible that long-lived parents have children at older ages than their shorter-lived peers. These correlations could confound the advanced parental age-offspring mortality association. Late reproduction may also signal age-independent problems in fecundity. Fecundity problems may be correlated with the general level of health. If some portion of poor health status is heritable—that is, if frailty is shared within the family—such correlations would confound the parental age-offspring health association. There is, however, no direct evidence that shared frailty would be connected to the aging of the reproductive system that is often hypothesized to be the primary explanation for the association between parental age at birth and offspring health.

Second, parental survival may also be a proxy for parental investments in their children. Parental survival is correlated with parental age. Other factors being held constant, a child

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

born to a 20-year-old mother will, on average, lose the mother at an age 20 years older than that of a child born to a 40-year-old mother. Thus, parental survival may be correlated with intergenerational transfers (e.g. time spent with homework and education, or financial transfers in early adulthood), and these transfers may contribute to the association between parental age and offspring mortality. To investigate more closely these two mechanisms (i.e., shared family frailty versus intergenerational transfers), we tested for the socioeconomic transfer mechanism by adjusting the regressions with the child's own socioeconomic attainment, which is at least partially a function of parental investments (results not shown). This control explained only 20–25% of the coefficients for parental survival. This result suggests that an important share of the observed protective association for parental survival may be related to factors other than intergenerational transfers; these may include shared family frailty, as discussed above.

Our results are consistent with several recent studies that have examined parental age effects on offspring health. Myrskyla and Fenelon (2012) analyzed the U.S. Health and Retirement Study, and found that, net of maternal education and maternal survival, the association between advanced maternal age and offspring adult mortality is weak. Hubbard et al. (2009) analyzed the health and mortality of Canadian 20th century cohorts, and found no maternal age effects on mortality. Robine et al. (2003) found no association between parental age and the probability of surviving to age 100. Westendorp and Kirkwood (2001), in an analysis of British historical aristocracy, also failed to find maternal age effects on longevity. Smith et al. (2009) analyzed the Utah Population Database, which consists mostly of people of the Mormon faith, and found that maternal ages above 35 were associated with an 8% increase in adult mortality for sons; but this association, while statistically significant, was small in terms of magnitude.

Pre-birth selection may partially explain the weak association between parental age and offspring adult health and mortality. The force of selection is by far strongest in utero. This selection—spontaneous abortions and stillbirths—increases with maternal age (de La Rochebrochard and Thonneau 2002). This maternal age-dependent quality control may partially explain why mortality differences among adults by parental age are small.

Our results pertain to a population with moderately high fertility and rapidly declining mortality. The analyzed cohorts were born between 1936 and 1950. During 1936–1944 Finnish total fertility rate fluctuated between 2 and 2.5, and for the period 1945–1950 following the second world war increased to above 3 (Statistics Finland 2013). Some of the earlier results on parental age and offspring mortality were obtained from high-fertility populations in which mortality advances were modest (e.g., Kemkes-Grottenthaler 2004) and it is possible that in such contexts the associations differ from those observed in modern populations. Within our data, however, the results were not cohort-specific, as the key result – that net of parental survival and parental socio-demographic characteristics, parental age does not predict offspring mortality – was obtained in cohorts that were born before, during, or after the second world war (results not shown).

Our analytical sample size was 89,737 persons, and the data selection process resulted in us losing 26,885 of the original sample of 116,622. Of these 16,066 were excluded because

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

they died or had moved out of the country before age 35. Of the remaining 10,819 persons that were excluded the majority, 7,729, were excluded because they had only one parent present in the 1950 census. Only 3,090, or less than 4%, were excluded because of missing data or very high or low parental age. Thus the analyzed sample of 89,737 persons is largely representative of the population that was aged 0–14 and resided in Finland in 1950 and was alive in Finland at age 35.

Our findings are not inconsistent with the studies that have shown that older mothers have worse birth outcomes than younger mothers in terms of, for example, higher probability of malformations or chromosomal abnormalities (Andersen et al. 2000). There are, however, two reasons why these negative birth outcomes generally have only a small effect on population-level adult mortality. First, these conditions are rare. The incidence of Down Syndrome, the most common chromosomal abnormality, is only about 1% at maternal age 40 (Trimble, Baird and Opitz 1978). Consequently, the population-level impact on health by individuals whose mothers were 40 years old when they were born cannot be large. The same applies to several other conditions (schizophrenia, bipolar affective disorder, childhood cancer) that have been linked to advanced parental age as their prevalence is not high. Second, life expectancy for those with poor initial health is lower than for those with good initial health, which results in a healthier sample of adults. For example, in the 1940s life expectancy with Down Syndrome was 12 years (Bittles et al. 2007). Thus early selection may drive some of the differences between our results that focus on adult ages and those that focus on earlier outcomes.

Limitations of the study

First, our data do not allow us to definitively address what parental survival proxies. Our analyses nevertheless clearly show the importance of parental survival on the advanced parental age-offspring adult mortality association, and suggest that the association reflects factors other than reproductive ageing. Second, we were not able to analyze the impact of changing parental characteristics. Powell et al. (2006) suggested that older parents transmit more economic and social resources to their children than younger parents. These positive factors may offset some of the negative effects induced by reproductive ageing. Third, we did not cover child and early adult mortality, for which the associations between parental age and mortality may be stronger than for older ages. Additional insights may be obtained from future research that focuses on younger ages. Fourth, we could not include birth order in the models because siblings who had died or moved out of the family before the census date of 1950 are not included in the data. This omission does not render our findings invalid because birth order is positively associated with both parental age and adult mortality (Modin 2002). Therefore not controlling for birth order results in an upward bias in the advanced parental age coefficients and our models are more likely to over- than underestimate the association between advanced parental age and offspring adult mortality. Thus, it is unlikely that advanced parental age would emerge as a predictor of increased mortality if we could control for birth order. Finally, our study focused on all-cause mortality. It is possible that the associations with parental age could be different for specific causes of death for which physiological or biological pathways are particularly important. Further studies should analyze more specific outcomes.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

In summary, this analysis suggests that advanced maternal and paternal ages are not associated with increased mortality for the offspring at adult ages after parental socioeconomic status and parental survival are controlled for. Instead, in our study population, a representative sample of Finnish cohorts born in 1936–1950, the association was shown to be driven by factors related to parental survival until the child reached age 35, and by older parents having lower socioeconomic attainment. The parental socioeconomic difference is indicative of confounding, whereas the parental survival mechanism could signal shared within-family frailty or truncated intergenerational transmission of socioeconomic resources. While we were able to measure intergenerational transfers only indirectly via the child's socioeconomic attainment, the results still suggested that only a fraction of this mechanism is related to such transfers. Moreover, with secular declines in mortality, the role of intergenerational transfers may further decrease as the risk of early parental loss decreases. These results suggest that the causal impact of being born to an older mother or father on adult mortality is not large. However, our findings focus on a population born before the use of modern assisted reproductive technologies (ART) or prenatal screening, both of which may change the parental age–offspring adult health association by allowing less fecund couples to reproduce and by screening for less fit fetuses. The balance of these opposing two processes on the parental age-offspring health should be the focus of later studies.

References

Allison, PD. Fixed Effects Regression Models. Sage; 2009.

American Society for Reproductive Medicine. Age and fertility: a guide for patients. Birmingham: American Society for Reproductive Medicine; 2003.

Andersen AMN, Wohlfahrt J, Christens P, Olsen J, Melbye M. Maternal age and fetal loss: population based register linkage study. Bmj. 2000; 320:1708–1712. [PubMed: 10864550]

Armstrong DT. Effects of maternal age on oocyte developmental competence. Theriogenology. 2001; 55:1303–1322. [PubMed: 11327686]

Benzies KM. Advanced maternal age: Are decisions about the timing of child-bearing a failure to understand the risks? Canadian Medical Association Journal. 2008; 178:183–184. [PubMed: 18195294]

Bittles AH, Bower C, Hussain R, Glasson EJ. The four ages of Down syndrome. The European Journal of Public Health. 2007; 17:221–225.

Bray I, Gunnell D, Davey Smith G. Advanced paternal age: How old is too old? Journal of Epidemiology and Community Health. 2006; 60:851–853. [PubMed: 16973530]

Breen, Richard; Karlson, Kristian Bernt; Holm, Anders. Total, Direct, and Indirect Effects in Logit and Probit Models. Sociological Methods & Research. 2013; 42(2):164–191.

Broström G. eha: Event History Analysis. R package version 2.4-1. 2014

Broström, G.; Edvinsson, S. A parametric model for old age mortality in mediation analysis. Paper presented at the IUSSP 2013 Conference in Busan; Korea. 2013. Available at http://www.iussp.org/sites/default/files/event_call_for_papers/AFT13.pdf

Case A, Paxson C, Ableidinger J. Orphans in Africa: Parental Death, Poverty, and School Enrollment. Demography. 2004; 41:483–508. [PubMed: 15461011]

Cutler D, Deaton A, Lleras-Muney A. The Determinants of Mortality. The Journal of Economic Perspectives. 2006; 20:97–120.

de La Rochebrochard E, Thonneau P. Paternal age and maternal age are risk factors for miscarriage; results of a multicentre European study. Human Reproduction. 2002; 17:1649–1656. [PubMed: 12042293]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Durkin MS, et al. Advanced Parental Age and the Risk of Autism Spectrum Disorder. American Journal of Epidemiology. 2008; 168:1268–1276. [PubMed: 18945690]

Gale EAM. Maternal age and diabetes in childhood. Bmj. 2010; 340:c623. [PubMed: 20181638]

Gavrilov, LA.; Gavrilova, NS. Human longevity and parental age at conception. In: Robine, J-M.; Kirkwood, TBL.; Allard, M., editors. Sex and longevity: sexuality, gender, reproduction, parenthood. Berlin, Heidelberg: Springer-Verlag; 2000. p. 7-31.

Gavrilov LA, Gavrilova NS. Biodemography of exceptional longevity: early-life and mid-life predictors of human longevity. Biodemography and Social Biology. 2012; 58:14–39. [PubMed: 22582891]

Hayward MD, Gorman BK. The Long Arm of Childhood: The Influence of Early-life Social Conditions on Men's Mortality. Demography. 2004; 41:87–107. [PubMed: 15074126]

Heffner LJ. Advanced Maternal Age - How Old Is Too Old? New England Journal of Medicine. 2004; 351:1927–1929. [PubMed: 15525717]

Hubbard RE, Andrew MK, Rockwood K. Effect of parental age at birth on the accumulation of deficits, frailty and survival in older adults. Age and Ageing. 2009; 38:380–385. [PubMed: 19307228]

Karlson Kristian B, Anders Holm, Breen Richard. Comparing Regression Coefficients Between Same-Sample Nested Models using Logit and Probit: A New Method. Sociological Methodology. 2012; 42:286–313.

Kato I, Toniolo P, Akhmedkhanov A, Koenig K, Shore R, Zeleniuch-Jacquotte A. Prospective study of factors influencing the onset of natural menopause. Journal of Clinical Epidemiology. 1998; 51:1271–1276. [PubMed: 10086819]

Kaytor MD, Burright EN, Duvick LA, Zoghbi HY, Orr HT. Increased trinucleotide repeat instability with advanced maternal age. Human Molecular Genetics. 1997; 6:2135–2139. [PubMed: 9328478]

Kemkes-Grottenthaler A. Parental effects on offspring longevity - evidence from 17th to 19th century reproductive histories. Annals of Human Biology. 2004; 31:139–158. [PubMed: 15204358]

Ulrich, Kohler; Karlson Kristian, B.; Holm, Anders. Comparing Coefficients of Nested Nonlinear Probability Models. The Stata Journal. 2011; 11:1–19.

Kong A, et al. Rate of de novo mutations and the importance of father's age to disease risk. Nature. 2012; 488:471–475. [PubMed: 22914163]

Liu Y, Zhi M, Li X. Parental age and characteristics of the offspring. Ageing Research Reviews. 2011; 10:115–123. [PubMed: 20887815]

Malaspina D, et al. Paternal age and intelligence: Implications for age-related genomic changes in male germ cells. Psychiatric Genetics. 2005; 15:117–125. [PubMed: 15900226]

Miller B, et al. Advanced paternal age, mortality, and suicide in the general population. Journal of Nervous & Mental Disease. 2010; 198:404–411. [PubMed: 20531118]

Modin B. Birth order and mortality. A life-long follow-up of 14,200 boys and girls born in early twentieth century Sweden. Social Science and Medicine. 2002; 54:1051–1064. [PubMed: 11999502]

Myrskylä M, Fenelon A. Maternal Age and Offspring Adult Health: Evidence from the Health and Retirement Study. Demography. 2012; 49:1231–1257. [PubMed: 22926440]

Ní Bhrolcháin M. Flexibility in the marriage market. Population - An English Selection. 2001; 13:9–47.

Powell B, Steelman LC, Carini RM. Advancing age, advantaged youth: Parental age and the transmission of resources to children. Social Forces. 2006; 84:1359–1390.

Robine JM, Cournil A, Henon N, Allard M. Have centenarians had younger parents than the others? Experimental Gerontology. 2003; 38:361–365. [PubMed: 12670622]

Rocca WA, et al. Maternal Age and Alzheimer's Disease: A Collaborative Re-analysis of Case-Control Studies. International Journal of Epidemiology. 1991; 20:S21–27. [PubMed: 1833350]

Saha S, et al. Advanced Paternal Age Is Associated with Impaired Neurocognitive Outcomes during Infancy and Childhood. PLOS Medicine. 2009; 6:e1000040.

Schmid TE, et al. The effects of male age on sperm DNA damage in healthy non-smokers. Human Reproduction. 2007; 22:180–187. [PubMed: 17053003]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Smith KR, Mineau GP, Garibotti G, Kerber R. Effects of childhood and middle-adulthood family conditions on later-life mortality: Evidence from the Utah Population Database, 1850–2002. Social Science & Medicine. 2009; 68:1649–1658. [PubMed: 19278766]

Statistics Finland. Vuoden 1950 väestölaskennan otosaineiston käsikirja (Handbook of the 1950 census sample). Helsinki, Finland: Statistics Finland; 1997.

Statistics Finland. Statfin Database. 2013. http://www.stat.fi/tup/tilastotietokannat/index_en.html Data downloaded on November 18 2013

Tarín JJ, Brines J, Cano A. Long-term effects of delayed parenthood. Human Reproduction. 1998; 13:2371–2376. [PubMed: 9806250]

Trimble BK, Baird PA, Opitz JM. Maternal age and down syndrome: Age-specific incidence rates by single-year intervals. American Journal of Medical Genetics. 1978; 2:1–5. [PubMed: 162533]

Westendorp RGJ, Kirkwood TBL. Maternal and paternal lines of familial longevity. Population: An English Selection. 2001; 13:223–235.

Yip BH, Pawitan Y, Czene K. Parental age and risk of childhood cancers: a population-based cohort study from Sweden. International Journal of Epidemiology. 2006; 35:1495–1503. [PubMed: 17008361]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Research highlights

- We study whether social selection or parental survival explains the higher adult mortality of those born to older parents.
- Analyzing a Finnish 1936–1950 birth cohort, we find that the excess adult mortality is explained by early parental loss.
- The results do not support the idea that reproductive ageing is responsible for the association.
- The timing of parental loss is likely to reflect both within-family frailty and intergenerational transfers.
- With increasing longevity, the population-level importance of early parental loss on offspring adult mortality may decline.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Soc Sci Med. Author manuscript; available in PMC 2015 October 01.

Table 1

Demographic and family characteristics by maternal age, 10% sample of the Finnish census of 1950.

	Maternal age						
	All	**14–19**	**20–24**	**25–29**	**30–34**	**35–39**	**40–49**
Demographic characteristics							
Sample size (%)	89,737	1,928 (2.1)	18,702 (20.8)	26,037 (29.0)	21,323 (23.8)	14,726 (16.4)	7,021 (7.8)
Time of birth (SD)	1944.4 (4.3)	1944.5 (4.5)	1944.7 (4.3)	1944.4 (4.4)	1944.2 (4.2)	1944.6 (4.1)	1944.5 (4.2)
Female, %	48.0	47.1	47.7	47.9	48.1	48.1	48.6
Paternal age, %							
14–19	0.6	6.7	1.7	0.3	0.1	0.1	0.1
20–24	9.4	46.4	28.9	6.9	1.2	0.2	0.1
25–29	24.5	33.7	45.0	38.1	11.7	2.8	1.0
30–34	25.8	9.5	17.8	35.6	37.0	14.5	4.5
35–39	20.3	2.4	4.6	13.7	33.1	37.4	16.3
40–49	17.0	1.2	1.7	4.8	15.4	40.5	63.2
50–64	2.5	0.2	0.2	0.6	1.5	4.5	14.8
Mortality							
Mean follow-up, years (SD)							
For those who died	19.4 (8.5)	18.6 (8.6)	19.5 (8.5)	19.8 (8.5)	19.6 (8.5)	18.9 (8.5)	18.7 (8.6)
For censored	28.3 (4.2)	28.2 (4.5)	28.1 (4.3)	28.4 (4.3)	28.6 (4.1)	28.2 (4.1)	28.3 (4.1)
Died, %	12.9	13.3	12.5	12.8	12.8	13.2	14.2
Independent variables							
Parental socioeconomic characteristics, %							
Family education							
Below primary	15.3	8.2	8.2	10.6	16.0	23.3	34.5
Primary	74.2	84.6	81.7	77.2	72.4	67.7	59.9
Beyond primary	10.5	7.1	10.1	12.3	11.7	9.0	5.6
Occupation of family head, %							
Professional/administrative	15.3	10.4	14.5	17.5	16.6	13.7	9.8
Agricultural/other workers	42.3	57.4	50.0	42.4	39.7	37.6	34.9
Farmers, < 10 hectares	25.9	19.9	21.2	24.2	26.5	30.3	34.8

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

	Maternal age						
	All	**14–19**	**20–24**	**25–29**	**30–34**	**35–39**	**40–49**
Farmers, ≥ 10 hectares	8.4	3.8	6.1	7.7	9.1	10.6	11.7
Other	8.2	8.5	8.2	8.1	8.2	7.8	8.7
Number of children aged<18, %							
1	14.2	29.2	20.4	12.3	10.3	12.0	17.4
2	26.2	30.6	33.3	28.4	22.6	20.3	21.0
3	22.4	19.2	22.1	24.4	22.9	20.4	19.8
4	37.2	21.0	24.3	34.9	44.2	47.4	41.8
Persons per heated room (SD)	2.7 (1.6)	2.7 (1.4)	2.6 (1.5)	2.6 (1.5)	2.7 (1.7)	2.9 (1.8)	3.0 (1.8)
Swedish speaking, %	7.2	6.0	6.7	7.7	7.7	7.2	6.0
Parental survival, %							
Mother alive when ego 35	81.9	89.3	89.6	87.0	82.2	73.5	57.5
Father alive when ego 35	59.6	74.3	73.5	67.0	56.6	44.4	31.7
Both alive when ego 35	50.9	67.5	66.8	59.2	47.6	33.9	19.4
Own socioeconomic characteristics							
Education at ages 30–35, %							
9 years – basic	47.2	50.8	45.6	44.8	47.4	49.9	53.7
10–11 years – lower sec.	26.4	27.8	27.3	27.0	25.6	26.0	24.9
12 years – upper secondary	13.1	12.0	14.0	13.7	13.2	11.9	11.4
13+ years – post secondary	13.2	9.4	13.2	14.4	13.9	12.1	10.0
Marital status 30–35, %							
Never married	15.3	10.7	12.3	14.5	16.2	18.3	18.7
Married	74.7	76.6	76.5	75.5	74.5	72.5	72.1
Separated/divorced/widowed	10.0	12.7	11.3	10.0	9.4	9.3	9.2
Own home, %	70.5	66.1	69.4	70.8	70.9	71.3	70.9
Has children, %	81.1	82.8	82.9	81.5	80.7	79.7	78.3

Soc Sci Med. Author manuscript; available in PMC 2015 October 01.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Soc Sci Med. Author manuscript; available in PMC 2015 October 01.

TABLE 2

Mortality odds ratios by maternal and paternal age. Data: 10% sample from Finnish census of 1950 with mortality follow-up from 1971 to December 31st, 2007 and over ages 35 to 49. N = 89,737 persons, 3,454 deaths.

	Model 1		Model 2		Model 3		Model 4		Model 5
	Maternal age	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal and paternal ages**
Maternal age (ref: 25–29)									
14–19	1.26*		1.20		1.17		1.20		1.15
20–24	0.99		0.98		0.98		1.00		0.98
30–34	1.05		1.04		1.03		1.00		1.02
35–39	1.17***		1.14*		1.11*		1.04		1.09
40–49	1.31***		1.25***		1.18*		1.06		1.09
Factor significance[a]	<0.001		<0.01		<0.05		>.10		>.10
Paternal age (ref: 25–29)									
14–19		1.29*		1.22		1.14		1.15	1.12
20–24		1.13^ t		1.08		1.06		1.08	1.08
30–34		1.07		1.07		1.04		1.02	1.00
35–39		1.11*		1.1^ t		1.05		0.99	0.95
40–49		1.21***		1.18**		1.10*		0.99	0.92
50–64		1.28*		1.22^ t		1.11^ t		0.95	0.87
Factor significance[a]		<0.01		<0.05		>.10		>.10	>.10
Parental SES									
Family educ. (ref: < primary)									
Primary			0.82***	0.81***	0.87**	0.86***	0.89*	0.87**	0.88**
Beyond primary			0.76***	0.75***	0.90	0.88	0.91	0.90	0.91
Factor significance[a]			<0.001	<0.001	<0.01	<0.001	<0.001	<0.05	<0.05
Occupation of family head (ref:									
Professional/admin.			0.87*	0.87*	0.99	0.99	0.99	1.00	1.00
Farmers, < 10 hectares			0.83***	0.83***	0.88**	0.88**	0.88**	0.89**	0.89**

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

	Model 1		Model 2		Model 3		Model 4		Model 5
	Maternal age	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal and paternal ages**
Farmers, >= 10 hectares			0.73***	0.73***	0.84*	0.84*	0.84*	0.85*	0.85*
Other			0.93	0.93	0.98	0.99	0.98	0.98	0.98
Factor significance[a]			<0.001	<0.001	<0.01	<0.05	<0.05	<0.05	<0.05
No. of children aged <18 (ref: 2)									
1			1.08	1.08	1.07	1.08	1.07	1.07	1.07
3			1.01	1.01	0.98	0.98	0.98	0.98	0.98
4+			0.99	0.99	0.94	0.95	0.94	0.95	0.95
Factor significance[a]			>.10	>.10	>.10	>.10	>.10	>.10	>.10
No. of persons /heated room			1.05***	1.05***	1.02^t	1.02^t	1.02^t	1.02^t	1.02^t
Swedish speaking (ref: Finnish)			0.81**	0.81**	0.81**	0.81**	0.81**	0.82**	0.81**
Own SES									
Education (ref: 9 years)									
10–11 years					0.83***	0.83***	0.84***	0.83***	0.83***
12 years					0.68***	0.68***	0.69***	0.70***	0.70***
13+ years					0.54***	0.54***	0.55***	0.56***	0.56***
Factor significance[a]					<0.001	<0.001	<0.001	<0.001	<0.001
Marital status (ref: Never marr.)									
Married					0.55***	0.55***	0.55***	0.55***	0.55***
Separated/divorced/widow					1.05	1.05	1.05	1.05	1.05
Factor significance[a]					<0.001	<0.001	<0.001	<0.001	<0.001
Has children					0.72***	0.72***	0.73***	0.73***	0.73***
Own home					0.72***	0.72***	0.72***	0.72***	0.72***
Parental survival									
Mother alive when ego 35							0.85***	0.84***	0.85***
Father alive when ego 35							0.83***	0.81***	0.82***
Factor significance[a]							<0.001	<0.001	<0.001

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

	Model 1		Model 2		Model 3		Model 4		Model 5
	Maternal age	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal and paternal ages**
Log likelihood	−23187.1	−23192.5	−22867.9	−22872.0	−22430.6	−22434.5	−22409.2	−22409.7	−22407.0

^ t p<.10

* p<.05

** p<.01

*** p<.001;

[a] Significance level of the factor variable; All models control for sex, year of birth and period effects

Soc Sci Med. Author manuscript; available in PMC 2015 October 01.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Soc Sci Med. Author manuscript; available in PMC 2015 October 01.

TABLE 3

Mortality odds ratios by maternal and paternal age. Data: 10% sample from the Finnish census of 1950 with mortality follow-up from 1971 to 31st December 2007 and over ages 50 to 72. N = 86,283 persons, 8,128 deaths.

	Model 1		Model 2		Model 3		Model 4		Model 5
	Maternal age	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal and paternal ages**
Maternal age (ref: 25–29)									
14–19	1.00		0.93		0.92		0.93		0.92
20–24	1.01		0.99		0.99		1.00		1.00
30–34	0.99		0.99		0.99		0.97		0.99
35–39	1.01		1.01		0.98		0.95		0.99
40–49	1.10*		1.07^ t		1.03		0.96		1.02
Factor significance[a]	<0.1		>0.1		>0.1		>0.1		>0.1
Paternal age (ref: 25–29)									
14–19		1.05		1.02		0.99		1.00	1.01
20–24		1.06		1.03		1.01		1.02	1.03
30–34		1.03		1.05		1.03		1.02	1.02
35–39		1.01		1.02		0.98		0.95	0.95
40–49		1.08*		1.07^ t		1.02		0.95	0.95
50–64		1.04		1.02		0.94		0.92	0.93
Factor significance[a]		<0.1		>0.1		>0.1		>0.1	>0.1
Parental SES									
Family educ. (ref: < primary)									
Primary			0.92*	0.92**	0.98	0.97	0.99	0.98	0.98
Beyond primary			0.88*	0.87*	1.04	1.03	1.05	1.04	1.04
Factor significance[a]			<0.05	<0.05	>.10	>.10	>.10	>.10	>.10
Occupation of family head (ref:									
Professional/admin.			0.82***	0.82***	0.92*	0.92*	0.92^ t	0.92^ t	0.92^ t
Farmers, < 10 hectares			0.84***	0.84***	0.86***	0.87***	0.87***	0.87***	0.87***
Farmers, >= 10 hectares			0.74***	0.74***	0.81***	0.81***	0.81***	0.82***	0.81***

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

	Model 1		Model 2		Model 3		Model 4		Model 5
	Maternal age	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal age**	**Paternal age**	**Maternal and paternal ages**
Other			0.95	0.95	0.99	0.99	0.98	0.99	0.99
Factor significance[a]			<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
No. of children aged <18 (ref: 2)									
1			1.05	1.06	1.05	1.05	1.04	1.05	1.05
3			0.96	0.96	0.94^ t	0.94^ t	0.94^ t	0.94^ t	0.94^ t
4+			0.97	0.96	0.96	0.96	0.96	0.96	0.96
Factor significance[a]			>.10	>.10	>.10	>.10	>.10	>.10	>.10
No. of persons /heated room			1.02**	1.02**	1.00	1.00	1.00	1.00	1.00
Swedish speaking (ref: Finnish)			0.81***	0.81***	0.80***	0.80***	0.80***	0.80***	0.80***
Own SES									
Education (ref: 9 years)									
10–11 years					0.80***	0.80***	0.80***	0.80***	0.80***
12 years					0.72***	0.72***	0.73***	0.73***	0.73***
13+ years					0.53***	0.53***	0.54***	0.54***	0.54***
Factor significance[a]					<0.001	<0.001	<0.001	<0.001	<0.001
Marital status (ref: Never marr.)									
Married					0.57***	0.57***	0.57***	0.57***	0.57***
Separated/divorced/widow					1.01	1.01	1.01	1.01	1.01
Factor significance[a]					<0.001	<0.001	<0.001	<0.001	<0.001
Has children					0.85***	0.85***	0.86***	0.86***	0.86***
Own home					0.76***	0.76***	0.76***	0.76***	0.76***
Parental survival									
Mother alive when ego 35							0.92**	0.92**	0.92**
Father alive when ego 35							0.89***	0.88***	0.88***
Factor significance[a]							<0.001	<0.001	<0.001
Log likelihood	−45972.8	−45973.2	−45477.7	−45477.5	−44823.8	−44823.2	−44806.7	−44803.6	−44802.8

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

^ t p<.10

* p<.05

** p<.01

*** p<.001;

[a] Significance level of the factor variable; All models control for sex, year of birth and period effects