Reproductive history and post-reproductive mortality: A sibling comparison analysis using Swedish register data

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ABSTRACT

A growing body of evidence suggests that reproductive history influences post-reproductive mortality. A potential explanation for this association is confounding by socioeconomic status in the family of origin, as socioeconomic status is related to both fertility behaviours and to long-term health. We examine the relationship between age at first birth, completed parity, and post-reproductive mortality and address the potential confounding role of family of origin. We use Swedish population register data for men and women born 1932—1960, and examine both all-cause and cause-specific mortality. The contributions of our study are the use of a sibling comparison design that minimizes residual confounding from shared family background characteristics and assessment of cause-specific mortality that can shed light on the mechanisms linking reproductive history to mortality. Our results were entirely consistent with previous research on this topic, with teenage first time parents having higher mortality, and the relationship between parity and mortality following a U-shaped pattern where childless men and women and those with five or more children had the highest mortality. These results indicate that selection into specific fertility behaviours based upon socioeconomic status and experiences within the family of origin does not explain the relationship between reproductive history and post-reproductive mortality. Additional analyses where we adjust for other lifecourse factors such as educational attainment, attained socio-economic status, and post-reproductive marital history do not change the results. Our results add an important new level of robustness to the findings on reproductive history and mortality by showing that the association is robust to confounding by factors shared by siblings. However it is still uncertain whether reproductive history causally influences health, or whether other confounding factors such as childhood health or risk-taking propensity could explain the association.

1. Introduction

In contemporary developed populations a growing body of evidence points to the influence of individual reproductive history on post-reproductive health and mortality. Previous studies have shown that an early age at first childbearing, childlessness, and having many children are associated with higher mortality (Grundy and Tomassini, 2005; Grundy and Kravdal, 2010). The association

between reproductive history and mortality is likely to be the result of multiple biological and social pathways, some of which may operate in opposing directions on health status, and may differ for men and women (Grundy and Read, 2015).

A potentially important dimension of the association may be confounding by early life socio-economic and health factors that increase both the chance of selection into specific reproductive patterns and later mortality. For example, those who become teenage parents are more likely to have grown up in socioeconomically disadvantaged households or non-intact families compared with non-teenage parents (Kiernan, 1997), and education is typically inversely related to both completed fertility (Nisen et al., 2014a), and mortality in adulthood (Torssander and Erikson,

2010).

Most previous studies on fertility and mortality have attempted to adjust for selection processes by controlling for parental education (Henretta, 2007), or the educational attainment of the index person (Doblhammer, 2000; Grundy and Kravdal, 2008; 2010). However, because detailed data on early life characteristics are often limited, and this is particularly true of the older cohorts that are used in mortality studies, there remains a risk of omitting important selection factors such as early life socio-economic status or family disruption that could produce biased estimates for the relationship between reproductive history and post-reproductive mortality.

We use Swedish population register data to examine the relationship between age at first birth, completed parity, and post-reproductive mortality for Swedish men and women, our index persons, born 1932–1960 over ages 40 to 80. We apply a sibling comparison design to compare mortality amongst siblings who grew up in the same family. Sibling comparisons have been used to investigate the socioeconomic consequences of fertility patterns (Geronimus and Korenman, 1993; Hoffman et al., 1993), but so far only one study has examined the relationship between reproductive history and health outcomes (Einiö et al., 2015). This recent study using Finnish register data found that early fatherhood is associated with increased mortality. However, the study focused only on men, who do not go through the physiological process of childbearing, and mortality at ages 45–54.

1.1. Early parenthood and mortality

Previous research has consistently found that women who experience early motherhood, usually defined as giving birth before the age of 20, have excess morbidity and mortality in mid- and later-life (Doblhammer, 2000; Grundy and Tomassini, 2005; Henretta, 2007; Grundy and Kravdal, 2008, 2010; Spence and Eberstein, 2009; Grundy and Kravdal, 2010; Read et al., 2011). The associations are similar for men (Grundy and Kravdal, 2010; Read et al., 2011; Einiö et al., 2015).

There are a number of potential explanations for the association between early age at first childbearing and post-reproductive mortality. Social mechanisms include the interruption of educational and labour market trajectories (Hobcraft and Kiernan, 2001; Kane et al., 2013), and a higher risk of single parenthood and partnership disruption (Hobcraft and Kiernan, 2001), which are associated with worse health (Berkman et al., 2015; Huisman et al., 2003). Socioeconomic selection may also explain part of the association. Teenage parents are more likely to come from deprived backgrounds, disrupted families, and have a lower education level (Kiernan, 1992; Imamura et al., 2007; Raymo et al., 2015), and these factors are associated with worse health in later life (Ploubidis et al., 2014). Men who become young fathers are also more likely to have adolescent educational or behavioural problems (Sigle-Rushton, 2005; Lehti et al., 2012).

It appears that these social mechanisms are particularly harmful to health, as physiological mechanisms actually suggest a protective effect against some health risks. Early pregnancy, childbirth, and breastfeeding are linked to lower risk of breast cancer (Grundy and Kravdal, 2010). The mechanism linking reproductive behaviours to breast cancer in women concerns exposure to estrogen and progesterone, which are produced by a woman's ovaries. These ovarian hormones stimulate cell growth, including the growth of cancerous tissues (Kelsey et al., 1993). Pregnancy and breastfeeding both reduce a woman's lifetime number of menstrual cycles, and thus her cumulative exposure to these ovarian hormones. Pregnancy and breastfeeding also have a direct effect on breast cells, causing them to differentiate, or mature so as to produce milk,

which may reduce the risk of those cells transforming into cancer cells (Russo et al., 2005). Although the same hormonal mechanism also influences the risk of uterine and ovarian cancer, the empirical evidence for the relationship between age at first birth and these cancers is mixed (Merrill et al., 2005; Grundy and Kravdal, 2010). A younger age at first birth has also been associated with cervical cancer (Grundy and Kravdal, 2010), with the mechanism thought to be related to sexual behaviour, a higher number of partners, and increased risk of exposure to human papillomavirus (Merrill et al., 2005).

1.2. Parity and mortality

Most studies on the relationship between completed parity and post-reproductive mortality in contemporary populations find a J-shaped or U-shaped relationship, where childlessness, having only one child, and high parities are associated with higher mortality (Doblhammer, 2000; Hurt et al., 2006; Read et al., 2011). However the association for high parity parents varies somewhat between studies, possibly due to contextual or methodological differences (Hurt et al., 2006; Grundy, 2009; Spence and Eberstein, 2009; Hank, 2010). For example, a study using Swedish register data found a small increase in mortality hazard for parents of 6 or 7 children compared to 2 children (Barclay and Kolk, 2015a), while similar studies using Norwegian register data found no evidence for such association (Grundy and Kravdal, 2008; 2010).

Explanations for the parity–mortality association include both physiological and social mechanisms. The disposable soma theory suggests a trade-off between reproduction and longevity (Westendorp and Kirkwood, 1998). The maternal depletion hypothesis also argues for a trade-off between reproduction and longevity primarily through nutritional deficiencies (Winkvist et al., 1992). Repeat childbearing may also have protective physiological effects due to decreased exposure to progesterone and estrogen, which lowers the risk of breast, uterine and ovarian cancer (Merrill et al., 2005) though this could be offset by permanent deficiencies in glucose metabolism, the cardiovascular system, and fat distribution (Lawlor et al., 2003; Lassek and Gaulin, 2006).

Since the relationship between parity and mortality is similar for women and men (Grundy and Kravdal, 2008), social mechanisms are likely to play a substantial role in this relationship. Short birth intervals are associated with higher mortality for both men and women (Grundy and Kravdal, 2014), suggesting that the emotional, psychological and social strains of raising multiple children plays a role in that association. On the other hand children could benefit parents by providing social and emotional support throughout the life course (Grundy and Read, 2012). Nulliparity, low parity (and in men, high parity) are associated with a higher risk of death from diseases stemming from poor health behaviours, including alcohol-related disease, circulatory disease and accidents and death, which could reflect both selection and an absence of social control of health related behaviours from close family members (Grundy and Kravdal, 2010). Childlessness may also be a consequence of an underlying health problem, or social or psychological factors that reduce the likelihood of finding a partner (Kiernan, 1989).

As with the relationship between age at first birth and mortality, the relationship between parity and mortality could be driven by selection mechanisms related to socioeconomic status in the family of origin. For example, childlessness in Sweden, as well as other European countries, is more common amongst women with higher educational attainment (Hoem et al., 2006; Neyer and Hoem, 2009), though the opposite is true for men (Nisen et al., 2014b).

1.3. Contribution

In this study we employ a within-family sibling comparison design to try and minimize confounding from shared family background characteristics that influence both fertility behaviours as well as mortality, and consider both women and men. Sibling fixed effects adjust for all factors that are shared by siblings, such as parental educational level, parental social class, religious belief in the household, the final size of the sibling group, as well as other factors that may be difficult to observe, such as parenting style. Previous applications of sibling comparisons in epidemiology and demography have been useful for showing that associations in earlier work have been spurious or overstated; these include the association between breastfeeding and subsequent child health in the US (Colen and Ramey, 2014), and teenage childbearing and educational attainment (Geronimus and Korenman, 1993; Hoffman et al., 1993).

Sibling comparisons may be particularly valuable for studying the relationship between reproductive history and post-reproductive mortality as fertility behaviours are related to both family socioeconomic status as well as mortality in adulthood. Furthermore, the nature of research on mortality requires data from cohorts born decades earlier, long before the widespread and reliable collection of data on family socioeconomic status, meaning that the findings of previous research on this topic could be biased.

Another important contribution of this study is our assessment of cause-specific mortality patterns, which can help to shed light on the mechanisms for the association between reproductive history and mortality. The association between reproductive history and cause-specific mortality has been studied using Norwegian data (Grundy and Kravdal, 2010), but that study was only able to follow participants to a maximum age of 68 years, which in a low mortality population like Norway means that only a small fraction of the population had died. Our follow up period includes individuals to a maximum of age 80.

2. Data and methods

2.1. Data

We used Swedish administrative register data for birth cohorts 1932–1960. For the analyses of age at first birth and mortality the analytical sample size is 59,436 for women, and 78,296 for men. For the analyses of completed parity and mortality the analytical sample size is 79,162 for women, and 120,297 for men. Details on how we reach our analytical sample are shown in Table 1. We use the Swedish multigenerational register to link the index persons to their children. This allows calculation of age at first birth and completed parity. The Swedish multigenerational register also enables us to link the index person to their parents, so we can link the index person to their siblings. We define siblings as those who share a biological mother and father. Our mortality follow-up covers the years 1990–2012 as the Swedish multigenerational register was incomplete before the 1990s.

The within-siblings design identifies the associations from variation between siblings. Therefore our sample may be drawn from a healthier section of the population, because at least two siblings need to survive to 1990. Furthermore, there may be a small amount of measurement error for completed parity, as the offspring have to survive to 1990 as well. Given that early adulthood mortality is very low in Sweden (for example, 86% of women and 80% of men of the 1932 birth cohort survived to age 60), we expect this bias to be small.

We analyse all-cause mortality, leading causes of death, and causes of death for which prior research suggests that reproductive history is important (Grundy and Kravdal, 2010). For men we examine mortality attributable to neoplasms, diseases of the circulatory system, external causes, and all remaining other causes. Mortality attributable to external causes includes accidents, suicides, and events of undetermined intent. For women we examine mortality attributable to neoplasms, diseases of the circulatory system, external causes, all remaining other causes, as well as mortality attributable to breast cancer, cervical cancer, uterus cancer, and ovarian cancer. For the analyses of women we have removed cancers of the breast, cervix, uterus and ovaries from the main neoplasms category.

Our mortality follow-up is from age 45 until death, the end of the study period in 2012, or until censoring due to out-migration. Our analysis population consists of individuals who come from siblings groups with at least two children, as there is no variance within a one-child sibling group. We also exclude sibling groups where none of the siblings have died, as variance on the outcome variable, mortality, is needed to produce the within-family estimates. Furthermore, we exclude sibling groups where all the members of that group have the same value for the explanatory variables, meaning age at first birth and completed parity, as there is no variation within the sibling group.

In further analyses we take account of factors that might mediate, moderate or confound the associations by fitting models including highest educational attainment, attained socioeconomic status, and post-reproductive marital status. Attained socioeconomic status is classified according to the Erikson, Goldthorpe, Portocarero class schema (Erikson et al., 1979). Marital status is taken from the civil status register, which covers the period

Table 1
Sample exclusion process.

	Exclusion criteria	Men		Women	
		N	N excluded	N	N excluded
AFB	Total in Swedish registers 1932–1960	1,932,220		1,816,574	
	ID for both parents	1,299,549	632,671	1,248,786	567,788
	No multiple births	1,275,989	23,560	1,224,996	23,790
	No only children	1,045,756	230,233	1,001,171	223,825
	No childless parents	836,577	209,179	861,685	139,486
	No variance on either mortality or AFB	78,296	758,281	59,436	802,249
	Final	**78,296**		**59,436**	
Parity	Total in Swedish registers 1932–1960	1,932,220		1,816,574	
	ID for both parents	1,299,549	632,671	1,248,786	567,788
	No multiple births	1,275,989	23,560	1,224,996	23,790
	No only children	1,045,756	230,233	1,001,171	223,825
	No variance on either mortality or parity	120,297	925,459	79,162	922,009
	Final	**120,297**		**79,162**	

1968–2012, and is included as a time-varying covariate (categories: unmarried, married, divorced, or widowed).

3. Methods

We use Cox proportional hazard regressions (Cox, 1972) stratified by the sibling group so that the baseline hazard is shared by the sibling group (Allison, 2009). We refer to the stratified model as a sibling fixed effect model.

The time scale is age. The follow-up period is between 1990 and 2012. Both men and women enter the analysis population in 1990, or at age 45 if they turn 45 after the year 1990. For the earliest cohort, born in 1932, we are able to follow them from age 58 to age 80, while for the latest cohort, born in 1960, we are able to follow them from age 45 to age 52. Individuals are censored if they emigrate from Sweden. In our analyses of cause-specific mortality, individuals are censored if they die from a cause other than the one under examination.

To study age at first birth and parity we use two different populations, with the age at first birth analyses based upon all parous women and men, and the completed parity analyses based upon all women and men, including those who are childless. We also use a different set of control variables for the analyses of age at first birth and parity. In the analyses of age at first birth we include a variable for completed parity, while in the analyses of parity we do not include a variable for age at first birth, as we want to include childless women and men in the analysis.

For each of our analyses of mortality we run four different models, the first two using a regular Cox proportional hazard models, which we refer to later as between-family comparisons, and the third and fourth using sibling fixed effects to estimate the hazards of mortality based upon a within-family comparison.

In Model 1 to examine the relationship between age at first birth and mortality we use a regular Cox model and adjust only for birth cohort. In Model 2 we adjust for parity as well as an observed measure of parental occupational class taken from the 1960 Swedish census. The purpose of this is to estimate a model that is comparable to that estimated in previous studies that have used a measure of parental socioeconomic status to control for background SES. In Model 3, the first sibling fixed effect model, we adjust for birth cohort, completed parity, as well as the age of the index person's mother at the time of their own birth. In Model 4, the second sibling fixed effect model, we adjust for birth cohort, completed parity, the age of the index person's mother at the time of their own birth, as this may be associated with mortality risks of offspring (Myrskylä and Fenelon, 2012) and there are known intergenerational continuities in fertility patterns in Sweden (Kolk, 2014). We also adjust for several variables that may mediate, moderate, or confound the association between age at first birth, parity, and mortality, which are attained socioeconomic status, educational attainment, and a time-varying covariate for post-reproductive marital status. The analyses for the relationship between parity and mortality are exactly the same, except they do not include a variable for age at first birth.

4. Results

4.1. Descriptives

Table 2 describes the analytical sample. In the analytical sample used to study age at first birth the highest mortality rate is found amongst teenage parents, while those aged 25 or older have similar rates of mortality. In the analytical sample used to study completed parity, mortality is highest amongst childless men and women, but follows a U-shaped pattern where men and women with none, one,

or four or more children have higher mortality than those with two children. In both analytical samples mortality rates are highest amongst those who attain the lowest levels of education and socioeconomic status. Mortality is highest for those who are widowed, and lowest for those who are married. Further descriptive information on the analytical sample can be found in the Supplementary Information, in Tables S1 and S2.

4.2. Age at first birth

4.2.1. Women
4.2.1.1. All-cause and cause-specific mortality. Women who had a first birth in the teenage years have the highest all-cause mortality, followed by women aged 20–24, and these differences are statistically significant in the regular Cox models as well as the sibling comparison model (Table 3). Women who had a first birth in their 30s have very similar all-cause mortality to women aged 25 to 29. A full results table, including the control variables, can be found in the Supplementary Table S3.

The cause-specific mortality results can be seen in Fig. 1 (regression coefficients shown in Supplementary Table S7). Teenage first-time mothers had significantly higher mortality from neoplasms than women aged 25 to 29, and this is consistent across the between-family comparison and within-family comparison models. Mortality attributable to diseases of the circulatory system is elevated among women who had a first birth below age 25. Mortality attributable to diseases of the circulatory system is also elevated for first time mothers aged 35 or older in the between-family comparison and in the sibling comparison model with mediators, but the difference is not statistically significant.

Teenage mothers also have elevated external cause mortality, and these differences were statistically significant (Fig. 1). The remaining category, mortality attributable to all other causes, also shows that younger first time mothers have significantly higher mortality, and this is consistent across models 1 to 4.

Fig. 2 shows the results for age at first birth and mortality attributable to cancers of the breast, cervix, uterus, and ovaries (detailed results in Table S8). The results for breast cancer show that women who have a first birth at ages 30–34 or 35+ have much higher mortality than women aged 25–29 at time of first birth, and this pattern is even stronger in the sibling comparison models than in the regular Cox models. The analysis for cancers of the cervix, uterus and ovaries is underpowered.

4.2.2. Men
4.2.2.1. All-cause and cause-specific mortality. Age at first birth is also related to all-cause mortality for men, with men who become fathers in the teenage years or at 20–24 having significantly elevated mortality compared to first time fathers aged 25–29, while older first time fathers have lower mortality (Table 3). The results from the cause-specific mortality analyses can be seen in Fig. 3 (details in Tables S4 and S9). The results are largely similar to those shown for women. Mortality attributable to neoplasms and diseases of the circulatory system show that men who become fathers in the teenage years or at 20–24 have elevated mortality compared to first time fathers aged 25–29 or older. Mortality attributable to external causes or other causes is lower among older fathers. Overall the results from the regular Cox models and the sibling comparison models are very similar.

4.3. Parity

4.3.1. Women
4.3.1.1. All-cause and cause-specific mortality. Compared to women with two children, childless women have the highest all-cause

Table 2
Descriptive statistics for analytical sample.

		Age at first birth analytical sample				Parity analytical sample			
		Men		Women		Men		Women	
		Deaths (%)	Rate (10^{-3})	Deaths (%)	Rate (10^{-3})	Deaths (%)	Rate (10^{-3})	Deaths (%)	Rate (10^{-3})
Age at first birth	<20	6.1	2.5	23.5	2.1				
	20—24	35.4	2.1	40.1	1.9				
	25—29	34.0	1.9	24.7	1.8				
	30—34	16.7	1.9	8.7	1.9				
	>34	7.9	1.9	3.0	1.9				
Parity	0					24.9	3.0	17.1	3.0
	1	21.1	2.3	21.0	2.2	16.9	2.3	18.9	2.3
	2	42.4	1.9	43.5	1.8	27.8	1.8	30.9	1.7
	3	23.2	1.9	23.2	1.8	18.8	1.9	20.6	1.8
	4	8.7	2.1	8.2	2.0	7.6	2.2	8.3	2.0
	5+	4.6	2.3	4.1	2.2	4.0	2.4	4.2	2.3
Cohort	1932—1934	11.9	3.1	11.7	2.7	11.2	3.3	11.5	2.8
	1935—1939	27.7	2.4	26.6	2.2	26.4	2.6	26.1	2.3
	1940—1944	27.4	1.9	27.6	1.8	26.6	2.0	26.9	1.9
	1945—1949	19.9	1.6	20.7	1.6	20.8	1.8	20.7	1.7
	1950—1960	13.1	1.7	13.5	1.7	15.1	2.0	14.9	2.0
Education	Primary (<9 years)	36.8	2.2	34.1	2.1	37.9	2.4	33.9	2.2
	Primary (9 years)	9.8	2.0	12.4	2.0	10.9	2.2	12.8	2.2
	Secondary (10—11 years)	26.1	2.0	34.3	1.8	25.5	2.2	32.8	1.9
	Secondary (12 years)	12.7	1.9	4.7	1.9	11.6	2.0	4.7	2.0
	Tertiary (13—15 years)	6.4	1.8	7.2	1.7	5.7	1.9	7.2	1.8
	Tertiary (15+ years)	7.0	1.8	7.0	1.6	6.6	1.9	7.4	1.7
	Post-graduate	0.5	1.6	0.1	1.3	0.5	1.7	0.2	1.9
	Missing	0.7	3.6	0.3	4.3	1.2	4.1	1.0	4.7
EGP	I	6.3	1.7	2.9	1.8	5.8	1.8	3.1	1.9
	II	15.9	1.9	15.8	1.9	13.7	2.0	15.2	2.0
	III	8.2	2.0	12.7	1.8	7.5	2.0	13.4	2.0
	IV	6.2	1.7	4.5	1.9	5.3	1.7	4.1	1.9
	VI—VII	52.7	2.0	42.9	2.0	53.0	2.2	41.2	2.0
	Unknown	10.6	2.8	21.2	1.9	14.7	3.3	23.1	2.2
Civil status	Unmarried	39.0	2.1	39.9	2.1	48.5	2.5	44.4	2.3
	Married	28.1	1.4	23.0	1.2	23.5	1.4	21.4	1.3
	Divorced	28.2	2.8	24.3	2.3	23.7	2.8	22.0	2.3
	Widowed	4.7	3.7	12.9	3.1	4.2	3.9	12.1	3.2

Note: EGP refers to the Erikson, Goldthorpe, Portocarero social class schema.

mortality, followed by women with one child, and this is true across models 1 to 4 (Table 3 and Figs. 4—5; details in Tables S6 and S10—S11). In Models 1 and 2, the between-family comparisons, women with four, or five or more children, also have higher mortality. In the sibling comparison analyses, mothers with two, three or four children have similar mortality, though mothers with five or more children have significantly elevated mortality.

The results for neoplasms and diseases of the circulatory system are similar to the all-cause mortality results. The results for mortality attributable to external causes show that mortality is only significantly elevated for childless women and mothers with one child, and this is consistent across the four models. In the between-family comparison models and the sibling comparison model without mediators, the point estimates show that mortality is higher for mothers with four or more children, though those differences are not significant. The results for mortality attributable to other causes also show that childless women and mothers with one child have higher mortality. In the sibling comparison analyses women with two or more children have similar mortality, but in the between-family comparisons women with four or more children have significantly elevated mortality.

Fig. 5 shows the results for women for cancers of the breast, cervix, uterus, and ovaries. Childless women have much higher mortality from breast cancer, and the estimates are similar across the sibling comparison models and the regular Cox models. Mothers with one child may also have elevated mortality. Overall, higher parity women have lower risk of mortality from breast cancer. The point estimates for cervical cancer in the between-family comparison and sibling comparison models are quite similar, with a U-shaped relationship between parity and mortality where women with two children have the lowest mortality. Although the pattern is statistically significant in the between-family comparison models, it is not significant in the sibling comparison models. The results for cancers of the uterus and ovaries show that childless women have the highest mortality from these cancers, while higher parity women have lower mortality. These differences are statistically significant for ovarian cancer, but not for cancers of the uterus.

4.3.2. Men
4.3.2.1. All-cause and cause-specific mortality. Childless men have the highest all-cause mortality in the between-family comparison and sibling comparison analyses, followed by fathers with one child (Table 3; details in Tables S6 and S12). Fathers with four, or five or more, children also have significantly higher mortality. The results for neoplasms are similar, though the gradient of the U-shaped pattern is less pronounced, and in the sibling comparison models fathers with four children do not have significantly elevated mortality. The results for mortality attributable to diseases of the circulatory system are also very similar to the all-cause mortality results. The results from models examining mortality attributable to external causes also show that childless men have the highest mortality, followed by fathers with one child. Fathers with two or three children have the lowest mortality, while fathers with four or more children have higher mortality, and this is consistent across the three models. Finally, mortality attributable to all remaining

Table 3
Results: relationship between age at first birth, parity, and all-cause mortality for Swedish men and women born 1932−1960.

|			M1		M2		M3		M4	
			RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
Women	Age at first birth	<19	1.16	1.12−1.21	1.19	1.15−1.24	1.29	1.22−1.37	1.20	1.13−1.28
		20−24	1.04	1.00−1.07	1.06	1.03−1.10	1.15	1.10−1.20	1.09	1.04−1.14
		25−29	1.00		1.00		1.00		1.00	
		30−34	1.02	0.97−1.07	0.97	0.92−1.02	0.92	0.85−0.99	0.96	0.89−1.04
		>34	1.07	0.99−1.15	0.96	0.88−1.03	0.95	0.84−1.07	1.03	0.91−1.17
	Parity	1			1.24	1.20−1.29	1.32	1.25−1.40	1.27	1.20−1.34
		2			1.00		1.00		1.00	
		3			0.97	0.94−1.01	0.96	0.91−1.01	0.96	0.91−1.01
		4			1.01	0.96−1.06	0.98	0.91−1.06	0.97	0.89−1.05
		5+			1.05	0.98−1.12	1.07	0.96−1.20	1.03	0.92−1.15
	N		59,436		59,436		59,436		59,436	
	Deaths		23,510		23,510		23,510		23,510	
Men	Age at first birth	<19	1.42	1.35−1.49	1.40	1.33−1.47	1.41	1.30−1.53	1.29	1.19−1.40
		20−24	1.12	1.09−1.15	1.12	1.09−1.15	1.14	1.09−1.18	1.08	1.04−1.12
		25−29	1.00		1.00		1.00		1.00	
		30−34	1.00	0.97−1.04	0.98	0.94−1.01	0.92	0.88−0.97	0.96	0.91−1.00
		>34	1.04	1.00−1.09	0.96	0.92−1.01	0.92	0.86−0.98	0.96	0.89−1.03
	Parity	1			1.27	1.23−1.31	1.31	1.24−1.37	1.23	1.17−1.30
		2			1.00		1.00		1.00	
		3			0.98	0.95−1.00	0.99	0.95−1.04	0.99	0.94−1.03
		4			1.05	1.01−1.10	1.09	1.02−1.17	1.07	0.99−1.14
		5+			1.12	1.06−1.19	1.16	1.06−1.28	1.12	1.02−1.23
	N		78,296		78,296		78,296		78,296	
	Deaths		31,787		31,787		31,787		31,787	
Women	Parity	0	1.79	1.73−1.85	1.79	1.73−1.85	1.94	1.85−2.05	1.77	1.67−1.86
		1	1.33	1.29−1.37	1.32	1.28−1.37	1.29	1.24−1.35	1.27	1.21−1.33
		2	1.00		1.00		1.00		1.00	
		3	1.04	1.00−1.07	1.04	1.00−1.07	0.97	0.93−1.02	0.96	0.92−1.00
		4	1.13	1.09−1.18	1.14	1.09−1.19	1.02	0.96−1.09	0.99	0.92−1.05
		5+	1.26	1.19−1.34	1.27	1.20−1.34	1.19	1.09−1.30	1.12	1.02−1.22
	N		79,162		79,162		79,162		79,162	
	Deaths		32,707		32,707		32,707		32,707	
Men	Parity	0	1.79	1.74−1.83	1.80	1.76−1.84	1.96	1.89−2.04	1.71	1.64−1.78
		1	1.33	1.30−1.37	1.33	1.29−1.36	1.29	1.24−1.34	1.22	1.17−1.27
		2	1.00		1.00		1.00		1.00	
		3	1.07	1.04−1.09	1.07	1.04−1.09	1.05	1.02−1.09	1.04	1.00−1.08
		4	1.19	1.14−1.23	1.19	1.15−1.23	1.16	1.10−1.23	1.12	1.06−1.19
		5+	1.29	1.24−1.36	1.29	1.23−1.35	1.21	1.13−1.30	1.14	1.06−1.23
	N		120,297		120,297		120,297		120,297	
	Deaths		52,034		52,034		52,034		52,034	

causes is also significantly elevated for childless men and fathers with one child, and for fathers with three or four children. Fig. 6.

4.3.2.2. Robustness checks. In addition to the models presented here, we have also examined the patterns of mortality with between-family comparisons using the full population data. Those results are extremely similar to the results obtained from the between-family comparisons using the analytical sample, and those results are available upon request. We have also run additional analyses where we adjust for the birth order of the men and women within their sibling groups of origin, since birth order is related to adult mortality (Barclay and Kolk, 2015b), and is directly correlated with maternal age amongst siblings. Those results are fully consistent with the results presented above. We focus on the models where we do not adjust for birth order since we can only adjust for birth order when analysing a more narrow range of cohorts due to the way that Swedish multigenerational register is constructed. Since the results are so similar, we find that the increased power from analysing a wider range of cohorts is the more favourable alternative.

We have also conducted additional analyses using cohorts born between 1945 and 1960 to check the robustness of our results. The results (shown in Tables S13 and S18) are consistent with those presented here.

5. Discussion

The results from this study using sibling comparison models to examine how fertility behaviours are related to post-reproductive mortality consistently corroborate the results from previous research on this topic not using a sibling comparison approach (Grundy and Kravdal, 2010). This finding is notable given the fact that some studies using sibling comparison models have shown very substantial differences from models comparing individuals across different families. For example, research examining how teenage childbearing is related to educational attainment has found that when comparing sisters, teenage childbearing itself is not actually that detrimental for subsequent educational achievement; it is the disadvantaged backgrounds that teenage mothers are disproportionately drawn from that explains the lower educational attainment (Geronimus and Korenman, 1993). The results of this study show that the previous finding that teenage childbearing is related to increased mortality is not simply a consequence of confounding by disadvantaged socioeconomic background or other family disruptions that are associated with an increased risk of young childbearing. Furthermore, additional analyses where we adjust for other lifecourse factors such as educational attainment, attained socioeconomic status, and post-reproductive marital history indicate that it is not simply the detrimental impact of teenage childbearing on subsequent socioeconomic or relationship
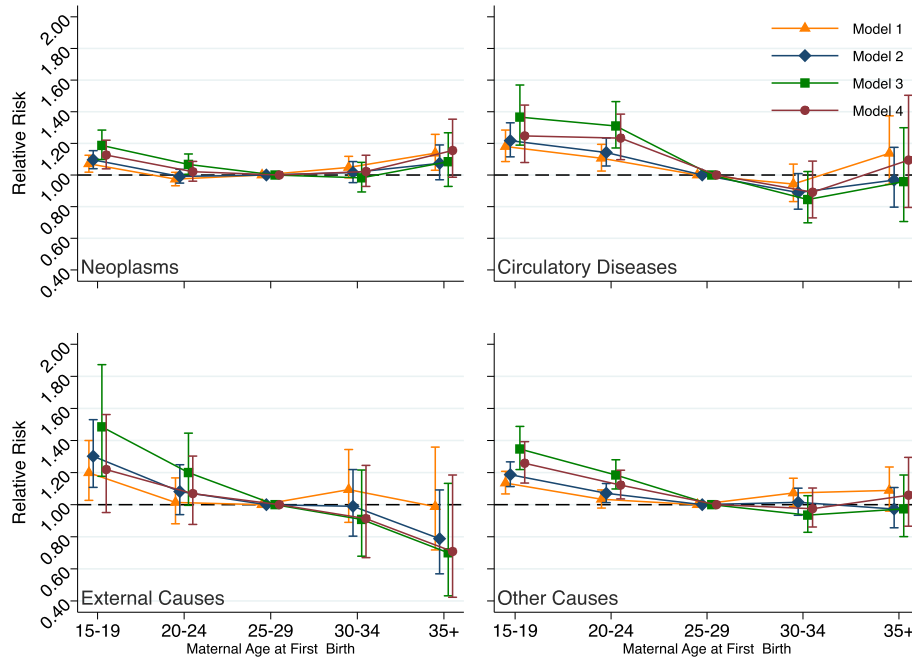
Fig. 1. Relationship between Age at First Birth and Post-reproductive Mortality Attributable to Neoplasms, Diseases of the Circulatory System, External Causes, and Other Causes, for Swedish Women Born 1932—1960. Error Bars are 95% Confidence Intervals.
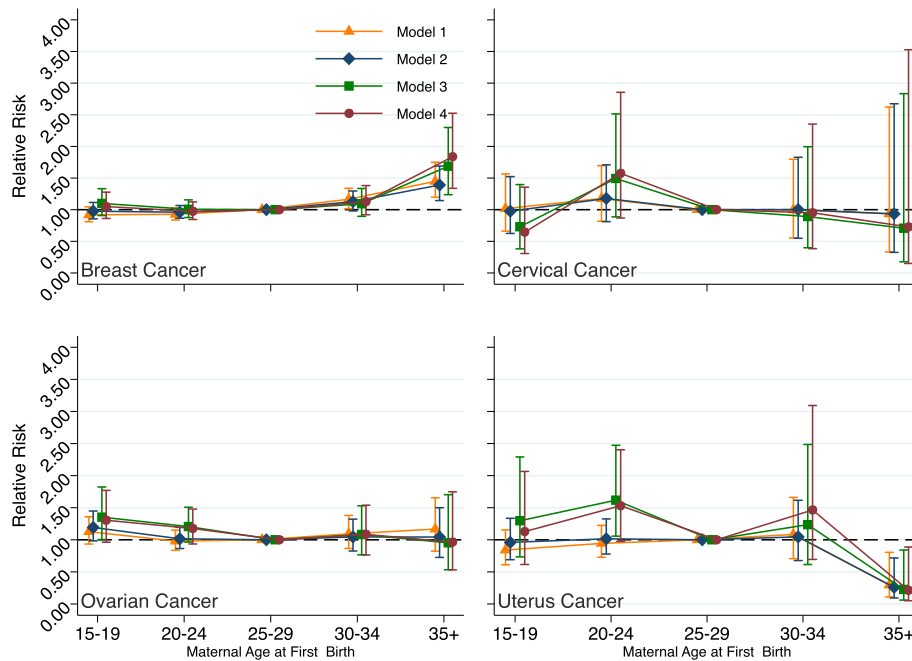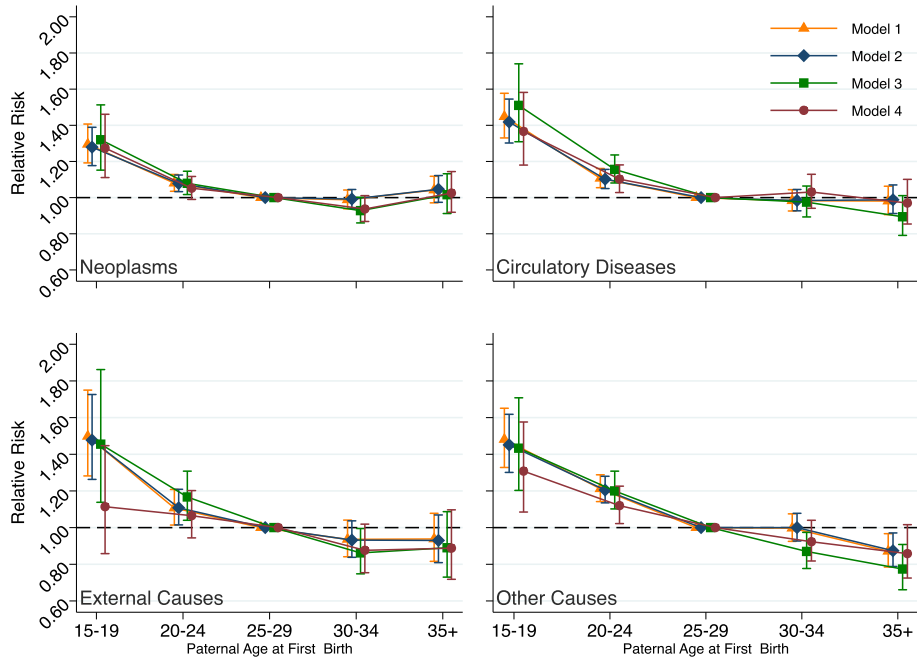

Fig. 2. Relationship between Age at First Birth and Post-reproductive Mortality Attributable to Breast Cancer, Cervical Cancer, Ovarian Cancer, and Uterus Cancer, for Swedish Women Born 1932—1960. Error Bars are 95% Confidence Intervals.

trajectories that is responsible for the higher mortality of young first time mothers.

Similarly to the results for age at first birth, we find that the relationship between parity and mortality is not driven by shared factors in the family of origin such as socioeconomic status. In the sibling comparison models we find the U-shaped relationship found in other studies: childless women and men have much higher mortality than mothers and fathers with two children, while mothers and fathers with one or five or more children also have

substantially elevated mortality. Although socioeconomic status is related to the probability of childlessness as well as having very many children, we again find that this is not the primary factor driving the mortality differences between these groups. A further notable result is that the relationship between age at first birth and mortality from breast cancer shows a stronger relationship in the sibling comparison models than in the between-family comparison models. Women who give birth for the first time at ages 30 or older have the highest mortality from breast cancer. Since women from

Fig. 3. Relationship between Age at First Birth and Post-reproductive Mortality Attributable to Neoplasms, Diseases of the Circulatory System, External Causes, and Other Causes, for Swedish Men Born 1932—1960. Error Bars are 95% Confidence Intervals.
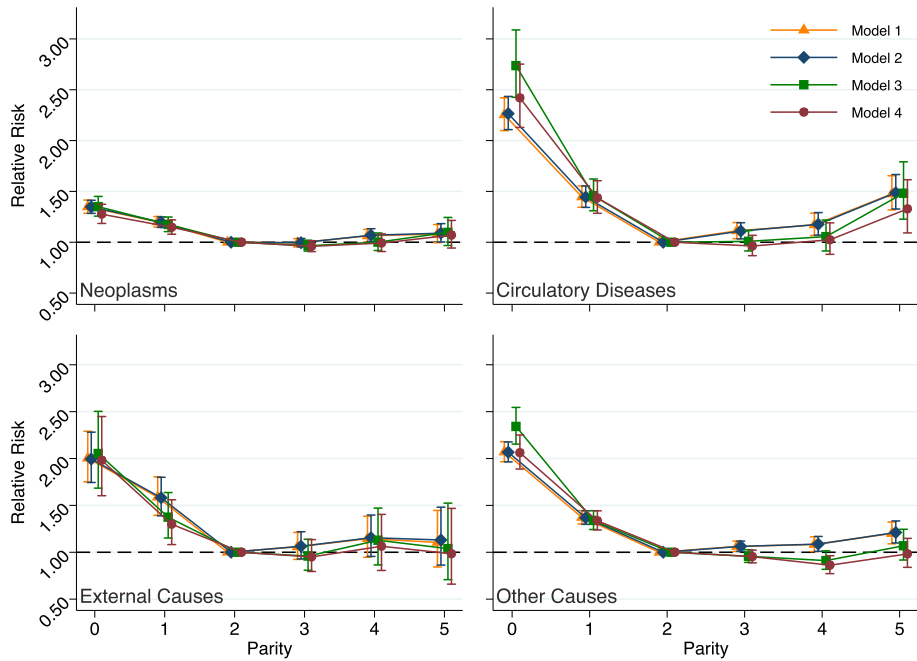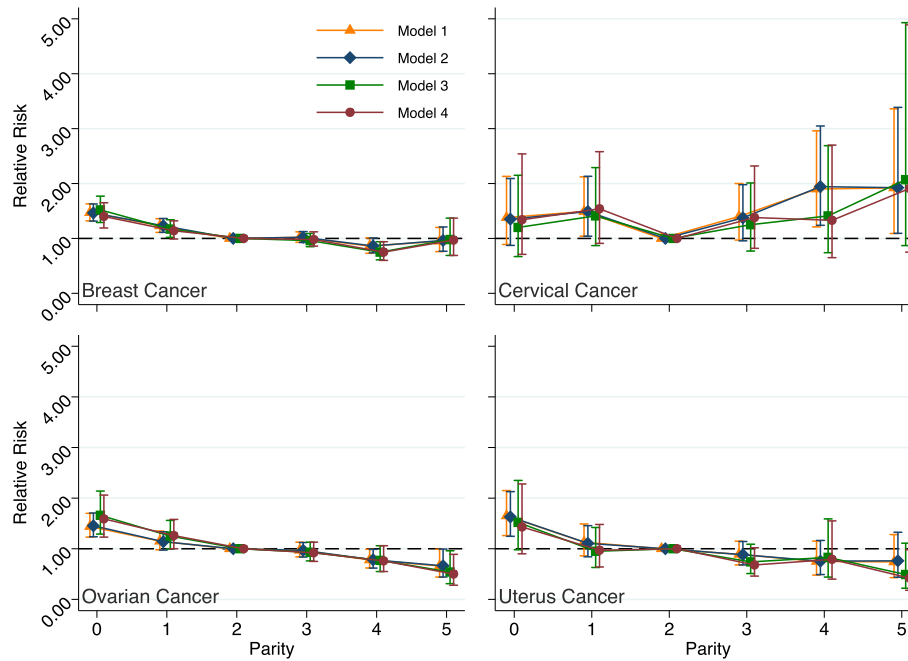

Fig. 4. Relationship between Completed Parity and Post-reproductive Mortality Attributable to Neoplasms, Diseases of the Circulatory System, External Causes, and Other Causes, for Swedish Women Born 1932—1960. Error Bars are 95% Confidence Intervals.

the highest socioeconomic status backgrounds are the most likely to delay childbearing to advanced ages, and the most likely to seek preventive medical care (Bradley et al., 2002), the results from these sibling comparison models suggest that previous research has been underestimating the impact of age at first birth on breast cancer mortality. After reducing confounding by background socioeconomic status, which was suppressing the relationship between age at first birth and mortality, we find a much stronger relationship between childbearing for the first time at advanced

ages and mortality from breast cancer.

Our finding that the relationship between age at first birth, and parity, and mortality is very similar whether you apply a sibling comparison approach or not suggests that the relationship between reproductive history and post-reproductive mortality is driven by factors within an individual's life course that are not shared by siblings. While these sibling comparison models do minimize residual confounding from background socioeconomic status and other factors shared within the family of origin, such as religious

Fig. 5. Relationship between Completed Parity and Post-reproductive Mortality Attributable to Breast Cancer, Cervical Cancer, Ovarian Cancer, and Uterus Cancer, for Swedish Women Born 1932—1960. Error Bars are 95% Confidence Intervals.
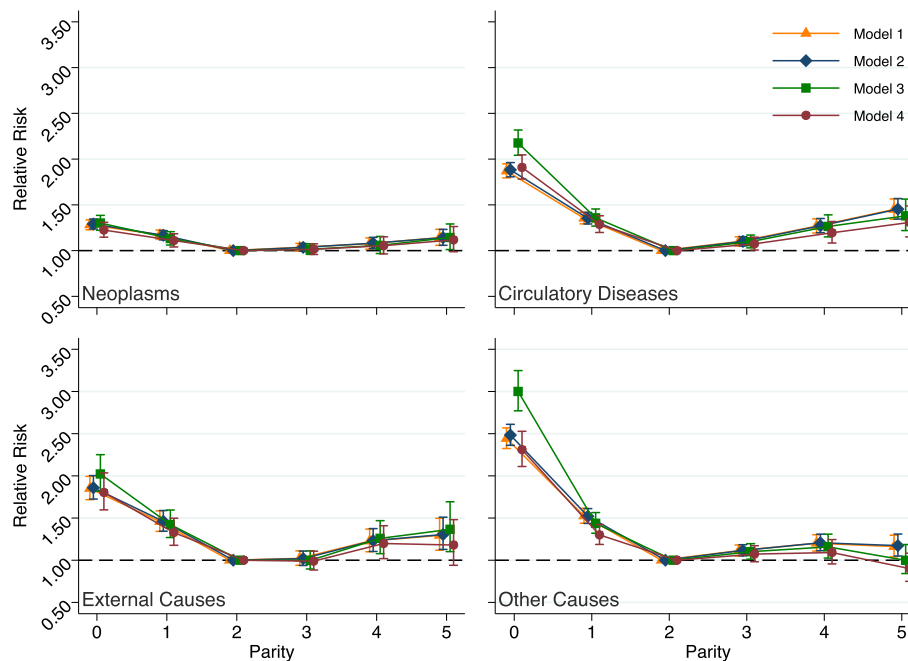
Fig. 6. Relationship between Completed Parity and Post-reproductive Mortality Attributable to Neoplasms, Diseases of the Circulatory System, External Causes, and Other Causes, for Swedish Men Born 1932—1960. Error Bars are 95% Confidence Intervals.

beliefs, there are other selection processes that could produce the pattern of results that we have observed in this study. The average pair of siblings is far from identical, and other differences between siblings that are not shared cannot be adjusted for simply by using a sibling comparison model. For example, siblings may differ in the propensity to engage in risky behaviours independent of any shared influence within the home environment that they occupy early in life, and this may affect the risk of teenage childbearing, or having children with multiple partners, as well has higher

mortality.

Our study is not without limitations. Although we are able to study our oldest cohort, those born in 1932, up to age 80, data limitations mean that we are only able to study members of our youngest cohort, those born in 1960, up to age 52. It is possible that the association between reproductive history and mortality, and particularly cause-specific mortality, may change over time, and could be different at the oldest ages. We also use data from Sweden, and the results may differ in other contexts. Furthermore, our

analyses are based on the portion of the population from the birth cohorts that we study that experienced at least one death in the sibling group in the study window, which is a minority of the total population. That selection procedure, necessary for the sibling fixed effects analyses, means that we focus on sibling groups that are on average less healthy than the general population. Nevertheless, robustness checks show that when using between-family comparisons the patterns of mortality that we have observed in our analytical sample are very similar in the full population. On the other hand, the size and the high quality of the data mean that we are able to use a study design that is far beyond the reach of most datasets, and also means that we have large numbers to study the association between parity and mortality for men and women with more than five children.

There is a tendency in the literature to ascribe a causal interpretation to results that persist after the application of a fixed effects model. Our study has only been able to minimize residual confounding from various factors, such as socioeconomic status, in the family of origin. Although we suspect that there is probably some causal effect of age at first birth and completed parity on post-reproductive mortality, we are hesitant to describe this relationship as causal, as various selection mechanisms may play an important role in producing the patterns shown in our results. Other potential sources of confounding could be related to personality, health, or even genetics. For example, mothers and fathers with five or more children both have higher mortality than mothers and fathers with two children. Men and women who have very many children may include subgroups of individuals who are on average more likely to engage in risky health behaviours. Likewise, childless men and women, or men and women who only have one child, may have health problems, such as infertility, or may have some other characteristic that makes it difficult for them to find a romantic partner. This means that there are likely to be confounding factors beyond shared family background that influence both fertility behaviours as well as adult mortality. Future research should explore the extent to which personality as well as underlying physical and mental health predict reproductive behaviours over the lifecourse as well as eventual mortality, which might be done by taking into account factors such as birth weight, hospital admissions, drug prescriptions, and relevant measures of personality. Alternatively, a stronger control for unobserved selection might be achieved by a twins-based study.

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Ethics approval

This study has not been required to obtain ethical approval by Statistics Sweden, the government administrative body overseeing the management of Sweden's administrative registers. The data used for this study was collected, prospectively, by various Swedish government bodies prior to the initiation of work on this manuscript. Researchers at Swedish institutions may apply to access this data for scientific research. The data that we have access to is securely stored, and is de-identified.

Researchers using this data are only allowed to export aggregated results from the secure storage location, meaning that no identification of individuals is possible from the results presented in this manuscript. Standard considerations for the ethical approval of a project state that if the dataset already exists, and no identification is possible, then no ethical approval is needed.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.socscimed.2016.02.043.

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