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Is young fatherhood causally related to midlife mortality? A sibling fixed-effect study in Finland

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ABSTRACT

Background Previous studies have shown that young fatherhood is associated with higher later-life mortality. It is unclear whether the association is credible, in the sense that mortality and young fatherhood appear to be associated because both are determined by family-related environmental, socioeconomic and genetic characteristics.

Methods We used a household-based 10% sample drawn from the 1950 Finnish census to estimate all-cause mortality of fathers born during 1940–1950. The fathers were followed from age 45 until death, or the end of age 54. We used a standard Cox model and a sibling fixed-effects Cox model to examine whether the effect of young fatherhood was independent of observed adulthood characteristics and unobserved early-life characteristics shared by brothers.

Results Men who had their first child before the age of 22 or at ages 22–24 had higher mortality as compared with their brothers who had their first child at the median or mean age of 25–26. Men who had their first child later at ages 30–44 had lower mortality than their brothers who had a first child before the age of 25. The pattern of results from a standard model was similar to that obtained from a fixed-effects sibling model.

Conclusions The findings suggest a causal effect of young fatherhood on mortality and highlight the need to support young fathers in their family life to improve health behaviours and health.

INTRODUCTION

Men who father a child early in life have been shown to have poorer physical health and higher later-life mortality than men who delay fatherhood.[1 2] Fatherhood often requires financial commitments over a long period of time and can reduce young men's ability to invest in their own human capital.[3 4] The need to provide immediate financial support for a family may interrupt investment in job skills or career development, and push young men into lower paid employment,[5] which could have adverse consequences on their later health. Early-timed parenthood may also disrupt psychological transition from adolescence to adulthood, and leave little time for exploration and appropriate individuation.[6 7] Young fathers, co-resident with their children, have been suggested to experience an increase in depressive symptoms during the years after the birth of their first child.[8]

Despite a growing interest in young fatherhood and its consequences, causal inference from observational data remains difficult due to the confounding effects of omitted variables, such as early-life conditions, intergenerational values and attitudes, norms of behaviour, parental practices and genetic characteristics. The effect of young fatherhood may be biased by factors that affect both the timing of fatherhood and mortality. For example, young fatherhood has been shown to relate to structural family characteristics, such as low socioeconomic status and educational level of own parents,[9–12] their divorce,[13] mother's or father's young age at first birth,[7 10] and living in disadvantaged neighbourhoods,[14] all of which could have adverse consequences on health and mortality.[15–18]

In addition to family structure, socialisation to certain norms and parental practices may also influence both entry into young fatherhood and mortality. Low parental monitoring has been shown to increase the likelihood of becoming a young father, independent of the family's socioeconomic status and mother's age at first birth.[7] Low parental monitoring is also known to affect risky health behaviour, such as alcohol use in adolescence and problem drinking in early adulthood,[19 20] which may increase mortality at later stages in life.

Although it is widely acknowledged that the lack of data on confounding factors may induce considerable bias in the estimates of the effects of early-timed fatherhood on mortality, no previous studies have used a sibling design to control for unobserved environmental and genetic family characteristics shared by brothers. A sibling design can help to reduce the potential bias caused by omitted variables because brothers share many characteristics of their early-life environment and also some of their genetic makeup.[21]

This study aims at analysing the association between the timing of a first child and midlife mortality in Finland by using (1) a standard Cox model that controls for observed adulthood characteristics, such as education, marital status, number of children and region of residence and (2) a fixed-effects model that also allows controlling for unobserved early-life characteristics shared by brothers. All-cause mortality is analysed from the age of 45 until 54 for men born during 1940–1950.

METHODS

Data

We used a 10% sample of households drawn from the 1950 Finnish census that included information allowing for the identification of family members. The original 1950 household census was organised into 7300 folders according to the region of residence. For example, over 1000 folders were needed to cover the capital and one folder to cover a small municipality. Every 10th folder was included in the sample. Of the 547 municipalities, 72% are represented in the sample. The sample

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well represents the demographic and socioeconomic characteristics of the total population.[22] The sample was further linked to information on mortality, fertility and quinquennial 1970–1995 censuses. The data linkage was carried out by Statistics Finland using personal identification codes that were introduced in the late 1960s. The use of data was approved by Statistics Finland (permission TK-53-704-10).

We studied men born during 1940–1950 in order to construct almost complete records of the timing of fatherhood. We had month-level information on the time of birth of fathers and their children. These dates were set on the 15th day of the corresponding month and year, and used to create a variable indicating the age of a father at the birth of his first child in completed years. Our analyses were restricted to men who were alive and had at least one child by the age of 45. Of the 30 565 fathers, 15% fathered a first child by the age of 22, 29% at ages 22–24, 18% at ages 25–26, 19% at ages 27–29 and 19% at ages 30–44. The median age on having the first child was 25 (IQR: 5), and the mean age was 26 (SD 5). A few men (0.8% of the fathers), who had the first child after having reached the age of 45, were excluded from our mortality analysis in order to avoid reverse causality between health and late fatherhood. Of the 30 565 fathers, approximately 38% were identified to be brothers. On the basis of this subsample of 11 743 brothers in 5301 families, we estimated fixed-effects models.

Control variables

We controlled for the year of birth and observed adulthood characteristics, such as education, marital status, number of children and region of residence in all our models. Educational level was controlled, because of its close links with both fertility timing and mortality,[16 23] although the direction of causality between education and fertility is ambiguous.[24] We further controlled for marital status, because young fatherhood was associated with unstable marriages in our sample, and marriage has been shown to be associated with decreased mortality.[25] The number of children was controlled, because those who have a child early in life tend to have more children, and it has been suggested, it increases all-cause mortality—at least among women.[26–28] Region of residence was taken into account, because of its close links with both fertility timing and mortality in Finland.[29 30]

All control variables, except for the number of children, were measured at ages 35–39 based on the censuses of 1975, 1980 and 1985, depending on the cohort. The number of children ever born was calculated at the end of 2009. The distribution of the control variables, excluding Finnish regions and the year of birth, by the timing of a first child, is shown in table 1.

Statistical methods

We had month-level information on the time of birth and death of the fathers. These dates were set on the 15th day of the corresponding month and year to create a variable indicating the age at death. The risk of death was estimated with Cox regression models with age as underlying time. The follow-up was calculated from the date of turning age 45 years until the date of death, or the date before turning age 55 years if still alive. Approximately 5% of the fathers died during the 10-year follow-up. The predominant causes of death were ischaemic heart diseases (21% of deaths) and alcohol-related diseases and alcohol poisonings (16% of deaths). Cause-specific mortality analyses fell, however, beyond the scope of the study.

We first used a standard Cox model to study the association between the timing of a first child and total mortality, controlling for observed adulthood characteristics and the year of birth. Each control variable was added to the model as a categorical, time-invariant variable. The results were presented in terms of HRs and their 95% CIs. The clustering in the sample by

Table 1 Sample characteristics of those who had at least one child by the age of 45, by the timing of a first child, men born in 1940–1950, Finland

	Men (N=30 565), % Timing of a first child (age of a father in completed years)					
	<22	22–24	25–26	27–29	30–44	All
Education						
Basic or less (≤9 years)	51.1	44.7	37.3	32.1	35.1	40.1
Lower secondary (10–11 years)	32.5	31.9	28.4	26.2	27.1	29.4
Upper secondary (12 years)	11.0	12.8	15.1	18.1	16.3	14.6
Tertiary (≥13 years)	5.4	10.6	19.2	23.7	21.5	15.9
Marital status						
Married	81.4	87.5	90.2	90.5	80.2	86.2
Never married	0.4	0.7	1.1	2.4	14.5	3.7
Divorced/separated	17.3	11.0	8.0	6.4	4.5	9.3
Widowed/unknown	1.0	0.8	0.7	0.7	0.8	0.8
Number of children						
1	14.7	17.6	18.9	23.3	38.5	22.5
2	41.4	46.5	50.0	50.0	43.6	46.5
3	27.3	24.4	22.2	19.6	13.6	21.5
4+	16.7	11.5	8.9	7.2	4.3	9.6
All (%)	100.0	100.0	100.0	100.0	100.0	100.0
Proportion of deaths (%)	6.6	5.3	4.3	4.1	3.7	4.8
Number of deaths (at ages 45–54)	306	467	236	237	219	1465
N	4654	8863	5435	5729	5884	30 565

households was taken into account in the estimation of robust SEs. Men who fathered a first child at the median or mean age of 25–26 were selected as the reference group. Unadjusted estimates were not of substantial interest to us, and they are in the online supplementary appendix 1.

Second, we used a Cox regression model with sibling fixed effects that allowed controlling for all predictor variables that were the same for brothers living in the same family in 1950, without having to measure them directly. In Stata software, this was implemented by a stratification option, allowing different families of origin to have different baseline hazard functions, while constraining the HRs to be the same across families.[31] Only families in which at least one brother died contributed to the estimation of HRs (n=1124). HRs were estimated using the variation in the timing of fatherhood within a family. Approximately 79% of brothers were discordant on the timing of fatherhood, at least with one of their siblings. Robust SEs were used to calculate the 95% CIs. The results from the Cox model with fixed effects were verified by using a conditional fixed-effects logistic model that produced similar results (results not shown).

RESULTS

Standard models

According to the standard Cox model, men who had a first child by age 22 had 26% higher mortality (HR=1.26, 95% CI 1.05 to 1.51), and men who had a first child at ages 22–24 had 14% higher mortality (HR=1.14, CI 0.97 to 1.34) than men who became first-time fathers at a median or mean age of 25–26, independent of adulthood characteristics and the year of birth (figure 1). The observed 14% excess among the fathers aged 22–24 years was not significant when compared with that of the reference group, although it was significant when compared with the mortality of the oldest fathers (results for CIs not shown). The oldest fathers who had a first child at ages 30–44 had 25% lower mortality (HR=0.75, CI 0.62 to 0.92) than men who fathered a first child at ages 25–26. The mortality of men who fathered a first child at ages 27–29 was the same as that of the reference group.

Sibling fixed-effects models

According to the model with sibling fixed effects, men who had a first child by the age of 22 had 73% higher mortality (HR=1.73, CI 1.18 to 2.54), and those who had a child at ages 22–24, a 63% higher mortality (HR=1.63, CI 1.16 to 2.30) than men who fathered a first child at the age of 25 or 26, independent of birth year, observed adulthood characteristics and unobserved early-life conditions shared by brothers (figure 2). Men who became first-time fathers at ages 30–44 had the lowest mortality (HR=0.78, CI 0.53 to 1.14), but it was not significantly different from the mortality of men who fathered a first child at the age of 25 or 26. The mortality of these oldest fathers was, however, significantly lower as compared with that of the youngest fathers who had a child by the age of 22 or at ages 22–24 (results for CIs not shown). The mortality of men who fathered a first child at ages 27–29 was not different from the reference group, although it seemed somewhat elevated.

DISCUSSION

We assessed the possibility that the association between fertility timing and mortality is spurious, in the sense that mortality and timing appear to be associated, because both are determined by family-related social and genetic characteristics. In a first study of its kind, it was shown that young fathers experienced higher midlife mortality than their brothers who became first-time fathers at a median or mean age. According to the fixed-effects sibling model, men who had had a first child by the age of 22 or at ages 22–24, had higher mortality at ages 45–54 as compared with their brothers who had a first child at the median or mean age of 25 or 26. The mortality of the oldest fathers was significantly lower as compared with that of their brothers who had had a child by the age of 24. These results from the fixed-effects sibling model confirm our results from the standard model suggesting a causal relationship between young fatherhood and mortality. Despite the differences in the size of the estimated HRs and their CIs, the overall pattern of results from both models is similar.

Our findings from the standard model correspond with the results of earlier studies suggesting an inverse relationship between the timing of a first child and later-life mortality. In a Norwegian register-based study, Grundy and Kravdal[1] showed that young men who had had a first child by the age of 23 had higher mortality later in life than men who fathered a first child at ages 23–28, independent of age, year, education, marital

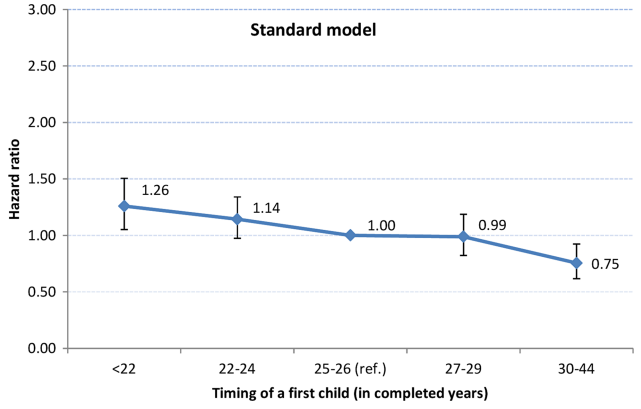

Figure 1 HRs and 95% CIs of mortality at ages 45–54 in relation to the timing of a first child (at ages 25–26: HR=1), standard model, men born in 1940–1950, Finland. Model controlled for own education, marital status, number of children, year of birth and region of residence.

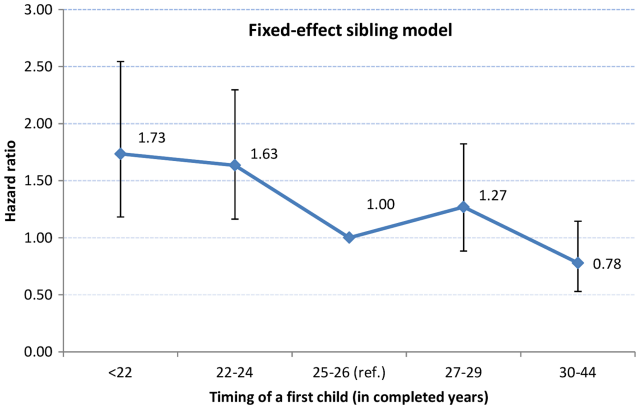

Figure 2 HRs and 95% CIs of mortality at ages 45–54 in relation to the timing of a first child (at ages 25–26: HR=1), fixed-effects sibling model, men born in 1940–1950, Finland. Model controlled for own education, marital status, number of children, year of birth, region of residence and unobserved early-life characteristics shared by brothers.

status, number of children and certain residential characteristics. Respectively, men who became first-time fathers after the age of 34 had the lowest mortality. Similarly, in a British study based on record linkage data, Grundy and Tomassini[32] showed that men in their long-term first marriages who had had their first child by the age of 23 had higher mortality than men who delayed fatherhood.

However, the results from our standard Cox model and those of earlier studies can be biased by early-life characteristics that affect both the timing of a first child and the risk of death. In a British study of men aged 30 years born in 1970, Sigle-Rushton[5] used a propensity score matching by a wide range of childhood characteristics to overcome the problem of selection, and compared young fathers who had had a first child by the age of 22 with men who had delayed fatherhood, or had no children. The study suggested that the selection into young fatherhood was substantial, but for some outcomes measuring subsequent disadvantage, such as malaise, for example, significant differences remained in disfavour of the young fathers. The study was, however, based on a survey of relatively young men as compared with our sample, and mortality was not among the outcomes studied. Furthermore, the reference group also included childless men, which may confound the comparison.

As far as we are aware, there are no previous mortality studies that use fixed-effects sibling models to study the effect of first-time fatherhood. However, our findings from the fixed-effects model are partly in line with those of Pudrovska and Carr,[33] who suggested that older age at first birth was positively associated with men's health in the USA, and this relationship was not explained by the confounding influences of unobserved early-life conditions shared by brothers. In their analysis, the effect of age at first birth on fathers' health was fully explained by men's socioeconomic and family statuses in adulthood. In our study, however, early timing of a first child carried a subsequent disadvantage on midlife survival that was independent of both unobserved early-life characteristics shared by brothers as well as observed adulthood characteristics. The differences between the studies can relate to differences in the health outcomes or other measures. Pudrovska and Carr[33] used an indicator of several chronic illnesses, including asthma, arthritis, thyroid disease, diabetes, hypertension, autoimmune disorders, heart conditions and ulcers, at ages 25–74 as their measurement of subsequent health disadvantage, while we used all-cause mortality in midlife. Furthermore, we used large register-based data that allowed sufficient statistical power even in the fixed-effects analyses.

The findings of our study suggest that the association between young fatherhood and midlife mortality is likely to be causal. The association was not explained by unobserved early-life characteristics shared by brothers or by certain adulthood characteristics known to be associated both with fertility timing and mortality. Although having a child early in life is thought to cause less disruption to educational and professional careers among men as compared with women, the impact on male mortality was strong. It is possible that fathering a child early in life caused accumulated psychological, social and economic stress in balancing the roles of a father, husband and breadwinner. Accumulated stress could have had more adverse consequences for young men not prepared for their new role. This idea is consistent with previous research documenting that young fathers have a declining pattern of subjective well-being even after the birth of their first child, whereas mature fathers experience an increase in well-being in the long run.[34]

In our study cohort, the link between fertility and marriage was still strong. According to a survey carried out in the 1970s, approximately 40–50% of ever-married women who got married by the age of 23 in 1955–1971 reported that they had been pregnant at the time of marriage.[35] It is thus likely that many young men experienced a normative pressure to marry their partner if a pregnancy occurred. It is possible that becoming the main breadwinner of a new family reduced young men's ability to invest in their own well-being. It remains unclear, however, whether our findings hold for recent cohorts of young fathers who may experience different kinds of stressors and responsibilities regarding their children than those in our study cohort. For example, current cohorts of young fathers are less likely to live with the mother of their child, but more likely to face expectations toward involvement in the home sphere if they decide to participate in parenting.

One of the main strengths of the study is the identification of male fertility. The birth of a child was systematically registered to the files of a father from the late 1960s onward owing to the introduction of personal identification codes. Children who were born earlier were included only if they lived with their father in the late 1960s. It is therefore possible that the proportion of young fathers is modestly underestimated in the sample. However, only 5% of children were born out of wedlock in the 1950s and 1960s, and approximately 10% of those who had married in 1960 ended in divorce within 10 years.[36 37] Furthermore, previous studies based on the 1950 census sample have shown that completed fertility rate is similar among men and women born during 1940–1950, and the bias attributable to unknown paternity is thus likely to be small.[23]

A register-based sample from the 1950 Finnish census carries several other advantages. Unlike many surveys, register-based data are representative of men across social strata, including those from more advantaged backgrounds, as well as those ending up in institutions. Personal identification codes allowed the linkage to fertility and mortality records, and to quinquennial censuses in 1970–1985, including demographic and educational measures in adulthood. Unfortunately, the marital status variable did not include information on cohabitation, and the cohabitees were thus classified with the never-married. However, an advantage over the previous mortality studies is that brothers living in a same household in 1950 could be identified. The identification of brothers allowed us to use the sibling design, which gave a unique possibility to verify that the association between the timing of a first child and mortality was not biased by unobserved early-life characteristics shared by brothers.

Despite the strengths of the sibling design, characteristics that brothers did not share in childhood may still have confounded the association.[21 38] Characteristics not entirely shared by brothers include psychological traits such as personality, which were not directly measured in our study. Of the five main personality traits (conscientiousness, extraversion, neuroticism, agreeableness and openness to experiences),[39] low conscientiousness, which reflects low persistence, poor self-control and lack of long-term planning, has been shown to be associated with mortality[40] and early pregnancies in women.[41] It has been suggested, however, that conscientiousness is unrelated to the age at which men conceive their first child.[41] In addition, despite the lack of direct measurement of personality, the brothers of our subsample are likely to be more similar to one another than a pair of boys selected randomly from a population. The intraclass correlation of conscientiousness for fraternal twins has

previously been reported to be around 0.10.[42] Furthermore, our subsample of brothers also included identical twins, although genetic links were impossible to identify.

CONCLUSIONS

In a register-based follow-up study of a large sample from the 1950 Finnish census, it was shown that young fatherhood was associated with an elevated mortality in midlife. The study was unique in that we were able to show that mortality was influenced by young fatherhood, independent of unobserved environmental and genetic characteristics shared by brothers in childhood and of observed demographic and educational characteristics in adulthood. The findings of our study provide evidence of a need to support young fathers struggling with the demands of family life in order to promote good health behaviours and future health. The promotion of good health behaviours in young fathers could also support healthy behaviours in their children. More research is needed that considers the processes leading to the excess mortality of young fathers at later stages in life.

What is already known on this subject

- Young fatherhood has shown associations with higher later-life mortality.
- It is unclear whether the effect of young fatherhood on mortality is causal, or related to early-life conditions.

What this study adds

- This study is the first to consider the association of young fatherhood and mortality, independent of the influences of unobserved early-life characteristics shared by brothers and observed characteristics in adulthood.
- This study uses a register-based sample from the 1950 Finnish census allowing the identification of brothers and their fertility histories.
- The findings suggest a causal effect of young fatherhood on midlife mortality.

Contributors EE planned the study, performed the statistical analyses and wrote the paper. JN contributed to collecting the literature and revising the paper. PM contributed to revising the paper.

Competing interests None declared.

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Data sharing statement Census 1950 longitudinal data file (version TK-53-704-10) can be accessed on-site at the Population Research Unit, University of Helsinki until 2018, by agreeing to confidential conditions and restrictions of Statistics Finland. Data requests should be addressed to Statistics Finland.

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