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Does differential mortality after parental investment affect sex ratio evolution? No

Alistair Pirrie[1,2] **and Ben Ashby**[1]

[1]*Department of Mathematical Sciences, University of Bath, Bath BA2 7AY, United Kingdom*

[2]*E-mail: arp59@bath.ac.uk*

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The classical view of sex ratio evolution, popularized by R. A. Fisher, is that the sex ratio at birth should be equal when males and females require the same level of parental investment. Thus, although differences in mortality between the sexes during parental investment will cause deviations from an equal sex ratio at birth, differential mortality after parental investment should have no effect. However, a recent theoretical model appears to contradict this view, suggesting that differential mortality after the period of parental investment does cause deviations from an equal sex ratio at birth. Moreover, the life stage at which mortality differs (juvenile vs. adult) is predicted to cause contrasting effects on sex ratio evolution. These results are in stark contrast with Fisher's hypothesis. Here, we resolve this disparity by analyzing a stage- and sex- structured model of population dynamics. We find that selection always drives the population to an equal sex ratio at birth regardless of differential mortality effects after parental investment, thus confirming Fisher's hypothesis. The disparity appears to be due to incorrect accounting of mutant-resident unions, which we avoid by considering separate union classes for different types of mutant-resident unions.

KEY WORDS: **Adult, age structure, evolution, juvenile, mortality, sex ratio.**

Offspring sex ratio has long been a subject of interest in evolutionary biology (Darwin 1871; Düsing 1884; Fisher 1930; Kolman 1960; Leigh 1970; Trivers 1972; Charnov 1982; Frank 1990; Hardy 2002; West 2009; Fawcett et al. 2011; Shyu and Caswell 2016). In essence, when should parents produce more sons than daughters, or more daughters than sons, or equal amounts of each sex? In 1930, R. A. Fisher argued that, given certain simplifying assumptions (e.g., randomly mixing populations), selection will favor equal parental investment in male and female offspring. Hence, when sons and daughters require the same level of parental investment, one should expect a 1:1 sex ratio at birth. Deviations from equal costs—for example, unequal demands in rearing sons and daughters, or differential mortality during the period of parental investment—are predicted to select for an uneven sex ratio. Thus, if sons are on average less costly to produce than daughters, then selection will favor a skewed sex ratio at birth in favor of males (Fisher 1930). Conversely, Fisher predicted that differential mortality between the sexes after parental investment will not affect selection for sex ratio at birth. Intuitively, this is because higher mortality in one sex, after parental investment has ended, does not affect the cost to the parent of producing that sex and the benefits are unchanged because a decrease in the number of offspring of one sex is perfectly compensated by an increase in the survivors' reproductive value. Although Fisher (1930) only proposed a verbal argument to support his view, mathematical models later confirmed his intuition (Kolman 1960; Leigh 1970).

Although the Fisherian view of sex ratio evolution is widely accepted (West 2009), in nature there are often deviations from an equal sex ratio at birth, which can have significant effects on mating dynamics and disease transmission (Halimubieke et al. 2021). Understanding when deviations from an equal primary sex ratio occur therefore remains an active area of research (Kahn et al. 2015; Shyu and Caswell 2016; Zietsch et al. 2020; Lehtonen 2021; Orzack and Hardy 2021). Various mechanisms have been shown to cause selection to favor unequal sex ratios, including local mate competition (Hamilton 1967), condition-dependent effects on reproductive success (Trivers and Willard 1973), and overlapping generations in the population alongside either seasonal variation or environmental perturbations (Werren

and Charnov 1978; Kahn et al. 2013) (see West [2009] for a detailed review of sex ratio theory and conditions that cause deviation from a 1:1 sex ratio at birth). In most cases, deviations from a 1:1 sex ratio at birth occur because the assumptions of Fisher's hypothesis are violated, and so there is no inconsistency between predictions.

Recently, however, Shyu and Caswell (2016) suggested that Fisher was incorrect, and that differential mortality after the period of parental investment does cause deviations from a 1:1 sex ratio at birth. By specifically modeling union formation and stage structure, Shyu and Caswell (2016) appear to show that differential mortality between the sexes postparental investment affects the evolution of sex ratio at birth. Specifically, their results suggest that the sex ratio at birth will evolve to be biased toward the sex that experiences: (1) lower mortality prior to reproduction but after parental investment has ended; and (2) higher adult mortality. These predictions directly contradict Fisher's hypothesis. Here, we use a stage-structured model with union formation to investigate the discrepancy between the predictions. We confirm the Fisherian view that differential mortality postparental investment does not cause selection to deviate from an equal sex ratio at birth. We suggest that the predictions made by Shyu and Caswell (2016) differ due to erroneous assumptions in their model regarding the dynamics of rare mutants, the most important of which causes a bookkeeping error when mutants die while in unions with the resident.

Models and Results

No stage structure or mating unions

We first present a simple model without stage structure or mating unions to demonstrate why there is no deviation from an equal sex ratio at birth despite differential mortality between the sexes. The model has two classes, males (M) and females (F), with the population dynamics given by

$$\frac{dM}{dt} = \underbrace{\frac{bs\,(1-qN)\,MF}{N}}_{\text{births}} - \underbrace{\mu_M M}_{\text{mortality}}, \tag{1}$$

$$\frac{dF}{dt} = \underbrace{\frac{b\,(1-s)\,(1-qN)\,MF}{N}}_{\text{births}} - \underbrace{\mu_F F}_{\text{mortality}}, \tag{2}$$

where $N = M + F$ is the total population size; b is the baseline birth rate; s is the sex ratio at birth (proportion of offspring that are male); q is the strength of density dependence on population growth; and μ_M and μ_F are the male and female per-capita mortality rates. Like Shyu and Caswell (2016), we assume that both males and females are limiting in the birth term, and that mating occurs at a frequency-dependent rate, with births manifesting as a product of the number of males and females divided by the total number of individuals ($\frac{MF}{N}$). Note that Shyu and Caswell (2016) refer to the sex ratio at birth as the primary sex ratio, and the sex ratio of the adult population as the secondary sex ratio. Standard naming convention refers to the sex ratio at birth as the secondary sex ratio, and the sex ratio in the adult population as the adult sex ratio (Székely et al. 2014). To avoid confusion, we refer throughout to s as the sex ratio at birth.

We assume that the sex ratio at birth, s, is a heritable trait under selection, and is autosomal with equal chances of being inherited from each parent. When two individuals have different sex ratio traits, offspring are born at the average of the two sex ratios. We use the next-generation method (Hurford et al. 2010), which calculates the fitness of a rare mutant phenotype as the long-term growth rate in the resident population. When the fitness of a rare mutant is greater than 1, it can invade the resident population. Using the next-generation method (see Supporting Information), we calculate the invasion fitness of a rare mutant, $\bar{s}$, as

$$\omega(\bar{s}, s) = \frac{2s^2 + (2\bar{s} - 3)\,s - \bar{s}}{4s\,(s-1)}. \tag{3}$$

The fitness gradient is then

$$\mathcal{F}(s) = \left.\frac{\partial \omega}{\partial \bar{s}}\right|_{\bar{s}=s} = \frac{1-2s}{4s\,(1-s)}. \tag{4}$$

The population will evolve in the direction of the fitness gradient until a singular strategy (s^*) is reached at $\mathcal{F}(s^*) = 0$ (i.e., the fitness gradient is equal to zero). Clearly, the singular strategy in this model is at $s^* = 0.5$, indicating that the population will evolve toward a 1:1 sex ratio at birth, in agreement with Fisher (1930).

With stage structure and mating unions

We now consider a more detailed model including stage structure and explicit modeling of mating unions (Fig. 1A), similar to the model analyzed by Shyu and Caswell (2016). Let M_J and F_J be the male and female juvenile classes, respectively; M_A and F_A be the male and female unpaired adult classes, respectively; U be the mating union class; $N_u = M_A + F_A$ be the density of unpaired adults; $N = M_J + F_J + N_u + 2U$ be the total population density; τ be maturation rate; δ be the union formation rate; d be the union dissolution rate; and μ_{MJ}, μ_{FJ}, μ_{MA}, and μ_{FA} be the respective morality rates. The population dynamics are then given by the following set of ordinary differential equations:

$$\frac{dM_J}{dt} = \underbrace{bs\,(1-qN)\,U}_{\text{births}} - \underbrace{(\tau + \mu_{MJ})\,M_J}_{\text{aging and mortality}}, \tag{5}$$

$$\frac{dF_J}{dt} = \underbrace{b\,(1-s)\,(1-qN)\,U}_{\text{births}} - \underbrace{(\tau + \mu_{FJ})\,F_J}_{\text{aging and mortality}}, \tag{6}$$

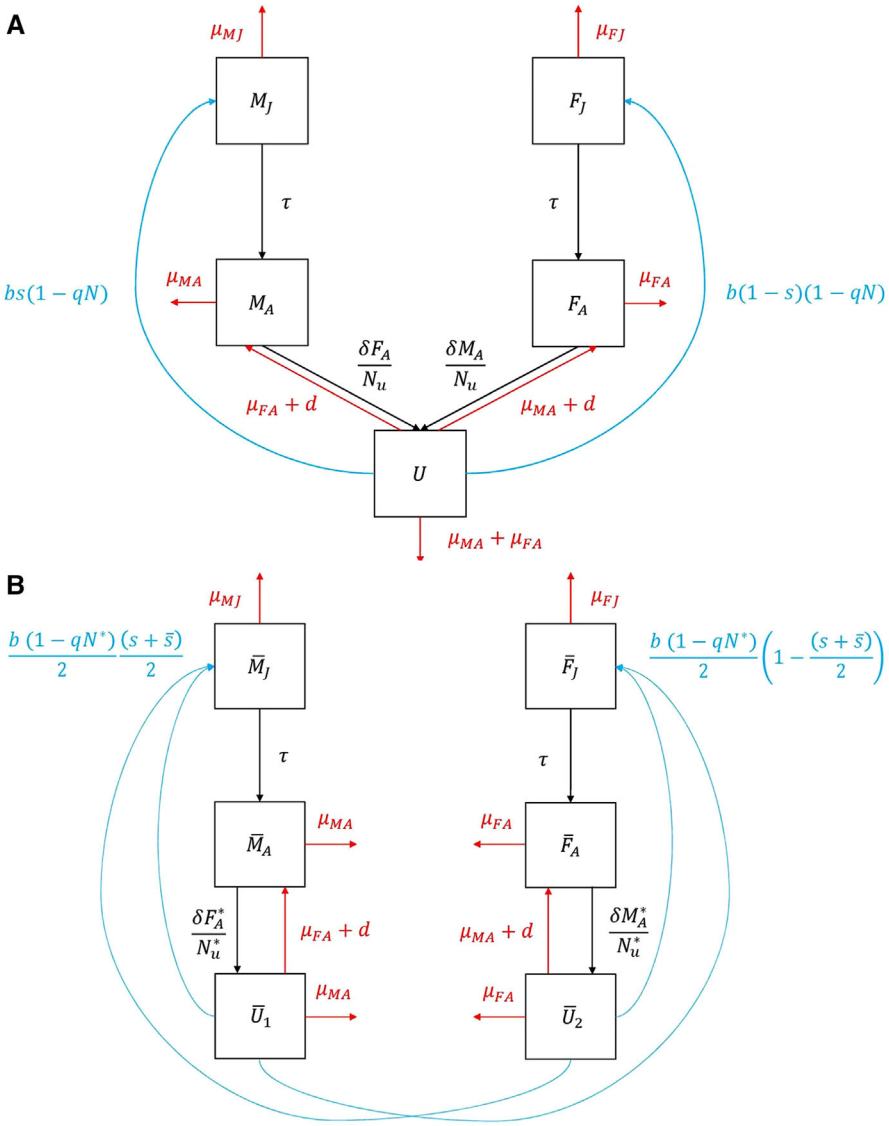

Figure 1. Transition diagrams for the model with stage structure and unions, showing the resident dynamics (A) and the rare mutant dynamics (B).

$$\frac{dM_A}{dt} = \underbrace{\tau M_J}_{\text{aging}} + \underbrace{(\mu_{FA}+d)U}_{\text{union dissolution}} - \underbrace{\mu_{MA}M_A}_{\text{mortality}} - \underbrace{\frac{\delta M_A F_A}{N_u}}_{\text{pairing}}, \tag{7}$$

$$\frac{dF_A}{dt} = \underbrace{\tau F_J}_{\text{aging}} + \underbrace{(\mu_{MA}+d)U}_{\text{union dissolution}} - \underbrace{\mu_{FA}F_A}_{\text{mortality}} - \underbrace{\frac{\delta M_A F_A}{N_u}}_{\text{pairing}}, \tag{8}$$

$$\frac{dU}{dt} = \underbrace{\frac{\delta M_A F_A}{N_u}}_{\text{pairing}} - \underbrace{(\mu_{MA}+\mu_{FA}+d)U}_{\text{mortality and divorce}}. \tag{9}$$

The first terms in equations (5) and (6) are the birth rates, which depend on the total number of unions, U, that have already formed through frequency-dependent pairing (eq. 9), whereas in the previous version of the model (eqs. 1 and 2) mating was treated as an ephemeral frequency-dependent process with no long-lasting unions. Individuals leave the juvenile classes through aging or mortality. Individuals enter the unpaired adult classes (eqs. 7 and 8) through aging or when unions end, either due to the death of a partner or when either partner leaves. Unpaired adults leave these classes through mortality or pairing to form mating unions, which occurs at a frequency-dependent rate (hence the N_u in the denominators of the pairing terms). This ensures that the pairing rate is independent of population size. Finally, unions end either due to the death of one partner or when one terminates the union.

Our model differs from the one proposed by Shyu and Caswell (2016) in three ways. First, we assume that the

population may experience density-dependent growth (when $q > 0$), which prevents unconstrained population growth and is therefore more realistic. This does not, however, qualitatively affect our results (see the Supporting Information for analysis of an exponentially growing population when $q = 0$). The other differences concern the mutant invasion dynamics (Fig. 1B), which are given by

$$\frac{d\bar{M}_J}{dt} = \underbrace{\frac{b(1-qN^*)}{2}\left(\frac{s+\bar{s}}{2}\right)\left(\bar{U}_1+\bar{U}_2\right)}_{\text{births}} - \underbrace{(\tau+\mu_{MJ})\bar{M}_J}_{\text{aging and mortality}}, \tag{10}$$

$$\frac{d\bar{F}_J}{dt} = \underbrace{\frac{b(1-qN^*)}{2}\left(1-\frac{s+\bar{s}}{2}\right)\left(\bar{U}_1+\bar{U}_2\right)}_{\text{births}} - \underbrace{(\tau+\mu_{FJ})\bar{F}_J}_{\text{aging and mortality}}, \tag{11}$$

$$\frac{d\bar{M}_A}{dt} = \underbrace{\tau\bar{M}_J}_{\text{aging}} - \underbrace{\mu_{MA}\bar{M}_A}_{\text{mortality}} - \underbrace{\frac{\delta\bar{M}_A F_A^*}{N_u^*}}_{\text{pairing}} + \underbrace{(\mu_{FA}+d)\bar{U}_1}_{\text{union dissolution}}, \tag{12}$$

$$\frac{d\bar{F}_A}{dt} = \underbrace{\tau\bar{F}_J}_{\text{aging}} - \underbrace{\mu_{FA}\bar{F}_A}_{\text{mortality}} - \underbrace{\frac{\delta M_A^*\bar{F}_A}{N_u^*}}_{\text{pairing}} + \underbrace{(\mu_{MA}+d)\bar{U}_2}_{\text{union dissolution}}, \tag{13}$$

$$\frac{d\bar{U}_1}{dt} = \underbrace{\frac{\delta\bar{M}_A F_A^*}{N_u^*}}_{\text{pairing}} - \underbrace{(\mu_{MA}+\mu_{FA}+d)\bar{U}_1}_{\text{mortality and divorce}}, \tag{14}$$

$$\frac{d\bar{U}_2}{dt} = \underbrace{\frac{\delta M_A^*\bar{F}_A}{N_u^*}}_{\text{pairing}} - \underbrace{(\mu_{MA}+\mu_{FA}+d)\bar{U}_2}_{\text{mortality and divorce}}, \tag{15}$$

where asterisks indicate the resident population at equilibrium; $\bar{M}_i$ and $\bar{F}_i$ represent the unpaired mutant classes; $\bar{U}_1$ represents unions between mutant males and resident females; and $\bar{U}_2$ represents unions between resident males and mutant females. Crucially, the mutant dynamics differ by modeling the dynamics of mutant males with resident females ($\bar{U}_1$) and resident males with mutant females ($\bar{U}_2$) separately (Fig. 1B), whereas Shyu and Caswell (2016) only have a single mutant union class. The importance of this difference can be seen in the transition diagram (Fig. 1B) where there is always feedback from the mutant union classes into the correct mutant population classes, which is not the case in Shyu and Caswell (2016) (see *Discussion*). Note that due to the initial scarcity of mutants in the population, interactions between mutants are assumed to be negligible (hence there are no mutant-mutant unions). Mutants are born at a rate $\frac{b(1-qN^*)}{2}(\bar{U}_1+\bar{U}_2)$, where the factor of $\frac{1}{2}$ represents equal probability of inheritance from either parent in a mutant-resident union. The other notable difference in our model is seen in the birth terms present in equations (10) and (11), where we average the resident and mutant sex ratios to give the actual sex ratio at birth arising from resident-mutant unions (i.e., the realized sex ratio at birth, $\frac{s+\bar{s}}{2}$, is additive based on the parents' traits). In Shyu and Caswell (2016), the sex ratio at birth for mutant individuals is always determined by the mutant (i.e., the mutant allele is always dominant), creating a bias toward the mutant sex ratio. The "resident" and "mutant" labels only refer to the frequency of each phenotype in the population and so they are interchangeable. Hence, there is no biological reason as to why a mutant allele should be dominant simply because it is rare.

Again, we use the next-generation method (see Supporting Information) to calculate the invasion fitness of a rare mutant, $\bar{s}$:

$$\begin{aligned}\omega(\bar{s},s) = {} & \zeta N_u^*(d+\mu_{MA}+\mu_{FA})\left(\mu_{FA}F_A^*(\tau+\mu_{FJ})(s+\bar{s})\right.\\ & \left.+\mu_{MA}M_A^*(\tau+\mu_{MJ})(2-s-\bar{s})\right)\\ & +\zeta\delta M_A^*F_A^*(\mu_{MA}(2-s-\bar{s})(\tau+\mu_{MJ})\\ & +\mu_{FA}(s+\bar{s})(\tau+\mu_{FJ})),\end{aligned} \tag{16}$$

where $\zeta = \frac{b\delta\tau(1-qN^*)}{4\mu_{MA}\mu_{FA}(\tau+\mu_{MJ})(\tau+\mu_{FJ})(\delta M_A^*+(d+\mu_{MA}+\mu_{FA})N_u^*)(\delta F_A^*+(d+\mu_{MA}+\mu_{FA})N_u^*)}$ for notational convenience. The fitness gradient, $\mathcal{F}(s) = \frac{\partial\omega}{\partial\bar{s}}|_{\bar{s}=s}$, is then

$$\begin{aligned}\mathcal{F}(s) = {} & \zeta N_u^*(d+\mu_{MA}+\mu_{FA})\left(\mu_{FA}F_A^*(\tau+\mu_{FJ})\right.\\ & \left.-\mu_{MA}M_A^*(\tau+\mu_{MJ})\right)+\zeta\delta M_A^*F_A^*\ (\mu_{FA}(\tau+\mu_{FJ})\\ & -\mu_{MA}(\tau+\mu_{MJ})).\end{aligned} \tag{17}$$

Note that the sex ratio does not explicitly appear in the fitness gradient, but the population equilibrium depends on the sex ratio, and hence the fitness gradient also depends on the sex ratio. It is not possible to find the nontrivial equilibrium of this model analytically, so we therefore analyze the fitness gradient numerically to determine whether differential mortality at the juvenile and adult stages affects the singular strategy, s^*.

We find that the singular strategy always occurs at $s^* = 0.5$ (provided the population is viable). The point $s^* = 0.5$ is always a fitness maximum and so the population therefore evolves to a 1:1 sex ratio at birth, independent of juvenile and adult mortality (Fig. 2). Moreover, the fitness gradient does not vary with differential juvenile or adult mortality between the sexes, and does not depend on whether population growth is density-dependent or exponential (see Supporting Information). Because our model has no period of parental investment, our results confirm the classical Fisherian view (Fisher 1930; Leigh 1970): all else being equal, differential mortality between the sexes after parental investment does not affect the evolution of sex ratio at birth.

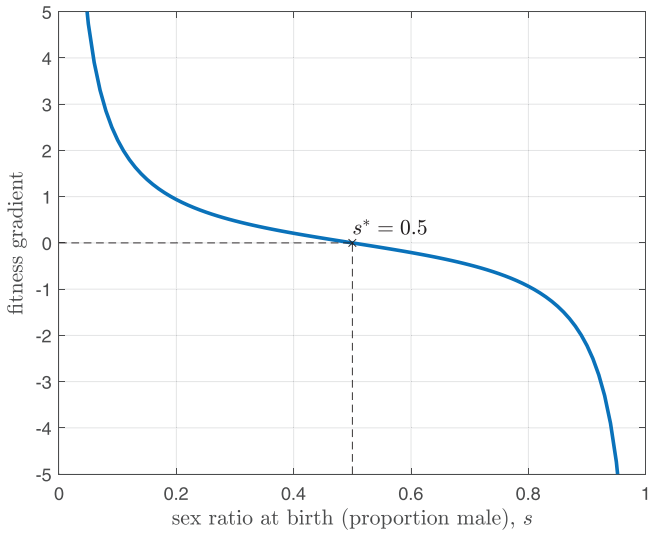

Figure 2. The fitness gradient ($\mathcal{F}(s)$) plotted as a function of sex ratio at birth (s; proportion male), using the model with stage structure and unions. Note that the sex ratio at birth (proportion male) will increase over evolutionary timescales when the fitness gradient is positive and will decrease when it is negative. The singular strategy, where the fitness gradient equals zero, always occurs at a 1:1 sex ratio, $s^* = 0.5$, regardless of the model parameters (provided the population is viable). Moreover, the fitness gradient does not vary with juvenile or adult mortality in either sex.

Discussion

Our study was motivated by the apparent contradiction between the classical Fisherian view of sex ratio evolution and the recent study by Shyu and Caswell (2016). We have shown that sex- and stage-specific mortalities after parental investment do not affect sex ratio evolution, in agreement with Fisher (1930) and in contrast with Shyu and Caswell (2016).

Although our model includes density dependence on population growth, this is not the cause of the discrepancy, as shown by our analytical derivation (see Supporting Information S3) and stochastic simulations of exponentially growing populations (Fig. S1). Instead, there appear to be two flaws in how Shyu and Caswell (2016) define the mutant population dynamics. First, Shyu and Caswell (2016) assume that mutant-resident unions always produce offspring at the mutant sex ratio, despite only one parent having the mutant sex ratio phenotype. The assumption of a dominant mutant allele introduces a bias toward the mutant sex ratio, but because "resident" and "mutant" are just labels indicating the respective frequency of the two phenotypes in the population, assuming dominance of the mutant phenotype is an unusual assumption. We are unaware of any biological mechanism by which dominant/recessive phenotypes are dependent on the relative frequencies of those phenotypes. We instead assumed that mutant-resident unions produce offspring at the average of the parental sex ratio (i.e., realized sex ratio at birth is additive based on both parent's traits). Second, and most importantly, Shyu and Caswell (2016) only have a single class for mutant-resident unions, consisting of mutant male-resident female pairs, and mutant female-resident male pairs. The disbanding of a mutant-resident union, either through divorce or mortality, therefore causes a counting error as it is impossible to determine whether the union consists of a mutant male or female. To illustrate this problem, consider the case where there are initially no mutant females in the population. Mutant adult males can form partnerships with resident adult females, causing them to move into the mutant-resident union class. Suppose now that the mutant adult male in the union dies, leaving a resident adult female behind. Instead, a mutant female is produced. We avoid this problem by modeling the two types of mutant-resident unions separately (mutant males with resident females, and resident males with mutant females). Incorrect counting of rare mutants has previously been shown to generate a frequency-dependent advantage in evolutionary models of infectious diseases with coinfections (Alizon 2013). We suggest that a similar problem may exist in the model analyzed by Shyu and Caswell (2016), which emphasizes the importance of carefully defining mutant dynamics to avoid unintended frequency-dependent advantage.

In conclusion, although a variety of mechanisms have been shown to cause a departure from equal sex ratios at birth (Hamilton 1967; Trivers and Willard 1973; Werren and Charnov 1978), stage structure and mating unions do not. Our model—which corrects the flaws in Shyu and Caswell (2016)—shows no deviation from a 1:1 sex ratio at birth due to differential mortality between the sexes following parental investment, thus confirming the classical Fisherian view of sex ratio evolution.

AUTHOR CONTRIBUTIONS

AP and BA conceived the study and wrote the manuscript. AP carried out the modeling work.

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DATA ARCHIVING

Source code is available in the Supporting Information and in the following GitHub repository: https://github.com/ecoevogroup/Pirrie_and_Ashby_2021.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Supplementary information