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Birth Order and Mortality: A Population-Based Cohort Study

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Abstract This study uses Swedish population register data to investigate the relationship between birth order and mortality at ages 30 to 69 for Swedish cohorts born between 1938 and 1960, using a within-family comparison. The main analyses are conducted with discrete-time survival analysis using a within-family comparison, and the estimates are adjusted for age, mother's age at the time of birth, and cohort. Focusing on sibships ranging in size from two to six, we find that mortality risk in adulthood increases with later birth order. The results show that the relative effect of birth order is greater among women than among men. This pattern is consistent for all the major causes of death but is particularly pronounced for mortality attributable to cancers of the respiratory system and to external causes. Further analyses in which we adjust for adult socioeconomic status and adult educational attainment suggest that social pathways only mediate the relationship between birth order and mortality risk in adulthood to a limited degree.

Keywords Birth order · Mortality · Fixed effects · Population register data · Sweden

Introduction

The relationship between birth order and outcomes in adulthood has been the subject of investigation in demography, sociology, psychology, and economics for many years. The past decade has seen growing attention to the importance of early-life and childhood conditions on adult outcomes, ranging from educational attainment and other measures of socioeconomic status (SES), through to health and disease. The evidence consistently demonstrates that social conditions within the family of origin

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have important consequences for adult health and mortality (Gluckman et al. 2008; Hayward and Gorman 2004). As we describe in more detail later, children of higher birth order, on average, have a lower IQ and lower educational attainment than their older siblings (Barclay 2015a; Black et al. 2005; Kristensen and Bjerkedal 2007). A number of theories have been developed to account for how birth order is related to later life outcomes, including the resource dilution hypothesis (Blake 1981), the confluence hypothesis (Zajonc and Markus 1975), and the hygiene hypothesis (Strachan 1989). Empirical research has shown that birth order is a marker of early-life relative resource deprivation (Buckles and Kolka 2014; Price 2008), and exerts an influence within families of all different financial resources (Bjerkedal et al. 2007; Myrskylä et al. 2013). As decades of research in demography and epidemiology has shown, adult health and mortality are often the path-dependent results of early-life disadvantage and cumulative resource deficiencies, with children who experience early resource deprivation, less cognitive stimulation, and a higher disease load performing less well than their more-advantaged peers or siblings (Bradley and Corwyn 2002; Knudsen et al. 2006; Willson et al. 2007). Abundant evidence suggests that early childhood investment is critical for long-term development, with randomized control trials showing that cognitive and health benefits attributable to early intervention can range from 0.2 to over 1 standard deviation (Campbell et al. 2014; Heckman 2006; Heckman et al. 2010). Although these relative advantages and disadvantages are typically studied between different families, studies on birth order show that relative advantage is equally important within families (Black et al. 2005; Härkönen 2014; Kristensen and Bjerkedal 2007).

However, despite this background, few studies have investigated the relationship between birth order and mortality in adulthood (Modin 2002; O'Leary et al. 1996; Smith et al. 2009), and very little research addresses birth order and cause-specific mortality in adulthood. A number of studies have demonstrated a link between birth order and cancer development, although not mortality attributable to cancer (Altieri and Hemminki 2007; Amirian et al. 2010; Bevier et al. 2011; Hemminki and Mutanen 2001; Richiardi et al. 2004). The overall pattern is mixed: the direction of the relationship between birth order and cancer development has been shown to vary according to the site of the cancer. This study is the first to address the relationship between birth order and all-cause mortality using a population data set, the first to use a within-family comparison design to study the relationship between mortality and birth order, and the first to use a sufficiently large database to address cause-specific mortality in adulthood. Using a within-family comparison approach—meaning that we only compare siblings born to the same parents to one another—allows us to rule out a wide range of potential confounding factors that may vary considerably between families, such as parental SES, as well as other unobserved family-specific characteristics. The specific causes of death that we study are mortality attributable to diseases of the circulatory system, external causes, and neoplasms, excluding cancers of the respiratory system, which we study separately. These cause-specific analyses are valuable both in terms of enabling us to partially discriminate between different causal mechanisms, and in allowing us to speculate about the importance of birth order effects on mortality beyond the ages that we observe in our data.

This study focuses on adult mortality at ages 30 to 69. Given that the life expectancy at birth in Sweden as of this writing is 79.7 for men and 83.8 for women (United

Nations 2013), we are studying premature mortality. Although premature mortality is relatively rare in Western developed societies such as Sweden, it remains a critical topic of interest given that premature mortality terminates the opportunity for individuals to enjoy long, satisfying lives and to make an economic contribution toward society. Premature mortality in adulthood is usually concentrated among the most vulnerable in society because those who suffer from premature mortality are typically more frail than the average individual. In this study, we choose to study the relationship between birth order and mortality separately for men and women, for several reasons. First, mortality patterns differ by gender. Second, previous research has also shown gender differences in the relative impact of early-life conditions, such as early socioeconomic disadvantage, on later-life mortality, with women being relatively more vulnerable (Hamil-Luker and O'Rand 2007). Read and Gorman (2010) suggested that the long-term chains of risk associated with early relative disadvantage may be greater among women than men because of the institutional disadvantages that women face in the household and the labor market. In this study, we will be able to examine whether relative disadvantage within the family produces a similar pattern. One reason why women may be more sensitive to relative disadvantage within the family is that previous research indicates that women demonstrate a greater tendency to root their social role within the family and private sphere than men (Hagestad 1986; Rossi and Rossi 1990; Young and Willmott 1957).

Previous studies on the relationship between birth order and mortality in adulthood have been mixed, with some finding that children of higher birth order have greater mortality (Modin 2002) and others finding no clear pattern of substantive or statistical significance (O'Leary et al. 1996; Smith et al. 2009). However, these studies have varied in quality as well as in the degree to which they focused on birth order as a key variable. Using the Utah Population Database, Smith et al. (2009) investigated how a range of early-life factors were associated with mortality in adulthood. The impact of birth order on adult mortality was not the main focus of the study. Operationalizing birth order as a binary variable indicating whether the individual was firstborn, that study found no statistically significant associations between birth order and adult mortality for either men or women. The study by O'Leary et al. (1996) found little relationship between birth order and mortality but used a small (n = 1,162) and non-representative sample, with insufficient statistical power to detect any patterns. Finally, a study using Swedish data (n = 14,192) from the Uppsala Birth Cohort Study found that birth order was associated with an increased risk of all-cause mortality for both men and women aged 20–54 and for men aged 55–80 (Modin 2002), although after adjusting for the SES of the ego in adulthood, the pattern was no longer statistically significant. However, sibship size was not included in the models. Because high birth orders are directly correlated with large family sizes, this leaves open the potential for confounding if sibship size is not adjusted for. Furthermore, none of these studies used the within-family comparison approach adopted in this study, leaving open the possibility that spurious associations could be observed even after the researchers adjusted for important variables, such as sibship size and parental SES. Nevertheless, given past research findings on the importance of birth order, we anticipate that all-cause mortality will increase with a rising birth order, and we also anticipate that we will observe the same pattern for cause-specific mortality.

Several hypotheses have been proposed for why birth order should be related to outcomes in adulthood. Among myriad explanations, two theories have gained particular scientific interest: the confluence hypothesis (Zajonc 1976; Zajonc and Markus 1975) and the resource dilution hypothesis (Blake 1981). These two theories explain discrepancies in achievement by birth order as being attributable to lower cognitive stimulation in early-life and cumulative resource disadvantage for later-born siblings. More specifically, the confluence hypothesis takes account of the fact that children are a part of their own dynamically changing environment, and states that as family size grows with an increasing number of children, the environment becomes steadily less cognitively stimulating (Zajonc 1976). This less-stimulating environment is hypothesized to impact intellectual development (Zajonc 1976). Previous findings of a negative relationship between cognitive ability and both education and longevity suggest that later-borns would have higher mortality in adulthood (Batty et al. 2007; Lager and Torssander 2012). The resource dilution hypothesis states that the pool of parental resources, which includes material, cognitive, and interpersonal resources (Hertwig et al. 2002), available to each child decreases as the sibship size increases (Blake 1981). First- and early-born children will spend the early years of life having the exclusive or near-exclusive attention of parents, whereas later-borns are forced to compete with siblings over resources from birth. Empirical research shows a negative relationship between birth order and the time that parents spend caring for their children (Price 2008), the amount of parental leave time that parents take (Sundström and Duvander 2002), and the likelihood of breast-feeding (Buckles and Kolka 2014). Although parents' incomes may increase as they age, this rising income is rarely sufficient to offset the dilution of resources as more children enter the household. Investment in childhood and early-life resource access has been shown to be associated with various measures of health in adulthood (Campbell et al. 2014), including mortality (Hayward and Gorman 2004).

In addition to the confluence hypothesis and the resource dilution hypothesis, several other explanatory theories have been offered. For example, the hygiene hypothesis (Strachan 1989) argues that a larger sibship increases the likelihood of communicable diseases being introduced into the family and that younger siblings may be more susceptible to these diseases (Holman et al. 2003; Strachan 1989). Another explanation, the family dynamics model (Sulloway 1996; Sulloway and Zweigenhaft 2010), assumes the fundamental aspects of the resource dilution hypothesis and extends it to argue that children tend to occupy different niches within the family environment, and that they also attempt to differentiate themselves from one another in order to avoid direct intersibling competition. It has been argued that these intrafamily dynamics tend to produce firstborns whose values are more closely aligned with those of their parents, and later-borns who are more rebellious and more likely to engage in risky or dangerous activities (Sulloway 1996; Sulloway and Zweigenhaft 2010; Zweigenhaft and Von Ammon 2000). Yet another explanation is that older siblings introduce younger siblings to developmentally inappropriate activities, such as smoking and alcohol consumption, at a younger age than they otherwise would have been, which may have both direct and indirect influences on health and mortality (Elliott 1992; Harakeha et al. 2007). We hypothesize that if there is support for this latter explanation then mortality attributable to cancers of the respiratory system, as well as external causes in the form of accidents, suicide, and events of undetermined

intent, should be positively associated with birth order; we will be able to test this hypothesis by looking at cause-specific mortality outcomes.

Another perspective in the literature is that the finding of a relationship between birth order and intelligence or educational attainment is a methodological artifact of drawing inferences about within-family patterns from between-family data and that these associations disappear after between-family heterogeneity is adjusted for (Rodgers 2001). However, recent research using high-quality, population-based, longitudinal Nordic administrative register data comparing siblings within the same family to one another suggests that within-family birth-order effects do exist and that later-born children fare worse on measures of both cognitive ability and educational attainment (Bjerkedal et al. 2007; Black et al. 2005, 2011; Kristensen and Bjerkedal 2007). The current study, using Swedish administrative register data, features the same advantages in terms of data and methodological approach. Given the strong and unambiguous evidence for the effect of IQ, educational attainment, and SES on health outcomes (Batty et al. 2007; Lager and Torssander 2012; Mackenbach et al. 1997; Marmot 2004; Torssander and Erikson 2010), we expect that mortality will increase for higher birth orders, and that this association will be mediated through social pathways in a way that is at least partially observable by using measures of SES and educational attainment. We hypothesize that this pattern will be clearest for cause-specific mortality associated with lifestyle and environmental conditions, such as cancers of the respiratory system and mortality attributable to external causes. We will be able to test this hypothesis by adjusting for SES and educational attainment in adulthood and by analyzing cause-specific mortality outcomes.

Data and Methods

Data

In this study, we use Swedish population register data to investigate the relationship between birth order and mortality. We conduct separate analyses for men and women. The individuals under analysis consist of cohorts born between 1938 and 1960, with 1938 being practically the earliest point for which we can obtain reliable information on parent–child linkages using the multigenerational Swedish registers. Here, we define a sibship as a group of siblings with the same biological mother–father pairing. We use the terms "set size" to refer to the size of the full sibling group and "set order" to refer to birth order within that sibling group. We do not restrict the calculation of set size or set order to these cohorts but instead use the full population registers to generate these measures. We link the population register to the Swedish mortality register, following them from 1990 to 2007 for both all-cause and cause-specific mortality. Some descriptive details on our data can be seen in Table 1. Although the Swedish mortality register contains data over the period 1960 to 2007, the multigenerational registers that allow family members to be linked to one another are incomplete before the 1990s (SCB 2011). We exclude families with plural births from our analyses because the meaning of birth order is less clear in these families. To maximize the quality of our birth-order measure, we also exclude sibling groups in which any of the children are born outside the Nordic region. For our main analyses, which use a within-family comparison

Table 1 Description of study population by set order, set size, birth year, study size, and causes of death for Swedish men and women born 1938–1960, observed over ages 30 to 69

		Birth Year					
Set Order by Birth Year	Set Order	1938–1945	1946–1950	1951–1955	1956–1960	Total	
	1	233,142	155,409	147,420	155,517	691,488	
	2	189,285	163,070	150,689	155,368	658,412	
	3	63,766	66,370	64,579	65,579	260,294	
	4	18,676	24,083	23,618	24,494	90,871	
	5	4,474	8,282	8,408	8,355	29,519	
	6	641	2,214	2,484	2,465	7,804	
	Total	509,984	419,428	397,198	411,778	1,738,388	
		Birth Year					
Set Size by Birth Year	Set Size	1938–1945	1946–1950	1951–1955	1956–1960	Total	
	2	218,224	181,607	169,982	180,049	749,862	
	3	150,876	126,946	123,730	134,127	535,679	
	4	80,870	65,219	62,104	62,060	270,253	
	5	40,028	30,600	28,101	24,668	123,397	
	6	19,986	15,056	13,281	10,874	59,197	
	Total	509,984	419,428	397,198	411,778	1,738,388	
		Set Size					
Set Order by Set Size	Set Order	2	3	4	5	6	Total
	1	391,014	195,385	71,711	24,305	9,073	691,488
	2	358,848	188,444	74,409	26,512	10,199	658,412
	3		151,850	69,593	27,661	11,190	260,294
	4			54,540	25,227	11,104	90,871
	5				19,692	9,827	29,519
	6					7,804	7,804

Table 1 (continued)

	Total	749,862	535,679	270,253	123,397	59,197	1,738,388
		Men			Women		
Study Size	Cause of Death	*N*	Deaths		*N*	Deaths	
	All-cause mortality	889,126	49,909		849,262	31,219	
	Diseases of the circulatory system		14,548			4,958	
	Neoplasms		15,461			17,350	
	Cancers of the respiratory system		2,952			3,123	
	External causes		8,983			3,189	

Note: Set order refers to birth order, and set size refers to the size of the sibling group.

Source: Swedish administrative register data, compiled by the authors.

approach (to be described in more detail later herein), we also exclude only-children. Finally, we study sibling groups with between two and six children because sibling sizes greater than six are rare, and including those few cases produced unreliable estimates for birth orders seven and higher. Based on these exclusion criteria, of the 2,166,948 individuals in the 1938–1960 birth cohorts living in Sweden in 1990, we have 1,788,388 individuals from sibling groups with between two and six children available for our analyses.

Aside from all-cause mortality, we address mortality attributable to the following causes: neoplasms; cancers of the respiratory system; diseases of the circulatory system; and external causes, which includes accidents, suicides, and events of undetermined intent. These cause-specific outcome variables were coded using the World Health Organization (WHO) International Classification of Diseases (ICD), versions 9 and 10, taking into account the transition between these versions in 1996 in Sweden (Janssen and Kunst 2004). Because we also study cancers of the respiratory system as a specific outcome, we remove this category of cancers from the larger category of neoplasms for the analyses presented here. Because the 1990s are the earliest point at which we can reliably link the multigenerational registers to the mortality register, we have both left- and right-censoring in our models, and the age at which individuals enter and exit the analysis varies for different birth cohorts. This means that the members of the earliest cohort in our study, born in 1938, enter the analysis at age 52 and are followed until age 69; and members of the latest-born cohort, born in 1960, are followed from ages 30 to 47. Because of the nature of the data, we are not able to observe mortality for the youngest ages of adulthood, from 18 to 29, and for the oldest ages, after 69. Although we are unlikely to lose a great deal of information on mortality attributable to diseases of the circulatory system and different cancers by having the earliest age of analysis at 30, we undoubtedly fail to fully capture all of the deaths attributable to external causes in the form of accidents and suicides. We also fail to observe a large proportion of the deaths of each cohort by not observing them later than age 69. Including all our birth cohorts, 4.7 % of our total study population died between the time they entered the analysis and the end of the follow-up period. This proportion is higher for the oldest cohort, at approximately 13 %.

Statistical Analyses

We conducted within-family analyses to estimate the relationship between birth order and mortality. The within-family analyses—meaning a within-sibship comparison—use fixed-effect discrete-time survival analysis. The hazard function—that is, the probability that individual i has an event y during interval t, given that no event has occurred before the start of t—is defined as

$$h_i(t) = Pr(y_i(t) = 1 | y_i(t-1) = 0).$$

We fit a discrete-time logistic regression model of the following form:

$$\text{logit}\left[h_{ij}(t)\right] = log\left(\frac{h_{ij}(t)}{1-h_{ij}(t)}\right) = \alpha(t) + \beta x_{ij}(t) + u_j,$$

where h_{ij} refers to the hazard of failure at discrete time t for respondent i in sibling group j; $\alpha(t)$ is the logit of the baseline hazard function; x_{ij} is a vector of covariates for

individual i; β is the vector of regression parameters; and u_j refers to the fixed-effect component for any given sibling group j. The estimator used is a conditional logistic regression model (Greene 2012; Hosmer et al. 2013), with fixed effects specified at the level of the sibling group. These models have been estimated using cluster-adjusted standard errors to account for any potential intragroup correlation (Primo et al. 2007). The clusters in this study are sibships.

The results from these discrete-time survival analyses are the main results presented in the Results section. Although we study individuals from different cohorts across different ages, using survival analysis allows us to adjust for differences in the number of person-years of exposure that different groups contribute to the analysis. We right-censor for the first out-migration of any individual from Sweden. In our main analysis of all-cause mortality, 5.7 % of men and 3.6 % of women out-migrated over the entire study period. Table 1 shows the study size as well as the number of deaths for men and women. Besides modeling all-cause mortality, we also estimate the cause-specific mortality of other causes of death. We can no longer assume independent right-censoring because our causes of death are dependent on each other; thus, we can no longer estimate the marginal effect (the effect of our covariates on a specific cause of death in the absence of other causes of deaths). We can, however, still examine the extent to which birth order mediates mortality for different causes of death.

Because the within-sibship comparison fixed-effects approach requires within-family variation, we are able to examine only those families in which at least one sibling has died. Thus, the frequency of the outcome is very high in the within-family logistic regression models. The procedure by which odds ratios are calculated means that when the incidence of an outcome is greater than 10 %, as it is in this study, any given odds ratio will be elevated relative to the corresponding difference in the probability of the outcome between two groups (Zhang and Kai 1998). For example, when the frequency of the outcome is 50 %, the odds ratio can be more than 150 % higher than the corresponding relative risk (Schmidt and Kohlmann 2008). Odds ratios are not problematic in and of themselves, but it is important that they are interpreted in terms of a relative increase or decrease in the odds of an outcome rather than a relative increase or decrease in the probability of an outcome between groups.

Covariates in Survival Analyses

We adjust our estimates of the relationship between birth order and mortality for a number of different variables that are theoretically confounders for this relationship. Correlation matrices for these variables are shown in Table S10 in Online Resource 1. In the analyses, we adjust for the age of the ego's mother in the birth year of the ego and for cohort. Theoretically, all other intrafamily characteristics, including sibship size, geographical location, and parental SES, are inherently accounted for by conducting a within-family comparison, allowing us to focus exclusively on the importance of birth order for mortality. This approach precludes concerns that the results for birth order may be a statistical artifact of drawing within-family inferences from a between-family comparison (Rodgers et al. 2000), thereby isolating the causal effect of birth order on mortality.

We adjust for cohort effects rather than period effects for two reasons. The first is burgeoning evidence that suggests the importance of *in utero* and early-life conditions

around the time of birth, which vary substantially by cohorts over time, on longevity (Bengtsson and Broström 2009; Bengtsson and Mineau 2009; Gluckman et al. 2008). Furthermore, previous research has indicated that cohort effects play a more significant role in mortality trends than period effects (Richards et al. 2006). In addition, because of changing fertility preferences, period-specific fertility patterns are also related to cohort size (Andersson et al. 2009; Andersson and Kolk 2011), which is related to birth order. Thus, we include a variable for birth year to account for these underlying patterns. We also implicitly adjust for period effects by adjusting for both cohort and age. We adjust for maternal age at birth because evidence suggests that this is an important factor influencing a wide range of adult health outcomes (Myrskylä and Fenelon 2012). In our within-family comparison analyses, we exclude only-children because variance in the outcome is required within the sibling group. We also do not include any sibling set that includes multiple births because the meaning of birth order is different in these families. The full results in Online Resource 1 show the association between birth order and mortality from pooled analyses for children born in sibling sets ranging in size from two to six, as well as results from sibship-size–specific analyses for both the within-family and between-family analyses.

Because previous research has shown that birth order influences education and IQ (Bjerkedal et al. 2007; Black et al. 2005, 2011; Kristensen and Bjerkedal 2007), we also conduct additional analyses to estimate the degree to which the relationship between birth order and mortality is mediated by socioeconomic class and educational attainment, measured in adulthood. To do this, we estimate models in which we adjust for a common measure of SES, the Erikson, Goldthorpe, and Portocarero occupational class scheme (EGP) (Erikson and Goldthorpe 1992; Erikson et al. 1979), measured between ages 30 and 40 using information on occupation from the Swedish censuses in 1960, 1970, 1980, and 1990. The EGP variable used in this study is divided into the following categories: upper service class, including self-employed professionals (EGP = I); lower service class (EGP = II); routine nonmanual (EGP = III); self-employed nonprofessionals, farmers, and fishermen (EGP = IV); skilled and unskilled workers (EGP = VI–VII); and unknown/other. We adjust for educational attainment using information from the Swedish educational register, which has been updated continuously since 1987, using information on the highest achieved educational level starting from age 51. These additional analyses adjusting for socioeconomic and educational attainment are limited to individuals aged 52 years or older. We conduct separate analyses for men and women. We also present results based on this same older sample group without the inclusion of the variables for adult socioeconomic and educational attainment for the sake of comparison.

Between-Family Analyses

Although the main analyses to be presented use a within-family comparison, we also conduct the full set of analyses described for the within-family comparisons using a between-family comparison approach to provide comparability to previous research on birth order and mortality. The between-family analyses also use discrete-time survival analysis, using sequential logistic regression. As in the within-family analyses, we estimate cluster-adjusted standard errors. In these between-family analyses, we include *singletons* (i.e., individuals from sibling groups with only one child) because this

statistical approach does not require variance within the sibling group. In these between-family analyses, we adjust for the age of the ego, the age of the ego's mother in the birth year of the ego, cohort, and the sibling set size of which the ego is a part. These results can be seen in Tables S4, S6, and S7 in Online Resource 1.

Results

The main analyses presented in this article use discrete-time survival analyses in the form of logistic regressions, specifying fixed effects at the sibship level, to perform a within-family comparison; we compare only those siblings born to the same biological mother and father to one another. The results from the within-family analyses for all-cause mortality are shown in Fig. 1 for men and women, in Table 2 for men, and in Table 3 for women. These results show a positive and statistically significant relationship between birth order and mortality, with the hazard rising steadily with an increasing birth order for both men and women. This relationship is considerably stronger for

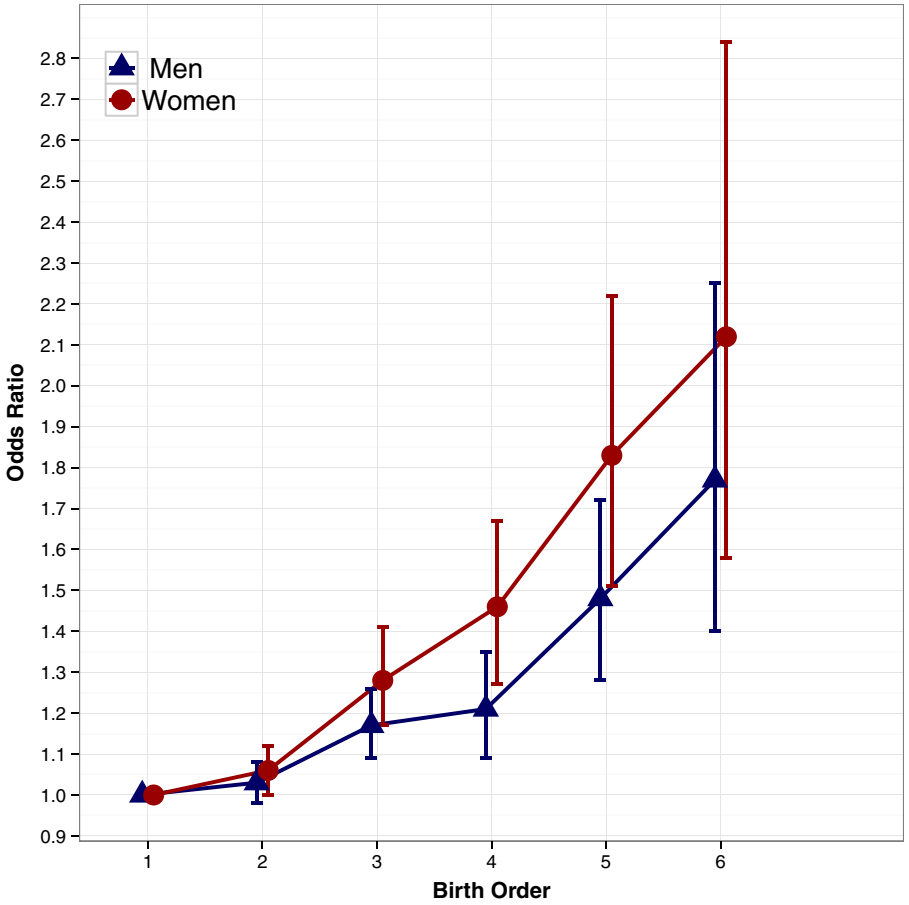

Fig. 1 Within-family discrete-time survival analyses: All-cause mortality by birth order, Swedish men and women born 1938–1960. Error bars are 95 % confidence intervals

Table 2 Within-family discrete-time survival analyses: All-cause mortality results by birth order, Swedish men born 1938–1960, observed over ages 30 to 69

		Model I: Cohorts 1938–1960 No Adult SES Controls			Model II: Aged 52+ Model III Sample, No SES Controls			Model III: Aged 52+ Adult EGP and Education		
Variable	Category	OR	SE	95 % CI	OR	SE	95 % CI	OR	SE	95 % CI
Set Order	1	1 (ref.)			1 (ref.)			1 (ref.)		
	2	1.03	0.02	0.98–1.08	0.99	0.04	0.92–1.07	0.95	0.04	0.88–1.02
	3	1.17	0.04	1.09–1.26	0.99	0.06	0.88–1.13	0.95	0.06	0.84–1.08
	4	1.21	0.07	1.09–1.35	1.07	0.10	0.89–1.28	0.96	0.09	0.80–1.16
	5	1.48	0.11	1.28–1.72	1.33	0.18	1.02–1.75	1.17	0.16	0.88–1.54
	6	1.77	0.21	1.40–2.25	2.17	0.50	1.38–3.40	1.83	0.43	1.15–2.91
Mother's Age	<20	0.87	0.06	0.76–1.01	1.33	0.17	1.03–1.72	1.24	0.16	0.95–1.61
	20–25	0.92	0.03	0.86–0.98	0.92	0.05	0.82–1.03	0.91	0.05	0.81–1.03
	26–30	1 (ref.)			1 (ref.)			1 (ref.)		
	31–35	1.07	0.04	1.00–1.15	0.86	0.05	0.76–0.96	0.92	0.05	0.82–1.04
	36–40	1.10	0.06	0.98–1.23	0.73	0.07	0.60–0.89	0.81	0.08	0.67–0.99
	>40	1.20	0.11	1.01–1.44	0.81	0.13	0.59–1.12	0.91	0.15	0.66–1.25
Birth Year	1938–1945	1 (ref.)			1 (ref.)			1 (ref.)		
	1946–1950	1.06	0.04	1.00–1.14	0.83	0.04	0.75–0.92	0.83	0.04	0.75–0.92
	1951–1955	1.25	0.07	1.13–1.39						
	1956–1960	1.34	0.10	1.16–1.56						
Age	30–35	0.00	0.00	0.00–0.00						
	36-40	0.02	0.00	0.02–0.02						
	41–45	0.05	0.00	0.05–0.05						
	46–50	0.13	0.00	0.12–0.13						

Table 2 (continued)

Variable	Category	Model I: Cohorts 1938–1960 No Adult SES Controls			Model II: Aged 52+ Model III Sample, No SES Controls			Model III: Aged 52+ Adult EGP and Education		
		OR	SE	95 % CI	OR	SE	95 % CI	OR	SE	95 % CI
	51–55	0.36	0.01	0.35–0.37	1 (ref.)			0.20	0.00	0.20–0.21
	56–60	1 (ref.)			4.78	0.10	4.58–4.99	1 (ref.)		
	61–65	3.08	0.05	2.98–3.19	18.01	0.47	17.10–18.95	3.90	0.08	3.76–4.05
	>65	9.93	0.32	9.32–10.58	64.46	2.53	59.68–69.63	14.24	0.50	13.29–15.26
Occupational Class	Upper service class							0.60	0.05	0.51–0.70
	Lower service class							0.64	0.03	0.58–0.72
	Routine nonmanual							0.74	0.05	0.65–0.84
	Self-employed, farmers							0.82	0.06	0.72–0.94
	Skilled/unskilled workers							1 (ref.)		
	Other/unknown							3.23	0.19	2.88–3.63
Education	Missing							1.88	0.28	1.40–2.51
	Primary							1 (ref.)		
	Secondary							0.77	0.03	0.71–0.83
	Tertiary							0.44	0.03	0.39–0.50
N				84,068			38,759			38,759
Deaths				49,086			25,657			25,657

Source: Swedish administrative register data, compiled by the authors.

Table 3 Within-family discrete-time survival analyses: All-cause mortality results by birth order, Swedish women born 1938–1960, observed over ages 30 to 69

		Model I: Cohorts 1938–1960 No Adult SES Controls			Model II: Aged 52+ Model III Sample, No SES Controls			Model III: Aged 52+ Adult EGP and Education		
		OR	SE	95 % CI	OR	SE	95 % CI	OR	SE	95 % CI
Set Order	1	1 (ref.)			1 (ref.)			1 (ref.)		
	2	1.06	0.03	1.00–1.12	0.99	0.05	0.90–1.09	0.97	0.05	0.88–1.07
	3	1.28	0.06	1.17–1.41	1.16	0.09	1.00–1.36	1.14	0.09	0.98–1.33
	4	1.46	0.10	1.27–1.67	1.17	0.14	0.93–1.47	1.17	0.14	0.93–1.48
	5	1.83	0.18	1.51–2.22	1.24	0.22	0.87–1.77	1.24	0.22	0.87–1.77
	6	2.12	0.32	1.58–2.84	1.54	0.44	0.88–2.70	1.41	0.41	0.80–2.50
Mother's Age	<20	0.95	0.09	0.79–1.15	1.01	0.17	0.73–1.40	0.98	0.16	0.71–1.35
	20–25	1.04	0.05	0.95–1.13	1.20	0.09	1.04–1.39	1.20	0.09	1.03–1.39
	26–30	1 (ref.)			1 (ref.)			1 (ref.)		
	31–35	1.01	0.04	0.93–1.11	0.82	0.06	0.71–0.94	0.85	0.06	0.74–0.98
	36–40	1.02	0.07	0.89–1.17	0.74	0.09	0.58–0.94	0.81	0.10	0.63–1.03
	>40	1.23	0.14	0.98–1.55	0.55	0.12	0.36–0.83	0.59	0.13	0.39–0.90
Birth Year	1938–1945	1 (ref.)			1 (ref.)			1 (ref.)		
	1946–1950	1.17	0.05	1.07–1.27	1.00	0.06	0.88–1.13	1.02	0.07	0.90–1.16
	1951–1955	1.27	0.09	1.11–1.46						
	1956–1960	1.43	0.14	1.18–1.74						
Age	30–35	0.00	0.00	0.00–0.00						
	36–40	0.01	0.00	0.01–0.01						
	41–45	0.04	0.00	0.04–0.04						
	46–50	0.12	0.00	0.11–0.12						

Table 3 (continued)

Variable	Category	Model I: Cohorts 1938–1960 No Adult SES Controls			Model II: Aged 52+ Model III Sample, No SES Controls			Model III: Aged 52+ Adult EGP and Education		
		OR	SE	95 % CI	OR	SE	95 % CI	OR	SE	95 % CI
	51–55	0.33	0.01	0.32–0.34	1 (ref.)			0.19	0.01	0.18–0.20
	56–60	1 (ref.)			5.26	0.14	4.99–5.55	1 (ref.)		
	61–65	2.90	0.06	2.78–3.02	18.65	0.62	17.48–19.91	3.60	0.09	3.44–3.78
	>65	8.67	0.35	8.00–9.39	64.53	3.21	58.54–71.13	12.62	0.56	11.57–13.76
Occupational Class	Upper service class							1.05	0.14	0.82–1.35
	Lower service class							0.94	0.07	0.81–1.08
	Routine nonmanual							0.88	0.06	0.77–1.00
	Self-employed, farmers							0.80	0.09	0.65–1.00
	Skilled/unskilled workers							1 (ref.)		
	Other/unknown							1.20	0.07	1.08–1.34
Education	Missing							6.07	1.50	3.74–9.86
	Primary							1 (ref.)		
	Secondary							0.71	0.03	0.65–0.78
	Tertiary							0.39	0.03	0.33–0.45
N				52,787			24,949			24,949
Deaths				30,897			16,479			16,479

Source: Swedish administrative register data, compiled by the authors.

women than for men. Although the analyses presented here pool individuals in sibship sizes ranging from two to six, we also conducted within-family comparison analyses that were specific to sibship size. These results are shown in Table S4 in Online Resource 1. The overall pattern of increasing mortality by birth order is consistent for the sibship-size-specific results. A substantially elevated hazard for higher birth orders remains even in families with two or three children, which are the most common family sizes in Sweden.

To test the extent to which the relationship between birth order and mortality is mediated by social pathways, we conduct additional analyses in which we adjust for SES and educational attainment in adulthood. The results for all-cause mortality from these additional analyses are shown in Fig. 2 and Table 2 for men and in Fig. 3 and Table 3 for women. These additional analyses of men and women aged 52 or older use a different sample group because variables for socioeconomic and educational attainment are consistently available for the entire sample only at an older age. These results show that the association between birth order and mortality is weaker than that seen in

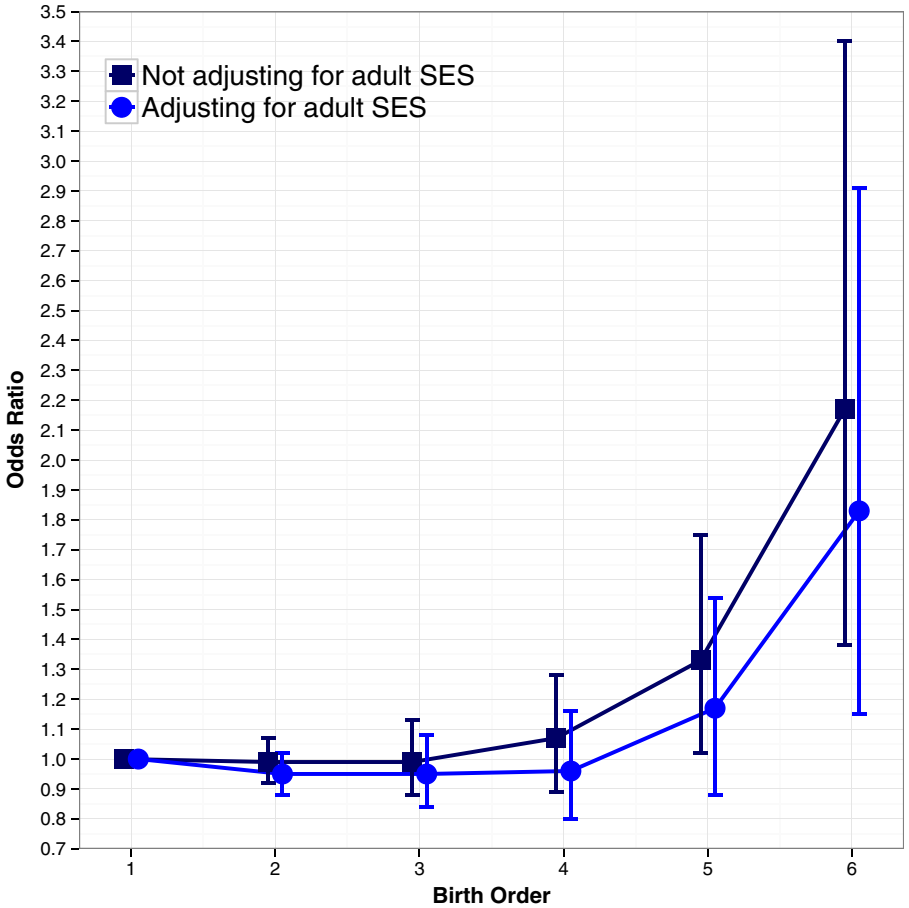

Fig. 2 Within-family discrete-time survival analyses: Swedish men aged 52 and older. All-cause mortality by birth order, with and without adjusting for SES in adulthood. Error bars are 95 % confidence intervals

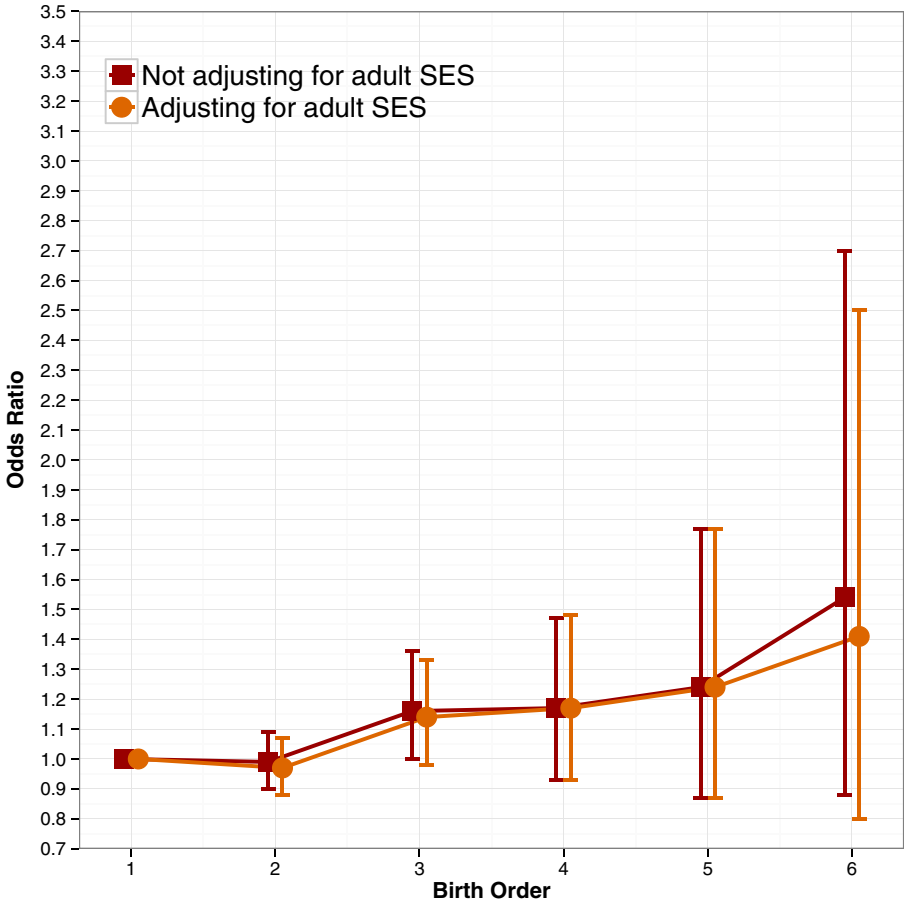

Fig. 3 Within-family discrete-time survival analyses: Swedish women aged 52 and older. All-cause mortality by birth order, with and without adjusting for SES in adulthood. Error bars are 95 % confidence intervals

the models based upon the full sample. Figure 2 shows that the relationship between birth order and mortality for men aged 52 and older is flat for birth orders 2 and 3 relative to firstborns, and then increases from birth order 4. However, the difference in the odds ratios is statistically significant only for birth orders 5 and 6. After we adjust for attained SES and educational attainment, the confidence intervals show that mortality is statistically significantly elevated only for individuals of birth order 6 relative to firstborns. Overall, adjusting for attained SES and educational attainment decreases the size of the parameter estimates, suggesting that this is a mediating factor between birth order and mortality for men in this age group. Nevertheless, the smaller and age-restricted nature of the sample means that the standard errors for these estimates are substantially larger than those seen in the results for the full sample. The results in Fig. 3 for women show that mortality is elevated from birth order 3 to birth order 6, but these results are not statistically significant. Adjusting for attained SES and educational attainment has little impact on the size of the parameter estimates, indicating that adult socioeconomic attainment is not an important mediating variable for birth order and mortality for women aged 52 and older.

In addition to the analyses of all-cause mortality, we study cause-specific mortality for several major causes of death. The cause-specific patterns for the within-family analyses are shown in Fig. 4 for men and Fig. 5 for women (see also Table S5 in Online Resource 1). For men, the odds of mortality attributable to diseases of the circulatory system are lower for birth orders 2 to 4 in comparison with firstborns, before leveling out for birth orders 5 and 6. In contrast, the odds of mortality attributable to neoplasms, and cancers of the respiratory system are flat until birth orders 5 and 6, at which point they increase substantially. However, the confidence intervals, shown in Table S5, show that the differences are not statistically significant. The strongest pattern of association for men is clearly mortality attributable to external causes, which includes accidents, suicides, and events of undetermined intent. The odds of mortality attributable to external causes rises steadily up to birth order 4 before decreasing slightly for birth orders 5 and 6. The results for the analyses for women were substantially larger than those observed for men. Although the odds of mortality attributable to diseases of the circulatory system are slightly negative for later-born siblings, the odds of mortality

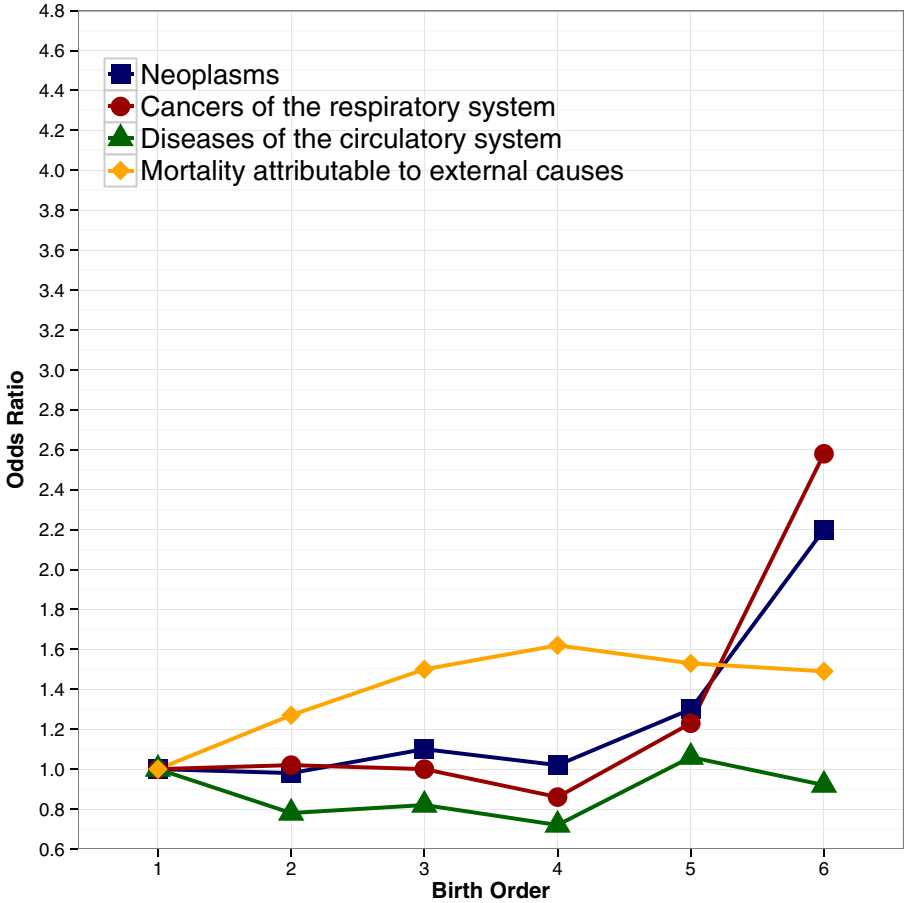

Fig. 4 Within-family discrete-time survival analyses: Cause-specific mortality by birth order, Swedish men born 1938–1960

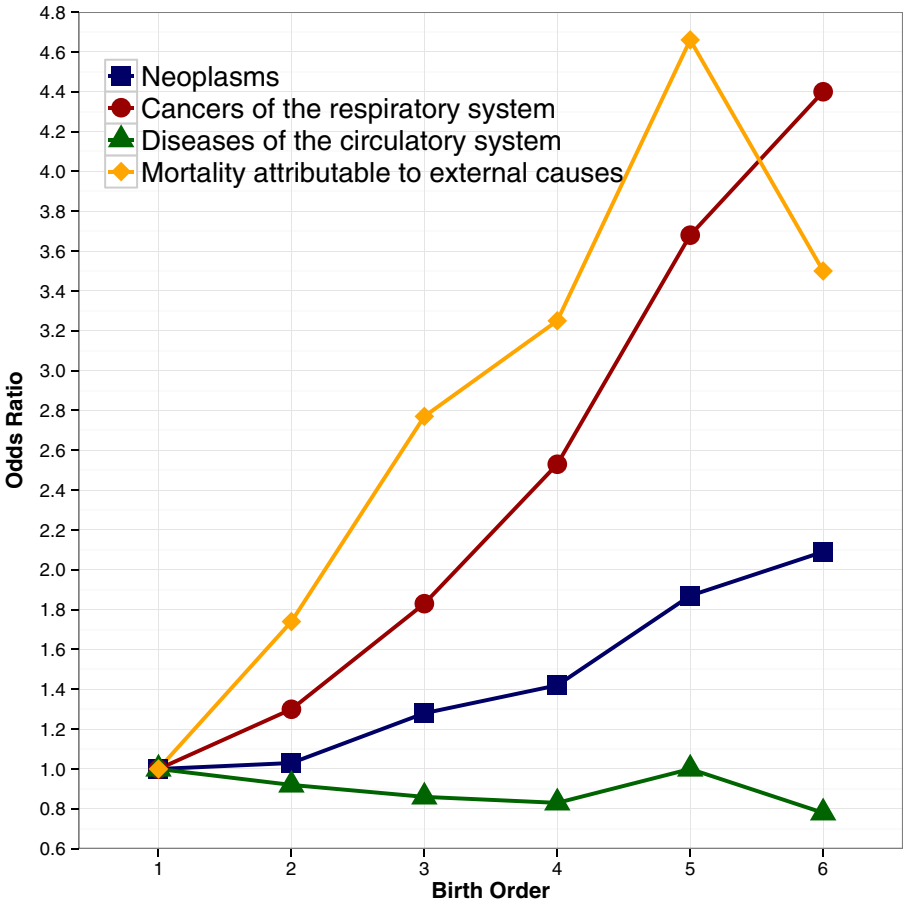

Fig. 5 Within-family discrete-time survival analyses: Cause-specific mortality by birth order, Swedish women born 1938–1960

attributable to neoplasms, cancers of the respiratory system, and external causes increase very substantially with an increasing birth order.

As described earlier in this article, we also conduct the full set of analyses using a between-family comparison approach, using discrete-time survival models in the form of logistic regressions, in order to provide a comparison to the previous research on birth order and mortality. This between-family analysis approach looks at the association between birth order and mortality across all families rather than conducting a within-family comparison of siblings born to the same mother and father. Online Resource 1 presents the all-cause mortality results from these between-family analyses for men (Table S6) and women (Table S7), as well as the cause-specific mortality results from the between-family analyses for men and women (Table S8). These results show that singletons often have mortality comparable to that of fourth-borns or later. We also conduct sibship-size-specific analyses using the between-family comparison approach, and these are presented in Table S4 alongside the sibship-size-specific analyses from the models using the within-family comparison approach.

Finally, we also conduct robustness checks to verify that the main results presented earlier are not skewed by the differences in the follow-up time for different cohorts. For these analyses, we used discrete-time survival models, meaning a between-family comparison, and restricted the follow-up period to age 65. We used the between-family comparison approach because the within-family approach requires that at least two children be alive in each sibship group. Given that they must have an opportunity to live to the age of 65, we must focus only on cohorts born from 1938 to 1942. Thus, larger sibship groups are particularly unusual, because they require that multiple siblings are born within a limited period, which introduces endogeneity problems. The results of these robustness checks are shown in Table S9 (Online Resource 1). The results are still fully consistent with the main results. We also conduct analyses in which we restrict the follow-up period to age 60, and age 55, with the analyses conducted on the 1938–1947 and 1938–1952 cohorts, respectively. These results, also shown in Table S9, are consistent with our main finding. We also considered that the interaction between the gender composition of the sibling group and birth order might have an impact on mortality. However, these extra analyses, performed on sibling groups of up to three children, showed no substantively or statistically significant patterns of association. These results are available upon request from the authors.

Discussion

The results of these analyses demonstrate that birth order matters for men's and women's mortality in adulthood after confounding attributable to factors shared among siblings are eliminated and other potential confounding factors are minimized. This is true for all-cause mortality as well as for several cause-specific mortality patterns, and it is particularly pronounced for mortality attributable to external causes for men and for mortality attributable to neoplasms, cancers of the respiratory system, and external causes for women. The overall pattern of these all-cause mortality results is consistent with those reported by Modin (2002). These results indicate that relative deprivation and cumulative disadvantage early in life can have long-term consequences, even extending into adulthood. Previous research has shown that sibship size is related to mortality both in childhood as well as adulthood, but few studies have had a sufficiently large database to investigate the impact of birth order itself on mortality, and particularly not to conduct a within-family analysis. We also find that the relative effect of birth order on mortality is greater between sisters than it is between brothers; and when looking at an older portion of our sample, we find that socioeconomic and educational attainment only mediates the relationship between birth order and mortality to a limited degree.

By using a within-family comparison approach, comparing only siblings born to the same biological mother–father pairing, we eliminate residual confounding with respect to shared factors among siblings. Furthermore, by adjusting for a number of confounders within the sibling group, such as maternal age at the time of birth and birth year, we minimize confounding in our results. However, although the results from the within-family analyses allow us to rule out confounding from factors shared among siblings, these results do not allow us to distinguish between the different hypotheses about how this relationship operates, including the confluence hypothesis (Zajonc

1976), the resource dilution hypothesis (Blake 1981), the hygiene hypothesis (Strachan 1989), and the family dynamics model (Sulloway 1996). Each of these hypotheses predicts that later-born siblings have poorer outcomes for IQ and educational attainment; that is, they predict that the observed association between birth order and mortality in adulthood is transmitted through social pathways, such as adult SES. The results from this study, however, show that the effect of birth order on mortality is largely the same after we adjust for adult SES and educational attainment. A limitation of this study is that we are able to adjust for socioeconomic and educational attainment only for individuals aged 52 or older. Previous research has indicated that the association between birth order and mortality is weaker in later stages of the life course than at younger ages (Modin 2002).

The resource dilution hypothesis and the confluence hypothesis in particular would predict that the degree to which parental resources are available for children by birth order and the degree to which they are exposed to an intellectually stimulating environment within the household at a crucial point of their lives are likely to have important implications for their long-term prospects for educational and intellectual development (Sénéchal and LeFevre 2002; Sénéchal et al. 1998). In addition, previous research on the relationship among birth order and IQ, educational attainment, and height has found that birth-order effects persist even among children raised in high-SES families (Bjerkedal et al. 2007; Kristensen and Bjerkedal 2010; Myrskylä et al. 2013). One the one hand, this would suggest support for the confluence hypothesis, given that even later-born children in high-SES families are unlikely to be left wanting in terms of access to resources. On the other hand, the observed birth-order effects are relative effects. Although the marginal gain from access to additional resources is likely to diminish past a certain threshold, presumably realized in high-SES families, there may still be an effect of relative access to resources. This would be consistent with research on social status and income inequality finding that even when all basic needs are satisfied, relative social standing and material resources still produce a gradient in health (Marmot 2004).

The results from the cause-specific mortality analyses, in which mortality attributable to external causes rises sharply by birth order for men and women and mortality attributable to cancers of the respiratory system rises sharply by birth order for women, suggest possible support for the family dynamics model. This model argues that children tend to occupy different niches within the family environment and that these intrafamily dynamics tend to produce firstborns whose values are more closely aligned with those of their parents and later-borns who are more rebellious and more likely to engage in risky activities (Sulloway 1996; Sulloway and Zweigenhaft 2010; Zweigenhaft and Von Ammon 2000). These predictions would be consistent with the patterns observed for mortality attributable to cancers of the respiratory system and mortality from external causes. Although this study has primarily focused on the social pathways by which birth order is linked to outcomes in adulthood, physiological pathways accounted for by prenatal or gestational factors are also possible (Gualtieri and Hicks 1985). However, the evidence for physiological pathways predicting a negative relationship between birth order and health is sparse. Research on sibling groups in which children have died in infancy and in fully adopted sibling groups indicate that it is social set order rather than biological birth order that explains the consistently observed birth-order effects (Barclay 2015b; Kristensen and Bjerkedal 2007).

The within-family comparison results show that the relative effect of birth order on mortality is greater between sisters than it is between brothers. Although the reason for this is not clear, previous research has shown that women are much more closely involved in kin work, such as maintaining kinship ties (Hagestad 1986; Rossi and Rossi 1990; Young and Willmott 1957). It may be that these closer ties to kin mean that women are more affected by intrafamily interactions than are men. Alternatively, the nature of our sample means that we do not observe mortality at the youngest ages. Given that men have higher rates of mortality at all ages, forces of selection mean that those who survive to enter our analysis are less frail. If part of this selection is related to birth order, this would partially account for the stronger effects observed for women. Another, related explanation is that because we observe mortality before very old age and because mortality in our study period is more common for men than for women, our observed mortality is likely to be concentrated in more vulnerable populations in the portion of our sample that is female. If birth-order effects are stronger in more vulnerable populations, this could partially explain these results. Previous research also indicates gender differences in the degree to which later-life outcomes, such as the risk of cardiac disease, diabetes, and obesity, are affected by early-life socioeconomic conditions (Hamil-Luker and O'Rand 2007; Khlat et al. 2009; Maty et al. 2008). This research indicates that women are more responsive to early-life conditions than men. It has been argued that because of institutionalized structures that disadvantage women relative to men—most prominently in terms of paid work and household conditions (Read and Gorman 2010)—women might be more heavily affected by the chains of risk that follow early-life disadvantage than are men (Hamil-Luker and O'Rand 2007). Although men have higher rates of mortality at all ages relative to women, when stratifying analyses by gender, the differences between women coming from different socioeconomic backgrounds are greater than they are for men. Given that birth order is a marker of early-life resource availability and intellectual stimulation within the household, the results presented in this article are consistent with that previous research on socioeconomic conditions in childhood and later-life health outcomes.

A potential alternative explanation for the pattern observed for mortality attributable to cancers of the respiratory system is sibling influence. Research in the fields of social psychology and social networks has consistently and convincingly demonstrated the importance of alters, including parents and siblings, for shaping health behaviors (Christakis and Fowler 2008; Leonardi-Bee et al. 2011; Rosenquist et al. 2010). Studies more particularly focused on sibling influences show that younger siblings—those with a higher birth order—are more likely to begin smoking if an older sibling already smokes, but this relationship is not reversed (Harakeha et al. 2007). There are also indications that, because of this pattern of smoking uptake by younger siblings, they are likely to begin smoking at younger ages (Bard and Rodgers 2003). Smoking initiation at younger ages is associated with a greater daily cigarette consumption as well as a stronger tendency toward smoking continuation, particularly when smoking initiation begins before age 16 (Chen and Millar 1998; Khuder et al. 1999). These findings suggest that an individual with a higher birth order will be more likely to smoke in the long term, with obvious implications for the future health conditions of that individual's respiratory system, regardless of his or her socioeconomic trajectory over the life course. Although smoking behavior would also impact the health of the

circulatory system, previous research indicates that younger siblings demonstrate both a higher rate of alcoholism and a greater proclivity to initiate developmentally inappropriate activities at younger ages (Blane and Barry 1973; Rodgers and Rowe 1988). The positive relationship between birth order and both cancers of the respiratory system and external causes for women suggests some support for this hypothesis, but unfortunately we do not have data on smoking or drinking behavior that would allow us to test the degree to which these factors may mediate that relationship.

The nature of the data used in this study means that we have studied adult mortality, at ages 30 to 69, with different birth cohorts contributing exposure for different ages. Although most deaths in Sweden take place after age 70, mortality before this age indicates that the exposure can explain variation in mortality among the healthiest and most robust section of the population. Unfortunately, we were not able to study mortality at age 70 and older because the data contained information on mortality only up to 2007, and our earliest cohort was born in 1938. In the future, it will be valuable to address whether the birth-order effect on mortality persists among the elderly. In Sweden, the majority of deaths at old ages are attributable to cancer and diseases of the circulatory system (Janssen and Kunst 2005; Socialstyrelsen 2010). Given that the strongest pattern for the relationship between birth order and mortality is seen for neoplasms in general, and for cancers of the respiratory system more particularly for women, one might speculate that the birth-order effect on mortality attributable to neoplasms could persist for women into older ages. For men, this is less clear because the relationship between birth order and mortality attributable to neoplasms generally, cancers of the respiratory system more particularly, and diseases of the circulatory system, is weaker than that seen for women. However, because we do not study individuals aged 70 and older, these suggestions about mortality patterns by birth order among the elderly should be interpreted as conjecture, particularly given that previous research has suggested that rates of mortality attributable to specific causes differ not only by age but also across cohorts (Janssen and Kunst 2005).

This study has many strengths, but certain factors are difficult to account for when using register data. For example, we have not been able to test the specific mechanisms that potentially link birth order to mortality, and this will be an important dimension of this research question for future studies to address. In this study, we look at birth order within sibships, where a sibship is defined as a group of children born from the same biological mother–father pairing. Our research excludes half-brothers or half-sisters who may, practically speaking, be part of a sibship. This can be seen as both an advantage and a disadvantage. Indeed, a general shortcoming is that we are not able to observe which children are in the household—an important factor when considering the potential importance of a shared pool of resources and how this might be related to later health outcomes. We also do not have access to information on birth weight. Firstborns consistently have a lower birth weight than later-born siblings (Magnus et al. 1985), and birth weight has been shown to be positively associated with educational attainment and earnings in adulthood (Black et al. 2007). Thus, the estimates for the effect of birth order on mortality presented in this study represent a conservative lower bound, and accounting for birth weight would increase the point estimates. An additional factor that we do not adjust for in our models is the potential role of the time interval between the births of siblings. However, birth intervals are endogenous and will be strongly related to the SES of the parents, meaning that the extent to which the results of further

analyses would further clarify the underlying processes might be limited. Furthermore, it is not possible to overcome this endogeneity by using a within-family comparison because the values for the interaction between birth order and birth intervals are constant within a sibling group. Overall, the results of this study demonstrate how social conditions within the family of origin can significantly influence long-term health outcomes.

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