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Journal

Proceedings of the Royal Society B, 282(1799)

ISSN

0962-8452

Authors

Bruckner, Tim A
Helle, Samuli
Bolund, Elisabeth
[et al.](#)

Publication Date

2015-01-22

DOI

10.1098/rspb.2014.0835

Peer reviewed

**Culled males, infant mortality and reproductive success in a
pre-industrial Finnish population**

Tim A. Bruckner¹

Samuli Helle²

Elisabeth Bolund³

Virpi Lummaa⁴

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

Theoretical and empirical literature asserts that the sex ratio (i.e., M/F) at birth gauges the strength of selection *in utero* and cohort quality of males that survive to birth. We report the first individual-level test in humans, using detailed life history data, of the “culled cohort” hypothesis that males born to low annual sex ratio cohorts show lower than expected infant mortality and greater than expected lifetime reproductive success. We applied time series and structural equation methods to a unique multigenerational dataset of a natural fertility population in 19th century Finland. We find that, consistent with culled cohorts, a one standard deviation decline in the annual cohort sex ratio precedes an eight percent decrease in the risk of 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. The offspring result, however, falls just outside conventional levels of statistical significance. In historical Finland, the cohort sex ratio gauges selection against males *in utero* and predicts male infant mortality. The reproductive success findings, however, provide weak support for an evolutionarily adaptive explanation of male culling *in utero*.

Keywords: culled cohorts, male frailty, sex ratio, lifetime reproductive success, infant mortality

INTRODUCTION

Mean male lifespan falls below female lifespan in all human societies [1]. Historical mortality series also 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, between 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 disasters [5, 6, 7]. In addition, male fetal deaths rise, and the sex ratio falls, following the terrorist attacks of September 11, 2001 [8]. These results indicate that ambient stressors raise the risk of fetal death more among male than female gestations and result in a lower birth sex ratio.

A separate but related literature builds on this evidence and assumes that a component of temporal variation in the sex ratio does not involve sampling error. Aggregate-level analyses of males born to low sex ratio cohorts report lower mortality in infancy than that of males born to high sex ratio cohorts [9, 10; 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 perturbations involves the conservation of adaptive maternal mechanisms that spontaneously abort less fit fetuses [11,12]. Maternal mechanisms presumably assess the fitness of a fetus and environmental challenges to its survival if carried to birth [12, 13]. These mechanisms would terminate gestations that, if continued to birth, would have yielded an infant with relatively low likelihood of surviving to reproductive age and producing offspring [14]. 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 [14].

The argument that male fetal loss and sex ratio variation reflects an adaptive response to stressors would enjoy empirical support if males born to the most “culled” (i.e., low sex ratio) cohorts showed greater offspring surviving to adulthood than did males born to the least culled cohorts. The literature reports no such test of lifetime reproductive success (LRS). This circumstance likely arises from the rarity of the requisite historical, multigenerational data in humans. We address this gap in the literature and examine high-quality life history data from a pre-industrial population in Finland.

We test two hypotheses implied by culled cohorts. First, we test whether 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 LRS due to their higher quality. To suggest potential mechanisms that may produce higher LRS in men from culled cohorts, we also examined the influence of cohort sex ratio on probability of marriage (i.e., sexual selection) and total fecundity. We focus our test on males given that the culled cohort hypothesis makes no prediction about females [10].

In addition to providing the first test of sex ratio variation and LRS, 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 [14]. Our step-wise 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 competing hypothesis that spurious findings arise due to shared temporal patterns in the sex ratio, cohort mortality and LRS.

METHODS

Variables and Data

We retrieved birth, reproductive success, and death data from historical church records in Finland. Since the 18th 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 (see electronic supplementary material for more details) [15].

To derive our sex ratio variable, we used data from five parishes that contained a sufficiently large number of births per year to permit stable annual 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 [10]. Figure S1 (electronic supplementary material) 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 yielded 7,824 Finnish males. This 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 (electronic supplementary material, Table S2). Significant improvements in the standard of living occurred primarily during the 20th century in Finland [16]. Almost all women finished reproduction by age 45 and few gave birth out of wedlock [17].

Previous literature on sex ratios and male lifespan indicates distinct age-specific mortality responses [9]. 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 [18] in which the force of natural selection declines monotonically with age as well as recent empirical findings in historical Finland [15], 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 indicates that total number of children may not approximate LRS [19]. This dependent variable also correlates strongly with the long-term contribution to the future population gene pool [20].

Approach

Mortality

Our regression approach 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, given their known relationship with survival and/or sex ratio in the study population. These factors included the following: month of birth [21], interbirth interval, first-born status [22] and family socioeconomic status at birth (i.e., “wealthy” farm owners and merchants; “middle class” craftsmen and tenant farmers, and “poor” crofters and laborers) [23]. We also controlled for region of birth (archipelago, northern region, or mainland region) given the different background levels of mortality across the rural parishes. None of the independent variables correlated above 0.35, which minimizes the threat of multicollinearity.

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 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 [24]. This purely empirical approach, employed frequently in biodemography [5, 25], identifies autocorrelation in the time series. We used this identification strategy to arrive at annual, best-fitted values of male mortality rates for each specific birth cohort. This time propensity 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 cohort year, we assigned these fitted values back to each individual and used it as a covariate in the logistic regression. The ARIMA routines detected unique autocorrelation “signatures” for each mortality series

(results available upon request), which supports the need to control such patterns with time propensity values.

LRS

We did not have access to reproductive histories after 1900, which led to substantial missing data on offspring yield among 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.

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 have less within-cohort competition for employment and goods at adult ages and therefore yield greater than average marital success and fertility [26]. 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 also include information on marital status and number of children born. These variables may serve as "bottlenecks" for our LRS measure in that, in the Lutheran Church culture in Finland, marriage correlates positively with the likelihood of bearing children, which in turn correlates positively with the number of children surviving to 15 years. The SEM permits testing these three dependent

variables simultaneously within the same analytic framework and an exploration of whether the cohort sex ratio also predicted marital status and 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.

The numbers of live offspring, and offspring surviving to 15 years, had a large excess of zeroes (58% and 62%, respectively). We compared models with different count distributions (Poisson vs. negative binomial) and with and without zero-inflation. We ultimately fitted these dependent variables in SEM using a zero-inflated Poisson distribution given that this model yielded the smallest AIC- and BIC-value. In addition, we included in the model the residual covariances of marital status, live offspring, and offspring surviving to 15 years. This inclusion accounts for the within-individual correlation among dependent variables and improves efficiency of standard error estimates (see additional details of SEM in electronic supplementary material). 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 handled missing data using full-information maximum likelihood. We performed the LRS analysis using Mplus version 7.11 [27].

We employed two sensitivity analyses to assess the robustness of mortality and LRS results. First, for results that rejected the null, we included twin births. Second, we included cohort size in the mortality analyses to control for the possibility that sampling variation drove results.

RESULTS

Males born during years with a relatively low annual sex ratio show higher quality in terms of early survival and later reproductive output. To aid 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 is reduced with a lower cohort sex ratio (likelihood ratio $\chi^2=6.25$, $p=.01$, see Table 1). 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. We cannot reject the null for other age spans.

Figure 1 shows this discovered relation by plotting 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 yields the positively signed sex ratio coefficient in Table 1. 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.

Males born to lower sex ratio cohorts raise slightly more offspring to 15 years than do males born to higher sex ratio cohorts (likelihood ratio $\chi^2=3.02$, $p=.08$; see Table 2). Although the result falls outside conventional levels of statistical significance, the sign of the coefficient lies in the hypothesized direction. 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, $p=.29$). 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 2 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.

Sensitivity analyses for both the mortality and LRS outcome yielded essentially the same inference as in the initial tests (see electronic supplementary material).

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 [10]. Using life-history 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 LRS. Findings support the culled cohort hypothesis in that birth in a low sex ratio year predicts lower than expected male infant mortality. Men from low sex ratio cohorts also 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 Finnish dataset. The pre-industrial time period we investigate better approximates human mortality and reproductive schedules over much of human history than do contemporary datasets [28]. 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 cohorts. 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 [34].

We lacked information on fetal deaths for each mother and therefore inferred excess culling of frail male fetuses among low sex ratio cohorts. Low sex ratios may arise due to a variety of other mechanisms (e.g., those that affect sex at conception) [30]. 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 larger than those of an ecological test in historical Sweden [9], but diverges from a null result in a Finnish study from 1865-2003 [6]. We attribute this divergence to the difference in time period used. We also caution against making direct comparisons given that we, unlike earlier work, use 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 [15]. Although we use a cohort measure of variation in male mortality, a more refined individual-level indicator of male frailty, such as low birthweight or prematurity, 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*. Vaupel and colleagues theorize [31], and others find empirical support for [32], 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 [33].

The LRS findings indicate 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 [13] depends on sons yielding marginal gains in potential LRS relative to that of daughters [34]. This condition typically holds in populations with high reproductive skew (e.g., polygynous societies). In Finland, the Lutheran church precluded these conditions, which yielded a conservative test for LRS. 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 [35, 36].

Our male infant mortality finding coheres with Hamilton's logic that natural selection exerts its greatest pressure on mortality at earlier ages, when the replacement cost to the parents is relatively low [18]. As this argument relates to our findings, the failure of a male to survive past infancy imposes fitness costs since it precludes him from engaging in competition for female mates. By contrast, mortality at an older age (i.e., in mid-adulthood) would allow that male an opportunity to reproduce.

We hesitate to speculate *post hoc* on why the data support the infant mortality more than the LRS aspect of our hypothesis. However, one review posits that the early-life environment could exert independent effects on infant

survival and later-life reproductive success (see Fig. 2 of [37]). In addition, after birth, parental investment or local resource levels may exert a relatively strong influence on LRS that acts separately from effects on infant morbidity.

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 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.

Acknowledgements: We thank Lasse Iso-livari, Kimmo Pokkinen and Aino Siitonen for their assistance with data collection and the European Research Council for funding the data collection. SH was supported by The Turku Collegium for Science and Medicine.

Data accessibility: We will make data available via the Dryad Data repository.

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Table 1. 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.

Variable	Death before 1yr		Death 1 to 4 yrs		Death 5 to 14 yrs		Death 15 to 49 yrs	
	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 1. 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.

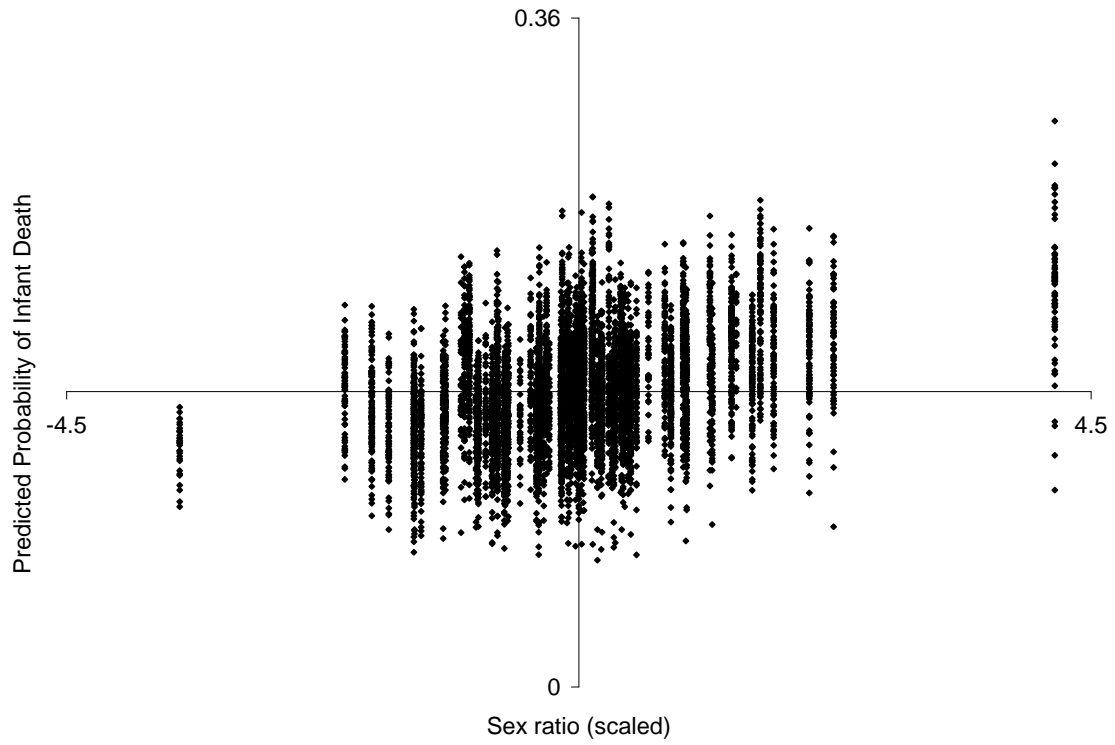
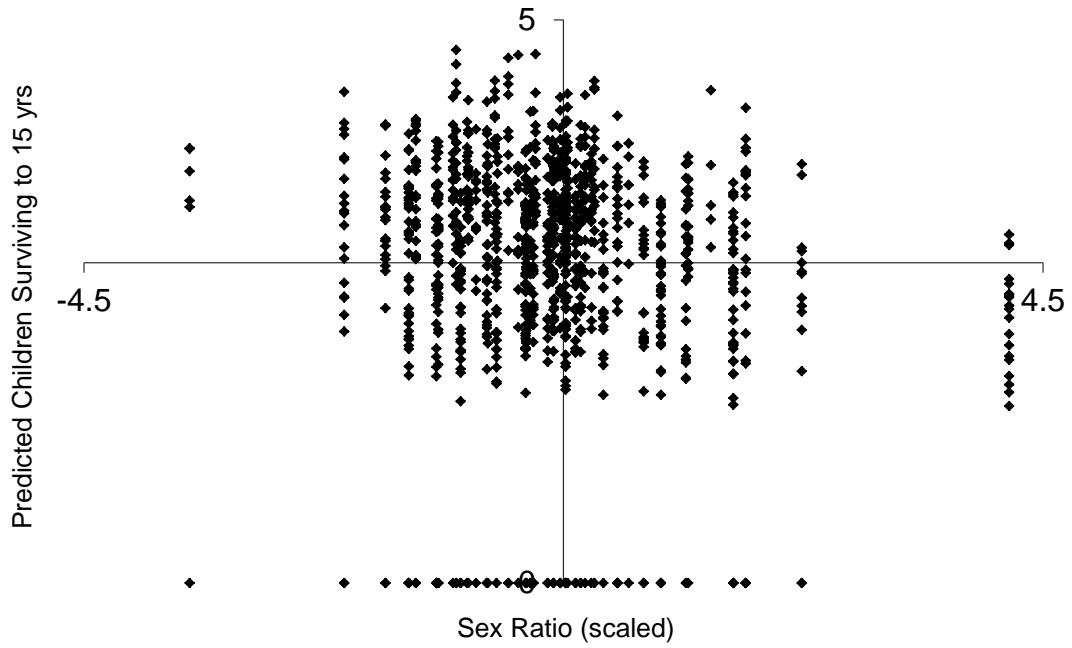


Table 2. Coefficients (SEs) from the structural equation model predicting number of fathered offspring surviving to 15 years as a function of the annual cohort's sex ratio at birth and control variables. Father's marital status and number of liveborn offspring used to estimate standard errors.

Variable	Father's total number of children surviving to 15 years		p-value
	Coef.	(SE)	
Cohort's sex ratio at birth	-0.042	(0.023)	.08
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 smallest AIC and BIC value relative to that of Poisson and negative binomial distributions. Results control for clustering of sibling observations within mother.

Figure 2. 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).



Supplementary Material for:

Bruckner TA, Helle S, Bolund E, Lummaa V. Culled males, infant mortality and reproductive success in a pre-industrial Finnish population.

Human Life History Database

Careful documentation of the Church registries allows for linkage of data between mothers and their children. The Finnish Human Life History 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 excluded twins based on their different life histories from that of singletons [Lummaa et al., 2001].

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.

Structural Equation Model

We further used marital status (1=married; 0= never married) to predict the zero-inflation component of number of born and survived offspring (1= had children; 0 = no children) 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.

Sensitivity Analyses

We performed two sensitivity analyses to assess the robustness of our findings. First, 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, $p = .105$). Second, we included cohort size in the infant mortality analysis to control for the possibility that sampling variation (due to small cohort sizes) drove the sex ratio result. Inference remained essentially unchanged (OR=1.09; 95% CI = 1.02—1.16, $p = .01$).

Figure S1. Annual Sex Ratio at Birth (M/F) for Five Finnish Parishes, 1790 to 1870.

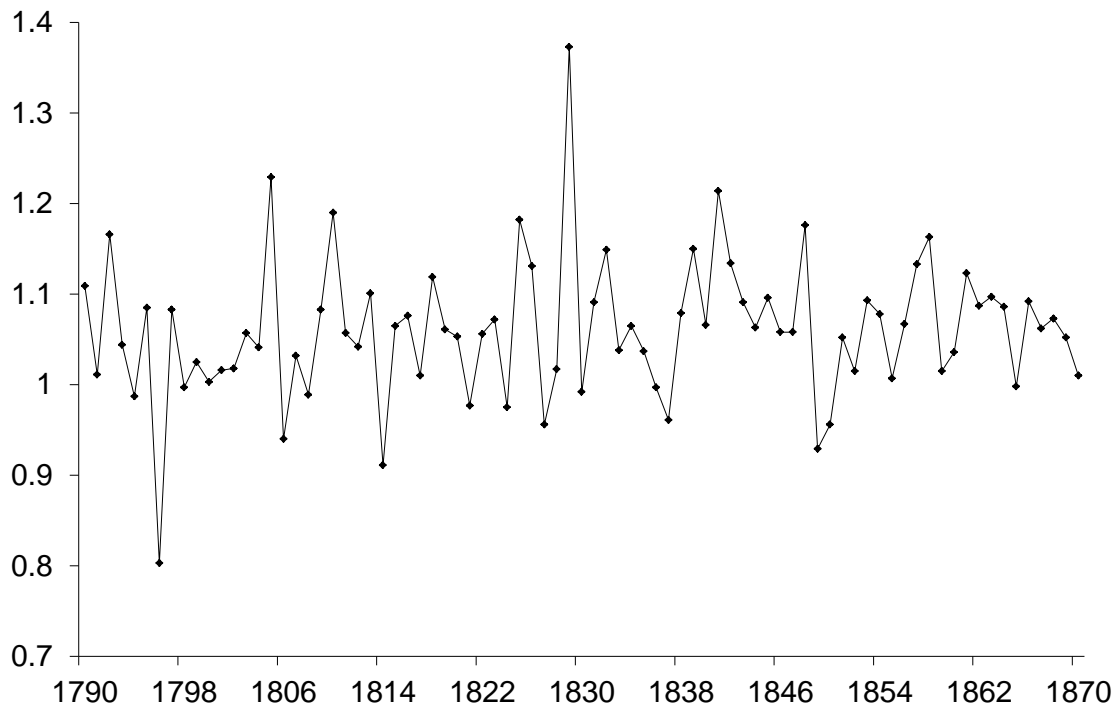


Table S2. Birth, fertility, and mortality characteristics of pre-Industrial Finns, 1790 to 1870.

	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, Tyrvaä)	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.