Half as high for twice as long: male bias in the fertile-age sex ratio

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Abstract

Humans are distinguished from our closest living relatives, other great apes, by our extended postmenopausal longevity, later first births, and shorter birth intervals. Those features likely evolved as ancestral grandmothers' foraging subsidised dependants in habitats lacking foods that youngsters could manage for themselves. If so, as female post-fertile years increased, older years increased in males too. Those still-fertile old males in the paternity competition pushed average male reproductive success below the female average. As R.A. Fisher explained, Mendelian inheritance requires equal contributions from both sexes to descendant gene pools. Higher lifetime reproductive success in one sex makes tendencies to overproduce it pass to more grandchildren, equalising averages in descendant generations. Yet, men's lower per-capita per-annum reproductive success persists, their fertility lasting decades longer than women's. Here, we present a simple mathematical model to investigate this phenomenon. We show that a male-biased adult sex ratio is consistent with an offspring sex ratio of 1:1. Assuming a stable age distribution, we show that if male fertile careers are twice as long as those of females, the extended male fertility soon balances the higher female average, maintaining Fisher's equilibrium under human life history conditions.

Keywords: Fisher's principle, overlapping generations, reproductive value, sex ratio evolution, diploidy, life history evolution.

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1 Introduction

Hamilton's rule is a simple inequality that W. D. Hamilton introduced to explain why altruism need not be a puzzle for natural selection [1]. From a gene's-eye view of its phenotype's social behaviour, the benefit supplied to another individual's fitness at a cost to its own will make more copies of the gene itself if $rb > c$, where r is the chance that a copy of the same gene is in the recipient; b is the direct fitness benefit to the recipient; and c is the cost of the behaviour to the donor's own direct fitness. With sexual reproduction, diploid offspring get half their genes from each parent making the chance a rare gene identical by descent is in an offspring ($r = 0.5$) twice the chance it is in a grandoffspring ($r = 0.25$).

Here, we apply Hamilton's rule to explore how our postmenopausal longevity may have shaped ancestral male mating strategies. The evolution of our grandmothering life history [2, 3, 4] can explain why human populations are biased toward males in the fertile ages [5, 6, 7, 8, 9]. With that male bias, unusual for a mammal, mate-guarding wins more paternities than multiple mating [10, 11, 12, 13, 14, 15]. If deference to older males' claims raise average paternal ages above average maternal ages, then generational steps from fathers put more genes from previous generations into generations produced by younger mothers.

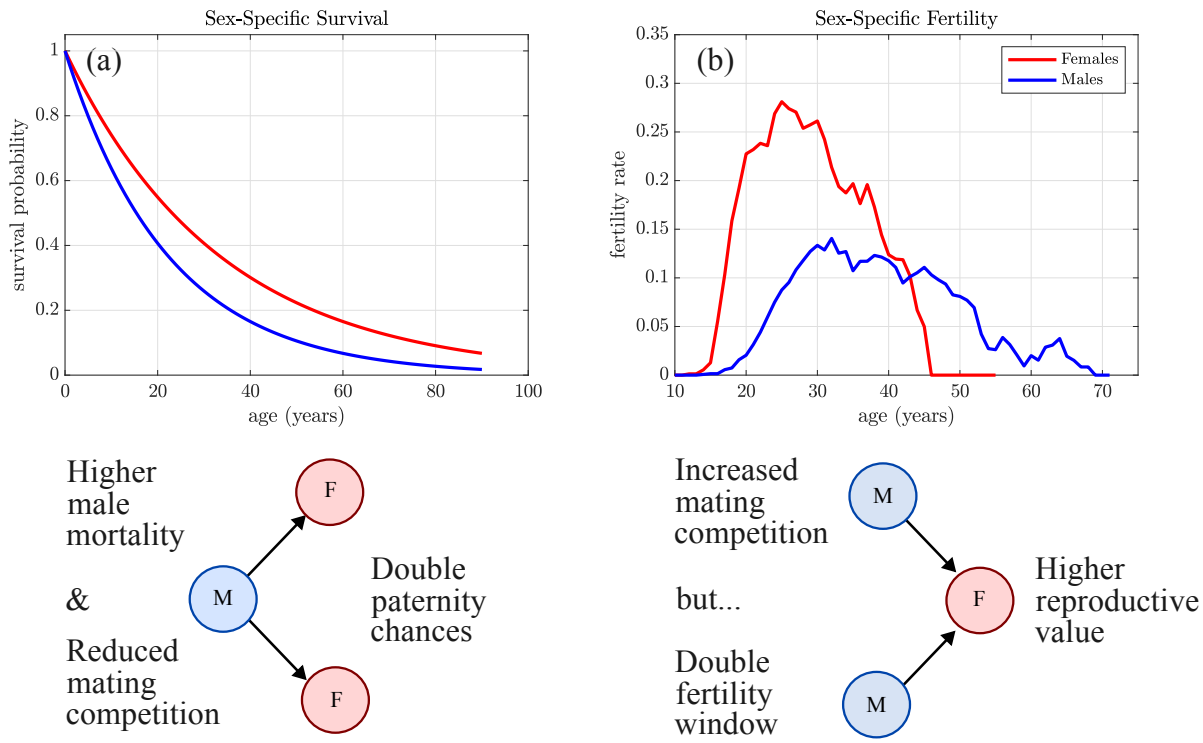

Figure 1: Sex-biased adult populations and reproductive consequences. Illustrations depict two demographic scenarios. (**a**) In a female-biased population, elevated male mortality means that surviving males have increased paternity opportunities, leading to decreased mating competition and more paternity chances over the long run. (**b**) In a male-biased population, intensified male competition reduces male reproductive success, increasing the fitness value of sons. Both scenarios reflect frequency-dependent selection consistent with Fisher's principle: reproductive value adjusts to maintain equal genetic contribution from males and females to subsequent generations, even in the presence of age-specific mortality or fertility asymmetries.

To clarify this and its consequences, we consider an extreme example. Assume that the fertile ages of males are twice as long as those of females. Modelling results resonate with a thought

experiment suggested by Alan Grafen [16], who proposed looking back at a gene's history from far in the future as it jumps back into a mother or a father at each generation. He showed that the reproductive value of individuals of any given age, defined by Fisher as their average future fertility, when totalled for all males and females depends on how old an average new mother and father are. If the evolution of postmenopausal longevity made deference to older males' mate-guarding more prevalent, then the foraging contributions of ancestral grandmothers may have facilitated conditions conducive to patriarchy.

It has long been recognised that sex differences in adult mortality need not alter Fisher's argument about equal investment in offspring of both sexes. If adult sons are twice as likely to die as adult daughters, then the sons that survive have double the paternity chances (see Figure 1(a)). Here, we investigate whether the age-specific fertility difference between the sexes in humans might balance in the same way (Figure 1(b))[17]. We show that the persistence of the male-biased sex ratio in human fertile ages is consistent with Fisher's theory and leads reproductive values to be higher at older ages in males than in females. Males continue to contribute offspring to the grandoffspring generation for females. Then, the number of aggregate replicas through sons begins to catch up to the number through daughters.

2 Background

R. A. Fisher, G. C. Williams, W. D. Hamilton and J. Maynard Smith, developed much of evolutionary life history theory. Williams showed why sexual reproduction is such a puzzle in his book *Sex & Evolution* [18] with an exposition that parallels Hamilton's rule. Meiosis is the reduction of diploid cells into the haploid gametes of sexual reproducers. Syngamy of two haploid gametes is required to form a diploid zygote, so offspring of a sexually reproducing mother carry only half her genes ($r = 0.5$). If she reproduced asexually, by cloning or budding, offspring would have the full complement ($r = 1$). The two different gamete types, one small and plentiful, the other large and few required for sexual reproduction make anisogamy the beginning of what has been called the sexual cascade to sexual selection, sex roles, and sexual conflict [19]. Later, Goodman [20] clarified how life-history questions can be expressed in formal demographic terms. He showed how reproductive value and age-structured population models can be used to measure fitness consistently and to compare alternative life-history strategies within the same mathematical framework.

Darwin had recognised the one-mother-one-father axiom for sexual reproduction [21]; however, it was Cobb[22] and Fisher [23], combining natural selection with Mendelian inheritance, who saw that each sex contributes half the autosomal genes to future generations which usually pushes natural selection to equal expenditure on sons and daughters. The important point here is that given the one-mother-one-father requirement, generally referred to as the "Fisher Principle", more of one sex in randomly mating diploids makes the average reproductive success of the more abundant sex lower than the average of the rarer one, so tendencies to bias offspring toward the rarer sex occur in more grandchildren. That rare sex advantage, first modelled by Karl Düsing [24], is widely considered the first identification of frequency-dependent selection [25].

More generally, rare sex advantage explains why there are so many males, even when–as in most primates–the number of babies depends only on the number of fertile females. If males are rare and one or a few males can fertilise all the females almost simultaneously, the average success of rare males must be much higher than the female average. Tendencies to bias offspring toward the rare males would be more common in the grandoffspring generation, reducing males' rarity. An equilibrium is reached when the average reproductive success of

the two sexes is equal, demonstrating an evolutionarily stable strategy (ESS).

We explore the possibility that inclusive fitness consequences of deference to older males' mate-guarding could balance a sex difference in average reproductive success with an extreme simplification. Assume that average reproductive success is half as high for twice as long in males as in females. Then, sons average half the reproductive success of daughters, but with fertile careers twice as long, they put more genes into future generations than do daughters.

This extreme example shows that the number of accumulated replicas through sons begins to catch up to the number through daughters. While a hypothetical offspring gene gets only half the replicas through sons as through daughters each generation at time t, it has double the chance of putting copies in the next generation at time $t + 1$. A gene is being favoured by natural selection if the aggregate of its replicas forms an increasing fraction of the total gene pool [26]. Population genetic models of senescence have shown that the selection pressure on survival is directly a function of reproductive value at any age [27]. However, these models make use of Fisher's explicit definition of reproductive value that leaves out the contributions of still-productive post-fertile females [17]. Fisher showed that when age-specific fertility and mortality remain constant and migration is negligible, populations attain a stable age structure in which one can compute the direct contribution from each age class to future gene pools by combining survival and fertility rates across generations. Although he briefly acknowledged a "...mother past bearing age may greatly promote the reproduction of her children" [23], he treated such indirect kin effects as marginal. By ignoring those indirect effects, he could take advantage of the renewal equation, which attends only to age-specific fertility and mortality. These are the effects that Hamilton would later elaborate and formalise [1].

Careful collection and analysis of demographic data for modern human foragers living under mortality regimes, more like those that prevailed through most of human experience, show that post-fertile women are not rare [28, 29, 30, 31]. Instead, females that survive to adulthood are much more likely to live beyond their fertility window. Human age structures include substantial fractions of post-fertile women. Thus, although postmenopausal women do not produce offspring, they must indeed be characterised by a *genetic* reproductive value not too different from that of males [32]. They also include substantial fractions of old men who are still fertile. Some out-compete younger men for paternities. The strong male bias of the sex ratio in the fertile ages which makes that possible distinguishes humans not only from the other great apes [33], but from most other mammals. This is illustrated for several modern hunter-gatherer populations in Figure 2.

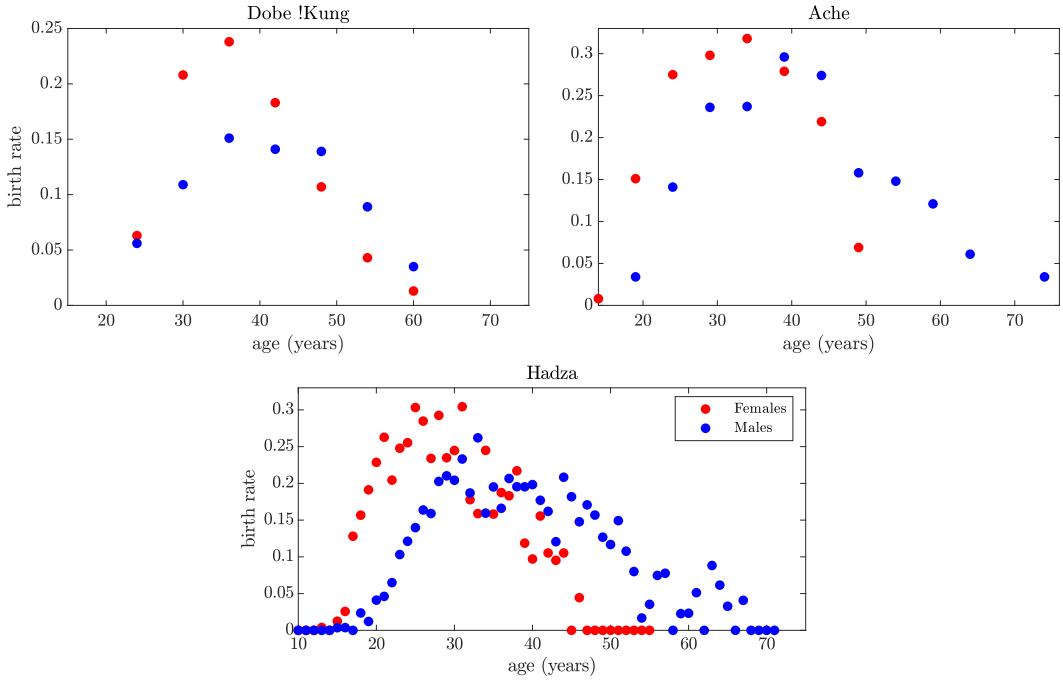

Figure 2: Age-specific fertility for women and men in three modern hunter-gatherer societies. Scatter plots show empirical estimates of age-specific fertility for women (red) and men (blue) among the Dobe !Kung [28] (top left), Ache [29] (top right), and Hadza [31] (bottom center). Female fertility data exhibit a narrower reproductive span, typically peaking in the late twenties and declining steeply by age 45. Male fertility spans a broader age range and averages decline more gradually, particularly in the Ache and Hadza. All data are derived from published demographic reconstructions and are plotted as observed. These distributions illustrate persistent sex differences in fertility timing and duration, which underpin the sex-specific structure of reproductive value in later analyses.

In humans, as is common in mammals generally [34, 35], mortality is higher in males. As noted above, higher male mortality in early life has been widely understood to imply that the slight male bias in human birth sex ratios regularly found in large samples is part of an adjustment from a higher male bias at conception with early male biased mortality allowing replacements that result in equal expenditure on sons and daughters as predicted by Fisher's account. However, a recent empirical study showed that an equal sex ratio at conception holds, contradicting the common claim that the sex ratio at conception is male-biased [36, 37].

However, our concern here is what happens in adult ages. Higher levels of adult mortality in males are widely known, and sufficiently remarked in humans to pose a "health-survival paradox" [38, 17]. However, it has long been recognised that sex differences in adult mortality need not alter Fisher's argument about equal investment in offspring of both sexes. If adult sons are twice as likely to die as adult daughters, then there are double the paternity chances for surviving sons. One of us [17] wondered whether the age-specific fertility difference between the sexes in humans might somehow cancel in the same way. Here, we address that question, explaining why it might. In the next section, we develop mathematical models that help us explore this possibility.

3 Model

Models of sex-ratio evolution fall into two distinct, yet complementary frameworks. The classic framework is based on expected future fitness; it evaluates sex-allocation strategies by predicting how often selectively neutral genes carried by a parent–one that produces a given sex ratio–will appear in future generations. The other framework utilises dynamical population-genetic models to determine under what conditions an allele that determines a specific parental sex ratio phenotype can invade against an allele that determines a different phenotype [39]. There is no inherently superior framework, the choice depends on personal preference and the specifics of the problem [40]. Here, we use the classic approach by considering population-level effects of the sex ratio phenotype.

We construct two models to investigate our extreme example. The first is a two-sex Leslie matrix model to demonstrate that the age-specific fertility differences between the sexes is consistent with a balanced offspring sex ratio. Then, we develop a Markov chain model that allows us to trace the lineage of a hypothetical gene back from a distant future generation to the present time. With the long-term behaviour of this model, we show that under our simplified assumptions, the expected future fertility of males is higher than that of females and that this remains compatible with an equal offspring sex ratio, satisfying Fisher's condition.

3.1 Leslie matrix model

In this section, we consider a diploid population in discrete time and introduce a two-sex Leslie matrix model. Using this model, we show that an equal offspring sex ratio is the optimal evolutionary strategy even when the age-specific fertility difference between the sexes is male biased (males have a longer fertility window).

Table 1 provides a summary of parameters and population variables used in our model.

Variable	Description
F_i	Number of females in the age class i
M_i	Number of males in the age class i
$s_{f,i}$	Fraction of females who survive from age class i to $i+1$
$s_{m,i}$	Fraction of males who survive from age class i to $i+1$
$l_{f,i}$	Probability of females surviving to age i
$l_{m,i}$	Probability of males surviving to age i
f_i	Per capita female fecundity
f_m	Per capita male fecundity

Table 1: Parameters and Population Variables

Consider a population of females and males divided into ω age groups, with the number in each group at time t given by the vector

$$\mathbf{P}_t = (F_{0t}, \ldots, F_{(\omega-1)t}, M_{0t}, \ldots, M_{(\omega-1)t})^T ,$$

where superscript T denotes the transpose. The change in population structure from time t to $t+1$ is then represented by the equation

$$\mathbf{P}_t = \mathbf{L}\mathbf{P}_{t-1} , \tag{1}$$

where $\mathbf{L}$ is a two-sex Leslie matrix in which f_i are the fecundities of mothers, $s_{f,i}$ are the fractions of females, and $s_{m,i}$ the fractions of males who survive from age class i to $i+1$. A more explicit description of this model is shown in equation 2.

$$\begin{bmatrix} F_0 \\ F_1 \\ \vdots \\ F_{\omega-2} \\ F_{\omega-1} \\ M_0 \\ M_1 \\ \vdots \\ M_{\omega-2} \\ M_{\omega-1} \end{bmatrix}_t = \left[\begin{array}{ccccc|ccccccc} f_0 & f_1 & \cdots & f_{\omega-2} & f_{\omega-1} & 0 & 0 & \cdots & 0 & 0 & 0 & 0 \\ s_{f,0} & 0 & \cdots & 0 & 0 & 0 & 0 & \cdots & 0 & 0 & 0 & 0 \\ 0 & s_{f,1} & \cdots & 0 & 0 & 0 & 0 & \cdots & 0 & 0 & 0 & 0 \\ \vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \vdots & \vdots & \ddots & \vdots & \vdots & \vdots \\ 0 & 0 & \cdots & s_{f,\omega-2} & 0 & 0 & 0 & \cdots & 0 & 0 & 0 & 0 \\ \hline f_0 & f_1 & \cdots & f_{\omega-2} & f_{\omega-1} & 0 & 0 & \cdots & & 0 & & 0 \\ 0 & \cdots & 0 & 0 & 0 & s_{m,0} & 0 & \cdots & & 0 & & 0 \\ 0 & \cdots & 0 & 0 & 0 & 0 & s_{m,1} & \cdots & & 0 & & 0 \\ \vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \vdots & \ddots & & \vdots & & \vdots \\ 0 & \cdots & 0 & 0 & 0 & 0 & 0 & \cdots & & s_{m,\omega-2} & & 0 \end{array}\right] \begin{bmatrix} F_0 \\ F_1 \\ \vdots \\ F_{\omega-2} \\ F_{\omega-1} \\ M_0 \\ M_1 \\ \vdots \\ M_{\omega-2} \\ M_{\omega-1} \end{bmatrix}_{t-1} . \tag{2}$$

Note that the $\omega \times \omega$ matrix in the upper left corner of $\mathbf{L}$ is the standard Leslie matrix model for a female population. The lower ω rows of the matrix account for the survival probabilities and births of new males from the female population. We assume a stationary population with no growth, so $\lambda = 1$. Let $l_{f,i}$ be the probability a female survives multiple age classes up through age class i, then $l_{f,i} = s_{f,0} \cdot s_{f,1} \cdots s_{f,i-1}$ for $i \geq 1$. Each female is born with certainty, so $l_{f,0} = 1$. Thus, the female parameters satisfy the Euler-Lotka equation

$$\sum_{i=0}^{\omega-1} l_{f,i} f_i = 1 \,. \tag{3}$$

This means that the expected lifetime reproductive success of a newborn individual is one and each individual exactly replaces itself. Additionally, we assume a balanced offspring sex ratio so that sons and daughters are produced in equal numbers from the same fertility distribution. Thus, the fecundity terms in rows 1 and $\omega + 1$ of L are symmetric.

Let

$$\mathbf{P}_* = (1,\, l_{f,1}, \cdots, l_{f,\omega-1}, 1,\, l_{m,1}, \cdots, l_{m,\omega-1})^T \,, \tag{4}$$

where $l_{f,0} = 1$, $l_{f,i} = s_{f,0} s_{f,1} \cdots s_{f,i-1}$, $l_{m,0} = 1$ and $l_{m,i} = s_{m,0} s_{m,1} \cdots s_{m,i-1}$ for $i \geq 1$. $\mathbf{P}_*$ is the stable age distribution, that is, it is an eigenvector of the two-sex Leslie matrix corresponding to the eigenvalue 1. Hence, any age distribution of the population will be proportional to $\mathbf{P}_*$.

For simplicity, we assume all surviving males are able to compete for paternities and do so with equal chance. In addition, assume the population has reached a stable age distribution. This means that the age-specific fertility is constant and males have equal chance of mating with every female. Thus, we can define ρ, the ratio of males competing for females who are able to conceive in the next time step (the adult sex ratio, ASR) as:

$$\rho = \frac{\sum_{i=0}^{\omega-1} l_{m,i}}{\sum_{i=0}^{\omega-1} l_{f,i} f_i} \,. \tag{5}$$

The denominator of this ratio is 1 from Eq. 3, so it becomes

$$\rho = \sum_{i=0}^{\omega-1} l_{m,i} \,. \tag{6}$$

We wish to construct a modified Leslie matrix to track how many descendants come from males and females. Using the assumption that all surviving males compete for paternities with equal chance, the fecundity of each male is

$$f_m = \frac{1}{\rho}. \tag{7}$$

Inserting this into the two-sex Leslie matrix above (Equation 2) to account for paternities and assuming an offspring sex ratio of 1:1, the modified Leslie matrix, K, that calculates how genetic contributions pass from one generation to the next is

$$K = \left[\begin{array}{ccccc|ccccc}
\frac{1}{2}f_0 & \frac{1}{2}f_1 & \cdots & \frac{1}{2}f_{\omega-2} & \frac{1}{2}f_{\omega-1} & \frac{1}{2}f_m & \frac{1}{2}f_m & \cdots & \frac{1}{2}f_m & \frac{1}{2}f_m \\
s_{f,0} & 0 & \cdots & 0 & 0 & 0 & 0 & \cdots & 0 & 0 \\
0 & s_{f,1} & \cdots & 0 & 0 & 0 & 0 & \cdots & 0 & 0 \\
\vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \vdots & \vdots & \ddots & \vdots \\
0 & 0 & \cdots & s_{f,\omega-2} & 0 & 0 & 0 & \cdots & 0 & 0 \\
\hline
\frac{1}{2}f_0 & \frac{1}{2}f_1 & \cdots & \frac{1}{2}f_{\omega-2} & \frac{1}{2}f_{\omega-1} & \frac{1}{2}f_m & \frac{1}{2}f_m & \cdots & \frac{1}{2}f_m & \frac{1}{2}f_m \\
0 & \cdots & 0 & 0 & 0 & s_{m,0} & 0 & \cdots & 0 & 0 \\
0 & \cdots & 0 & 0 & 0 & 0 & s_{m,1} & \cdots & 0 & 0 \\
\vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \vdots & \ddots & \vdots & \vdots \\
0 & \cdots & 0 & 0 & 0 & 0 & 0 & \cdots & s_{m,\omega-2} & 0
\end{array}\right]. \tag{8}$$

The first ω elements of the first row represent the number of daughters produced by females in the current time step multiplied by a factor of $1/2$ as descendants carry half the genes of their mothers. The last ω elements of the first row are the number of daughters produced by males in the current time step. These elements are also multiplied by $1/2$ since descendants carry half the genes of their fathers. We have assumed an equal offspring sex ratio, thus the $(\omega+1)$th row, which corresponds to the number of sons produced in the current time step, is the same as the first row. K has the same stable age distribution as L, that is, they have the same eigenvalue of 1.

To determine the contribution of one newborn female to subsequent descendants at time step n, we start with the initial condition

$$\mathbf{P}_0 = (1, 0, \cdots, 0, \cdots, 0)^T \tag{9}$$

and calculate $K^n\mathbf{P}_0$, with the contribution of one newborn female to subsequent descendants. By construction, the population will approach a stable age distribution. Thus,

$$\lim_{n\to\infty} K^n\mathbf{P}_0 = c\mathbf{P}_*\,, \tag{10}$$

for some constant c. Note that over her lifetime, one newborn female will have a total of one female descendant and one male descendant. Thus, she will replace herself (we assumed a stationary population). Also, by the way we defined fecundity in terms of the sex ratio, over his lifetime, one newborn male will also have a total of one female and one male descendant.

Since the property of self-replacement is invariant, i.e., future descendants will also replace themselves, both initial conditions of one newborn female and one newborn male will approach the stable age distribution $c\mathbf{P}_*$ with

$$c = \left[\sum_{i=0}^{\omega-1} \left(f_i \sum_{j=i}^{\omega-1} \ell_{f,j} \right) + f_m \sum_{i=0}^{\omega-1} \sum_{j=i}^{\omega-1} \ell_{m,j} \right]^{-1}, \tag{11}$$

where c is the reciprocal of the total lifetime number of descendants produced by the initial population vector $\mathbf{P}_0$. Thus, whether you start with one newborn male or one newborn female, the long-term reproductive output converges to the same stable age distribution. This means that each will have the same long-term contribution to future offspring, which implies that an equal offspring sex ratio is the optimal evolutionary strategy. Hence, an offspring sex ratio of 1:1 is consistent with Fisher's principle. An illustration of this is shown in Figure 3 with a numerical simulation in which an equally weighted carrying capacity is implemented. Under this regime, when the carrying capacity is achieved, individuals are removed from the population with equal probability from all age classes.

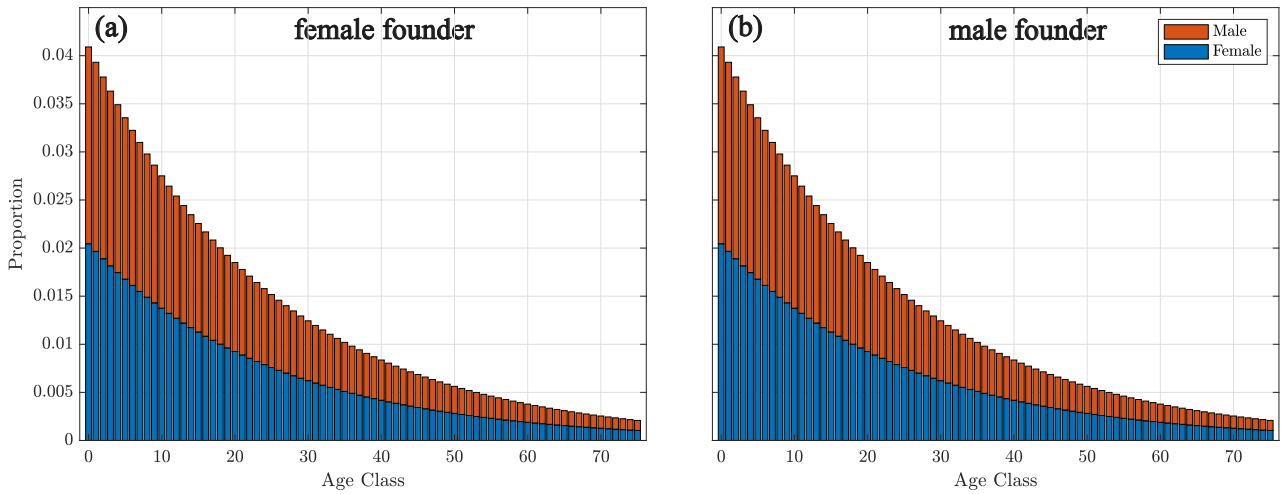

Figure 3: Stable age distributions produced by the two-sex Leslie matrix model under sex-specific founder conditions. Each subplot displays the normalized age structure at $t = 500$ following initialization with a single newborn individual of either a female founder (a) or a male founder (b). Stacked bars represent the proportion of females and males in each age class. The model uses a $2\omega \times 2\omega$ Leslie matrix, incorporating sex-specific survival rates, s_f and s_m, a constant per capita female fecundity (f_i) from ages 15–45, and constant male fertility (f_m) extending from ages 15–75. For convenience, we set $s_f = s_m = 0.98$, $f_i = 0.2$ for $i \geq 1$, and $f_m = 0.1$. Note that both males and females will have the same long-term contribution to future offspring, implying an equal offspring sex ratio is the optimal strategy.

3.2 Markov Chain Model

Here, we consider the trajectory of a gene backward in time and show that reproductive values of all males and of all females depend on the age of an average new father and new mother. Following a thought experiment suggest by Alan Grafen [16], we look back at a gene's history from far in the future as it jumps back into a mother or father in each generation. He pointed out that although non-overlapping generations ensure equal reproductive value of females and males in each generation, overlapping generations will cause differences to arise between them. In this work, we assume an idealised situation in which males have a fertility window that is exactly double that of females. To investigate the consequences of this scenario, consider a simplified model in which females are fertile for one time step and reproduce with probability 1. This allows for mathematical simplicity and greater clarity. Males are fertile for two generations and all males have an equal chance to compete for a paternity.

We also assume that there is an equal offspring sex ratio and the population is stationary, so in each time step a female will give birth to one son and one daughter. An illustration of this is shown in Figure 4 for discrete time steps t and $t+1$.

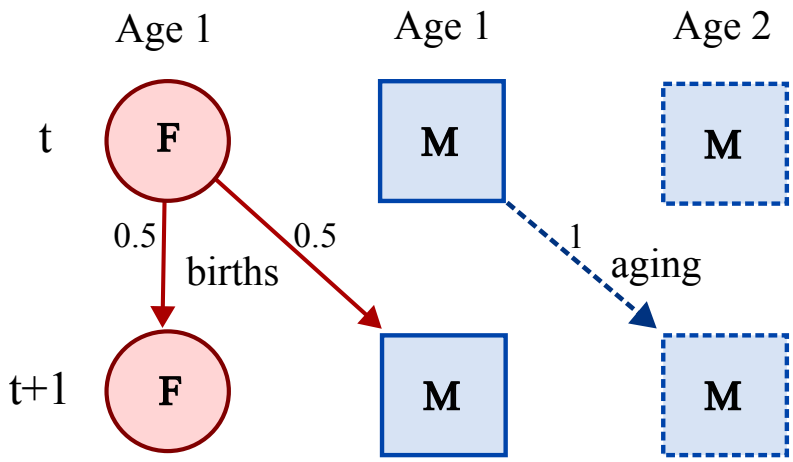

Figure 4: Simplified age-structured population under extended male fertility. A conceptual model of a stable diploid population in which females are fertile for one age class and males for two. At each discrete time step, one female of age 1 and two males (ages 1 and 2) comprise the reproductively active population. Numbers on the arrows represent probabilities.

Following [16], we define the probability that a new parent is of age $a \in \{1, 2, 3, \ldots\}$ and sex $s \in \{m, f\}$ at time t as p_{tsa}. Since we assume a diploid dioecious population and all females are fertile and males eligible for a paternity,

$$p_{tf1} = 1 \qquad p_{tfa} = 0 \quad (\text{for all } a > 1) \tag{12}$$

$$p_{tm1} = 0.5\,,\ p_{tm2} = 0.5 \qquad p_{tma} = 0 \quad (\text{for all } a > 2)\,. \tag{13}$$

Additionally, we define h_t as the chance an ancestral path moves between individuals at time t. In other words, this is the fraction of paths at time t that transition from a parent to a newborn. Since we assume a stable age distribution, h_t is the same for all t, which is an explicit calculation of this quantity.

Figure 5 illustrates the path a single gene may take starting from time T back to time $T-1$. The ancestral path passing through a female or male of age 1 has a 50% chance of transitioning back to a female parent at the previous time step, a 25% chance of transitioning back to a male parent of age 1, and a 25% chance of transitioning back to a male of age 2 (this idealised assumption of equal chance for both old and young males is further explored in the appendix). A male of age 2 has probability of 1 of transitioning back to his younger self, a male of age 1.

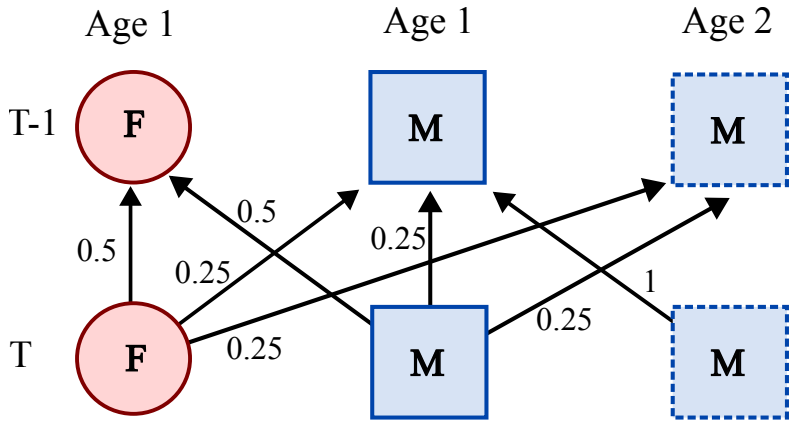

Figure 5: Ancestral pathways in a backward-time Markov model. Diagram showing the probabilistic paths of a single autosomal gene tracing back one generation from a future time T. Probabilities on arrows reflect the frequency with which a gene is expected to pass through individuals of each sex and age, supporting the inference of sex-specific reproductive contributions over time.

The probabilities of Figure 5 define a transition matrix for the Markov chain

$$P = \begin{pmatrix} 0.5 & 0.5 & 0 \\ 0.25 & 0.25 & 1 \\ 0.25 & 0.25 & 0 \end{pmatrix} , \tag{14}$$

where each column corresponds to the transitions for a gene copy currently carried by a female, an age-1 male, or an age-2 male in generation T. This conveniently allows us to determine the long-term stationary distribution of the system. Each step backward is represented by an application of this matrix. Thus, if $\mathbf{x}$ is the initial distribution of types at some point in the future, then k steps back to the present time can be written as $\mathbf{x} = P^k\mathbf{x}$. Following this logic, when k is large we reach a stable equilibrium of the system,

$$\pi = \begin{pmatrix} 0.4 \\ 0.4 \\ 0.2 \end{pmatrix} . \tag{15}$$

This means that at the stable age distribution, 40% of the paths pass through a female of age 1, 40% pass through a male of age 1, and 20% through a male of age 2. Therefore, 80% of paths involve a transition between individuals, and 20% a transition within a male of age 1 to age 2. Hence, $h_t = 0.8$.

Next, define u_{tsa} as the probability that an autosomal gene's unique ancestral path passes to an offspring from an individual of a given age and sex at time t. In order for this to happen, the ancestral path must move between individuals (not due to aging) and additionally, there must be a new available parent of the correct age, a, and sex, s, at time t. Thus, since h_t is the chance that the path moves between individuals and p_{tsa} is the probability that a specific parent is available, we can define

$$u_{tsa} = \frac{h_t}{2} p_{tsa} \, . \tag{16}$$

Note that we divide h_t by 2 since the gene has an equal chance of moving through a mother

or a father. Thus,

$$u_{tf1} = 0.4 \qquad p_{tfa} = 0 \quad (\text{for all } a > 1) \tag{17}$$

$$u_{tm1} = 0.2\,,\ u_{tm2} = 0.2 \qquad p_{tma} = 0 \quad (\text{for all } a > 2)\,. \tag{18}$$

Recall that we're tracing a single gene on its journey jumping back in time. To simplify this thought experiment, we've made the assumption that such a gene can only go back to a mother of a single age class and a father belonging to one of two possible age classes. Thus, in each generational step, p_{fta} and p_{mta} are zero for all other age classes, a.

Next, define the probability that a random ancestral path at time t is present in an individual of a given age, a, and sex, s, to be v_{tsa}. Thus,

$$v_{tsa} = \sum_{i=0}^{\infty} u_{t+i,s,a+i} \tag{19}$$

and in particular when $a = 0$,

$$v_{ts0} = \sum_{i=0}^{\infty} u_{t+i,s,i}\,, \tag{20}$$

where we sum over an arbitrary number of years starting from a distant point in the future. Assuming a stable age distribution, the t dependence drops and we obtain the equality

$$v_{f0} = \sum_{i=0}^{\infty} u_{fi} = \frac{h}{2}\sum_{i=0}^{\infty} p_{fi} = \frac{h}{2} = \frac{h}{2}\sum_{i=0}^{\infty} p_{mi} = \sum_{i=0}^{\infty} u_{mi} = v_{m0}. \tag{21}$$

These equations imply that

$$v_{f0} = u_{f1} = 0.4 = u_{m1} + u_{m2} = v_{m0}\,, \tag{22}$$

which means that the total reproductive value of all age-zero females equals the total reproductive value of all zero-age males.

We can now determine the total reproductive value over all ages given our idealised example. By definition, when we consider summing over all ages, we have

$$\sum_a v_{sa} = \sum_a \sum_{i=0}^{\infty} u_{a+i,s} = \sum_a a u_{sa}\,. \tag{23}$$

Since most of these terms are zero from equations 17 and 18, we obtain the following

$$\sum_a v_{fa} = 1 \cdot u_{f1} = 0.4 \tag{24}$$

$$\sum_a v_{ma} = 1 \cdot u_{m1} + 2 \cdot u_{m2} = 0.2 + 2 \cdot 0.2 = 0.6\,. \tag{25}$$

Thus, even though the per-capita per-annum reproductive success of males is half that of females, their lifetime reproductive value is higher since older fathers put more genes from previous generations into generations through younger mothers. Thus, the number of aggregate replicas through sons begins to catch up to the number through daughters. This effectively balances the sex difference in annual reproductive success.

4 Discussion

It has long been recognised that sex differences in adult mortality do not necessarily violate Fisher's argument for equal parental investment in sons and daughters. Our results suggest that age-specific fertility differences between the sexes in humans may function in the same way. Although males typically have lower average reproductive success per year, they also remain fertile for longer than females. This extended fertility enables surviving males to accumulate genetic contributions over a longer period, thereby compensating for their lower reproductive success per time step. We explored this possibility using two mathematical models, showing that if male fertile careers are twice as long as those of females, the corresponding reproductive value of males is larger. Thus, extended male fertility balances the sex differences in average reproductive success, preserving Fisher's equilibrium for human life histories. Although we illustrate this with the 'half as high for twice as long' scenario, this is a worked example chosen for simplicity; the modelling framework applies to any sex difference in the duration or intensity of fertility.

While our models use simplified assumptions, they suggest a plausible evolutionary scenario in which sex differences in the duration of fertility are consistent with a balanced offspring sex ratio and equal long-term genetic contributions from males and females. We omit explicit male hierarchy or other specific deference mechanisms. These effects would extend male reproductive opportunity further, amplifying the sex-specific difference in reproductive value that our simple demographic asymmetry already produces. Male competition for reputation, which places older men at an advantage, has been modelled in a previous study [41], and would likely reinforce the same demographic dynamics described here. This opens the door to a broader view of life history evolution, in which the genetic reproductive value of older individuals, plays a central role in shaping sex allocation strategies over time. Taken together, these results suggest that human life history evolved under conditions that maintain a genetic balance between the sexes, shaped by sex-specific patterns of ageing and fertility.

Declaration of competing interest

The authors declare that they have no known competing financial or personal relationships that could have appeared to influence the work reported in this paper.

A Appendix

Figure 6 illustrates the evolution of ancestry paths across generations under different assumptions about the transmission probabilities w_1 and w_2. Here, we generalise the transition matrix from the main text (Eq. 14); w_1 denotes the probability that a female offspring traces her ancestry through a young male parent (m_1), while $w_2 = 0.5 - w_1$ corresponds to the probability of tracing through an older male parent (m_2). The Markov transition matrix P incorporates these probabilities to govern the dynamics of ancestry paths over time. Panels (a)–(c) present simulations for $w_1 = 0.05$ (older males have a large advantage), $w_1 = 0.25$ (all males have an equal chance), and $w_1 = 0.45$ (younger males have a large advantage). Note that in all cases, asymptotic dynamics lead to the same steady state.

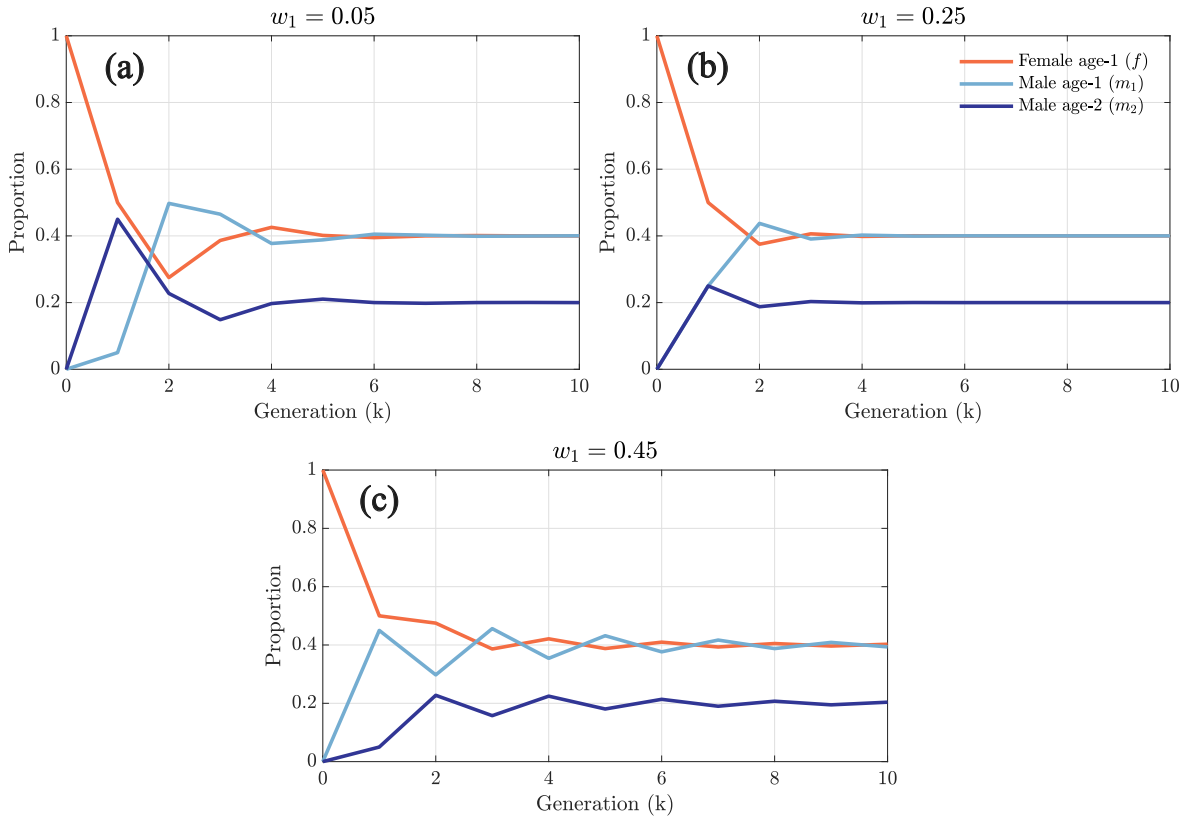

Figure 6: Evolution of ancestry paths under varying values of w_1 and w_2, where w_1 is the probability that a female offspring traces ancestry through a young male (m_1), and $w_2 = 0.5 - w_1$ is the probability of tracing through an older male (m_2). Panels (a)–(c) show the proportion of ancestry paths associated with females (f), young males (m_1), and older males (m_2) across generations. (a) Older males are heavily favored with only 5% of male contributions going through young males, $w_1 = 0.05$, and 45% through older males, $w_2 = 0.45$. (b) Old and young males are equally likely $w_1 = w_2 = 0.25$. (c) Young males are heavily favored with 45% of male contributions going through young males, $w_1 = 0.45$ and only 5% through older males, $w_2 = 0.05$. In all cases, less than 10 generations are needed to reach the same equilibrium.

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