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AGE-SPECIFIC GENETIC AND MATERNAL EFFECTS IN FECUNDITY OF PREINDUSTRIAL FINNISH WOMEN

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A population's potential for evolutionary change depends on the amount of genetic variability expressed in traits under selection. Studies attempting to measure this variability typically do so over the life span of individuals, but theory suggests that the amount of additive genetic variance can change during the course of individuals' lives. Here we use pedigree data from historical Finns and a quantitative genetic framework to investigate how female fecundity, throughout an individual's reproductive life, is influenced by "maternal" versus additive genetic effects. We show that although maternal effects explain variation in female fecundity early in life, these effects wane with female age. Moreover, this decline in maternal effects is associated with a concomitant increase in additive genetic variance with age. Our results thus highlight that single over-lifetime estimates of trait heritability may give a misleading view of a trait's potential to respond to changing selection pressures.

KEY WORDS: ***Homo sapiens*, maternal effects, natural selection, reproduction, senescence.**

Estimating the heritability of traits is fundamental to understanding evolutionary change. In general, studies assume that the heritability of traits does not change throughout the life of an organism. However, evolutionary theories of senescence predict that the additive genetic variance in fitness traits is age dependent. According to theories of senescence, aging leads to increased additive genetic variance in late life due to the accumulation of late-acting mutations (Medawar 1952; Williams 1957; Rose 1991). Quantitative genetic evidence for such age-related increases in the expression of genetic variation is well documented for life-history traits in domestic animals (e.g., Arbuquerque and Meyer 2001) and laboratory populations of *Drosophila* (Tatar et al. 1996; Hughes et al. 2002, but see Shaw et al. 1999). Similar studies of wild animals are generally lacking, presumably because of the difficulty of following dispersing offspring and the fact that few individuals commonly survive to an old age under natural conditions (Kirkwood and Austad 2000). However, a few recent studies of wild animals suggest that heritability of important life-history traits can increase with age. For example, age-dependent increases in additive genetic variances were shown for timing of egg laying in mute swans (*Cygnus olor*) (Charmantier et al. 2006), for body mass in bighorn sheep (*Ovis canadensis*) (Reale et al. 1999),

and for annual fitness in female collared flycatchers (*Ficedula albicollis*) (Brommer et al. 2007). The presence of such an age-dependent additive genetic variation suggests that the common practice of using estimates of heritability assumed constant over all age classes can give an incomplete picture of whether and how selection can lead to an evolutionary change in trait mean values (Williams 1957). Although selection may be strong earlier in an individual's life, there might be little genetic variation that can lead to evolutionary change, while later in life selection can be weaker but genetic potential for evolutionary change may be higher.

Human life-history evolution has attracted attention, in part because of its obvious wide-reaching relevance and its unusual nature (Hill and Kaplan 1999; Kaplan et al. 2000; Mace 2000). However, such studies have typically investigated human evolution from a phenotypic perspective. For example, studies on selection for age at first and last reproduction and fecundity (number of offspring born) in humans living both in modern and traditional settings are now well established (reviewed in Lummaa, 2007). Such studies reveal that the most important component of female fitness (i.e., that with the highest selection differential) in both traditional and modern settings alike is the number of offspring delivered over an individual's lifetime (Käär et al. 1996; Helle et al. 2005).

However, the evolution of human life-history traits is not only a consequence of phenotypic selection, but most importantly depends on the level of additive genetic variance for these traits. Despite this, this latter area of human life-history evolution has been almost wholly devoid of attention. A lack of information on the heritability and genetic correlations of reproductive traits in human populations has resulted in a limited understanding of whether the documented phenotypic selection could lead to evolutionary changes over time. Two recent exceptions provide interesting insights into the evolution of human life-history traits, including fecundity. First, Kirk et al. (2001) used a twin study designed to estimate heritabilities for a set of life-history traits in a contemporary Australian population. They found age at menarche, age at first reproduction, and age at menopause to be heritable to some extent (23–45%). Second, Pettay et al. (2005) used maximum-likelihood (REML) based techniques implementing an animal model (Lynch and Walsh 1998) on pedigree records of preindustrial Finns to investigate heritabilities and maternal effects on life-history traits. In these women, fecundity had a significant heritability of 0.31, potentially permitting rapid evolutionary response to selection (Pettay et al. 2005).

However, female fertility in humans also shows clear changes with age, possibly affecting such calculations. First, parental effects may be more important for successful reproduction in young rather than older women. For example, wealth of the parents correlates both with female body condition and their related age at menarche (reviewed in Voland 1998) and with reproductive success (Voland 1990; Pettay et al. 2007; Gillespie et al. 2008). Furthermore, family help, such as presence of grandmothers, which is likely to be greatest early rather than later in an individual's reproductive life, may be an important determinant of female reproductive rate: daughters with alive, close-living postreproductive mothers show reduced interbirth intervals (Voland and Beise 2002; Lahdenperä et al. 2004) and increased breeding probability (Sear et al. 2003). Second, female fertility shows clear senescence with age: natural conception rates fall rapidly from the mid-30s onwards (Sievert 2001), and the risk of unsuccessful pregnancy (miscarriage) increases, whereas the developmental and genetic problems of offspring may increase with maternal age (Holman and Wood 2001). Consequently, a single over-lifetime estimate of heritability for female fecundity in humans may give an oversimplistic view of its expected response to the documented selection pressures.

Although it therefore seems likely that the heritability of fecundity will vary with age in humans, whether this is the case is currently unknown. In this study, we investigate the importance of maternal effects in relation to the additive genetic variance in fecundity displayed in women of varying ages living under natural fertility conditions without availability of modern contraceptive methods and medical care. To estimate the heritabilities, we applied restricted maximum-likelihood (REML) estimation and animal models extended to random regression models in which additive genetic and maternal variances were a function of age (Meyer and Hill 1997). The advantage of animal models compared with more traditionally used parent–offspring and sibling regression analyses in human studies is that it simultaneously incorporates information from a variety of relationships of different degrees, such as offspring, parents, grandparents, full-siblings, and half-siblings, which makes it a more powerful method than the traditional approaches (Lynch and Walsh 1998; Kruuk 2004). REML analyses also have less strict assumptions about selection patterns or inbreeding and do not require balanced datasets, which makes them considerably more amenable to data from nonexperimental populations (Lynch and Walsh 1998; Kruuk 2004).

Materials and Methods

DATA

We studied the age-specific maternal effects and heritability in fecundity of preindustrial women using extensive demographic pedigree records available for a historical Finnish population. The study period runs from 1745 to the late 19th century, a time when industrialization had not yet begun in Finland, and fertility and mortality rates are considered natural, before the introduction of effective contraception or modern medical care (Soininen 1974). We collected pedigrees of life-history data following four

generations using a large genealogical database (Kojonen 1971; Genealogia Sursilliana CD-2000 database, I. B. Voipio, pers. comm.). Our dataset includes 194 base individuals, who were born between 1745 and 1765 in four different parishes (Oulu, Kokkola, Kaarlela, and Kuusamo), and all of their descendants up to the great-grandchildren level, as well as spouses of all married individuals, which amounts to a total of 5619 individuals over four generations. Complete individual life histories (birth, death, and all reproductive events) were recorded for three generations, resulting in a total of 1894 individuals, of which 904 are females. Of those 904 females with complete life histories, 352 survived to adulthood and reproduced.

The data include known paternities for all individuals. We expect the incidences of extra-pair paternities (EPPs) to be low in the study population, given that the mean estimate for EPPs around the world using modern human data fall between 3% and 9% (Anderson 2006). Extra-marital affairs were a strict taboo of the Lutheran church and punishable by the society (Sundin 1992) and the historical Finns, with strict social and religious norms, are likely to be positioned at the lower end of this EPP range. The EPP rates detected in human populations are not sufficient to trigger significant biases in the heritability estimates, and even if the EPPs had been common, this should be a conservative error, because in that case heritabilities would be underestimated (Charmantier and Réale 2005). Furthermore, even if young women had higher infidelity rate this would affect the whole pedigree and all age-specific genetic variance would be affected at all ages.

STATISTICAL ANALYSES

We conducted two types of analyses using (1) fecundity pooled over young versus old ages, and (2) age-specific fecundity records. Both approaches made use of animal models in which the phenotype of each individual is broken into components of fixed and random effects (Knott et al 1995; Lynch and Walsh 1998). A key premise of these models is that they use pedigree information with relatedness between individuals to quantify the component of additive genetic (co)variances in the focal traits, while controlling for a suite of other fixed and random effects (Kruuk 2004). Hence an individual's genetic merit will be based on phenotypic measurements from itself as well as from its relatives, allowing in theory the estimation of its additive genetic value even in the absence of any measurement on the focal individual.

In our population, the mean and median age at which women gave birth was 31 years. Consequently, using the first approach, we summed the number of children born to a woman before and at the age of 31 to obtain "early age fecundity" and after the age of 31 to obtain "late age fecundity." This means that the whole lifetime fecundity is split into two and each woman has a value for both early age fecundity and late age fecundity (if she has reproduced after 31). Variance components and heritability values for early and late age fecundity, as well as genetic covariance between the two traits, were estimated in a bivariate animal model using a multivariate REML mixed model procedure . Study parish, birth cohort, and social class (four classes, from rich to poor; see Pettay et al. 2007) were fitted as fixed effects because general linear models indicated that the first factor had a significant effect on early age fecundity and the two others on late age fecundity (with late fecundity highest for intermediate wealth and lowest in the richest class). Maternal identity was included as a random effect. Because in most cases each woman had only one husband and father of her children, the "maternal effect" discussed here also includes paternal effects and other environmental variation shared by siblings. For individual i with mother j, the phenotype y was modeled as:

$$y_i = \mu + (\textit{fixed effects}) + a_i + m_j + e_i,$$

where μ is the population mean, a_i is the individual's additive genetic value, m_j is the maternal effect, and e_i is the random residual value. Hence the total phenotypic variance (V_P) was portioned as: $V_P = V_A + V_M + V_R$, with additive genetic variance (V_A), maternal effect variance (V_M), and residual variance (V_R), respectively. Although the traits used in this analysis showed departure from a normal distribution, the restricted maximum likelihood estimation procedure that is used is fairly robust to departures from normality; deviations may affect optimality properties, but estimates remain unbiased (Shaw 1987; Lynch and Walsh 1998). In fact, no data transformation improved the normality of the data, and in the case of late age fecundity, residuals of the best-fit model were normal. Variance components and their ratio over V_P and standard errors were computed using ASReml release 2.0 (Gilmour et al. 2002). Variance components were constrained to positive values, resulting in a narrow sense heritability ($h^2 = V_A/V_P$) varying between 0 and 1. Significance of the variance components and heritabilities were determined by one-tailed t-tests. Coefficients of additive genetic variance (CVA; Houle 1992) were also estimated to facilitate comparison of genetic variance between the two traits while accounting for the difference in their means.

In the second approach, age-specific fecundity was used in a random regression extension of the animal model (Kirkpatrick et al. 1990; Meyer and Hill 1997; Wilson et al. 2005). Random regressions allow the variance of random effects to be a function of a covariate, here the age of an individual. Their application to senescence analysis in wild populations is very recent (Brommer et al. 2007; Wilson et al. 2007; Nussey et al. 2008), providing a powerful means to test for predictions of senescence evolution theory, such as increase in V_A in late ages (Charlesworth 1990). To analyze fecundity as a binary variate (0,1), twins and triplets (1.23% of all births) were considered similarly as single child births (i.e., fecundity = 1). The models described below to

analyze these binary data were Generalized Linear Mixed Models (GLMMs) with a logit link, run with ASReml 2.0. The minimum age of reproduction was 16 with a maximum of 54 and an average age of last reproduction of 40 (Pettay et al. 2005). However, the mean incidence of reproduction in a given year was very low (i.e., close to zero) for the earliest and latest ages, and this may be expected to cause poor performance of generalized models. We therefore restricted the analysis to ages 19 to 45 inclusive and used a subset of 236 reproductive women for whom complete data were available (i.e., no missing fecundity records).

The additive genetic, maternal, and permanent environment (due to persistent between-individuals differences over and above additive genetic effects, Kruuk 2004) effects were modeled as polynomial regressions on female age of order 0 (i.e., constant) or 1 (i.e., a linear function). Fixed effects included mean-centered age, study parish, birth cohort, and social class. Fecundity of individual i at age x was modeled as:

$$y_{ix} = \mu + (\textit{fixed effects}) + f_1(a_i, n_1, x) + f_2(pe_i, n_2, x) + m_j + e_{ix},$$

where $f_1(a_i, n_1, x)$ is the random regression function based on orthogonal Legendre polynomials of degree n_1 of additive genetic values over age (x); f_2 the equivalent for permanent environment effects; m_j is the maternal effect; and e_{ix} is the random residual value for individual i at age x. Residual effects were modeled using a diagonal error structure, such that separate residual variances were estimated for each of five age classes (19–24, 25–29, 30–34, 35–39, 40–45). Although designation of age classes is arbitrary here, this partitioning avoids the otherwise implicit assumption that residual variance is homogenous with age, an assumption that, if violated, could bias estimation of genetic parameters. Covariance matrices for the additive genetic function (f_1) were estimated and transformed to give the corresponding G matrix for age-specific fecundities, with approximate standard errors determined following Fischer et al. (2004).

Results and Discussion

When considering early and late age fecundity as distinct reproductive traits, early female fecundity was influenced by a significant maternal effect variance whereas the estimate of additive genetic variance (V_A) for this trait was small, and the heritability was nonsignificant (Table 1). The reverse was true for fecundity at late age, which was characterized by a larger V_A (and CV_A) and significant heritability, whereas there was no statistical support for maternal effects (Table 1). These results suggest that life-history traits, such as female fecundity, can have varying potential to evolve during an individual's lifetime. Nevertheless, this conclusion should be made cautiously because differences in additive and maternal components of variance were not themselves significant. Furthermore, although CV_A was larger at late age (suggesting the increase in V_A is not purely a scale effect associated with an increased phenotypic mean), approximating standard errors for coefficients of variation is particularly problematic (Vangel 1996), and we are unaware of any randomization test appropriate for use with complex pedigrees. Consequently, we were unable to assess the statistical significance, or lack thereof, of the observed difference in CV_A between early and late age fecundities.

Early in an individual's reproductive career, maternal effects were more important than additive genetic effects for female fecundity, a result that was not perceivable in previous studies in which fecundity was estimated over the whole life span (Pettay et al. 2005). Maternal effects occur when the phenotype of the mother affects the phenotype of her offspring over and above the direct effect of genes she passes on (Falconer and Mackay 1996). For example, maternal care and investment in offspring can play a major role in early life (Mousseau and Fox 1998; Lummaa and Clutton-Brock 2002), whereas later on offspring phenotype may be determined mainly by the genotype and/or by other environmental factors such as habitat or partner quality (e.g., Price and Grant 1985; Health et al. 1999). In humans, maternal effects such as grandmother effects (Hawkes et al. 1998) may be important in early reproductive life when a mother of a woman

Table 1. Mean, sample size, estimates for (co)variance components for female early and late fecundity (V_P=phenotypic variance, V_A, additive genetic variance; V_M, maternal effect variance; V_R, residual variance), their CV_A (coefficient of additive genetic variance), heritability (h^2) and ratio of maternal over total phenotypic variance (m^2), and their associated *t*-test *P*-values.

	Mean	*N*	V_P (SE)	V_A (SE)	V_M (SE)	V_R (SE)	CV_A (SE)	h^2 (SE)	*P*-value	m^2 (SE)	*P*-value
Early age fecundity	2.7	352	4.07 (0.33)	0.53 (0.65)	1.01 (0.44)	2.52 (0.47)	26.85	0.13 (0.16)	0.20	0.25 (0.10)	0.01
Late age fecundity	3.5	302	4.82 (0.41)	1.20 (0.74)	0.09 (0.45)	3.54 (0.41)	31.60	0.25 (0.14)	0.04	0.02 (0.09)	0.42
Covariances between early and late fecundity			1.20 (0.28)	0.49 (0.53)	0.39 (0.34)	0.32 (0.41)					

(or that of her husband) is still alive and potentially able to help her offspring in their reproductive attempts. Accordingly, daughters with living postreproductive mothers show reduced interbirth intervals (Lahdenperä et al. 2004) and increased breeding probability (Voland and Beise 2002; Sear et al. 2003). Consistent with this idea, a previous study failed to detect significant additive genetic effects on the age at first reproduction in historical Finnish women, but instead highlighted significant maternal effects affecting this trait (Pettay et al. 2005). The absence of maternal effects influencing late age female reproduction (Table 1) is in line with evidence that grand-maternal effects appear to weaken with age. For example, Lahdenperä et al. (2004) found that in historical Finns, living grandmothers failed to have any detectable effect on the interbirth intervals of their daughters' late reproductive career, and only influenced the survival of grand-offspring up to the age of 65 years. Although the evidence for such grandmother effects is correlational, based on the assumption that alive mothers help their adult daughters, and this may underestimate or overestimate the true effects of grandmaternal assistance, the effects are unlikely to arise due to confounding genetic or nongenetic sources of variation (e.g., between family-variation in health). For example, the grandmother effects detected in the historical Finnish population are robust to controlling for individual differences in socioeconomic status, birth order, or within-family death rates from infectious diseases (most common cause of death), and hold when comparing the success of different offspring from the same family when their mother was alive versus when she was dead, or offspring from the same family who lived in the same village as their alive postreproductive mother versus those that lived elsewhere (Lahdenperä et al. 2004). Furthermore, the presence of the (grand)father is not related to similar higher reproductive success of their adult offspring and does not aid grandchild survival, even though such associations would also be predicted if the grandmother effects arose purely due to genetically transmitted health characteristics or other familial/environmental similarities (Lahdenperä et al. 2007). Other studies able to record the amount of food provided by grandmothers or other type of help directed toward the adult offspring provide further support for the positive effects of mothers on the success of their daughters (Hawkes et al. 1997; Gibson and Mace 2005).

In a subsequent analysis, we used random regression animal models in which all data from early and late life were pooled and additive genetic and permanent environment effects were modeled as linear functions of age. Using zero order (i.e., constant) functions of age, additive and permanent environment effects were constrained to be constant and the associated variance components (± SE) were estimated as $V_A = 0.372 \pm 0.138$ and $V_{PE} = 0.061 \pm 0.105$. Maternal effects were not statistically significant (based on SE) when fitted across all ages. However, with maternal effects specified as present only in earlier life (age ≤ 31; based on results discussed above) maternal identity did explain a significant amount of the phenotypic variance ($V_M = 0.370 \pm 0.102$). Thus both modeling approaches used indicate that maternal effects on female fecundity are present in early reproductive life but may be less important later on.

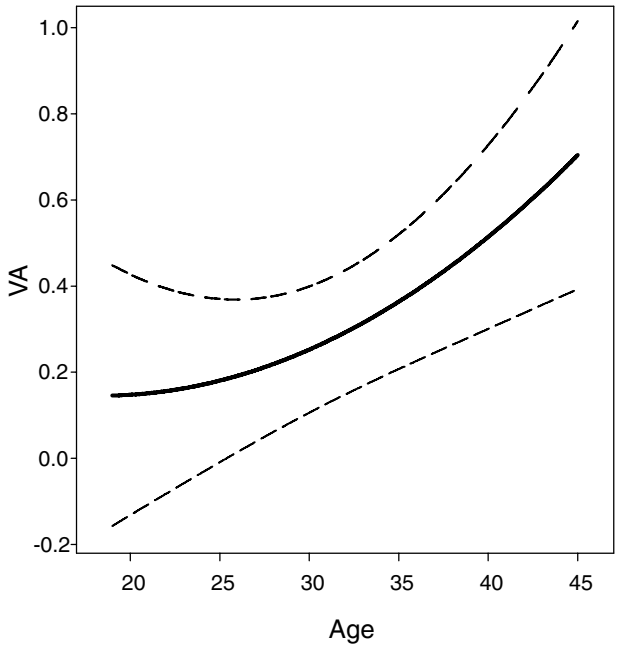

Figure 1. Age-specific additive genetic variance (with 95% confidence limits) in fecundity of preindustrial Finnish women.

Using first-order functions of age in the random regression model revealed a pattern of increasing V_A for fecundity with age (Fig. 1). An important limitation of the current analysis is that methods of model comparison for GLMMs are not yet available. Specifically, likelihood-ratio tests are not valid with nonnormal errors, and deviance-based tests used for generalized linear models are inappropriate when random effects are included. Thus, we are unable to test at present whether the first-order functions produce a significantly improved model, although confidence intervals around age-specific V_A (Fig. 1) certainly suggest that a cautious interpretation is appropriate. Nevertheless, with this statistical caveat in mind, the parameter estimates generated show an increase in additive genetic variance with age, consistent with the above analyses of early and late age fecundity. Based on the estimated standard error, the maternal effect (specified only on younger woman as discussed above) was again statistically significant ($V_M = 0.258 \pm 0.109$).

Analyses of fertility-related behaviors and fertility outcome using human twin designs have previously shown that these components of human fertility display a genetic variance (reviewed in Kohler et al. 2006) that can vary according to the societal and economic context. Here, we show that genetic variation in fecundity varies also during the life stages of women. Such increases in

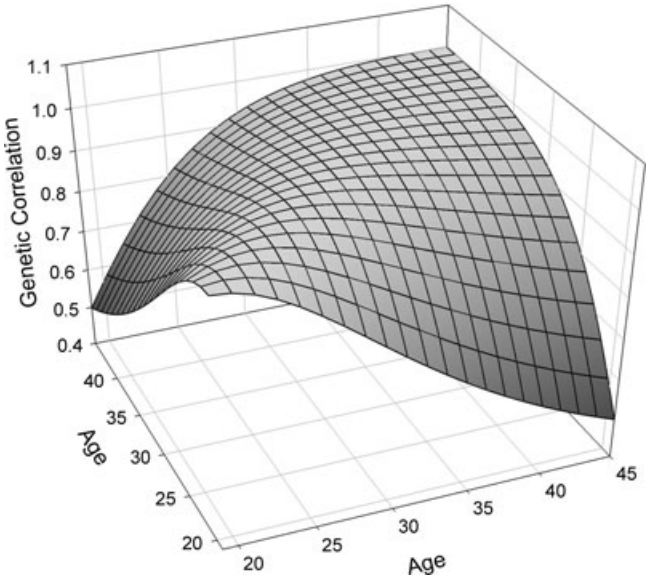

Figure 2. Additive genetic correlations across age-specific fecundities for preindustrial Finnish women.

the expression of genetic variation with age are concordant with previous results from studies investigating age-specific genetic variance in female fecundity on laboratory (Tatar et al. 1996) or wild animals (Charmantier et al. 2006). The increase in late V_A in fecundity is in agreement with the documentation of high heritabilities of both the ages at last reproduction (Pettay et al. 2005) and menopause in humans (Kirk et al. 2001), and predicted by the senescence theories (Rose 1991; see also Snoke and Promislow 2003).

Finally, the random regression animal models in which additive genetic and permanent environment effects were modeled as linear functions of age also provide more detailed results on correlations between fecundity at different ages. In our initial animal model, additive genetic, maternal, and residual covariances between early and late fecundity were nonsignificant (Table 1), whereas there was a significant positive phenotypic correlation providing no support for a trade-off between early and late fecundity. The random regression approach suggests overall positive genetic covariances between close ages, with corresponding genetic correlations declining with time between measurement ages from +1 to a minimum of 0.50 between ages 19 and 45 (Fig. 2). Hence there is no evidence here of a genetic trade-off (Stearns 1989) which would translate into negative genetic correlations between early and late fecundity, rather simply the positive correlations among traits expressed at close ages as witnessed here.

This is, to our knowledge, the first attempt to estimate age-specific variation in the genetic make-up of a human life-history trait. Our results suggest that heritability of female fecundity is strongly age dependent, with high levels late in life, whereas the importance of maternal effects is restricted to the early reproductive life. Classically, reproduction early in individuals' lives is given more emphasis in terms of selection and fitness, especially in growing populations (individual lambda; McGraw and Caswell 1996), due to the decline with age of the force of natural selection on fitness traits (Williams 1957). In theory, small amounts of heritability in early life fecundity, when selection is strongest, can be counterbalanced by larger significant heritability later in life when selection is weakest, implying that the scope for an evolutionary response might be minimal (see also discussion in Helle et al. 2005). Selection may deplete additive genetic variation of traits that are closely associated with fitness (Falconer and Mackay 1996), which could explain the lack of heritability in middle-age fecundity. In addition, a more relaxed selection in later age would lead to the accumulation of deleterious mutations resulting in an increase of additive genetic variation (Medawar 1952). In this study, higher estimate for heritability of late age fecundity is also explained by the decline in maternal effects, a process not mutually exclusive from the increase in additive genetic variance. However, contrary to many animals, women have high survival to later age classes: data from modern hunter–gatherers suggest that a large proportion (one-third or more) of the adulthood population may be over age 50 even in populations with short overall life expectancy at birth (Hawkes and Blurton Jones 2005; Gurven and Kaplan 2007). Hence, the evidence brought forward by this study of higher additive genetic variation in fecundity late in life emphasizes the importance of late age reproductive events for evolution by natural selection in human populations.

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LITERATURE CITED

Anderson, K. G. 2006. How well does paternity confidence match actual paternity? Evidence from worldwide nonpaternity rates. Curr. Anthropol. 47:513–520.

Arbuquerque, L. G., and K. Meyer. 2001. Estimates of covariance functions for growth from birth to 630 days of age in Nelora cattle. J. Anim. Sci. 79:2776–2789.

Brommer, J. E., A. J. Wilson, and L. Gustafsson. 2007. Exploring the genetics of aging in a wild passerine bird. Am. Nat. 170:643–650.

Charlesworth, B. 1990. Optimization models, quantitative genetics, and mutation. Evolution 44:520–538.

Charmantier, A., and D. Réale. 2005. How do misassigned paternities affect the estimation of heritability in the wild? Mol. Ecol. 14:2839–2850.

Charmantier, A., C. Perrins, R. H. McCleery, and B. C. Sheldon. 2006. Age-specific genetic variance in life-history trait in the mute swan. Proc. R. Soc. Lond. B. 273:225–232.

Falconer, D. S., and T. F. C. Mackay. 1996. Introduction to quantitative genetics. Longman, Essex, UK.

Fischer, T. M., J. H. J. Van der Werf, R. G. Banks, and A. J. Ball. 2004. Description of lamb growth using random regression on field data. Livest. Prod. Sci. 89:175–185.

Gibson, M. A., and R. Mace. 2005. Helpful grandmothers in rural Ethiopia: a study of the effect of kin on child survival and growth. Evol. Hum. Behav. 26:469–482.

Gillespie, D. O. S., A. F. Russell, and V. Lummaa. 2008. When fecundity does not equal fitness: evidence of an offspring quantity versus quality trade-off in pre-industrial humans. Proc. R. Soc. Lond. B. 275: 713–722.

Gilmour, A. R., B. J. Gogel, B. R. Cullis, S. J. Welham, and R. Thompson. 2002. ASReml user guide release 1.0. Hemel Hemstead, UK: VSN International Ltd., Hemel Hempstead, UK.

Gurven, M., and H. Kaplan. 2007. Longevity among hunter-gatherers: a cross-cultural examination. Pop. Dev. Rev. 33:321–365.

Hawkes, K., and N. J. Blurton Jones. 2005. Human age structures, paleodemography and the grandmother hypothesis. Pp. 118–140 *in* E. Voland, A. Chasiotis, and W. Schiefenhovel, eds, Grandmotherhood: the evolutionary significance of the second half of female life. Rutgers Univ. Press, New Brunswick, NJ.

Hawkes, K., J. F. O'Connell, and N. G. Blurton Jones. 1997. Hadza women's time allocation, offspring provisioning, and the evolution of long postmenopausal life spans. Curr. Anthropol. 38:551–577.

Hawkes, K., J. F. O'Connell, N. G. Blurton Jones, H. Alvarez, and E. L. Charnov. 1998. Grandmothering, menopause, and the evolution of human life histories. Proc. Natl. Acad. Sci. USA 95:1336–1339.

Heath, D. D., C. W. Fox, and J. W. Heath. 1999. Maternal effects on offspring size: variation through early development of Chinook salmon. Evolution 53:1605–1611.

Helle S., V. Lummaa, and J. Jokela. 2005. Are reproductive and somatic senescence coupled in humans? Late but not early, reproduction correlated with longevity in historical Sami women. Proc. R. Soc. Lond. B. 272:29–37.

Hill, K., and H. Kaplan. 1999. Life history traits in humans: theory and empirical studies. Annu. Rev. Anthropol. 28:397–430.

Holman, D. J., and J. W. Wood. 2001. Ecology, reproduction, and human evolution. Pp. 1–12 *in* Ellison, P. T. (ed.) Reproductive ecology and human evolution. Aldine de Gruiter, New York.

Houle, D. 1992. Comparing evolvability and variability of quantitative traits. Genetics 130:195–204.

Hughes, K. A., J. A. Alipaz, J. M. Drnevich, and R. M. Reynolds. 2002. A test of evolutionary theories of aging. Proc. Natl. Acad. Sci. USA 99:14286–14291.

Käär, P., J. Jokela, T. Helle, and I. Kojola. 1996. Direct and correlative phenotypic selection on life-history traits in three pre-industrial human populations. Proc. R. Soc. Lond. B. 263:1475–1480.

Kaplan, H., K. Hill, J. Lacaster, and A. M. Hurtado. 2000. A theory of human life history evolution: diet, intelligence, and longevity. Evol. Anthropol. 9:156–185.

Kirk, K. M., S. P. Blomberg, D. L. Duffy, A. C. Heath, I. P. F. Owens, and N. G. Martin. 2001. Natural selection and quantitative genetics of life-history traits in western women: a twin study. Evolution 55:423–435.

Kirkpatrick, M., D. Lofsvold, and M. Bulmer. 1990. Analysis of the inheritance, selection and evolution of growth trajectories. Genetics 124:979–993.

Kirkwood, T. B. L., and S. N. Austad. 2000. Why do we age? Nature 408:233–238.

Knott, S. A., R. M. Sibly, R. H. Smith, and H. Møller. 1995. Maximum likelihood estimation of genetic parameters in life-history studies using the 'animal model.' Funct. Ecol. 9:122–126.

Kohler, H. P., J. L. Rodgers, W. B. Miller, A. Skytthe, and K. Christensen. 2006. Bio-social determinants of fertility. Int. J. Androl. 29:46–53.

Kojonen, E. (ed.) 1971. Sursillin suku. Genealogia Sursilliana. Weiling & Göös, Tapiola.

Kruuk, L. E. B. 2004. Estimating genetic parameters in wild populations using the 'animal model'. Phil. Trans. R. Soc. Lond. B 259:873–890.

Lahdenperä, M., V. Lummaa, S. Helle, M. Tremblay, and A. F. Russell. 2004. Fitness benefits of prolonged post-reproductive lifespan in women. Nature 428:178–181.

Lahdenperä, M., A. F. Russell, and V. Lummaa. 2007. Selection for long lifespan in men: benefits of grandfathering? Proc. R. Soc. Lond. B. 274:2437–2444.

Lummaa, V. 2007. Life-history theory, reproduction and longevity in humans. *in* L. Barrett and R. I. M Dunbar, eds. Oxford handbook of evolutionary psychology. Oxford Univ. Press, Oxford.

Lummaa, V., and T. Clutton-Brock. 2002. Early development, survival and reproduction in humans. Trends Ecol. Evol. 17:141–147.

Lynch, M., and B. Walsh. 1998. Genetics and analysis of quantitative traits. Sinauer Associates, Inc, Sunderland, MA.

Mace, R. 2000. Evolutionary ecology of human life history. Anim. Behav. 59:1–10.

McGraw, J. B., and H. Caswell. 1996. Estimation of individual fitness from life history data. Am. Nat. 147:47–64.

Medawar, P. B. 1952. An unsolved problem in biology. H. K. Lewis & Co, London.

Mousseau, T. A., and C. W. Fox. 1998. Maternal effects as adaptation. Trends Ecol. Evol. 13;403–407.

Meyer, K., and W. G. Hill. 1997. Estimation of genetic and phenotypic covariance functions for longitudinal or 'repeated' records by restricted maximum likelihood. Livest. Prod. Sci. 47:185–200.

Nussey, D. H., A. J. Wilson, A. Donald, J. M. Pemberton, T. H. Clutton-Brock, and L. E. B. Kruuk. 2008. Testing for genetic trade-offs between early- and late-life reproduction in a wild red deer population. Proc. R. Soc. Lond. B. 275:745–750.

Pettay, J. E., L. E. B. Kruuk, J. Jokela, and V. Lummaa. 2005. Heritability and genetic constraints of life-history trait evolution in pre-industrial humans. Proc. Natl. Acad. Sci. USA 102:2838–2843.

Pettay, J. E., S. Helle, J. Jokela, and V. Lummaa. 2007. Natural selection on female life-history traits in relation to socio-economic class in pre-industrial human populations. PLoS One, 2:e606.

Price, T. D., and P. R. Grant. 1985. The evolution of ontogeny in Darwin's finches: a quantitative genetic approach. Am. Nat. 125:169–188.

Réale, D., M. Festa-Bianchet, and J. T. Jorgenson. 1999. Heritability of body mass varies with age and season in wild bighorn sheep. Heredity 83:526–532.

Rose, M. R. 1991. Evolutionary biology of aging. Oxford Univ. Press, New York.

Sear, R., R. Mace, and I. A. McGregor. 2003. The effects of kin on female fertility in rural Gambia. Evol. Hum. Behav. 24:25–42.

Shaw, F. H., D. E. L. Promislow, M. Tatar, K. A. Hughes, and C. J. Geyer. 1999. Toward reconciling inferences concerning genetic variation in senescence in *Drosophila melanogaster*. Genetics 152:553–566.

Sievert, L. L. 2001. Aging and reproductive senescence. *in* P. T. Ellison, (ed.) Reproductive ecology and human evolution. Aldine de Gruiter, New York.

Snoke, M. S., and D. E. L. Promislow. 2003. Quantitative genetic test of recent senescence theory: age-specific mortality and male fertility in *Drosophila melanogaster*. Heredity 91:546–556.

Soininen, A. M. 1974. Old traditional agriculture in Finland in the 18th and 19th centuries. Forssan Kirjapaino Oy, Forssa.

Stearns, S. C. 1989. Trade-offs in life-history evolution. Funct. Ecol. 3:259–268.

Sundin, J. 1992. Legal prosecution of extramarital sex in preindustrial Sweden. Soc. Sci. Hist. 16:99–128.

Tatar, M., D. E. I. Promislow, A. A. Khazaeli, and J. W. Curtsinger. 1996. Age-specific patterns of genetic variance in *Drosophila melanogaster*. 2. Fecundity and its genetic covariance with age-specific mortality. Genetics 143:849–858.

Vangel, M. G. 1996. Confidence intervals for a normal coefficient of variation. Am. Stat. 50:21–26.

Voland, E. 1990. Differential reproductive success within the Krummhörn population (Germany, 18th and 19th centuries). Behav. Ecol. Sociobiol. 26:65–72.

———. 1998. Evolutionary ecology of human reproduction. Annu. Rev. Anthropol. 27:347–374.

Voland, E., and J. Beise. 2002. Opposite effects of maternal and paternal grandmothers on infant survival in historical Krummhörn. Behav. Ecol. Sociobiol. 52:435–443.

Williams, G. C. 1957. Pleiotropy, natural selection, and the evolution of senescence. Evolution 11:398–411.

Wilson, A. J., L. E. B. Kruuk, and D. W. Coltman. 2005. Ontogenetic patterns in heritable variation for body size: using random regression models in a wild ungulate population. Am. Nat. 166:E177–E192.

Wilson, A. J., D. H. Nussey, J. M. Pemberton, J. G. Pilkington, A. Morris, F. Pelletier, T. H. Clutton-Brock, and L. E. B. Kruuk. 2007. Evidence for a genetic basis of ageing in two wild vertebrate populations Curr. Biol. 17:2136–2142.

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