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Fertility decline and the emergence of excess female survival in post-reproductive ages in Italy

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Abstract

In Italy, at least in the cohorts born up to the beginning of the twentieth century, women's mortality in post-reproductive ages was influenced by fertility, with large progenies (and, to a lesser extent, childlessness) leading to markedly lower survival chances. This relationship proved strong enough to affect the female-to-male ratio in old age as fertility declined. In this paper, we show that various measures of extra female survival at high ages are closely connected to the fertility transition in Italy, and to its peculiar historical and geographical evolution.

Keywords: Women's mortality, Post-reproductive ages, Fertility, Disposable soma

Introduction

In modern populations, women live longer than men do, and they end up outnumbering them: this inflates the value of the female-to-male ratio in old age (Robine & Caselli, 2005; Robine et al., 2006).

Male disadvantage has both biological and social causes. Proponents of the biological explanation argue that men are less fit to live past reproduction, although disagreement emerges as to the specific causal factor: the Y chromosome, mitochondrial DNA, sexual hormones (Giuliani et al., 2018; Marais et al., 2018; Vina et al., 2005). While genes certainly matter, doubts remain as to their relative importance. Research on Danish twins, for instance, led to the conclusion that only a small share of the observed variability in the distribution of ages at death depends on genetic factors, possibly about 25% (Herskind et al., 1996). Sex, which is also linked to genetics, may therefore play only a limited role in differential mortality. Beltrán-Sánchez et al. (2014), for instance, show that a significant female advantage in late survival emerged only in the cohorts born at the end of the nineteenth century, and only in a handful of developed countries. Furthermore, at least in England and Wales, the emergence of the female survival advantage started from higher social strata (Martin, 1951). Both the rapidity of the change, with ups and, recently, downs, and its socio-economic connotation seem to conflict with a purely biological interpretation.

The alternative set of explanations pertains to behavioural differences and culturally defined gender roles, responsibilities and experiences. Men are more likely to participate in the labour market, doing hazardous jobs and holding managerial positions, which exposes them to higher health risk and stress (Hemström, 1999). Besides, men, who rarely perform health-preserving activities (Liang et al., 1999; Vaidya et al., 2012), indulge more frequently than women in risk-taking behaviours, such as consumption of tobacco, alcohol and psychoactive substances, dangerous driving and insalubrious eating habits (Courtenay, 2000; Waldron, 1985; Wardle et al., 2004). Cigarette smoking, in particular, directly linked with mortality (Janssen, 2019), spread rapidly among males, starting from the cohorts born towards the end of the nineteenth century (Ipsen, 2016; Preston & Wang, 2006). Later on, smoking prevalence increased among women and subsided among men (Pampel, 2002, 2010), a convergence that fits well with the recent reduction of the gender difference in survival, and points to smoking as a likely causal factor.

However, even if a substantial part of the female advantage in late survival is attributable to limited smoking, possibly between 30 and 40% in the peak years (Beltrán-Sánchez et al., 2014), and even accounting for the possible role of genetics and other behavioural factors, a large part of the female survival advantage still remains unexplained. Part of the explanation may be the fertility decline. This is the conjecture that Bolund et al. (2016) advance for the cohorts born between 1820 and 1919 in Utah (USA): in this paper we will try to substantiate our claim that the validity of this conjecture extends to Italy and its fertility transition.

The idea that completed family size (CFS) and post-reproductive survival are related is not new. More than 30 years ago, for instance, Kirkwood (1977) elaborated the “disposable soma” theory, suggesting that animals allocate their (finite) energetic resources “trading off” between reproduction and survival, and this explains why species with high fertility typically have shorter life spans and vice versa. These ideas were later developed to argue that even within species, including humans, females devoting more resources to reproduction exhaust their reserves sooner: they tend to die earlier (Westendorp & Kirkwood, 1998) and are more frequently affected by health problems, such as diabetes and cardiovascular diseases (Jasienska et al., 2017), although unobserved demographic factors (Gavrilov & Gavrilova, 1999) and frailty (Doblhammer & Oeppen, 2003) may confound this relationship. This interpretation is also consistent with the results of genetic studies, leading to the conclusion that reproduction may accelerate aging in women by affecting their cellular turnover (measured by telomere length, a bio-marker of ageing) or their epigenetic regulation (DNA methylation age; Ryan et al., 2018), or both.

So far, however, empirical research on the link between fertility and survival has yielded mixed results. Some studies found an association between higher CFS and telomere shortening (Pollack et al., 2018), higher biological age (Ryan et al., 2018), higher risks of type-2 diabetes (Mueller et al., 2013) and, in general, higher mortality (Doblhammer & Oeppen, 2003; Gagnon et al., 2009; Smith et al., 2002). Others, instead, concluded that the association between CFS and survival is positive (Müller et al., 2002; Sear, 2007), or absent (Helle et al., 2002), or negative, but only for high parities, five and over (Zeng et al., 2016) or for women in lower social classes (Dribe, 2004). To increase confusion, Barclay and Kolk (2019), found a negative association

between CFS and survival (Swedish birth cohorts, 1915–1960), not only for biological mothers, but also for biological fathers and even for adoptive parents, both mothers and fathers.

According to Gagnon et al., (2009), among others, these conflicting results may depend on the intrinsic difficulty of testing the association between CFS and mortality. Inverse causation is a factor, because pregnancy and breastfeeding protect women from certain types of cancer, such as uterus and breast.

Selection may act in several ways. In the simplest version, sick women are more likely to have fewer children or to remain childless—and to die earlier in their old age. However, there is also a possible counteracting mechanism: as women in poor health who had more children than their health would allow die during childbearing ages, those with high parities who survive to old age are selected. Conversely, among the frail ones, those who had fewer children survive to post-reproductive ages and tend to be concentrated among low parities, but, because they are frail, their mortality is higher (Barclay et al., 2016; Doblhammer & Oeppen, 2003; Grundy & Kravdal, 2010; Kelsey et al., 1993; Kravdal et al., 2012). Moreover, adult children may provide support to their ageing mothers (Caselli et al., 2013) and the relation is probably context-dependent: in recent cohorts, better nutrition, life conditions and medical assistance during pregnancy and at delivery may have weakened, if not cancelled, the link between fertility and survival later in life.

Against this background, Bolund et al. (2016)'s contribution has some distinct merits, in our opinion. First, they conjecture that the negative association between fertility and survival is so strong that declining fertility may significantly improve later female survival, and confer upon women a distinct advantage over men, at least in the specific (pre-transitional and transitional) historical phase that they analyse. In addition, their hypothesis is consistent with the top-down diffusion of both processes, because lower fertility and greater excess female survival appeared first in high SES niches. Finally, it combines biological and behavioural elements: women are more robust than men are, but before the fertility transition this “natural” advantage of theirs was offset by their specific “risky behaviour”: too many pregnancies and births.

In this paper, we will test the hypothesis that fertility decline was one of the drivers of the emergence of female survival advantage in Italy, a country that constitutes an interesting laboratory, for two main reasons: on the one hand, because of the large regional variation in the onset of the fertility transition, which started in last decades of the nineteenth century in the north, but much later in the south, around mid-twentieth century (Livi Bacci & Breschi, 1990); on the other, because the Italian official statistics allow, with only a few limitations, to track the fertility evolution of the cohorts who experienced the fertility transition.

This paper proceeds as follows. In Sect. 2, we present the sources and indicators that we exploit to carry out our analysis. In Sect. 3, we document the existence of strong negative association between previous fertility and later (old age) survival for several cohorts of women involved in the fertility transition. The ecological analysis of Sect. 4 shows that the emergence of the female excess survival can be linked to the process of fertility decline. In the conclusions of Sect. 5, we argue that the fertility transition gave a substantial impulse to the improvement of female survival in post-reproductive ages, and

Table 1 Italian administrative regions and macro-regions

Macro-regions	Regions
North:	Piedmont, Liguria, Lombardy, Veneto, Emilia R. (Val d'Aosta, Trentino A.A., Friuli V.G.)
Centre:	Tuscany, Marche, Umbria, Lazio
South:	Abruzzi, Campania, Puglia, Basilicata, Calabria (Molise)
Islands:	Sardinia, Sicily

Regions in parenthesis were created later: they can be observed in recent years (e.g., 2011), but they are not reported separately in historical sources (e.g., the 1911 population census)

Table 2 Observation of selected birth cohorts through the Italian censuses, at various post-reproductive ages

Cohort	Census										
	1911	1921	1931	1941	1951	1961	1971	1981	1991	2001	2011
1862–1866	45–49	55–59	65–69	–	85–89	95–99	–	–	–	–	–
1872–1876	–	45–59	55–59	–	75–79	85–89	95–99	–	–	–	–
1882–1886	–	–	45–49	–	65–69	75–79	85–99	95–99	–	–	–
1892–1996	–	–	–	–	55–59	65–69	75–79	85–89	95–99	–	–
1902–1906	–	–	–	–	45–49	55–59	65–69	75–79	85–89	95–99	–
1912–1916	–	–	–	–	–	45–49	55–59	65–69	75–79	85–89	95–99
1922–1926	–	–	–	–	–	–	45–49	55–59	65–69	75–59	85–89
1932–1936	–	–	–	–	–	–	–	45–49	55–59	65–69	75–79

Source: Italian censuses

that this factor contributed to inflate the female-to-male ratio in old ages, with different timings in the various Italian regions.

Sources and indicators

Sources

In this paper, we tap several data sources. Some of them refer to Italy as a whole, while others allow to break the information down by administrative region. Currently Italy has 20 such regions, often grouped into 4 macro-regions, but back in 1911, when we start our observation, there were only 16. Table 1 reports the detailed classification.

Population censuses are probably our most important source. The first national Italian census was conducted in 1861 (year of the Italian Unity). Successive censuses took place every 10 years thereafter, with few exceptions (not in 1891 and in 1941). We use the censuses of the period 1911–2011, which contain data related to cohort fertility. We also use the “extra” census of 1936, but only for consistency checks. In our analyses, we focus on the number of respondents by age, gender, marital status and administrative region at specific age classes (e.g., 100 years and over) both in a period and in a cohort perspective, by following birth cohorts enumerated at successive rounds and progressively older ages.

Table 2 shows the census subset for each of the cohorts under study. For instance, we know how many members of the 1862–1866 birth cohorts were alive and still in Italy in 1911 (at 45–49 years), and we can “follow” them up to age 95–99 (1961 census). Unfortunately, truncation affects our data, in part because the 1941 census is missing and in part because in 2011 the youngest cohorts had not yet reached the

Table 3 Sources of information on fertility (and lung cancer mortality) for selected Italian cohorts

Birth cohort	Fertility			Mortality
	1931 survey	1961 survey	I_f	Lung cancer
Regressions of Table 4				
1862–1866	–	–	1891	–
1872–1876	50–59	–	1901	–
1882–1886	45–49	–	1911	–
1892–1996	–	65–69	1921	–
1902–1906	–	55–59	1931	–
1912–1916	–	45–49	1936	1991
1922–1926	–	–	1951	2001
1932–1936	–	–	1961	2011
Parity-specific survival (Sect. 4)				
Before 1887	45+	75+		
1887–1891	40–44	70–74		

“1931 survey” and “1961 survey” refer to the Italian retrospective fertility surveys carried out in 1931 and 1961, together with the population census, covering ever-married women. In these columns, the ranges refer to the age classes for which information on complete cohort fertility exists. “ I_f ” is the general fertility index as defined in the framework of the European Fertility Project and calculated by Livi Bacci (1977). This is the (period) fertility measure that we use as a proxy for the fertility of the cohorts born 25–29 years before: better measures are available for some cohorts, but not for all of them. Each cell in this column indicates the year when I_f was computed and the cohorts to which we associate this (period) measure. The last column refers to lung cancer mortality at 75–79 years (a proxy for the impact of smoking), derived from ISTAT’s “Health for All” database. The table displays the years when cancer mortality was calculated

ending age group that we use in this part of our analysis (95–99 years). As most of the Italian censuses of the past century adopted a final open class “100 years and over”, we cannot include centenarians in our study. Note, incidentally, that in all the sources that we use, age is not asked directly of respondents. What they indicate is their date of birth (day, month, year), which drastically reduces the risk of age misreporting, and, in all cases, makes it virtually independent of CFS.

To refine our analyses, we introduce controls. One of these is gender differential mortality (within a cohort) attributable to smoking, which we proxied with lung cancer. However, as information on regional cause-specific mortality became available only after 1990 (in the “Health-for-All” database) in Italy, the oldest birth cohort for which we could introduce this control is that of 1912–1916, aged 75–79 years in 1991. For reasons of consistency, this is the age class used also for subsequent (younger) birth cohorts, as specified in Table 3.

For fertility, the main “causal” variable in this analysis, we use completed cohort fertility for the cohorts from 1872 to 1916. This is calculated from two retrospective fertility surveys administered together with the 1931 and 1961 censuses. Both surveys asked two different questions: (1) number of children ever had and (2) number of children still alive at the time of the survey. In our analysis, we use the former (children ever had), which includes stillbirths (but excludes miscarriages). Strangely, the 1961 retrospective survey reported data for women who had been married only once (including widows). For reasons of comparability, we applied the same restriction also to the 1931 survey, and we focused on women married only once. In the rest of the paper, however, for the sake of simplicity, we will simply call them ever-married women.

As Table 3 shows, we can determine the CFS of the 1912–1916 cohort, for instance, thanks to the 1961 survey, when these (ever-married) women were 45–49 years old. In the same source, the 1892–1896 birth cohort reports this information at 65–69 years. For the 1872–1876 cohort, whose completed fertility is known through the 1931 survey, we do not have precisely the information that we need (at 55–59 years): as a proxy, we resorted to the decennial age class 50–59 years.

For the cohorts 1862–1866, 1922–1926 and 1932–1936, unfortunately, we cannot calculate completed cohort fertility. In these cases, in some parts of our analysis we use as a proxy the general fertility index I_t as defined in the framework of the European Fertility Project (Coale & Cotts Watkins, 1986), and calculated by Livi Bacci (1977) for the Italian regions. While this index is a period measure, it correlates well with the fertility of a properly chosen cohort, born 25–29 years before, roughly corresponding to the average age at childbearing. Of the cohorts for which we have both variables, we measured the correlation, which proved to be very high (about 0.93).

The lower part of Table 3 shows the cohorts (born before 1887 and in the years 1887–1891) whose completed fertility we can calculate in two separate rounds (1931 and 1961) and for which we can also compute parity-specific survival.

Finally, for the 1912–1916 birth cohort (not in Table 3), to derive parity-specific survival we combine the 1961 fertility survey and the general-purpose national *AVQ Survey (Aspetti della Vita Quotidiana, or Aspects of Everyday Life)* of 1993 and 2003, when these women were aged 77–81 years and 87–91 years, respectively.

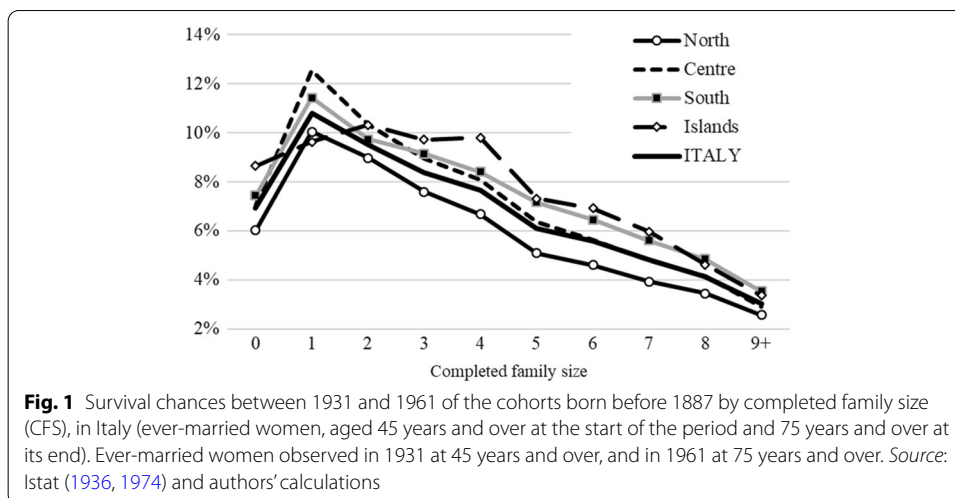
Indicators

For fertility, as mentioned, we use completed cohort fertility whenever this is known, or the general fertility index I_t as proxy for it, when the first indicator is not available.

For mortality, we are interested in female mortality in post-reproductive ages, because we want to relate it to past fertility. For the years and the cohorts for which data are available, we calculate absolute, parity-specific measures of survival by comparing the number of women of the same cohorts who survive at various post-reproductive ages. In other cases, we use a cruder measure: the female-to-male ratio $\Phi_x = F_x/M_x$. This indicator has an implicit reference value, $\Phi_0 = F_0/M_0 \approx 0.95$, the female-to-male ratio at birth, a biological constant: higher values at later ages, such as $\Phi_{100+} \approx 3$, signal higher survival among women. The shortcoming of this simple measure is that it does not reveal when the female advantage emerged in the life course of a cohort and why. It could depend on “external” causes, such as war or gender selective out-migration¹ that took place several decades before, all factors that are (largely) independent of fertility.

A better indicator of the same type is $EFS_x = \Phi_x/\Phi_e$, or post-reproductive extra female survival, which measures (the ratio of) differential survival from slightly after the end of the reproductive survival “ e ” (in this paper 55–59) up to age x , where x is a conveniently advanced age (e.g., 85–89 years). A simple rearrangement of terms shows that $EFS_x \left(= \frac{F_x/M_x}{F_e/M_e} = \frac{F_x/F_e}{M_x/M_e} \right)$ restricts the observation to what happens at relatively late

¹ In theory also in-migration. In the Italian cohorts covered in this study, however, emigration was largely prevalent.



ages, where confounding factors such as wars, migration and maternal mortality play only a minor role, if at all.

The link between mortality and completed family size (CFS)

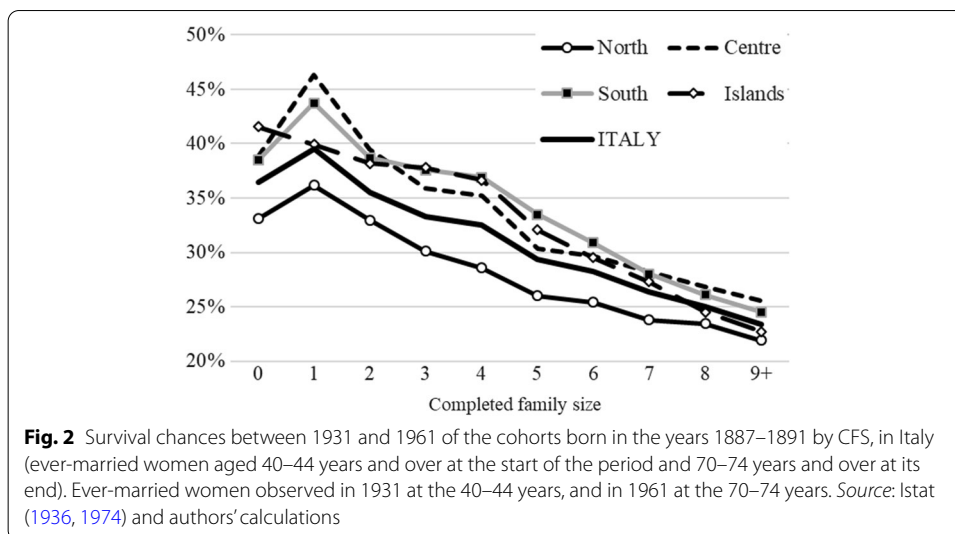
Fertility decline can be a major contributor to the emergence of the female survival advantage at advanced ages only if mortality depends on the number of children women had in the course of their life (or CFS—completed family size). We will now show that this was indeed the case in all the Italian cohorts of women that we could investigate.

Figure 1 refers to the cohorts born before 1887, and shows their survival chances between 1931 and 1961, when these women passed from 45 years and over to 75 years and over. These probabilities are calculated as a simple ratio between two enumerations of the same birth groups of women from two population censuses. Both in 1931 and in 1961, the supplementary fertility questionnaire also reveals the number of children these women had ever had, which permits us to compute their CFS-specific survival chances.

On average, survival was close to 6%, over 30 years, for this group of adult-to-old women. However, it varied significantly by parity: from 10.8% for parity one to 3.0% for parity 9 and over. Levels differ slightly, but the pattern is the same for all the macro-regions (with the partial exception of the Islands, Sicily and Sardinia, at parity 4). Only (ever-married) childless women deviate from this scheme: their survival chances are comparatively low (6.9% on average). Unfortunately, we cannot determine why this happens: we submit (but cannot prove) that their health was already poor at younger ages, and this is why they did not have children, despite their marital status.

Of course, this rough analysis is subject to biases from several possible confounders. Let us consider a few.

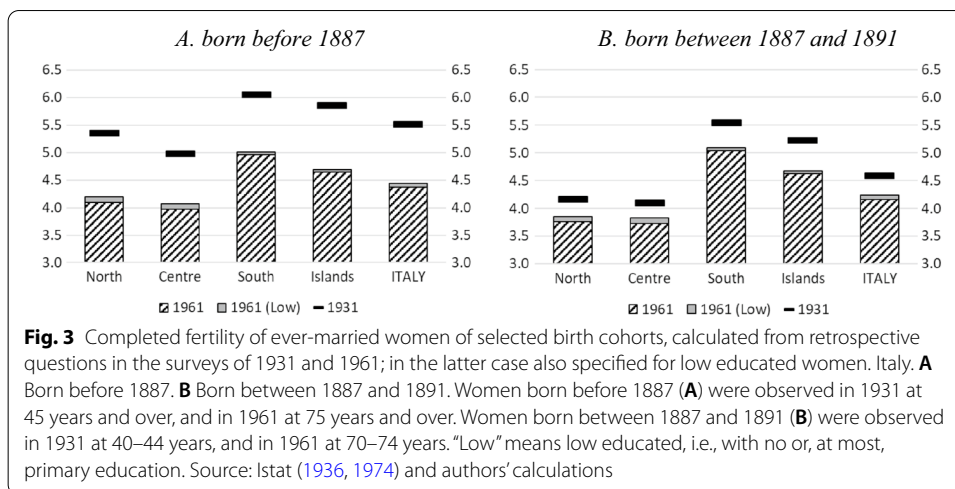
Migration Italy has traditionally been a country of emigration, at least until 1973. However, in the years between 1931 and 1945 international emigration was negligible, because fascism discouraged it; because the United States (the main destination country) had introduced restrictions already in the early 1920s; and because, after the economic crisis of 1929, only a few working opportunities were available abroad. After the



end of the Second World War, emigration rose again, but at that point the cohorts of women covered by our analysis were already relatively old (59 years and over): only very few of them moved abroad. Besides, migratory movements were not uniform over the territory: international emigration was large from the south, but virtually absent from other regions. Internal migration was, once again strong from the south and directed towards Rome (the capital city) and towards the industrial north. Migration (of all types: international, return and internal) is unlikely to be a relevant confounder in our analysis, for two main reasons. The first is that it needs to be parity dependent, which it was not at that time, when migratory movements were essentially driven by men (for work reasons), with their families following (or accompanying) them, regardless of the extent of the progeny. The second reason is that, if it were a confounder, we could not observe the same pattern of parity-dependent survival all over Italy.

Change of marital status between 1931 and 1961 This, too, is unlikely to be a major confounding factor, because these women were already relatively old in 1931 (45 years and over): with permanent celibacy (i.e., at 45 or 50 years) traditionally close to 10% (De Santis & Rettaroli, 2008), the margin of error is negligible, here. As non-marital fertility was also extremely rare in these old cohorts, late marriages may, at most, have inflated the number of childless ever-married women in 1961, whose (relatively poor) survival chances may therefore have been slightly overestimated. In all cases, this does not affect the relation that we are studying here, between past fertility and current survival.

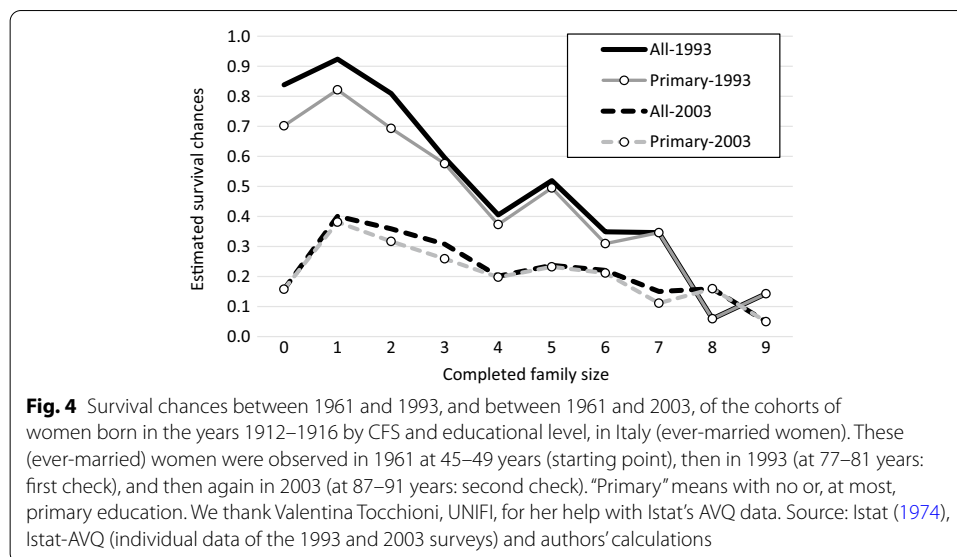
Selection on younger, less fertile cohorts The cohorts considered in Fig. 1, born before 1887, were aged 45 and over in 1931 and 75 and over in 1961. If older cohorts (those that we can no longer observe in 1961 because already extinct) were more fertile, this can bias our observation, because the apparently higher survival chances of women with low CFS may depend on their younger age at the start, given that high CFS women were predominantly old (and from older birth cohorts). Unfortunately, we cannot examine this objection in detail because of the large final age class used in the tabulation of the 1961 fertility survey: “women born in 1886 or before”. However, we can repeat the analysis for a younger group of cohorts, born in the years 1887–1891 (Fig. 2). These women



were “only” 40–44 years old in 1931 (first observation), which means that some of them may have had additional children later on (very unlikely to be a first child, however). Therefore, survival with high CFS may be slightly overestimated, while survival at low CFS may be slightly underestimated: if anything, this potential bias runs counter to our hypothesis. However, the general pattern of Fig. 2 is the same as in Fig. 1: apart from childless women, the relationship between CFS and survival is negative and differences are large: for instance, women aged 40–44 with just one child have markedly higher chances to survive for the next 30 years than women with 9 children and over: (39.5% and 23.4%, respectively).

Selection on education Another possible confounder is education: if highly educated women have fewer children and live longer, the relationship between fertility and survival may be spurious, at least in part. Due to data limitation, we cannot fully control for this variable, but we can indirectly appreciate its effects by looking at Fig. 3. Education level was not reported in the 1931 fertility survey, but it was in the 1961 version. Therefore, we can calculate total cohort fertility on both sources, and cohort fertility of low-educated women only in 1961. We did this for the two groups considered before: born before 1887 (panel A), and born between 1887 and 1891 (panel B). Note that cohort fertility depends on when retrospective questions are asked: it declines over time, because women with high CFS die earlier than others (selective mortality). The effect is very strong on older cohorts: 30 years later, their fertility seems to have declined from 5.5 to 4.4 children per ever-married woman. This effect is not negligible even on more recent cohorts (about half a child less), despite their younger age, which is associated with lower general mortality, and their still incomplete “original” fertility, which was calculated in 1931 when these women were “only” aged 40–44 years.

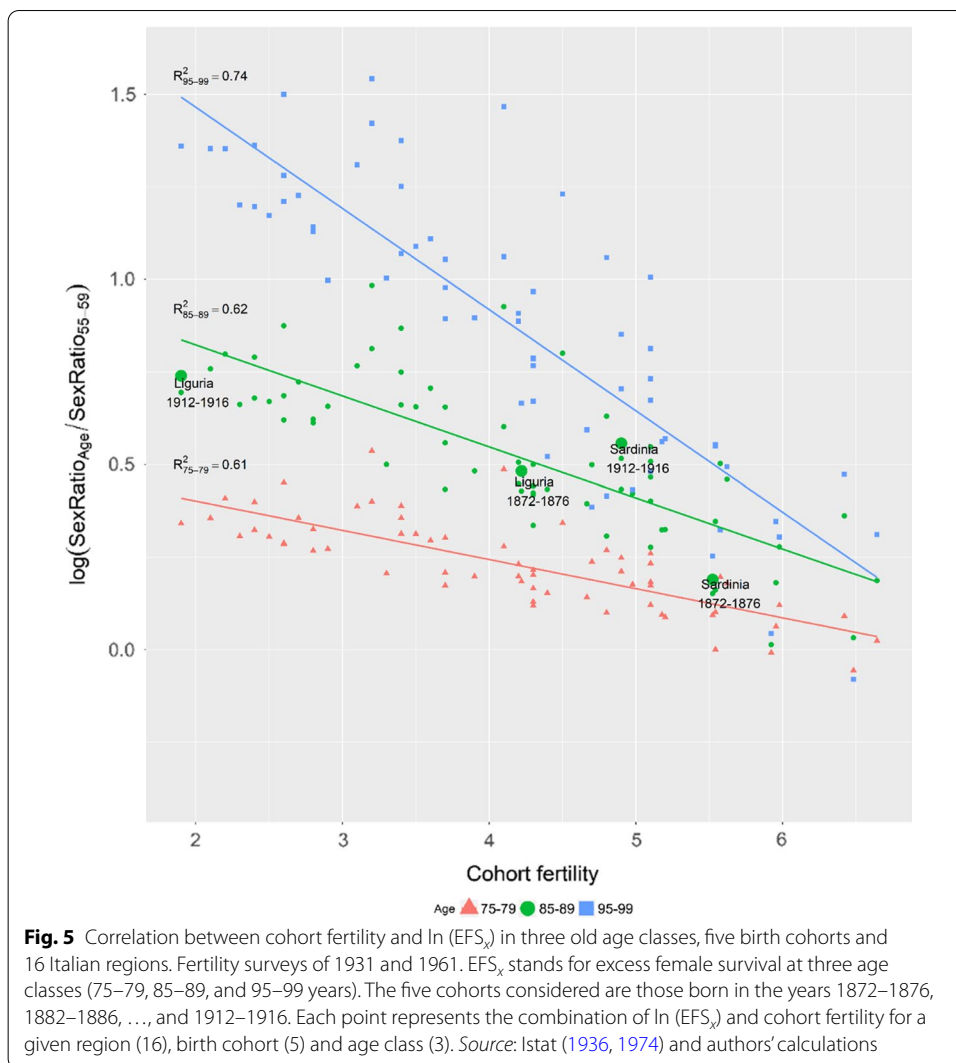
Second, the general picture changes only marginally if we restrict the analysis to ever-married women who were poorly educated in 1961, where “poorly” means that they had at most completed the primary cycle (5th grade). This selection on low education offsets, to the best of our possibilities, selective mortality: without correction, highly educated women (with low fertility and low mortality) would be over-represented in 1961.



Restricting the analysis to those who were only poorly educated in 1961 may introduce a small bias in the opposite direction, because, for these cohorts, we are now comparing general fertility in 1931 to education-specific fertility in 1961 (low education, associated with higher fertility). However, this does not alter the general conclusion: selective mortality operates strongly, and women with high fertility die earlier.

Tapping a different source (Istat’s survey *Aspects of Everyday Life*, 1993 and 2003 rounds), we can replicate the analysis for the birth cohorts of 1912–1916 (Fig. 4), and measure survival chances between 1961 and 1993 (from 45–49 to 77–81 years) and between 1961 and 2003 (from 45–49 to 87–91 years). In this case, the margin of error is greater, because the 1993 and 2003 surveys interviewed only a sample of women, whereas the 1931 and 1961 fertility surveys were conducted together with the population census, and questionnaires were administered to all respondents. However, the general pattern is the same: childless women are not particularly well off while high CFS’s are associated with markedly lower survival chances. As shown in Fig. 4, the relation holds also if we restrict the analysis to low-educated women (with primary education or less): their survival chances are slightly lower but the general pattern is the same.

The relatively poor survival performance of childless married women does not seem to depend, as Bolund et al. (2016) suggest, on misclassification (“individuals may be classified as non-reproducers when there is a lack of data”, p. 6), for several reasons. First, the pattern emerges also on recent datasets (see Fig. 4), arguably of better quality. Second, it has been noted also elsewhere, for instance in Norway (Lund et al., 1990), Israel (Di Jaffe et al., 2009), Bangladesh (Shih et al., 2020), and in Zeng et al. (2016)’s meta-analysis. Third, it makes sense, biologically speaking, because the estrogens activated by pregnancy exert a protective role against various forms of cancer (above all, breast cancer; Zeng et al., 2016). Finally, parity 0 may be a marker (proxy) for health problems in the woman, which may well lead to (slightly) higher mortality at later ages.



The association between female extra survival and the fertility decline

Having identified a link between completed family size and post-reproductive female survival, we can now assess its role in the emergence of the female survival advantage at later ages. To do this, we regressed the natural logarithm of EFS_x (with $x = 75-79$, $x = 85-89$, $x = 95-99$) on cohort fertility.² Each point in Fig. 5 represents cohort fertility and $\ln(EFS_x)$ for a specific combination of region (16), cohort (5) and age class (3): 240 points overall. We highlighted a few points to ease the interpretation of the figure: ages 85–89 years of the 1872–1876 and 1912–1916 birth cohorts in Liguria, a forerunner in the fertility transition, and Sardinia, a latecomer.

Two main indications emerge from Fig. 5. First, higher cohort fertility is systematically associated with lower relative female survival chances in post-reproductive ages. Second, the influence of fertility on the sex ratio becomes stronger with age: for

² We used a logarithmic transformation because logarithms can be negative, which confers more flexibility to regression models.

Table 4 Checks on the relation between fertility and excess female survival at various old ages

	Dependent variable								
	ln (EFS ₇₅₋₇₉)			ln (EFS ₈₅₋₈₉)			ln (EFS ₉₅₋₉₉)		
	Estimate	SE	p value	Estimate	SE	p value	Estimate	SE	p value
All eight cohorts (1862–1866, ..., 1932–1936)									
Intercept	0.774	0.104	<0.001	1.072	0.114	<0.001	1.713	0.157	<0.001
Fertility (<i>I_t</i>)	- 1.112	0.142	<0.001	- 1.865	0.256	<0.001	- 3.336	0.399	<0.001
Survival	- 0.409	0.118	<0.001	- 0.104	0.205	0.612	7.734	2.157	<0.001
	df=93	R ² =0.46	S=0.43	df=93	R ² =0.54	S=0.67	df=77	R ² =0.70	S=0.93
Three youngest cohorts (1912–1916, 1922–1926, 1932–1936)									
Intercept	0.727	0.109	<0.001	0.698	0.193	0.001	0.861	0.254	0.006
Fertility (<i>I_t</i>)	-0.511	0.124	<0.001	-0.669	0.282	0.013	-1.389	0.394	0.002
Survival	- 0.685	0.106	<0.001	- 0.667	0.300	0.035	4.281	4.158	0.325
Male LCM	0.003	0.001	0.001	0.007	0.002	0.002	0.011	0.003	0.004
Female LCM	- 0.006	0.003	0.019	- 0.009	0.010	0.192	- 0.008	0.022	0.361
	df=43	R ² =0.78	S=0.09	df=27	R ² =0.71	S=0.18	df=11	R ² =0.84	S=0.53

Regression of EFS_x (excess female survival) at various ages (75–79 years; 85–89 years; 95–99 years). The main independent variable is Princeton’s index of general fertility (*I_t*), used as a proxy for cohort fertility, while survival is introduced as a control. The upper part of the table refers to all the cohorts in our dataset. The lower part considers only the three youngest cohorts, for which we can add an extra control on lung cancer mortality (LCM) at 75–79 years, used as a proxy for the prevalence of smoking. Standard errors (SE) have been computed with the heteroskedasticity- and autocorrelation-consistent sandwich estimator (Cribari-Neto, 2004). The *p* values for the coefficients of “*I_t*”, “male lung mortality” and “female lung mortality” refer to a one-tail t test. For each regression, we show the degrees of freedom (df), the explained variance (*R*²) and Shapiro’s test on the normality of regression residuals (*S*), where *S* < 0.05 signals a probable departure from normality

example, at age 95–99 fertility alone explains about three-quarters of the observed variance in differential gender mortality (*R* squared = 0.74).

To go beyond a simple correlation and have a more robust and informative analysis we should include more birth cohorts. Unfortunately, as shown in Table 3, we do not have completed fertility histories for all the eight cohorts of potential interest. However, as a proxy for cohort fertility we can use Princeton’s index of total fertility *I_t* (in paragraph 2.1 we showed that the two measures are strongly correlated).

In Table 4, extra female survival (EFS_x) at 75–79, 85–89 and 95–99 years is regressed on (the proxy for) cohort fertility and on general survival, measured with the *N_x/N_e* index, where *N* is the number of members of each cohort at ages “e” (end of reproductive period, 55–59 years in this case) and *x* = 75–79, 85–89 and 95–99, respectively. The introduction of a general measure of survival captures a possible spurious correlation: if societal progress involves both lower fertility and lower mortality, the latter could be the true driver of the changes in the dependent variable considered here, EFS_x. Results are mixed: holding other variables constant, the general increase in survival per se tends to reduce extra female survival up to the age class 85–89; at later ages the effects reverses or, with additional covariates, becomes non-significant. While the change in sign may depend on selection (Manton & Stallard, 1981), for the sake of our argument the important thing is to keep this potential source of bias under control.

Getting back to the focus of this article, the most important result of Table 4 is that higher cohort fertility, consistently and very significantly lowers extra female survival at all post-reproductive ages. This holds not only for all the eight birth cohorts under

study taken together (first section), but also for the three most recent birth cohorts of the second section, where we could control, separately, for men's and women's lung cancer mortality (LCM). Note that these covariates appear to affect the dependent variable EFS_x significantly and with the expected sign: positive for men and negative for women (the more women smoke, the less they survive, the lower the dependent variable).

However, what matters here is that these additional controls do not affect our relation of interest: high fertility consistently and significantly reduces excess female survival at all post-reproductive ages.

Conclusions

In the cohorts that went through the fertility transition in Italy, i.e., those born up to 1912–1916, higher completed family size (CFS) was associated with higher mortality in women's post-reproductive years. Therefore, the fertility transition, which consisted essentially in the rapid decline and eventual disappearance of large progenies, may also have had a long-term beneficial effect on late female survival. This may well have contributed to the historical increase in the female-to-male ratio in old age observed until the end of the twentieth century in Italy. We cannot say exactly how important this effect was, but the good fit of our regression results (Fig. 5 and Table 4) suggests that it was not negligible, at least for certain age classes and birth cohorts.

Our results are all in all consistent with those of Gagnon et al. (2009), who also found a trade-off between longevity and fertility, except that they concluded that “fertility may need to be fairly high for the trade-off to be revealed”. In our case, instead, the trade-off between fertility and late-life mortality is apparent already at low parities (see Figs. 1, 2 and 4). A possible explanation for this difference is that, contrary to in Gagnon et al. (2009) we do not assume monotonicity in the relationship between parity and late-life mortality. Parity 0, in particular, is systematically associated with lower survival than parity 1, a non-linearity that cannot be ignored, if one wants to build an all-encompassing model.

Our analysis is based on aggregate data: the advantage is that we deal with a very large number of observations (with the exception of Fig. 4), but the downside is that we cannot control for all possible confounders. However, the relationship that we posit here did pass all the tests that we tried, for instance using different sources, different indicators, different ages, focusing on selected subgroups, and introducing controls.

Completed fertility does not represent the full health cost of childbearing: miscarriages, for instance, are ignored in our analysis, for lack of data. However, if these events move more or less in parallel with live births, as it seems reasonable in historical birth cohorts, our conclusions remain unaffected. If instead, some women had few live children because of repeated episodes of miscarriages, our conclusions are reinforced: these women ended their reproductive careers with relatively few children, and, despite these episodes that could have impaired their health, they benefitted from a higher-than-average survival. Of course, the exception of nulliparous women should be stressed: their mortality is relatively high and we cannot assess the possible impact of poorly terminated pregnancies.

Another factor that is worth mentioning is early widowhood (not followed by remarriage, because data limitation forced us to exclude re-married women from our

analysis). Early widowhood can be a cause of both low fertility and worse health and survival: this notwithstanding, with the exception of nulliparous women, low fertility characterizes women with better survival chances in their post-reproductive ages, which suggests that the causal relationship that we are positing here (high CFS leading to higher post-reproductive mortality) is stronger than possible offsetting forces. A by-product of our analysis is the note of caution that emerged from the study of “completed” cohort fertility, when calculated through retrospective questions: selective mortality has the effect of reducing this variable as (female) respondents get older and increasingly selected towards low parities.

The CFS–high mortality relation that we submit existed in Italy until not so long ago need not persist to our days or in the future. Pregnancy and delivery were an exacting and risky activity in the past, but things have greatly improved nowadays, in all respects (nutrition, prevention, medical assistance, etc.), to the point that the negative health consequences that (we think) existed in the past have now practically disappeared.

Medical progress may explain why female survival peaks at parity one in our analysis, but shifts to parity 2 in the analysis of the Swedish cohorts born between 1915 and 1960 (Barclay & Kolk, 2019) and rises further to parity 3–4 in articles using surveys conducted after 1960 (meta-analysis of Zeng et al, 2016).

Anyway, all the evidence up to date consistently shows that $CFS=0$ is associated with lower survival than $CFS=1$ and, to a lesser extent, 2. This seems to suggest that part of the reversal observed in the evolution of female extra survival may also depend on the recent increase in the proportion of nulliparous women (see also Luy, 2003). However, if a link between poor health in late life and childlessness seems plausible for the past, when childlessness was mostly involuntary, it is hard to tell whether the same holds among recent cohorts, who increasingly *choose* not to have children (Sobotka, 2017). In all cases, this is something that needs to be investigated with more refined information than we could access here: individual data, true panels, controls for possible confounders, etc.

In conclusion, our findings indicate that, before the end of the fertility transition, higher fertility led to higher female mortality in Italy. Fertility decline may therefore have triggered a new phase in female survival, and explain, at least in part, the evolution of the female-to-male ratio in old age. This connection has been scarcely considered recently, but it may be worth investigating more in depth, if new data become available, and in other countries.

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Author contributions

GS had the original idea, later refined after extensive discussion with all authors: CF and CG aided on the interpretation of results at biological level and worked on the manuscript; VZ focused on sources and methods and MB and GDS on the general structure of the paper. The paper itself was written by GS, GDS and VZ, and revised and approved by all others. All authors read and approved the final manuscript.

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Availability of data and materials

The data used in this study are freely available.

Declarations

Competing interests

None, on part of all authors.

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