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Two roads to two sexes: unifying gamete competition and gamete limitation in a single model of anisogamy evolution

Jussi Lehtonen · Hanna Kokko

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Abstract Recent studies have revealed the importance of self-consistency in evolutionary models, particularly in the context of male–female interactions. This has been largely ignored in models of the ancestral divergence of the sexes, i.e., the evolution of anisogamy. Here, we model the evolution of anisogamy in a Fisher-consistent context, explicitly taking into account the number of interacting individuals in a typical reproductive group. We reveal an interaction between the number of adult individuals in the local mating group and the selection pressures responsible for the divergence of the sexes. The same underlying model can produce anisogamy in two different ways. Gamete competition can lead to anisogamy when it is relatively easy for gametes to find each other, but when this is more difficult and gamete competition is absent, gamete limitation can provide another route for anisogamy to evolve. In line with earlier models, organismal complexity favors anisogamy. We argue that the early contributions of Kalmus and Scudo, largely dismissed as group selectionist, are valid under certain conditions. Linking their work with the contributions of Parker helps to explain why precisely males keep producing more sperm than can ever lead to offspring: sperm could evolve to provision zygotes but this brings little profit for the effort required, because sperm would have to be equipped with provisioning ability before it is known which sperm will make it to the fertilization stage. This insight creates a logical link between paternal care under uncertain paternity (where again investment is selected against when some investment never brings about genetic benefits) and gamete size evolution.

Keywords Anisogamy · Sperm competition · Parental care · Sex roles

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J. Lehtonen (✉) · H. Kokko
Laboratory of Ecological and Evolutionary Dynamics,
Department of Biological and Environmental Sciences,
University of Helsinki,
P.O. Box 65 (Viikinkaari 1), 00014, Helsinki, Finland
e-mail: jussi.lehtonen@helsinki.fi

J. Lehtonen · H. Kokko
Evolution, Ecology and Genetics, Research School of Biology,
The Australian National University,
Canberra, ACT 0200, Australia

Introduction

Sexual reproduction can occur without distinct males and females. The two sexes, with the labels "male" and "female", only exist if gametes of two distinct sizes fuse to form a zygote (Bell 1982). Why maleness and femaleness exists in the first place is a question of gamete size evolution (Lessells et al. 2009; Jennions and Kokko 2010): males by definition are the sex producing the small gametes (e.g., Bell 1982) in such anisogamous species. Unicellular, sexually reproducing organisms are usually isogametic with no separate sexes (Parker et al. 1972), although most such organisms have gametes of two kinds, + and −. These mating types are morphologically indistinguishable, but fusion takes place only between unlike types (Wiese et al. 1979; Maynard Smith 1982; Hoekstra 1987). Hermaphroditism is a well-known alternative, but hermaphroditic organisms still have clearly distinct male and female functions (or "roles").

The recent review by Lessells et al. (2009) states that models for the evolution of anisogamy fall into three categories: they focus on gamete competition (Parker et al.

1972; Maynard Smith 1982; Bulmer 1994; Bulmer and Parker 2002), sperm limitation (argued to increase egg size, Cox and Sethian 1985; Levitan 1993; Dusenbery 2000), or intracellular conflicts (Cosmides and Tooby 1981; Hurst 1996; Law and Hutson 1992). Lessells et al. (2009) compiled a list of outstanding questions in the field, recommending fruitful avenues for empirical work (on the considerable difficulties to test between these hypotheses empirically see also Randerson and Hurst 2001a, b). Here, however, we shall argue that there is still much theoretical work to be conducted as well.

The reason behind this view is the recent recognition of the importance of the so-called Fisher condition in explaining male and female roles in reproduction and parental care. The Fisher condition states that in a given period of time, the number of offspring sired by all males must be the same as the number of offspring produced by all females. Queller (1997) realized that many earlier theories of sex roles in parental care violate this condition: they inexorably lead to the logically impossible conclusion that, despite an even sex ratio, males have on average more mating partners than do females. Self-consistency has since been argued to be an essential feature of models in evolutionary theory (Houston and McNamara 2005; Houston et al. 2005), and subsequent work has shown that this simple principle can lead to complex and unexpected consequences (Kokko and Jennions 2008). In particular, the Fisher condition predicts that a mate-limited sex should not automatically evolve to avoid provisioning offspring: since members of this sex are (by definition) not guaranteed a high mating rate, provisioning offspring may be a more profitable route to fitness than attempting ever more matings. We should therefore expect the same to apply for theories of the evolution of the sexes: under an even sex ratio, the average number of gametic fusions achieved by males must be exactly the same as that of females. The explanation for why males keep producing more gametes than can possibly ever be fertilized has to be answered with this in mind.

In much of the classic work on anisogamy, the numbers of mature individuals producing gametes, and hence the number of gametes available, is not explicitly considered (e.g. Parker et al. 1972; Maynard Smith 1982; Bulmer 1994; Bulmer and Parker 2002). This means that we do not know if an explicit consideration of mating difficulties of the sex with the more numerous gametes would change the conclusions (while so far, some models have been based on mating difficulties of the *larger* gametes, classified under "sperm limitation" in Lessells et al. 2009). Our aim is to begin to fill in this gap by producing a model that is *Fisher-consistent*, by which we mean that the Fisher condition is taken explicitly into account (see Houston and McNamara 2005). In doing so, we also revisit the issue of the shape of the functions that relate gamete or zygote size to its fitness.

The model

The historical sequence of events leading from isogamy without mating types to anisogamy with mating types is unclear, and consequently several different pathways have been modeled in the past. We begin the construction of our sequential invasion analysis model (Otto and Day 2007) with the claim by Hoekstra (1987) that models of the evolution of anisogamy should start with an ancestral isogamous population with two mating types, although we note that a similar model could be built for other cases too. The same starting point was used in later revisions of Parker et al.'s (1972) theory by Maynard Smith (1982), Bulmer (1994), and Bulmer and Parker (2002). We mainly use the work of Bulmer and Parker (2002) as the starting point for our Fisher-consistent model, but we also incorporate attributes of the older models by Kalmus (1932) and Scudo (1967). We model competition among gametes and the scarcity (or abundance) of gametes of the opposite mating type explicitly in continuous time (for an overview see Fig. 1). Our continuous-time formulation applies to a population in which the number of parents of either mating type (+ or −) is at equilibrium, such that the local vicinity contains A_x and A_y adult (gamete-producing) individuals of the + and − type, respectively. (We prefer the notation x and y for subscripts indicating mating types in equations, to avoid confusion with the mathematical meaning of the symbols + and −. Henceforth we will refer to the mating types only as x and y). While we will formulate gamete

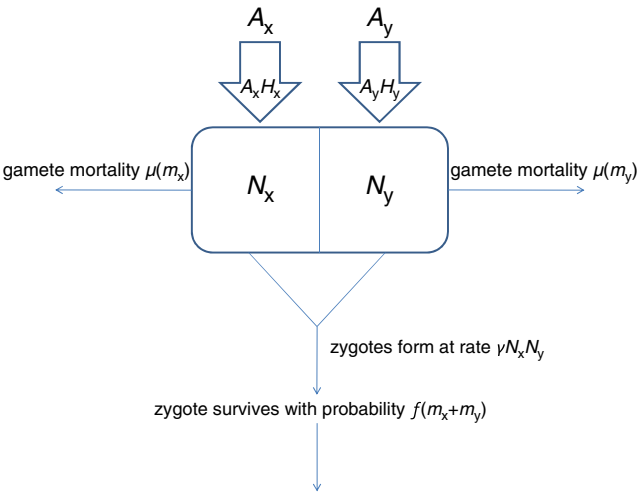

Fig. 1 The description of the model. The number of parents of + and − (which we henceforth call x and y) mating types is A_x and A_y, respectively, and each parent produces an influx of $H_x = H(m_x)$ and $H_y = H(m_y)$ gametes per unit time into the local volume where gametes seek fertilization opportunities. Gametes become depleted by dying (rate $\mu(m_x)$ and $\mu(m_y)$, respectively) and by mating via a process of random encounters, with γ modifying the encounter rate. The survival of the resulting zygote is a function of its size

dynamics explicitly, we will not do the same for the number of adults A_x and A_y. Instead, we assume that deaths of adults occur independently of their gamete production strategy and are compensated for by a corresponding number of new recruits into the adult population. These assumptions allow us to state that parental fitness is maximized when the instantaneous rate of fitness gain is maximized, which will depend on the rate of gamete formation and the subsequent success of these gametes.

We assume that both mating types (x and y) have a budget M per time unit to spend on gamete production. A parent that produces gametes of size m can produce a number $H(m)=M/m$ of them per unit time. We assume that M/m is sufficiently large in comparison to the rate of gamete fusion, and the mortality rates of adults, zygotes, and gametes that the population does not become limited by lack of recruits; values that would lead to a persistent decline of at least one mating type A_x or A_y in the population would not sustain a population and we thus exclude such cases. In other words, despite considering gamete limitation, we assume it is not so severe that the population cannot persist (this is in keeping with reality as sperm-limited populations are argued to be common in broadcast spawners, Bode and Marshall 2007).

As the local vicinity contains A_x and A_y adult individuals, it follows that the total rate of gamete production in the locality is $A_xH(m_x)=A_xM/m_x$ and $A_yH(m_y)=A_yM/m_y$. Our explicit accounting of the numbers of adults allows us to consider cases where, e.g., sperm competition (or more broadly, competition among gametes) does not occur: this is achieved by setting $A_x=A_y=1$. To derive the fitness gradients for calculating the evolutionary trajectories of gamete sizes, we consider how (if at all) the fitness of a single focal (mutant) individual changes with a small increase or decrease in gamete size.

Since we consider here finite local population sizes, gametes of the same mating type as the focal individual will determine the depletion rate of gametes of the opposite mating type. It follows that gamete availability for each mating type is dependent on the gamete sizes of both mating types, as well as their mortality rates. This also calls for a critical re-examination of the functions for zygote and gamete survival. Bulmer and Parker (2002) use a function derived from the work of Vance (1973) and Levitan (2000) for zygote survival. The function is based on two main assumptions: (1) instantaneous zygote mortality rate is constant and independent of zygote size; (2) the time required for a zygote to develop from fertilization to adulthood is inversely proportional to zygote size. From these assumptions, it follows that larger zygotes are more likely to survive the entire process from fertilization to adulthood as they spend less time experiencing constant mortality. We refer to Vance (1973) and Levitan (2000) for the exact derivation for the survival function that ensues, and simply give it here as

$$f(m_x, m_y) = e^{-\frac{\beta}{m_x+m_y}} \quad (1)$$

Note that this survival is a probability, rather than a rate. The parameter β scales the relationship between survival and size such that large values of β only permit high survival for very large zygotes. β is therefore interpreted as the "complexity" or size of the organism: complex, multicellular organisms in particular will experience a survival advantage to the reproductive stage if they, already as zygotes, are well provisioned (Bulmer and Parker 2002).

While one could conceivably question the assumption of size-independent mortality, our approach does not as such affect these assumptions. Therefore, we model zygote survival identically with earlier work, which is also the approach adopted by Bulmer and Parker (2002), and accept Eq. (1) as our description of zygote survival.

These authors then state that the same relationship is probably realistic for the probability of a gamete surviving until fertilization. This, however, would require conditions analogous to the two mentioned above: (1) instantaneous gamete mortality rate should be constant, and independent of gamete size; (2) the time required for a gamete to find and fuse with another gamete should be inversely proportional to gamete size. The first condition is again reasonable, at least as a first approximation, although not universally accepted (Dusenbery 2006). The second, however, is debatable.

One could argue that the time for a gamete to find opposite gametes is indeed shorter for large gametes because they present a target that is larger in cross-sectional area (Lessells et al. 2009). Nevertheless, the assumption that the time required for a gamete to be fertilized is inversely proportional to gamete size seems rather arbitrary. That said, there are probably other combinations of time and survival that would lead to a fitness function that has similar biologically reasonable properties of a minimum viable size and a saturating shape as the ones used in earlier models. Our main critique is thus not the shape itself, but the fact that *any* gamete survival function of a fixed shape is ill suited to capturing the idea that the time to find a gamete of the opposite type must also depend on the number of gametes of the opposite types. Therefore, it is preferable to derive the fitness of each gamete based on explicit tracking of gamete numbers, mortalities, and zygote-forming possibilities.

Explicit accounting of the fate of gametes over time is also required because gamete survival (over any given time unit) will matter less to gametes of a mating type that will mate much sooner than another type, as such a gamete will then proceed to the stage in the life cycle where zygote

rather than gamete survival matters. Thus, we need a function to describe the instantaneous mortality rate of gametes. As mentioned above, a reasonable first approximation is that it is relatively independent of gamete size. However, realistically, there must be a lower limit for gamete size corresponding to nuclear size and minimum possible cytoplasm (Parker et al. 1972). Therefore we seek a function for *instantaneous* mortality (not a probability as in Eq. 1) that is approximately constant across most of the size range, but increases steeply at the minimum gamete size threshold. There are several candidates for such a function, and if they lead to the same conclusions, this makes the model more robust. We base the results shown here on the function

$$\mu(m) = \varphi e^{\left(\frac{\alpha}{m}\right)^k} \tag{2}$$

where φ is the minimum gamete mortality rate, α is the (approximate) minimum gamete size below which mortality increases sharply, and k determines the steepness of the rise of the mortality rate at the minimum size threshold. Here, m refers to either m_x or m_y for the two different gamete sizes produced by the two mating types. As k increases, μ will approximate a step function more and more closely, thus the interpretation of α as an absolute minimum size for viability is better when k is large, while with low k, there is no such step-wise threshold but α nevertheless scales the size below which survival is very low. Note that from instantaneous mortality rates we can obtain survival probabilities over a specific time period by calculating survival over a time span of T units as $e^{-T\mu(m)}$. Thus, we can phrase the same results using survival rather than instantaneous mortality: using, e.g., k=100, gamete survival is essentially an all-or-nothing affair, whereas with lower values of k we can set it to be a more gradually increasing function of gamete size (Fig. 2a). This way, we can see that our approach allows for survival functions of very similar shape to earlier theory (Fig. 2b presents the Bulmer and Parker 2002 assumption) while allowing for more flexibility. The more important improvement in our model compared with earlier theory is explicit accounting of time. Our model does not assume that the probability of surviving over any prespecified time span (e.g., one time unit, as depicted in Fig. 2a) equals the probability of surviving to fertilization. The latter has to be computed taking into account additional information on the time it takes to find a gamete of the opposite mating type. In our model this probability is determined through the time dynamics of the model, and it is affected by gamete sizes of both mating types, as well as the encounter rate γ.

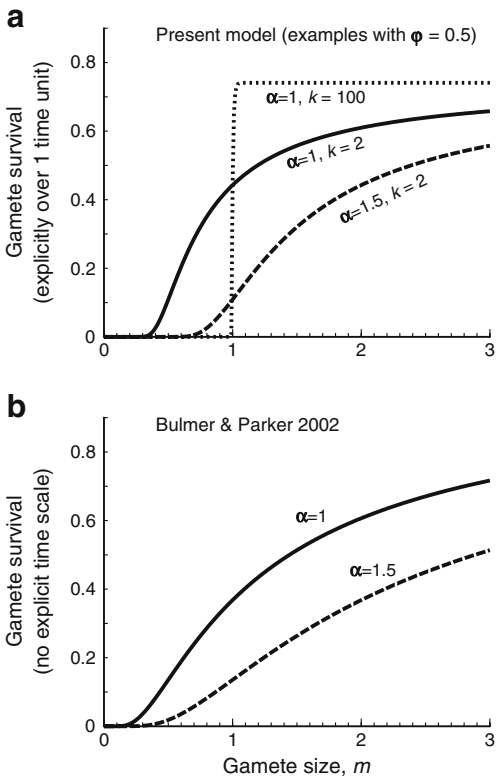

Fig. 2 The gamete mortality function used in this model, rephrased as survival probability over one unit of time (**a**), and the gamete survival function used in Bulmer and Parker (2002), which has no explicit time scale (**b**). The approach in **a** allows for survival functions of similar shape to earlier theory while allowing explicit accounting of time

We assume gametes to behave similarly to randomly colliding particles, which are removed from the particle pool after a successful collision. We begin by following Kalmus (1932) and use statistical mechanics on particle collisions to determine the population-wide rate of zygote formation (see also Dusenbery 2006 for further similar derivations). From the perspective of one individual gamete of type, say, x, the rate of encountering gametes of the opposite type is assumed to increase linearly with their density N_y, multiplied by the encounter rate coefficient γ that describes the ease with which gametes find each other. When there is a total number N_x per unit volume of gametes that experience such encounters, the overall rate of encounters in the entire population becomes $\gamma N_x N_y$ where N_x and N_y denote the equilibrium density of gametes in the locality (which without loss of generality is assumed to have a volume of unit size). The parameter γ can be used to adjust the level of gamete limitation in the model. Note that similarly as an instantaneous mortality rate can exceed 1, the rate $\gamma N_x N_y$ can exceed the local number of gametes in a continuous-time model, without this being a mistake. If a

larger number of gametes are removed from a pool of gametes per time unit than there are gametes in this pool, this simply means that gamete numbers deplete very quickly. The high depletion rate applies over an infinitesimally short time period only, thus preventing the removal of too many gametes. The rate $\gamma N_x N_y$ then instantaneously decreases to account for the fast decrease in available numbers.

Before asking evolutionary questions about selection to change gamete size, we need to derive equations that govern the number of gametes available for fertilizations per unit volume, N_x and N_y. The above assumptions imply that the dynamics of gamete numbers N_x and N_y are governed by (1) a continual input of $A_x H(m_x)$ and $A_y H(m_y)$ per time unit, respectively, reflecting gamete output by parents; (2) an instantaneous mortality rate of $\mu(m_x)$ for each x gamete and $\mu(m_y)$ for each y gamete, which leads to a loss $\mu(m_x)N_x$ and $\mu(m_y)N_y$ of the gametes present in the local population; and (3) fertilizations (collisions of opposite mating type gametes) which remove gametes of either type at a rate $\gamma N_x N_y$. The numbers N_x thus increase (decrease) over time if $A_x H(m_x) - \mu(m_x)N_x - \gamma N_x N_y$ is positive (negative). A similar equation, with x- and y-indices reversed, holds for N_y. The gamete numbers are at equilibrium when there is no net change, i.e., the rate of change over time is zero:

$$\begin{cases} \frac{dN_x}{dt} = A_x H(m_x) - \mu(m_x)N_x - \gamma N_x N_y = 0 \\ \frac{dN_y}{dt} = A_y H(m_y) - \mu(m_y)N_y - \gamma N_x N_y = 0 \end{cases} \quad (3a–3b)$$

The total rate of offspring produced by each mating type is $\gamma N_x N_y f(m_x, m_y)$, i.e., the fertilization rate multiplied by the survival probability of the zygotes. As this term is identical for the two mating types, the Fisher condition is fulfilled.

However, Eqs. (3a–3b) describe the situation only when all individuals within a mating type are producing gametes of the same size. To determine the evolutionary dynamics of the system, we expand the equations to accommodate a mutant individual, which we denote with a "hat". For example, $\hat{m}_y$ refers to the gamete size of a mutant of mating type y, whereas $\hat{N}_y$ refers to the density of available gametes produced by this mutant. The equations below are derived for the case where a single mutant individual of mating type y produces gametes of size $\hat{m}_y$. All other y-individuals produce gametes of size m_y, and all x-individuals produce gametes of size m_x. Switching all x- and y-indices gives the equivalent equations for a mutant x-individual.

$$\begin{cases} \frac{dN_x}{dt} = A_x H(m_x) - \mu(m_x)N_x - \gamma N_x(N_y + \hat{N}_y) = 0 \\ \frac{dN_y}{dt} = (A_y - 1)H(m_y) - \mu(m_y)N_y - \gamma N_x N_y = 0 \\ \frac{d\hat{N}_y}{dt} = H(\hat{m}_y) - \mu(\hat{m}_y)\hat{N}_y - \gamma N_x \hat{N}_y = 0 \end{cases} \quad (4a–4c)$$

Again, the Fisher condition is fulfilled because the total rate of offspring production for both mating types is $\gamma N_x N_y f(m_x, m_y) + \gamma N_x \hat{N}_y f(m_x, \hat{m}_y)$.

The latter term in this equation is the fitness of the mutant y-individual:

$$\hat{W}_y = \gamma N_x \hat{N}_y f(m_x, \hat{m}_y) \quad (5)$$

To proceed with the sequential invasion analysis, we must derive the fitness gradients, i.e., the derivative of the mutant individual's fitness with respect to the mutant gamete size for both mating types. We also derive the second derivatives and the convergence stability matrix to determine the evolutionary stability and convergence stability (Eshel 1983; Otto and Day 2007) of equilibria. The calculations and resulting equations are lengthy, and we refer the interested reader to Appendix 1 for details; here we show only the final equations for the fitness gradient of mating type y (again we get the equivalent equation for the opposite mating type by exchanging all x- and y-indices).

$$\frac{\partial \hat{W}_y}{\partial \hat{m}_y}\Big|_{\hat{m}_y = m_y} = \left[\left(H(\hat{m}_y) - \hat{N}_y \mu(\hat{m}_y) \right) \frac{\partial f(m_x, \hat{m}_y)}{\partial \hat{m}_y} + f(m_x, \hat{m}_y) \left(\frac{dH(\hat{m}_y)}{d\hat{m}_y} - \hat{N}_y \frac{d\mu(\hat{m}_y)}{d\hat{m}_y} - \mu(\hat{m}_y) \frac{\partial \hat{N}_y}{\partial \hat{m}_y} \right) \right] \Big|_{\hat{m}_y = m_y} \quad (6)$$

where

$$\frac{\partial \hat{N}_y}{\partial \hat{m}_y} = - \frac{\left(\gamma N_x \left[\gamma \hat{N}_y + \mu(m_x) \right] + \left[\gamma N_y + \gamma \hat{N}_y + \mu(m_x) \right] \mu(m_y) \right) \left(-\frac{dH(\hat{m}_y)}{d\hat{m}_y} + \hat{N}_y \frac{d\mu(\hat{m}_y)}{d\hat{m}_y} \right)}{\gamma^2 N_x^2 \mu(m_x) + \left(\gamma N_y + \gamma \hat{N}_y + \mu(m_x) \right) \mu(m_y) \mu(\hat{m}_y) + \gamma N_x \left(\gamma N_y \mu(m_y) + \gamma \hat{N}_y \mu(\hat{m}_y) + \mu(m_x) \left[\mu(m_y) + \mu(\hat{m}_y) \right] \right)} \quad (7)$$

and

$$\begin{cases} N_x\big|_{\hat{m}_y=m_y} = \dfrac{A_x\gamma H(m_x)-A_y\gamma H(m_y)-\mu(m_x)\mu(m_y)+\sqrt{4A_x\gamma H(m_x)\mu(m_x)\mu(m_y)+\left[-A_x\gamma H(m_x)+A_y\gamma H(m_y)+\mu(m_x)\mu(m_y)\right]^2}}{2\gamma\mu(m_x)} \\ N_y\big|_{\hat{m}_y=m_y} = (A_y-1)(\hat{N}_y\big|_{\hat{m}_y=m_y}) \\ \hat{N}_y\big|_{\hat{m}_y=m_y} = \dfrac{-A_x\gamma H(m_x)+A_y\gamma H(m_y)-\mu(m_x)\mu(m_y)+\sqrt{4A_x\gamma H(m_x)\mu(m_x)\mu(m_y)+\left[-A_x\gamma H(m_x)+A_y\gamma H(m_y)+\mu(m_x)\mu(m_y)\right]^2}}{2Ay\gamma\mu(m_y)} \end{cases} \quad (8a–8c)$$

The fitness gradients are derived with the assumption that offspring disperse far enough to be outside the locality where the parents resided. In this case we can use the derivative of absolute fitness as the fitness gradient: we compare a parent's fitness against the entire population. If the offspring did not disperse, local fitness differences would determine the course of evolution (fitness of the mutant relative to the individuals it interacts with and influences the fitness of, see e.g. Johnstone 2008 for an example).

We use the fitness gradient to search numerically for evolutionary equilibria and to plot evolutionary trajectories, the latter assuming that evolutionary change within each mating type is proportional to the fitness gradient of this mating type. The second derivatives and the convergence stability matrix (see Appendix 1) can then be used to check the evolutionary stability and convergence stability of the equilibria.

Although the model allows the local population sizes of both mating types to be defined independently, in this study we focus on the case $A_x=A_y=A$. We thus exclude a priori biases that would result in a skewed adult mating type ratio. Considering them would require specifying processes that could lead to such an asymmetry, and this is beyond the scope of our current work.

In the following we will show representative examples of evolutionary trajectories using the fitness gradient approach (Eqs. 6, 7, and 8a–8c, for details see Appendix 1). In these examples we systematically change only one parameter at a time. We also determined the evolutionary and convergence stability of each equilibrium that these examples yield. We then corroborated the insight thus gained by examining a large set of parameter combinations and computing evolutionary equilibria numerically using the fitness gradient approach. The randomness of our exploration of the parameter space avoids the danger that the initial examples might have presented a biased subset of possible evolutionary equilibria.

Results

In line with earlier models, organismal complexity favors anisogamy

Our model repeats two key predictions of earlier models (e.g. Parker et al. 1972; Bulmer and Parker 2002): increasing organismal complexity favors the evolution of anisogamy, and simultaneously complexity is predicted to increase the *degree* of anisogamy by increasing egg size (and thus the egg/sperm volume ratio). As in Bulmer and Parker (2002), we assume that a large value of β reflects complexity: zygotes then need relatively larger reserves to increase their chances of survival until adulthood. Although our definition of gamete mortality is explicit as a rate over time and thus our definition of α is somewhat different from theirs, a large difference between β and α in both models implies qualitatively the same statement: that a viable zygote requires a large investment in comparison to the minimum requirements of a gamete. It is thus reassuring that we repeat the finding that $\beta>4\alpha$ is a minimum condition for the evolution of anisogamy; no matter what the other parameter values are, anisogamy will not evolve unless $\beta/\alpha>4$ (Fig. 3, 4). However, in a departure from Bulmer and Parker (2002), Fig. 4 shows that $\beta>4\alpha$ is not a sufficient condition to guarantee the divergence of gamete sizes, as a large number of cases with this condition fulfilled remain isogamous (Fig. 4; for specific examples of such cases see Fig. 5a–c). Anisogamous solutions are characterized by either the x or the y mating type evolving to a large size (the exact size increases with the complexity parameter β, Fig. 3b–c) while the opposite mating type evolves to a value close to the minimum viable size for a gamete.

Gamete competition is a route to anisogamy

Figure 3 was derived assuming $A=20$, so that local interactions contain many parents of each mating type; likewise Fig. 4 contains mostly cases with $A>1$. This implies strong competition between gametes. By reducing A to low levels gamete competition can be diminished (top left panels in Fig. 5), and low values predict a return to stable isogamy (despite the complexity condition $\beta>4\alpha$ remaining fulfilled in Fig. 5a). Intermediate values of A can yield multiple stable equilibria: an isogamous solution as well as two anisogamous ones with either x or y types evolving to become females (clearest in Fig. 5b with $A=2$). The isogamous basin of attraction diminishes with increasing A, until very small deviations from isogamy are sufficient to trigger disruptive selection towards anisogamy

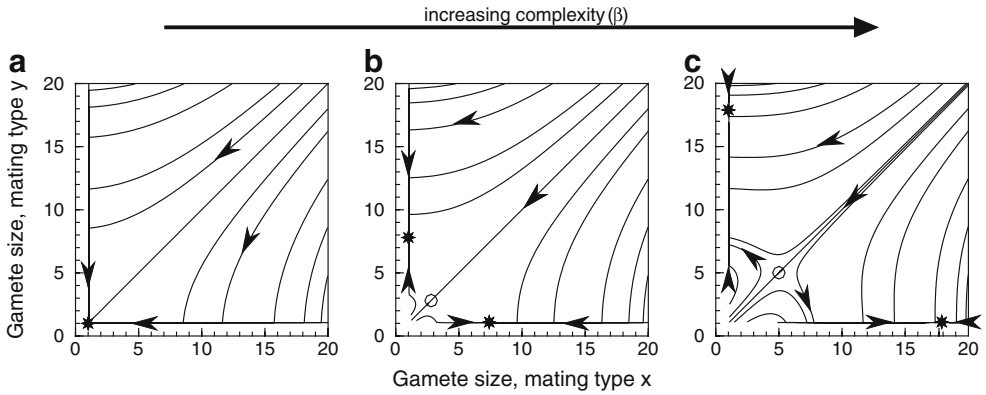

Fig. 3 In line with earlier results, the model predicts that increasing organismal complexity (parameter β) selects for anisogamy. Anisogamy always evolves with two possible equilibria, with either the x or the y type evolving to a large size (female). Egg size increases with β, **a** β=3.5, **b** β=10, **c** β=20. Other parameters: A=20 (there is gamete competition), α=1, γ=1, φ=1, k=100, M=100. Note that in a deterministic model such as this, isogamy is always an equilibrium due to symmetry, but not necessarily a stable equilibrium. Stable equilibria are indicated with *stars*, unstable equilibria with *open dots*. Given suitable conditions (**b,c**), small deviations easily lead to anisogamy. Numerical values for the equilibria and stability analysis are shown in Table 1 of Appendix 1

(Fig. 5c) and finally the isogamous equilibrium becomes convergently unstable (Fig. 5d). Thus anisogamy invading an ancestral isogamous population becomes more likely with increasing gamete competition (Fig. 5a–d). The existence of gamete competition (A>1) does not immediately produce anisogamy as the sole solution, although competition is sufficient to maintain anisogamy once evolved (Fig. 5b). This is in agreement with the results of (Parker 1982).

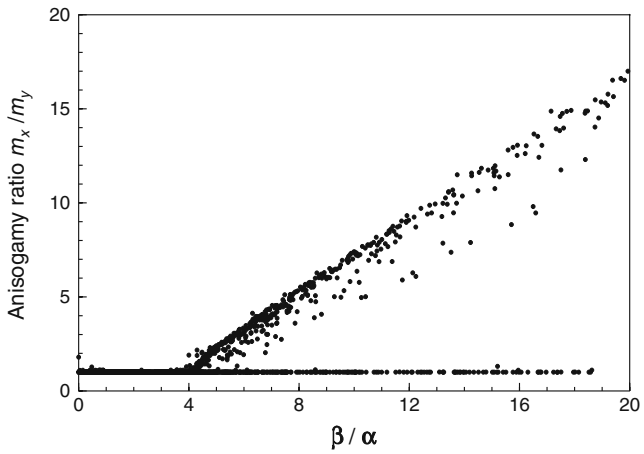

Fig. 4 Evolutionary endpoints for the gamete size ratio as a function of β/α in a set of 1,800 randomly chosen parameter combinations. The parameters are picked from the following uniform distributions: $0<\alpha<5$; $0<\beta<20$; $0<\gamma<10$; $0<\varphi<10$; $0<M<1000$; $0<k<100$; $0<A<10$ (integer values only for k and A). Here $A_x=A_y=A$, which imposes a 1:1 sex ratio of adults. The gamete size ratio was allowed to evolve to equilibrium with each random parameter combination, starting from nearly isogamous initial values: $m_y=(\beta+\alpha)/2$, $m_x=1.1m_y$. Despite the fact that our definition of α somewhat differs from earlier studies, the approximate minimum requirement for the evolution of anisogamy is $\beta/\alpha>4$, as found in Bulmer and Parker (2002). Beyond this ratio, increasing β further leads to an approximately linear increase of the gamete size ratio. However, all parameter combinations do not lead to anisogamy even when $\beta>4\alpha$ (see Fig. 5a–c for examples of why this is not always a sufficient condition)

Gamete limitation also produces anisogamy

Starting from the same situation as Fig. 5a, evolution can also proceed to anisogamy even in complete absence of gamete competition (A=1). This outcome can be achieved in three ways: by reducing γ, the encounter rate of gametes (Fig. 5e); by reducing M, the gamete production budget per time unit (also shown in Fig. 5e); or by increasing φ, the minimum mortality rate experienced by gametes (Fig. 5f). This is an intriguing finding because there is no gamete competition involved (A remains at 1 throughout the sequence 5a–e–f). Instead, for any given availability of gametes of the opposite mating type, these changes in γ, φ, and M all have the effect of decreasing the number of gametes that reach a suitable gamete to fuse with before dying. If at the same time the developmental requirements of the zygote are high ($\beta>>\alpha$), then the chances of simultaneously reaching a reasonable number of fusions and giving the zygotes good probabilities for survival can best be maximized when one mating type specializes in zygote provisioning, and the other specializes in producing a large number of gametes. As with Figs. 3 and 4, this result can also be corroborated by a random search of the parameter space. By fixing the value of A at 1 and letting all other parameters vary randomly, we confirm the result that in the absence of gamete competition the mortality rate experienced by gametes must be high in comparison to the gamete encounter rate for anisogamy to evolve (Fig. 6). Similar figures can be produced for the pairs γ-M and φ-M.

If there is gamete competition (high A) then anisogamy prevails regardless of the values of φ, γ, and M (not

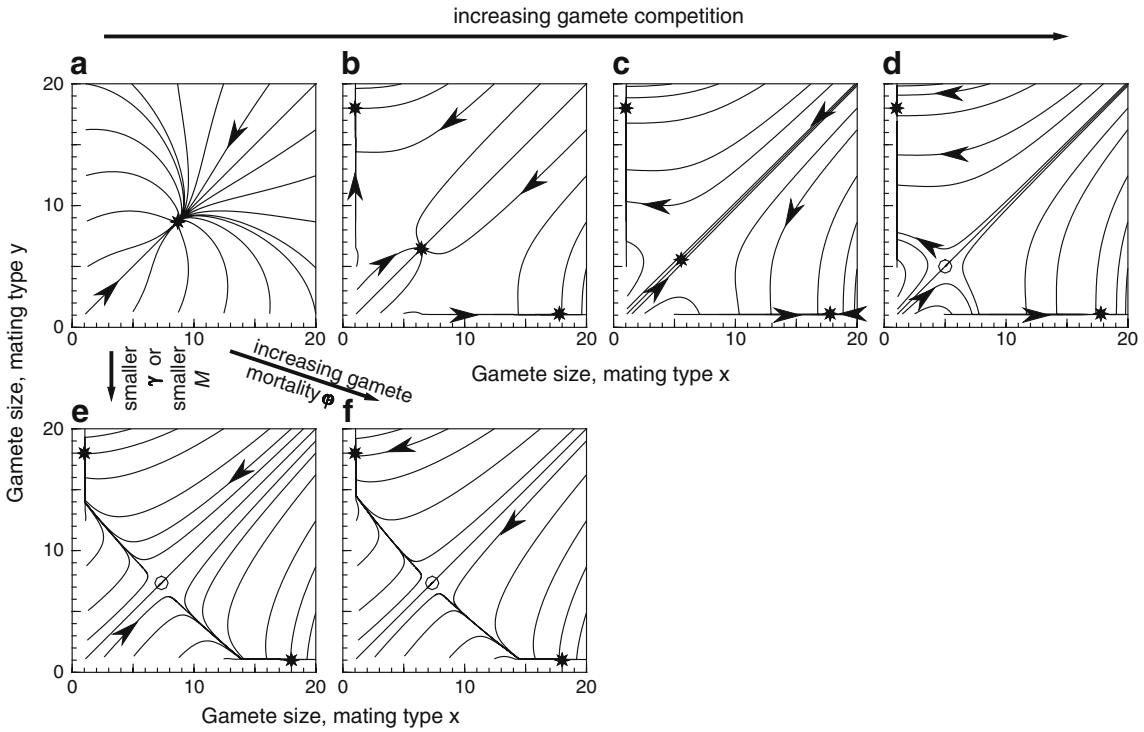

Fig. 5 The two routes to anisogamy: increasing gamete competition (*top row*) or gamete limitation (*difference between the two rows*). *Top row* shows the effect of increasing the number of individuals producing gametes in the locality, **a** A=1, **b** A=2, **c** A=5, **d** A=20 (other parameters: $\alpha=\gamma=\varphi=1$, β=20, k=100, M=100). As competition increases from **a** to **d**, the initial stability of the isogamous solution becomes increasingly sensitive to small deviations in gamete sizes until in **d** the smallest deviations trigger evolution towards one or the other anisogamous solution. But anisogamy evolution can also be triggered in the absence of any gamete competition if gametes fusions occur at a low rate: this can be achieved by reducing γ to 0.1 which decreases gamete encounter rates (**e**), by reducing M to 10 which decreases the gamete production rate (**e**, the evolutionary trajectories and equilibria are identical in these two cases) or by increasing gamete depletion through mortality, (**f**) φ=3. Other parameters in (**e–f**) as above, with A=1. Stable equilibria are indicated with *stars*, unstable equilibria with *open dots*. Numerical values for the equilibria and stability analysis are shown in Table 1 in Appendix 1

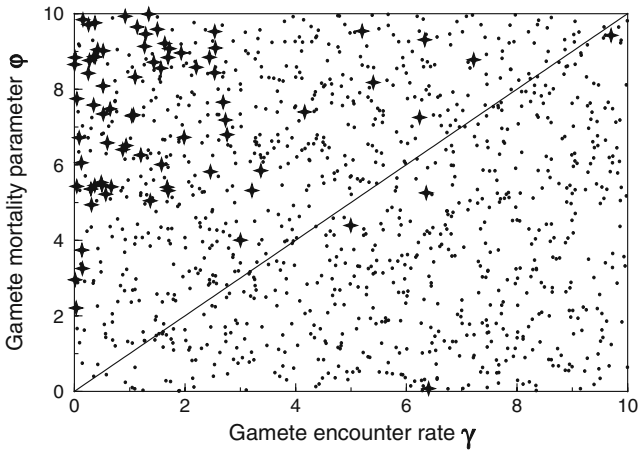

Fig. 6 The effect of gamete limitation on the evolution of anisogamy in the absence of gamete competition. The figure was produced from 1,200 parameter combinations with a procedure identical to Fig. 4, with the exception that gamete competition was excluded by setting $A_x=A_y=A=1$. *Dots* and *stars* indicate isogamous and anisogamous equilibria respectively. Gamete encounter rate increases on the x-axis, whereas gamete mortality increases on the y-axis. Therefore gamete limitation is strongest in the *upper left corner*. The figure indicates the robustness of the result that in the absence of gamete competition, gamete limitation is an alternative path to anisogamy

shown). Our findings are also robust with respect to the precise value of k which measures how accurately gamete survival matches the assumption of independence of its size.

Discussion

The modeling of anisogamy (the emergence of males and females after sexual reproduction has become established) has proceeded from arguments that derive gamete sizes that lead to a maximum number of gametes finding each other (Kalmus 1932) to an explicit individual-level consideration of how much to provision a zygote when the opposite mating type provisions zygotes as well (Parker et al. 1972; Maynard Smith 1982; Bulmer 1994; Bulmer and Parker 2002). Additionally, modeling has considered several alternative explanations, such as a eggs evolving to a large size because smaller eggs are difficult targets for sperm and might remain unfertilized (Cox and Sethian 1985; Levitan 1993; Dusenbery 2000; for a review of further hypotheses that are beyond our current focus see Lessells et al. 2009).

The contribution of Parker and his co-workers was of undisputed fundamental importance as it removed worries that argumentation might be based on group selection. Simultaneously, however, some valuable aspects of earlier work have been lost. Kalmus (1932) applied classical statistical mechanics on the collision of particles to maximize the number of successful gamete unions with various degrees of anisogamy. Scudo (1967) added gamete mortality and depletion due to previous gamete fusions to the same framework. These insights remain valid even though the group-level fitness argumentation has caused these old models to be largely dismissed in more recent updates of Parker's ideas (Maynard Smith 1982; Bulmer 1994; Bulmer and Parker 2002). Gamete fusion and depletion rates and the total numbers of zygotes formed are not explicitly derived in these later models.

This has two implications: gamete limitation or depletion is not accounted for explicitly, from which it follows that the Fisher condition is not accounted for. For example, consider the following type of fitness equations of x- and y-individuals used in several models (e.g. Maynard Smith 1982; Bulmer 1994; model (d) in Bulmer and Parker 2002)

$$\begin{cases} W_x(m_x) = \dfrac{M}{m_x} f(m_x + m_y) \\ W_y(m_y) = \dfrac{M}{m_y} f(m_x + m_y) \end{cases}$$

This implies that $\frac{w_x}{w_y} = \frac{m_y}{m_x}$, or equivalently $w_x = \frac{m_y}{m_x} w_y$. Now, assuming an anisogamy ratio (of gamete sizes) of, say 1:4, we have $w_x = 4w_y$, which clearly violates the requirement that male and female average fitness must be equal if the sex ratio is unity.

There is a conceivable counterargument for this critique: since we are considering mating types that have already diverged before the evolution of anisogamy, there is only competition for fertilizations within each mating type. Therefore only the relative fitnesses within a mating type matter for the end result, not the relative fitnesses between the two mating types. In other words, the absolute values of fitness could be argued not to matter as dividing the fitness of the more fit type by (in this case) four would restore equality without changing the shape of the fitness function. Our results show, however, that the matter is not quite that easily resolved. An explicit treatment of gamete depletion is required to derive how many fertilization opportunities are left locally for mutants that, say, decrease their own gamete size and thus produce more proto-sperm. If the number of adults in the local vicinity is small (low A in our model), it is inconsistent to assume that the success of each proto-sperm remains constant across all sperm sizes of the focal mutant. This is why applying a single correction factor (dividing by, say, 4) will not resolve the issue unless the population is well mixed such that gamete competition is ubiquitous and strong enough so that no focal individual has a significant impact on local gamete depletion. The strength of gamete competition and gamete depletion is thus worthwhile to consider explicitly.

Parker et al. (1972) and later models by Maynard Smith (1982), Bulmer (1994), and Bulmer and Parker (2002) assumed, at least implicitly, that competition is strong and depletion negligible. Parker later modeled what would happen in a limited population of external fertilizers after anisogamy has already evolved (Parker 1982), although the main aim of this extension was to model the stability of anisogamy once internal fertilization has evolved. Our current contribution shows that it is worthwhile to keep investigating the numbers of (proto)females and (proto) males in each mating group in broadcast spawners too, as it is by no means guaranteed that primitive broadcast spawning isogamous species experienced limitless gamete competition (Levitan 2010, see also Bode and Marshall 2007, Crean and Marshall 2008). We show that considerations of local competition are probably also relevant for broadcast spawners, as our explicit gamete depletion model predicts that isogamous solutions do not disappear immediately as soon as local interactions involve more than one individual of each mating type.

Given this background, it is perhaps not surprising that our model produces results identical to Bulmer and Parker (2002) when gamete competition is strong (large A): for example, our results are compatible with the finding that anisogamy is favored when the complexity of organisms increases ($\beta > 4\alpha$). A focus on the Fisher condition makes it necessary, however, to give a clearer, rephrased description of why exactly sperm can evolve to become "parasites" of eggs instead of contributing to zygote viability. This is an insightful way of phrasing the trade-off (e.g. Pizzari and Parker 2009), but when it is combined with recent modeling of post-zygote care (Kokko and Jennions 2008) it raises a new question: why precisely do proto-sperm keep evolving smaller when in a post-zygotic parental care context difficulties of males finding mates select for males who provide *more* care (all else being equal)? In other words, why does the Fisher condition not produce frequency-dependent selection that makes the smaller gametes evolve towards the size of the larger ones, in a similar manner to it producing frequency dependence on primary sex ratios (Fisher 1930) and frequency dependence on care that has to be overcome through other mechanisms, e.g. uncertain paternity, to explain sex-biased care (Kokko and Jennions 2008)?

One answer is that the mathematics work out that way (for this line of reasoning see e.g. Parker 1982, or p. 210 in Pizzari and Parker 2009): Parker's models as well as our results show this to be the case. It is nevertheless useful to

sketch a heuristic answer to why precisely selection becomes disruptive. When either sex could benefit by producing either many or well provisioned gametes (but between these goals there is a trade-off), why is the sex that is already overproducing gametes the one that keeps being selected to invest in ever more gametes rather than somewhat better provisioned ones? The verbal answer is illuminating as it shows that there is an important parallel between paternity uncertainty for sperm and for males tending their young. The sex that overproduces gametes will have difficulties having them fuse successfully, and this selects against equipping each "lottery ticket" with much provisioning ability because the provisioning decision has to be made *before it is known* which sperm will lead to positive fitness. The uncertain fate of each sperm makes it impossible to concentrate provisioning ability on those few that end up successful, and this is precisely analogous to a male's situation who has a brood of young and care has to be divided among the young *without knowledge* which ones carry the father's genes. The principle of little investment as a result of uncertain parentage (Queller 1997) thus extends to little investment in each proto-sperm as a result of highly uncertain returns of trying to improve the fate of each of them.

The situation is very different when gamete competition becomes weak (small A). In such a case, the fate of each gamete is far more certain, particularly if gametes survive relatively well (low mortality φ), if gametes encounter each other frequently (large γ) and if gametes are produced at a high rate (large M). This is analogous to a male who is relatively certain of parentage; his situation reverses to comparing a certainty of existing young vs. his uncertain success of finding new mates, and this favors care by the mate-limited sex (Kokko and Jennions 2008). These conditions indeed predict that isogamy evolves. The evolving patterns are interesting because intermediate parameter values predict that isogamy can coexist with anisogamy, and ancestral conditions then determine which equilibrium is maintained—this implies that phylogenetic inertia may play a significant role: reaching e.g. Fig. 5c from the right (large A that decreases) would predict that anisogamy will be retained, while reaching it from the left (small A that increases) predicts that isogamy is still stable, assuming that each mutation only has a small effect on gamete size.

The heuristic of uncertain investment can also help understand why regions of isogamy and anisogamy can coexist (the clearest example being Fig. 5b). If gamete sizes differ from each other relatively little, the relative overproduction of gametes by one sex remains limited. The problem of most gametes being destined to a dead end thus does not act with full force on either sex. Consequently, the relatively high chances of each gamete making it to the zygote stage makes investment in provisioning profitable for both sexes, and the system returns to isogamy. But if there is a sufficient deviation in current gamete sizes and consequently their numbers, then uncertainty (low average success of each gamete) will hit the overproducing and underinvesting sex hardest, selecting further against investing in each gamete.

There above explanation views our findings from the perspective of parental investment theory. One can, equally well, consider an approach more often used in deriving sex allocation patterns (West 2009). In the absence of other adults of the same mating type ($A=1$), gametes produced by the same parent compete with each other—a form of competition between relatives. This diminishes the returns of producing many gametes as they simply hamper each other's success (analogous to local mate competition; see also Schärer 2009). If $A>1$, much of competition happens between unrelated gametes, and the returns from gamete numbers do not diminish at the same rate for the parent producing them.

Our model also reveals another set of conditions that can lower the chances of each gamete making it to the zygote stage. In addition to evolving through gamete competition in a relatively large local population (the assumption of Parker et al. 1972), anisogamy can also arise in the absence of gamete competition if gametes do not survive well, if gamete encounter rates become low and/or if gametes are produced at a low rate. This route implies gamete limitation. It is a qualitatively different explanation from gamete competition discussed above, yet again the low investment in proto-sperm can be explained by a low effort in each gamete that has an uncertain future. The reduced size and increased number of proto-sperm then makes it easier for proto-eggs to become fertilized, which for mothers decreases the uncertainty that they face about the fate of eggs, and selects for increased investment in each. Thus both gamete limitation and gamete competition, while being superficially very different routes to anisogamy, can be understood using a similar framework of uncertain investments that influences parental care decisions too (Queller 1997; Kokko and Jennions 2008).

Note that by solely relying on this investment principle our model is a different version of gamete limitation than those discussed by Lessells et al. (2009); in typical gamete limitation models (Cox and Sethian 1985; Levitan 1993; Dusenbery 2000) larger eggs are assumed to be more likely to found by sperm. We assume no such effect, instead selection for large egg size is a simple result of higher maternal investment when each proto-egg has higher chances of success (due to evolution of numerous sperm). This form of disruptive selection could, obviously, interact with selective processes that favor large "targets" that are more likely to be found and fertilized than smaller gametes of the same mating type (review: Lessells et al. 2009).

In general, our model should be seen as a first step in taking the Fisher condition into account in all types of anisogamy models—not as a statement that the modeled processes are the only fundamental ones. In addition to assuming that the size of a "target" cell does not impact the likelihood that it is found, we have also made several other simplifying assumptions. The modeling approach we have used shows that the equilibria cannot be invaded by mutant strategies that are close to the equilibrium values, but it does not guarantee that large-effect mutations cannot invade. We have not considered the impact of any type of stochasticity in either the number of adults or the number of gametes. For example, we assume negligible spatial variation in the number of adult types. We consequently also assume no *within*-individual variation in gamete size, which could in principle be adaptive in unpredictable environments (Marshall et al. 2010). Given lack of theoretical work examining effects of stochasticity, we do not know if local variation in fertilization prospects forms a type of unpredictability that could select for such variation in gametic investment. Current empirical evidence is likewise scant. On the female side, there are some examples where within-brood egg size variation has been argued to reflect environmental variability (but not variation in fertilization prospects per se, Crean and Marshall 2009). On the male side, a broadcast spawner *Stuela plicata* has been shown to adaptively adjust sperm size across densities of sperm competitors (Crean and Marshall 2008). This does not yet, however, imply that there are multiple local optima for sperm size within a local population, and we did not find evidence for disruptive selection within a mating type in our theoretical work either. Future work could fruitfully relax our assumption of equilibrium numbers of adults and gametes to investigate this question.

Finally, it is also worth commenting on the oldest model of anisogamy, Kalmus (1932). This model was published decades before biologists were trained to realize that individuals or their genes can be in conflict with each other. Kalmus' model has been largely dismissed as group selectionist, yet this model is surprisingly relevant to this day in the special case of $A=1$. When there is only one individual of each mating type present, both "paternal" and "maternal" fitness are maximized when parents behave in a way that maximizes the number of viable zygotes, even though each parent is acting "selfishly" in its own best interests. This can again be seen as a consequence of the Fisher condition: when $A=1$, fitness of the only male in the locality must be exactly the same as the fitness of the only female (even though they are competing with individuals in other localities). Therefore maximizing one maximizes the other, and the group selectionist argument by Kalmus converges with our individual selection model when $A=1$. In that conflict-free case the analysis of Kalmus (1932) is valid to this day. Incorporating the Fisher condition into models of the evolution of anisogamy shows that the selection pressures that can lead to the divergence of gamete sizes form a continuum from Kalmus' principle of maximizing the number of successful zygotes, to the gamete competition principle of Parker et al. (1972). Which type prevails in nature is an interesting question for further study.

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Appendix 1

Our aim is to find the fitness gradients $\frac{\partial \hat{W}_y}{\partial \hat{m}_y}\Big|_{\hat{m}_y=m_y}$ and $\frac{\partial \hat{W}_x}{\partial \hat{m}_x}\Big|_{\hat{m}_x=m_x}$, the second derivatives $\frac{\partial^2 \hat{W}_y}{\partial \hat{m}_y^2}\Big|_{\hat{m}_y=m_y}$ and $\frac{\partial^2 \hat{W}_x}{\partial \hat{m}_x^2}\Big|_{\hat{m}_x=m_x}$ and the matrix $C = \begin{pmatrix} \frac{\partial}{\partial m_x}\left(\frac{\partial \hat{W}_x}{\partial \hat{m}_x}\Big|_{\hat{m}_x=m_x}\right) & \frac{\partial}{\partial m_y}\left(\frac{\partial \hat{W}_x}{\partial \hat{m}_x}\Big|_{\hat{m}_x=m_x}\right) \\ \frac{\partial}{\partial m_x}\left(\frac{\partial \hat{W}_y}{\partial \hat{m}_y}\Big|_{\hat{m}_y=m_y}\right) & \frac{\partial}{\partial m_y}\left(\frac{\partial \hat{W}_y}{\partial \hat{m}_y}\Big|_{\hat{m}_y=m_y}\right) \end{pmatrix}$ for evaluating the direction of evolution, evolutionary stability, and convergence stability respectively. We derive here the equations for the case where the mutant individual is of mating type y. Equivalent equations for type x are obtained simply by exchanging the indices x and y in all equations. A "hat" (such as in $\hat{m}_y$) is used to denote the mutant individual. For gamete numbers we will use the notation N_x, N_y, and $\hat{N}_y$ instead of the longer forms $N_x(m_x, m_y, \hat{m}_y)$, $N_y(m_x, m_y, \hat{m}_y)$, and $\hat{N}_y(m_x, m_y, \hat{m}_y)$. However, it is important to keep in mind that these are functions of all three variables when carrying out the differentiations below.

Note that although in the main text we use fixed functions for gamete production rate, gamete mortality rate, and zygote survival probability, the equations below are given in a general form, and can flexibly accommodate alternative forms for these functions. The model also allows independent values for A_x and A_y, but in the main text we limit ourselves to cases with an even adult sex ratio ($A_x=A_y$).

All the equations that follow are derived from the time dynamics Eqs. (A1a–A1c). For their biological interpretation, see the main text concerning Eqs. (3a–3b) and (4a–4c). Despite the fact that some of the resulting equations are relatively complex and difficult to interpret by themselves, the full biology of the system is already contained in Eqs. (A1a–A1c) together with the functions for gamete production, gamete mortality, and zygote survival. The following

equations are simply tools to determine the behavior of the system.

$$\begin{cases} \frac{dN_x}{dt} = A_x H(m_x) - \mu(m_x)N_x - \gamma N_x(N_y + \hat{N}_y) = 0 \\ \frac{dN_y}{dt} = (A_y - 1)H(m_y) - \mu(m_y)N_y - \gamma N_x N_y = 0 \\ \frac{d\hat{N}_y}{dt} = H(\hat{m}_y) - \mu(\hat{m}_y)\hat{N}_y - \gamma N_x \hat{N}_y = 0 \end{cases} \quad \text{(A1a–A1c)}$$

The fitness of the mutant individual is given by its rate of fertilizations ($\gamma N_x \hat{N}_y$, or alternatively $H(\hat{m}_y) - \mu(\hat{m}_y)\hat{N}_y$ from Eq. A1c) multiplied by the survival probability $f(m_x, \hat{m}_y)$ of its zygotes:

$$\hat{W}_y = \gamma N_x \hat{N}_y f(m_x, \hat{m}_y) = (H(\hat{m}_y) - \mu(\hat{m}_y)\hat{N}_y) f(m_x, \hat{m}_y) \quad \text{(A2)}$$

Therefore the selection differential is

$$\frac{\partial \hat{W}_y}{\partial \hat{m}_y}\Big|_{\hat{m}_y = m_y} = \left[(H(\hat{m}_y) - \hat{N}_y \mu(\hat{m}_y)) \frac{\partial f(m_x, \hat{m}_y)}{\partial \hat{m}_y} + f(m_x, \hat{m}_y) \left(\frac{dH(\hat{m}_y)}{d\hat{m}_y} - \hat{N}_y \frac{d\mu(\hat{m}_y)}{d\hat{m}_y} - \mu(\hat{m}_y) \frac{\partial \hat{N}_y}{\partial \hat{m}_y} \right) \right] \Big|_{\hat{m}_y = m_y} \quad \text{(A3)}$$

and the second derivative

$$\begin{aligned} \frac{\partial^2 \hat{W}_y}{\partial \hat{m}_y^2}\Big|_{\hat{m}_y = m_y} = \Bigg[& (H(\hat{m}_y) - \hat{N}_y \mu(\hat{m}_y)) \frac{\partial f^2(m_x, m_y)}{\partial \hat{m}_y^2} + 2 \frac{\partial f(m_x, \hat{m}_y)}{\partial \hat{m}_y} \left(\frac{dH(\hat{m}_y)}{d\hat{m}_y} - \hat{N}_y \frac{d\mu(\hat{m}_y)}{d\hat{m}_y} - \mu(\hat{m}_y) \frac{\partial \hat{N}_y}{\partial \hat{m}_y} \right) \\ & + \ldots\ldots f(m_x, \hat{m}_y) \left(\frac{d^2 H(\hat{m}_y)}{d\hat{m}_y^2} - \hat{N}_y \frac{d^2 \mu(\hat{m}_y)}{d\hat{m}_y^2} - 2 \frac{d\mu(\hat{m}_y)}{d\hat{m}_y} \frac{\partial \hat{N}_y}{\partial \hat{m}_y} - \mu(\hat{m}_y) \frac{\partial^2 \hat{N}_y}{\partial \hat{m}_y^2} \right) \Bigg] \Big|_{\hat{m}_y = m_y} \end{aligned} \quad \text{(A4)}$$

Everything in equations (A3) and (A4) is straightforward to derive, with the exception of $\frac{\partial N_x}{\partial \hat{m}_y}$, $\frac{\partial N_y}{\partial \hat{m}_y}$, $\frac{\partial \hat{N}_y}{\partial \hat{m}_y}$ and $\frac{\partial \hat{N}_y}{\partial \hat{m}_y^2}$. These can be found by using implicit differentiation with respect to $\hat{m}_y$ on equations (A1a–A1c). Differentiating once leads to the equations

$$\begin{cases} \gamma(N_y + \hat{N}_y) \frac{\partial N_x}{\partial \hat{m}_y} + \mu(m_x) \frac{\partial N_x}{\partial \hat{m}_y} + \gamma N_x \left(\frac{\partial N_y}{\partial \hat{m}_y} + \frac{\partial \hat{N}_y}{\partial \hat{m}_y} \right) = 0 \\ \gamma N_y \frac{\partial N_x}{\partial \hat{m}_y} + (\gamma N_x + \mu(m_y)) \frac{\partial N_y}{\partial \hat{m}_y} = 0 \\ \hat{N}_y \left(\frac{d\mu(\hat{m}_y)}{d\hat{m}_y} + \gamma \frac{\partial N_x}{\partial \hat{m}_y} \right) + (\gamma N_x + \mu(\hat{m}_y)) \frac{\partial \hat{N}_y}{\partial \hat{m}_y} = \frac{dH(\hat{m}_y)}{d\hat{m}_y} \end{cases} \quad \text{(A5a–A5c)}$$

and after differentiating twice we have

$$\begin{cases} 2\gamma\frac{\partial N_x}{\partial \hat{m}_y}\left(\frac{\partial N_y}{\partial \hat{m}_y}+\frac{\partial \hat{N}_y}{\partial \hat{m}_y}\right)+\gamma(N_y+\hat{N}_y)\frac{\partial^2 N_x}{\partial \hat{m}_y^2}+\mu(m_x)\frac{\partial^2 N_x}{\partial \hat{m}_y^2}+\gamma N_x\left(\frac{\partial^2 N_y}{\partial \hat{m}_y^2}+\frac{\partial^2 \hat{N}_y}{\partial \hat{m}_y^2}\right)=0 \\ 2\gamma\frac{\partial N_x}{\partial \hat{m}_y}\frac{\partial N_y}{\partial \hat{m}_y}+\gamma N_y\frac{\partial^2 N_x}{\partial \hat{m}_y^2}+\left(\gamma N_x+\mu(m_y)\right)\frac{\partial^2 N_y}{\partial \hat{m}_y^2}=0 \\ 2\left(\frac{d\mu(\hat{m}_y)}{d\hat{m}_y}+\gamma\frac{\partial N_x}{\partial \hat{m}_y}\right)\frac{\partial \hat{N}_y}{\partial \hat{m}_y}+\hat{N}_y\left(\frac{d^2\mu(\hat{m}_y)}{d\hat{m}_y^2}+\gamma\frac{\partial^2 N_x}{\partial \hat{m}_y^2}\right)+(\gamma N_x+\mu(\hat{m}_y))\frac{\partial^2 \hat{N}_y}{\partial \hat{m}_y^2}=\frac{d^2H(\hat{m}_y)}{d\hat{m}_y^2} \end{cases} \quad \text{(A6a–A6c)}$$

Now we can solve $\frac{\partial N_x}{\partial \hat{m}_y}$, $\frac{\partial N_y}{\partial \hat{m}_y}$, and $\frac{\partial \hat{N}_y}{\partial \hat{m}_y}$ from Eqs. (A5a–A5c):

$$\begin{cases} \frac{\partial N_x}{\partial \hat{m}_y}=\frac{\gamma N_x\left(\gamma Nx+\mu(m_y)\right)\left(-\frac{dH(\hat{m}_y)}{d\hat{m}_y}+\hat{N}_y\frac{d\mu(\hat{m}_y)}{d\hat{m}_y}\right)}{\gamma^2N_x^2\mu(m_x)+\left(\gamma N_y+\gamma\hat{N}_y+\mu(m_x)\right)\mu(m_y)\mu(\hat{m}_y)+\gamma N_x\left(\gamma N_y\mu(m_y)+\gamma\hat{N}_y\mu(\hat{m}_y)+\mu(m_x)\left[\mu(m_y)+\mu(\hat{m}_y)\right]\right)} \\ \frac{\partial N_y}{\partial \hat{m}_y}=-\frac{\gamma^2N_xN_y\left(-\frac{dH(\hat{m}_y)}{d\hat{m}_y}+\hat{N}_y\frac{d\mu(\hat{m}_y)}{d\hat{m}_y}\right)}{\gamma^2N_x^2\mu(m_x)+\left(\gamma N_y+\gamma\hat{N}_y+\mu(m_x)\right)\mu(m_y)\mu(\hat{m}_y)+\gamma N_x\left(\gamma N_y\mu(m_y)+\gamma\hat{N}_y\mu(\hat{m}_y)+\mu(m_x)\left[\mu(m_y)+\mu(\hat{m}_y)\right]\right)} \\ \frac{\partial \hat{N}_y}{\partial \hat{m}_y}=-\frac{\left(\gamma N_x\left[\gamma\hat{N}_y+\mu(m_x)\right]+\left[\gamma N_y+\gamma\hat{N}_y+\mu(m_x)\right]\mu(m_y)\right)\left(-\frac{dH(\hat{m}_y)}{d\hat{m}_y}+\hat{N}_y\frac{d\mu(\hat{m}_y)}{d\hat{m}_y}\right)}{\gamma^2N_x^2\mu(m_x)+\left(\gamma N_y+\gamma\hat{N}_y+\mu(m_x)\right)\mu(m_y)\mu(\hat{m}_y)+\gamma N_x\left(\gamma N_y\mu(m_y)+\gamma\hat{N}_y\mu(\hat{m}_y)+\mu(m_x)\left[\mu(m_y)+\mu(\hat{m}_y)\right]\right)} \end{cases} \quad \text{(A7a–A7c)}$$

and $\frac{\partial^2 \hat{N}_y}{\partial \hat{m}_y^2}$ from equations (A6a–A6c):

$$\frac{\partial^2 \hat{N}_y}{\partial \hat{m}_y^2}=\frac{-\left(\gamma N_x\left[\gamma\hat{N}_y+\mu(m_x)\right]+\left[\gamma N_y+\gamma\hat{N}_y+\mu(m_x)\right]\mu(m_y)\right)\left(-\frac{d^2H(\hat{m}_y)}{d\hat{m}_y^2}+\hat{N}_y\frac{d^2\mu(\hat{m}_y)}{d\hat{m}_y^2}+2\left(\frac{d\mu(\hat{m}_y)}{d\hat{m}_y}+\gamma\frac{\partial N_x}{\partial \hat{m}_y}\right)\frac{\partial \hat{N}_y}{\partial \hat{m}_y}\right)+2\gamma^2\hat{N}_y\frac{\partial N_x}{\partial \hat{m}_y}\left(\gamma N_x\frac{\partial N_y}{\partial \hat{m}_y}+\mu(m_y)\left(\frac{\partial N_y}{\partial \hat{m}_y}+\frac{\partial \hat{N}_y}{\partial \hat{m}_y}\right)\right)}{\gamma^2N_x^2\mu(m_x)+\left(\gamma N_y+\gamma\hat{N}_y+\mu(m_x)\right)\mu(m_y)\mu(\hat{m}_y)+\gamma N_x(\gamma N_y\mu(m_y)+\gamma\hat{N}_y\mu(\hat{m}_y)+\mu(m_x)\left[\mu(m_y)+\mu(\hat{m}_y)\right])}. \quad \text{(A8)}$$

Next, we solve equations (A1a–A1c) for N_x, N_y, and $\hat{N}_y$ when $\hat{m}_y = m_y$:

$$\begin{cases} N_x\big|_{\hat{m}_y=m_y}=\frac{A_x\gamma H(m_x)-A_y\gamma H(m_y)-\mu(m_x)\mu(m_y)+\sqrt{4A_x\gamma H(m_x)\mu(m_x)\mu(m_y)+\left[-A_x\gamma H(m_x)+A_y\gamma H(m_y)+\mu(m_x)\mu(m_y)\right]^2}}{2\gamma\mu(m_x)} \\ N_y\big|_{\hat{m}_y=m_y}=\left(A_y-1\right)\left(\hat{N}_y\Big|_{\hat{m}_y=m_y}\right) \\ \hat{N}_y\Big|_{\hat{m}_y=m_y}=\frac{-A_x\gamma H(m_x)+A_y\gamma H(m_y)-\mu(m_x)\mu(m_y)+\sqrt{4A_x\gamma H(m_x)\mu(m_x)\mu(m_y)+\left[-A_x\gamma H(m_x)+A_y\gamma H(m_y)+\mu(m_x)\mu(m_y)\right]^2}}{2A_y\gamma\mu(m_y)} \end{cases} \quad \text{(A9a–A9c)}$$

Finally we get $\frac{\partial \hat{W}_y}{\partial \hat{m}_y}\Big|_{\hat{m}_y=m_y}$ by sequentially plugging Eqs. (A9a–A9c) into (A7c), and then plugging this result together with (A9c) into (A3). Similarly $\frac{\partial^2 \hat{W}_y}{\partial \hat{m}_y^2}\Big|_{\hat{m}_y=m_y}$ is calculated by the sequence (A9)→(A7)→(A8)→(A4).

An analytic expression for the convergence stability matrix C can be found by following steps similar to the ones described above, but both the methods and the resulting equations are more complex. We omit them from this appendix, but the equations are available from the authors upon request.

We now have the tools to find the equilibrium points and evolutionary trajectories for gamete sizes, and to determine their stability. The existence of a closed form analytic solution for the equilibrium points depends on the form of zygote fitness, gamete mortality, and gamete production functions used. With the functions we use in the main text, analytic solutions are not possible. However, we can use the following equation to find the equilibriums and plot the evolutionary trajectories to an arbitrary level of accuracy:

$$\begin{pmatrix} m_{x_{i+1}} \\ m_{y_{i+1}} \end{pmatrix} = \begin{pmatrix} m_{x_i} \\ m_{y_i} \end{pmatrix} + \begin{pmatrix} \frac{\partial \hat{W}_x}{\partial \hat{m}_x}\Big|_{\hat{m}_x=m_{x_i}} \\ \frac{\partial \hat{W}_y}{\partial \hat{m}_y}\Big|_{\hat{m}_y=m_{y_i}} \end{pmatrix} \Delta \qquad \text{(A10)}$$

where Δ is a sufficiently small number.

Once the equilibria m_x^* and m_y^* are found, the following conditions must be satisfied:

1. $\frac{\partial \hat{W}_x}{\partial \hat{m}_x}\Big|_{\hat{m}_x=m_x^*} = 0$, $\frac{\partial \hat{W}_y}{\partial \hat{m}_y}\Big|_{\hat{m}_y=m_y^*} = 0$; this means there is no directional selection on gamete size
2. $\frac{\partial^2 \hat{W}_x}{\partial \hat{m}_x^2}\Big|_{\hat{m}_x=m_x^*} \le 0$, $\frac{\partial^2 \hat{W}_y}{\partial \hat{m}_y^2}\Big|_{\hat{m}_y=m_y^*} \le 0$; this shows that the equilibrium is an ESS.
3. The real parts of the eigenvalues of the matrix C must be negative to show convergence stability.

Table 1 Conditions for the equilibria in Figs. 3 and 5

	m_x^*	m_y^*	$\frac{\partial \hat{W}_x}{\partial \hat{m}_x}\Big\vert_{\hat{m}_x=m_x^*}$	$\frac{\partial \hat{W}_y}{\partial \hat{m}_y}\Big\vert_{\hat{m}_y=m_y^*}$	$\frac{\partial^2 \hat{W}_x}{\partial \hat{m}_x^2}\Big\vert_{\hat{m}_x=m_x^*}$	$\frac{\partial^2 \hat{W}_y}{\partial \hat{m}_y^2}\Big\vert_{\hat{m}_y=m_y^*}$	Eigenvalue 1	Eigenvalue 2	Stability
3a (I)	1.0295556	1.0295556	0.0000000	0.0000000	−220.1571560	−220.1571560	−290.5661482	−228.8912094	ESS, CS
3b (I)	2.5629265	2.5629265	0.0000000	0.0000000	−0.0332809	−0.0332809	−0.7948520	0.2258166	ESS, not CS
3b (A)	7.7643734	1.0472158	0.0000000	0.0000000	−0.0523215	−307.5952457	−0.0522994	−307.6528428	ESS, CS
3c (I)	5.1248802	5.1248802	0.0000000	0.0000000	−0.0035626	−0.0035626	−0.0979423	0.0483636	ESS, not CS
3c (A)	17.8442571	1.0471633	0.0000000	0.0000000	−0.0054256	−157.9421049	−0.0054252	−157.9472522	ESS, CS
5a (I)	8.7251872	8.7251872	0.0000000	0.0000000	−0.0304892	−0.0304892	−0.0392724	−0.0217061	ESS, CS
5b (I)	6.4733466	6.4733466	0.0000000	0.0000000	−0.0304003	−0.0304003	−0.0514911	−0.0401946	ESS, CS
5b (A)	17.8463470	1.0477984	0.0000000	0.0000000	−0.0053972	−78.1954535	−0.0053960	−78.1895663	ESS, CS
5c (I)	5.5233488	5.5233488	0.0000000	0.0000000	−0.0149510	−0.0149510	−0.0793415	−0.0039228	ESS, CS
5c (A)	17.8445040	1.0472940	0.0000000	0.0000000	−0.0054164	−131.1748599	−0.0054158	−131.1769317	ESS, CS
5d (I)	5.1248802	5.1248802	0.0000000	0.0000000	−0.0035626	−0.0035626	−0.0979423	0.0483636	ESS, not CS
5d (A)	17.8442571	1.0471633	0.0000000	0.0000000	−0.0054256	−157.9421049	−0.0054252	−157.9472522	ESS, CS
5e (I)	7.1922359	7.1922359	0.0000000	0.0000000	−0.0091769[a]	−0.0091769[a]	−0.0228255[a]	0.0044718[a]	ESS, not CS
5e (A)	17.9403142	1.0550288	0.0000000	0.0000000	−0.0048372[a]	−7.3920755[a]	−0.0048251[a]	−7.3920876[a]	ESS, CS
5f (I)	7.2727273	7.2727273	0.0000000	0.0000000	−0.0097619	−0.0097619	−0.0229472	0.0034234	ESS, not CS
5f (A)	17.9285515	1.0564173	0.0000000	0.0000000	−0.0048910	−5.9459726	−0.0048756	−5.9459880	ESS, CS

All values are presented to seven decimals, although the values were computed to a much higher accuracy

The leftmost column refers to figures, with the additional capital letters in brackets (*I*) and (*A*) referring to the isogamous and anisogamous equilibria respectively

All equilibria are evolutionarily stable strategies (*ESS*), but some isogamous equilibria are not convergence stable (*CS*)

[a] These values refer to the case γ=0.1 in Fig. 5e. For the alternative of M=10, the marked values must be divided by 10. Stability is not affected, as this does not change the sign. Other values remain unchanged.

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