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Reproduction and longevity among the British peerage: the effect of frailty and health selection

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Whether a cost of reproduction exists among humans is still questionable. A major study of aristocratic British families finds a significant positive correlation between parity and late-life mortality, which indicates a trade-off between reproduction and longevity. This result is supported by four other studies, while earlier studies have not found a relationship or came to the opposite conclusion. We show that in natural fertility populations the relationship between fertility and late-life mortality cannot be studied correctly without considering the effects of differences in health and of mortality selection during childbearing ages because these two effects lead to a dampening of the true relationship. If these effects are controlled in Hollingsworth's genealogy of the British peerage a significant trade-off between reproduction and longevity exists for females but not for males.

Keywords: reproduction; longevity; fertility; old-age mortality; frailty

1. INTRODUCTION

A leading biological theory of the evolution of senescence stresses that resources have to be directed either towards somatic maintenance or towards reproduction (Kirkwood 1977; Kirkwood & Rose 1991). Since natural selection places priority on maximizing reproduction rather than on maximizing longevity, organisms may 'trade' a long lifespan for enhanced reproduction. Experiments have indicated that such trade-offs between reproduction and longevity exist in non-human species (Partridge & Barton 1993; Carey *et al.* 1998; Chen *et al.* 2001).

If a cost of reproduction exists among humans then it should be strongest in a natural fertility population where fertility and mortality are high. In the past, however, results for historical populations have been ambiguous. Two previous studies find a negative relationship (Voland & Engel 1986; Müller *et al.* 2002); four studies, a positive (Westendorp & Kirkwood 1998; Korpelainen 2000; Lycett *et al.* 2000; Smith *et al.* 2003). A recent and problematic study of the Finnish Sami population finds a positive phenotypic correlation between the number of sons who survive to adulthood and late-life mortality, and a negative for the number of surviving daughters (Helle *et al.* 2002). This result was partly confirmed by Beise & Voland (2002). A third series of studies found no relationship (Henry 1956; Gautier & Henry 1958; Le Bourg *et al.* 1993; J. Oeppen, personal communication) who studied both the inhabitants of German villages (Knodel 1988) and English parish data (Wrigley *et al.* 1997). All studies of contemporary populations with controlled fertility find a significant positive relationship between parity and late-life mortality (Kitagawa & Hauser 1973; Beral 1985; Green *et al.* 1988; Lund *et al.* 1990; Kvale *et al.* 1994; Friedlander 1996; Doblhammer 2000). A recent article that reviews the evidence for energy trade-offs between somatic and reproductive functioning in a range of human societies cites eight articles that report a positive relationship or no relationship between maternal energy reserves and parity, and eight studies which find a negative relationship (Tracer 2002).

Researchers have suggested that unobserved demographic factors such as marriage duration and husband's age significantly confound the observed relationship (Gavrilov & Gavrilova 1999). We provide evidence that one main unobserved factor that disturbs the observed relationship between parity and late-life mortality is difference in health (frailty). Two possible pathways exist concerning how health affects the relationship between reproduction and longevity. One is that both parity and late-life mortality depend on health; the second, that mortality selection during reproductive years depends on parity. Historical demographers define a 'natural fertility' population as one where fertility behaviour is not parity-specific. The main determinant of fertility is health rather than conscious social factors. Thus, the true relationship between parity and later-life mortality is always confounded by health, which is unobserved. A typical woman, but with poor health, will be 'selected' into lower parity, whereas a similar, but robust, woman will probably exhibit higher parity. With the frail concentrated at low parities raising mortality, and the robust at high ones lowering it, we may observe clockwise rotation and a dampening of the true positive relationship between parity and late-life mortality. At the extreme, it is possible that the phenotypic correlation will even be negative.

In historical populations, the analysis of the relationship between fertility and mortality late in life is confined to a highly selected group of women: those who survive to old age. The parish register studies of Wrigley *et al.* (1997) for England from 1580 to 1837 show that only 50–70% of women survived from age 20 to age 50. Thus, it was often the case that death occurred long before the end of a woman's reproductive phase. In these populations, the main cost of reproduction may have lain in the risks of

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pregnancy and childbirth during the pre-menopausal phase of life rather than in accumulated negative effects on old age. Mortality selection due to maternal mortality during childbearing ages may also result in a dampening of the true relationship at a later age. Suppose that frail women who had more children than their susceptibility would permit, die during childbearing ages. Then, after age 50, among the frail women only those who had few children would have survived while the strong women would be concentrated among the high parity women.

None of the existing studies has addressed the problem that health affects both mortality and parity, and that mortality selection during childbearing ages depends on parity. We account for the unobservable effects of health on parity and late-life mortality by specifying a simultaneous equation model that treats parity as an endogenous variable. This approach was developed and widely used by Lillard and colleagues (Lillard 1993; Lillard & Waite 1993; Lillard *et al.* 1995; Lillard & Panis 1996).

We demonstrate the effect of health by using what is probably the most accurate and most complete genealogy available today—Hollingsworth's study of the British peerage. We show the completeness of this genealogy by comparing it with the Bloore genealogy of the British peerage, which was used in an earlier major study (Westendorp & Kirkwood 1998) about cost of reproduction among humans.

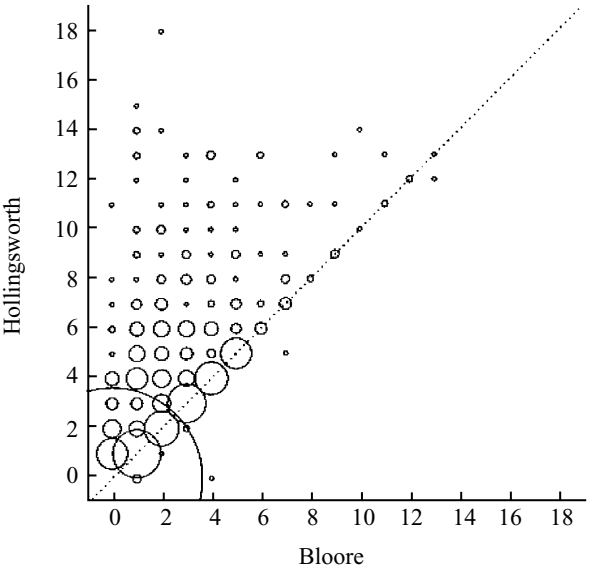

Figure 1. Comparison of the number of children born to Dukes and Marquesses in the Hollingsworth and Bloore genealogies of the British peerage. The comparison of the number of children of 694 Dukes and Marquesses that are included in both the Hollingsworth and the Bloore genealogy of the British peerage shows the incompleteness of the Bloore genealogy. It is based on the titles and the names of the peers. The circles on the diagonal indicate those peers with the same number of children in both genealogies. All circles above the diagonal refer to peers for whom Hollingsworth recorded more children than Bloore. The diameters of the circles are proportionate to the number of peers.

2. MATERIAL AND METHODS

Hollingsworth computerized the genealogies of the British peerage from 1603 to 1959 (Hollingsworth 1962, 1965). All 30 000 forms have been re-entered by the Cambridge Group for the History of Population and Social Structure. The details are contained in the original publications, but some features should be emphasized with regard to bias, accuracy and completeness. The family trees are descendant from founders, rather than ascendant from survivors, which eliminates the bias towards reproductive success. The rules for inclusion are precisely defined and follow the line of succession, including all known legitimate descendants down to the 15th birthday of the grandchildren of peers, even if they died young, or failed to marry, or have children. All known events were dated, even if the date had to be imputed, but Hollingsworth attached a code that indicated the imprecision. These codes range from zero days to ± 32 years. However, it is in the nature of genealogies of male succession that some children who died young, and particularly girls, may have been unrecorded. Hollingsworth thought that the data were relatively accurate and complete from 1750 onwards. The present study is based on all peers (1854 females, 2202 males) with only one marriage and with a maximum uncertainty in birth and death dates of up to 1 year.

The final model excludes childless marriages and marriages with only one child and draws from the records of the 3078 peers who had at least two children. Restricting the analysis to first marriages guarantees that the exposure time of the risk of becoming pregnant or becoming a father is correctly measured. The design of the data does not permit the calculation of the number of years at risk for all marriages of spouses not born in the peerage. To confine the analysis to a largely natural fertility population, birth cohorts up to 1850 are included. In this set, the first female peer is born in 1641, the first male in 1636. Violent deaths are excluded when recorded. Electronic Appendix A (available on The Royal Society's Publications Web site) gives an overview of the data and contains the average/maximum number of children, the proportion childless/parity one, the proportion of births before age 20/after age 40, mean age at marriage, and the total number of observations.

The force of mortality after age 50 is modelled by equation (2.1):

$$\ln \mu_j(x, \delta) = \alpha_0 + \alpha' T_j(x) + \beta' Y_j + \delta. \quad (2.1)$$

Let $\ln \mu_j(x, \delta)$ denote the logarithm of the force of mortality at age x, $\alpha' T_j(x)$ the age-dependent baseline hazard, and $\beta' Y_j$ the observed covariates and their parameters for individual j. All our calculations are performed in aML (http://www.applied-ml.com); thus the baseline hazard is a piecewise-linear spline function where $T_j(x)$ is a vector of piecewise-linear spline transformations of age x

$$T_j(x) = \begin{pmatrix} (\min[x, \nu_1]), \\ (\max[0, \min[x - \nu_1, \nu_2 - \nu_1]]), \ldots, \\ (\max[0, \min[x - \nu_{n-1}, \nu_n - \nu_{n-1}]]), \\ (\max[0, x - \nu_n]) \end{pmatrix}, \quad (2.2)$$

with the nodes ν_n of the splines set at ages 60, 70 and 80. The vector α' of the slopes of the splined linear segments is the average percentage increase in the mortality risk over 1 year of life. Let δ be the heterogeneity component that accounts for unobserved factors such as differences in health.

The observed covariates include as categorical variables 25-year birth cohorts, a birth after age 40, and a birth before age

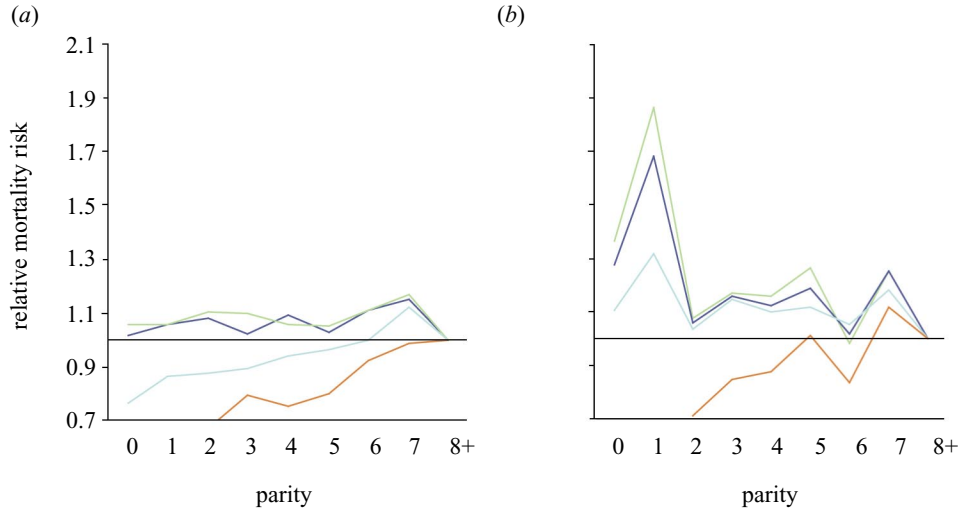

Figure 2. Relative mortality risks, by parity, for the British peerage, using different mortality models. (*a*) Among the female British peerage, parity does not influence mortality in a proportional hazard model without correction for unobserved heterogeneity (dark blue line). The model in equation (2.1), which corrects for unobserved heterogeneity (green line), yields a similar result. The model that corrects for the unobserved effect of health on parity and mortality (equations (2.1) and (2.3)) finds a positive relationship between parity and longevity. For all parities including childless women and mothers of one child, mortality increases by 1.9% ($p = 0.24$) for each additional child (light blue line). If the model is restricted to parities two and above (red line) then the increase in mortality per child is 3.8% and statistically significant at $p = 0.04$. (*b*) Among the male British peerage, childless peers and fathers of one child experience a significant excess mortality as compared to fathers of eight or more children when applying a proportional hazard model (dark blue line) or the proportional hazard model with unobserved heterogeneity in equation (2.1) (green line). This excess mortality becomes insignificant when both equations (2.1) and (2.3) are estimated simultaneously (light blue line). A positive, but statistically insignificant, relationship between parity and mortality appears when the model is restricted to parities two and higher (red line). Mortality increases by 2.7% for each additional child ($p = 0.16$).

20. In our final model, parity is included as a numerical variable after having verified that the relationship between fertility and mortality is approximately linear. We also included a tempo variable of fertility, since it is possible that a large number of births will be a proxy for the damaging effect of pace in childbearing rather than of quantity. It is defined as the number of children divided by the number of years between the first and the last child, plus one.

Differences in health and mortality selection before age 50 affect both mortality and parity; thus we treat parity as an endogenous variable. The ordered probit model in equation (2.3) estimates parity as a function of marriage cohort, marriage duration and marriage age. Consider the latent variable y_j^* where

$$y_j^* = \gamma' Z_j + \varepsilon + u_j \qquad (2.3)$$

and $y_j = i$ for $i = 2, \ldots, 8$ if $\tau_i \leqslant y_i^* \leqslant \tau_{i+1}$. Let $\tau_2 < \tau_3 < \ldots < \tau_8$ and $\tau_2 = -\infty$ and $\tau_9 = \infty$.

Let y_j^* denote the propensity of the number of children, $\gamma' Z_j$ the observed covariates and their parameters, ε the unobservable heterogeneity component and u_j a random variable which follows a standard normal distribution. Let τ_i be the thresholds of the ordered probit model, which need to be estimated.

The unobserved heterogeneity components ε and δ are assumed to follow a bivariate normal distribution with mean zero and variance–covariance matrix Σ, which consists of the standard deviations σ_δ and σ_ε and the correlation coefficient $\rho_{\delta\varepsilon}$ (equation (2.4)).

$$\Sigma = \begin{pmatrix} \sigma_\delta^2 & \rho_{\delta\varepsilon}\sigma_\delta\sigma_\varepsilon \\ \rho_{\delta\varepsilon}\sigma_\delta\sigma_\varepsilon & \sigma_\varepsilon^2 \end{pmatrix}. \qquad (2.4)$$

A non-zero correlation $\rho_{\delta\varepsilon}$ between the errors for each individual across the two equations indicates one or more unobserved variables, and the sign of the correlation shows us their aggregate effect. In particular, a negative sign indicates that an unobserved variable increases mortality and decreases parity, which is in accordance with the suggested effects of health and mortality selection before age 50.

The estimation of the model is based on the maximization of the joint likelihood function for the mortality and the fertility model.

3. RESULTS

(a) *Completeness of the Hollingsworth genealogy of the British peerage*

The number of children who die young tends to be underreported in genealogies in general, and particularly in a system concerned with survival in the male line. It cannot be assumed that underreporting and family size are independent, as large families are less likely to have missing births. If there is a positive relationship between parity and late-life survival, the slope will be biased towards zero by differential underreporting.

We compared 694 families that are included in both the Hollingsworth and the Bloore genealogy used by Westendorp and Kirkwood. Bloore knew that children were under-recorded in his work and we find that for a large proportion of these families Hollingsworth records considerably more children (figure 1), which explains the unexpectedly low figure of 2.14 (Westendorp & Kirkwood 1998) for the average number of children in the Bloore genealogy as compared to 4.8 in Hollingsworth's data.

Table 1. Results from the simultaneous estimation of the mortality and fertility model defined in equations (2.1) and (2.3).

	female peers		male peers	
	parameter estimates	*p*-value	parameter estimates	*p*-value
mortality: proportional hazard model (equation (2.1))				
age (baseline hazard)				
50–60	0.085	0.00	0.066	0.00
60–70	0.129	0.00	0.110	0.00
70–80	0.132	0.00	0.117	0.00
80+	0.199	0.00	0.171	0.00
constant	−6.034	0.00	−4.817	0.00
parity	0.038	0.04	0.027	0.16
birth cohort				
≦ 1650	1.141	0.00	0.636	0.00
1651–1675	0.573	0.09	0.550	0.03
1676–1700	1.002	0.00	0.792	0.00
1701–1725	1.058	0.00	0.722	0.00
1726–1750	0.491	0.03	0.481	0.00
1751–1775	0.618	0.00	0.182	0.16
1776–1800	0.459	0.00	0.199	0.09
1801–1825	0.313	0.01	0.183	0.06
1826–1850 (RG)				
origin of title				
Irish	0.234	0.03	0.034	0.68
Scottish	−0.029	0.81	−0.062	0.60
British (RG)				
birth after age 40	0.063	0.52	−0.088	0.28
birth before age 20	0.215	0.11	−0.034	0.91
tempo	0.341	0.17	0.093	0.62
fertility: ordered probit model (equation (2.3))				
thresholds				
T2	−1.695	0.00	1.303	0.15
T3	−0.968	0.08	2.182	0.02
T4	−0.207	0.70	2.875	0.00
T5	0.355	0.51	3.444	0.00
T6	0.865	0.11	3.891	0.00
T7	1.221	0.03	4.335	0.00
marriage duration	0.067	0.00	0.097	0.00
marriage cohort				
≦ 1675	0.225	0.30	0.555	0.00
1676–1700	0.124	0.65	0.940	0.00
1701–1725	0.594	0.02	0.259	0.27
1726–1750	−0.453	0.05	0.030	0.88
1751–1775	0.304	0.15	0.699	0.00
1776–1800	0.471	0.00	0.887	0.00
1801–1825	0.485	0.00	0.828	0.00
1826–1850	0.416	0.00	0.551	0.00
≧ 1850 (RG)				
marriage age	−0.060	0.00	0.040	0.03
variance covariance matrix				
σ_δ^2	1.108	0.00	0.808	0.00
σ_ε^2	1.187	0.00	1.348	0.00
$\rho_{\delta\varepsilon}$	−0.227	0.00	−0.198	0.03
log likelihood	−13 930.410		−17 108.920	

Despite the doubling of the number of children when compared with Bloore, Hollingsworth himself concluded that a significant number of children who died young were not recorded, especially before 1750 (Hollingsworth 1962, 1965).

(**b**) *Unobserved differences in health*

Table 1 shows that the estimated correlation coefficients $\rho_{\delta\varepsilon}$ of the variance–covariance matrix of the random factors are significant for both sexes. The negative value of −0.23 ($p = 0.001$) for females indicates that health influences both fertility and late-life mortality. For men, unobserved behavioural factors may produce the significant negative correlation of −0.20 ($p = 0.03$). Unpublished research suggests that marriage can be protective as well as selective in lowering male mortality for the British peerage. Thus, a smaller family may be associated with absence from the family home and suggest higher

exposure to morbidity in military service, from foreign travel, urban life and sexually transmitted diseases.

(c) *The relationship between reproduction and late-life mortality*

Figure 2*a* and *b* shows how the effect of parity on mortality after age 50 depends on the model specification. Neither the simple proportional hazard model nor the hazard model with unobserved heterogeneity, as defined in equation (2.1), yields a relationship between parity and mortality for females, but there is a strong and significant negative relationship for males. The full model (equations (2.1) and (2.3)) finds a significant positive relationship for females and a non-significant positive relationship for males. Among females, mortality is 33% lower for parity two ($p = 0.084$) and 29% lower for parity four ($p = 0.064$) than for women with eight or more children (results based on the specification of parity as indicator variables, and not shown). The slope of the increase in the force of mortality per child is 3.8% and it is significant at a level of $p = 0.04$ (table 1). Similar to Westendorp and Kirkwood we find a tendency that the 29% of female peers who gave birth before their 20th birthday experience an increase in late-life mortality of 22% ($p = 0.11$). We do not find a mortality advantage for late mothers (at least one birth after age 40). In none of the models were the variables related to fertility significant for males at a conventional significance level of 0.05.

In the parity part of our model (equation (2.3)) we find, for both sexes, a significant effect of marriage duration and marriage age on the number of children, with a long marriage duration and a low marriage age resulting in more children as expected for females (for males low marriage age results in significantly less children). When we consider the marriage age of the spouse in the female model (results not shown) marriage age of the woman loses significance while all other parameter values remain unchanged. While it seems surprising that male marriage age would be a more important determinant of female fertility than female marriage age, one has to keep in mind that marriage ages of spouses are highly correlated which causes collinearity in the independent variables and may thus lead to biased results.

4. DISCUSSION

The Hollingsworth data form a rare example of a descendant genealogy, specifically constructed for demographic research and following a precisely defined set of rules for inclusion and exclusion. Although based on a male succession, it aims to record the full demographic histories of peers and all their legitimate children. Our comparison with the Bloore genealogy demonstrates the relative completeness of the Hollingsworth genealogy.

Our analysis is restricted to first marriages only. Conditioned on survival to age 50, 23% of men and 12% of women married more than once, which implies that remarriage among the peerage was less common than in the general population. The reason for this difference is probably economic: in the general population women needed an income, widows were economically attractive if they inherited their husband's businesses, and both men and women needed support for children. Furthermore, some occupations would have been very difficult without a wife, for example, farming. There is almost no marital dissolution. We decided to exclude all peers with higher-order marriages to avoid confounding by selection into new marriages. Remarriage is possibly based on the fertility outcomes of previous marriages; for example, peers are less likely to remarry when male children are present from the first marriage. Remarriage also depends on age because peers are more likely to remarry when they are young and it is therefore confounded with parity. Restricting the analysis to first marriages assures a greater homogeneity of the peers in terms of their lifestyle and their fertility biography.

Our final models exclude childless peers and parents of one child because confounding factors specific to genealogies at low parities seem to affect the observed relationship between parity and longevity. A recording system concerned with male primogeniture is likely to have paid less attention to females and non-heir males, especially if they have not contributed significantly to the future survival of their line. The children of daughters and younger sons may be of little dynastic interest. This is particularly true if the children die young and have little impact on the historical record. In other words, the lower the parity the higher the likelihood that an individual is omitted from the genealogy or that the parity was under-recorded. A second problem arises if parity cannot be regarded as representing the full costs of childbearing. Conventional definitions of parity count live births only. Foetal losses, abortions and stillbirths are not included. Thus, we believe that under-recording of child costs may also be associated with health and cannot be regarded as random. It is unlikely that women with very high parity have suffered many miscarriages and stillbirths or that their parity has been under-recorded. Low parity women, under natural fertility conditions, are more likely to have borne higher fertility costs than their recorded number of live births reveals, leading to an underestimate of the slope of the true relationship. This is supported by our finding that the strength of the relationship between parity and late-life mortality increases if the model is restricted to higher parities only.

It is possible that the effect of reproduction on female late-life mortality may have been even stronger in the general historic population. Peers' nutrition may have been poor by modern standards, but they would not have been subject to caloric stress and may have had other physical advantages related to lifestyle. Although fashions changed, it is likely that a significant proportion of peerage women avoided the full physiological cost of breastfeeding, either through wet-nursing before 1800 or through bottle-feeding later. Wet-nursing among the upper classes in Britain peaked in the seventeenth and early eighteenth centuries, although it was never as widespread as in parts of Europe. Although not quantifiable, there was an upper-class movement towards maternal breastfeeding from the middle of the eighteenth century (Fildes 1988; Matthews Grieco 1991).

We do not find a beneficial effect of a late birth. Restricting the group of late mothers to the 5% who had a child after age 45 does not change the result. This finding is contrary to earlier results from female contemporary populations (Perls *et al.* 1997) and from three historical

populations (Voland & Engel 1986; Müller *et al.* 2002; Smith *et al.* 2003). In contemporary populations, late mothers are a small, socially selected group. In 1981, only *ca*. 7% of Austrian women aged over 50 had a child after their 40th birthday and in 1971 in England and Wales only 8% (Doblhammer 2000). Among the female peerage late mothers were much more frequent: 32% of the women gave birth in their 40s. The social factors that caused contemporary women to have a child comparatively late may also positively influence their late-life mortality. Thus, giving birth after age 40 may not necessarily be an indicator of slower biological ageing (Perls *et al.* 1997).

In historical populations, non-biological factors such as income or education may have created social and economic reproductive costs. However, many of these factors do not apply to the peerage because they are above some kind of economic threshold. In the peerage, the relationship between fertility and late-life mortality may primarily result from the accumulated physiological costs of repeated pregnancies or childbirths or it may stem from a genetic disposition towards reproduction at the cost of longevity (Williams 1957). Westendorp *et al.* (2001) report genetic evidence that a stronger immune response to infection among women may be associated with a lower probability that pregnancy will proceed. This mechanism for the trade-off between fertility and survival allows a different interpretation of our model and results. Reproductive success may not only have a direct cost of higher mortality, but could also be a proxy for characteristics of a woman's immune system.

In our data, we find a similar trend for both sexes but it is non-significant for males. Among males, the energetic costs of producing and raising offspring are small and the evolutionary costs in terms of survival may rather have lain in deleterious courtship behaviour (Partridge & Farquhar 1981; Cordts & Partridge 1996). For men the cost of reproduction is associated with a high testosterone level, which reduces the immune system and makes them more susceptible to infectious disease (Campbell *et al.* 2001). By contrast, a recent study shows that marriage, fatherhood, and longer periods spent with wives and children are all linked to lower testosterone levels (Gray *et al.* 2002). In addition to the results by Westendorp *et al.* (2001) concerning women's immune systems, our sex-specific finding is supported by another study. Christensen *et al.* (1998) reveal that the number of remaining teeth in old age is negatively correlated with parity among women but not among men.

After statistical correction for the effects of differences in health and of mortality selection before age 50, our data suggest a strong and significant positive correlation between parity and late-life mortality for peerage women. It is possible that the effect may have been even stronger outside this elite group, and if better proxies of the true costs of fertility could be defined.

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