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Evolution and Human Behavior 32 (2011) 433 – 443

Evolution and Human Behavior

Theoretical Contribution

Age at first reproduction and probability of reproductive failure in women

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Initial receipt 3 February 2010; final revision received 23 October 2010

Abstract

Life history theory predicts a trade-off between fitness benefits and costs of delaying age at first reproduction (AFR). In many human populations, maternal AFR has been increasingly delayed beyond sexual maturity over the past decades, raising a question of whether any fitness benefits accrued outweigh costs incurred. To investigate the cost–benefit trade-off concerning AFR in women, we construct a theoretical model and test its predictions using pedigree data from historical Finnish mothers. The model predicts that the probability of reproductive failure (no offspring produced reaching breeding) will increase with AFR if the benefit with delaying in terms of improvement to offspring quality (i.e., breeding probability) cannot offset the cost from decline in offspring quantity. The data show that offspring quantity declined significantly with delayed reproduction, while offspring quality remained initially constant before declining when AFR was delayed beyond 30. Consistent with the theoretical model's predictions, reproductive failure probability increased markedly with delaying AFR after 30, independently of maternal socioeconomic status. Our study is the first to investigate the associations between delay in AFR after sexual maturity and changes in not only offspring quantity but also offspring quality and suggest a significant evolutionary disadvantage of delayed AFR beyond 30 for lineage persistence in a predemographic transition society.

Keywords: Delayed reproduction; Sexual maturity; Life history; Lineage persistence

1. Introduction

Reproduction is fundamental for evolutionary success, and reproductive scheduling is central for the evolution of life histories (Roff, 1992). Among the components of reproductive scheduling, age at first reproduction (AFR) is particularly important, having a pivotal effect on reproductive success or fitness (Roff, 1992; Stearns, 1992). For females, AFR should evolve to maximize fitness subject to the benefits and costs of delayed reproduction (Stearns, 1992). The costs of delaying reproduction may include increased accumulated mortality hazard before reproduction, reduced reproductive span, reduced reproductive output, and longer generation time, while the benefits may include larger body size/weight, higher initial fecundity, and lower offspring mortality brought by longer growth (Kawecki, 1993; Kozlowski, 1992; Migliano, Vinicius & Lahr, 2007; Stearns, 1992; Stearns & Hoekstra, 2005).

In human females, AFR is among the most important life-history traits affecting between-female variation in fitness (Käär, Jokela, Helle, & Kojola, 1996; Migliano et al., 2007; Pettay, Helle, Jokela & Lummaa, 2007) as a result of the costs and benefits mentioned above. By considering the trade-off between such costs and benefits, Hill and Hurtado (1996) were the first to combine theoretical analysis and testing this with empirical data on the optimality of female AFR in humans using Malthusian parameter or instantaneous rate of increase as the fitness measure. Here, the costs of delayed AFR included longer generation time and higher chance of dying before reproducing, whereas the major benefit included higher initial fecundity brought by increased body weight with delay in AFR. Hill and Hurtado (1996) predicted the optimal maternal AFR to be 18 years, close to the observed mean AFR (17.5 years) in Ache, their studied traditional hunter–gatherer tribe of Paraguay. A similar study was carried out on Gambian females by Allal, Sear, Prentice and Mace (2004), who found that female height could be a better indicator than weight in predicting optimal AFR. However, as noted by Hill and Hurtado (1996), such

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doi:10.1016/j.evolhumbehav.2010.10.007

analyses apply to human populations where AFR is tightly linked with age at sexual maturity (with a short lag; e.g., in the case of Ache females, this lag was 1 year used for pregnancy), but not to an increasing number of populations where AFR varies independently of age at sexual maturity (Hill & Hurtado, 1996; Wood, 1994). Maternal AFR has become increasingly delayed beyond sexual maturity in postdemographic transition Western societies, especially during recent decades (Frejka & Sardon, 2006; Sobotka, 2004). Similar delays in AFR were also common in 18th and 19th European countries, such as Finland (Lutz, 1987). For such cases, the evolutionary benefits of delays in first reproduction remain currently unclear. Firstly, clinic data indicate that female fecundity remains constant from age at sexual maturity to age 30 and then declines significantly from 30 onwards (Schwartz & Mayaux, 1982; van Noord-Zaadstra et al., 1991). Secondly, after controlling for confounding factors, later maternal age is associated with higher risk of producing offspring with low birth weight (<2500 g) (Geronimus, 1996; Lee, Ferguson, Corpuz & Gartner, 1988), possibly leading to increased infant mortality (Vanlandingham, Buehler, Hogue & Strauss, 1988). Finally, reproduction at advanced maternal age (beyond 35) is also associated with higher risk of stillbirth (Raymond, Cnattingius & Kiely, 1994).

Given the disadvantages of delaying AFR, it seems that women should begin to reproduce when reaching sexual maturity (Fisher, 1999), but this is increasingly not observed worldwide. One emerging evolutionary hypothesis for the increasing delay in female AFR beyond age at sexual maturity is socioeconomic benefits. These benefits have been suggested to include the opportunity to accumulate more social resources so as to improve offspring performance or, in other words, "favorable eventual placement of children in the mating market" (Kaplan, Lancaster, Tucker & Anderson, 2002). Thus, mothers who delay reproduction may be trading off a rise in socioeconomic status against a fall in reproductive physiology. Such a rise is assumed to be able to compensate for the costs of delayed reproduction by improving the quality of offspring in order to pay off in terms of long-term likelihood of spreading genes into future generations, i.e., maximizing lineage persistence likelihood.

Investigating empirically whether this is true is difficult because it requires, by definition, individual-based lifelong records on survival and fecundity for whole lineages across several generations, and such data are difficult to obtain for any species, let alone for humans with exceptionally long lifespan. Two previous studies shed some light on this issue. First, Kaplan et al. (2002) used a sample of men and women from the National Survey of Families and Households in the USA and another sample of interviewed men in Albuquerque, NM, USA, to show that delayed reproduction in both men and women was associated with lower fertility. For the Albuquerque men, lower fertility was not compensated by income level to increase numbers of grandchildren born (Kaplan, Lancaster, Bock & Johnson, 1995). There were two important limitations, however: the direct link between AFR and numbers of grandchildren was not investigated to address how a delay in reproductive onset is associated with both offspring quantity and quality, and the analysis of grandchildren numbers concerned only men whose reproductive physiology is less constrained by age than that of women and whose fitness may be limited by trade-offs different to women (Lahdenperä, Russell & Lummaa, 2007). It is thus unknown how the conclusion on grandchildren applies to women. Second, using simulations based on data on education, income, AFR, and lifetime fertility of modern American women, Low, Simon and Anderson (2002) showed that despite of socioeconomic benefits brought by delayed AFR, mothers with later AFR were predicted to have reduced fertility and longer generation time and, consequently, increasingly lower proportion of descendants in the future population. Another simulation indicated that even longer generation time itself as the result of delayed AFR could lead to the same result (Low, Simon & Anderson, 2003). However, these simulations by Low et al. (2002) and (2003) considered only offspring survival rate but not their breeding success, which might be fundamentally affected by the suggested benefits of delayed maternal AFR.

In summary, at present, no empirical studies exist on women to show how delays in AFR relate to offspring quality, depicted not only by their survival to breeding age but also by their recruitment to the breeding population. Such information is crucial for determining whether delays in AFR could improve offspring quality, for example, through socioeconomic benefits and, despite reduced offspring quantity, thus lead to evolutionary benefits in the long-run, i.e., lineage persistence. We thus know currently little about whether and how the relationship between delayed reproduction, socioeconomic factors, and offspring quality can influence a woman's lineage persistence over time.

Here, we focus on investigating the association for females between delaying AFR and change in the vulnerability of their lineages to extinction, the opposite of lineage persistence likelihood. First, we introduce a quantitative index to assess this vulnerability and construct a generalized mathematical model to identify the association between delaying AFR and change in lineage extinction vulnerability. Secondly, we evaluate the predictions of the model using data from a multigenerational, individual-based dataset of preindustrial Finns from the 18th and 19th centuries where the average AFR, similarly as modern societies, was significantly beyond maturity. Specifically, we investigate the associations between maternal AFR and (i) offspring quantity (lifetime number of children), (ii) offspring quality (measured by their probability of being recruited to breeding population) and its major component (survival rate at reproductive age 15), and (iii) the probability of maternal reproductive failure (lineage extinction or no breeding children). Additionally, we represent environmental conditions by socioeconomic status and investigate how this influenced the above associations.

2. Theoretical models

First, we investigate the association between a mother's delayed AFR and the vulnerability of her lineage to extinction by constructing a theoretical model, predictions from which will be tested using empirical data. We quantify vulnerability of lineage to extinction as the probability of reproductive failure P_{rf} where the subscript is the acronym of "reproductive failure," which is defined as not raising any breeding offspring in a lifetime. Naturally, no breeding grand-offspring, great-grand-offspring, and so on, can also lead to lineage extinction. However, in this study, we focus only on the lineage extinction as a result of no breeding children, which is the most paramount concern of parenting (Buss, 1999) and could be relevant to reproductive decision making in modern low-fertility societies (Kaplan et al., 2002). Such low reproductive rates in many industrialized countries are hard to explain in terms of descendant dominance (such as lifetime fertility, reproductive success or number of grandchildren; Kaplan et al., 1995). Reproductive failure in terms of no breeding children might thus provide a complementary perspective to investigate individual reproductive strategies. Additionally, probability of reproductive failure may be a major determinant of ultimate measures of lineage persistence, such as expected number of generations to extinction (Mueller, 2001; Stearns 1992).

Before deriving mathematical formula for P_{rf}, we will introduce two concepts. For a mother that begins to reproduce at age α (her AFR), we define her offspring quantity R_α as her expected lifetime number of children, and offspring quality p_α as the expected probability for her offspring to recruit to the breeding population. In practice, this probability will be represented by the recruitment proportion of all produced children. The AFR-specific probability of reproductive failure of a mother is the probability that all her R_α expected number of children fail to be recruited to breeding, given the expected offspring recruitment probability if she begins to reproduce from age α. Hence, AFR-specific probability of reproductive failure of the mother can be formulized as follows,

$$P_{rf} = (1-p_\alpha)^{R_\alpha} \tag{1}$$

It should be noted that the probability of recruitment failure ($1\text{-}p_\alpha$) may be expressed as a function of various aspects of the environment or culture. For example, in pre-industrial societies with high marriage and reproductive rates, variation in offspring recruitment probability may be determined mainly by offspring mortality rate at pre-reproductive ages, but in modern societies, it may be determined mainly by variation in failure to find a partner or to have a child. In any case, the probability of reproductive failure can be seen as a quantitative criterion of maladaptiveness, and lineage persistence likelihood $1\text{-}P_{rf}$ can be seen as a measurement of adaptiveness. $1\text{-}P_{rf}$ will, in turn, be determined by both offspring quantity and quality.

We can investigate the association between probability of reproductive failure (P_{rf}) and AFR (α) by examining the associations between α and R_α and p_α. The following partial differentiation depicts the process:

$$\begin{aligned}\frac{\partial P_{rf}}{\partial \alpha} &= \frac{\partial P_{rf}}{\partial p_\alpha}\cdot\frac{\partial p_\alpha}{\partial \alpha} + \frac{\partial P_{rf}}{\partial R_\alpha}\cdot\frac{\partial R_\alpha}{\partial \alpha} \\ &= \left[-(1-p_\alpha)^{R_\alpha-1}\right]\cdot\left\{R_\alpha\cdot\frac{\partial p_\alpha}{\partial \alpha} - (1-p_\alpha)\cdot\left[\frac{\partial R_\alpha}{\partial \alpha}\cdot ln(1-p_\alpha)\right]\right\}\end{aligned} \tag{2}$$

$\frac{\partial p_\alpha}{\partial \alpha}$ is the change in offspring quality associated with the change in the timing of reproduction onset. Thus, the product of this change in quality and the number of offspring, $R_\alpha\cdot\frac{\partial p_\alpha}{\partial \alpha}$, represents the potential benefit to reproductive success from delaying reproduction (see Hamilton, 1966; Mace, 1996). Correspondingly, $\frac{\partial R_\alpha}{\partial \alpha}$ is the change in lifetime number of children associated with the change in AFR. Thus, the product of this and offspring quality, $(1-p_\alpha)\cdot\left[\frac{\partial R_\alpha}{\partial \alpha}\cdot ln(1-p_\alpha)\right]$, can be seen as the potential cost of delaying reproduction to reproductive success. Combining these two terms, $R_\alpha\cdot\frac{\partial p_\alpha}{\partial \alpha} - (1-p_\alpha)\cdot\left[\frac{\partial R_\alpha}{\partial \alpha}\cdot ln(1-p_\alpha)\right]$ generates an explicit representation of the potential trade-off between the benefit and cost to reproductive success associated with delaying reproduction. Importantly, both the benefits and the costs related to delayed AFR are possible, but not necessarily realized; thus, there may be no benefit or cost at all. Additionally, associations in Model 2 are not necessarily causal relationships. However, if the associations between α and R_α and p_α can be determined as causal, then, the association between α and P_{rf} may be causal, too.

It is worth noting that the sign of $\left[-(1-p_\alpha)^{R_\alpha-1}\right]$ is negative, so the sign of $\frac{\partial P_{rf}}{\partial \alpha}$ (the change in probability of reproductive failure with a change in AFR) will be determined by the sign of the trade-off. Consequently, we make the following predictions. (1) The probability of reproductive failure P_{rf} will decrease with delaying AFR when the associated benefit, $R_\alpha\cdot\frac{\partial p_\alpha}{\partial \alpha}$, offsets the cost $(1-p_\alpha)\cdot\left[\frac{\partial R_\alpha}{\partial \alpha}\cdot ln(1-p_\alpha)\right]$; by contrast, P_{rf} will increase with delaying AFR where the benefit cannot offset the cost. (2) A minimum value of P_{rf} will be achieved where the associated benefit is equivalent to cost and consequently $\frac{\partial P_{rf}}{\partial \alpha}$ is zero, and an optimal AFR for lineage persistence likelihood should thus be achieved at such point(s).

We have no a priori information about the explicit functional formulae of R_α and p_α with respect to α. Consequently, in the next section, we estimate them statistically for women living in pre-health care and pre-family planning conditions and use the results to test the above predictions.

3. Empirical data and statistical methods

3.1. Dataset of life history records

We test the predictions of the theoretical models with empirical pedigree data on rural Finnish women collected from historical parish records. These data were originally maintained by the Lutheran Church and have been compiled and checked for errors by professional genealogists. These records provide one of the most reliable and detailed sources of data for individual-level variation in survival and reproduction across several generations in pre-industrial humans (Käär et al., 1996; Lutz, 1987).

The data come from four small-scale farming and fishing based parishes, three of which are located on southwest archipelago and one on the mainland of Finland (archipelago: Hiittinen 60°N, 22°39'E, Kustavi 60°39'N, 21°39'E and Rymättylä 60°15'N, 22°E; mainland: Ikaalinen 61°45'N, 23°E) (Lummaa, 2001; Rickard, Russell & Lummaa, 2007). The fertility period (1746–1860) preceded the demographic transition in Finland and was thus characterized by natural fertility since modern birth control methods and advanced health care were not available (Käär et al., 1996; Lummaa, 2001; Lutz, 1987).

Living conditions were highly variable with unpredicted crop failures and associated famines occurring throughout the fertility period. Infant mortality rates were particularly high during the study period, with nearly 40% of the children dying before reaching reproductive age (15), similarly to contemporary human populations living without modern medical care (Luttbeg, Borgerhoff Mulder & Mangel, 2000; Sear, Mace & McGregor, 2000). The main causes of death were infectious diseases (Kannisto, Nieminen & Turpeinen, 1999). This high rate of infant mortality was accompanied by high fertility, a typical characteristic of societies prior to the demographic transition. The population was strictly monogamous with remarriage permitted only after the death of a spouse. Inheritance usually favored the eldest son, and the predominant household contained two parents, grandparents, and one or more children. All siblings usually lived close by (Moring, 1993).

Our study includes 388 founder mothers (born from 1709 to 1815) and all of their 2538 offspring (born from 1746 to 1860), reconstructed from three-generation family lines (parents, F_1-offspring and F_2-offspring) (see Table 1). We have full life-history records for mothers (birth date, all marriage events, fertility schedule, death date) and records on their children's recruitment to the breeding population (information on all grandchildren born, N=6486). We can classify each mother having either a rich or poor socioeconomic status based on her husband's occupation (Gillespie, Russell & Lummaa, 2008): mothers whose husband owned land themselves are classified as rich mothers (189 in total), and those mothers whose husband did not own land are classified as poor mothers (199 in total). The latter category mainly includes crofters that worked for other farmers, as well as servants, sailors, and beggars.

Average maternal AFR in the population was around 26 years and, thus, significantly beyond sexual maturity, resembling many current modern Western societies (Sobotka, 2004). Such a late AFR was primarily caused by a late marriage, a unique feature throughout most of Europe in the 18th and 19th centuries ["European marriage pattern"; see Hajnal (1965)], not just limited to Finland. Such delays in Finland, similarly to the whole of Scandinavia, were thought to have been promoted by a long work service necessary before marriage to accumulate necessary capital for the establishment of a future household. For daughters of landowners, the service was often at the parents' farm; for daughters of crofters or other landless people, the service was at other farmers' household as a maid (Moring 1996, 2009).

3.2. Statistical methods

We investigate the associations between delaying AFR and five variables: lifetime number of children, reproductive span, offspring survival, offspring recruitment probability and maternal probability of reproductive failure. To account for

Table 1
Summary of the life history parameters of the P mothers

Socioeconomic status		Age at first reproduction (AFR, years)	Lifetime number of children	Reproductive span (years)	Recruitment probability of children	Probability of children surviving to 15
Rich mothers	min	16.59	1	0	0	0
	max	41.98	16	28.3	1	1
	Mean±SE	25.14±0.31	7.50±0.21	14.86±0.44	0.46±0.017	0.59±0.017
Poor mothers	min	17.78	1	0	0	0
	max	40.47	16	25.94	1	1
	Mean±SE	27.02±0.34	5.63±0.19	12.07±0.44	0.48±0.020	0.61±0.019
All mothers	Mean±SE	26.11±0.23	6.54±0.15	13.43±0.32	0.47±0.013	0.60±0.013
Rich mothers versus poor mothers		$t_{384.45}$=-4.14 p<.001	$t_{380.34}$=6.74 p<.001	$t_{385.96}$=4.48 p<.001	$t_{379.16}$=-0.85 p=.39	$t_{382.37}$=-0.65 p=.52

Reproductive span as 0 denotes to one pregnancy over lifetime (twin or singleton); t test results are obtained by Welch Two-Sample t test in R program language (see Statistical methods section); recruitment and survival probabilities here are calculated as recruitment and survival proportions of all the produced children of a mother.

the fact that the mothers belonged to several parishes (four levels) and to different cohort years (84 levels), we use mixed-effects models, with AFR modeled as a fixed effect and spatial (parish) and temporal (cohort year) scales modeled as crossed random effects. Additionally, we investigate how socioeconomic status influenced such associations by including it (two levels, rich or poor) as a fixed effect.

Because we have no a priori prediction about the shape of associations between AFR and the five response variables mentioned above, we first use nonparametric generalized additive models (GAM) to identify plausible shapes (e.g., linear, quadratic, etc.; see Crawley, 2007). We then use polynomial expressions of AFR where necessary in parametric generalized linear mixed effects models (GLMM) testing the hypothesis that such associations depend on socioeconomic status (interaction terms; see Crawley, 2007; Gillespie et al., 2008). To minimize multicollinearity in polynomial regressions, maternal AFR will be firstly centered around its mean. All coefficients and significance levels are of AFR using centered scale. This treatment will improve the stability of coefficients of predictors significantly, but it will not change AFR-specific estimated values for response variables; thus, to aid reading, in all graphs, AFR is plotted using its original scale. In GLMMs, lifetime number of children is modeled as a Poisson variable and reproductive span as a Gaussian variable. Proportions of offspring recruitment and survival to age 15 years and reproductive failure (binary with some vs. no offspring recruited to the breeding population) are modeled as binomial variables. In other words, regarding offspring quality, because the focus of our study is at the mother rather than offspring level to be able to investigate the overall fitness benefits of a given maternal reproductive strategy (i.e., a given AFR), we investigate the proportion among all born offspring that survived to age 15 or breeding in response to a given maternal AFR. However, an alternative analysis (results not shown separately) at the offspring level controlling for maternal age for each birth and maternal ID shared by all offspring of the same mother reaches similar conclusions.

We use backward stepwise regression based on Likelihood Ratio Tests (see χ^2 values in results) to generate a minimum adequate model (MAM) in each analysis. In plotting, estimated values from regression models are calculated from fixed effects and standard errors of the estimated values are calculated using variance–covariance matrix (Fisher Information Matrix) of fixed-effects of GLMMs (Faraway, 2006; Liao, 2000). All analyses were carried out in the statistical environment R (Version 2.10.0, R Development Core Team, 2009) using statistical packages mgcv for GAM and lme4 for GLMM.

4. Results

4.1. AFR and offspring quantity

The mean AFR for women during our study period was 26±0.23 years, but AFR varied widely among women ranging from 16 to 42 and rich women starting reproduction on average significantly younger than poor women (Table 1; Pettay et al., 2007). Women who reproduced at least once in their lifetime produced, on average, 6.54±0.15 children altogether, but again, this varied among women and was significantly dependent on their socioeconomic status (Table 1; Pettay et al., 2007).

Fig. 1A depicts the association between AFR and lifetime number of children (MAM: lifetime number of children ~AFR+AFR^2+socioeconomic status; see Table 2). Delaying AFR was associated with reduced number of children over the whole range of AFR (AFR: z=-9.65, p<.001; AFR^2: z=-1.71, p=.087). Socioeconomic status had a significantly positive effect on lifetime number of children: for a given AFR, rich mothers had, on average, more children than poor mothers (z=4.69, p<.001). However, there was no significant interaction between socioeconomic status and linear (χ^2_1=0.11, p=.75) and quadratic (χ^2_1=0.067, p=.80) terms of AFR indicating that delaying AFR reduced

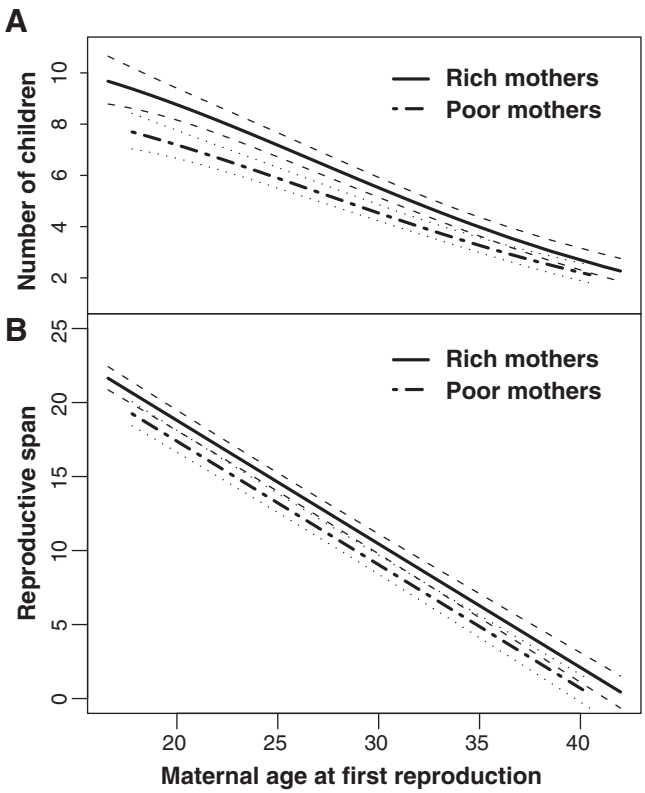

Fig. 1. The associations between a mother's AFR α and her lifetime number of children R_α (A) and reproductive span (difference in years between first and last birth) (B) for rich and poor women. In both panels, bold solid line represents estimations for rich mothers and thin dashed lines represent standard errors; bold dash-dotted line represents estimations for poor mothers and thin dotted lines represent standard errors. In panel (A), standard errors are firstly calculated in logarithm scale because Poisson family is used in GLMM. Then, such errors are transformed back to original scale, which gives rise to slightly asymmetric standard error bands around the regression lines.

Table 2
Regression equations of response variables with respect to centered AFR ($_c\alpha$)

Response variables	Rich mothers	Poor mothers
Lifetime number of children R_α	$e^{1.72-0.0490_c\alpha-0.00128_c\alpha^2}$	$e^{1.92-0.0490_c\alpha-0.00128_c\alpha^2}$
Lifetime reproductive span	$13.70-0.83_c\alpha$	$12.29-0.83_c\alpha$
Offspring probability of surviving to 15	$\frac{e^{0.442+0.0237_c\alpha-0.00145_c\alpha^2-0.000484_c\alpha^3}}{1+e^{0.442+0.0237_c\alpha-0.00145_c\alpha^2-0.000484_c\alpha^3}}$	
Offspring probability of recruitment to breeding p_α	$\frac{e^{-0.181+0.0186_c\alpha+0.000156_c\alpha^2-0.000447_c\alpha^3}}{1+e^{-0.181+0.0186_c\alpha+0.000156_c\alpha^2-0.000447_c\alpha^3}}$	
Probability of reproductive failure P_{rf}	$\frac{e^{-3.57+0.0896_c\alpha+0.0130_c\alpha^2}}{1+e^{-3.57+0.0896_c\alpha+0.0130_c\alpha^2}}$	

lifetime number of children in a similar way in both socioeconomic groups.

The presumable reason for the negative association between lifetime number of children and AFR may be that delaying AFR reduced the reproductive span of women (Fig. 1B) (MAM: maternal reproductive lifespan ~AFR+socioeconomic status; see Table 2). There was no significant interaction between AFR (χ^2_1=0.28, p=.60) and socioeconomic status and delaying AFR reduced reproductive span of women at the same rate in both socioeconomic groups (t=-15.62, p<.001; note that for the case of normally distributed variables, statistical package lme4 does not provide degrees of freedom for t values). Rich mothers had, on average, a longer reproductive span than poor mothers (t=2.81, p<.01).

Fig. 1 also shows that after an AFR of 35, standard error bands for rich and poor mothers cross in cases of both number of children and reproductive span, suggesting possibly less significant difference between them in these two traits. Statistical analysis confirms this point: after 35, for a given AFR, there were no significant differences between rich and poor mothers in their lifetime number of children (χ^2_1=0.14, p=.71) and reproductive spans (χ^2_1=0.69, p=.41).

4.2. AFR and offspring quality

On average, of all offspring born, 60% survived to age 15, and 47% were successfully recruited to the breeding population. Variance component analysis indicates that survival rate at age 15 can account for about 60% of the variance in recruitment probability.

Our analysis shows no association between the probability of offspring surviving to age 15 and maternal AFR in mothers starting reproduction by age 30 (χ^2_1=0.21, p=.65). However, beyond this age, delaying AFR was associated significantly with decreased offspring survival rate at age 15 (z=-2.92, p<.01). The above pattern is shown by a significant cubic term in the model explaining offspring survival probability by maternal AFR (Fig. 2) (MAM: survival probability ~AFR (z=1.36, p=.17)+AFR^2 (z=-0.76, p=.45) +AFR^3 (z=-2.01, p<.05); see Table 2). Both socioeconomic status (χ^2_1=0.032, p=.86) and its interactions with linear term (χ^2_1=0.0092, p=.92), quadratic term (χ^2_1=1.16, p=.28), and cubic term (χ^2_1=0.92, p=.34) of AFR were nonsignificant, indicating that on the whole, offspring survival was not significantly influenced by maternal socioeconomic status.

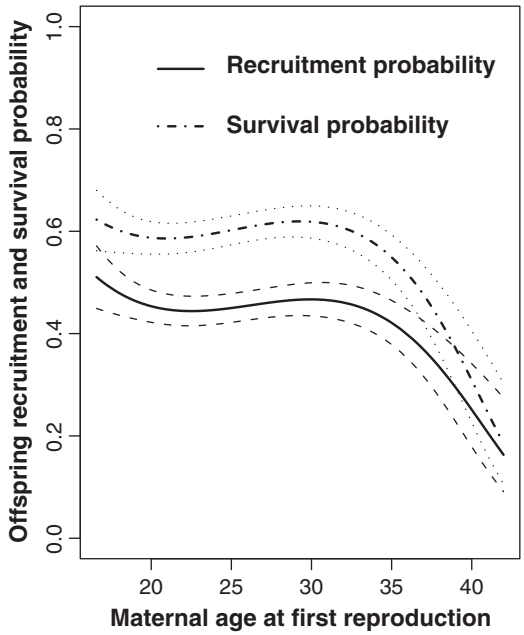

Fig. 2. The associations between a mother's AFR and her offspring quality, measured by probabilities of offspring being recruited to the breeding population and surviving to reproductive age (15). Solid line represents estimated recruitment probability and dashed lines represent standard errors. Dash-dotted line represents estimated survival probability and dotted lines represent standard errors. Since there was no difference between the two types of mothers, only one grouped regression line is plotted for recruitment and survival probability. Back transformation of standard errors in logit scale into original probability scale gives rise to asymmetry in the standard error bands.

The probability of offspring being recruited to the breeding population was also not associated significantly with maternal AFR in mothers starting reproduction by age 30 (χ^2_1=0.43, p=.51). However, beyond this age, delaying AFR was significantly associated with decreased offspring recruitment (z=-2.98, p<.01). The above pattern is illustrated by a quasi-significant cubic term in the model explaining the recruitment probability (Fig. 2) (MAM: recruitment probability ~AFR (z=1.09, p=.28)+AFR^2 (z=0.084, p=.93)+AFR^3 (z=-1.87, p=.062); see Table 2). Both socioeconomic status (χ^2_1=0.26, p=.61) and its interactions with linear term (χ^2_1=0.10, p=.75), quadratic term (χ^2_1=0.22, p=.64) and cubic term (χ^2_1=1.70, p=.19) of AFR were nonsignificant.

4.3. AFR and probability of reproductive failure

Overall, 6% of all first-generation mothers in our dataset (all of whom produced at least one offspring in their lifetime) produced no grandchildren. The number of grandchildren of these mothers ranged from zero to 62 with mean at 16.72±0.03.

The probability of complete failure to gain grandchildren became increasingly higher the longer AFR was delayed

[Fig. 3; MAM: maternal reproductive failure probability ~AFR (z=1.33, p=.18)+AFR^2 (z=2.00, p<.05), see Table 2]. Along the whole scale of AFR (16–42), both socioeconomic status (χ^2_1=1.45, p=.23) and its interactions with linear term (χ^2_1=0.48, p=.49) and quadratic term (χ^2_1=0.15, p=.70) of AFR were nonsignificant.

4.4. Matching data to models

Finally, we compare the theoretical predictions of Model 2 on the association between AFR and probability of reproductive failure with the empirical regression result in the above Section 4.3. To do this, we focus firstly on Model 1 $P_{rf}=(1-p_\alpha)^{R\alpha}$ that describes AFR-specific probability of reproductive failure of a mother. Including the AFR-specific fitted values of probability of offspring recruiting and of number of children produced from the regression equations (see Table 2) into our Mathematical Model 1 allows generating AFR-specific reproductive failure probabilities. Because AFR-associated numbers of children differed significantly between the rich and poor mothers, we generate predictions of reproductive failure probability for the rich and poor mothers separately. Fig. 3 shows that there is a good match between the curves predicted by the model and regression curve of the empirical data: for mothers of both socioeconomic statuses, both the empirical regression and the theoretical prediction indicate that the probability of reproductive failure remained low before AFR of 30; after this point, the probability accelerated.

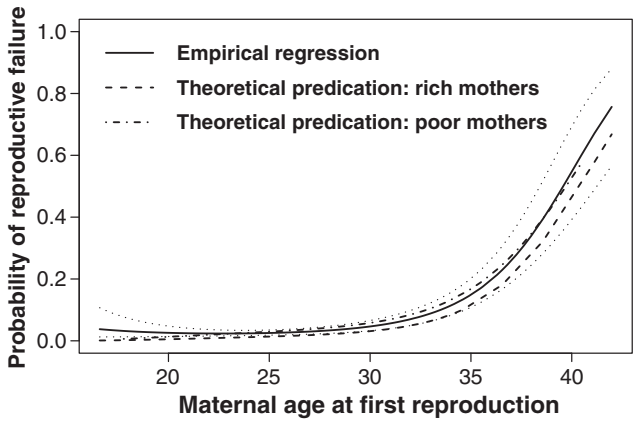

Fig. 3. Empirical logistic regression and theoretical prediction of the association between a mother's AFR and her probability of reproductive failure. Bold solid line represents estimated reproductive probability from logistic regression using empirical data, bold dashed line represents theoretical prediction of this probability for rich mothers and bold dash-dotted line represents theoretical prediction of this probability for poor mothers. Thin dotted line represents standard error lines of estimation from logistic regression. For logistic regression, only one regression line is plotted since there was no significant difference between rich and poor mothers. For theoretical prediction, two lines are plotted since the information of number of children is used, which showed significant difference between rich and poor mothers. Back transformation of standard errors in logit scale into original probability scale gives rise to asymmetry in standard error bands.

Since Model 1 is supported by the empirical data, predictions of Model 2 that concern any change in the probability of reproductive failure associated with the change in the timing of reproduction onset are also supported, since the latter model is a partial derivation of the former one. Delayed AFR was associated non-positively with offspring recruitment probability and negatively with expected lifetime number of children along the whole range of AFR (see Figs. 1A and 2). Thus, according to our theoretical Model 2, the partial derivative of P_{rf} with respective to AFR α will be positive at all AFRs (this derivative can be numerically calculated in an EXCEL form using regression equations in Table 2; this form is available from authors), and consequently, the probability of reproductive failure P_{rf} will increase with delaying AFR along the whole range of AFR. In other words, the earlier AFR was, the lower P_{rf} would be and the average AFR did not minimize P_{rf} in this population. The above predictions by Model 2 are supported by the monotonically upward curve in Fig. 3 that describes the association between a mother's reproductive failure status (binary; see Section 3.2) and her AFR using empirical data. This match between theory and data suggests that, at least in this population, a simple trade-off between potential benefit and cost associated with delayed reproduction underpins whether and how AFR will be associated with specific likelihood of maternal reproductive failure.

5. Discussion

The fundamental role of reproduction is to ensure lineage persistence. Ceteris paribus, the more members a lineage has, the safer it is from extinction (Low et al., 2003). It has been noticed for over 200 years that in humans, delaying marriage and consequently first reproduction is an effective method to reduce the number of children and to control population size (Malthus, 1803). Via its negative association with offspring quantity, delayed AFR may thus be associated with higher vulnerability of lineages to extinction. However, any costs of delays in AFR to offspring quantity should be evaluated against any potential benefits from enhancing offspring quality, for example, through prolonged opportunity to accumulate more social resources so as to improve offspring performance or mating success (Kaplan et al., 2002). Therefore, we combined mathematical models with detailed pedigree records available for three generations of preindustrial Finns to consider the associations between delaying AFR, socioeconomic status and both offspring quantity and quality in determining maternal lineage vulnerability to extinction.

With respect to offspring quantity, for both rich and poor mothers, delayed AFR beyond maturity was associated with reduced number of children, which is consistent with findings from both other pre-industrial societies (Hamilton, 1966) and modern industrialized societies (Kaplan et al., 2002; Low et al., 2002; Sobotka, 2004).

Two points suggest this association to be at least partly causal. Firstly, delayed reproduction led to reduced reproductive span (see Fig. 1B) and thus reduced available reproductive opportunity before menopause, given that menopause has no association with AFR (Borgerhoff Mulder, 1989a; Hill & Hurtado, 1996). Secondly, female fecundity declines with maternal age (Kaplan et al., 2002; Schwartz & Mayaux, 1982; van Noord-Zaadstra et al., 1991).

We also find that the environment played a significant role in modifying offspring quantity, with rich mothers producing on average more children in their lifetime than poor ones. This was likely caused by the earlier AFR and delayed age at last reproduction among rich mothers (Pettay et al., 2007); some noble women (although very few in our study population) might have also adopted the custom of hiring wet nurses to breast-feed their babies (Wirilander, 1974), resulting in shorter inter-birth intervals and higher fertility (Hrdy, 1992). Consequently, rich mothers could achieve the same number of children as poor mothers even if they started reproduction later. However, this advantage for rich mothers disappeared if they delayed their AFR later than 35 (see Section 4.1). This result is consistent with the finding that in this population, late-age (>31) female fecundity (probability of giving birth at a given age) was characterized by large additive genetic variance and significant heritability, but not by significant parental effects (Pettay, Charmantier, Wilson & Lummaa, 2008). The reverse was true for fecundity at young age (<31), which was influenced significantly by wealth and help from kin (Lahdenperä et al., 2004), whereas the estimate of additive genetic variance for this trait was small and the heritability was not significant (Pettay et al., 2008).

With respect to offspring quality, child survival probability remained constant before maternal AFR at 30 and thereafter declined significantly with delayed AFR. The association between offspring recruitment probability and maternal AFR followed a similar pattern; this is not surprising, since in this population recruitment probability was mainly accounted for by survival probability (see Section 4.2). There is no evidence in our dataset that a third contingent factor such as war or famine could have caused both late maternal AFR and low quality of offspring. Consequently, we suggest our survival–AFR association to be at least partly causal, supported by previous studies focusing on changes in offspring survival with maternal age per se. For example, in women, clinical observations indicate that the probability of producing a live birth per pregnancy declines significantly with maternal age after age 30 (Schwartz & Mayaux, 1982; van Noord-Zaadstra et al., 1991; also Raymond et al. (1994) for reproduction after 35). Also, later maternal age is associated with higher risk of low birth weight of infants, which may be linked with lower survival rate in the future (Geronimus, 1996; Lee et al., 1988). Such previous studies on humans documenting age-related changes in offspring quality have however typically investigated the consequences of maternal age per se across a women's reproductive lifespan but have failed to consider how any offspring quality outcome is modified by the onset age of reproduction for these mothers. Similarly, in other large mammals such as the red deer (*Cervus elaphus*), a calf's probability of survival to its second year declines if born to mothers beyond a certain age (Clutton-Brock et al., 1982), but less is known in animals, too, about the consequences of delayed AFR on their offspring ultimate quality. To our knowledge, our study is the first one in any species to document an association between delayed maternal AFR and offspring long-term quality in terms of breeding probability.

The environmental conditions, captured in mother's socioeconomic status, had no significant effect on AFR specific offspring survival rate: rich mothers did not have a significantly higher offspring survival rate than poor mothers for a given AFR. This result is consistent with other studies on the same population (Gillespie et al., 2008; Pettay et al., 2007). This may have been caused by a highly variable environment during the study period: famines were both unpredictable and frequent, while effective medical care was not available in cases of infectious disease outbreak (Soininen, 1974). According to Kannisto et al. (1999), the main causes of death among children during the study period were infectious diseases (e.g., small pox and tuberculosis), against which wealth provided little protection. So even if rich mothers were able to invest more in their children, this investment may not have brought about a significant return in terms of improved survival probability per offspring. That external socioeconomic status did not have significant effect on offspring survival further suggests that maternal AFR might have a significant effect on offspring survival through its effect on internal maternal physiology.

Finally, maternal probability of reproductive failure increased with increasing AFR and Theoretical Model 2 explained how this occurred as the result of associations between offspring quantity and quality and AFR, as indicated in Section 4.4. The lack of difference between the rich and poor mothers in the positive association between reproductive failure probability and AFR may be caused by several factors. For AFRs earlier than 30 years, the numbers of children for both rich and poor mothers were large; for example, even with AFR at 30 years, the number of children for poor mothers was around 4.5 and that for rich mothers about 5.5 (Fig. 1A). Thus, according to Model 1, probabilities of reproductive failure were very low for both types of mothers. For mothers starting their reproduction in their 30s, the difference in the numbers of children between rich and poor mothers was less pronounced or nonexistent. The same recruitment probability of offspring for both mothers resulted in a similar probability of reproductive failure. Consequently, even if a mother could get into a higher socioeconomic status by delaying her AFR, her final adaptive value, measured by $1\text{-}P_{rf}$ (probability of maintaining her lineage), may decline with increasing AFR, especially after 30. Thus, on the whole, our conclusion from this population is consistent with Low et al. (2002): after sexual maturity,

delaying AFR did not bring evolutionary benefits, and the costs for lineage persistence from delayed reproduction cannot be compensated by improvement in socioeconomic status, especially after age 30. However, our study adds to previous studies by providing the first empirical evidence on how delays in AFR relate to not only offspring quantity but also offspring quality in terms of breeding performance in determining long-term lineage persistence. Furthermore, since both offspring quantity and quality were associated nonfavorably with AFR, delayed AFR has detrimental fitness consequences whatever fitness measure is used, such as the number of grandchildren (statistics not shown). Thus, our main conclusion is robust to the choice of a fitness measure.

In contrast to the hypothesis that delays in AFR may be a strategy to raise socioeconomic status to improve future offspring quality (Kaplan et al., 2002), delayed female AFR appeared to have resulted from constraints on early reproduction in this population. One of the most important constraints was age at marriage, which explained about 80% variance in maternal AFR and longer delay in marriage and consequently later AFR did not result in marrying a rich husband. Actually, females whose husbands were rich married on average earlier and then had their first child younger than females whose husbands were poor (Table 1; Moring 1996; Pettay et al., 2007). This is consistent with selection for young wives by German landowners in the same period (Voland & Engel, 1990) and early-maturing women by rural Kipsigis men with more bridewealth payments in Kenya (Borgerhoff Mulder, 1989b). Since we have controlled a factor relevant for marriage constraint to some degree (i.e., socioeconomic status), our result, together with above discussion, suggests that delays in AFR per se at least partly led to lower offspring quantity and quality and then increased probability of reproductive failure. However, it is still possible that individuals with earlier AFR might have been of higher phenotypic quality than those with later AFR, leading them to be more successful in producing offspring of higher quantity and quality, or our classification of socioeconomic status may be too rough to grasp the exact character of environmental conditions. Furthermore, although women in our study population were unable to increase their offspring quality by delaying reproduction given the very limited options of education or economic autonomy and inability to improve opportunities to marry a rich husband by delaying AFR, it is possible that such effect is present in other societies. Further studies investigating long-term measures of offspring quality as well as quantity as a function of maternal AFR are thus warranted.

Our models give a general formalization of the relationship between AFR and the probability of reproductive failure. That is, even in societies other than preindustrial Finland, the probability of reproductive failure can be linked with AFR-specific offspring quantity and quality in the form of Model 1 and how the probability will change with delayed AFR can be depicted with Model 2. Thus, the findings of this paper could be used as a platform for predicting probability of reproductive failure in other populations by analyzing the reproductive success costs and benefits associated with delayed AFR. However, how offspring quantity and quality change with AFR in other ecological or cultural contexts may be different from curve patterns in Figs. 1A and 2, and so, the pattern of how probability of reproductive failure changes with AFR may be also different from Fig. 3. What is especially worth mentioning is that, in modern societies where the environment may be comparatively more constant and predictable and advanced medical care is available, higher investment in offspring by delaying AFR (given this can improve socioeconomic status) may bring more return in offspring quality, such as their physiological and psychological development, income, and socioeconomic status (Kaplan et al., 2002; Nettle, 2010), which all may contribute to higher recruitment probability of offspring. Thus, there may be an intermediate AFR that leads to a minimum probability of reproductive failure, which could be one explanation for increasingly delayed AFR in modern society. However, advanced maternal age (>35 years) at first reproduction will likely manifest similarly high costs to maternal lineage persistence as documented in this study. The reason is that compared with mothers with earlier AFRs, mothers with advanced AFRs will have highly reduced numbers of children, and their children may have higher mortality due to lower birth weights (Geronimus, 1996; Lee et al., 1988; Raymond et al., 1994; Vanlandingham et al., 1988). Also, such kind of reproduction may bring a higher probability of producing offspring with birth defects (Griffiths, Wessler, Lewontin & Carroll, 2008; Hollier, Leveno, Kelly, Mclntire & Cunningham, 2000; Tarin, Brines & Cano 1998). Thus, mothers with advanced maternal AFRs may still have higher probability of reproductive failure.

Like reproductive success, probability of reproductive failure provides another perspective to investigate individual fitness (Stearns 1992) or in other words, “knowing what not to do sometimes makes clearer what should be done” (Stearns & Hoekstra, 2005). Along this line of reasoning, further work should be undertaken to investigate (1) the link between reproductive success and probability of reproductive failure, (2) how natural selection works through reproductive failure, and (3) evolutionary psychology of individual reproductive decision making relative to reproductive failure.

Acknowledgments

We thank Alexandra Alvergne, Andrew Beckerman, Fansuo Geng, Duncan Gillespie, Ben Hatchwell, Bobbi S. Low, Jenni Pettay, Ian Rickard, Matt Robinson, Andrew F. Russell, Jessica Stapley and Masao Yamashita for comments. We thank Lasse Iso-livari, Kimmo Pokkinen, Aino Siitonen and Timo Verho for collecting the genealogical data. We thank the University of Sheffield (JL), Kone Foundation (VL, JL), the European Research Council and the Royal Society of London (VL) for funding.

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