Culled males, infant mortality and reproductive success in historical Finland

Tim A. Bruckner[1]

Samuli Helle[2]

Elisabeth Bolund[3]

Virpi Lummaa[4]

[1] Public Health & Planning, Policy and Design
University of California, Irvine, 202 Social Ecology I, Irvine, CA 92697, USA
Email: tim.bruckner@uci.edu

[2] Section of Ecology, Department of Biology
University of Turku, Finland

[3] Evolutionary Biology Centre
Uppsala University, 752 36 Uppsala, Sweden

[4] Department of Animal and Plant Sciences
University of Sheffield, S10 2TN Sheffield, UK

ABSTRACT

Theory asserts that the sex ratio (i.e., M/F) at birth gauges selection *in utero* and cohort quality of surviving males. We report the first individual-level test in humans of the "culled cohort" hypothesis that males born to low sex ratio cohorts show lower than expected infant mortality but greater than expected reproductive success. We examined a unique multigenerational dataset in 19th century Finland (n=7,824 males). A one standard deviation decline in the cohort sex ratio precedes an eight percent decrease in male infant mortality. Males born to lower cohort sex ratios also successfully raised four percent more offspring to reproductive age than did males born to higher cohort sex ratios, but the offspring finding falls just outside conventional statistical significance. Whereas the sex ratio gauges selection *in utero* and predicts male infant mortality, the reproductive success findings provide weak support for an evolutionarily adaptive explanation of male culling *in utero*.

INTRODUCTION

In humans, mean male cohort lifespan falls below female lifespan in all societies for which we have reliable data [1]. Historical mortality series, moreover, show greater risk of male relative to female mortality across virtually all age groups. This persistent and widespread male disadvantage has generated much research to identify mechanisms, and test underlying theories, regarding its causes [2]. Less research, however, examines male frailty *in utero* despite its potential to shape variation in human life history traits including mortality and reproduction.

In humans, an estimated thirty to seventy percent of conceptions do not survive to birth [3]. Male fetal losses outnumber female losses by nine to twenty percent [4]. Recent work indicates that the secondary sex ratio (i.e., the ratio of male to female live births; hereafter referred to as the sex ratio) may gauge important variation over time in male frailty *in utero*. The sex ratio falls in populations encountering ambient shocks including cold, earthquakes, and natural and man-made disasters [5 - 8]. In addition, male fetal deaths rise, and the sex ratio falls, following ambient economic downturns and stressful events such as the terrorist attacks of September 11, 2001 [9, 10]. These results indicate that ambient stressors raise the risk of fetal death more among male than female gestations and result in a lower than expected birth sex ratio.

Aggregate-level analyses of males born to these low sex ratio cohorts report lower mortality rates in infancy than that of males born to high sex ratio cohorts [11, 12, 13; but also see 6]. Researchers infer from these results that low birth sex ratios may reflect greater than expected “culling” of frail males *in utero*.

This culling would thereby result in a relatively robust cohort of males that survive to birth.

One popular but controversial argument for selective male loss following ambient perturbations involves the conservation of adaptive maternal mechanisms that spontaneously abort less fit fetuses [14 - 17]. Maternal mechanisms presumably assess the fitness of a fetus and environmental challenges to its survival if carried to birth [16]. These mechanisms would terminate gestations that, if continued to birth in the environments humans occupied through most of our history, would have yielded an infant with relatively low likelihood of surviving to reproductive age and yielding offspring [18]. According to theory, if ambient stressors raise the risk of death more among male than female infants and children, then aborting these males *in utero* would increase the woman's overall yield of offspring because frail sons produce fewer offspring than do frail daughters. Natural selection would favor any mechanism that enabled a woman to abort a frail male fetus if it allowed the mother either to invest more in existing children or to conceive future offspring with greater lifetime reproductive success than the frail son [18].

The argument that male fetal loss and sex ratio variation reflects a potentially adaptive response to stressors would enjoy empirical support if males born to the most "culled" (i.e., low sex ratio) cohorts showed greater number of offspring surviving to adulthood than do males born to the least culled cohorts. The literature reports no such test of lifetime reproductive success (LRS). We presume this circumstance arises from the rarity of the requisite historical, multigenerational data in humans. We address this gap in the literature and

examine high-quality, individual-level life history data from a pre-industrial population in Finland.

We test two hypotheses implied by culled cohorts. First, we test whether the risk of male mortality rises with the cohort sex ratio at birth, implying that males from culled (i.e., low-sex ratio) cohorts appear relatively more robust. Second, we test whether LRS rises above expected values among males born to the most culled (i.e., low sex ratio) cohorts, implying that such males would also enjoy relatively improved reproductive success due to their higher quality. We focus our test on males given that the culled cohort hypothesis makes no prediction about females [12].

In addition to providing the first test of sex ratio variation and LRS among humans, we improve upon earlier work on age-specific mortality by examining a historical, natural fertility population that more closely approximates the evolution of human life histories than do post-industrial societies [10, 11]. Our integrated time-series and structural equation methods also advance earlier work by using rich individual-level information contained in the Finland dataset to rule out a key rival that spurious findings arise due to shared temporal patterns in the sex ratio, cohort mortality and reproductive success.

METHODS

Variables and Data

We retrieved birth, reproductive success, and death data from historical church records in Finland. Since the 17th century in Finland, the Lutheran Church

required the submission of accurate registers of all births, inter-parish movements, marriages and deaths in the country. From these records, we compiled life histories for individuals from rural parishes previously used in analyses of life-history traits [19]. Careful documentation of the Church registries allows for linkage of data between mothers and their children. The dataset includes information on birth characteristics (including date of birth), social class, exact age at death, age at marriage, number of live (and stillborn) offspring and age of death of offspring. A detailed description of the quality and provenance of the historical data appears elsewhere (http://www.huli.group.shef.ac.uk/study-pop.html). We restricted our study sample only to males with non-missing values for all analytic variables (described below). We also excluded twins based on their fundamentally different life histories from that of singletons [20].

To derive our independent variable, we gained access to data from five parishes that contained a sufficiently large number of births per year to permit stable annual sex ratio estimates. These data included two archipelagic (Kustavi and Hiittinen), two mainland (Ikaalinen and Tyrvää) and one Northern (Pulkkila) parish. Consistent with the literature, we used the sex ratio for each annual birth cohort as our independent variable [11]. Figure 1 plots the annual sex ratio of the study population. The mean sex ratio was 1.06 (range: 0.80 to 1.37).

Given our interest in a cohort whose fertility and mortality schedules did not experience the industrial transition, we restricted our analytic sample to males born from 1790 to 1870. This restriction resulted in a study sample of 7,824 Finnish males. During the study period, the population depended on small-scale farming and supplemented their food supply by fishing. This preindustrial

period in Finland shows high birth and death rates. For example, mean male lifespan was 29.1 years for this population, and over 17 percent of males died in infancy (Table 1). Significant improvements in the standard of living, as well as the demographic transition, occurred primarily during the 20th century in Finland [21]. Almost all women finished reproduction by age 45 and few gave birth out of wedlock [22].

Previous literature on sex ratios and male lifespan indicates distinct age-specific mortality responses [11]. We therefore analyzed the relation between birth cohort sex ratio and male mortality by distinct age groups. We analyzed male mortality in infancy (before 1 year of age), childhood (ages 1–4), youth (ages 5–14), and over peak reproductive ages (15–50 years). Consistent with Hamilton's logic [23] in which the force of natural selection declines monotically with age as well as recent empirical findings in historical Finland [19], we hypothesized that any relation between the sex ratio and male mortality would appear strongest in infancy and decline thereafter.

We used as the LRS outcome the father's total number of children that were successfully raised to 15 years of age. We chose this measure over other indicators of fecundity (e.g., total number of live births) since high background infant and child mortality during the 18th and 19th centuries indicates that total number of children may not approximate LRS [24]. Total number of children surviving to 15 years also correlates strongly with the long-term contribution to the future population gene pool [25]. Previous research on the Finnish population, moreover, uses this measure of LRS [26].

Approach

Mortality

For all mortality analyses, we employed a regression approach that models the odds of male death within a specific age interval (died before end of interval=1; survived to end of interval=0) and uses a “logit” link function for the binary outcome. Only individuals alive at the beginning of the age interval qualify for inclusion in that age-specific test. We controlled for many individual-level variables that could confound the relation between the annual cohort sex ratio and male mortality. These factors included the following: month of birth, interbirth interval, whether a first born son and family socioeconomic status at birth (i.e., “wealthy” farm owners and merchants; “middle class” craftsmen and tenant farmers, and “poor” crofters and laborers). We also controlled for region of birth (archipelago, northern region, or mainland region) given the different background levels of mortality across the rural parishes [20].

Although the demographic transition arrived in Finland in the 20th century, initial gains in cohort lifespan may have begun in Finland before 1870. To the extent that annual sex ratios and male mortality may share secular patterns (including trend), this pattern could lead to spurious associations. To control for these secular patterns, we applied autoregressive, integrated, moving average (ARIMA) time series routines to annual aggregate values of the risk of age-specific male mortality over our test period [27]. This purely empirical approach, outlined by Box, Jenkins and Reinsel [27] and employed frequently in biodemography [5, 11, 28], identifies autocorrelation in the time series. We used this identification strategy to arrive at annual, best-fitted values of male mortality rates. This time propensity essentially gauges the predicted value, conditional on

time, of age-specific mortality based solely on secular patterns from 1790-1870. After yielding a time-propensity of male mortality for each birth year, we assigned these fitted values back to each individual and used it as a covariate in the logistic regression.

LRS

We did not have access to reproductive histories after 1900 from Finnish Lutheran church records. This circumstance led to a large amount of missing data on offspring yield among our cohorts born after 1850. We, therefore, restricted the LRS analysis to cohorts born from 1790 to 1850. This process yielded a sample of 3,503 males with non-missing information on the analytic variables (described below).

We included all covariates as in the mortality analyses with the addition of total annual cohort size. According to Easterlin's hypothesis, relatively small annual birth cohorts may yield greater than average marital success and fertility given the expectation of relatively greater economic prospects in adulthood [29]. This favorable expectation arises from less within-cohort competition for employment and goods at adult ages. We, therefore, included total cohort size as a control variable. As with the mortality analyses, we used ARIMA time-series routines to arrive at fitted, time-propensity value for offspring that survived to 15 years. We then included this time-propensity value as a control variable in the individual-level analyses to ensure that secular trends in LRS do not confound our tests.

We used structural equation modeling (SEM) to test the relation between cohort birth sex ratio and offspring surviving to 15 years. The Finnish data include information on marital status and number of children born. These variables may serve as successive "bottlenecks" for our LRS measure in that, in the Lutheran Church culture in Finland during this period, marriage correlates positively with the likelihood of bearing children, which in turn correlates positively with the number of children that survive to 15 years. Because both the numbers of live offspring, and offspring surviving to 15 years, had a large excess of zeroes (58% and 62%, respectively), we fitted these variables in SEM using a zero-inflated Poisson distribution. In addition, we included in the model the residual covariances of marital status, live offspring, and offspring surviving to 15 years, as well as the residual covariances from all other independent variables in the model. The inclusion of the full covariance matrix accounts for the within-individual correlation of this set of variables when estimating LRS.

We further used marital status (1=married; 0= never married) to predict the zero-inflation component of number of kids and live offspring (1= had kids; 0 = no kids) to predict the zero-inflation component of LRS. Inclusion of these variables in the SEM assists with predicting the likelihood of having zero children that survive to age 15 years, given that most men yielded offspring only if they first married. In addition to the LRS outcome, we explored whether the cohort sex ratio predicted marital status and, separately, number of live offspring. We reasoned that the coefficients of the sex ratio on marital status, and number of live offspring, could suggest potential pathways by which the sex ratio ultimately affects LRS. We used robust maximum likelihood estimation with Monte-Carlo

integration (5,000 integration points) that allows for non-normal data and clustering of observations within mother (e.g., siblings). We performed the LRS analysis using Mplus version 7.11 [30].

RESULTS

We find that males born during years with a relatively low annual sex ratio show higher quality in terms of early survival and later reproductive output. To assist with interpretation, we standardized the sex ratio coefficient such that the mean is zero and a one unit change reflects a movement of one standard deviation (SD). First, we analyzed survival and found that, consistent with the culled cohort hypothesis, the risk of male infant mortality varies positively with the cohort sex ratio (odds ratio for a 1 SD increase: 1.085; 95% Confidence Interval [CI] = 1.02 to 1.15; see Table 2). This coefficient implies that a 1 SD decrease in the sex ratio predicts an eight percent reduction in the odds of male infant death. This relation appears confined to infancy in that we cannot reject the null for other age spans (i.e., 1 to 4 years, 5 to 14 years, and 15 to 50 years).

To assist the reader with visualizing the discovered relation, Figure 2 plots the predicted values of the probability of infant death, based on our regression model, as a function of the scaled sex ratio. The best fitting line of these data points yields the positively signed sex ratio coefficient in Table 2. At the lowest sex ratios (i.e., far left), the probability of infant death falls below the mean (i.e., 0.16). By contrast, males born in the highest sex ratio years show a probability of infant death that disproportionately lies above the mean.

As shown in Table 3, males born to higher sex ratio cohorts raise slightly fewer offspring to 15 years than do males born to lower sex ratio cohorts (coef. = -0.042, SE= 0.023, p = 0.073). Although the result falls outside conventional levels of statistical significance, the sign of the coefficient lies in the hypothesized direction of culled cohorts. The magnitude of this finding is modest in that a 1 SD decrease in the cohort sex ratio predicts a 4.2 percent increase in offspring that fathers raised to 15 years (95% CI: -0.3 to 8.7 percent). This finding offers some support that birth during a "low sex ratio" year precedes increased LRS.

Exploration of the relation between the sex ratio and marital success, and number of live offspring, could not reject the null. The sex ratio was neither related to the likelihood of marriage (coef. = 0.017, SE = 0.046, p=.72) nor to the number of offspring (coef: -0.022, SE = 0.020, p=.29). These results suggest that the slight LRS gain for males born to low sex ratio cohorts does not likely accrue via higher marriage rates or number of born offspring.

Figure 3 displays the scatter plot of the fathers' predicted number of children surviving to 15 years (from the SEM analysis) and the scaled cohort sex ratio. Several LRS values are zero given that many Finnish males did not marry and yielded no offspring. Predicted LRS at extremely high and low sex ratios shows the hypothesized inverse association in that LRS is above the mean (i.e., 2.9 children) at low sex ratios and is below the mean at high sex ratios. The pattern of results at more modest deviations of the sex ratio, however, suggests a weak, non-significant inverse association.

We performed sensitivity analyses to assess the robustness of our findings. First, we used time-series methods to inspect whether secular patterns

(e.g., trend) in the annual sex ratio might have induced a spurious relation between the sex ratio and male infant mortality and LRS. Time-series methods detected no autocorrelation in the sex ratio. Second, for LRS, we evaluated whether results appear sensitive to the choice of functional form for the dependent variable (e.g., negative binomial or zero-inflated Poisson). Inference for the sex ratio coefficient did not change. Third, for the two results that suggest rejection of the null (i.e., infant mortality and LRS), we repeated the analyses but included twins. For male infant mortality, the sex ratio coefficient became slightly stronger than the initial test (odds ratio for a 1 SD increase = 1.09; 95% CI = 1.03—1.16, $p<.01$). For total offspring surviving to age 15 years, the sex ratio coefficient remained negative but became attenuated in magnitude and statistical significance (coef. = -.038, SE = .023, $p = .105$).

DISCUSSION

The culled cohort hypothesis contends that the sex ratio at birth gauges male cohort quality. According to this argument, lower than expected cohort sex ratios reflect an excess of male fetal loss that occurs disproportionately among frail gestations [12]. Using life-history, intergenerational data from 18th and 19th century Finland, we examined whether males born to low sex ratio cohorts show greater than expected survival. We also tested whether these males show relatively greater lifetime reproductive success. Findings support the culled cohort hypothesis in that birth in a low sex ratio year predicts lower than expected male infant mortality. In addition, men from low sex ratio cohorts show a slightly greater than expected number of offspring reared to reproductive age.

This finding, however, falls outside of conventional levels of statistical significance. Taken together, results indicate that the sex ratio may sensitively gauge the quality of male cohorts that survive to birth. The relatively small LRS finding among men, however, offers only modest support that population-level sex ratio variation predicts fathers' yield of robust offspring.

Strengths of our analyses include the high quality of the multigenerational Finnish dataset. The pre-industrial time period we investigate, moreover, better approximates human mortality and reproductive schedules over much of human history than do contemporary datasets [31]. This circumstance allows for more refined tests of evolutionary hypotheses that assume high background rates of mortality. We also employ advanced methods using both time-series and individual-level control variables, which minimizes the likelihood of spurious findings.

Limitations include that we did not have complete life history information on all birth cohorts. This missing data led to smaller sample sizes when examining mortality and reproductive outcomes later in life. We used the total number of offspring reared to age 15 years as a proxy of fitness, which includes both the quantity as well as the quality of the produced offspring. Other rate-sensitive measures of reproduction (i.e. individual lambda) may more sensitively capture fitness in the Finnish population that experienced expansion over the 19th century [32]. Nevertheless, research reports that total offspring surviving to 15 years corresponds well with total individual contribution to the future gene pool [33].

Absent detailed information on fetal deaths for each mother, we inferred excess culling of frail male fetuses among low sex ratio cohorts. We acknowledge that low sex ratios may arise due to a variety of other mechanisms (e.g., those that affect sex at conception) [34]. We, however, know of no other hypothesized mechanism for sex ratio variation that would predict its inverse relation with male infant mortality and LRS.

The magnitude of our infant mortality result appears somewhat larger than those of an ecological test in historical Sweden, a country with similar background levels of mortality during this time period [11]. Findings, however, diverge from a null result in a Finnish study of sex ratios from 1865-2003 [6]. We attribute this divergence to the difference in time period used. We also caution against making direct comparisons to previous literature on culled cohorts given that we, unlike earlier work, use detailed, individual-level life history data and its attendant methodology.

We view the male infant mortality finding as important for two reasons. First, results converge with previous research which finds that individual variation in relative fitness arose largely from variation in male mortality rates before reproductive age [19]. Although we use a cohort measure of variation in male mortality, it remains plausible that a more refined individual-level indicator of male frailty could hold implications for the conservation of a maternal selection mechanism *in utero.* Second, findings extend the "heterogeneity of frailty" argument to the developmental stage in which mortality selection appears the strongest—*in utero*. Briefly, Vaupel and colleagues theorize [35, 36], and others find empirical support for [37], the notion that the compositional change of

cohorts, through the selective early mortality of its more frail members, leave behind a smaller but more robust cohort with decelerating mortality rates. If this argument pertains to pregnancy, the sex ratio may sensitively gauge male cohort morbidity well beyond infancy [38, 39].

Whereas we found only modest support for improved reproductive success among males born to low sex ratio cohorts, results remain sensitive to inclusion of twins. This pattern of findings indicates that culled males may not reflect a facultative adjustment conserved by natural selection. We note, however, that support for culled males *in utero* as an individual maternal adaptation, rather than as a by-product of male frailty in dimorphic species [17], depends on sons yielding marginal gains in reproductive success relative to that of daughters [40]. This condition typically holds in populations that exhibit high reproductive skew (e.g., polygynous societies, extensive serial monogamy) [41]. In our population, the Lutheran church precluded these conditions. These religious norms may have yielded a conservative test for the reproductive success outcome. We further note that rigorous tests of sex ratio variation as an adaptive maternal mechanism would want to account for any marginal fitness benefit of having a son and the tradeoff cost to the mother's future reproduction of having a son (including maternal mortality) [42, 43]. We expect that availability of historical life history data among populations with high reproductive skew would permit such a test.

Scant work examines the role of mortality selection *in utero* on cohort quality over the life course. We report that, in a pre-industrial, natural fertility population, the sex ratio predicts male infant mortality and, to a much lesser

extent, number of fathered offspring that survive to reproductive age. These findings strengthen the evidence base that sex ratio variation sensitively gauges culling *in utero* among male gestations. We encourage more refined tests, especially in historical populations, of the extent to which variation in selection during pregnancy precedes cohort mortality and reproductive success.

References

1. Human Mortality Database. University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Available at: www.mortality.org (Accessed on 11/5/13).

2. Maklakov, A. A. & Lummaa, V. 2014 Evolution of sex differences in lifespan and aging: causes and constraints. *Bioessays* **35**, 717-24.

3. Boklage, C. E. 1990 The survival probability of human conceptions from fertilization to term. *International Journal of Fertility* **35**, 75-94.

4. Byrne, J. & Warburton, D. 1987 Male excess among anatomically normal fetuses in spontaneous abortions. *Am J Med Genet* **26**, 605-11.

5. Catalano, R., Bruckner, T. & Smith, K. R. 2008 Ambient temperature predicts sex ratios and male longevity. *Proc Natl Acad Sci U S A* **105**, 2244-7.

6. Helle, S., Helama, S. & Lertola, K. 2009 Evolutionary ecology of human birth sex ratio under the compound influence of climate change, famine, economic crises and wars. *J Anim Ecol* **78**, 1226-33.

7. Mocarelli, P., Brambilla, P., Gerthoux, P. M., Patterson, D. G., Jr. & Needham, L. L. 1996 Change in sex ratio with exposure to dioxin. *Lancet* **348**, 409.

8. Fukuda, M., Fukuda, K., Shimizu, T. & Moller, H. 1998 Decline in sex ratio at birth after Kobe earthquake. *Hum Reprod* **13**, 2321-2.

9. Catalano, R., Bruckner, T., Anderson, E. & Gould, J. B. 2005 Fetal death sex ratios: a test of the economic stress hypothesis. *Int J Epidemiol* **34**: 944-948.

10. Bruckner, T. A., Catalano, R. & Ahern, J. 2010 Male fetal loss in the U.S. following the terrorist attacks of September 11, 2001. *BMC Public Health* **10**, 273.

11. Bruckner, T. & Catalano, R. 2007 The sex ratio and age-specific male mortality: evidence for culling in utero. *Am J Hum Biol* **19**, 763-73.

12. Catalano, R. & Bruckner, T. 2006 Secondary sex ratios and male lifespan: damaged or culled cohorts. *Proc Natl Acad Sci U S A.* **103**, 1639-43.

13. Catalano, R., Ahern, J., Bruckner, T., Anderson, E. & Saxton, K. 2009 Gender-specific selection in utero among contemporary human birth cohorts. *Paediatr Perinat Epidemiol* **23**, 273-8.

14. Stearns, S.C. 1987 The selection-arena hypothesis. In The Evolution of Sex and Its Consequence*s*. (ed. S.C. Stearns). Basel: Birkhauser Verlag.

15. Forbes, L.S. 1997 The evolutionary biology of spontaneous abortion in humans. *TREE*; **12**:446-450.

16. Wells, J. 2000 Natural Selection and Sex Differences in Morbidity and Mortality in Early Life. *Journal of Theoretical Biology* **202**, 65-76.

17. Kruuk, L. E., Clutton-Brock, T. H., Albon, S. D., Pemberton, J. M. & Guinness, F. E. 1999 Population density affects sex ratio variation in red deer. *Nature* **399**, 459-61.

18. Lummaa, V. 2001 Reproductive investment in pre-industrial humans: the consequences of offspring number, gender and survival. *Proc Biol Sci* **268**, 1977-83.

19. Courtiol, A., Pettay, J. E., Jokela, M., Rotkirch, A. & Lummaa, V. 2012 Natural and sexual selection in a monogamous historical human population. *Proc Natl Acad Sci U S A* **109**, 8044-9.

20. Lummaa, V., Jokela, J., Haukioja, E. 2001 Gender difference in benefits of twinning in pre-industrial humans: boys did not pay. *J Anim Ecol* **70**, 739–746.

21. Liu, J., Rotkirch, A. & Lummaa, V. 2012 Maternal risk of breeding failure and life-history shifts during demographic transitions in Finland. **PLOS One** 7 (4), e34898.

22. Lahdenpera, M., Russell, A. F., Tremblay, M. & Lummaa, V. 2011 Selection on menopause in two premodern human populations: no evidence for the Mother Hypothesis. *Evolution* **65**, 476-89.

23. Hamilton, W.D. 1966 The moulding of senescence by natural selection. *J Theor Biol* **12**, 12-45.

24. Gillespie, D. O., Russell, A. F. & Lummaa, V. 2008 When fecundity does not equal fitness: evidence of an offspring quantity versus quality trade-off in pre-industrial humans. *Proc Biol Sci* **275**, 713-22.

25. Brommer, J. E., Gustafsson, L., Pietiainen, H. & Merila, J. 2004 Single-generation estimates of individual fitness as proxies for long-term genetic contribution. *Am Nat* **163**, 505-17.

26. Rickard, I. J., Holopainen, J., Helama, S., Helle, S., Russell, A. F. & Lummaa, V. 2010 Food availability at birth limited reproductive success in historical humans. *Ecology* **91**, 3515-25.

27. Box, G., Jenkins, G. & Reinsel, G. 1994 *Time Series Analysis: Forecasting and Control*. 3rd edn. London: Prentice Hall.

28. Catalano, R., Ahern, J. & Bruckner, T. 2007 Estimating the Health Effects of Macrosocial Shocks: A Collaborative Approach In *Macrosocial Determinants of Population Health* (ed. S. Galea), pp. 375-397. New York: Springer

29. Easterlin, R.A. 1976 The Conflict Between Aspirations and Resources. *Population and Development Review* **2**, 417– 425.

30. Muthén, L. K., & Muthén, B. O. (1998-2013). Mplus User's Guide. Seventh Edition. Los Angeles, CA: Muthén & Muthén.

31. Partridge, L. 1997 Evolutionary Biology and Age-Related Mortality. In *Between Zeus and the Salmon: The Biodemography of Longevity*. (ed. K. W. Wachter & C. E. Finch). Washington, DC: National Academy Press.

32. McGraw, J.B. & Caswell, H. 1996 Estimation of Individual Fitness from Life-History Data. *Am Nat* **147**, 47-64.

33. Brommer, J. E., Gustafsson, L., Pietiainen, H. & Merila, J. 2004 Single-generation estimates of individual fitness as proxies for long-term genetic contribution. *Am Nat* **163**, 505-17.

34. James, W. H. 2012 Hypotheses on the stability and variation of human sex ratios at birth. *J Theor Biol* **310**, 183-6.

35. Vaupel, J. W., Manton, K. G. & Stallard, E. 1979 The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography.* **16**, 439-54.

36. Vaupel, J. W., & Zhang, Z. 2010 Attrition in heterogeneous cohorts. *Demographic Research*, **23**, 737–748.

37. Zajacova, A. & Burgard, S.A. 2013 Healthier, Wealthier, and Wiser: A Demonstration of Compositional Changes in Aging Cohorts Due to Selective Mortality. *Popul Res Policy Rev* **32,** 311–324.

38. Song, S. 2010 Mortality consequences of the 1959-1961 Great Leap Forward famine in China: Debilitation, selection, and mortality crossovers. *Soc Sci Med* **71**, 551-8.

39. Bruckner, T. A. & Nobles, J. 2013 Intrauterine stress and male cohort quality: the case of September 11, 2001. *Soc Sci Med* **76**, 107-14.

40. Roff, D.A. 2002 Trade-offs. In: *Life History Evolution*. Sunderland, Massachusetts: Sinauer Associates.

41. Pen, I. & Weissing, F.J. 2002 Optimal sex allocation: steps towards a mechanistic theory. In: *Sex Ratios: concepts and research methods*. pp. 26-45. (ed. I.C.W. Hardy) Cambridge, UK: Cambridge University Press.

42. Penn, D. J. & Smith, K. R. 2007 Differential fitness costs of reproduction between the sexes. *Proc Natl Acad Sci U S A* **104**, 553-8.

43. Harrell, C.J., Smith, K.R., & Mineau, G.P. 2008 Are girls good and boys bad for parental longevity? Human Nature **19**, 56-69.

Figure 1. Annual Sex Ratio at Birth (M/F) for Five Finnish Parishes, 1790 to 1870.

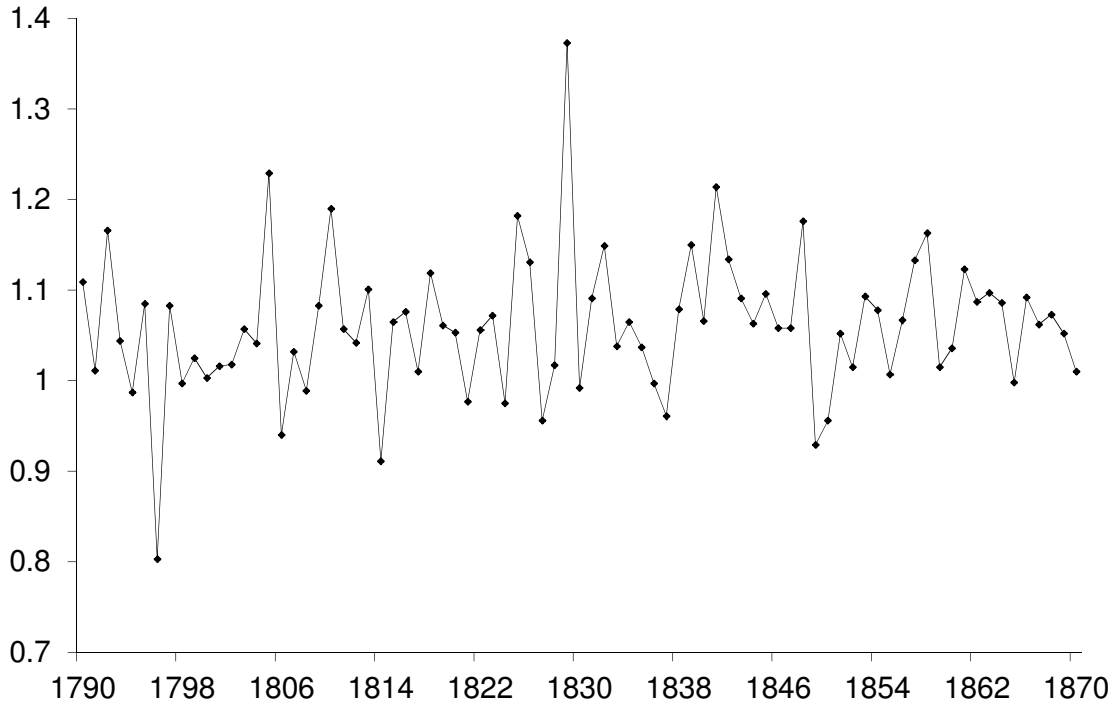

Table 1. Birth, fertility, and mortality characteristics of pre-Industrial Finns, 1790 to 1870.

	n	%	Mean (SD)
Annual sex ratio			1.06 (0.07)
Annual number of births			921.6 (218.4)
Males			
Lifespan in years			29.1 (28.5)
Region of Birth			
Archipelago (Kustavi, Hiittinen)	3,018	35.6	
Mainland Region (Ikaalinen, Tyrvaa)	4,478	52.8	
Northern Region (Pulkkila)	981	11.6	
Social Class at birth			
Poor	1,023	12.1	
Middle Class	3,566	42.1	
Wealthy	3,671	43.3	
Died before 1 year	1,463	17.3	
Died before 15 years	3,012	35.5	
Died before 50 years	4,242	50.4	
Ever Married	1,872	44.5	
Married and ≥1 live birth	1,460	41.0	
Married and ≥ 1 birth survived to 15 yrs	1,255	35.2	

Note: column percents may not sum to 100% due to rounding and non-exhaustive nature of categories.

Table 2. Coefficients (SEs) predicting the log-odds of death at specific age intervals for males as a function of the annual cohort sex ratio at birth and control variables.

	Death before 1yr		Death 1 to 4 yrs		Death 5 to 14 yrs		Death 15 to 49 yrs	
Variable	**Coef.**	**(SE)**	**Coef.**	**(SE)**	**Coef.**	**(SE)**	**Coef.**	**(SE)**
Cohort sex ratio at birth	0.08	(0.03)*	0.04	(0.04)	-0.05	(0.05)	-0.03	(0.04)
Firstborn son (ref: all others)	-0.06	(0.10)	-0.07	(0.12)	-0.33	(0.15)*	0.01	(0.13)
Interbirth interval (in years)	-0.06	(0.03)*	0.01	(0.03)	0.01	(0.04)	-0.01	(0.03)
Socioeconomic status at birth (1= poor; 2=middle 3=wealthy)	0.09	(0.05)†	-0.10	(0.05)†	-0.31	(0.07)***	-0.11	(0.07)
Region of birth (ref: Mainland)								
Archipelago	-0.10	(0.07)	-0.11	(0.08)	-0.01	(0.10)	0.58	(0.10)***
Northern region	0.12	(0.10)	0.55	(0.11)***	0.63	(0.14)***	0.67	(0.15)***
Time propensity of death	7.07	(2.62)**	8.14	(0.82)***	15.82	(2.45)***	4.57	(1.09)***
Indicators for birth month	Included; not shown		Included; not shown		Included; not shown		Included; not shown	
Number of observations used	7,824		6,500		5,446		2,292	

† p<.10; * p<.05; ** p<.01; *** p<.001; two-tailed tests. Sex ratio scaled as a z-score (i.e., one unit change is 1 SD). All analyses control for clustering of sibling observations within mother.

Figure 2. Scatter diagram of predicted probability of male infant death as a function of the cohort sex ratio at birth. Sex ratio is mean-centered at 0 and scaled in standard deviation units.

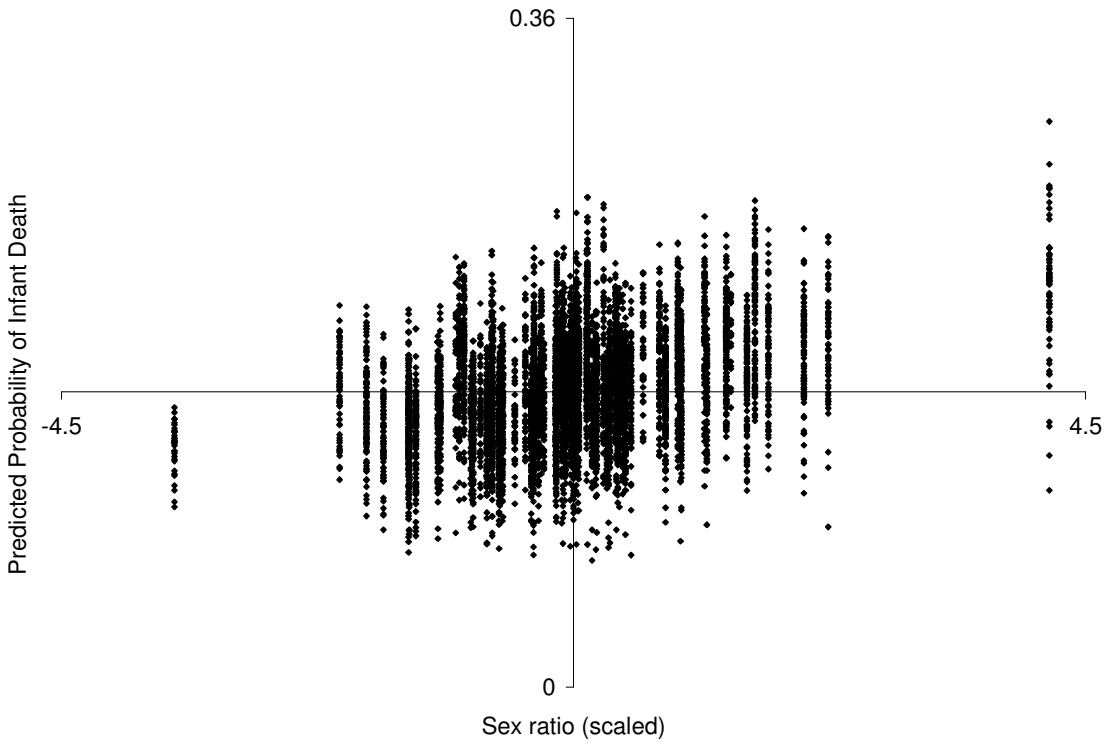

Table 3. Coefficients (SEs) from the structural equation model predicting number of fathered offspring surviving to 15 years as a function of the annual cohort's sex ratio at birth and control variables. Father's marital status and number of liveborn offspring used to estimate standard errors.

	Father's total number of children surviving to 15 years		
Variable	**Coef.**	**(SE)**	**p-value**
Cohort's sex ratio at birth	-0.042	(0.023)	.07
Firstborn son (ref: all others)	-0.22	(0.07)	.003
Interbirth interval (in years)	-0.04	(0.02)	.04
Socioeconomic status at birth (1= poor; 2=middle 3=wealthy)	0.09	(0.04)	.02
Annual cohort size	-0.06	(0.02)	.003
Region of birth (ref: Mainland)			
Archipelago	-0.27	(0.05)	<.001
Northern region	-0.63	(0.08)	<.001
Time propensity of outcome	0.10	(0.05)	.03
Month of birth (ref: December)			
January	0.19	(0.10)†	.08
February	0.04	(0.11)	.73
March	0.08	(0.11)	.49
April	0.19	(0.11)†	.08
May	0.10	(0.12)	.42
June	0.05	(0.12)	.67
July	0.27	(0.11)*	.27
August	0.22	(0.11)†	.05
September	0.12	(0.10)	.25
October	0.17	(0.11)	.14
November	0.14	(0.11)	.20
Number of observations analyzed		3,503	

Sex ratio scaled as a z-score. We fitted a zero-inflated Poisson distribution given its improved fit of the data relative to Poisson and negative binomial distributions. Results control for clustering of sibling observations within mother.

Figure 3. Scatter diagram of father's predicted number of children surviving to age 15 years as a function of the cohort sex ratio at birth (mean-centered at 0 and scaled in standard deviation units).