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Separating the Bruce and Trivers-Willard effects in theory and in human data

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Abstract

Objectives: Theories of reproductive suppression predict that natural selection would conserve mechanisms that abort the gestation of offspring otherwise unlikely to thrive in prevailing environments. Research reports evidence among humans of at least two such mechanisms—the Trivers-Willard and Bruce Effects. No literature, however, compares the mechanisms nor estimates their relative contribution to observed characteristics of human birth cohorts. We describe similarities and differences between the Trivers-Willard and Bruce Effects and explore high quality historical data from Sweden to determine which mechanism better describes temporal variation in the ratio of males to females in birth cohorts.

Methods: We measure Trivers-Willard exposures with the death rate among women of reproductive age. We measure Bruce exposures with the death rate among children. We use time-series regression methods to estimate the relative contribution of the Trivers-Willard and Bruce Effects to temporal variation in historical Swedish secondary sex ratio data.

Results: We find that the Bruce Effect appears to be a better predictor of the secondary sex ratio than does the Trivers-Willard Effect.

Conclusions: Attempts to identify mechanisms by which reproductive suppression affects fetal loss and characteristics of human birth cohorts should consider the Bruce Effect as an alternative to the Trivers-Willard Effect.

1 | INTRODUCTION

Theories of reproductive suppression posit that natural selection conserved mechanisms that reduce fertility when offspring would otherwise fail to thrive (Beehner & Lu, 2013; Haig, 1999; Wasser & Barash, 1983). This paper focuses on two mechanisms, the Bruce (1959) and Trivers and Willard (1973) Effects, thought to implement reproductive suppression in several species, including humans. Although the lines of literature describing these mechanisms rarely acknowledge each other, they have much in common. Both, for example, assume that pregnant women vary in their capacity to invest in children and that children vary in their need for maternal investment to thrive in prevailing environments. Both mechanisms also assume that low-resource mothers with high-need children will have relatively few grandchildren because their children will more frequently die before reproductive age than will the children of other mothers. These shared assumptions lead to the inference that natural selection would conserve mutations that spontaneously abort gestations in which the needs of the prospective child would otherwise exceed the resources of the prospective mother.

The arguments embedded in these two lines of literature also have important differences. We describe these differences and explore historic data to determine which argument better describes temporal variation in an important characteristic of human birth cohorts—the secondary sex ratio.

Reproductive suppression predicts that the likelihood of spontaneous abortion will increase when the fraction of pregnant women with relatively few resources increases, while the distribution of fetuses needing maternal resources remains unchanged. This circumstance, referred to in the literature as the Trivers-Willard Effect, implies that fewer high need infants will be born when the environment weakens women of reproductive age (Cameron, 2004; Trivers & Willard, 1973).

Reproductive suppression also predicts that the frequency of spontaneous abortion will increase when the fraction of pregnant women with relatively few resources remains unchanged, but the distribution of fetuses needing maternal investment shifts upward (Ebensperger, 1998; Labov, 1981). This prediction arises, in part, from animal literature reporting increased fetal loss when gravid females encounter signals of death among young conspecifics (Labov, 1981). This "Bruce Effect," first noted among laboratory animals (Bruce, 1959), has since been generalized to humans (Catalano, Saxton, Gemmill, & Hartig, 2016b). A Bruce Effect in humans implies that fewer high need infants will be born when the environment increases morbidity and mortality among children (Becker & Hurst, 2008; Saxton, Gemmill, & Catalano, 2017).

In sum, environmental circumstances that reduce maternal resources should trigger the Trivers-Willard Effect, while circumstances that increase children's need for maternal resources should trigger the Bruce Effect. Environmental circumstances that both reduce maternal resources and increase offspring need for such resources would activate both the Trivers-Willard and Bruce Effects.

Although empirical observation supports the argument that human populations confronting threats to maternal and offspring well-being exhibit signals of reproductive suppression via spontaneous abortion (Arck, 2001; Bruckner, Mortensen, & Catalano, 2016; Catalano et al., 2005; Forbes, 1997; Grech, 2015; Kozlowski & Stearns, 1989; Nepomnaschy et al., 2006), we know of no attempt to estimate the relative contribution of the Trivers-Willard and Bruce Effects. Here, we use high quality historic data from Sweden to estimate the separate and combined association of threats to maternal resources and to child survival with one widely accepted signal of strategic spontaneous abortion—the secondary sex ratio (i.e., ratio of male to female live births).

Half or more of human conceptions fail to yield a live birth (Boklage, 1990; Wang et al., 2003). Conceptuses that survive to birth do not represent their conception cohort (Wilcox et al., 1988; Zinaman, Clegg, Brown, O'connor, & Selevan, 1996). Early in gestation, spontaneous abortion targets conceptuses with chromosomal, genetic, and morphological abnormalities that would make survival to reproductive age unlikely even in benign environments (Coulam, 2016; Quenby, Vince, Farquharson, & Aplin, 2002; Teklenburg et al., 2010). Later in gestation, spontaneous abortion selects mostly against small for gestational age, but otherwise normal, males (Bukowski et al., 2014; Mondal, Galloway, Bailey, & Mathews, 2014; Räisänen, Gissler, Saari, Kramer, & Heinonen, 2013). Explanations of male predominance among later spontaneous abortions characterize the loss as a strategic adjustment to optimize the fitness of birth cohorts, given environmental conditions (Catalano et al., 2016a). Prior work (Bruckner, Helle, Bolund, & Lummaa, 2015; Lummaa, 2001; Lummaa, Pettay, & Russell, 2007) suggests that sons born in uncertain environments produce fewer grandchildren for their mothers than do daughters because, despite receiving relatively great maternal investment (Clutton-Brock, 1991; Helle, Lummaa, & Jokela, 2002; Powe, Knott, & Conklin-Brittain, 2010), males die more frequently as infants and children than females in virtually every society and every year for which we have dependable vital statistics (Human Mortality Database, 2017). These facts have led to the assumption that the spontaneous abortion of prospective daughters in a conception cohort appears due primarily to endemic abnormalities in female fetuses whereas the loss of prospective sons, while also due to endemic abnormalities, additionally reflects that male fetuses predominate in the mismatches of low-resource mothers and high-need prospective offspring (Trivers & Willard, 1973; Wells, 2000).

Recognizing the circumstances summarized above, measurements of the association between stressful environments and spontaneous abortion frequently use the secondary sex ratio (i.e., number of male births per 100 female births) as the dependent variable (Catalano & Bruckner, 2006; Catalano, Bruckner, & Smith, 2008; Sanders & Stoecker, 2015). Although temporal variation in the secondary sex ratio arises from several circumstances (James & Grech, 2017), the literature offering explanations of declines during stressful times has cited the Trivers and Willard Effect as one connecting mechanism (James, 2013). Indeed, Trivers and Willard offered their theory to explain, at least in part, low sex ratios in stressed human, as well other mammalian, populations (Trivers & Willard, 1973).

We know of one invocation of the Bruce Effect as a potential driver of the human secondary sex ratio (Saxton et al., 2017). The authors reasoned that if the Bruce Effect suppressed the birth of less fit males, the sex ratio should exhibit an inverse correlation over time with death rates among children. They tested this argument using annual Swedish data from the late 18th and early 19th centuries when modern public health and medicine had yet to induce strong secular trends in mortality that would overwhelm signals of more endemic mechanisms such as the Bruce Effect. They report support for their argument.

We explore historical data from Sweden in search of evidence that either the Trivers-Willard Effect, or Bruce Effect, or both affected the secondary sex ratio. More specifically

we test the hypothesis that the sex ratio varied inversely, controlling for autocorrelation, with the odds of death in the same and previous year among children as well as among women of reproductive age and with the interaction of these two variables.

2 | METHODS

2.1 | Data and variables

We extracted data for our tests from the Human Mortality Database (2017) that includes historical life table data from countries with dependable vital statistics. Among countries with vital statistics that meet the standards for inclusion in the Database, Sweden's begin the earliest, with the cohort born in 1751. Our test ends with the cohort born in 1840 because the outmigration of Swedes of reproductive age accelerated to relatively high levels in the ensuing decade and persisted well into the 20th century, implying that temporal variation in our variables could, after 1840, arise from changes in the composition of the population (Statistics Sweden, 1999).

We specified our dependent variable, the annual secondary sex ratio, by multiplying the annual odds of a male live birth by 100. We constructed our independent variable for the Bruce Effect from the odds of death among Swedes aged 1 to 9. We excluded infants to avoid confounding our test with the reported high infant mortality among males from high sex ratio birth cohorts (Bruckner & Catalano, 2007). We used the odds of death among Swedish women aged 20 to 49 years to construct our independent variable for the Trivers-Willard Effect.

2.2 | Analyses

Tests of association measure the extent to which variables move away from their statistically expected values in the same cases (Stigler, 1989). "Cases" in our tests include 90 years (i.e., 1751 through 1840) for which we know the sex ratio of birth cohorts as well as the death rates among children and women of reproductive age. The Bruce and Trivers-Willard Effects predict that the observed secondary sex ratio will fall below expected when the observed odds of death among children and women of reproductive age rise separately and together above their expected values.

Tests of association typically assume normal and independent distribution of variables. These assumptions allow specifying the expected value of a variable as its mean. Variables measured over time, however, often violate these assumptions by exhibiting "autocorrelation" in the form of secular trends, cycles, or the tendency to remain elevated or depressed, or to oscillate, after high or low values. The expected value of an autocorrelated series is not its mean, but rather the value predicted by autocorrelation. Following the practice dating at least to Fisher (1921), we solve this problem by identifying time-series models that best fit observed autocorrelation in our three series and by using the values the models predict as expected values. We used the most developed and widely disseminated type of such modeling. The method, devised by Box and Jenkins (1976), identifies which of a very large family of models best fits measurements made serially in time. The modeling procedure essentially assumes that measurements independent of each other and normally distributed passed through an unobserved "filter" that imposed patterns upon them. Box-Jenkins methods identify the filter, *post hoc*, from the imposed pattern. The methods use well-developed rules to narrow the likely filters to a few and then applies estimates of "fit" to arrive at the most likely candidate. The model residuals approximate the values that passed through the filter. These "unexpected" values meet the assumptions of traditional tests of association because they are independent of each other (i.e., exhibit no autocorrelation), their expected value equals their mean (i.e., 0), and they exhibit constant variability over time.

The Box and Jenkins approach attributes autocorrelation to integration as well as to "autoregressive" and "moving average" parameters. Integration describes secular trends and strong seasonality. Autoregressive parameters best describe patterns that persist for relatively long periods, while moving average parameters parsimoniously describe less persistent patterns.

Our test proceeded through the following steps. First, we used Box and Jenkins methods (1976) to detect and model autocorrelation in the Swedish secondary sex ratio, odds of child death, and odds of death among women of reproductive age, for the years 1751 through 1840. The residuals of these models estimate the degree to which observed values exceed or fall below values expected from autocorrelation and match the assumptions, made by tests of association, of independent and normal distribution.

Second, we created Bruce and Trivers-Willard exposure variables by rescoring negatively signed residuals from the two models for odds of death to 0 and positively signed residuals to 1. This scoring left variables that indicated birth cohorts exposed *in utero* to unexpectedly great threats to maternal (i.e., Trivers-Willard Effect) or child (i.e., Bruce Effect) wellbeing. The product of these two variables indicated cohorts exposed to both the Bruce and Trivers-Willard Effects.

Third, we estimated the equation formed by adding, as predictor variables, the 3 binary variables created in the step above to the model developed in Step 1 for the secondary sex ratio. We specified each of the 3 variables in the same year (i.e., year t) and year before (i.e., year $t-1$) the observed sex ratio. This specification reflects the fact that a large

TABLE 1 Means, standard deviations, and best fitting Box-Jenkins models for variables used in test ($n = 90$ years beginning 1751)

	Mean (Standard Deviation)	Box-Jenkins Model
Swedish male live births per 100 female live births	104.5892 (0.7484)	$Z_t = 104.5892 + a_t$
Odds of death among Swedes aged 1 through 9	0.0246 (0.0092)	$Z_t = 0.0232 + 1/(1-0.3073B)(1-0.3363B^{11}) + a_t$
Odds of death among Swedish women aged 20 through 49	0.0111 (0.0024)	$Z_t = 0.0111 + 1/(1-0.4762B) + a_t$

fraction of the gestations yielding births in year t began and progressed in year $t - 1$.

Fourth, we inspected the residuals of the model estimated in step 3 for autocorrelation and formed the test model shown below by adding any indicated Box-Jenkins parameters to the model estimated in step 3.

$$(1-\phi B^p)[100(M_t/F_t)] = C + \omega_1 X_{1t} + \omega_2 X_{1t-1} + \omega_3 X_{2t} + \omega_4 X_{2t-1} + \omega_5 X_{3t} + \omega_6 X_{3t-1} + (1-\theta B^q)a_t$$

M_t is the number of male births in year t. F_t is the number of female births in year t. C is constant. ω_1 to ω_6 are effect parameters. X_{1t} is the Bruce exposure in year t. X_{2t} is the

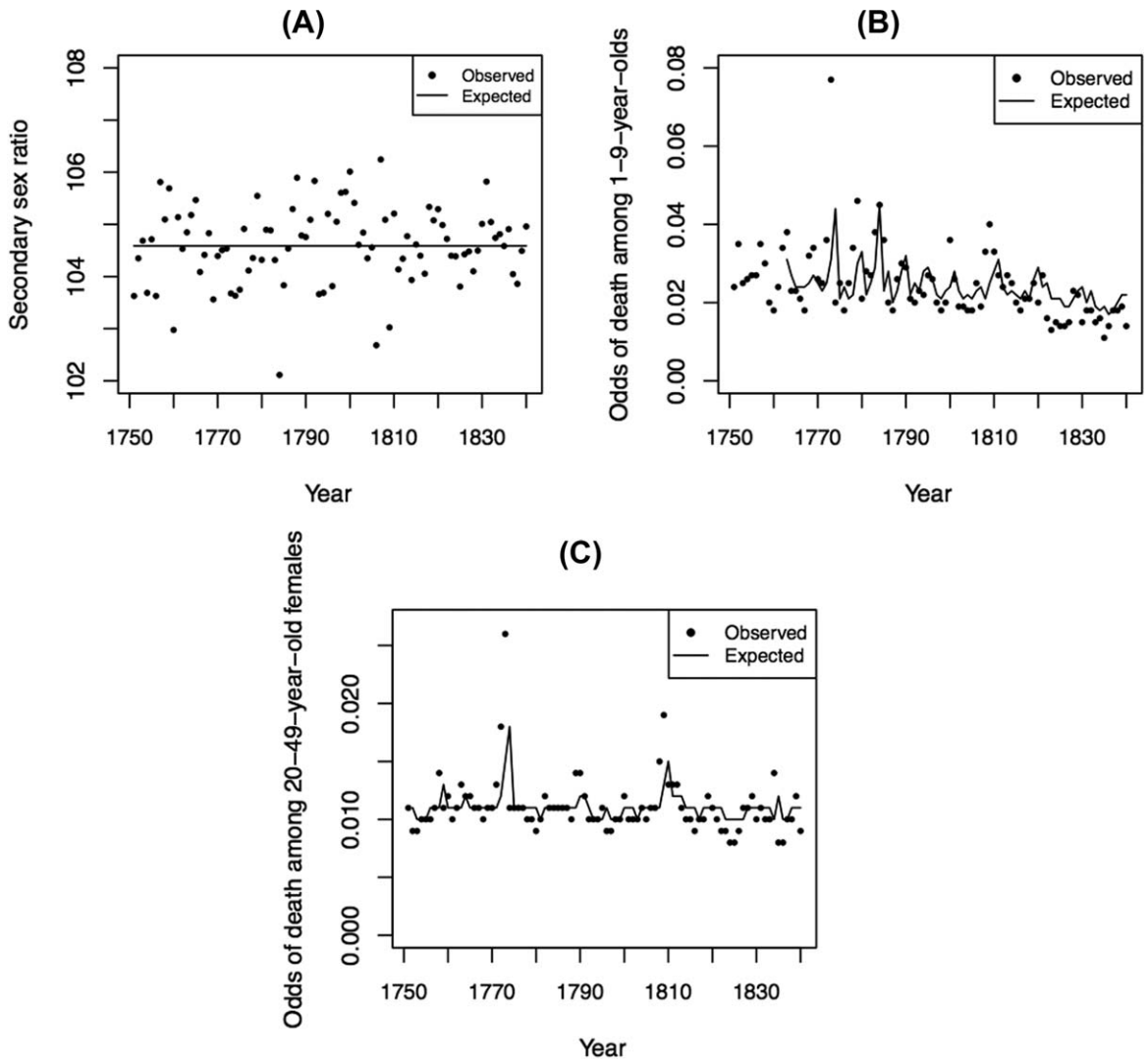

FIGURE 1 Observed and expected (i.e., from series mean in panel A and from Box-Jenkins models in panels B and C) values of the annual Swedish secondary sex ratio, odds of death among Swedes aged 1 through 9, and odds of death among Swedish women aged 20 through 49 ($n = 90$ years beginning 1751)

Trivers-Willard exposure in year *t*. X_{3t} is the interaction of the Bruce and Trivers-Willard exposures at year *t*. θ is added if autocorrelation best modeled by a moving average parameter were detected in step 4. ϕ is added if autocorrelation best modeled by an autoregressive parameter were detected in step 4. *B* is the "backshift operator" or value of $100(M_t/F_t)$ at year *t-p* or of *a* at year *t-q*. a_t is the residual of the model at year *t*.

3 | RESULTS

Table 1 shows the means and standard deviations of the 3 variables used in our tests. Table 1 also shows the Box-Jenkins models identified in step 1 above for each of the series. The sex ratio exhibited no autocorrelation implying that its mean was its expected value.

The separate panels of Figure 1 plot the observed and expected (i.e., mean in panel A, from Box-Jenkins models in panels B and C) values of the 3 variables. As noted in step 2 above, we created Bruce and Trivers-Willard exposure variables by subtracting the expected from observed values shown in panels B and C of Figure 1 and rescoring negatively signed differences to 0 and positively signed differences to 1. The product of these two binary variables indicated cohorts exposed to both effects. Forty-three of the 90 test years qualified as either or both Bruce or Trivers-Willard Effect years.

Table 2 shows the 11 years in which only the Bruce Effect appeared, the 15 years indicated by only the Trivers-Willard Effect, and the 17 joint effect years. Table 3 shows the results of estimating the test equation described in step 4 above. As shown, the data suggest that the Bruce exposure, but not the Trivers-Willard exposure or their interaction, shares unique variance with the sex ratio. The association appears such that the sex ratio drops in the year following unexpectedly high death rates among children. We understand these findings to mean that, consistent with the Bruce Effect literature, women pregnant when, or close after, deaths among Swedish children rose above expected levels gave birth to fewer sons than expected from the number of daughters and from autocorrelation. The sex ratio dropped by approximately 0.62 males per hundred females in the year following unexpectedly high odds of child death.

Table 3 also show that the residuals of the model estimated in step 4 exhibited autoregression at years 4 and 5. This suggests that Bruce and Trivers-Willard exposures may have, during our test period, masked autocorrelation in the sex ratio induced by forces that exhibit autoregression in which high or low values echo, although diminished, 4 or 5 years later. Any attempt to characterize these forces would be speculative and *post hoc*. We used the methods of Chang, Tiao, and Chen (1988) to determine if outliers in the sex ratio distorted our test estimations. The methods detected no outliers.

TABLE 2 Years in which only the Bruce and Trivers-Willard Effects, as well as their interaction, appeared in Sweden during our test period (i.e., 90 years beginning 1751)

Bruce effect only	Trivers-Willard effect only	Both effects
1768	1765	1763
1769	1767	1770
1778	1790	1771
1779	1795	1772
1783	1807	1773
1788	1811	1775
1810	1812	1777
1813	1817	1781
1814	1819	1782
1818	1827	1784
1821	1829	1785
	1831	1789
	1834	1800
	1837	1806
	1839	1808
		1809
		1828

Table 3 also shows the estimated parameters for a model that excluded the Trivers-Willard Effect, same-year Bruce Effect, and joint effect parameters because none of these appeared to contribute to variation in the sex ratio. Estimating this model yielded a coefficient of −0.52 for the Bruce Effect in years following unexpectedly high odds of death among children.

The secondary sex ratio fell below expected values (i.e., its mean) in 40 of our 90 test years. The average of these declines was −0.83. As implied by the coefficient for the Bruce Effect, the sex ratio fell by an average of −0.52 in the years after unexpectedly high odds of death among children. The expected Bruce Effect on the Swedish sex ratio equaled, therefore, about 62 percent (i.e., −0.52/–0.83) of the average of annual declines of the sex ratio below expected values.

A common metric of strength of effect is the change in a model's R^2 attributable to the independent variable. The model that included the Bruce Effect variable at year $t-1$ as well as the autoregressive parameters at years 4 and 5

TABLE 3 Coefficients and standard errors of equation expressing the annual Swedish secondary sex ratio as a function of Bruce and Trivers-Willard Effects, their interaction, and autocorrelation ($n = 90$ years beginning 1751)

Predictor	Full test model	Pared model
Constant	104.906** (0.103)	104.793** (0.074)
Bruce Effect exposure at year t	−0.057 (0.297)	
Bruce Effect exposure at year $t-1$	−0.632** (0.205)	−0.524** (0.143)
Trivers-Willard Effect exposure at year t	−0.001 (0.185)	
Trivers-Willard Effect exposure at year $t-1$	−0.121 (0.184)	
Interaction Effect exposure at year t	−0.506 (0.287)	
Interaction Effect exposure at year $t-1$	0.498 (0.284)	
Box-Jenkins Parameters		
Autoregression at 4 years	−0.286* (0.113)	−0.241* (0.109)
Autoregresssion at 5 years	−0.354** (0.110)	−0.331** (0.107)

*$P < .05$, 2-tailed test.
**$P < .01$, 2-tailed test.

accounted for 14 percent of the variance in the secondary sex ratio. Removing the Bruce Effect variable reduced the R^2 to 2 percent.

4 | DISCUSSION

As noted at the outset, much of the literature testing an association between population stressors and the secondary sex ratio cites the Trivers-Willard Effect as the explanatory mechanism, implying that reduced maternal resources connect the two phenomena. Our findings, based on dependable historical data from Sweden, suggest, however, that the association may instead arise from the Bruce Effect induced by threats to child survival.

Our use of high-quality historical data from Sweden—the country with the longest historical vital statistics records unaffected by mass migration—allowed us to estimate the association between mortality and nativity in a closed population over a long period. These data may add to the internal validity of our tests, but they may also detract from external validity. The fact that Swedes kept such detailed data earlier than other societies raises questions of how well Sweden represented those societies during our test period. That test period, moreover, ended a century and a half ago thereby raising questions concerning the meaning of our results for contemporary populations. For these reasons, we acknowledge the exploratory rather than confirmatory contribution of our work.

Our test estimates the unique contribution of the Bruce and Trivers-Willard Effects to the observed sex ratio. We estimate effects for each mechanism based on years in which mortality for either reproductive aged women or children increased above expected levels while mortality for the other did not. Such age and sex specific mortality arises from "normal" variation in, for example, infectious disease outbreaks. Counterintuitively, large shocks to the population, such as the famine of 1773, reflected in our joint effect variable, did not appear to affect the sex ratio beyond their contribution to the Bruce Effect alone. We speculate that this finding may reflect the depletion of vulnerable cases.

Both the Bruce Effect and the Trivers-Willard Effect describe adaptive mechanisms of reproductive suppression whereby females of several species avoid investing in offspring unlikely to thrive in prevailing environments. We focused on the secondary sex ratio as an indicator of reproductive suppression in women. Further research in humans should examine the effects of environmental threats to children on other indicators of greater clinical or societal interest such as amenorrhea, timing of puberty, or periviable birth.

We acknowledge that this work has more basic than applied implications. The former include support for reproductive suppression in humans, or the argument that natural selection has conserved mechanisms by which women, like females from several species, assess environmental threats to offspring and use that information to regulate their reproductive biology (Forbes, 1997). The nature of these assessments, however, remains unclear. The animal literature suggests that the standard of fetal hardiness females set for continued investment in gestation reflects signals of environmental virulence as diverse as the visible corpse of a young conspecific (Fashing et al., 2011) or changes in humidity that increase

the presence of parasites (Masi et al., 2012). Work in humans suggests that human chorionic gonadotropin (i.e., hCG) signals fetal hardiness (Forbes, 2017) and that the value needed to continue gestation depends on threats to infants in prevailing environments (Catalano, Currier, & Steinsaltz, 2015). We speculate that cognition may mediate assessments of environmental threats because "news" describing the death of children appears to be associated with fetal loss (Catalano et al., 2016b).

Our findings may, however, also contribute to the applied conversation over where society should focus attempts to prevent fetal loss. Much of the empirical literature that cites the Trivers-Willard Effect to explain low sex ratios in stressed human populations also invokes a "dysregulation" narrative that implies spontaneous abortion signals maternal failure to cope with environmental stressors. This, in turn, suggests interventions to help pregnant women manage stress. Several small studies report, for example, that supportive services may reduce recurrent miscarriage (Clifford, Rai, & Regan, 1997; James, 1963; Liddell, Pattison, & Zanderigo, 1991). A Cochrane review of 11 randomized clinical trials, including 10,429 women thought to be at risk for any adverse pregnancy outcome, however, found no effect of such services on miscarriage or perinatal death (Hodnett, Fredericks, & Weston, 2010).

Our results imply that a significant fraction of spontaneous abortion in stressful environments may result from the Bruce Effect—a well-regulated mechanism that protects the reproductive fitness of healthy women. Indeed, the association between population stressors and low secondary sex ratios may reflect more a failure of society to regulate environmental threats to children than a failure of the reproductive biology of prospective mothers. This perspective suggests that any accounting of costs and benefits of proposals to invest in community improvement should include assessment of implications for fetal loss.

COMPETING INTERESTS

We have no competing interests.

AUTHORS' CONTRIBUTIONS

RC conceived the paper, conducted the data analysis, and drafted all sections except the discussion. AG contributed to the methods, results, and discussion sections. JC contributed to the methods and discussion sections. DK contributed to the introduction and discussion sections. HS contributed to the discussion and edited the manuscript. KS contributed to the introduction and discussion.

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