Sex of the first child and mental health of the parents later in life. A natural experiment.

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Abstract

This study investigates whether the sex of the firstborn child has a causal effect on parental cognitive decline in later life. Leveraging a standard natural experiment design, we treat the sex of the firstborn as a natural randomizer, which allows for comparisons across groups while controlling for both observed and unobserved confounders. Using data from the Survey on Health and Retirement in Europe (SHARE) concerning over 60,000 individuals, we find that having a firstborn son is associated with significantly worse outcomes in immediate and delayed word recall, verbal fluency (among fathers), depression, and perceived quality of life. The effect sizes are small but statistically robust. In addition to identifying the overall effect, we explore potential causal mechanisms using a causal triangulation strategy. We find no evidence supporting biological or marital-status-based pathways. Instead, our findings suggest that the observed effect is primarily mediated by two factors: the higher mortality risk of sons and the more frequent contact between daughters and their aging parents. These results indicate that cognitive functioning in old age may be shaped by the accumulation of social and emotional shocks across the life course. Our study contributes to the literature on family structure and aging, and highlights the value of natural experiments in causal inference.

1. Introduction

The link between fertility and health of the parents has been extensively investigated. Most of the studies so far have focused on the effect of the number of children on parents' mortality and physical health. Recently, growing attention has been devoted to the effects of the number of children on mental health, particularly on cognitive decline (Bonsang and Skirbekk, 2022, Read and Grundy, 2017, Saenz et al., 2021), backed up by a rich biological literature examining the effect of childbirth on the brain structure, function and aging process among mothers (de Lange et al., 2019, de Lange et al., 2020, Kim et al., 2010, Luders et al., 2020, Pawluski et al., 2016), fathers (Kim et al., 2014) and both parents (Rotondi 2024).

No matter what the outcome of interest, the main focus has been on the number of children. The genesis of this approach can be identified in the evolutionary theories that predict a trade-off between survival and reproduction: individuals who invest more resources in fertility would have fewer resources for maintenance and survival, and vice versa (Ellison and Ellison, 2009, Kirkwood, 1977, Kirkwood and Rose, 1991, Westendorp and Kirkwood, 1998). But, while evidence for such a trade-off exists for different animals and plants (Golet et al., 2004, Lester et al., 2004, Obeso, 2002, Partridge et al., 1987), for humans, evidence is inconclusive. Some studies identify a cost of reproduction in terms of a shorter survival (Doblhammer and Oeppen, 2003, Doblhammer et al., 2000, Gagnon et al., 2009, Smith et al., 2002, Barclay and Kolk, 2019, Bolund et al., 2016), accelerated aging (Pollack et al., 2018, Ryan et al., 2018) or adverse health (McArdle et al., 2006, Mueller et al., 2013). Other studies, on the contrary, find either no effect (Helle et al., 2002), or a negative effect only on modern populations (Le Bourg, 2007), for parity above five (Zeng et al., 2016) or for mothers in lower social classes (Dribe, 2004). Other studies find that higher parity might even have protective health effects (Müller et al., 2002, Sear, 2007, Aviv, 2004, Barclay et al., 2016, Barha et al., 2016, Grundy and Kravdal, 2010). Finally, some analyses found that more than the number of children, it is rather fertility timing and age at first birth that matter the most in terms of health outcome of the parents later in life (Reibling and Möhring, 2018, Sironi, 2019).

The conflicting results might depend on the intrinsic difficulty of measuring trade-offs in human life history research and testing the association between fertility and health, and mortality of the parents (Bolund, 2020, Gagnon et al., 2009, Jasienska, 2020) because of a number of factors such as how confounding variables are accounted for (Hurt et al., 2006), reverse causality and selection of healthier individuals into parenthood. This is particularly true for women, as their ability to give birth could be an indicator of possessing an higher-than-the-average health (Hyppönen et al., 2005, Ronsmans et al., 2001), which would exhibit a selection bias in the estimation.

Another confounding factor, so far under-investigated, could be the sex of the children. There is a lack of studies on the effect of the sex of the child on the health of the mothers (and of parents, in general), but the few analyses that exist indicate this variable might have a differential effect. A study on the number and sex of the offspring in bighorn sheep, a

mammalian species characterized by pronounced male-biased sexual dimorphism, found that among the females with the same number of offspring, those who weaned more males showed an accelerated aging than those who weaned more females (Douhard et al., 2020). Also among humans sons appear to be more costly than daughters for the mothers, both in terms of decline of the self-perceived health (Galbarczyk et al., 2019) and of increase of aging-related inflammation markers (Galbarczyk et al., 2021). These results suggest sons might be more energetically and immunologically demanding than daughters, in both pregnancy and lactation, but some scholars highlight the possibly equally important role of social mechanisms such as gendered differences in the type and amount of stress, social support, and caregiving to and from sons and daughters (Thomas and Thomeer, 2019).

To our knowledge, the relationship between the sex of the children and cognitive health of the parents has not been systematically explored at the population level. The paucity of such studies is rather surprising, given the rich biological literature examining the effect of childbirth on the brain functioning (mentioned above) and the recently emerging evidence that the sex of the fetus may affect differently the process of cognitive decline of the mothers and ultimately the development of Alzheimer diseases at the molecular level, through mechanisms such as the production of inflammatory cytokines and micro-chimerism (Duarte-Guterman et al., 2019), in ways that yet need to be fully uncovered.

The investigation of the molecular level is not the object of our analysis. The focus of our analysis is, instead, the population level, where we want to understand if and how the sex of the child affects the cognitive health of the parents later in life.

One reason for focusing on the effect of children sex comes from the fact that parents in developed countries generally do not select the sex of their children (whereas they may select their number). This means that the sex of children, and particularly the sex of the first-born child, as we will see, produces a spontaneous randomization of parents into a control and treated group. Thanks to this fact, the multitude of factors that may potentially perturb the causal connection between children sex and parents' cognitive health are, thus, spontaneously controlled for.

The causal pathways connecting the sex of the children to physical and cognitive health of the parents appear similar to those hypothesized in the case of the relationship between fertility and mental health.

Sons are energetically more costly than daughters. According to the disposable soma theory (Westendorp and Kirkwood, 1998), given the same number of children, mothers with more sons would show a faster ageing process than mothers with more daughters. There is evidence that hormone concentrations in pregnancy differ according to the sex of the fetus as early as three weeks after fertilization (Yaron et al., 2002) and such difference could translate into different effects on the health of the mother. Furthermore, having a son, compared to having a daughter, significantly increases the probability to suffer from miscarriage or complications in the following pregnancy (Christiansen et al., 2004). Both are traumatic and stressful events that could affect the health of the parents, both physical and mental. Finally, the sex of the fetus could affect the risk of developing Alzheimer disease (Duarte-Guterman et al., 2019).

Sons are more physically active and turbulent than daughters (Campbell and Eaton, 1999, Eaton and Enns, 1986), already in the womb (Ellis and He, 2014). Boys are also more likely to be diagnosed with ADHD than girls (Ellis et al., 2013). It is reasonable to hypothesize that this may affect parents' cognitive functioning through higher levels of stress and sleep deprivation and lower time devoted to leisure activities.

The likelihood of death is higher for sons than for daughters. This is due both to the overall male higher mortality (Austad, 2006, Austad, 2011, Austad and Fischer, 2016, Zarulli et al., 2021) and to the higher recklessness and tendency to engage in risky behaviors, typical of young males (Legato, 2008, Wilson and Daly, 1985, Byrnes et al., 1999, Geary, 2010). Biological factors (Natterson-Horowitz and Bowers, 2019) and social norms (Courtenay, 2000) may impel males to more risky behaviors. Whatever the reason, this means that parents of boys, more than parents of girls, are at a higher risk of experiencing the emotional shock represented by the loss of a child. This may favor, later in life, cognitive decline through the depression caused by the fateful event. Even though the relationship between depression and dementia is complex and still not well understood, there is convincing evidence that early life depression can act as a risk factor for later life dementia, while later life depression may be associated with dementia and can be seen as a prodrome to dementia (Bennett and Thomas, 2014, Byers and Yaffe, 2011).

The sex of the first-born child has been found to be associated to parents' marital status and family structure. Having a first-born daughter increases the likelihood for a woman to live without a partner (Dahl and Moretti, 2008), to not receive enough support from the partner and to live in an unsatisfactory relationship (Raley and Bianchi, 2006). The combination of these factors may be associated with later cognitive decline, because of the protective effect of partnership and higher risk of falling below the poverty line for single parents, especially mothers.

The sex of the children might also affect the parents' health later in life due to different care offered to the parents by sons and daughters. There is evidence that sons offer smaller support to their elderly parents than what daughters do (Wolf et al., 1997, Xie and Zhu, 2009, Yi et al., 2016, Silverstein et al., 2006), even though the higher involvement of daughters in parent care can be partially explained by the fact that adult children are more likely to provide care to a parent of the same gender, when the substantial majority of elderly parents requiring care are mothers (Lee et al., 1993).

Another possible way through which the sex of the children can influence the health status of their elderly parents is when their adult children become parents themselves. Taking care of the grandchildren has a protective effect against cognitive decline (Arpino and Bordone, 2014, Bordone and Arpino, 2019, Bordone and Weber, 2012), but since a daughter is more likely to be close to and to involve more her parents in the care of her children, parents of female children may benefit more from the tending role to their grandchildren than parents of male children.

By examining the sex of children as a determinant of parental cognitive aging, this study extends theoretical perspectives in both life course analysis and evolutionary demography.

It broadens the scope of existing research by looking not just at how many children parents have, but at how differences among children—particularly their sex—may shape long-term health trajectories. This approach contributes to refining theories such as the disposable soma theory and parental investment theory, by emphasizing the long-term social and emotional consequences of parenting sons versus daughters. It also offers a rare population-level exploration of how early-life biological and social exposures shape neurocognitive aging trajectories.

The study of the effect of children sex offers, thus, the opportunity to explore some of the causal mechanisms which are thought to produce cognitive decline. Since, as we have already noted, these mechanisms resemble those involved in the studies on the number of children, the present analysis will also help to shed light, indirectly, on the relationship between fertility and cognitive health.

2. Methods

This study adopts a standard natural experiment design (Dunning, 2012), which shares key features with randomized experiments, except for the fact that the treatment assignment is not under the researcher's control (Imbens and Rubin, 2015). In this case, assignment to treatment is based on the sex of the first biological child: parents whose first-born is a daughter form the control group, while those with a first-born son form the treatment group. Since parental selection on child's sex is assumed to be negligible, this setup approximates random assignment and allows for causal inference.

The objective is to estimate the causal effect of the first child's sex on parental cognitive decline in later life. The core advantage of (quasi-)randomization is the statistical independence between the treatment and all pre-treatment covariates. For instance, pre-birth income should be similarly distributed across the control and the treatment groups, reducing concerns of omitted variable bias.

By contrast, post-treatment variables may be affected by the treatment itself. For example, the sex of the first child might influence the probability of separation (Dahl and Moretti, 2008), which in turn could affect household income. If a post-treatment variable lies on the causal pathway between treatment and outcome, it qualifies as a mediator. These mediation channels constitute causal mechanisms (Imai et al., 2011).

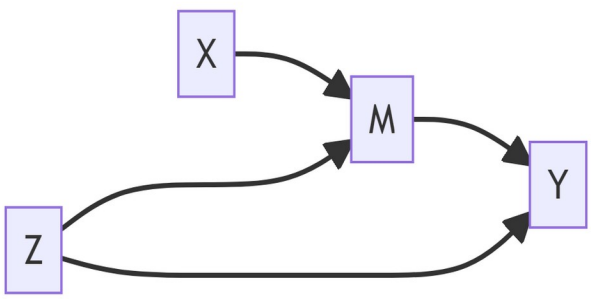


Figure 1 – Causal diagram. *X represents the treatment, Z a set of pre-treatment variables, M a set of mediators, and Y the outcome. Due to randomization, X and Z are independent (no edge between them).*

The structure of the causal relationships is summarized in Figure 1. The independence between treatment *X* and covariates *Z* is represented by the absence of a directed edge from *Z* to *X*. The treatment affects the outcome *Y* through a set of mediators *M*. We assume no direct effect from *X* to *Y*, as we assume all mediators are accounted for. Paths from *X* to *M* and from *X* to *Y* are unconfounded by design, whereas the path from *M* to *Y* may be confounded by *Z*. Consequently, we can identify the effects of *X* on *M*, and of *X* on *Y*, without adjustment; the effect of *M* on *Y*, instead, requires conditioning on *Z*.

We empirically test the marginal independence between *X* and *Z* through covariate balance checks. For numerical variables, we apply clustered versions of the t-test; for categorical ones, a clustered chi-squared test (Gregg et al., 2020), both implemented in the *htestClust* R package (Gregg et al., 2022). This accounts for within-cluster correlation due to parental dyads.

Provided balance holds, we estimate the Average Causal Effect (ACE) of *X* on *Y*. Following the Neyman potential outcomes framework, we use a t-test for the difference in means between groups (Athey and Imbens, 2017; Freedman, 2006). Again, clustering is accounted for in inference.

To investigate treatment effect heterogeneity by parent's sex, we fit the following linear model:

$$E(Y \vee x, s) = \alpha + \beta s + \gamma x + \delta xs, (1)$$

where *Y* denotes cognitive decline, *X* is the sex of the first child (0 = female, 1 = male), and *S* is parent's sex (0 = female, 1 = male). Standard errors are robust to heteroskedasticity and clustering (Cameron and Miller, 2015). A significant interaction term δ indicates that the effect of the child's sex differs by parent's sex—potentially suggesting biological or psychosocial mechanisms, especially if concentrated among mothers.

Finally, we examine whether the treatment causally influences hypothesized mediators through an approach called *causal triangulation* (Dunning 2012). For example, if cognitive decline is partly driven by the death of a child, and such risk is associated with the child's sex, we would expect the treatment to affect both the mediator (child mortality) and the outcome. We test for these intermediate effects using t-tests or chi-squared tests, depending on the variable type. Given the unconfoundedness between *X* and *M*, no adjustment is necessary. We also explore treatment effects on auxiliary outcomes such as depressive symptoms and subjective well-being, which may reflect broader pathways linking the treatment to cognitive health. Detecting such effects would further support the validity of the proposed causal mechanisms.

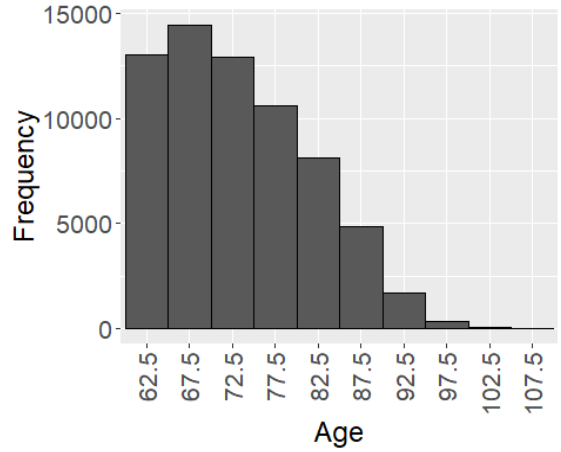
3. The data

We employed the data from the Survey on Health and Retirement in Europe (SHARE). This panel, which focuses on the population aged 50+, is organized in 9 waves covering 29 countries (27 countries from the European Union plus Israel and Switzerland) over the period from 2004 to 2022. Only 10 countries participated from the beginning, whereas the remaining countries joined later. In this analysis, we focus on the first eight waves to avoid potential distortions introduced by the COVID-19 pandemic.

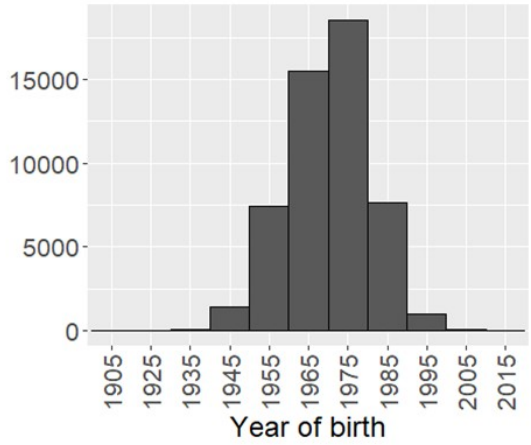
Each wave of the survey is composed of about 25 modules reporting several characteristics about individuals, couples and households. To ease data-handling, SHARE also provides *easySHARE*, a module that includes a simplified version of the dataset, with fewer variables for each individual. We used *easySHARE* as a starting point for the construction of our data set, to which we then added the variables from other modules. We first added the variables from the *SHARELIFE* module. The information in this module was collected, only for waves 3 and 7, with retrospective interviews performed to gather information about past life events and characteristics of the individuals. We then added the information contained in the *Children (CH)* and the *cognitive function (CF)* modules, for which information were collected in all waves, except for wave 3 and 7.

The SHARE panel contains 412,110 observations concerning 140,125 subjects. This means that for each subject we have, on average, about three observations; among these, we selected the observation associated with the oldest age and dropped those individuals for which the oldest age was below 60 because, at the population level, cognitive decline is practically absent before age 60 but increases rapidly after this age (Wu et al., 2016). Subsequently, we removed the individuals without children or for which the information on the sex or the year of birth of the first child was not available. At the end of these operations our study group resulted in 66,020 individuals.

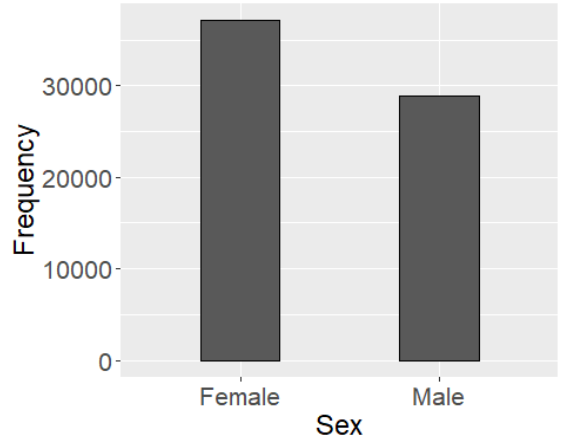
Figure 2 shows some features of our study group: the mean age of parents is 73.1 years, whereas their modal age class is 65-69 (Figure 2 (a)). Females, 56.2 percent of the sample, slightly outnumber males in our study group (Figure 2 (c)). This can be considered normal given the female advantage in survival especially in post-reproductive age (Beltrán-Sánchez et al., 2015, Salinari et al., 2022, Bolund et al., 2016). The parents in the study group had 51,671 first-born children, of which 24,934 females and 26,737 males, which corresponds to a male to female ratio of 107.2 (Figure 2 (d)). The modal period of birth of the children is represented by the decade 1970-1979, but only 16.8 percent of them are born after 1979 (Figure 2 (b)).



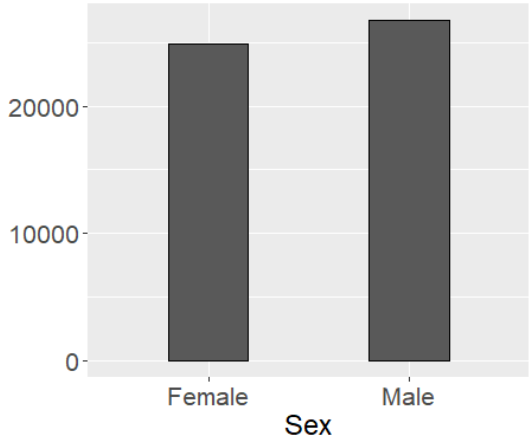
(a) Distribution of parents' age



(b) Distribution of first-born children's year of birth



(c) Distribution of parents' sex



(d) Distribution of first-born children's sex

Figure 2: Parents and first-born children's characteristics.

The SHARE dataset contains several variables related to cognitive health. However, for some of them the information was collected only in a few waves and for a few individuals. Therefore, we decided, following Bonsang and Skirbekk (2022), to focus only on three variables: immediate words recall, delayed words recall and verbal fluency.

In the assessment of immediate word recall, the participants were asked to listen to a list of 10 words and to repeat them immediately after. For delayed word recall, instead, the same question was repeated after a delay. In verbal fluency test, the participants were asked to mention as many animals as they could. The variables associated to these three questions report the number of words recalled and the number of animals mentioned by the survey participants. In case of immediate and word recall the test was performed on 62,473 individuals (the test was not performed in wave 3) and the number of missing values resulted of 2,772 and 2,555 which corresponds to about 4% of our study group. As for verbal fluency, the information on this variable is not available for waves 3 and 7. Among the remaining 38,648 individuals, the missing values are 1,468, that is, 3.8 % of the study group.

An additional group of variables was then used to test the effect of treatment on a set of hypothesized mediators and auxiliary outcomes: first-born child is alive, marital status, frequency of contacts with the first-born child, depression, and self-rated quality of life. These were selected based on the causal mechanisms discussed in the introduction.

4. Checking the treatment assignment mechanism

In this study, we use the sex of the first child as an *as-if random* assignment to a "treatment" (the birth of a male child) and a "control" (the birth of a female child). This approach builds on an established tradition in economic and demographic research that considers the sex of the firstborn to be essentially exogenous with respect to parents' pre-existing characteristics (Angrist and Evans 1998; Dahl and Moretti 2008; Bonsang and Skirbekk 2022). Specifically, several studies have documented that in contemporary Western populations, the probability that the first child is male is extremely stable (approximately 51–52%), and there is no evidence of systematic correlation between the sex of the firstborn and the parents' socioeconomic, cultural, or behavioral factors (Rodgers and Doughty 2001; Maconochie and Roman 1997; Jacobsen et al. 1999; Orzack et al. 2015). These features make this variable well suited to being interpreted as a natural randomizer. In principle, however, there exist several methods parents could use to select the sex of their child, which would alter the sex ratio at birth.

Historically, the most widely used method was sex-selective infanticide. Although this behavior is deemed rare in the European Ancien Régime (Lynch, 2011), recent historical demography studies have identified signs of selective infanticide in some Southern European countries: Spain (Beltrán Tapia and Marco-Gracia, 2022), Greece (Beltrán Tapia and Raftakis, 2022), but not Italy (Derosas et al., 2010). However, the literature agrees on the fact that this phenomenon rapidly vanished between the end of the 19th century and the first decades of the 20th century. Since most children considered in this study are born after the 1940s (see Figure 2), it is unlikely that selective infanticide affected the data.

A second method is sex-selective abortion (Bongaarts, 2013), which requires that sex can be determined during pregnancy, for example with amniocentesis or ultrasound scan, and that parents can opt for abortion after having determined the sex of the fetus. Amniocentesis is generally performed between the 15th and 20th week of pregnancy, whereas ultrasound scanning can determine the sex quite accurately after week 13. In most European countries the time limit for legal abortion is 12 weeks; 14 weeks in France, Spain and Romania, 18 weeks in Sweden and 24 in the Netherlands. In principle some parents could use sex-selective abortion, but in reality, this possibility is hampered by the timing imposed by the legislation on abortion. Methods for determining the sex of a fetus were introduced in the 1970s (Cukrowska-Torzewska and Grabowska, 2023) and became normal practice from the late 1980s. Therefore, for most (83%) of our study group the sex of the first-born children was simply unknown because they were born before.

A third, more subtle, way of manipulating the sex distribution of children consists in adopting a "stopping strategy": parents can continue having children until they get the child of the desired sex. Bongaarts (2013) has shown that if parents desire a boy and all families conform to this stopping strategy, then the proportion of males among the last-born children approaches 1. Stopping strategies have recently been identified in several European countries (Cukrowska-Torzewska and Grabowska, 2023). This is why we decided to focus on the sex of the first-born, because this variable is not affected by stopping strategies.

The sex ratio at birth may be, to some extent, determined by external environmental conditions or by the health conditions of the mother. The "frail male" theory (Catalano et al., 2005, Song, 2012) claims that male fetuses are more affected by external calamities than female ones. For this reason, it is expected that in good times the proportion of males is higher compared to bad periods. The "adaptive sex-biased investment" theory states, instead, that mothers in better health and nutritional condition should invest more resources in males (Trivers and Willard, 1973). The proportion of sons is thus expected to be higher among affluent and younger mothers than among poor and older ones. A recent analysis (Schacht et al., 2019) finds evidence of the first phenomenon (frail male) but not of the second (adaptive sex-biased investment). Once more, our analysis is sheltered from this phenomenon, because the first-born children here considered are born mostly in an epoch (1950-1990) without major crises or epidemics.

Finally, Hamoudi and Nobles (2014) argue that maternal stress during pregnancy may lead to higher mortality among male fetuses, thereby reducing the male-to-female ratio at birth. A recent article (Salinari, Carboni, and Zarulli), however, suggests that the observed association between prenatal stress and the sex of the newborn might partly reflect a statistical artifact due to collider bias. Moreover, this study does not find evidence consistent with the association reported by Hamoudi and Nobles (2014).

Even if it is overall unlikely that parents controlled in some way the sex of their offspring in Europe during the period covered by the present analysis, this issue cannot be decided solely based on logical or historical arguments. To assess the plausibility of treating the sex of the first child as an exogenous variable, we carried out several preliminary checks using both external population-level data and internal consistency tests within our dataset. These checks are designed to assess whether any of the previously mentioned mechanisms may influence the sex distribution of offspring in our data. Sex ratio at birth (SRB) trends across European countries between 1940 and 1999 show no evidence of systematic sex selection. Country-level tests on the proportion of first-born males reveal only three significant deviations (Romania, Slovakia and Spain), which disappears after multiple-testing correction in all but one case (see Table A.1 of the supplementary material). Further tests on last-born children and maternal age at first birth also fail to support hypotheses of sex preference or biologically-driven sex selection. For a detailed account of these robustness checks, see the supplementary material.

If parents are not manipulating the sex distribution of their offspring in any systematic way, we should expect the control group not to differ significantly from the treatment group in terms of relevant pre-treatment characteristics. This is the most important assumption in our analysis. The SHARE data provides several insights about socio-demographic characteristics and early life conditions of the parents in our study group. To these variables we then added a set of variables aimed at evaluating parents' personality: extraversion; agreeableness; conscientiousness; neuroticism; openness. These variables, the big five personality traits, are measured on an ordinal scale ranging from "low" to "high". This taxonomy was proposed in the 1980s by different scholars and has been found to be associated to numerous variables such as educational attainments (Caprara et al., 2011), income (Bakker, 2017) and fertility (Alvergne et al., 2010, Skirbekk and Blekesaune, 2014).

Table 2: Balance tests. Author's computations on SHARE data

Macro-category	**Var. name**	**Var. description**	**Test**	**P value**
Socio-demographic	Year of birth	numeric	t	0.402
	Month of birth	ordinal	χ^2	0.613
	Sex	binary	χ^2	0.509
	Education	ordinal	χ^2	0.773
	Country of birth 1	nominal	Perm.	0.001
	Country of birth 2	nominal	Perm.	0.111
Childhood conditions	Childhood health	ordinal	χ^2	0.194
	Book age 10	ordinal	χ^2	0.482
	Math age 10	ordinal	χ^2	0.755
	Language age 10	ordinal	χ^2	0.106
	Vaccinated	binary	χ^2	0.781
Personality traits	Extraversion	ordinal	χ^2	0.468
	Agreeableness	ordinal	χ^2	0.304
	Conscientiousness	ordinal	χ^2	0.666
	Neuroticism	ordinal	χ^2	0.299
	Openness	ordinal	χ^2	0.576

Note: this table reports the results of a series of tests aimed at verifying that our treatment (a binary variable indicating the sex of the first-born child) is not associated to pre-treatment variables. Most of the time we performed a χ^2 test of independence for clustered data between our treatment and the non-numeric variables listed in the column "Var.name". In case of numeric variable (Year of birth) we used instead a t-test for clustered data. In the case of the variable "Country of birth" we recurred to a Monte Carlo mutual information permutation test because of the elevated number of countries with an

insufficient number of observations. “Country of birth 1” refers to all countries of birth in our dataset, whereas we removed Romania, Slovakia and Spain from "Country of birth 2”.

We checked our assumption of independence by performing the series of balance tests, shown in Table 2. We considered 15 distinct pre-treatment variables, organized in three macro-categories: a) socio-demographic variables; b) childhood conditions; c) personality traits. To verify the independence between the sex of the first-born child and these variables, we performed a Chi-squared test of independence for clustered data, except for the case of country of birth, for which we used a Monte Carlo mutual information permutation test (Edwards, 2012) because of the elevated number of countries with an insufficient number of observations. In the case of the year of birth, we proceeded instead with a t-test for clustered data on the difference of means.

In all cases but one, the tests indicate that our treatment and pre-treatment variables are statistically independent. The only variable for which the null hypothesis of independence is rejected was the country of birth (variable: country of birth 1). This is the same problem that we have already identified in Table A.1 of the supplementary material and it is likely to be due to the anomalous value of the SRB index observed for Romania, Slovakia and Spain. Indeed, if we remove them (variable: country of birth 2) the hypothesis of independence cannot any longer be rejected.

Summing up, this first series of checks, we conclude that they do not identify any problem in 26 out of 29 countries. In Romania, Slovakia and Spain, the sex ratio among first-born children appears significantly higher than expected. This may be due to a sampling error or because in this populations some groups are actively selecting the sex of their children. Here we opted for the first, more optimistic, hypothesis because our main results do not change significantly if we remove these three countries from our dataset.

5. Main results

The main findings of our analysis are presented in Table 3. This table is divided into three sections. The first, *Cognitive Health*, reports the primary results supporting the existence of a causal link between the sex of the firstborn child and cognitive decline in old age. The second section, *Auxiliary Outcomes*, explores other variables associated with cognitive functioning, serving as robustness checks. The third, *Mediators*, investigates possible causal mechanisms that could explain how the sex of the firstborn affects parental cognitive health. In the remainder of this section, we summarize the results related to each of these three areas.

5.1 Cognitive Health

We begin by examining cognitive performance, focusing on immediate and delayed word recall. Both measures are significantly lower among parents whose firstborn is a son. Specifically, the average number of words recalled immediately is 4.83 for the treated group (firstborn male) and 4.88 for the control group (firstborn female). For delayed recall, the

means are 3.41 and 3.46, respectively. Although the differences are small (approximately -0.05), they are statistically significant. Estimations from Equation 1 confirm that this effect is not modified by the sex of the parent (see Table 3 - Effect modifier); both fathers and mothers exhibit similar declines when the firstborn is male.

The case of verbal fluency is more nuanced. On average, the treated group scores 19.22 and the control group 19.31, a non-significant difference. However, the analysis reveals a significant interaction with parent's sex. Among fathers, the average scores are 19.68 (treated) and 19.93 (control), a difference that is statistically significant at the 5% level. Among mothers, the scores are virtually identical (19.66 vs. 19.62). Thus, the adverse effect of having a male firstborn on verbal fluency appears to be concentrated among fathers.

Table 3: Main results. Author's computations on SHARE data

	Var. name	**Var. description**	**Test**	**P value**	**Effect modifier**
Cognitive health	Words recall1	numeric	t	0.005	No
	Words recall2	numeric	t	0.033	No
	Verbal fluency	numeric	t	0.330	Yes
Auxiliary outcomes	Depression	numeric	t	0.001	No
	Quality of life	numeric	t	<0.001	No
	Age	numeric	t	0.618	No
Mediators	Never married	binary	χ^2	0.479	No
	Alone	binary	χ^2	1	No
	Death child 1	binary	χ^2	<0.001	-
	Contact child 1	ordinal	χ^2	<0.001	Yes

Note: This table reports the results of our tests on the effect of the sex of the first-born child on different outcomes listed in the "Var. Name" column. The column "Effect modifier" specify whether the effect changes according to the sex of the parent. The presence of a different effect for mothers and fathers was identified, most of the times, by estimating eq. (1) and by checking that the coefficient on the multiplicative interaction term was significantly different from zero. In the case of two variables, "Never married" and "Alone", we simply repeated the test for mothers and fathers alone. In the case of the binary variable Death child 1, indicating whether the first-born child is dead, testing for a heterogeneous effect is meaningless.

5.2 Auxiliary Outcomes

To assess the robustness of the main results, and following the suggestion of Dunning (2012), we examined whether the treatment also affects other variables known to be closely related to cognitive health: depression, perceived quality of life, and age. The general idea behind this kind of analysis is that higher levels of depression and lower life satisfaction are both known causes and consequences of cognitive decline. If these variables also differ by the sex of the firstborn, this would provide additional support for a causal interpretation of our findings. This does indeed appear to be the case.

Depression is measured using twelve yes/no questions, with the total number of positive responses indicating the overall level of depression. The mean depression score is 2.60 for the control group and 2.68 for the treated group. While the absolute difference is modest, it is highly statistically significant (see Table 3).

Perceived quality of life, measured as the sum of four subscales (control, autonomy, pleasure, and self-realization), ranges from 12 to 48. The average score is 36.35 for the control group and 36.59 for the treated group. Again, the difference is small but statistically significant.

For both depression and quality of life, we found no evidence that the treatment effect varies by the sex of the parent.

Finally, we considered whether the sex of the firstborn affects parental age as a proxy for differential survival. The mean age is nearly identical across groups: 73.55 for the control and 73.51 for the treated. This result holds even when restricting the analysis to mothers, suggesting no significant effect on survival-related age differences.

5.3 Mediators

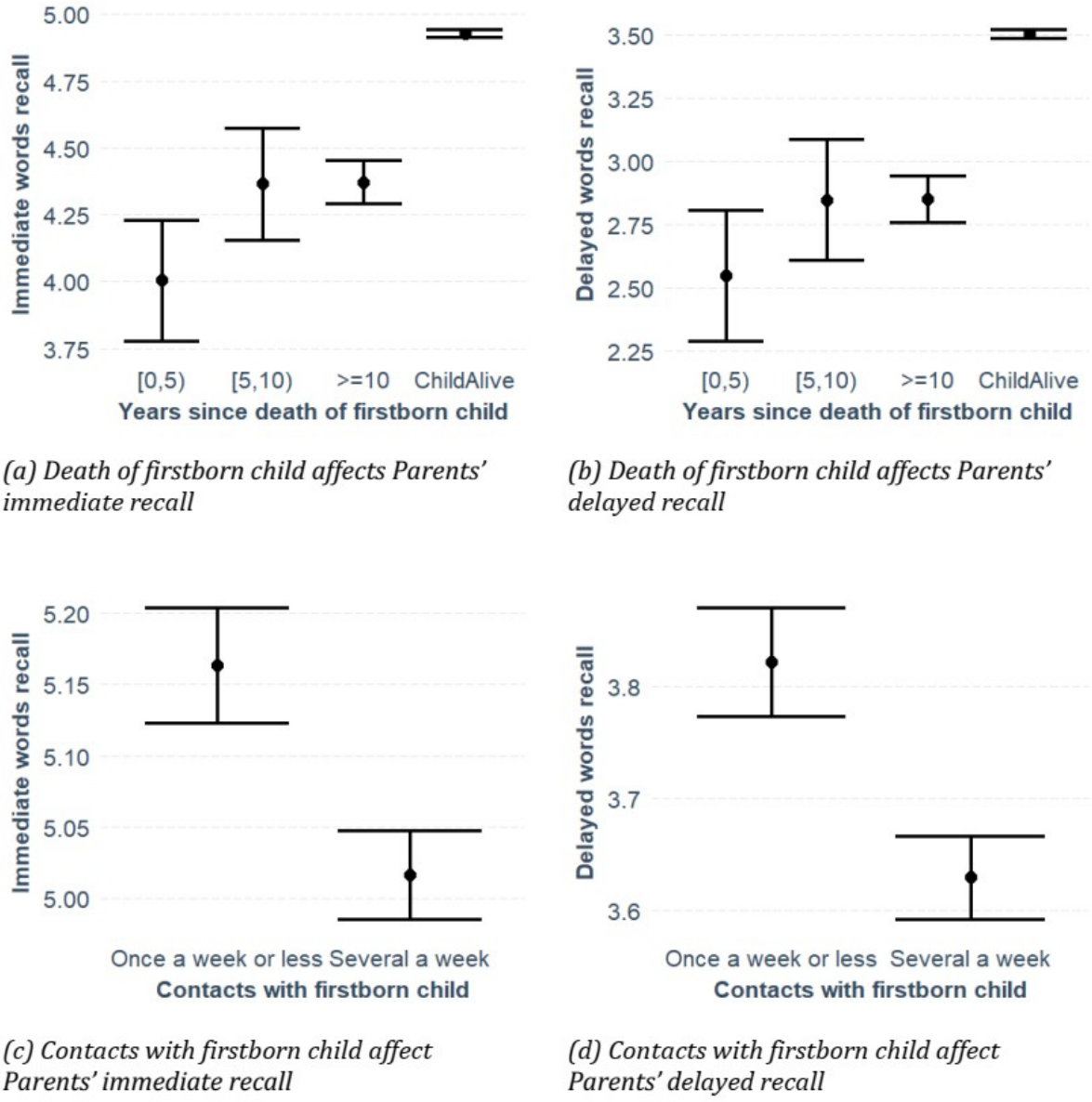
We then explored potential causal mechanisms linking the sex of the firstborn to cognitive outcomes. Table 3 includes results for three hypothesized mediators: marital status, child mortality, and frequency of contact.

The first mechanism relates to marital status. Dahl and Moretti (2008) found that in the U.S., women with firstborn daughters are more likely to live without a partner. We tested whether similar patterns exist in our data by comparing the proportion of never-married and non-cohabiting mothers across treatment groups. Both tests yielded non-significant differences: the proportion of never-married women is 1.67% in both groups; for non-cohabiting women, the treated group shows a slightly lower proportion (43.24%) than the control (43.45%). These findings align with Dahl and Moretti (2008), but our results are not statistically significant. Moreover, this mechanism would imply that daughters negatively affect maternal cognitive health, whereas we observe the opposite.

The second mechanism concerns the differential mortality risk of firstborn sons. The share of parents who have lost a firstborn son is significantly higher (5.66%) than for daughters (3.22%). This association is likely causal, given that treatment assignment is exogenous. Figure 3 shows that the death of a firstborn child is strongly associated with reduced

cognitive performance, particularly when the death occurred within the five years preceding the survey. In this group, parents recall only 4.00 words immediately and 2.55 after a delay, compared to 4.92 and 4.95 among parents whose firstborn is still alive.

The third mechanism hypothesizes that sons provide less support to aging parents. Figure 4 illustrates that frequent contact (several times per week or daily) is more common with firstborn daughters (67.8%) than with sons (59.2%). This difference is statistically significant (Table 3). However, Figure 3.c and Figure 3.d suggests an unexpected pattern: parents who are in contact less frequently tend to have better cognitive scores.



(a) Death of firstborn child affects Parents' immediate recall

(b) Death of firstborn child affects Parents' delayed recall

(c) Contacts with firstborn child affect Parents' immediate recall

(d) Contacts with firstborn child affect Parents' delayed recall

Figure 3: The association between mediators and outcomes

This may reflect reverse causality, where children increase contact in response to parental cognitive decline, complicating the interpretation.

Overall, our findings suggest that having a firstborn son is associated with worse cognitive performance in later life. The effect appears to operate through mechanisms such as higher child mortality and reduced parental support. These results are consistent across a range of robustness checks and do not appear to be driven by parental sex differences or survival selection.

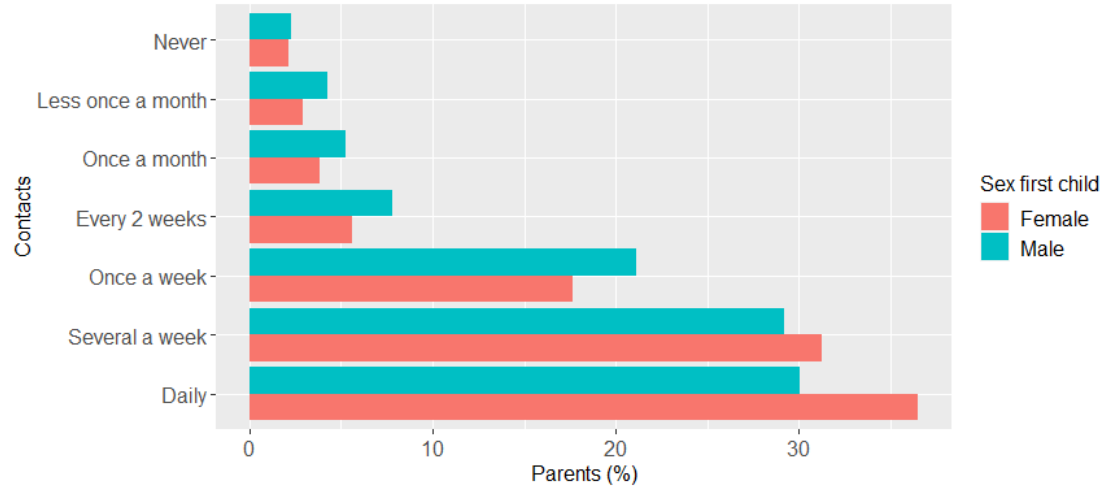


Figure 4: Contacts with first-born child over the past twelve months.

6. Discussion

In this study, we adopted a standard *natural experiment* design to assess whether the sex of the firstborn child affects the cognitive decline of parents. This approach offers two key advantages over other methods commonly used in similar studies.

The first advantage lies in the fact that the sex of the firstborn can be considered a natural randomizer. This implies that all covariates—both observed and unobserved—should have the same mean in the treatment and control groups (up to sampling error). The proportions of males and females, high school and university graduates, engineers and librarians, and so on, are expected to be identical across the two groups. In other words, the comparison between treatment and control groups takes place while implicitly holding constant virtually all pre-treatment variables that might otherwise bias the causal relationship of interest.

Some studies have questioned the randomness of firstborn sex. For example, Hamoudi and Nobles (2014) suggest that maternal stress prior to childbirth may reduce the likelihood of a male birth. However, the existence of such effects remains uncertain, and even if they do exist, they are likely to be extremely weak—too weak, in fact, to be detectable in a large sample like ours, which includes over 60,000 observations. Even if minor violations of the random assignment assumption were present, we argue that our estimates remain more credible than those obtained using alternative methods. Consider, for instance, the covariate adjustment approach (controlling for observables). In that framework, one might adjust for educational attainment by including in the model a variable for years of formal

schooling or highest degree earned. However, such models rarely adjust for *type* of diploma or degree, even though it is well known that different educational tracks are associated with very different income levels. These adjustments, therefore, are only approximations. Moreover, such models cannot account for unmeasured confounders unknown to the researcher. In short, any small deviations from randomness in the sex of the firstborn are likely more than compensated for by the broad array of potential biases that this natural experiment design helps to control for. For this reason, we believe this approach is preferable to covariate-adjustment-based methods.

The results of our analysis indicate that the sex of the firstborn child affects parental cognitive health in later life, across three primary outcomes (immediate and delayed word recall, and verbal fluency) and two auxiliary outcomes (depression and perceived quality of life). The effect is consistent: having a firstborn son reduces cognitive performance and perceived quality of life, and increases the average level of depression. The absolute magnitude of the effect is generally small but highly statistically significant (except for verbal fluency among mothers).

The second advantage of our approach is that it enables mediation analysis to explore the specific causal mechanisms linking treatment to outcome. In this study, we employed a strategy known as *causal triangulation*: we tested for the presence of associations between the treatment and a specific mediator, and between the mediator and the outcome. As discussed in the methodological section, this type of analysis is not sufficient to confirm the existence of a specific causal mechanism, but it can rule out mechanisms that are not involved in the phenomenon under investigation.

Our analysis sheds light on some of the potential mediators of the observed effect. Perhaps the most important result is the reappraisal of biological mechanisms linked to pregnancy. We found no clear evidence of a biological pathway: in most outcomes, the effect did not differ between mothers and fathers. In the case of verbal fluency, we observed an effect only among fathers; however, if a biological mechanism were at play, one would expect a stronger or exclusive effect among mothers. Additionally, we detected no difference in the average age of parents based on the sex of their firstborn, suggesting that this variable does not influence the ageing process.

We also found no support for the hypothesis that marital status mediates the effect. In our data, treatment and mediator were independent: firstborn daughters are not more likely to grow up in father-absent households, contrary to what has been reported in the literature. However, those earlier findings referred to the U.S. context (Dahl and Moretti, 2008), which may differ from the European setting considered here.

We did find support for the mechanism suggesting that firstborn daughters maintain more frequent contact with their parents than firstborn sons. However, in this case, the mediating path is complicated by the possibility of reverse causality: increased contact may reflect a response to cognitive decline rather than a cause of it.

The mechanism for which we found the strongest support relates to the differential survival of sons and daughters. Our analysis shows that the death of a firstborn child is associated with cognitive decline among parents, and that sons are more likely to die than daughters. A similar mechanism may be at work in the association between number of children and cognitive health (Bonsang and Skirbekk, 2022), as a larger number of children increases the probability of experiencing the death of at least one. We believe this mechanism is important to highlight, as it suggests that the loss of a child may have long-lasting effects on cognitive function. More broadly, our findings support the idea that cognitive functioning in old age may be shaped by the cumulative exposure to shocks experienced across the life course. This could also help explain why the overall effect size we observe is small: the death of a child is a rare event in the time span covered by this study, and the effect we identify relies on the small survival differences between male and female children.

In conclusion, our analysis not only provides evidence of a causal link between the sex of the firstborn child and cognitive decline in later life, but also narrows down the set of plausible causal mechanisms. Specifically, our results suggest that two mechanisms deserve further investigation: the higher mortality rate among sons, and the more frequent contact between daughters and their parents. These distinct causal pathways can now be explored more precisely through formal mediation analysis, which we intend to pursue in a future study.

These findings also hold significant policy implications for aging societies. First, they point to the need to incorporate family structure—especially offspring sex composition—into cognitive risk assessments and elder care planning. Daughters' greater involvement in intergenerational care may provide a buffer against cognitive decline, while the higher mortality risk associated with sons may signal the need for additional psychosocial support. Second, the evidence that child loss contributes to cognitive aging suggests a need for long-term mental health follow-up and cognitive screening among bereaved parents. More broadly, our findings reinforce the value of life-course approaches in public health, emphasizing how cumulative emotional and social exposures shape aging trajectories. Policy interventions that recognize these dynamics—especially those aimed at supporting single parents, caregivers, and parents experiencing loss—can contribute meaningfully to cognitive resilience in later life.

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