Early-life reproduction is associated with increased mortality risk but enhanced lifetime fitness in pre-industrial humans

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The physiology of reproductive senescence in women is well understood, but the drivers of variation in senescence rates are less so. Evolutionary theory predicts that early-life investment in reproduction should be favoured by selection at the cost of reduced survival and faster reproductive senescence. We tested this hypothesis using data collected from preindustrial Finnish church records. Reproductive success increased up to age 25 and was relatively stable until a decline from age 41. Women with higher early-life fecundity (ELF; producing more children before age 25) subsequently had higher mortality risk, but high ELF was not associated with accelerated senescence in annual breeding success. However, women with higher ELF experienced faster senescence in offspring survival. Despite these apparent costs, ELF was under positive selection: individuals with higher ELF had higher lifetime reproductive success. These results are consistent with previous observations in both humans and wild vertebrates that more births and earlier onset of reproduction are associated with reduced survival, and with evolutionary theory predicting trade-offs between early reproduction and later-life survival. The results are particularly significant given recent increases in maternal ages in many societies and the potential consequences for offspring health and fitness.

1. Introduction

Senescence is a decline in physiological function, survival and reproductive success with increasing age [1], and a large body of work has shown that senescence in survival and reproductive success is commonly detectable in wild animals [2]. Recent research has switched from establishing whether senescence occurs to quantifying variation between individuals in senescence rates and determining the drivers of such variation. Accelerated senescence has been associated with experience of adverse environmental conditions during early life in wild mammals [3,4] and birds [5]. In addition, individuals with higher reproductive success in early life may experience faster rates of reproductive senescence: red deer (Cervus elaphus) producing more calves in early adulthood showed more rapid senescence [6], and guillemots (Uria aalge) raising more chicks before the age of peak success had lower success once reproductive senescence began [5]. These results support the prediction of evolutionary theories of senescence that, since selection is stronger in early life than in later life, early reproduction should be favoured over later survival [7,8]. Despite these observations, this trade-off may not be apparent: instead, there may be positive covariance between reproduction and survival [9,10] since individuals with plentiful resources experience little constraint while those with fewer resources face more pressing resource allocation decisions [11]. Thus, variation in resources and reproductive investment may create variation in senescence rates.

Understanding reproductive senescence in humans is of increasing interest and importance because the age at which women desire children is increasing...
in modernized society [12]. The physiological factors underlying senescence in humans have been relatively well studied. These include endocrinological changes associated with reproductive senescence and the menopause [13,14], and the cessation of female fertility that occurs around the age of 50 across human populations [15]. Women experience a gradual decline in fertility with age similar to most other placental mammals, including our closest relatives, chimpanzees [16]. The physiology underpinning this is well studied: menstrual cycles gradually change in character, resulting in an increased frequency of anovulatory cycles and ultimately cessation of reproduction [17,18]. Large variation between women in age at menopause [15] suggests that there is likely to be variation between women in their rates of reproductive senescence, but the social and ecological factors which drive between-individual variation in ageing rates have not been well studied. Higher parity throughout reproductive life is associated with variation in susceptibility to different kinds of mortality in humans, particularly heart disease and cancer [19]. However, the consequences of high parity in early reproductive life in terms of a trade-off with later survival and reproductive success, as predicted by evolutionary theory, invoking genes with antagonistic effects on fitness in early and later life [7], selection on resource allocation strategies [8] and energetic costs of reproduction, is unknown. Testing this evolutionary hypothesis could offer fresh insight into the evolution of human life histories and provide information about the long-term consequences of early parity.

We used longitudinal data from a preindustrial human population exhibiting natural mortality and fertility rates, collected from church records during the eighteenth and nineteenth centuries in Finland, to examine age-related variation in reproductive success. We tested the hypothesis that high investment in reproduction in early life would be associated with reduced future survival and a more rapid rate of reproductive senescence, but be favoured by selection through enhanced lifetime reproductive success (LRS). We aimed to quantify: (i) changes in reproductive success with age; (ii) associations between reproductive investment in early life and reproductive success in later life; and (iii) the strength of natural selection on early-life fecundity (ELF).

2. Material and methods

(a) Study population and data collection

We examined age-related variation in reproductive success in women living in Finland during the eighteenth and nineteenth centuries, using data collected from Lutheran church records which has recorded all births, marriages and deaths across the country since 1750 [20]. We used data collected from five ‘parishes’: Hiittinen, Ikaalinen, Kustavi, Pulkila and Rymättylä, in order to construct individual life histories for 2695 women born from 1702 to 1830.

We only analysed data from females who married at least once, since females who never married have an extremely low probability of reproducing. All women in the sample had a known fate up to the age of at least 50. Analysis was restricted to females born before 1851, so that their reproductive lifespan ended by 1900 when improved healthcare and contraception began to influence birth and survival rates [21]. One of the key correlates of fitness in this population is social class and so women were assigned a social class based on their husband’s occupation: rich individuals included farm owners and craftsmen; poor individuals included labourers and crofters [22].

(b) Statistical analysis

(i) Age-related variation in annual breeding success across the whole reproductive lifespan

We began by exploring changes in reproductive success across the reproductive lifespan of all 2695 women in our sample. Our aim was to determine the age at which women were most likely to produce children, controlling for selective disappearance and appearance. Data were structured with a single year for each year of life of each female, giving a total of 86 776 female-years. We first analysed the probability of a female reproducing each year between the age of 16 and 50. We included all years for each individual, including years in which they were not married, in order to examine age-related variation in investment in reproduction rather than fertility per se. We used the ‘glmmer’ function in the ‘lm4’ package in R v. 3.1.1 to perform generalized linear mixed-effects models (GLMMs) with annual breeding success (ABS) as the response variable (0, female did not give birth in a given year; 1, female gave birth), with a binomial error structure and logit link function. We constructed a base model, containing fixed effects of parish (five levels, as described above) and social class (as above) as categorical variables, and age at first reproduction (AFR) and last reproduction (ALR) as fixed covariates. These account for covariance between ABS and the timing of onset (selective appearance) and cessation (selective disappearance) of breeding [23]. We also included random effects of individual identity and year to account for repeated measures and variation in breeding success across individuals and years. Descriptive statistics associated with these data are shown in the electronic supplementary material, table S1.

To this model, we added functions describing the change in breeding success with age and tested which best described the ageing trajectory. We tested models with (i) linear, (ii) quadratic, and (iii) cubic functions of age. We then fitted a series of one-threshold models [24], in which ABS varied as a function of age in two stages (e.g. an increase to the threshold age and a subsequent decline). We tested models varying the threshold between ages 18 and 47. We then fitted two-threshold models, in which ABS varied with age in three stages (e.g. an increase to the first threshold; a plateau to the second threshold; and a subsequent decline). The first threshold varied between 18 and 44, and the second between 21 and 47. We compared a total of 412 models using Akaike’s information criterion (AIC) to describe ageing-related variation in female ABS. A model was deemed to be a statistically better fit to the data if the AIC value was at least ~2 relative to the next best model [25]. In all models, AFR, ALR and age were divided by 100 in order to aid model convergence.

(ii) Association between early-life fecundity and later-life fitness

Our analyses supported a three-stage ageing trajectory for ABS: an increase from age 16 to 25; a plateau between ages 25 and 41; and a decline from age 41 onwards. We therefore considered children born before age 25 to constitute a female’s ELF, which was on average 0.76 ± 0.02 children per female. There were 1507 women who did not reproduce before the age of 25 (55.92% of our 2695 women); 583 (21.63%) produced one child; 409 (15.18%) produced two children; and 146 (5.42%) produced three; 50 (1.86%) produced four or more. We therefore considered children born before age 25 to constitute a female’s ELF, which was on average 0.76 ± 0.02 children per female. There were 1507 women who did not reproduce before the age of 25 (55.92% of our 2695 women); 583 (21.63%) produced one child; 409 (15.18%) produced two children; and 146 (5.42%) produced three; 50 (1.86%) produced four or more. We then determined whether variation in ELF was associated with differences in survival, ABS and child survival in later life. Note that this is separate from AFR: ELF is the number of children a female produced before age 25, while AFR is the age at which a female produced her first child.

First, we assessed the association between ELF and survival in married women from age 25 onwards. We used the R package ‘survival’ to perform Cox proportional hazards models (function ‘coxph’) to determine the predictors of mortality risk in the 2660
women in our sample who lived to at least the age of 25 (35
women from our original sample died before this age). Thus,
we analysed survival from the peak age of ABS as defined by
our initial model. The models included parish and social class,
plus ELF. We statistically compared models fitting ELF as a
factor with two levels (ELF2; no children before age 25, versus
at least one child); three levels (ELF3; zero, one or at least two
children before 25); four levels (ELF4; zero, one, two, three+ chil-
dren); or as a covariate (ELF, a linear function of the number
of children born). We also investigated whether the impact of ELF
varied across social classes by fitting interactions between
social class and the different variables describing ELF, predicting
that any effect would be strongest in poor individuals. We
compared models using likelihood ratio tests (LRTs), where the
χ²-test statistic is calculated as $-2(\text{LogLik}_{\text{model1}} - \text{LogLik}_{\text{model2}})$,
and by comparing model AICs [25].

Second, we investigated the effects of ELF on ABS from the
age of 41 onwards, the age at which our models suggested
ABS began to decline. We restricted this analysis to years in
which women were married: thus, we considered their actual fer-
tility, given the strong effect of being married on probability of
reproduction. We analysed 16 348 female-years in 1922 women.
Once again, we used GLMMs with binomial errors and logit
link, with ABS as the response variable, random effects of indi-
vidual identity and year, and fixed effects of parish, social
class, AFR and ALR. We fitted age as a linear covariate, to test
for a significant decline in ABS, and ELF as a three-level variable
(ELF3, comparing women who produced zero, versus one,
versus at least two children before the age of 25). We predicted
that high ELF would accelerate senescence in ABS, and that
this would be most pronounced in poor individuals. To test
this prediction, we fitted a three-way interaction between age,
social class and ELF3 and all two-way interactions between
these three variables. The model was simplified by removing
non-significant terms in the order of least significance, assessed
by LRTs as outlined above.

Third, we used binomial GLMMs to analyse the association
between ELF and child survival to the age of 15, at which
point individuals are considered independent. We treated off-
spring survival as a maternal trait, based on the fact that it
reflects maternal ability to produce a robust offspring and suc-
cessfully rear it. We analysed the survival of 1614 children
born to 1162 women aged 41–50, to match the ABS analysis
above. The unit analysed was the child, rather than the female-
year (0, the child died before age 15; 1, the child survived to
age 15). The fixed- and random-effect structures of the model
were the same as the model for ABS, including the two-
and three-way interactions. The model also included, as fixed effects,
age as a quadratic covariate, to test the hypothesis that the late-
life change in offspring survival was nonlinear, and the time
since the birth of the focal child’s elder sibling as a fixed factor
(0, the child was firstborn; 1, less than 1 year since the birth of
the last child, up to 7, more than 6 years since the birth of the
last child). This allowed us to distinguish firstborn children
from later-born children, and then the intervals which later-
born children were born at, in the same explanatory variable.
We also fitted the child’s sex and whether or not the child was
a twin as categorical fixed effects. Finally, we controlled for
effects of maternal survival on child survival: maternal presence
affects child survival in this population, but this effect diminishes
after the first few years of life [26]. We fitted a two-level categori-
cal fixed effect describing maternal survival in the first five years
of a child’s life (0, mother was alive until the child was aged 5; 1,
mother died before the child was 5). Again we simplified the
model by removing non-significant terms in the order of least
significance using LRTs. Descriptive statistics associated with
these data are shown in the electronic supplementary material,
table S2.

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(iii) Early-life fecundity and lifetime reproductive success
Finally, we determined whether ELF was under phenotypic
selection through LRS, defined as the number of children born
to a female across her lifetime that survived to age 15. We
regressed LRS on life-history traits potentially associated with
fitness to calculate selection gradients [27]. We fitted linear
mixed-effects models (LMMs, using the ‘lmer’ function in
‘lme4’) with relative LRS, calculated by dividing individual
LRS by its mean, as the response variable with Gaussian error
structure. As explanatory variables, we included social class as
described above, plus AFR and ALR, longevity and ELF as cov-
ariates, standardized to mean = 0 and standard deviation = 1, in
order to account for selection on correlated traits [27]. We also
fitted quadratic effects of all of these covariates in order to test
for nonlinear selection. We tested the significance of each
model term by sequentially removing them from the model and
comparing models using LRTs.

3. Results
(a) Age-related variation in annual breeding success
across the reproductive lifespan
The best-supported model describing ageing-related variation in
breeding success between ages 16 and 50 had thresholds at 25
and 41 years (table 1). This model was statistically supported
over linear, quadratic, cubic and all one- and two-threshold
models, with $\Delta$AIC = −15.80 relative to the next best-fitting
model (electronic supplementary material, table S5). The model
predicted an increase in ABS from almost 0 at age 16 to around
0.3 at age 25, a relatively stable period followed until age 41 before a subsequent steep decline (figure 1). Parameter
estimates from the final model (electronic supplementary
material, table S3) suggested that poor females were less likely
to reproduce at a given age than rich females; females with an
earlier AFR were more likely to reproduce in any given year;
females with a later ALR were more likely to reproduce at a
given age. Thus, women who began reproduction early and con-
tinued childbearing until old age were more likely to reproduce
at any given point in their lives than women who began later and
finished earlier. Comparisons of one- and two-threshold models
are shown in the electronic supplementary material, tables S4
and S5, respectively. These results led us to define ELF as the
number of children born to a female before the peak age of 25
(i.e. reproduction up to and including age 24).
produce any children (hazard before age 25 had similar survival rates to those who did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether they did not reproduce before age 25, whether 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Parameter estimates and test statistics for these models are shown in the electronic supplementary material, table S7. There was generally non-significant evidence that the decline in child survival to age 15 with maternal age was intensified by high ELF (χ² = 5.64, p = 0.060; figure 2). Compared with women who did not reproduce before age 25, there was no statistical evidence for a steeper decline in child survival with age in women who produced one child (age × ELF = 1 estimate = −8.15 ± 9.00), but there was some evidence for a steeper age trajectory in women who produced at least two children before age 25 (age × ELF = 2 estimate = −18.09 ± 7.91). There was no statistical support for the three-way interaction between age, social class and ELF (χ² = 0.03, p = 0.985), providing no support for our prediction that the effect of ELF on senescence was greatest in poor women. There was no evidence that poor women experienced a different pattern of child survival with age compared to rich women (age × poor χ² = 0.16, p = 0.686). Finally, there was no statistical support to suggest that child survival varied as a nonlinear function of maternal age (age² χ² = 0.48, p = 0.488). The final model controlled for lower survival to age 15 among twins compared with

(b) Associations between early-life fecundity and later-life fitness

First, high ELF was associated with an increased mortality risk from the age of 25 onwards: the statistically best-supported Cox proportional hazards model contained ELF as a three-level factor (table 2). Women who produced one child before age 25 had similar survival rates to those who did not produce any children (hazard = 0.96, 95% confidence interval [CI] = 0.87–1.06), but those who produced at least two children before age 25 had a significantly higher hazard of mortality (hazard = 1.11, 95% CI = 1.01–1.22). Poor women had a significantly higher hazard of mortality than rich women (hazard = 1.17, 95% CI = 1.05–1.32), but an interaction between social class and ELF3 did not improve model fit, indicating that the ELF effect on mortality did not differ between rich and poor individuals (table 2).

Second, we investigated associations between ELF and ABS in married women after the age of 40. Parameter estimates and test statistics for these models are shown in the electronic supplementary material, table S6. The decline in ABS after the age of 40 was strong and significant (χ² = 1549.50, p < 0.001).
singletons, and among children whose mother died before they reached age 5 compared with those whose mother was still alive when they reached age 5.

(c) Early-life fecundity and lifetime reproductive success
Our selection analyses revealed that ELF was significantly positively associated with relative LRS, suggesting that it was under positive selection. We tested for a nonlinear association between ELF and LRS, but this was not statistically supported (table 3); instead, the model predicted a linear increase in LRS with increasing ELF (figure 3). Women who did not reproduce before the age of 25 had a mean LRS of $3.00 \pm 0.05$ s.e. children, while women at the top end of the scale, who produced more than three children before the age of 25, produced $5.02 \pm 0.34$ surviving children on average.

This analysis accounted for higher relative LRS in rich individuals; a nonlinear effect of AFR which suggested that starting reproduction earlier enhanced lifetime fitness; and positive selection on ALR, suggesting that individuals who ceased reproduction later also have higher lifetime fitness. There was also a nonlinear effect of longevity, which supported a decelerating increase in LRS with increasing lifespan.
4. Discussion

In this study, we investigated age-related variation in female reproductive success in a pre-industrial population experiencing natural rates of mortality and fertility. ABS increased from age 16 to age 25, followed by a relatively stable phase until age 41 and a subsequent decline. ELF, defined as the number of children born before age 25, was not significantly associated with fecundity after the age of 40 or the rate of senescence in ABS: females with high ELF were no less fecund in later life than females with low ELF. However, higher ELF was associated with increased mortality risk from age 25 onwards and a (statistically marginal) more rapid decline in child survival rate with maternal age. Ultimately, ELF was under positive phenotypic selection: women who produced more children before the age of 25 had overall higher LRS despite apparent survival costs.

The annual probability of giving birth increased from age 16 to age 25. The main driver of this was almost certainly the increase in the proportion of women who were married: in our sample, less than 1% of women were married at age 16; this rose to approximately 51% by the age of 24. We did not include marriage status in these initial models as: (i) all women in the sample married at some point; (ii) we were interested in determining the peak age of breeding success, rather than peak fertility; including marriage status in the model would have predicted probability of reproduction when married, rather than probability of reproduction overall; and (iii) we included AFR to control for selective appearance in the models, which is highly correlated with marriage age (electronic supplementary material, table S1). The predicted probability of giving birth was relatively stable from age 25 until age 40, from which point the probability of giving birth declined.

By age 24, 44% of women had produced their first child and thus had reproduced in what we defined as ‘early reproductive life’ [6,28]. Women who produced two or more children before age 25 had an 11% higher mortality risk in each subsequent year of life than women who did not reproduce in early life, suggesting that investment in reproduction during early adulthood carried a survival cost. Support for the link between lifetime number of pregnancies (gravidity) or births (parity) and long-term health in women is mixed: some have reported the predicted positive association between parity and mortality risk [29], while others have found no association [30], and yet others have found a lower risk associated with very high gravidity [31]. In addition to these analyses of the consequences of total gravidity/parity, are studies of the association between AFR and later-life survival. Such studies have found that early AFR is associated with reduced longevity [32,33]. In the United States, cohorts born from 1931 to 1941, controlling for social status, education, marital status and parity, women who gave birth during their teens had a 42% greater hazard of mortality from the age of 50 onwards, largely due to increased cardiovascular disease, lung disease and cancer [34]. Similarly, a comparison of three modern populations in the UK, Norway and the USA found that mortality risk between ages 50 and 70 was increased 21–57% in women who had their first child as a teenager [35]. However, other studies have found the association only in certain subsets of the population [36] or that later-life survival or longevity is independent of AFR [37,38]. Nevertheless, a general pattern emerges whereby more births and earlier onset of reproduction are associated with a later-life survival cost, which is consistent with our finding that high ELF was associated with increased mortality risk in later life. A potential evolutionary mechanism underpinning this trade-off could be selection for genes promoting early fecundity at the expense of late survival [7], while a likely physiological explanation could be that physiological ‘wear and tear’ leads to an increase in hazard of death from metabolic disease or cancer.

We found that fertility declined rapidly after age 40 in married women. The biological basis of the decline may have been accompanied to some extent by a reduced frequency of intercourse at these ages [39]. However, reduced frequency of intercourse does not significantly affect the likelihood of pregnancy, if intercourse occurs during the fertile window [40]. Despite this, the desire for more children may be lower at later ages, and there is, therefore, likely to be a cultural aspect to the age-related decline in ABS in addition to the biological basis of reduced fertility and eventual menopause. ABS declined significantly from age 41 to 50 in this group, with approximately 20% of women giving birth at age 41, approximately 10% at 45, and zero at age 50. This decline is consistent with the observation of complete cessation of female fertility, across human societies, by age 50 [15]. The gradual decline was probably due to changes in the hormonal profiles of menstrual cycles leading up to menopause: the final 30 cycles before menopause increasingly exhibit delayed ovulation or are anovulatory [18]. However, we did not find that this decline was affected by ELF, and thus found no support for the hypothesis that there would be an accelerated senescence cost of early reproduction [6]. Nor did we find any association between ELF and the annual probability of giving birth during this period. Such a lack of negative association between early reproductive output and later birth rate is not surprising, given that early AFR was associated with higher ABS across all ages, probably reflecting that those females with young age at the onset of reproduction were more fertile overall. This is in line with findings from another long-lived mammal, the Asian elephant (Elephas maximus), where individuals producing many offspring in early life were more likely to produce offspring in later life [28]. However, these results are to our knowledge the first indication of the association between ELF and later-life reproductive success in humans.

Contrary to the results for later-life ABS, we found that high ELF was associated with faster senescence in child survival in older mothers. The probability of child survival declined with maternal age, and this decline was most rapid in women with high ELF (figure 2), suggesting that high investment in early reproduction carried a cost in terms of ability to successfully rear children born in later life. This could reflect social factors: for instance, these children may have experienced competition with elder siblings [41], or their grandparents were less likely to still be alive, compared with their older siblings [42,43]. Our results do not reflect older mothers dying before their offspring and therefore not being present to care for them, since we controlled for this known effect [26] in our models. Physiological explanations could include wear and tear induced by earlier births, which may reduce maternal ability to produce and raise a robust offspring [14]. It is interesting to contrast this result with the finding that ELF did not affect
investing heavily in reproduction before the age of peak fertility had higher lifetime breeding success, despite a negative association between early-life fertility and later maternal survival as found in our study [28].

In this study, we found evidence for contrasting associations between early-life reproductive success and later-life survival and reproduction. Women who produced more children before the age of 25 had a higher risk of mortality after this age, but overall had higher LRS. A caveat to these results is that phenotypic associations may not reflect underlying genetic correlations, and therefore cannot be used to predict past and future trajectories of evolution. For example, non-genetic variation in exposure to disease or ability to access and use resources, and resulting effects on health and physiology, may create positive phenotypic associations where negative genetic associations exist [11,56]. We attempted to counter such effects by controlling for factors consistently associated with variation in resource acquisition, fitness traits and health in our population, chiefly birth year [58–60] and social class [22]. We also note that there is evidence for a genetic correlation between early reproduction and late survival in both human and non-human primates. Previous work on this population observed a positive genetic correlation between AFR and longevity, suggesting that genes for early reproduction are also associated with reduced lifespan [61]. Similarly, a study of Rhesus macaques (*Macaca mulatta*) found a positive genetic correlation between AFR and survival to various stages of adulthood [62]. While AFR and ELF are different traits, these results do suggest that to some extent the trade-off between reproduction at early ages and survival in later life may have a genetic basis, though we do not suggest that our results here provide evidence for such a trade-off at the genetic level. This could be explored further by determining the genetic association between ELF and later-life performance, including senescence rates and longevity: a study of red deer revealed a genetic trade-off between ELF and the rate of senescence in birth weight [63], but similar studies on humans are lacking. Other opportunities for further research include determining changes in the strength of the reproduction-survival trade-off with age [64], which may enable a test of the hypothesis that the menopause evolved as an early cessation of reproduction due to the survival costs of reproduction increasingly outweighing the benefits with increasing age [65]. In short, while the proximate mechanisms of human reproductive maturity and senescence are already relatively well understood, tests of evolutionary theory will enable us to determine the ultimate mechanisms underpinning the unusual human life history.

Data accessibility. The data used for this study are available on request by contacting Dr V. Lummaa (v.lummaa@sheffield.ac.uk).

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