The Effect of Number of Siblings on Mortality Risk: Evidence from Swedish Register Data

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ABSTRACT

Demographic research has paid a lot of attention to the impact of childhood conditions on adult mortality. This paper focuses on one of the key aspects of early life conditions, i.e. family size, and to examine the causal effect of growing up in a large family on mortality risk. We use Swedish register data and a quasi-experimental approach that exploits multiple births as a source of exogenous variation in the number of siblings. While previous studies have examined the effects of family size on health and mortality in low or middle income countries, we show whether growing up in a large family may be considered as a disadvantage in a country context where the resources of most parents are not dramatically scarce and are complemented by a generous welfare state. Overall our results indicate a significant, but rather small, effect of number of siblings on mortality.

INTRODUCTION

Recent years have seen researchers across the social sciences directing their attention towards the long-term consequences of early life conditions, such as the in utero environment, birth weight, socioeconomic status in childhood, birth order, and family size. Researchers have previously documented a strong negative association between family size and measures of human capital, such as cognitive skills and education attainment (Steelman et al., 2002; Jaeger, 2008). While there is a growing body of literature examining the relationship between early life conditions and later life mortality (Gluckman et al., 2008; Bengtsson and Broström, 2009; Barclay and Kolk, 2013), there have been very few studies examining the relationship between the number of siblings and longevity. Our paper aims to show whether the necessity to divide the parental resources across a large number of children has a long-lasting effect on the quality and duration of their children's lives (Grundy and Sloggett, 2003; Van den Berg et al., 2009).

Previous studies that examine the way in which family size is related to child health have mainly used contemporary data from low or middle-income countries. While this strand of research provides a number of interesting insights, the results discussed in these studies should be interpreted with reference to the context of harsh economic conditions and limited access to state support. There is little evidence on how family size may affect health in developed, rich countries, and the few available studies show rather mixed results. Moreover, previous studies focused on the health outcomes that can be observed among infants and small children, and hence they provide little evidence on the long term effects of growing up in a large family. The aim of this study is to demonstrate the impact of family size on health in the late life course stage in a context of a rich and developed country with a welfare state system that provides generous support for families with children. Specifically, we examine the impact of family size on mortality using Swedish administrative register data and event history analysis techniques as well as instrumental variable models.

THEORY AND EMPIRICAL RESEARCH

Theory

Several theories have been proposed to account for why children reared in large sibling groups should have poorer later life outcomes, independent of confounding factors such as the socioeconomic status of the parents. These include the resource dilution hypothesis (Blake, 1981) and the model of trade-off between child quality and quantity (Becker & Lewis 1973; Becker & Tomes, 1976), and the hygiene hypothesis (Strachan, 1989).

According to the resource dilution model (Blake, 1981), the development of the child is heavily influenced by the degree of parental resource investment. These resources include not only financial means but also the amount of time and attention paid to the child, which may in turn translate into the child having a lower propensity to engage in risky behaviours and increase the likelihood of the child adopting a healthy life style (Mercy and Steelman, 1982; Evans, 2006). The resource dilution model is also applicable to household space; the negative effect of family size on the physical and mental health of the child may be moderated through household overcrowding (Solari and Mare, 2012; Burström et al., 1999). Living in a home environment that provides insufficient space and privacy for the inhabitants has been shown to raise the level of stress, impede interpersonal relations within family, and to have detrimental consequences for academic performance and health (Evans et al., 1998; Jaine et al., 2011; Solari and Mare, 2012). Moreover, research shows that household overcrowding is a risk factor in the transmission of serious diseases even in developed nations (Baker et al., 2008; Jaine et al., 2011). Exposure to aggressive pathogens early in life may have an overwhelmingly detrimental effect on long term health (Mucci et al., 2004; Bengtsson and Broström, 2009).

Given that parental material and non-material resources are constrained, each additional child decreases the per child investments made by parents (Hertwig et al., 2002). The larger the family, the greater the dilution of resources, and the more limited are the opportunities for the healthy development of a child. Despite the inevitable dilution of resources that comes with increasing family size, it has also

been argued that if the resource dilution hypothesis is true, it would operate through birth order rather than family size (Rodgers 2001). The size of the sibling group is not fixed at birth unless you happen to be the last born child, and a first born in a five-child sibling group will spend the majority of his or her childhood without four other siblings. Previous research indicates that there is an independent effect of birth order on adult mortality (Barclay and Kolk, 2013), and this may mediate the importance of the size of the sibling group.

The predictions of a negative impact of family size on child health can be also derived from the economic literature. According to theoretical models proposed by Becker and Lewis (1973) as well as Becker and Tomes (1976), parents face trade-offs between investing in quantity and quality of children face when making decisions regarding the size of their family. Early life health is a key dimension of investment in offspring's "quality" because regular access to health care, adopting a healthy life style and enjoying favorable home environment have important consequences for future productivity and opportunities of achievement of life goals in aulthood (Case and Paxson 2010; Currie 2000; Currie and Madrian 1999; Yeaung et al. 2002).

Although most interpretations of the resource dilution hypothesis as well as quality-quantity trade-off model focus upon this framework in the context of the childhood experience, it is also possible to extend the application of these theories over a more extended period of the life course. This is particularly clear when considering how any potential inheritance from the parents may be divided amongst the adult siblings. The health of children may also be influenced by the health of their parents. Being raised in a large family necessarily means that the parents have raised a large number of children, and the number of children born and raised has been shown to be associated with mortality risk for both mothers and fathers (Doblhammer, 2000; Grundy and Kravdal, 2008; Grundy, 2009; Hank, 2010). The fact that parents who have had many children have worse health is likely to directly impact the health of their children, as research shows that the degree to which the lifespans of parents and their children overlap has a protective effect on the health of the offspring (Myrskylä and Fenelon, 2012), and that parental death increases the mortality risk of children (Rostila and Saarela, 2011).

However, it is also possible that having a large number of siblings might be beneficial in adulthood. Previous research shows that social support from kin is related to health in adulthood (Rostila et al., 2012). Furthermore, while sibling relationships in adulthood have not been the focus of much research, siblings usually share a common background and history, and this means that sibling relationships are social ties that commonly extend across the life course (Knipscheer and van Tilburg, 2013). Although there is variance in the degree of contact and support that siblings provide to one another in adulthood, which varies by the gender composition of the sibling group as well as age differences (Knipscheer and van Tilburg, 2013), it can be presumed that having a greater number of siblings increases the possibility of being able to seek social and financial support in adulthood if and when that support might be needed.

Another argument for a positive impact of growing up in a large family on health has been formulated within hygiene hypothesis (Strachan, 1989). argues that the number of siblings increases exposure to diseases which are spread by means of human interactions. The hygiene hypothesis has two opposing potential interpretations for the relationship between family size and health. The first predicts a positive influence on later life health. In the case of many exposures, having experienced them early in life contributes to the development of the immune system and diminishes the risk of more severe health problems in adulthood. This can reduce susceptibility to atopic diseases, or hyperallergenic diseases, such as hay fever.

Empirical Research

Previous research on the relationship between family size and health has mainly used contemporary data from low or middle-income countries including Romania, Colombia, China, and Indonesia (Glick et al., 2007; Baez, 2008; Henderson et al., 2008; Rosenzweig and Zhang, 2009; Millimet and Wang, 2011), or historical data (Hart and Smith 2003; Smith et al., 2009; Hatton and Martin, 2010). This strand of literature focuses also on the health outcomes of very young children. The results of these studies generally show that the impact of an additional child in the household negatively affects nutrition (Glick et al., 2007; Henderson et al., 2008), weight

(Rosenzweig and Zhang, 2009) and height (Millimet and Wang, 2011; Hatton and Martin, 2010). Studies using more historical data have also found that an additional sibling in the household is positively associated with mortality (Hart and Smith 2003; Smith et al., 2009). In developing countries, growing up in a large family increases the likelihood of sharing a room and reduces the chance of having access to clean water and sanitary sewer facilities (Baez 2008).

While these studies do demonstrate a negative effect of family size on child health, the context is one of harsh economic conditions and limited access to state support. There is little evidence on how family size may affect health in developed, rich countries, and the few available studies show rather mixed results. On the one hand, research shows that a larger number of siblings increases the risk of tooth loss and periodontal disease (Mucci et al., 2004), injuries at home due to poisoning, falls, scalding and ingestion of foreign objects (Hjern et al 2003), multiple sclerosis (Montgomery et al., 2004) or leukemia (Altieri et al. 2006). Primary care-givers of children living in large households tend to rate the physical health of children lower than parents of children in small families (Solari and Mare 2012). On the other hand, there seems to be an inverse relation with number of siblings of eczema, asthma and wheezing, hay fever and other forms of allergic sensitization (Karmaus and Botezan, 2002). In the Swedish context, unlike in less affuent societies, siblings seem to have a positive effect on child height (Lundborg et al. 2012). Moreover, according to findings reported by Ringbäck et al. (2003), in Sweden the death rate among children living in larger families is lower than among their peers living in small families.

The poor conditions for health in childhood may have profound long term consequences and decrease the quality and duration of life among adults (Case and Paxson 2010). However, given ambiguous conclusions from studies that describe associations between the number of siblings and child health, it is very difficult to predict what is the long term impact of family size on health outcomes in adult life in more developed nations. It is therefore unclear whether growing up in a large family may negatively affect child well-being when parents have adequate resources and access to support from the welfare state system.

DATA, STUDY DESIGN, AND METHODS

Main analyses

We use data from Swedish administrative registers that cover the full population and provide links between children and their biological parents as well as siblings. In total our analysis consists of 2,279,125 men and 2,168,609 women. Swedish administrative register data is overall of very high quality, and linkage proportions between children, parents and siblings are near 1 for the cohorts in our study. The sample consists of individuals born in Sweden whose personal identity number can be linked to the personal identity numbers of their parents and siblings. We link these individuals to their biological children and full siblings through a unique personal identification number. The registers contain information on year and month of birth, which makes it possible to identify children born in multiple births. We base all our family size variables on sets of siblings which all share their biological mother and father. We also exclude all individuals with half siblings, thus restricting our population to sibling sets in which the parents did not have children with other partners. In addition to covariates on baseline hazard (age), cohort, and number of siblings, we additionally include covariates on age of the mother, and birth order in the sibling set.

Our main analyses consists of piece-wise exponential hazard models. We follow all cohorts born in Sweden between 1938-1994, starting at age 20 or year 1991, whichever is later. The nature of Swedish register data puts certain constraints on our selection of cohorts. The linkage between parents and their children is only reliable after 1991. Thus, we can only focus on individuals that survived to that point. We also right censor individuals at first emigration. Descriptive information for men and women, including number of failures, and baseline mortality rates for different covariates are presented and discussed in table 1 in the beginning of the results section.

Causal analyses

Beyond looking at the association between number of siblings and mortality we have also carried out statistical analyses aiming to identify the causal relationship between an additional exogenous sibling and mortality. We do this through the use of econometric models which deal with the endogeneity of family size. One of the most promising approaches has been proposed by Rosenzweig and Wolpin (1980) and named as "twin-first approach". The basic idea is to use the data on multiple births in order to construct a "control group" for parents with a given number of children. As long as decisions on higher party births are generally non-random, parents who experienced multiple births may be regarded as a random "sample" that may be used for comparisons with families that experienced births of singletons. Twin births are an outcome of a random process and not a result of deliberate decisions driven by a calculus considering future child welfare. Thus, information on twin births can be applied to estimate instrumental variable models that reveal the causal effect of the number of siblings on the child health outcomes such as mortality. The twin-first approach proposed by Rosenzweig and Wolpin (1980) can be regarded as comparable to a natural experiment and has opened up new opportunities for research on the consequences of family size (Schulz 2007, Moffit 2005). An important factor of the twin instrumental variable approach is that it measures the effect of an unplanned additional child. Thus, the child is in some sense "unplanned" for which is unlike most children born in contemporary Sweden.

An important challenge that arises using the approach proposed by Rosenzweig and Wolpin (1980) is related to the fact that, as it has been shown in the medical literature; children born in multiple births tend to have lower birth weight which is in turn related to poorer health outcomes later in life. A solution proposed by Black et al. (2010) relies on restricting the sample to the older siblings of children born in the multiple births. Specifically, we construct an indicator of a multiple birth at n birth, limit the analytical sample to children born in families that experienced at least n births and then examine the mortality of children born before the nth birth. In other words, multiple births are used only to construct an instrumental variable, but the mortality of these children is not analysed. Using this restrictive research strategy we run statistical models on the following samples: (1) 1st born in sibling sets of at least 2 siblings, where a birth at parity 2 is the instrumental variable, (2) 1st and 2nd born in sibling sets of at least 3 siblings, where a birth at parity 3 is the instrumental variable, (3) 1st, 2nd, and 3rd born in sibling sets of at least 4 siblings, where a birth at parity 4 is the instrumental variable, and (4) 1st, 2nd, 3rd, and 4th born in sibling sets of at least 5 siblings, where a birth at parity 5 is the instrumental variable.

Our first causal analyses will be based on what is often referred to as the reduced form relationship between an instrumental variable and the outcome, in this case a twin birth and mortality. Thus, we do not aim to identify the effect of exactly one additional sibling, but rather estimate the effect of the natural experiment itself, the exogenous birth of a twin. As a twin birth is a very strong predictor of family size (ranging from 0.75 to 0.9, see Appendix), which means that in practical terms the differences is not very big. This approach has for example been used by (Jacobsen et al 1999, Glick et al 2007). Thus, in a strict sense we are identifying the causal relationship and effect size of a following twin birth and mortality, rather than the relationship between one additional exogenous sibling and mortality. Reduced form estimates have many attractive features, though they are less easily interpreted than traditional IV-estimates (Chernozhukov & Hansen 2008). Our models are similar to previously mentioned survival analyses models and use the same period, cohort, and age restrictions. However, unlike earlier survival analyses these models use the sample restrictions described in the previous paragraph. The effect of an exogenous twin birth on mortality is based on the inclusion of a binary covariate indicating the existence or absence of a following twin birth.

A large number of Swedish, and Scandinavian studies have used and evaluated the exogeneity of a twin birth and found that it meet stringent demands on exogeneity (Black et al 2005, Black et al. 2010, Åslund & Grönqvist 2010, Hirvonen 2009, Holmlund et al 2013, Lundborg et al. 2012). The most important sociodemographic determinant (and potential confounder) of the probability a twin birth, maternal age, is controlled for in our models. We study cohorts born before the introduction of assisted reproductive technologies (the first in vitro fertilization birth in Sweden took place in 1982), as their introduction reduce the degree to which multiple births can be considered an exogenous shock.

We also show results from instrumental variable analyses estimated using two-stage least square procedure (2SLS) as well as instrumental variable probit models estimated with maximum likelihood (results are overall very similar, we will only show results from probit models). Unlike, earlier mentioned these models do estimate the effect of an exogenous increase of sibling size by one. As these models are not

easily estimated by non-linear models we instead model the probability to survive in a 10 year age interval (age 20-30 for cohorts 1972-1977, 30-40 for cohorts 1962-1967, 40-50 for cohorts 1952-1957, and 50-60 for cohorts 1942-1947), using similar period, cohort, and age restrictions as in our survival analyses models. The instrument variable is variable set to unity for multiple births at the nth birth (for n equal to at least two) and zero otherwise. Separate estimations are carried out for children from families with at least n births and for mortality rates in ten years intervals. In the following section we present the results from our main analyses on the relationship between family size and mortality, as well as our causal models estimating the exogenous effect of another sibling.

RESULTS

Table 1 shows summary statistics for women and men based upon the analytical sample population. As can be seen, the rate of mortality for women increases as the size of the sibling group increases from two to eight, before decreasing slightly amongst women from nine- and ten-child sibling groups. Women from a one-child sibling group have a mortality rate approximately equivalent to women from a six-child sibling group. As would be expected, the mortality rate increases with age, and decreases with cohort group. These unadjusted rates shows that mortality increases as the age of the mother at the time of birth increases from 21-25 to >40. Women who were born to mothers aged 16-20 have a mortality rate similar to that of women born to mothers aged 26-30. The mortality rate increases as birth order increases from 2 to 10, but is also relatively high amongst first born women. This is because the category for first born women includes women from a one-child sibling group.

The summary statistics for men show that the mortality rate increases as the size of the sibling group increases from two to seven, before leveling off amongst men from sibling groups with seven to ten children. Men from a one-child sibling group have a mortality rate that is approximately equivalent to men from a six-child sibling group, as was true for women. As the descriptive statistics show for women, the mortality rate amongst men rises with increasing age, and decreases for later born cohort

groups. Men born to mothers aged 26-30 at the time of birth have the lowest mortality rate, and the mortality rate is higher amongst both younger and older mothers, both the increase in the rate is more pronounced amongst the latter. Finally, the mortality rate by birth order shows that the rate increases as birth order increases from two to seven, before decreasing slightly for birth order eight and nine, and increasing again for men with birth order ten. First born men have a mortality rate that is greater than that seen for fourth-born men, though this is also attributable to this category including men from a one-child sibling group.

Table 2 shows the results from piece-wise exponential models examining the relationship between the size of the sibling group and adult mortality for men. Model 1 shows the results without adjusting for birth order. The reference category is men in sibling groups with two children. We find that mortality is 16% higher for men from a one-child sibling group than for men from a two-child sibling group. Mortality is the same for men from a three-child sibling group as for men from a two-child sibling group, but amongst men from larger sibling groups with more than three children, the relative risk increases. Men from a four-child sibling group have a 6% increased relative risk of mortality, men from a seven-child sibling group have a 24% increased relative risk of mortality, and men from a ten-child sibling group have a 21% increased relative risk of mortality. In all comparisons, there is a statistically significant increase in the relative risk of mortality, with the exception of men from three-child sibling groups.

Table 1. Descriptive Statistics for Men and Women.

		Women		Men	
Variable	Category	Failures	Rate	Failures	Rate
Size of the	1	10,063	2.33E-03	15,878	3.57E-03
Sibling Group	2	22,069	1.34E-03	35,313	2.06E-03
	3	16,004	1.40E-03	25,576	2.12E-03
	4	8,226	1.70E-03	13,550	2.69E-03
	5	4,100	2.11E-03	6,666	3.35E-03
	6	2,077	2.31E-03	3,199	3.50E-03
	7	1,051	2.36E-03	1,796	4.00E-03
	8	626	2.78E-03	875	3.84E-03
	9	321	2.72E-03	459	3.99E-03
	10	163	2.64E-03	256	4.00E-03
Age	20-25	1,209	2.19E-04	3,246	5.54E-04
	26-30	1,185	2.53E-04	2,856	5.77E-04
	31-35	1,556	3.25E-04	3,209	6.36E-04
	36-40	2,475	5.08E-04	4,441	8.68E-04
	41-45	4,146	8.37E-04	6,810	1.32E-03
	46-50	7,122	1.47E-03	10,838	2.15E-03
	51-55	10,239	2.40E-03	15,966	3.64E-03
	56-60	12,163	3.73E-03	18,615	5.61E-03
	61-65	12,509	5.60E-03	19,452	8.68E-03
	66-70	9,349	8.23E-03	14,145	1.27E-02
	70-75	2,747	1.07E-02	3,990	1.64E-02
Cohort	1938-1949	43,438	3.95E-03	66,554	5.98E-03
	1950-1959	13,413	1.43E-03	21,557	2.21E-03
	1960-1969	5,221	5.19E-04	9,419	8.91E-04
	1970-1979	1,962	2.75E-04	4,170	5.53E-04
	1980-1989	630	2.07E-04	1,770	5.50E-04
	1990-1994	36	1.35E-04	98	3.47E-04
Mother Age	16-20	4,753	1.51E-03	7,926	2.42E-03
	21-25	17,062	1.43E-03	28,047	2.25E-03
	26-30	19,205	1.45E-03	30,659	2.22E-03
	31-35	13,996	1.76E-03	21,798	2.63E-03
	36-40	7,357	2.05E-03	11,685	3.16E-03
	>40	2,327	2.44E-03	3,453	3.58E-03
Birth Order	1	31,199	1.69E-03	49,948	2.60E-03
	2	20,079	1.44E-03	32,367	2.23E-03
	3	8,426	1.52E-03	13,243	2.31E-03
	4	2,997	1.63E-03	4,852	2.55E-03
	5	1,171	1.78E-03	1,875	2.78E-03
	6	494	1.86E-03	756	2.78E-03
	7	209	1.86E-03	332	2.84E-03
	8	79	1.68E-03	132	2.58E-03
	9	39	1.93E-03	47	2.47E-03
	10	7	1.06E-03	16	2.65E-03
Total		64,700	1.58E-03	103,568	2.44E-03

Table 2. Results from Piece-wise Exponential Models: Swedish Men, Born 1938-1994.

			Model 1			Model 2	
Variable	Category	RR	SE	95%CI	RR	SE	95%CI
Size of the	1	1.16	0.01	1.14-1.19	1.21	0.01	1.18-1.23
Sibling	2	1.00			1.00		
Group	3	1.00	0.01	0.98-1.01	0.97	0.01	0.95-0.99
	4	1.06	0.01	1.04-1.09	1.02	0.01	1.00-1.05
	5	1.15	0.02	1.12-1.18	1.09	0.02	1.06-1.13
	6	1.13	0.02	1.08-1.17	1.06	0.02	1.01-1.10
	7	1.24	0.03	1.17-1.30	1.14	0.03	1.08-1.21
	8	1.16	0.04	1.08-1.24	1.06	0.04	0.98-1.14
	9	1.17	0.06	1.06-1.29	1.06	0.06	0.95-1.18
	10	1.21	0.08	1.06-1.37	1.07	0.07	0.94-1.23
Age	20-25	0.41	0.01	0.39-0.43	0.41	0.01	0.39-0.43
	26-30	0.40	0.01	0.38-0.42	0.40	0.01	0.38-0.42
	31-35	0.41	0.01	0.39-0.42	0.40	0.01	0.39-0.42
	36-40	0.50	0.01	0.49-0.52	0.50	0.01	0.48-0.52
	41-45	0.67	0.01	0.65-0.70	0.67	0.01	0.65-0.69
	46-50	1.00			1.00		
	51-55	1.58	0.02	1.54-1.62	1.58	0.02	1.54-1.62
	56-60	2.35	0.03	2.30-2.41	2.36	0.03	2.30-2.41
	61-65	3.48	0.04	3.40-3.57	3.49	0.04	3.40-3.58
	66-70	5.05	0.07	4.92-5.19	5.08	0.07	4.95-5.22
	70-75	6.47	0.12	6.24-6.72	6.54	0.12	6.30-6.79
Cohort	1938-1949	1.00			1.00		
	1950-1959	0.83	0.01	0.81-0.84	0.82	0.01	0.81-0.84
	1960-1969	0.65	0.01	0.63-0.67	0.65	0.01	0.63-0.67
	1970-1979	0.53	0.01	0.51-0.56	0.53	0.01	0.50-0.55
	1980-1989	0.56	0.02	0.52-0.59	0.55	0.02	0.52-0.59
	1990-1994	0.35	0.04	0.28-0.43	0.35	0.04	0.28-0.43
Mother	16-20	1.19	0.02	1.16-1.22	1.24	0.02	1.21-1.27
Age	21-25	1.07	0.01	1.06-1.09	1.10	0.01	1.08-1.11
	26-30	1.00			1.00		
	31-35	1.01	0.01	0.99-1.03	0.99	0.01	0.98-1.01
	36-40	1.05	0.01	1.02-1.07	1.02	0.01	1.00-1.04
	>40	1.09	0.02	1.05-1.13	1.05	0.02	1.01-1.09
Birth Order	1				1.00		
	2				1.07	0.01	1.05-1.09
	3				1.12	0.01	1.10-1.15
	4				1.12	0.02	1.08-1.16
	5				1.14	0.03	1.08-1.21
	6				1.19	0.05	1.10-1.29
	7				1.24	0.07	1.11-1.40
	8				1.23	0.11	1.03-1.48
	9				1.31	0.20	0.97-1.76
	10				1.52	0.39	0.92-2.52
N			2,279,125			2,279,125	
Deaths			103,568			103,568	
Person-mont	ths		509,828,183			509,828,183	

Table 3. Results from Piece-wise Exponential Models: Swedish Women, Born 1938-1994.

			Model 1			Model 2	
Variable	Category	RR	SE	95%CI	RR	SE	95%CI
Size of the	1	1.14	0.01	1.11-1.17	1.17	0.02	1.14-1.21
Sibling	2	1.00			1.00		
Group	3	1.01	0.01	0.99-1.03	0.98	0.01	0.96-1.00
-	4	1.02	0.01	0.99-1.05	0.98	0.01	0.95-1.01
	5	1.09	0.02	1.06-1.13	1.04	0.02	1.00-1.08
	6	1.13	0.03	1.08-1.18	1.06	0.03	1.00-1.11
	7	1.09	0.04	1.03-1.17	1.01	0.04	0.94-1.08
	8	1.27	0.05	1.17-1.38	1.17	0.05	1.07-1.27
	9	1.20	0.07	1.07-1.34	1.08	0.07	0.95-1.22
	10	1.25	0.11	1.06-1.48	1.14	0.10	0.95-1.36
Age	20-25	0.23	0.01	0.22-0.25	0.23	0.01	0.22-0.25
	26-30	0.25	0.01	0.23-0.27	0.25	0.01	0.23-0.27
	31-35	0.29	0.01	0.28-0.31	0.29	0.01	0.28-0.31
	36-40	0.42	0.01	0.40-0.44	0.42	0.01	0.40-0.44
	41-45	0.62	0.01	0.60-0.65	0.62	0.01	0.60-0.65
	46-50	1.00			1.00		
	51-55	1.54	0.02	1.49-1.59	1.54	0.02	1.49-1.59
	56-60	2.31	0.04	2.24-2.38	2.31	0.04	2.25-2.38
	61-65	3.32	0.05	3.22-3.42	3.32	0.05	3.22-3.43
	66-70	4.82	0.08	4.67-4.99	4.85	0.08	4.70-5.01
	70-75	6.22	0.14	5.94-6.50	6.28	0.15	6.00-6.57
Cohort	1938-1949	1.00			1.00		
	1950-1959	0.83	0.01	0.81-0.85	0.82	0.01	0.80-0.84
	1960-1969	0.68	0.01	0.65-0.70	0.67	0.01	0.64-0.69
	1970-1979	0.58	0.02	0.54-0.61	0.57	0.02	0.54-0.61
	1980-1989	0.52	0.03	0.47-0.58	0.52	0.03	0.47-0.57
	1990-1994	0.35	0.06	0.25-0.49	0.35	0.06	0.25-0.49
Mother	16-20	1.13	0.02	1.10-1.17	1.18	0.02	1.14-1.22
Age	21-25	1.04	0.01	1.02-1.06	1.06	0.01	1.04-1.08
	26-30	1.00			1.00		
	31-35	1.02	0.01	1.00-1.05	1.01	0.01	0.98-1.03
	36-40	1.03	0.01	1.00-1.06	1.00	0.01	0.97-1.03
	>40	1.11	0.02	1.06-1.16	1.08	0.02	1.03-1.12
Birth Order	1				1.00		
	2				1.06	0.01	1.04-1.08
	3				1.13	0.02	1.10-1.17
	4				1.11	0.03	1.06-1.16
	5				1.14	0.04	1.06-1.22
	6				1.19	0.06	1.08-1.31
	7				1.23	0.09	1.07-1.43
	8				1.13	0.13	0.90-1.43
	9				1.44	0.24	1.03-2.00
	10				0.90	0.35	0.42-1.93
N			2,168,609			2,168,609	·
Deaths			64,700			64,700	
Person-mont	ths		490,131,802			490,131,802	

Model 2 shows the results for the relationship between the size of the sibling group

and mortality when adjusting for birth order in addition to the other covariates. As can be seen, the size of the coefficients decreases quite substantially, except for in the case of men from a one-child sibling group, who now have a 21% increased risk of mortality relative to men from a two-child sibling group. Men from a three-child sibling group have a statistically significantly lower relative risk of mortality than men from a two-child sibling group. Men from sibling groups with four or more children still have an elevated risk of mortality relative to men from a two-child sibling group, but the size of the relative risk has decreased in comparison to model 1, and the results are no longer statistically significant for men from sibling groups with greater than seven children. The decrease in the size of the risk after adjusting for birth order is consistent with the theoretical argument made in the introduction that if resource dilution is the underlying explanation for the relationship between the size of the sibling group and mortality, that would be experienced through an interaction of birth order and final size of the sibling group.

Table 3 shows the results for the relationship between the size of the sibling group and mortality for women. Model 1 in Table 3 shows the results without adjusting for birth order. Relative to women from a two-child sibling group, women from a one-child sibling group have a 14% higher risk of mortality. There is no substantive or statistically significant difference for women from three- or four-child sibling groups, but the relative risk is greater for women from sibling groups with five or more children. Women from a five-child sibling group have a 13% higher risk of mortality, women from a eight-child sibling group have a 27% higher risk of mortality, and women from a ten-child sibling group have a 25% higher risk of mortality. Amongst women there is a smaller difference, at least in comparison to individuals from a two-child sibling group, amongst the most common sibling group sizes found in Sweden, i.e. those with 1 to 4 children.

Model 2 in Table 3 shows the results for the relationship between the size of the sibling group and adult mortality after adjusting for birth order. As with the results from Model 1, women from a one-child sibling group have an elevated risk of mortality relative to women from a two-child sibling group. As before there is no statistically significant difference in the relative risk for women in three or four-child sibling groups, and in Model 2 there is no longer a statistically significantly elevated

risk of mortality for women from five-child sibling groups (p=0.06). Women from a six-child sibling group have a statistically significant 6% increased risk of mortality, and women from an eight-child sibling group have a 17% increased risk of mortality, but the difference for women from seven-, nine-, and ten-child sibling groups is not statistically significantly different from that of women from two-child sibling groups. This lack of statistical significance is notable given the very high statistical power in this analysis. Again, these results support the assertion that if resource dilution is the underlying explanation for the relationship between the size of the sibling group and mortality, that would be experienced through the interaction of birth order and the final size of the sibling group.

Figure 1 shows the interaction between the size of the sibling group and the gender composition of the sibling group. The gender composition of the sibling group is split into three categories: all male, all female, or mixed. While the analyses shown in Tables 2 and 3 examined men and women in sibling groups with up to ten children, in these analyses we restrict the maximum size of the sibling group to six. The reason for this is that the number of all-male or all-female sibling groups with more than seven children is very small, meaning that the estimates are somewhat erratic. The probability of having seven male or female children in a row is approximately 0.78%1. As can be seen there is very little difference in the association between the size of the sibling group and mortality by the gender composition of the sibling group.

¹ Using a sex ratio, of 0.5; a more accurate probability would be higher for sons, and lower for daughters.

Figure 1. Interaction between the Size of the Sibling Group and the Gender Composition of the Sibling Group on Mortality for Men and Women.

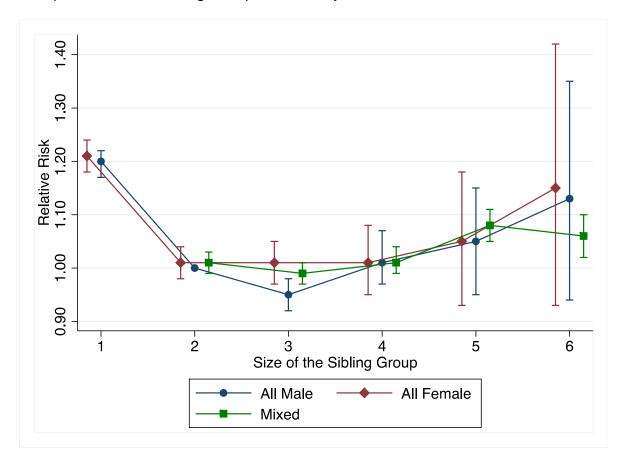


Table 4. Men: Results from Natural Experiment of Twin Birth

			First B	orn	First	and Se	cond Born	Fi	rst, Seco	ond and	First, Second, Third			
		Sample Samp			ole	Th	ird Born	Sample	and Fourth Born Sample					
Variable	Category	RR	SE	95% CI	RR	SE	95% CI	RR	SE	95% CI	RR	SE	95% CI	
Twin Birth	No	1.00			1.00			1.00			1.00			
	Yes	1.02	0.05	0.92-1.14	0.93	0.05	0.83-1.04	0.94	0.07	0.82-1.07	0.83	0.08	0.69-1.00	
Age	20-25	0.42	0.02	0.38-0.46	0.45	0.02	0.40-0.50	0.43	0.04	0.37-0.51	0.42	0.06	0.32-0.54	
	26-30	0.42	0.02	0.38-0.45	0.43	0.02	0.39-0.47	0.44	0.03	0.38-0.51	0.45	0.05	0.36-0.56	
	31-35	0.42	0.02	0.38-0.45	0.43	0.02	0.40-0.47	0.43	0.03	0.38-0.49	0.51	0.05	0.43-0.60	
	36-40	0.51	0.02	0.47-0.54	0.52	0.02	0.49-0.56	0.49	0.02	0.44-0.54	0.54	0.04	0.47-0.62	
	41-45	0.69	0.02	0.65-0.73	0.71	0.02	0.67-0.76	0.70	0.03	0.65-0.76	0.75	0.04	0.67-0.83	
	46-50	1.00			1.00			1.00			1.00			
	51-55	1.65	0.04	1.58-1.72	1.64	0.04	1.57-1.72	1.61	0.05	1.52-1.70	1.66	0.07	1.53-1.80	
	56-60	2.43	0.05	2.33-2.54	2.39	0.06	2.28-2.50	2.31	0.07	2.18-2.45	2.36	0.10	2.18-2.56	
	61-65	3.63	0.08	3.47-3.80	3.59	0.08	3.42-3.76	3.41	0.10	3.22-3.61	3.52	0.14	3.25-3.81	
	66-70	5.42	0.13	5.17-5.68	5.38	0.13	5.13-5.65	5.12	0.16	4.82-5.44	4.89	0.21	4.50-5.32	
	70-75	7.03	0.22	6.61-7.47	6.72	0.22	6.30-7.16	6.18	0.26	5.70-6.70	6.34	0.35	5.69-7.07	
Cohort	1938-1949	1.00			1.00			1.00			1.00			
	1950-1959	0.80	0.01	0.78-0.83	0.82	0.01	0.79-0.85	0.84	0.02	0.80-0.87	0.82	0.02	0.78-0.87	
	1960-1969	0.60	0.02	0.57-0.64	0.61	0.02	0.58-0.65	0.63	0.03	0.58-0.68	0.69	0.04	0.61-0.78	
	1970-1979	0.49	0.02	0.45-0.53	0.52	0.02	0.47-0.57	0.55	0.04	0.48-0.63	0.61	0.07	0.50-0.76	
	1980-1989	0.53	0.03	0.47-0.59	0.50	0.03	0.45-0.57	0.51	0.05	0.42-0.62	0.52	0.08	0.39-0.70	
	1990-1994	0.37	0.06	0.26-0.52	0.28	0.06	0.18-0.43	0.30	0.10	0.15-0.58	0.36	0.19	0.13-1.00	
Mother	16-20	1.29	0.02	1.24-1.33	1.27	0.02	1.23-1.32	1.19	0.03	1.13-1.25	1.18	0.04	1.10-1.26	
Age	21-25	1.11	0.01	1.08-1.14	1.13	0.02	1.10-1.16	1.12	0.02	1.08-1.16	1.10	0.03	1.04-1.15	
	26-30	1.00			1.00			1.00			1.00			
	31-35	1.05	0.02	1.01-1.09	1.01	0.02	0.98-1.05	1.01	0.02	0.96-1.05	1.00	0.03	0.94-1.06	
	36-40	1.07	0.04	1.00-1.14	1.08	0.04	1.01-1.16	1.00	0.04	0.93-1.08	1.06	0.05	0.97-1.16	
	>40	1.21	0.14	0.96-1.53	1.22	0.13	0.99-1.50	1.29	0.14	1.04-1.60	1.16	0.16	0.89-1.51	
N			798,6	521		650,3	396		297,6	503		131,6	517	

Table 5. Women: Results from Natural Experiment of Twin Birth

			First B	orn	First	and Se	cond Born	Fi	rst, Seco	ond and	First, Second, Third			
			Sam	ole		Sam	ple	Th	ird Born	Sample	and F	ourth B	orn Sample	
Variable	Category	RR	SE	95% CI	RR	SE	95% CI	RR	SE	95% CI	RR	SE	95% CI	
Twin Birth	No	1.00			1.00			1.00			1.00			
	Yes	1.07	0.07	0.94-1.22	1.01	0.07	0.88-1.16	1.09	0.09	0.93-1.29	1.11	0.12	0.91-1.37	
Age	20-25	0.20	0.01	0.17-0.23	0.24	0.02	0.20-0.28	0.25	0.03	0.20-0.32	0.31	0.06	0.21-0.45	
	26-30	0.25	0.02	0.22-0.28	0.28	0.02	0.24-0.32	0.28	0.03	0.23-0.35	0.32	0.05	0.23-0.44	
	31-35	0.30	0.02	0.27-0.33	0.32	0.02	0.29-0.36	0.30	0.03	0.25-0.36	0.29	0.04	0.22-0.38	
	36-40	0.43	0.02	0.40-0.47	0.46	0.02	0.42-0.50	0.44	0.03	0.39-0.50	0.48	0.05	0.39-0.57	
	41-45	0.63	0.02	0.59-0.68	0.63	0.02	0.58-0.67	0.62	0.03	0.56-0.69	0.58	0.04	0.50-0.67	
	46-50													
	51-55	1.55	0.04	1.47-1.64	1.52	0.04	1.43-1.61	1.54	0.06	1.43-1.65	1.63	0.09	1.47-1.81	
	56-60	2.33	0.06	2.20-2.46	2.37	0.07	2.24-2.51	2.32	0.09	2.15-2.49	2.42	0.12	2.19-2.68	
	61-65	3.38	0.10	3.19-3.57	3.48	0.10	3.28-3.68	3.50	0.13	3.25-3.77	3.65	0.19	3.30-4.04	
	66-70	5.06	0.15	4.78-5.36	4.97	0.15	4.68-5.28	5.00	0.20	4.63-5.40	5.39	0.29	4.86-5.98	
	70-75	6.45	0.25	5.97-6.96	6.49	0.26	5.99-7.02	6.58	0.33	5.97-7.27	6.64	0.45	5.81-7.59	
Cohort	1938-1949													
	1950-1959	0.81	0.02	0.78-0.84	0.85	0.02	0.82-0.89	0.87	0.02	0.82-0.92	0.87	0.03	0.81-0.94	
	1960-1969	0.64	0.02	0.60-0.69	0.68	0.03	0.63-0.73	0.76	0.04	0.69-0.84	0.80	0.06	0.68-0.94	
	1970-1979	0.60	0.03	0.54-0.67	0.56	0.04	0.50-0.64	0.52	0.05	0.43-0.64	0.46	0.08	0.33-0.65	
	1980-1989	0.57	0.05	0.48-0.68	0.54	0.05	0.44-0.65	0.55	0.08	0.42-0.73	0.51	0.11	0.33-0.79	
	1990-1994	0.49	0.13	0.29-0.83	0.43	0.14	0.23-0.80	0.39	0.20	0.14-1.06	0.20	0.20	0.03-1.44	
Mother	16-20	1.19	0.03	1.14-1.24	1.20	0.03	1.14-1.26	1.18	0.04	1.10-1.25	1.17	0.05	1.08-1.28	
Age	21-25	1.05	0.02	1.02-1.09	1.08	0.02	1.04-1.12	1.07	0.02	1.03-1.12	1.09	0.03	1.02-1.15	
	26-30													
	31-35	1.04	0.03	0.99-1.09	1.01	0.02	0.96-1.06	1.02	0.03	0.97-1.08	1.06	0.04	0.99-1.14	
	36-40	1.07	0.05	0.99-1.17	1.05	0.04	0.97-1.15	0.98	0.05	0.90-1.08	0.94	0.06	0.84-1.06	
	>40	1.12	0.16	0.84-1.49	1.22	0.17	0.94-1.60	0.81	0.14	0.58-1.14	1.06	0.19	0.75-1.49	
N			758,3	358		611,8	382		284,6	553		128,9	907	

Tables 4 (men) and 5 (women) shows the results from models examining whether a twin birth has an effect on mortality for the preceding children. In earlier models we are not able to clearly distinguish between the effect of number of siblings, from other socioeconomic and sociodemographic factors that are associated with number of siblings. In Tables 4 and 5 we look at how a random exogenous shock to a person's sibling size, the birth of a later born twin, affects mortality. These models answer a different question, the effect on mortality of an additional sibling per se, but lack the statistical power of our earlier analysis due to the relative scarcity of twins in our population.

We use several different samples due to the previously described research design. Our first born sample consists of first born men from a sibling group with two or more children. The first and second born sample consists of first and second born men from a sibling group with three or more children. The first, second and third born sample consists of first, second, and third born sons from a sibling group with four or more children. Finally, the first, second, third and fourth born sample consists of first to fourth born men from sibling groups with five or more children. The binary variable for whether there was a twin birth indicates whether there was a twin birth directly

after the birth of the individuals included in the sample. So, for the first born sample, if there was a twin birth, this meant that the twin birth was the second birth. In the sample of first, second and third borns, a twin birth means that the fourth birth was a twin birth. These samples will be referred to by the birth order of the index population, e.g. the population of first born, in families with at least two children, where we look at a twin birth at parity 2 will be referred to as the first born sample.

Table 4 shows that a twin birth had no substantial impact upon the mortality of the earlier born sons. In fact, the relative risk is lower for all sample groups except the first born sample, though it is not statistically significant. Table 5 shows that a twin birth increased the relative risk of mortality for individuals born prior to the twin, but that the difference from those individuals who did not experience a twin birth in their sibling group was not statistically significant. Overall, while these analyses are lacking in statistical power despite the use of the complete Swedish population (due to the small number of twin births), our results are consistent with the earlier finding of weak or no causal effect of an additional sibling on one's own mortality. For men we can rule out a moderate to strong positive effect of an exogenous sibling on mortality, though our estimates are not very precise. For women, our results are more ambiguous and we are unable to make any speculation about the relationship between the experience of a twin birth and mortality. Because of the specific nature of these samples, we also estimate models similar to our main analyses for the specific analytical sample used to test whether a twin birth has an association with mortality. These results are shown in the appendices, in Tables S1 and S2.

We additionally run a number of traditional two-stage least square (2SLS) instrumental variable probit models. These models are not compatible with our earlier non-linear regressions, and we thus use a binary variable on survival from age 30-40, 40-50, 50-60, and 60-70. We used the same research design as earlier, with first born with at least one additional sibling, and a twin at parity 2 as an instrumental variable, etc. We show the results from these models in Table 6. The full results table, including first stage estimates and non-instrumental ordinary least square (OLS) models, can be found in Appendix QWERTY. Our probit 2SLS results are ambiguous, largely insignificant, and are broadly consistent with the lack of a causal effect in our results from our survival analysis models, presented in Tables 4 and 5.

Table 6. Results from Two-stage Least Square (2SLS) probit models, Men and Women.

women	• 			1			
		Men				Women	
Sample	1st born	1st & 2nd born	1st, 2nd, & 3rd born		1st born	1st & 2nd born	1st, 2nd, & 3rd born
IV	Twin at 2	Twin at 3	Twin at 4		Twin at 2	Twin at 3	Twin at 4
			death between	30	-40		
coefficient	0.240	0.009	0.201		-1.096***	-0.047	0.042
SE	(0.159)	(0.165)	(0.212)		(0.008)	(0.252)	(0.438)
95% CI.	-0.072 - 0.552	-0.314 - 0.332	-0.215 - 0.617		-1.1111.081	-0.540 - 0.447	-0.817 - 0.900
			death between	 40	 -50		
coefficient	0.217	-0.139	-0.171		-0.156	-0.278	-0.262
SE	(0.146)	(0.154)	(0.236)		(0.258)	(0.247)	(0.361)
95% CI.	-0.069 - 0.502	-0.442 - 0.163	-0.633 - 0.292		-0.663 - 0.350	-0.762 - 0.206	-0.971 - 0.446
			death between	50	 -60		
coefficient	-0.169	0.056	-0.035		0.093	-0.081	0.077
SE	(0.147)	(0.103)	(0.137)		(0.167)	(0.143)	(0.150)
95% CI.	-0.458 - 0.120	-0.147 - 0.258	-0.305 - 0.234		-0.234 - 0.419	-0.362 - 0.200	-0.217 - 0.371
	ļ		death between	60	 - 70		
coefficient	-0.119	0.108	0.075		-0.126	0.275***	0.014
SE	(0.090)	(0.071)	(0.078)		(0.103)	(0.081)	(0.105)
95% CI.	-0.295 - 0.057	-0.030 - 0.247	-0.077 - 0.227		-0.328 - 0.076	0.116 - 0.435	-0.191 - 0.219

DISCUSSION

Economic, sociological and epidemiological literature provides a number of arguments for a disadvantage of growing up in a large family. Having many siblings implies the necessity to compete for parental financial resources, time and attention. Moreover, it increases the risk of infections early in life, which may have long lasting damaging consequences for health outcomes in adulthood. Previous research has tested these theoretical ideas using data for low- or middle-income countries where the welfare state provides little support for families. There is little evidence on how family size may affect health in developed, rich countries, and the few available studies show rather mixed results. Moreover, previous research on the effects of family structure examined health of infants and young children, which opens the question whether the effects of growing up in a large family are persistent and can be observed also in the later life course stages.

In this paper, we focus on the impact of family size on mortality, which reflects the long term health consequences of early life conditions. We carry out our study using data for Sweden, one of the riches countries of the world, where parents with children are provided with very generous support from the welfare state. Regardless of social and ethnic background, families with children may take advantage of a publicly funded early child education and care system as well as health care services that are free of charge for children. We argue that in such institutional setting, parents may not necessarily face a strong trade-off between having many children and providing each child with favorable conditions for living in good health.

Indeed, our results do not support the idea of a strong negative impact of family size on mortality. While we do observe some disadvantage of children born at higher parities, after controlling for birth order the effect of family size is not statistically significant among women except at high parities, and it is of small to moderate size for men. We also provide results from models that control for possible confounders using information on multiple births that cause an exogenous shock to family size and exogenously shift the number of siblings. These estimates on the exogenous effect of an additional sibling suggest no impact of growing up in a larger family among women and less clearly for men.

Summing up, according to our findings, even if having many siblings does have a detrimental effect on child health, this impact is not long lasting and universal. The mechanisms causing a disadvantage of children raised in large families seem to be primarily at work only in countries where families receive only limited support from the welfare state.

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APPENDICES

Table S1. Men: Population from Natural Experiment of Twin Birth

	Category		First Bo Samp		Fi	rst and Seco			First, Secon		First, Second, Third and Fourth Born Sample		
Variable		RR	SE	95% CI	RR	SE	95% CI	RR	SE	95% CI	RR	SE	95% CI
Size of the	2	0.89	0.02	0.84-0.94									
Sibling Group	3	0.87	0.02	0.82-0.92	0.89	0.02	0.86-0.93						
	4	0.93	0.03	0.87-0.99	0.95	0.02	0.91-0.99	0.94	0.02	0.91-0.97			
	5	1.00			1.00			1.00			1.00		
	6	0.88	0.05	0.80-0.98	0.93	0.03	0.87-1.00	0.95	0.03	0.89-1.00	0.95	0.02	0.91-1.00
	7	1.11	0.07	0.98-1.26	1.11	0.05	1.02-1.22	1.06	0.04	0.98-1.14	1.06	0.04	1.00-1.14
	8	0.99	0.10	0.81-1.21	1.01	0.07	0.88-1.15	1.01	0.06	0.91-1.13	0.99	0.05	0.90-1.09
	9	0.91	0.14	0.67-1.23	1.02	0.11	0.83-1.25	1.13	0.09	0.97-1.32	1.02	0.07	0.89-1.17
	10	1.07	0.22	0.72-1.58	1.14	0.15	0.87-1.48	0.99	0.11	0.80-1.22	0.91	0.09	0.76-1.10
Age	20-25	0.42	0.02	0.38-0.46	0.45	0.02	0.40-0.50	0.43	0.04	0.36-0.51	0.43	0.06	0.33-0.55
· ·	26-30	0.41	0.02	0.38-0.45	0.43	0.02	0.39-0.47	0.44	0.03	0.38-0.51	0.45	0.05	0.36-0.56
	31-35	0.42	0.02	0.39-0.45	0.43	0.02	0.40-0.47	0.43	0.03	0.39-0.49	0.51	0.05	0.43-0.61
	36-40	0.51	0.02	0.47-0.54	0.52	0.02	0.49-0.56	0.49	0.02	0.44-0.54	0.54	0.04	0.47-0.62
	41-45	0.69	0.02	0.65-0.73	0.71	0.02	0.67-0.75	0.70	0.03	0.65-0.76	0.74	0.04	0.66-0.83
	46-50	1.00			1.00			1.00			1.00		
	51-55	1.64	0.04	1.57-1.71	1.63	0.04	1.56-1.71	1.61	0.05	1.52-1.71	1.67	0.07	1.54-1.81
	56-60	2.42	0.05	2.31-2.52	2.38	0.06	2.28-2.49	2.31	0.07	2.18-2.44	2.36	0.10	2.18-2.56
	61-65	3.62	0.08	3.47-3.79	3.58	0.08	3.42-3.75	3.41	0.10	3.21-3.61	3.53	0.14	3.26-3.82
	66-70	5.39	0.13	5.15-5.65	5.37	0.13	5.12-5.63	5.12	0.16	4.82-5.45	4.92	0.21	4.52-5.35
	70-75	7.01	0.22	6.59-7.46	6.68	0.22	6.26-7.13	6.22	0.26	5.73-6.74	6.40	0.35	5.74-7.13
Cohort	1938-1949	1.00			1.00			1.00			1.00		
	1950-1959	0.80	0.01	0.78-0.83	0.82	0.01	0.80-0.85	0.84	0.02	0.80-0.87	0.82	0.02	0.77-0.87
	1960-1969	0.61	0.02	0.58-0.64	0.62	0.02	0.59-0.66	0.63	0.03	0.58-0.68	0.68	0.04	0.60-0.77
	1970-1979	0.49	0.02	0.45-0.53	0.52	0.02	0.48-0.57	0.55	0.04	0.48-0.63	0.62	0.07	0.50-0.76
	1980-1989	0.53	0.03	0.48-0.60	0.51	0.03	0.45-0.58	0.52	0.05	0.43-0.62	0.51	0.08	0.38-0.69
	1990-1994	0.37	0.06	0.27-0.52	0.29	0.06	0.19-0.44	0.30	0.10	0.15-0.59	0.36	0.18	0.13-0.98
Mother Age	16-20	1.27	0.02	1.22-1.31	1.29	0.03	1.24-1.34	1.23	0.03	1.17-1.30	1.25	0.05	1.16-1.36
.	21-25	1.10	0.01	1.07-1.13	1.13	0.02	1.10-1.16	1.14	0.02	1.10-1.18	1.13	0.03	1.07-1.19
	26-30	1.00			1.00		0.00-0.00	1.00			1.00		
	31-35	1.05	0.02	1.01-1.09	1.01	0.02	0.98-1.05	1.00	0.02	0.95-1.04	0.98	0.03	0.92-1.04
	36-40	1.07	0.04	1.00-1.15	1.08	0.04	1.01-1.15	0.98	0.04	0.91-1.06	1.03	0.05	0.94-1.13
	>40	1.12	0.14	0.88-1.43	1.23	0.13	1.00-1.52	1.22	0.14	0.98-1.52	1.14	0.15	0.87-1.48
Birth Order	1				1.00			1.00			1.00		0.00-0.00
	2				1.06	0.01	4.74-1.08	1.04	0.02	1.01-1.08	1.04	0.03	0.98-1.10
	3				2.00	0.02	2.00	1.11	0.02	1.06-1.15	1.11	0.04	1.04-1.18
	4								J.J_	1.00 1.10	1.11	0.04	1.03-1.19
N			798,62	71		650,39	96		297,60)3		131,61	

Table S2. Women: Population from Natural Experiment of Twin Birth

			First Born Sample			irst and Seco			First, Secon		First, Second, Third and Fourth Born Sample		
Variable	Category	RR	SE	95% CI	RR	SE	95% CI	RR	SE	95% CI	RR	SE	95% CI
Size of the	2	0.93	0.03	0.86-0.99									
Sibling Group	3	0.92	0.03	0.86-0.98	0.92	0.02	0.87-0.97						
	4	0.90	0.04	0.84-0.98	0.91	0.03	0.86-0.96	0.93	0.02	0.89-0.98			
	5	1.00			1.00			1.00			1.00		
	6	1.10	0.07	0.98-1.24	1.06	0.05	0.97-1.15	1.04	0.04	0.97-1.12	1.04	0.03	0.98-1.11
	7	0.88	0.08	0.73-1.06	0.91	0.06	0.80-1.03	0.96	0.05	0.87-1.05	0.97	0.04	0.89-1.05
	8	1.00	0.13	0.78-1.29	1.01	0.09	0.85-1.20	1.07	0.07	0.93-1.22	1.08	0.06	0.97-1.21
	9	1.40	0.23	1.02-1.92	1.05	0.13	0.82-1.35	1.14	0.11	0.95-1.37	1.09	0.09	0.93-1.27
	10	1.48	0.33	0.96-2.29	1.21	0.22	0.85-1.72	1.17	0.17	0.88-1.56	1.23	0.15	0.97-1.55
Age	20-25	0.20	0.01	0.17-0.23	0.24	0.02	0.20-0.28	0.26	0.03	0.20-0.33	0.31	0.06	0.21-0.45
	26-30	0.25	0.02	0.22-0.28	0.28	0.02	0.24-0.32	0.28	0.03	0.22-0.35	0.32	0.05	0.23-0.44
	31-35	0.30	0.02	0.27-0.33	0.32	0.02	0.29-0.36	0.30	0.03	0.25-0.36	0.29	0.04	0.22-0.38
	36-40	0.43	0.02	0.40-0.47	0.46	0.02	0.42-0.51	0.44	0.03	0.39-0.51	0.47	0.05	0.39-0.57
	41-45	0.63	0.02	0.59-0.68	0.63	0.02	0.58-0.67	0.62	0.03	0.56-0.69	0.58	0.04	0.50-0.67
	46-50	1.00			1.00			1.00			1.00		
	51-55	1.54	0.04	1.46-1.63	1.51	0.04	1.43-1.60	1.54	0.06	1.43-1.65	1.64	0.09	1.47-1.81
	56-60	2.32	0.06	2.19-2.44	2.36	0.07	2.23-2.50	2.32	0.09	2.16-2.49	2.43	0.12	2.20-2.68
	61-65	3.35	0.09	3.17-3.54	3.48	0.10	3.29-3.69	3.50	0.13	3.25-3.76	3.65	0.19	3.30-4.04
	66-70	5.03	0.15	4.75-5.33	4.96	0.15	4.67-5.27	4.99	0.20	4.63-5.39	5.41	0.29	4.87-6.00
	70-75	6.42	0.25	5.95-6.92	6.44	0.26	5.95-6.98	6.55	0.33	5.94-7.23	6.65	0.45	5.82-7.60
Cohort	1938-1949	1.00			1.00			1.00			1.00		
	1950-1959	0.81	0.02	0.78-0.84	0.85	0.02	0.82-0.89	0.86	0.02	0.82-0.91	0.88	0.03	0.82-0.95
	1960-1969	0.64	0.02	0.60-0.69	0.68	0.03	0.63-0.73	0.76	0.04	0.68-0.84	0.80	0.06	0.69-0.94
	1970-1979	0.60	0.03	0.54-0.67	0.56	0.04	0.50-0.64	0.53	0.05	0.43-0.64	0.47	0.08	0.34-0.65
	1980-1989	0.57	0.05	0.48-0.68	0.53	0.05	0.44-0.65	0.55	0.08	0.41-0.73	0.51	0.11	0.33-0.79
	1990-1994	0.48	0.13	0.28-0.81	0.43	0.14	0.23-0.80	0.38	0.20	0.14-1.04	0.20	0.20	0.03-1.45
Mother Age	16-20	1.18	0.03	1.13-1.23	1.21	0.03	1.15-1.27	1.24	0.04	1.15-1.32	1.21	0.06	1.09-1.34
	21-25	1.05	0.02	1.02-1.09	1.08	0.02	1.05-1.12	1.10	0.03	1.05-1.15	1.10	0.04	1.03-1.17
	26-30	1.00			1.00			1.00			1.00		
	31-35	1.04	0.03	0.99-1.09	1.01	0.02	0.96-1.06	1.02	0.03	0.96-1.08	1.07	0.04	0.99-1.15
	36-40	1.08	0.05	0.99-1.18	1.06	0.05	0.98-1.15	0.96	0.05	0.87-1.06	0.95	0.06	0.84-1.08
	>40	1.14	0.16	0.86-1.51	1.27	0.17	0.97-1.65	0.83	0.14	0.60-1.16	1.11	0.20	0.78-1.58
Birth Order	1				1.00			1.00			1.00		
	2				1.05	0.02	3.19-1.08	1.05	0.03	1.01-1.10	1.01	0.04	0.94-1.09
	3							1.14	0.03	1.08-1.20	1.07	0.04	0.98-1.15
	4										1.03	0.05	0.94-1.13
N			758,35	58		611,88	32		284,65	53		128,90	