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THE EFFECT OF MATERNAL AGE AND REPRODUCTIVE HISTORY ON OFFSPRING SURVIVAL AND LIFETIME REPRODUCTION IN PREINDUSTRIAL HUMANS

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Senescence is one of the least understood aspects of organism life history. In part, this stems from the relatively late advent of complete individual-level datasets and appropriate statistical tools. In addition, selection against senescence should depend on the contribution to population growth arising from physiological investment in offspring at given ages, but offspring are rarely tracked over their entire lives. Here, we use a multigenerational dataset of preindustrial (1732–1860) Finns to describe the association of maternal age at offspring birth with offspring survival and lifetime reproduction. We then conduct longitudinal analyses to understand the drivers of this association. At the population level, offspring lifetime reproductive success (LRS) declined by 22% and individual λ, which falls with delays to reproduction, declined by 45% as maternal age at offspring birth increased from 16 to 50 years. These results were mediated by within-mother declines in offspring survival and lifetime reproduction. We also found evidence for modifying effects of offspring sex and maternal socioeconomic status. We suggest that our results emerge from the interaction of physiological with social drivers of offspring LRS, which further weakens selection on late-age reproduction and potentially molds the rate of senescence in humans.

KEY WORDS: **Aging, birth order, fertility, senescence, sex**

Senescence arises when an individual's physiological function declines with age. Evolutionary theory predicts such a decrease because the strength of natural selection on alleles that affect reproduction weakens at older ages (where selection strength is quantified by the impact of an allele on population growth; Hamilton 1966; Charlesworth 1994). Empirical evidence for reproductive senescence in wild iteroparous species is now abundant, yet the social and ecological factors that affect age-specific contributions to population growth remain less clear (Jones et al. 2008; Monaghan et al. 2008; Nussey et al. 2008, 2013). An individual's genetic contribution to future generations (termed individual fitness) strongly correlates with grand-offspring numbers (i.e., the numbers of second-generation offspring, hereafter, G2 offspring). This is itself a function of the numbers of first-generation offspring (G1 offspring) produced and their lifetime reproductive success (LRS; Ridley 2007; Gillespie et al. 2008). Thus, descriptions of reproductive senescence, as well as the selection shaping it, should consider the effect of maternal age on all components of

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LRS (Nussey et al. 2008). Failure to do so risks misrepresenting the age-specific strengths of natural selection on reproduction and survival.

Previous wild animal studies have primarily investigated the effects of reproductive senescence on offspring survival to independence or first reproduction in various species of birds and mammals (Nussey et al. 2013). These studies have shown that offspring born to old mothers, or mothers with high levels of previous reproduction, can be less likely to reach adulthood. However, aside from laboratory organisms (Hercus and Hoffmann 2000; Benton et al. 2008), few studies have investigated the effects of maternal age over the entire lifetimes of offspring (Rodel et al. 2009; Bouwhuis et al. 2010; Hoffman et al. 2010). One such study on great tits, *Parus major*, found that the reproductive success of G1 offspring (defined by their production of breeding G2 offspring) senesced earlier and faster among offspring with older mothers (Bouwhuis et al. 2010). However, overall, there was no net effect of maternal age on G1 offspring LRS, because the offspring of older mothers also had relatively higher reproductive success before the onset of senescence. Such studies are vital because the LRS of offspring born at a given maternal age determines the contribution of later-life reproduction to population growth (Arnold and Wade 1984; Charlesworth 1994). Furthermore, the extent to which, across species, the maternal age or reproductive history at an offspring's birth affects the offspring's entire lifetime are unclear. Theoretically, we might expect that such reproductive senescence is more pronounced in longer-lived species where parents make costly investments in few offspring, often involving long periods of dependence (Hamel et al. 2010).

Humans are a species combining long life span and protracted offspring dependence with unusually pronounced reproductive senescence at female menopause. Understanding the causes and consequences of human reproductive senescence also has current social ramifications due to the increasing postponement of childbearing to older ages (Sobotka 2004). However, there are few data on the consequences of delayed motherhood to the offspring born. The potential effects of advanced maternal age at childbirth on offspring survival and reproductive success can be divided broadly into two factors (as illustrated by Goodman and Koupil 2009; Myrskylä and Fenelon 2012). The first is physiological: older mothers can produce lighter offspring with higher mortality risk (Tarin et al. 1998; Djahanbakhch et al. 2007), and/or impaired fecundity and life span in adulthood (Gavrilov and Gavrilova 2000; Tarin et al. 2001; Smits et al. 2002; Nassar and Usita 2009). The same outcomes could also be due to cumulative somatic costs of previous reproduction (Kiely et al. 1986; Jasienska 2009). The second is social: because of protracted dependence, offspring born to older mothers might suffer reduced reproduction or survival if born with older siblings who compete for family resources (e.g., nutrition or wealth; Mace 1996; Hagen et al. 2001; Gibson and Gurmu 2011). Inequality in the inheritance of wealth can be particularly strong among adult male siblings, with later born males experiencing delayed and reduced reproduction (Mace 1996; Gibson and Gurmu 2011). Either effect, physiological or social, could be modified by ecological differences among mothers, such as socioeconomic status (SES), and among time periods or populations due to differences in culture or means of subsistence (Hrdy and Judge 1993; Goodman and Koupil 2009; Gibson and Gurmu 2011). In general, we expect the strength of selection on late-life reproduction and survival to be weakened if later-born offspring are disadvantaged, and so contribute relatively little to population growth (Käär and Jokela 1998; Pavard and Branger 2012). However, despite the rising number of longitudinal, multigenerational human datasets, there remain few studies of how maternal age and/or reproductive history at childbirth affect the survival and lifetime reproduction of G1 offspring.

Here, we use our dataset of preindustrial Finns to investigate the relationship of maternal age at childbirth to G1 offspring survival and subsequent production of G2 offspring who reach adulthood (15 years). We divide our study into three sections. The first describes the population-level relationships of maternal age at childbirth to G1 offspring LRS and individual λ. LRS is the number of G2 offspring alive at 15 years. Individual λ is a similar metric but, unlike LRS, is also sensitive to the timing of G2 offspring production (McGraw and Caswell 1996; Käär and Jokela 1998). Earlier reproduction increases individual λ; accounting for reproductive timing makes individual λ a more accurate measure of individual fitness in a growing population. The second section decomposes G1 offspring LRS into components of: (i) survival to 15 years, (ii) production of a G2 offspring by a G1 offspring who had survived to 15 years, and (iii) number of G2 offspring alive at 15 years, given at least one G2 offspring was born. We estimate the variation in these traits predicted by maternal age at first reproduction (AFR) and G1 offspring birth order. Finally, we show how the results of these analyses explain the population-level trend in LRS by mapping the predictions from each model back to the maternal age at each G1 offspring's birth.

Materials and Methods

STUDY POPULATION

Our data come from church registers where local clergymen recorded dates of marriage, birth, and death for each parish area, and details of interparish migration. From an initial sample of 560 mothers who produced a G1 offspring, we tracked the lifetimes of their 3751 G1 offspring and the survival of their 8709 G2 offspring to 15 years (Lummaa 2007). We sampled mothers from four parishes: 98 mothers with 724 G1 offspring from Hiittinen, 238 mothers with 1816 G1 offspring from Ikaalinen, 114 mothers with 607 G1 offspring from Kustavi, 110 mothers with

604 G1 offspring from Rymättylä. These mothers were born in years 1710–1816, gave birth to G1 offspring in years 1732–18--59, and their latest born G2 offspring reached 15 years in 1917. This period largely precedes the declines in fertility and mortality associated with industrialization (Korpelainen 2003; Liu et al. 2012). We used these data to build a social pedigree that linked offspring survival and reproduction to maternal age at childbirth. Given the serious punishments for adultery (Sundin 1992), we had high confidence that our social pedigree closely reflected the underlying genetic pedigree of our population sample. At most, it is unlikely that extra-pair paternities exceeded the median rate of 3% in populations where men have high confidence of being the genetic father (Anderson 2006).

Based on the occupation of each mother's first-husband, we categorized maternal SES as either landless (renting or with no access to land) or landowning (Gillespie et al. 2008). These socioeconomic categories were therefore broad, comprising roughly equal proportions of our data sample. Land inheritance generally occurred at marriage and favored first-born sons, although daughters could inherit in the absence of a male heir (Moring 2009). Divorce was virtually impossible; remarriage occurred only after spouse death and rarely affected the inheritance rights of the first-husband's offspring (Moring 2002). As in other preindustrial European societies, land ownership reduced the age at marriage and increased the numbers of both G1 and G2 offspring (Voland 1990; Pettay et al. 2007; Gillespie et al. 2008). Migration rates were low: approximately 50% of individuals married within the village of their birth; those that married outside this village commonly settled in a neighboring area (Nevanlinna 1972). Although this limited the loss of individuals from our dataset due to migration, migrants were most likely to be of low SES and/or high birth order (Beise and Voland 2008; Moring 2009). Full reproductive histories could be tracked for 87% of the 1841 G1 offspring of landless parents, and 94% of the 1910 G1 offspring of landowning parents. Here, we define "full" reproductive history by whether an individual was tracked in the church registers until death or the 95th percentile of reproductive life, 45 years for females, 56 years for males.

Statistical Analysis

POPULATION-LEVEL ANALYSIS

First, we investigated how the production of G2 offspring alive at 15 years by G1 offspring varied with maternal age at the G1 offspring's birth. We considered G1 offspring born to all consecutive husbands of each mother. We then restricted our full sample of 560 mothers and 3751 G1 offspring to 554 mothers and 3391 G1 offspring with full reproductive histories. For each 1-year maternal age class between 16 and 50 years, we calculated the weighted mean and variance of LRS and individual λ among the G1 offspring produced. In computing individual λ, we counted fertility as G2 offspring who survived to 15 years and assigned these offspring to the maternal age at their birth (Käär and Jokela 1998). We also multiplied the number of G2 offspring produced at each G1 offspring age by 0.5, which accounts for the average genetic relatedness between parent and offspring (McGraw and Caswell 1996). The weights used in computing these metrics were proportional to the percentage of each G1 offspring's G2 offspring successfully tracked to 15 years. Thus, more weight was given to records that were more complete. Analyses of the trends in LRS and individual λ by maternal age at the G1 offspring's birth were linear regressions, with a further set of weights input into the model. These latter weights were inversely proportional to the variance of LRS or individual λ among the G1 offspring born at each maternal age. Maternal ages with less than five observations were assigned one-tenth of the smallest weight among the more abundant ages (Jones et al. 2008). Thus, more weight was given to data points with greater measurement accuracy.

INDIVIDUAL-LEVEL ANALYSES

Here, we considered only offspring from each mother's first-husband. This simplified the interpretation of our individual-level results by removing any "husband effects" or effects of the interval between maternal marriages. To target our analyses further at the trends within mothers, we selected only mothers with at least three longitudinal data points. This removed mothers whose childbearing history could not reliably inform the longitudinal trends. Table S1 gives the final sample size in each analysis. Multiple births (e.g., twins / triplets) were always assigned the same maternal age at childbirth and offspring birth order. We implemented our analyses with the R package MCMCglmm (Hadfield 2010), which fits Bayesian generalized linear mixed models (GLMMs). The most likely fixed-effect structures were determined using the Deviance Information Criterion (DIC; Spiegelhalter et al. 2002). See the Supplementary Information for full details on the analysis and model fitting. Survival to 15 years and reproduction given survival to 15 years were binary variables, analyzed with a logit link function. The number of G2 offspring alive at 15 years, conditional on at least one G2 offspring birth, was analyzed with a Poisson error structure, a log link function and the estimation of additive overdispersion. All models had a single random effect: maternal identity, which accounts for the nonindependence of G1 offspring with the same mother.

We considered the control variables: total number of each mother's G1 offspring (as a linear and quadratic function), the parish of a G1 offspring's birth (as a 4-level factor), and the year of a G1 offspring's birth (as a linear function). See the Supplementary Information for explanation of these variable specifications. The most important control variable was each mother's total offspring number, the estimation of which is essential for the reliable

description of the within-mother trend by G1 offspring birth order (van de Pol and Verhulst 2006; Nussey et al. 2008). We therefore fixed the linear function of total offspring number to appear in all analyses; all other control variables were subject to selection by DIC.

We structured the linear and quadratic functions of maternal AFR and offspring birth order (BO), to form the surface

$$AFR + AFR^2 + BO + BO^2 + AFR * BO, \quad (1)$$

where + shows terms combined additively and * shows interactions. The advantage of this specification is that it separates the (strongly collinear) effects of maternal age at offspring birth from those of offspring birth order. Some studies analyze both competing variables in the same model (e.g., Kiely et al. 1986). However, our approach leads to a clearer interpretation by estimating how maternal AFR affected the mean offspring trait value, and modified the subsequent effects of offspring birth order. Because our focus is within-mother variation, we fixed offspring birth order to be in all models; other terms were selected by DIC. We used the estimates from this analysis to plot our figures, and for mapping the model predictions back to the maternal age at each G1 offspring's birth. As the offspring age, some die and others never reproduce. Thus, not all offspring born to each mother resulted in data on the trait under analysis. Our definitions of maternal AFR and offspring birth order incorporated the consequent changes to offspring age ranking: Maternal AFR was the effect of a mother's age at the birth of her oldest offspring in each analysis. Birth order was the age rank among each mother's offspring who were present in an analysis. This loss of offspring between analyses also explains the change in sample size between analyses (Table S1).

We then investigated how G1 offspring sex modified our main results, by expanding the most likely main effects model with the terms

$$SEX + SEX * BO + SR + SR * BO + SEX * SR + SEX * SR * BO, \quad (2)$$

and also including the interactions between these and other terms that contained maternal AFR or offspring birth order. SEX denotes G1 offspring female or male. SR is a linear continuous variable that describes the sex ratio of each G1 offspring's older siblings. SR is 0 for all-female older siblings, 1 for all-males, and for analysis was set at 0.5 for first-borns. This structure thus investigates how the trait value of each G1 offspring depended on its sex, birth order and the sex of its older siblings.

Finally, we investigated how maternal SES (defined as a two-level factor with levels landless vs. landowner) modified the most likely main effects model. To focus on modifications to the within-mother trends by G1 offspring birth order, we fixed the additional structure SES + SES * BO in all models.

To contrast the fits of each DIC-selected model between analysis stages, Table S2 presents relative model likelihoods. These are evidence ratios, which calculate the relative likelihood of two alternative model structures from their difference in DIC (Burnham and Anderson 2002). Tables S3–S5 present parameter estimates and 95% confidence intervals (95% CIs).

MAPPING MODEL PREDICTIONS BACK TO MATERNAL AGE AT G1 OFFSPRING BIRTH

Finally, we mapped the model predictions from our main effects analysis back to the maternal age at each G1 offspring's birth. This step showed how our model predictions underlie the population-level trends in G1 offspring LRS by the maternal age at each G1 offspring's birth. To do so, we extracted a predicted value for each G1 offspring data point and smoothed these across the maternal age at each G1 offspring's birth using a regression spline (Wood 2003).

Results

POPULATION-LEVEL ANALYSIS

Mothers gave birth to a mean of 6.7 G1 offspring during their lifetime (standard deviation, [SD] = 2.8, range 1–16) at a mean maternal age of 32.5 years (SD = 6.3 years). Only 11% of mothers reproduced in both their first and second marriages, and only two mothers did so in three consecutive marriages. On average, the birth of a G1 offspring gave rise to 1.5 G2 offspring (SD = 2.2, range = 0–11). This value is low because it incorporates zeros for G1 offspring who did not survive to reproduce, and who produced G2 offspring who all died before 15 years. The mean individual λ of G1 offspring was 0.89 (SD = 0.33, range = 0–1.06). Values exceeding 1 indicate that the newborn G1 offspring succeeded in replacing itself with a G2 offspring who reached 15 years. The fact that the mean individual λ was less than 1 indicates that most G1 offspring did not succeed in this replacement. The linear regressions of G1 offspring LRS and individual λ showed that both metrics decreased with maternal age at the G1 offspring's birth (Fig. 1). The regression coefficients (r) showed that LRS decreased by 22% (Fig. 1A) and individual λ by 45% (Fig. 1B) between the G1 offspring born to mothers aged 16 and 50 years. The fact that individual λ decreased approximately twice as fast as LRS indicates a strong association of old maternal age with delayed G1 offspring reproduction. Later we investigate the within-mother variation underlying the decrease in G1 offspring LRS, in terms of traits that describe G1 offspring survival to adulthood and their subsequent production of G2 offspring.

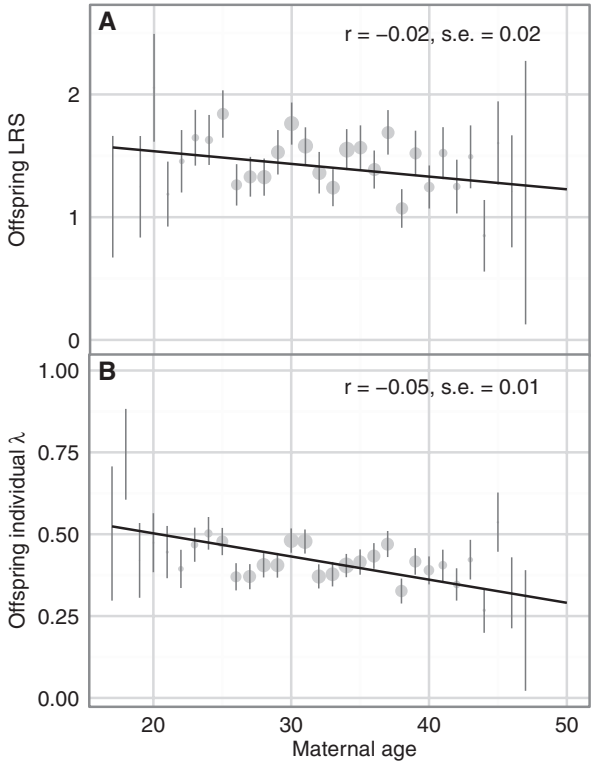

Figure 1. Population-level analysis: older mothers produce less fit offspring. Regressions of G1 offspring (A) lifetime reproductive success (LRS) and (B) individual λ on maternal age at G1 offspring birth. LRS was the number of G2 offspring alive at 15 years that arose from the birth of a G1 offspring. Individual λ incorporates the timing of G1 offspring reproduction into this measure. The size of the gray data points for each 1-year maternal age class are proportional to the sample sizes used to calculate the means of each metric; error bars show standard errors. The regression lines show predicted values from least squares linear regression through the means of offspring LRS or individual λ for G1 offspring born in 1-year maternal age classes (35 data points). Both the dependent and explanatory variables were standardized by subtracting their mean and dividing by their standard deviation before analysis. In each panel, we present the regression coefficient, *r*, indicating the slope of the relationship alongside the standard error (SE) around each predicted slope.

INDIVIDUAL-LEVEL ANALYSES

Survival to 15 years

Of the G1 offspring born, 62% survived to 15 years. The most likely main effects model did not contain an association of offspring survival with maternal AFR, but did contain a curvilinear association of offspring survival with birth order, which improved model fit (Table S2), and reached significance at the 95% level (Table S3). It showed that the expectation of offspring survival increased slightly from 0.63 (95% CI = 0.53–0.72) for first borns to 0.65 (95% CI = 0.53–0.76) at birth order 6 (Fig. 2a). Survival then declined substantially from birth order 7, reaching values computed from the raw data of ca. 0.4 at birth orders 10 and 11 (Fig. 2A). We note that despite the loss of sample size at high birth orders, and thus increasing prediction uncertainty, this decline in survival was evident in the fit of our quadratic function. Over the 95% range of offspring birth orders (orders 1–9), the predicted mean rise and then fall of offspring survival spanned a probability difference of 0.05, equivalent to a difference of 50 deaths per 1000 births. This represents nontrivial variation in juvenile mortality, to the disadvantage of the earliest and latest born offspring.

Additional investigation of how the above main effects differed by G1 offspring sex led to a small improvement in model fit (Table S2). However, none of the parameter estimates for these sex effects differed from zero with 95% confidence (Table S3). At the average birth order of a G1 offspring (fourth born), female G1 offspring survived to 15 years with a probability that was 0.04 (95% CI = −0.01 to 0.09) higher than male G1 offspring. The most likely model incorporated only the interaction between offspring sex and the curvilinear function of offspring birth order. The fit indicated that the sex difference in survival to 15 years was evident only around birth orders 4–7, where female offspring had the higher survival (Fig. S1). This suggests that increasing birth order had a greater positive effect on the survival of female than male offspring, but that the resulting survival advantage of female offspring was not present beyond birth order 7. However, the overlap of standard errors computed from the binary raw data in Figure S1 emphasizes the low confidence surrounding this fit.

Investigation of the differences between the maternal SES groups (landless and landowners) also led to a small improvement in model fit (Table S2). However, as with the effects of G1 offspring sex, none of the additional parameter estimates differed from zero with 95% confidence (Table S3). At the average G1 offspring birth order (fourth born), the G1 offspring of landowning mothers survived to 15 years with a predicted probability that was 0.01 (95% CI = −0.04 to 0.05) higher than those of landless mothers. The most likely model featured only maternal SES and its interaction with G1 offspring birth order, which we had fixed in all model structures. The fit indicated two trends (Fig. S2): (i) That the first-born offspring of landowning mothers had a survival probability 0.03 (95% CI = −0.02 to 0.04) *lower* than the first-born offspring of landless mothers. Notably, in the raw data, the magnitude of this difference was 0.10 and there was little evidence of maternal SES effects beyond first-born G1 offspring (Fig. S2). (ii) Beyond approximately birth order 5, the predicted decline in offspring survival to 15 years was more rapid among landless than landowning mothers. The result was that by birth order 8 (the 95% limit of childbearing by landless mothers), the G1 offspring of landowning mothers had a predicted probability of survival to 15 years 0.05 (95% CI = 0.04–0.06) higher than the G1 offspring of landless mothers.

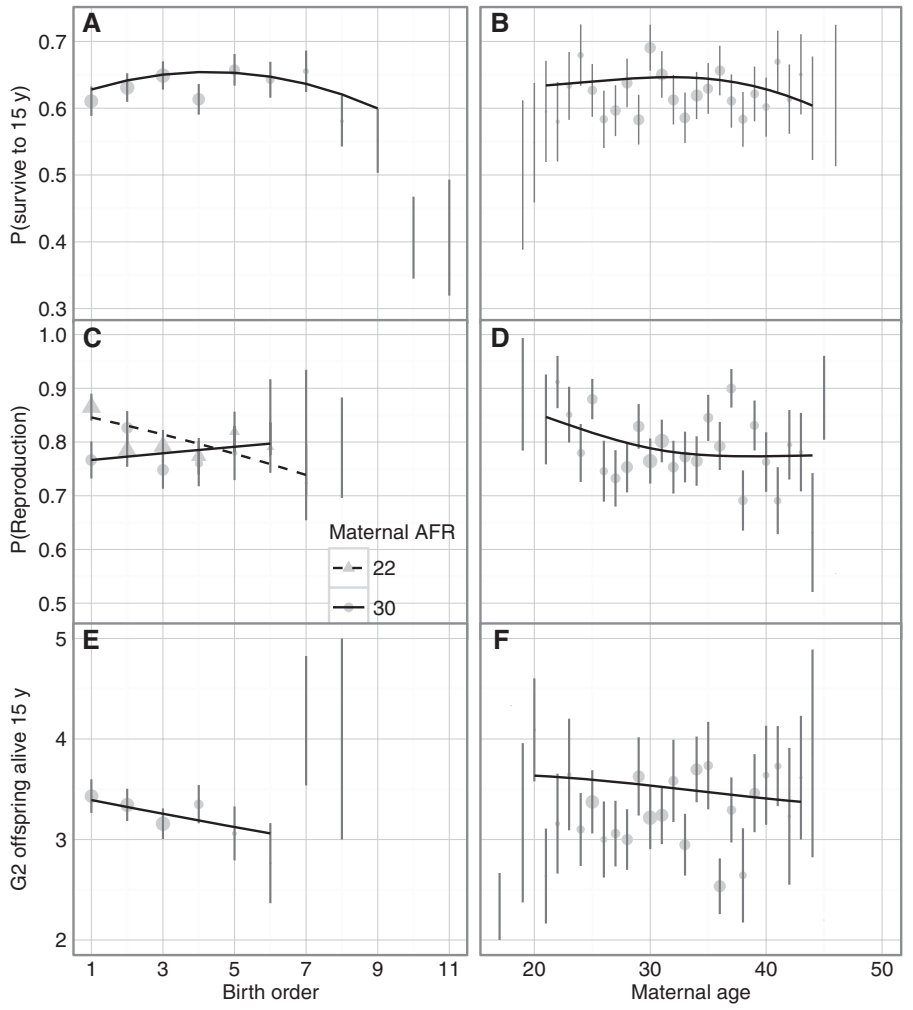

Figure 2. **Individual-level analyses: offspring birth order and maternal age at offspring birth effects. Analyses of G1 offspring survival to 15 years (A and B); reproduction, given survival to 15 years (C and D); and number of G2 offspring alive at 15 years, given at least one G2 offspring born (E and F). The left-hand plots (A, C, E) show the regression of each trait on G1 offspring birth order, from generalized linear mixed models fitted using the R package MCMCglmm. Note that the birth-order effect on the probability of reproduction (C) was dependent on maternal age at first reproduction, which we illustrate with solid and dashed lines. The right-hand plots (B, D, F) show the outcome of mapping the predicted G1 offspring birth-order effects to maternal age at each G1 offspring's birth, then smoothing these predictions over maternal age. Each line spans the 95% range of raw data; sizes of the gray data points indicate the relative sample sizes for each birth order or maternal age; points and error bars show means and standard errors. See Supporting Information for parameter estimates.**

REPRODUCTION IF ALIVE AT 15 YEARS

Of the G1 offspring who survived to 15 years, 79% produced at least one G2 offspring. The most likely main effects model contained the linear functions of maternal AFR and offspring birth order, and their interaction, which improved model fit (Table S2). This interaction was just significant at the 95% level (Table S4). Mothers relatively young at the birth of their oldest G1 offspring who survived to 15 years had G1 offspring with a steeper birth-order decline in the probability of reproduction (Fig. 2C). When the oldest G1 offspring was born to a mother aged 22 years (1 SD before the mean maternal AFR), the predicted G1 offspring probability of reproduction decreased from 0.85 (95% CI = 0.75–0.91) to 0.76 (95% CI = 0.63–0.85) between birth orders 1 and 6. At the mean maternal AFR, the effect of G1 offspring birth order was close to zero. When the oldest offspring was born to a mother aged 30 years (1 SD after the mean maternal AFR), the predicted probability of reproduction increased slightly from 0.77 (95% CI = 0.65–0.85) to 0.80 (95% CI = 0.52–0.94) between birth orders 1 and 6. Thus, the significance of the interaction between maternal AFR and offspring birth order appeared to be driven mainly by the steep birth-order decline among mothers who were younger than average at their first birth. Furthermore, Figure 2c indicates that the difference associated with maternal AFR was localized to first-born

offspring; the predicted difference in the probability of reproduction by first borns was consistent across the range of maternal AFR at 0.08 (95% CI = 0.08–0.08). This equates to a difference of 80 reproducing G1 offspring for every 1000 who survived to 15 years. Note that the potential causes of this difference include G1 offspring death after 15 years, infertility as well as social effects.

Investigation of how these main effects differed by G1 offspring sex produced a substantial improvement in model fit (Table S2); the sex effects also differed from zero with 95% confidence (Table S4). At the average birth order among G1 offspring who survived to 15 years (third born), female G1 offspring produced a G2 offspring with a probability 0.04 (95% CI = 0–0.08) higher than male G1 offspring. The most likely model contained older sibling sex ratio and its interactions with G1 offspring sex and G1 offspring birth order. For female G1 offspring, the clearest effect was seen among second borns, that is females with one older sibling alive at 15 years (Fig. S3). The predicted probability of reproduction by a second-born female was 0.09 (95% CI = 0.06–0.13) higher for those with an older brother compared to an older sister. For male offspring, the effect of older sisters was approximately neutral, but rising numbers of older brothers reduced the probability of reproduction. The effect was clearest among fourth-born males where the predicted probability of reproduction was 0.14 (95% CI = −0.12 to 0.40) lower for males with three older brothers compared to three older sisters.

Investigation of the difference in main effects between maternal SES groups produced no improvement in model fit (Table S2). At the average birth order among G1 offspring who survived to 15 years (third born), the G1 offspring of landowning mothers had a probability of reproduction 0.01 (95% CI = −0.03 to 0.07) lower than the G1 offspring of landless mothers. Nor did the interaction between maternal SES and G1 offspring birth order differ from zero with 95% confidence (Table S4). Inspection of Figure S4 further supports the absence of a modifying effect of our two-level SES variable of landless versus landowners.

G2 OFFSPRING ALIVE AT 15 YEARS

If G1 offspring did produce a G2 offspring, they achieved a lifetime mean of 3.3 (SD = 2.2, range = 0–10) G2 offspring who survived to 15 years. None of our main effects variables caused improvement in model fit, and so we investigated only the linear function of G1 offspring birth order (Table S2). This birth-order effect did not differ from zero with 95% confidence (Table S5). However, Figure 2(e) does indicate a trend for decline in the number of G2 offspring alive at 15 years with increasing G1 offspring birth order (mean estimate = −0.031, 95% CI = −0.080 to 0.015). This equated to a predicted decline of 0.33 surviving G2 offspring between G1 offspring birth orders 1 and 6. Despite the weak statistical support of this effect, and apparent small effect size, it predicts a 9.8% decline between birth orders 1 and 6, or a loss of 330 G2 offspring alive at 15 years per 1000 G1 offspring who reproduced. Thus, despite the low prediction certainty, it appears to be an effect of potential demographic significance.

Investigation of the differences by G1 offspring sex did improve model fit (Table S2). At the average birth order among the G1 offspring who produced a G2 offspring (third born), female G1 offspring produced 0.4 (95% CI = 0.1–0.8) fewer G2 offspring alive at 15 years than male G1 offspring. The most likely model did not include interactions with G1 offspring birth order, but did include the interaction between G1 offspring sex and older sibling sex ratio (Fig. S5). These effects also differed from zero with 95% confidence (Table S5). Females with only older brothers produced 0.7 (95% CI = 0–1.3) more G2 offspring alive at 15 years than those with only older sisters. Males with only older sisters produced 0.6 (95% CI = −1.1 to 2.3) more G2 offspring alive at 15 years than those with only older brothers. These effects are likely to be due to the negative effects of having at least one older same-sex sibling, although could also reflect positive effects of older opposite-sex siblings.

Investigation of how the main effects differed between maternal SES groups did not improve model fit (Table S2). At the average birth order among the G1 offspring who produced a G2 offspring (third born), the G1 offspring of landowning mothers produced 0.18 (95% CI = −0.17 to 0.56) more G2 offspring alive at 15 years than the G1 offspring of landless mothers. The interaction between maternal SES effects and G1 offspring birth order did not differ from zero with 95% confidence (Table S5). However, Figure S6 indicates that, for first-born G1 offspring only, the offspring of landowning mothers produced 0.4 (95% CI = 0.3–0.4) more G2 offspring alive at 15 years than the offspring of landless mothers. This first-born advantage for landowners clearly underlies the trend for a birth-order decline seen in the main effects analysis. The SES difference between first-borns was relatively large, equating to a difference of 400 G2 offspring alive at 15 years per 1000 G1 offspring who reproduced.

MAPPING MODEL PREDICTIONS BACK TO MATERNAL AGE AT G1 OFFSPRING BIRTH

When we mapped the above predicted mean effects onto the maternal age at G1 offspring birth, we found each effect visible clearly in terms of maternal age (Fig. 2B, D, F). G1 offspring born to mothers aged 32 years had the highest probability of survival to 15 years, 0.65 (95% CI = 0.54–0.74; Fig. 2b). The largest change in survival to 15 years over maternal age occurred between 32 and 44 years, when the probability of survival declined by 0.04 (95% CI = −0.02 to 0.11). In addition, the raw data indicated that the probability of

G1 offspring survival was particularly low, at around 0.50, for those born to mothers younger than age 21 ($n = 69$ offspring). For the probability of reproduction by G1 offspring who survived to 15 years, the combined effects of maternal AFR and G1 offspring birth order produced a curvilinear relationship with maternal age (Fig. 2d). The predicted probability of reproduction declined from 0.85 (95% CI = 0.75–0.91) if born to a mother aged 21 years, to 0.79 (95% CI = 0.66–0.88) if born to a mother aged 30 years, after which age there was little change. For the number of G2 offspring alive at 15 years given that at least one G1 offspring was born, there was an approximately linear decline with maternal age at G1 offspring birth (Fig. 2f). Between G1 offspring born to mothers aged 21 and 43 years, there was a difference in G2 offspring number of 0.26 (95% CI = −0.14 to 0.59), which equates to a decline of 7.2% G2 offspring alive at 15 years or 260 fewer per 1000 G1 offspring who reproduced. Finally, we multiplied the predicted mean values for each of the above three traits to recapitulate the trend in G1 offspring LRS by maternal age at their birth. This showed an approximately linear 18% decline in G1 offspring LRS between G1 offspring born to mothers aged 21 and 43 years.

Discussion

Here, we show the first evidence in a long-lived species that older mothers produce offspring with lower individual fitness. The population-level decreases in offspring LRS and individual λ with maternal age at childbirth were affected by both maternal AFR and longitudinal changes with offspring birth order. The main implication of our study is that failure to quantify survival and reproduction over the entire lifetimes of offspring risks misrepresenting: (1) the effects on an offspring of maternal age and reproductive history at childbirth; (2) the effects on population growth and dynamics of changes to the timing and amount of maternal lifetime reproduction. These implications are likely to be most relevant in species such as humans, where mothers invest somatic resources heavily into reproduction, and where offspring of different ages compete for extra-somatic resources, such as wealth. However, the nature and severity of sibling competition for both nutritional and economic resources varies widely among periods and cultures. Incorporating these additional effects on the entire lifetimes of offspring into mathematical models of population growth will undoubtedly bring insights into the molding of senescence by natural selection.

Lower offspring survival to 15 years at the lowest and highest birth orders is also known from several other human population studies (Modin 2002; Mahy 2003; Faurie et al. 2009). It was notable, however, that we found that wealthier landowner mothers experienced lower survival of their first-born offspring to adulthood, despite their expected better energetic condition (Moring 1998). The younger age at first reproduction of landowning mothers could play a role in this result, as this has been linked to incomplete maternal development, inexperience or a greater risk of maternal mortality in childbirth (Kramer 2008; Lahdenperä et al. 2011). Alternatively, landowners and the landless could have differed behaviorally in birth spacing or their care for offspring, which are known in general to affect offspring energetic condition and survival (Alam 1995; Gibson and Mace 2006). At high birth orders, especially after the seventh birth, offspring survival to 15 years declined rapidly. Physiologically, this could arise either as mothers accumulate somatic costs of reproduction, for example mechanical damage or energetic depletion, or due to the negative effects of maternal aging per se (Djahanbakhch et al. 2007). Socially, increasing numbers of codependent offspring could reduce the expected survival of newborns through competition, for example by negatively affecting the pooled household energy budget (Reiches et al. 2009). The expectation of kin care, which improves offspring survival, is also lower for offspring with older mothers, and hence for higher birth-order offspring (Lahdenperä et al. 2004; Pavard et al. 2005; Shanley et al. 2007; Sear and Mace 2008). In addition, death of the father could be particularly detrimental to the survival of young offspring, particularly in landless families where men made a vital contribution to household sustenance (Moring 2002). The net product of all these potential causes was a decline in offspring survival to 15 years at advanced birth orders and advanced maternal ages at offspring birth. This survival variation among young offspring should play a key role in shaping the impact on population growth of age-specific maternal reproduction.

Another effect of offspring failing to reach 15 years is a rearrangement of the age-ranking among siblings. The outcome of this rearrangement is crucial in determining the subsequent social mediation of reproductive output, as cultural rules on marriage and wealth inheritance often operate by adult sibling age-rank (Hrdy and Judge 1993). Among the G1 offspring who reached 15 years, those with more older siblings were less likely to reproduce and had fewer G2 offspring alive at 15 years. Our results are consistent with previous research on preindustrial Finns showing the negative effects on male offspring of having older brothers, due largely to a lower likelihood of attaining landowning SES (Faurie et al. 2009; Rickard et al. 2009; Nitsch et al. 2013). Our findings also indicate that older sisters constrained the reproduction of female offspring, potentially by diminishing the available dowry (Moring 2009; Nitsch et al. 2013). The probability of producing a G2 offspring was highest among first-born G1 offspring with relatively young mothers, suggesting that a common socioeconomic advantage could have facilitated both early maternal reproduction and a high probability of first-born offspring reproduction. For example, marriage to a landowning husband was associated with a younger female age at marriage, and hence at first birth (Pettay et al. 2007;

Gillespie et al. 2010). Landowning could then have increased the probability of first-born reproduction (Faurie et al. 2009). This suggests that the dynamics of wealth inheritance, even if not captured by our broad landowners versus landless variable, were an important aspect of Finnish population dynamics. Our population-level analyses indicated that such effects also modified the timing of reproduction in the life histories of G1 offspring, as individual λ decreased more steeply over maternal age than LRS. The impact of SES on the timing of life-history events is well documented in a range of societies (e.g., Pettay et al. 2007; Gibson and Gurmu 2011). Sibling inequality in wealth inheritance is also likely to have diminished the heritability of traits such as age at first reproduction.

Together, our results indicate that throughout the lifetime of G1 offspring, their sibling age rank, and/or maternal age at their birth, predicted the influence of factors that modified their contribution to population growth. Early in the life history, this was largely an all-or-nothing modification, driven by variation in survival to maturity and subsequent achievement of at least one G2 offspring (Liu and Lummaa 2011). Among adult G1 offspring, there were also declines in their lifetime production of G2 offspring alive at 15 years. We found that these factors explained most of the population-level decline in G1 offspring LRS with advancing maternal age at G1 offspring birth.

In classical life-history theory, senescence evolves in response to extrinsic environmental changes that modify the age-specific probabilities of reproduction or survival (Hamilton 1966). Our results suggest that if such factors also modify the association of offspring reproduction with parental ages at childbirth, then the evolution of senescence is similarly modified. However, the variability of environmental, economic, and cultural conditions both among and within human societies emphasizes the need for robust mathematical modeling to test this assertion. Such a model would be likely to build on existing population models that quantify the effects of kin on offspring survival (Pavard and Branger 2012). These models are also expandable to account for wealth inheritance using recently developed methods for tracking phenotypic dynamics (Coulson et al. 2010). Our results show that there are sufficient effects of maternal reproductive timing on the entire lifetime of their G1 offspring to make such extensions extremely worthwhile.

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Supporting Information

Additional Supporting information may be found in the online version of this article at the publisher's website:

Table S1. Final sample sizes.
Table S2. Summary of model fits over the sequence of analyses for each trait.
Table S3. Probability of survival to 15 years.
Table S4. Probability of reproduction, given survival to 15 years.
Table S5. Number of G2 offspring alive at 15 years, given at least one G2 offspring born.
Figure S1. The probability that a G1 offspring survived to 15 years by its sex and birth order.
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Figure S3. The probability of reproducing by G1 offspring sex, birth order, and the sex of its older siblings.
Figure S4. The probability of reproducing by G1 offspring birth order and maternal socioeconomic status.
Figure S5. G2 offspring alive at 15 years by G1 offspring sex, birth order, and the sex of older siblings.
Figure S6. G2 offspring alive at 15 years by G1 offspring birth order and maternal socioeconomic status.