Parallel Ageing: The Synchronised Postponement of Fertility and Mortality

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Abstract

Births and deaths have been traditionally studied separately. Not only are they considered two largely disconnected, if not opposite, demographic processes, the use of different and non-comparable measures has also hindered cross-disciplinary studies. We take advantage of a newly proposed analytical framework to illustrate the concrete benefits of comparing mortality and fertility trends. Specifically, we propose leveraging the known limit to female fertility to shed light on the unknown limit to survival. Using percentile-based aggregate measures of mortality and fertility schedules to compare their trends since the 1940s in various low-mortality and low-fertility female populations, we show that for the last 35 years, the pace of mortality and fertility postponement has been surprisingly similar and consistent, forming two parallel lines. This pace persists throughout almost the entire period of fertility postponement and therefore appears independent of the proximity to an upper age-limit. These results exemplify the new avenues of investigation that a common study of mortality and fertility could open. Based on them, we also highlight shortcomings in previous approaches to locate a limit to human lifespan using aggregate measures, thereby refining future research on the subject.

Introduction

As people live longer and delay parenthood to later ages, questions about impending limits to human lifespan and fertility become increasingly pertinent. In this paper, we compare the postponement of births and deaths and leverage the similarities we find to examine how aggregate trends behave at the approach of a limit.

Despite much research effort, no impending limit to human lifespan has been found (Eisenstein, 2022; Vaupel et al., 2021; Zuo et al., 2018; Oeppen & Vaupel, 2002), while the limit to fertility is better known, at least for women. Most women reach menopause between ages 40 and 60 (Daan & Fauser, 2015; Towner et al., 2016). The last birth typically happens a few years before menopause, even in the absence of contraception, due to declines in fecundability and the increased likelihood of foetal loss. Studies of European natural fertility populations consistently show that the average woman had her last child between ages 38 and 41 (Larsen & Vaupel, 1993; Larsen & Yan, 2000; Wood, 1994) and aggregate fertility becomes negligible around age 50 (Eijkemans et al., 2014; ESHRE Capri Workshop Group, 2005; Leridon, 2008; Pittenger, 1973; Wood, 1994). We

consider these ages stable in time. Ages at childbearing have shifted closer to these limits during the fertility postponement over recent decades, although the level of late fertility has not yet reached pre-1950s values (Beaujouan & Sobotka, 2019; Prioux, 2005).

A rich literature explores the factors that (may have) led to the postponement of ages at death and childbearing. In Western countries, ages at death have been postponed over at least the last two centuries (Oeppen & Vaupel, 2002), as a result of different long-run processes. Increased living standards have been put forward as a major factor of mortality decline through the first half of the XX century (McKeown, 1979), but so have medical advances, e.g. vaccination, and improvements in public health and personal hygiene (Mackenbach, 1996). During this time, mortality was largely driven by infectious diseases and so was its decline (Shaw-Taylor, 2020). New leading causes of death emerged after the 1950s in low mortality countries, such as cardiovascular diseases and, more recently, cancer, but mortality continued being postponed through further medical advances and lifestyle changes (Cao et al., 2017; Ezzati et al., 2015).

Fertility has been postponed at various points in recorded history. Most recently, ages at childbearing started rising in Western countries only after the 1970s, much later than ages at death (Sobotka, 2017). There is widespread agreement that lengthening education contributed to this latest postponement, both with a direct and an indirect effect (Ní Bhrolcháin & and Beaujouan, 2012), although this relationship may be partly driven by other factors, such as family values (Tropf & Mandemakers, 2017). Female labour market participation may also affect the likelihood of having children, although this relationship is mediated by country-level policies, cohort and parity (Tomatis & Impicciatore, 2023). Other economic factors that may affect fertility are employment uncertainty (Vignoli et al., 2020) and changes in the perceived economic pre-requisites to parenthood (van Wijk & Billari, 2024). Wider cultural shifts have also been put forward to explain the postponement of ages at childbearing, notably as changes in fertility-related norms and attitudes (Lesthaeghe, 2014) and in patterns of partnership formation (Kuang et al., 2025). Finally, medical advances such as improvements in contraceptive technology have likely facilitated, if not encouraged, recent fertility postponement (Sobotka, 2004). These factors are varied and have probably interacted in various ways as ages at childbearing continued to shift upwards throughout the last three decades.

The long-run mortality postponement and the most recent fertility postponement have been studied as largely separate processes, with distinct driving factors and consequences. Some of these factors, however, seem to overlap more than could be expected. Education is an important driver of both, as it is connected to delayed parenthood and lower chances of death at younger ages (Balaj et al., 2024; Ní Bhrolcháin & and Beaujouan, 2012). Medical advances and healthier lifestyles may also positively affect the ability to have children at older ages, just as they have positively affected the chance to die older ages. Finally, a positive link was found between longevity and late fertility , likely driven by physiological factors (Gagnon, 2015): an interaction between somatic and reproductive ageing seems to take place also in the follicles themselves, which consist of both somatic and germline cells (Fraser et al., 2020; Banerjee et al., 2014). In fact, linked constraints between survival and reproduction form the basis of life

history biology, resting on the premise that evolution balances trade-offs between survival and reproduction to maximise evolutionary fitness (Roff, 2001). Hence, we hypothesise that although the current scientific practice is to study mortality and fertility in separation, their postponement may be more similar than expected. Our results seem to support this claim.

We investigate whether trends of delay in comparable aggregate mortality and fertility schedules share common patterns. To quantify fertility and mortality schedules, we analyse survival percentiles. These capture the fraction of total fertility, or mortality, that happens after a certain age. We use survival percentiles between the 90^{th} and the 5^{th} percentiles (avoiding more erratic data outside that range, as well as the influence of fixed age limits to life and fertility tables) to investigate whether the presence of a limit is influencing the shape of mortality and fertility distributions. This approach allows a direct comparison with previous research on the limits to lifespan (Zuo et al., 2018). If fertility and mortality age distributions share common patterns of postponement, we can examine how the (female) fertility distribution changes as it approaches its known limit to accordingly hypothesise how the mortality distribution might change at a similar distance from the unknown, potential limit to human lifespan. Following (Zuo et al., 2018), we expect the distribution to contract when nearing a limit, leading to decelerating or stagnant postponement of percentile ages, that is the ages when a certain percentage of total fertility, or mortality, is left to occur. Based on results from (Zuo et al., 2018), we expect the mortality distribution to maintain a constant shape over time, suggesting that a potential limit to mortality is not yet in sight. For fertility, we know that ages at childbearing are getting closer to a physiological age limit beyond which women (generally) cannot give birth. Hence, we expect the postponement of the highest fertility percentile ages to slow down or stagnate in more recent years.

We are interested in processes common to all (female) humans, independent of culture, religion, political system or other factors that drive the variation often observed for mortality and fertility patterns across populations (Medford et al., 2019; Rindfuss & Choe, 2015). Therefore, we pool individual populations into what we call a meta-population. We do not pool the individuals that make up each population. Rather, each population contributes with its own population-specific characteristics (e.g. a population-specific age-at-childbearing distribution). Although not explicitly named, this approach has been used before in population studies (Oeppen & Vaupel, 2002). Results may be influenced by changes in the composition of the meta-population (i.e. the individual populations used to create it). We will discuss our case at the end of the results section.

Data and Methods

We use annual period data on female deaths and exposures for ages 65+ from the Human Mortality Database (HMD, (Barbieri et al., 2015)), for 38 populations with at least one million inhabitants in 2019. We further use annual period data on births and female exposures for ages 20+ from the Human Fertility Database (HFD, (Jasilioniene et al., 2016)). In 2019, ages below 65 and 20 accounted for similar proportions of deaths and

births respectively (between 5% and 13% of deaths and between .03% and 10% of births). For both of these data sources, we focus on years between 1985 and 2019, in order to focu on a period of fertility postponement and exclude the COVID-19 pandemic.

For each of these years and populations, we smoothed the age distributions of counts and exposures to the second decimal using the splines R-package. From these, we estimated mortality and fertility survival distributions. The survival curve of mortality was calculated using standard demographic methods as $S(x) = e^{-H(x)}$, where $H(x)$ is the cumulative hazard function. The corresponding fertility survival curve is equal to the cumulative sum of age-specific fertility rates, as a proportion of the Total Fertility Rate (TFR), as shown by (Baudisch & Polizzi, 2025). Note that their equations [1], [24] and [26] are derived for the gross reproductive rate Gross reproduction Rate (GRR) in a one-sex population and for cohorts. Assuming a constant sex ratio and a synthetic cohort, their framework likewise holds for the TFR with period data and a two-sex population, as long as survival is high for women in reproductive ages. This is the case in the populations used in this study. Using survival percentiles as shared measures of fertility and mortality patterns is part of a newly developed, integrated framework, which rests on the Born once, die once approach (B1D1) to quantify fertility (Baudisch & Alvarez, 2021). Baudisch and Polizzi (2025) showed that the B1D1 approach can be translated into conventional notation based on age-specific fertility rates when maternal survival is high. Using the survival curve as defined within the B1D1 framework did not substantively change our results, which are derived within the conventional formulation.

After obtaining the population-specific survival curves, we averaged them within percentiles to obtain year-specific survival curves for the meta-population (results did not change when using the median value). Using this meta-population survival curve, we first estimated a linear model for each percentile, regressing the yearly percentile age on the calendar year. This yielded a single coefficient of the speed of postponement for each percentile and for mortality and fertility separately. We then divided the observation period into 11 five-year windows starting from 1985, each overlapping the previous one by two years. Within each of these windows, we fit a linear model to estimate the coefficient of postponement for different percentile ages (e.g. regressing the year-specific age at which 10% of deaths, or respectively births, had not yet happened on calendar years), again using the metapopulation survival curve.

For our last measure, we were interest in how variable the speed of postponement is across populations. We therefore estimated the delay coefficient for each individual population within five-year rolling windows as described above and extracted the highest and lowest value observed across all populations for each percentile. The range is the difference between the two. All analyses were run on R 4.5.2.

Results

Because the starting and end chronological ages of the lifetables and fertility tables represent fixed limits, we focus on percentiles 90^{th} to 5^{th} throughout the paper. Figure 1 shows the time-trends of mortality and fertility average percentile ages, i.e. the age at which different percentiles are reached, from 1980 to 2019, averaged across our populations. These ages have increased continuously throughout this period[1]. More strikingly, this increase has been largely linear and parallel across percentiles, suggesting that the different percentile ages have shifted upwards at a similar pace.

This visual inspection is confirmed by a set of linear regressions, modelling the average percentile age on year for each mortality and fertility percentile during the observation period. These yield R-squares of above 0.97 for all percentiles, showing that linear models fit the pace of postponement in percentile ages extremely well. Figure 2 shows the associated coefficients. These are clustered closely together: even when comparing mortality and fertility, coefficients largely remain in the range between 0.08 and 0.15. This range narrows to between 0.1 and 0.15 when focusing on the older half of the distribution, i.e. percentiles between the 50^{th} and the 5^{th}. Within this range, the difference between the mortality and fertility coefficients for the same percentile age is always less than 10% of the mortality coefficient (the absolute difference also increases for younger percentile ages). Appendix B shows these differences, as well as the distribution of coefficients and R-squared for each model.

It is important, however, to consider how these coefficients have changed in time. Figure 3 shows the coefficients of linear models estimated in 5-year rolling windows, as described in the *Data and Methods* section. Following Zuo *et al.* (2018)'s hypothesis, we expect fertility, but not mortality, coefficients to show some decline in more recent years, especially at the oldest percentiles, indicating a slowdown in the postponement of ages at childbearing as these approach the known limit to female fertility. This is not what we find. For both mortality and fertility, coefficients remain positive throughout the observation period, indicating a continuous shift upwards of percentile ages. Focusing on the latter part of this period, we see little indication of a compression in the age distributions of either mortality or fertility. There is a slight decline in the values of the mortality coefficients during the most recent time period, but this is in line with previous fluctuations and affects the oldest percentiles less than the younger ones. In fact, coefficients for the oldest percentiles remain above those for younger percentiles, indicating that the right tail is shifting faster than the left (especially the youngest 25% of deaths). The same is true for fertility, although coefficients for younger and older percentiles seem to be converging towards values between 0.11 and 0.13. The pace of postponement has remained comparable across percentiles throughout this period, especially for mortality, and, more recently, for fertility. This pace is also comparable for mortality and fertility, largely remaining in a range of values between 0.07 and 0.15. Similarities between mortality and fertility coefficients also emerge for population-

[1] The only exception are the highest fertility percentiles, which only started increasing in the 1980s and 1990s, but have been steadily going up since

specific patterns, but with more variation, highlighting the advantage of using a meta-population (Appendix D in the Supplementary materials). Similar coefficients across time, percentile and between mortality and fertility indicate a shared and constant pace of delay of births and deaths with no signs of a contraction in either distribution.

Figure 4 shows trends in the range between the maximum and minimum delay coefficients for all percentiles between the 5^{th} and the 90^{th}, for every five-year window and for mortality (on the left) and fertility (on the right). For both, the range has remained relatively low for the youngest percentiles, with relatively little variation. In contrast, the range was higher for the oldest percentiles (in lighter colours) in the earlier observation years, but has since decreased. By the 2010s, range values for all percentiles had converged towards 0.2 for fertility, while coefficients for the oldest percentiles have recently become more varied for mortality, suggesting that the convergence of their coefficients may have been only temporary.

The number of populations included in the HFD and HMD varies by year, as shown in Appendix A. Yet, our results are robust to changing the sample of populations. We considered only the populations with the highest and lowest median ages at death and childbearing in 2019 (after ages 65 and 20, respectively). We also restricted our analyses to years between 1960 and 2019 for populations present in the databases throughout these years. These results support our conclusions (Appendices E-G). Our results are also robust to changing the starting age of the mortality and fertility distributions (Appendix C), although values may differ.

Discussion

In this paper, we set out to examine how ages at death and childbearing have been postponed throughout the last 30 years. We then investigated how the shape of the fertility distribution changed as it approaches the physiological age-limit to female fertility, to contribute to the search for a similar limit to human survival. For both mortality and fertility, we examined the rate at which percentile ages are increasing and found strikingly similar coefficients across ages. Percentile ages have been increasing continuously and at a comparable pace since 1985. Linear regressions throughout the observation period and in 5-y rolling windows yield coefficient values of between 0.7 and 0.14. Time trends show no clear signs of a compression of the age distribution for either mortality or fertility. Finally, we found that delay coefficients at the highest percentiles have become increasingly similar across populations.

Our results support three main conclusions. All of them are unexpected.

Firstly, we see that, from the years when female fertility started being postponed, the mortality and fertility distributions have been shifting to later ages linearly and at a comparable pace. There is no obvious reason why mortality and fertility delay should move at the same pace, nor in the same linear pattern. In line with previous evidence, actuarial and reproductive senescence may be two expressions of the same underlying physiological ageing process (Fraser et al., 2020; Gagnon, 2015; Banerjee et al., 2014).

Thus, some physiological processes may influence both actuarial and reproductive ageing and constrain the speed of postponement of deaths and births.

However, mortality and fertility postponement are complex phenomena, influenced by other distinct physiological mechanisms and various socio-cultural factors (de Bruin et al., 2001; Leone et al., 2023; Benjamin, 2018; Arbeev et al., 2016; Depmann et al., 2016; Shobeiri & Nazari, 2014; Walter et al., 2012; Pakarinen et al., 2010). That the interaction between all these factors should result in a common pace of delay is frankly surprising. What is also surprising is that fertility postponement follows this pace from the 1990s, when ages at childbearing were farther away from the physiological limit to fertility. We would expect to see stronger evidence of common physiological constraints in more recent years, as ages at childbearing shift upwards. Instead, our results suggest that similarities in postponement exist regardless of how close its physiological limit is. It is possible that fertility and mortality do not only share specific physiological mechanism, but also socio-cultural factors, whose influence has so far been studied separately for mortality and fertility postponement. Appendix H shows the trends of the delay coefficients for mortality and fertility from 1900 to 2019. These were not only more variable across time, but also much more different between mortality and fertility. In this perspective, the similarity in pace that we find since 1985 is even more striking. Crucially, we are not arguing that our results show incontrovertible evidence of such a connection. It also possible that the similarities in the postponement of mortality and fertility are due to the casual concourse of distinct processes. However, the possibility that these two processes, and potentially mortality and fertility themselves, have more in common than currently assumed, especially since the 1980s, holds enough potential for future research to be worth exploring.

Our second conclusion shows the potential of exploring such links in practice. Since the 1990s, the mortality and fertility distributions have maintained a largely constant shape, despite their continued postponement. According to (Zuo et al., 2018)'s hypotheses, this does not support the existence of an approaching limit for either distribution. For mortality, this fits our initial hypothesis and qualitatively confirms results by (Zuo et al., 2018). Not so for fertility. Even when constraining our analyses to fertility at older ages, our results do not detect signals of a limit (see Supplementary materials)[2]. We know that there is an age above which each woman cannot have children, so why do we see no sign of this limit? That the age distribution should contract at the approach of a limit, as proposed by (Zuo et al., 2018), remains a sensible expectation. We believe that the answer is to be found elsewhere.

We propose that the limit of fertility is still too far away to have a visible influence on even the right-most tails of the fertility distribution. Where is this limit? We consider two values. Firstly, age 40.8 is the highest value recorded in the HFD for the 10^{th} percentile age[3]. The average woman in natural fertility European populations also had her last child

[2] The increasing slope at younger percentiles suggests a slight contraction for the fertility distribution, but not as a consequence of slowing improvements at older percentiles.

[3] Until 1916, only data for Sweden are available in the HFD. We chose to use this value because the Swedish 90^{th} percentile age aligns with what is known from other sources.

at age 41 (Wood, 1994). However, infertility between age 40 and menopause was to some extent due to foetal loss (Wood, 1994), which may be mitigated by modern medical advances (Haavaldsen et al., 2010). Moreover, current fertility levels after age 40 remain considerably lower than levels in the late XIX and early XX century (Beaujouan & Sobotka, 2019), suggesting that there remains space for postponement beyond age 41. An alternative benchmark for the limit is age 50, which is the average age at menopause for Caucasian women and the age at which aggregate fertility was close to zero in European natural fertility settings (Eijkemans et al., 2014; Leone et al., 2023). Since this age would determine physiological sterility for a large number of women, it is less likely to have been considerably influenced by public health and medical progress. Moreover, a recent study suggests that Artificial Fertility Treatments (AFTs) have so far contributed little to overall fertility after age 50 (Lazzari et al., 2021). We take these two ages as the lower and upper bounds of the interval where most women will become sterile. There are cases falling outside these confines, but they also fall outside the focus of this paper. The lower bound of this interval is only 26 months above the maximum age of the 10^{th} percentile in 2019 (at 38.8 years for Spanish women). The upper bound is about 11 years above it, still close enough that we expected to detect some sign of it. The maximum age of the 10^{th} percentile is even higher when left-truncating the distribution at older ages (41.6 years for Italian women when only considering births after age 35). Yet, we see no sign of a compression in these cases either.

Despite continuous progress, aggregate trends move slowly. At the current pace, it takes 10 years to shift the distributions by one year of age. If the highest 10^{th} percentile age continues to increase at a constant rate of 0.1 every year, it would take a little over 20 years to reach the lower bound of the fertility limit (age 41), and over 100 years to reach the upper one (age 50). Because of the inertia of aggregate trends, what is close in terms of age, may indeed be quite far in terms of time. Because mortality and fertility share a similar pace of postponement, the same conclusion translates to mortality. This may be the answer to a question we did not think to ask. Previous studies have attempted to find evidence for a looming limit or questioned it : but what does looming mean in this case? Our results suggest that if we ever see signs of a limit in aggregate trends of mortality or fertility, this limit will likely be less than 11 years of age above the current maximum 10^{th} percentile age (14 years for mortality, if we consider the distance to the limit relative to the age range affected by mortality), at least as long as the pace of delay remains unchanged. The question remains as to how much closer in age a limit should be to become visible using aggregate trends, but we are not currently able to answer it. As fertility continues to be postponed, this may change.

Further restricting the ages considered (e.g. deaths after age 85 and births after age 30) affected the trends of the coefficients, but not the main conclusions (see Appendix G). Coefficient values decrease the older the left-truncation is but remain comparable between mortality and fertility. Moreover, the older percentiles are consistently higher than for the younger ones and are also increasing at a faster rate, challenging the hypothesis of a contracting distribution. In fact, the distribution after such higher ages seems to be expanding, as the right tail moves faster than the rest.

Our third conclusion is that the pace of delay for the highest percentiles is increasingly similar across populations and, for fertility, across percentiles. This convergence suggests that while we may not be finding a limit to the delay in mortality and fertility, we may be hitting a limit to its pace. Because this trend is much more marked and stable for fertility than mortality, it is possible that it may be the first sign of an approaching limit. It is also possible that the recent increase in variability for coefficients of the higher percentile ages in mortality is a temporary fluctuation and that they will decline again in the near future. These results are not due to declines in the number of populations included in the datasets, as this number continually increased (including after 2000) and range values are lower in 2019 than in the 1950s. The decline in range for the highest percentiles for fertility is also confirmed when considering higher left-truncation ages, or when focusing only on the 10 populations with the highest and lowest median ages at childbearing and death at ages 20+ (see Supplementary Materials). If the pace of delay is indeed reaching a limit, it may indicate a "stiffer resistance to further progress", especially for populations with the highest ages at childbearing, which however does not necessarily "*[set] any definite limit to it"* (Kannisto, 2001, p. 169) in the near future. At the same time, populations that experienced slower paces of delay in the past, may be benefitting from the experience and medical advances of forerunners and catching up to them. This "stiffer resistance" does not affect lower percentiles, leaving room for the continuing influence from socio-cultural, population-specific factors.

Beyond the broad reason initially mentioned, several further reasons motivated choosing aggregate trends. By not relying on individual exceptional values, we avoid the need for individual level data and involved methods to handle extreme values (Medford et al., 2019). Further, considering the whole mortality and fertility distributions enables comparison of patterns across ages. Thus, we can distinguish changes that affect young and older ages alike from the effect of a limit, which is unlikely to affect trends at younger ages. We also conceive the limits to mortality and fertility not as hard lines, but rather as fuzzy intervals. At the aggregate level, substantial variation in ages at death (van Raalte et al., 2018) and in the onset of menopause (Daan & Fauser, 2015; Dratva et al., 2009) exist and is unlikely to ever reach zero. The established approach of focusing on a few exceptionally longevous individuals (Barbi et al., 2018; Dong et al., 2016; Lenart & Vaupel, 2017) may help determine an upper limit to human lifespans, but the great majority of the population will not reach those ages. By considering the whole mortality (and fertility) distribution, we aim to explore the rule, not the exception.

We believe that using aggregate trends is conceptually more robust than focusing on exceptional cases, but our results suggest that it presents empirical limitations. Decreasing cross-country variation in the speed of postponement may serve as the early signal of an approaching limit. Alternatively, it may be that as long as a potential limit to longevity remains far enough in terms of time, the experience of exceptional individuals will be the only operational tool at the disposal of population scientists looking for a longevity limit. Hopefully, individual variation will be kept into account. Other disciplines (e.g. medicine or biology) may of course be able to theorise a limit to longevity through wholly different methods.

In this paper, we have jointly investigated aggregate trends in fertility and mortality to shed light on their similarities. Our results suggest that these may be more substantial than currently assumed. We show how these similarities may be used to investigate the question of a limit to human lifespans. We believe that if even more such similarities are found, they may open the way for exciting new research, for example in forecasting and modelling fertility. Investigating mortality and fertility jointly is still uncommon, however, and only more research on the topic can show whether similarities between them are pervasive or whether we have stumbled upon the only one. In this paper, we highlight a striking convergence in the pace of delay for fertility and mortality, but we can only hypothesise as to its source. More research is needed to understand whether it is the product of common underlying factors or mere randomness. By virtue of the datasets at hand, we also largely focus on Western populations. Since these populations happen to show the most advanced ages in mean ages at childbearing and death, they are also the most appropriate populations to examine in our case. However, this restriction likely affects the generalisability of our results, as other populations may have different characteristics (e.g. in reproductive health and menopause trends (Leone et al., 2023)). Finally, we have also assumed that the limit to fertility is stable. Despite poor data, various studies find an increasing menopause age in highly industrialised countries (Dratva et al., 2009; Leone et al., 2023; Shen et al., 2019; Varea et al., 2000) and two find a pace of 0.1 per birth year (Gottschalk et al., 2020; Rödström et al., 2003). If the age at menopause is being delayed at the same or at a faster pace than the fertility schedule, it becomes unreachable. This could explain why we see no sign of a limit in fertility trends. If the limit to fertility moves, the same could be true for the limit to longevity. In this case, we may never see its effects. In fact, we may question whether this would be a limit at all.

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Figures

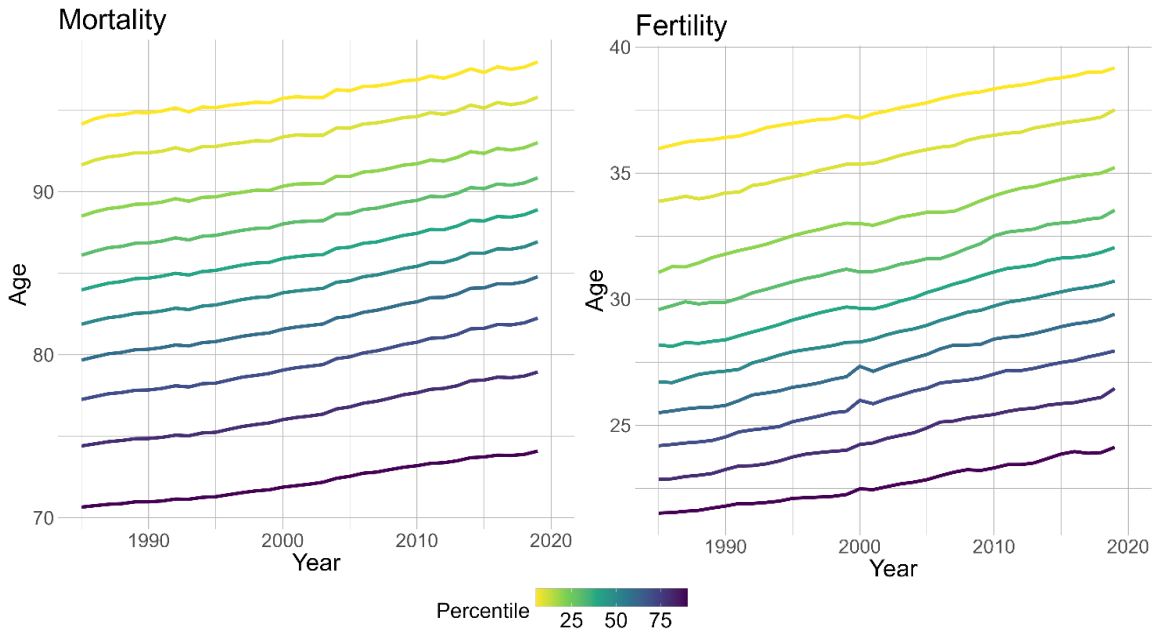

Figure 1. Percentile ages for the mortality (left) and fertility (right) distributions. From darkest to lightest: percentiles 90th, 80th, 70th, 60th, 50th, 40th, 30th, 20th, 10th and 5th. For example, the age at which 30% of birth have not yet happened (fourth from the top) has increased from 29.1 in 1985 to 33.5 in 2019.

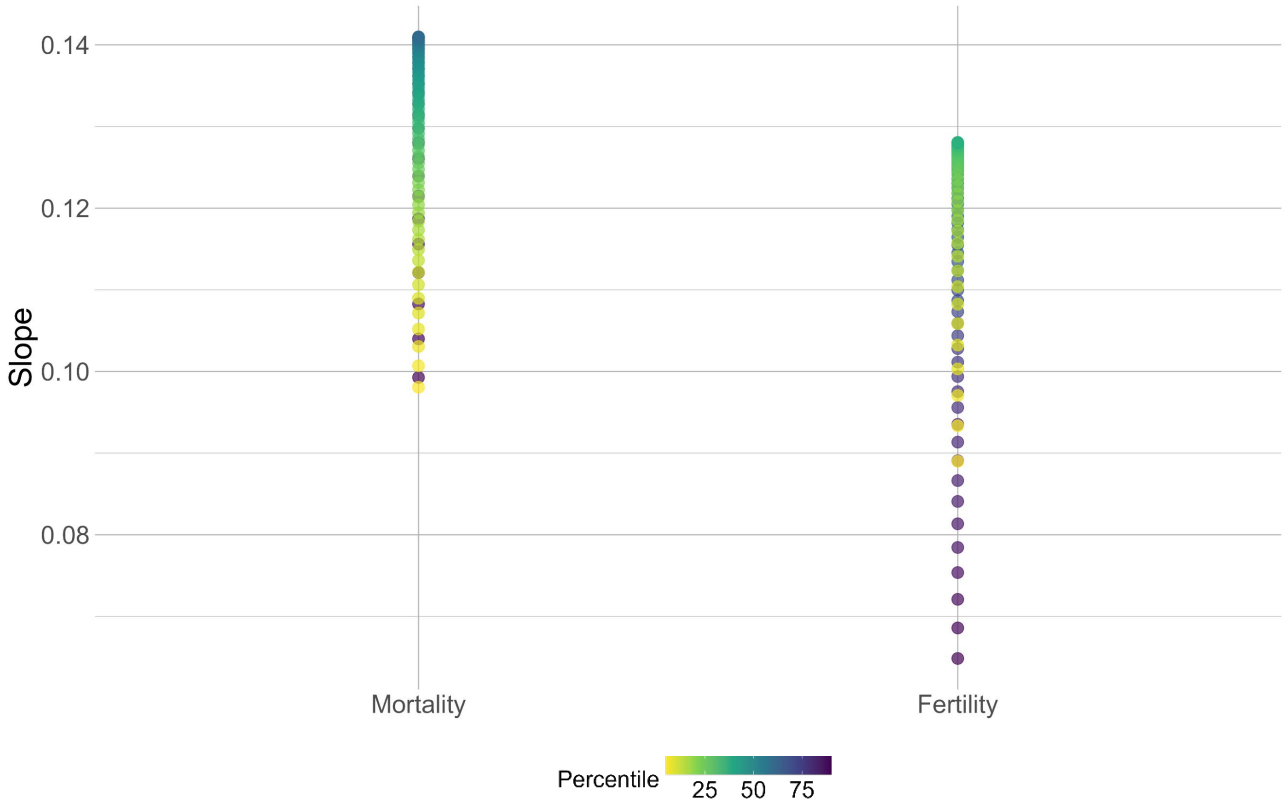

Figure 2. Coefficients of linear models predicting percentile ages with calendar years for mortality (left) and fertility (right), for each percentile age.

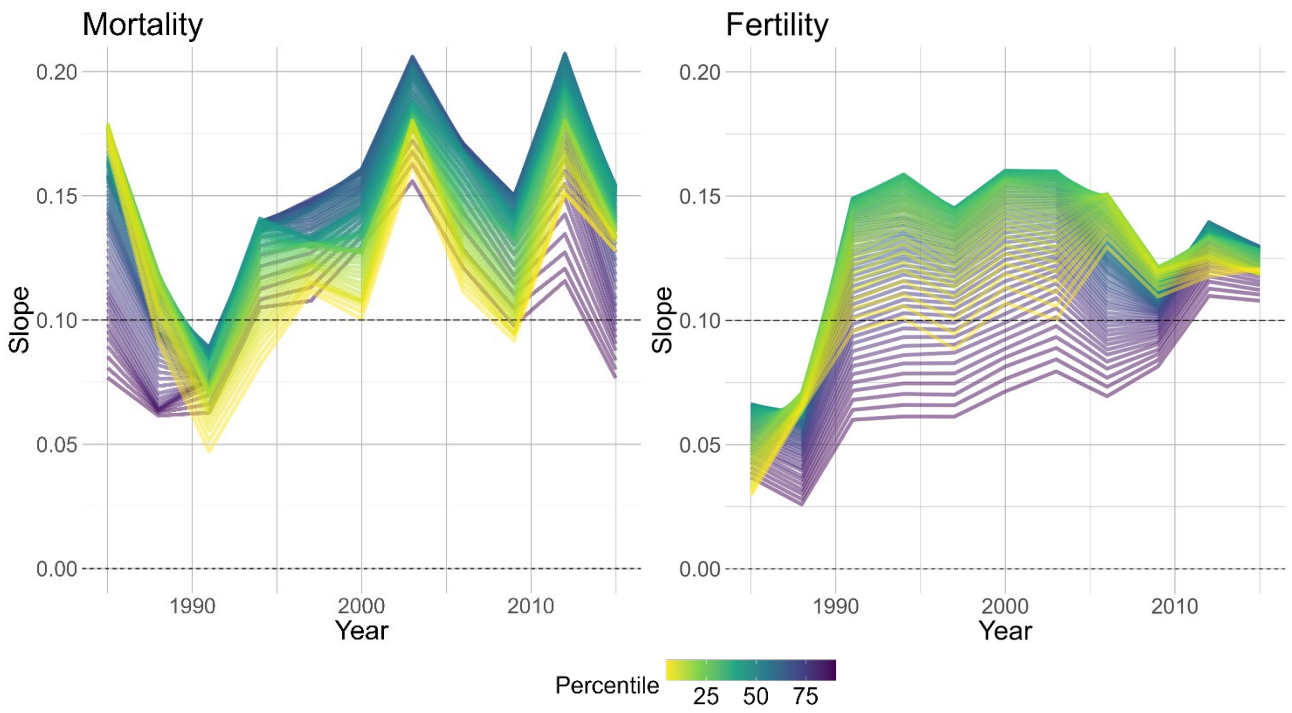

Figure 3. Coefficients of linear models predicting percentile ages with calendar years within five-year rolling windows for mortality (left) and fertility (right), for each percentile age.

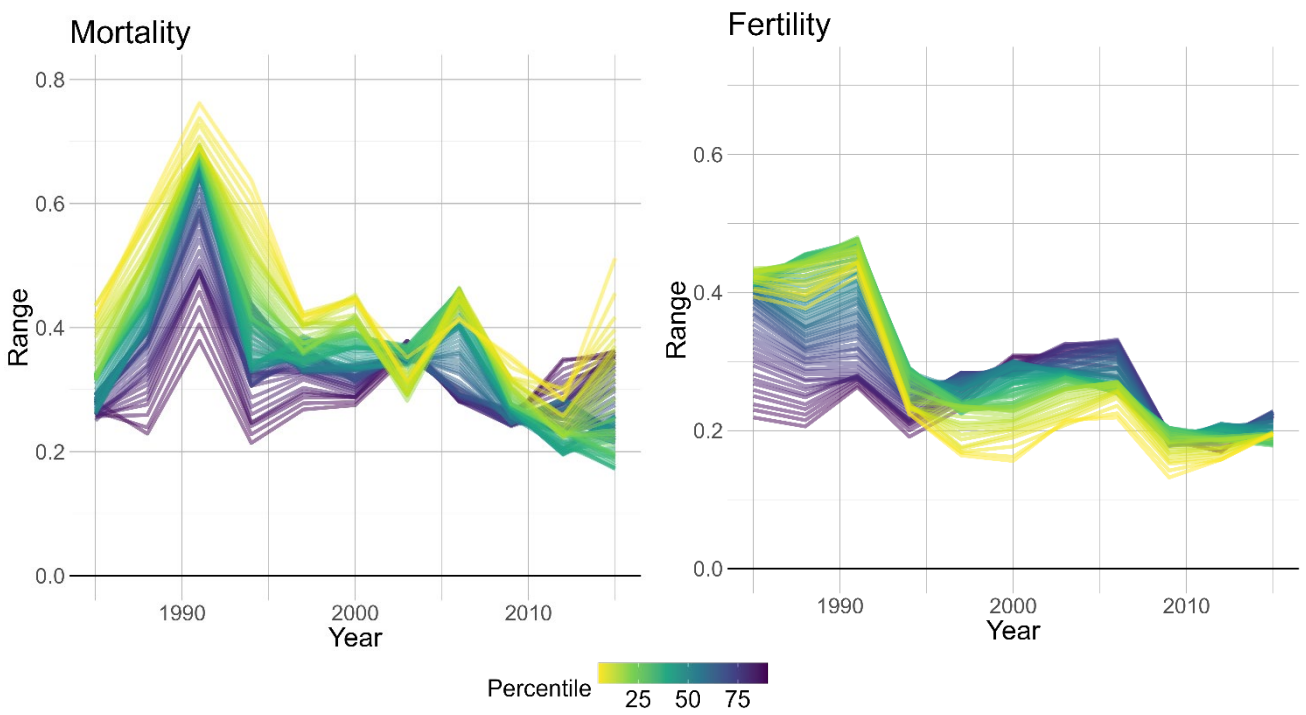

Figure 4. Range of population-specific coefficients, from linear models predicting percentile ages with calendar years within five-year rolling windows for mortality (left) and fertility (right), for each percentile age.

Appendices

See https://osf.io/preprints/socarxiv/b7shw_v2