The evolution of anisogamy: The adaptive significance of damage, repair and mortality

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Abstract

Classic theory on the evolution of anisogamy focuses on the trade-off between gamete productivity and provisioning and mechanisms associated with post-zygotic survival. In this article, the role of mortality acting on both zygotes and gametes is explored as a factor influencing the evolution of different sized gametes. In particular, variable mortality through differential survival or metabolic damage is shown to affect the persistence of isogamy, the evolution of more than two sexes and the evolution of anisogamy. Evolutionary stable isogamous states are shown to be locally unstable and disruptive selection can induce the evolution of anisogamy. Analysis of both the isogamous and anisogamous ESS points reveals that the persistence of either of these conditions is not always assured. The implications of variable survival on the evolution of anisogamy are discussed.

1. Introduction

The ‘evolution of sex’ is a contemporary theme in evolutionary biology and covers at least four broad areas (Felsenstein, 1988): sex differentiation (Bull, 1981), anisogamy, costs of mating and recombination (Maynard Smith, 1971) and outcrossing (Berstein et al., 1988). The ‘evolution of sex’ has predominantly focused on the latter two processes (Michod and Levin, 1988; Burt, 2000). However, central to all these problems and controversies over costs of mating and recombination (e.g. Barash, 1976; Maynard Smith and Williams, 1976; Birdsell and Willis, 2003) is the adaptive significance of sexual dimorphism and particularly how selection has favoured the evolution of anisogamous (different sized) gametes in higher organisms.

A variety of explanations have been proposed for the evolution of anisogamy. These include the role of differential mating types (Charlesworth, 1980, 1994; Hoekstra, 1982, 1990), the effects of cytoplasmic conflict (Hurst and Hamilton, 1992; Hutson and Law, 1993) and gamete motility or attraction (Cox and Sethian, 1984, 1985; Dusenbery, 2000, 2002). Most recently, Czárá and Hoekstra (2004) have shown that evolution of mating systems and the evolution of anisogamy might rely on heterogeneous or spatially explicit differences in the distribution of gametes, reminiscent of the ‘Strawberry-coral model’ (Williams, 1975).

However, the classic theory for the origin of anisogamy is based on a productivity versus provisioning conflict (Parker et al., 1972; Bell, 1978). Given that a fixed amount of resource is available for producing gametes then the evolutionary dilemma is whether to invest in a small number of well-provisioned gametes or...
a large number of small gametes. There may be selective advantages to producing many gametes, provided this increases the future reproductive success of a given individual (Parker et al., 1972). A further condition necessary for the evolution of gamete dimorphism is that zygote fitness increases in a nonlinear way with changes in zygote size (Parker et al., 1972; Bell, 1978; Charlesworth, 1978). While evidence for the evolution of anisogamy as a function of resource availability and zygote survival is relatively well established (e.g. Madsen and Waller, 1983), recent phylogenetically controlled analyses reveal that the basic tenets of the theory may rely on specific untested assumptions (Randerson and Hurst, 2001a). Recent commentaries on the evolution of gamete dimorphism (Randerson and Hurst, 2001b; Bulmer et al., 2002) highlight that understanding the nature of the relationship between zygote fitness and size is integral for anisogamy to evolve. However, this remains unverified. Further, it is entirely plausible that other factors affecting gamete survival are also of equal importance in driving disruptive selection acting on isogamous gametes (Bulmer and Parker, 2002).

2. Metabolic damage and evolution of sex

One broad way that zygote and gamete survival might be impaired is through damage associated with metabolic and cellular processes. There is a growing body of theory and experiments showing that metabolic damage, particularly damage associated with oxidative processes, can have major consequences that cascade from the molecular and cellular levels to affect fitness of the whole organism (von Schantz et al., 1999; de Boer et al., 2002; Alonso-Alvarez et al., 2004). Metabolic damage through the production of oxygen or nitrogen-derived free radicals can affect all biomolecules including DNA, proteins and lipids, it can affect cell behaviour (restricting cell division) and induce cell death (Halliwell and Gutteridge, 1999). There is also increasing evidence that metabolic damage can influence a number of biological characters such as ageing (e.g. Beckman and Ames, 1998), susceptibility to disease (e.g. Parthasarathy et al., 1992) and sex (e.g. Alonso-Alvarez et al., 2004).

Damage to biomolecules, such as DNA, and repair hypotheses have been proposed as explanations for the ‘evolution of sex’ (Berstein et al., 1988; Michod et al., 1988; Wojciechowski et al., 1989; Hoelzer and Michod, 1991). Recently, it has been argued that metabolic damage might be a major determinant of the ‘evolution of sex’ (Allen, 1996; Nedelec and Michod, 2003; Nedelec et al., 2004). For example, in Volvox carteri, metabolic and, in particular, oxidative stresses have recently been shown to induce sexual reproduction (Nedelec and Michod, 2003). In particular, increased levels of oxygen free radicals trigger gamete formation, the development of a diploid zygospore and DNA repair mechanisms (through meiosis). An interesting finding here is that gamete formation and facultative sex is a derived state as V. carteri produces anisogamous (sperm, eggs) reproductive cells.

3. Biological motivation

Gamete dimorphism is a widespread phenomenon and bipolar differentiation of gametes (into male and female types) is undeniably the characteristic separating the sexes. However, in motivating the study of the evolution of different sexes, it is important to distinguish between the evolution of different sized gametes (pseudo-anisogamy) and the evolution of different types of gametes (anisogamy, oogamy).

Distinguishing between different sized and different types of gametes on the evolution of anisogamy can be resolved by defining the types of isogamous or anisogamous gametes. Bell (1978) highlights four different strategies: isogamy (where the gametes are of equal size), pseudo-anisogamy (where gametes are of different size), anisogamy (where fusion occurs between gametes of different sizes) and oogamy (where fusion occurs between large and small gametes).

In the former two cases (isogamy and pseudo-anisogamy), fusion occurs randomly between gametes. There is no distinction between different types of gametes and the evolution of anisogamy occurs through additional evolutionary processes acting on preferential mating strategies. For instance, recent work has suggested that additional biologies (e.g. phototactic responses) are necessary in order to understand how pseudo-anisogamy might evolve and persist (Togashi and Cox, 2004).

In the latter two cases (anisogamy and oogamy), fusion occurs between gametes of different sizes. As such, it is implicit that distinct gametes exist. Gametic dimorphism is often correlated with physiological complexity (Bell, 1978; Randerson and Hurst, 2001a,b). For instance, complex multicellular algae are more likely to have anisogamous (or oogamous) gametes than unicellular species (Bell, 1978). However, while this pattern has been observed (Bell, 1978), appropriately structured evolutionary analyses of these observations remain scarce (Randerson and Hurst, 2001a).

Here, in this study, the presence (of at least) two types of gametes, the functional relationship between gamete fitness and frequency, and zygote fitness and size are argued to be fundamental in understanding the evolution of anisogamy. Central to this objective is how metabolic damage might be the driver for the evolution of anisogamy. In particular, the goal is to determine the conditions under which disruptive selection leads to the evolution of an anisogamous condition by coupling an
ESS approach for the evolution of anisogamy (Maynard Smith, 1978; Bulmer, 1994; Matsuda and Abrams, 1999; Bulmer and Parker, 2002) with the ideas associated with cellular biochemistry and metabolic damage. Underpinning the theoretical framework developed here are the gamete and zygote survival functions. The derivation of these functions from associated theory on metabolism and growth is introduced. It is shown that damage can be described as a set of linear chains of ordinary differential equations (ODEs). In the results section, the evolution of anisogamy is explored in terms of static and dynamic anisogamous and isogamous ESSs. It is shown that although metabolic damage can induce disruptive selection on the isogamous state, a fixed anisogamous condition might also not be evolutionary stable. These findings are discussed with reference to the consequences for the persistence of fixed anisogamous strategies and, recent advancements in the ‘evolution of sex’.

4. Model framework

The model is a development of the size-number trade-off framework introduced by Parker et al. (1972) and developed by Maynard Smith (1978), Matsuda and Abrams (1999), and most recently by Bulmer and Parker (2002).

Gamete fitness (defined as net reproductive potential) is assumed to be a function of the number of gametes produced (with a fixed amount of resource for reproduction), gamete viability and zygote survival. Individuals produce a fixed number ($n_i$) of gametes of size $s_i$ with available resources $R_i$ that survive (with probability $g(s_i)$) to fuse and form a zygote. Zygotes of size $\sum_j s_j$ (the fusion of $s_j$ gametes) have a finite probability of surviving to become adult that is related to their size ($f(\sum_j s_j)$). The fitness of different gametes is measured by the number of surviving zygotes that they contribute to, and as such the fitness ($w$) of a gamete of type $i$ is

$$w_i = \frac{R_i g(s_i)}{s_i} f\left(\sum_j s_j\right),$$

where $g(s_i)$ and $f(\sum_j s_j)$ are the gamete and zygote survival functions, respectively. In particular, in the baseline model (following Bulmer and Parker, 2002) it is assumed that these survival functions are related to size such that

$$g(s_i) = \exp\left(-\frac{\alpha}{s_i}\right)$$

and

$$f\left(\sum_j s_j\right) = \exp\left(-\frac{\beta}{\sum_j s_j}\right),$$

where $\alpha$ and $\beta$ are the background mortality rates of gametes and zygotes, respectively. The broad aim here is to consider how different probabilities of survival affect the evolution of anisogamy. To test this hypothesis, the effects of more complicated survival functions that encapsulate different biologies are explored.

4.1. Complex survival functions

Central to understanding how anisogamy might evolve is the survival of gametes and zygotes: larger gametes or zygotes are expected to have higher survival probabilities (Maynard Smith, 1978). In this section, explanation for the derivation of more complex survival functions is presented: three survival functions are used to explore different biologies on the evolution of anisogamy. First, a generic function to explore how variability in survival affects the evolution of anisogamy is introduced. Next, how distributed mortality affects the evolution of anisogamy is presented. In specifying this functional form no underlying mechanism for mortality is proposed other than that the probability of survival is distributed over the lifespan of the gametes (or zygotes). Finally, a more explicit function exploring how damage (which impairs functionality) might accumulate is developed. In particular, it is assumed that damage might accumulate through metabolic processes associated with size. These different survival functions are compared and contrasted with the baseline model (Eqs. (1)–(3)).

4.2. Variable mortality

Morphologically, isogamy is relatively widespread, particularly amongst unicellular organisms (Scudo, 1967; Bell, 1978). However, this does not preclude the possibility that individual variability in gamete size (and consequently survival) might occur, nor does it exclude the likelihood that fusion between different types of gametes (pseudo-anisogamy) might occur. As such, gamete or zygote survival is unlikely to be constant or fixed at some background level (as in Eqs. (2)–(3)), and one way to describe variability in survival (or mortality) of gametes as a function of their size is by

$$g(s_i) = \exp\left(-\frac{(s_i - G_0)^2}{2\sigma_j^2}\right),$$

where $G_0$ is the optimal gamete size and $\sigma_j$ is the variability around a mean gamete size. Similarly, variability in zygote survival can be described by

$$f\left(\sum_j s_j\right) = \exp\left(-\frac{(\sum_j s_j - Z_0)^2}{2\sigma_z^2}\right),$$

where $Z_0$ is the optimal zygote size and $\sigma_z$ is the variability around the optimum zygote size.
4.3. Distributed mortality

The accumulation of gamete and zygote damage and mortality could be manifest through a range of different cellular processes and reactions (Halliwell and Gutteridge, 1999). If the accumulation of damage through the multiple mechanisms associated cellular metabolism leads to functional impairment that ultimately affects survival, then the accumulation of mortality can in the most general setting be considered to depend upon gamete size, number and the environment. Thus, in this most general case, changes in gamete (or zygote) numbers can be expressed as:

\[
\frac{dn_i}{dt} = - \left[ \int_0^T \mu(z, n_i(z), E(z)) \, dz \right] n_i(t),
\]

where \(\mu(z, n_i(z), E(z))\) is the mortality over the interval \(z\) to \(z + \delta z\) when the number of gametes of size \(i\) is \(n_i\), and the environment is of type \(E\) (MacDonald, 1978, 1989). MacDonald (1978, 1989) introduced a technique, the linear chain trick, for dealing with such a situation. Although, this technique has been used to explore a range of processes such as the distribution of disease incubation periods, insect maturation times and population dynamics (MacDonald, 1989), the linear chain trick method is used here in a phenomenological way to account for the suite of complex biochemical processes as a source of damage (Mangel and Bonsall, 2004) that might affect gamete (or zygote) survival.

This technique works by decomposing the integral (Eq. (6)) into a series (or chain) of linear ODEs. For instance, distributed mortality on the number of gametes \((n_j)\) of size \(s_i\) (or zygotes of size \(\sum s_j\)) can be described by the following linear chain:

\[
\frac{dn_j}{dt} = - \gamma_q n_j(t), \quad \frac{d\gamma_q}{dt} = v(\gamma_q - \gamma_{q-1}), \quad \frac{d\gamma_0}{dt} = \left( \frac{\mu}{s_i} - \gamma_0 \right),
\]

where \(\mu/s_i\) is the baseline mortality, \(\gamma_q\) is an auxiliary variable \((\gamma_q = 0, 1, 2, \ldots, q,\) where \(q\) is the length of the chain) describing the distribution of mortality and \(v\) is the shape of the distribution. Both the integral and set of ODEs are identical and allow mortality to be evaluated over a particular interval of time \([0, T]\).

Linear chains are described by two main parameters: the length of the chain \((q)\) and the shape of the distribution \((v)\). Each of these parameters within the chain have a clear biological interpretation. Increases in chain length can be viewed as novel physiological innovations while changes in the shape parameter govern the rate at which damage cascades through a chain. This shape parameter can be thought of as a descriptor of biochemical function. The solution of these equations for a chain of length \(q = 1\) over the lifespan \((T)\) of the gametes (or zygotes) is

\[
n(T) = \exp \left( -\frac{\mu}{s} (1 - \exp(-vT))(1 - \exp(-vT)) \right) n(0), \quad (10)
\]

where \(n(0)\) is the initial number of gametes (or zygotes) produced with energy allocation \(R\) \((n(0) = R/s)\). Given the lifespan of the gametes is implicitly assumed in the simpler survival functions (Eqs. (2) and (3)) to be a constant, \(T\) is assumed to be of fixed, constant time. If distributed mortality acts on gametes then the fitness of gametes of size \(i\) is described by

\[
w_i = \frac{R_i}{s_i} \exp \left( -\frac{\mu}{s_i} (1 - \exp(-v)) \right) \left( \sum_j s_j \right). \quad (11)
\]

Similarly, if distributed mortality operates on zygotes then fitness is given by

\[
w_i = \frac{R_i}{s_i} g(s_i) \exp \left( -\frac{\mu}{\sum_j s_j} (1 - \exp(-v)) \right). \quad (12)
\]

4.4. Metabolic damage

As mentioned, one specific way in which mortality and survival might be affected is through the processes associated with metabolism. There is increasing evidence that metabolic damage and oxidative stress can impair the function of DNA (Halliwell and Gutteridge, 1999). DNA strand cleavage occurs due to the effects of a range of free radical generating factors such as ozone, hydrogen peroxide and radiation. Hydroxyl free radicals, for instance, can affect DNA by altering the biochemical function and structure of sugars, purines and pyrimidines. This oxidative damage is highly detrimental to the functionality of DNA and a suite of repair mechanisms (such as the role of polymerase and photolyase enzymes, excision repair mechanisms and mismatch repair mechanisms) act to restrict and control the extent of metabolic damage (Halliwell and Gutteridge, 1999).

Recently it has been proposed that the way in which these fundamental cellular processes might cascade up to affect survival is through power-law scalings (West et al., 2001). As such damage \((D)\) might accumulate as:

\[
\frac{dD}{dr} = k_s^0.75 - p_0 s, \quad (13)
\]

where \(k\) is a measure of the baseline effects of metabolism on damage, \(s\) is gamete (or zygote) size and \(p_0\) is the (constant) rate of damage repair (Mangel and Bonsall, 2004). To explore how the accumulation of damage might be affected by the rate of biochemical reactions and/or the degree of physiological innovation,
Again a linear chain can again be used to described this distributed damage \((D)\)
\[
\frac{dD}{dt} = \gamma_q,
\]
\[
\frac{d\gamma_q}{dt} = \omega (\gamma_q - \gamma_{q-1}),
\]
\[
\frac{d\gamma_0}{dt} = \omega (k_s^{0.75} - p os - \gamma_0),
\]
where \(\gamma\) is an auxiliary variable \((\gamma_0 = 0, 1, 2, \ldots, q;\) where \(q\) is the length of the chain) describing the distribution of damage on gametes (or zygotes) and \(\omega\) is the shape of the distribution. The solution to this chain (of length \(q = 1\)) is
\[
D = (k^{0.75} - p os)(1 - \exp(-\omega T))(1 - \exp(-\omega T)) + C,
\]
where if \(T = 0\) and \(D = 0\) then \(C = 0\), and \(g(s_i) = D\) or \(f(\sum_j s_j) = D\). Again, \(T\) is assumed to a fixed constant representing the total lifespan of a gamete (or zygote). In both the distributed mortality and metabolic damage scenarios, survival is a mixture of exponentials. Model analysis proceeds by determining the conditions for the isogamous and anisogamous ESSs. Under static fitness models, stability of an ESS is determined by evaluating the second derivative of the fitness function. Continuous stability is assured if this slope at the ESS is negative and greater than \(-1\). Solutions of the first derivative of the fitness function (derived either analytically or using a root-solving algorithm) provide the conditions for identifying the evolutionary stable conditions for anisogamy. The dynamics of the ESS points are explored using stability and bifurcation analysis techniques.

5. Results

Analysis of the models proceeds by determining how the log fitness of gamete size \((\ln(w_i))\) is shaped by different gamete or zygote survival functions, and how this affects the persistence of isogamy or the evolution of anisogamy.

5.1. Persistence of isogamy: Static fitness models

5.1.1. Variable mortality

The log change in gamete fitness with respect to size when variable mortality acts on gametes (Eq. (4)) is given by
\[
\frac{d \ln(w_i)}{ds_i} = -\frac{1}{s_i} - \frac{s_i - G_0}{\sigma_s} + \frac{\beta}{(\sum_j s_j)^2}.
\]
The isogamous ESS (when \(s_i = s^*\) and \(i = 1, 2\)) is the solution to this expression when there is no change in fitness \((w_i)\) with respect to size \((s^*)\)
\[
-\frac{1}{s^*} - \frac{s^* - G_0}{\sigma_s} + \frac{\beta}{2s^2} = 0.
\]
Similarly, the log change in gamete fitness with respect to size when variable mortality acts on zygotes (Eq. (5)) is given by
\[
\frac{d \ln(w_i)}{ds_i} = -\frac{1}{s_i} - \frac{2(s_i - Z_0)}{\sigma_z} + \frac{\alpha}{s_i^2}
\]
and the isogamous ESS when variable mortality acts on zygotes is evaluated from
\[
-\frac{1}{s^*} - \frac{2(2s^* - Z_0)}{\sigma_z} + \frac{\alpha}{s^*^2} = 0.
\]
Compared to the fixed survival models (Eqs. (1)–(3)), introducing variability in survival can influence gamete fitness and the evolution of anisogamy. This occurs simply because there is an optimum size for gametes or zygotes, and departure from this leads to a decline in fitness (Fig. 1A) as size departs from the optimum. At the isogamous ESS, variability in mortality acting on gametes is unstable and disruptive selection away from this point is expected to occur (as the slope of the second derivative is less than \(-1\)). In contrast, isogamy under variability in mortality on zygotes is considered to be at least locally stable (as the slope of the second derivative of Eq. (19) is greater than \(-1\)) (Fig. 1B).

5.1.2. Distributed mortality

Under distributed mortality acting on gametes (Eq. (10)), the log change in gamete fitness with respect to size is given by
\[
\frac{d \ln(w_i)}{ds_i} = \frac{1}{s_i} + \ln(g'(s_i)) - \frac{\beta}{\sum_j s_j},
\]
where \(g'(s_i)\) is the derivative of the gamete survival function. The isogamous ESS is evaluated when \(s_i = s^*\) for \(i = 1, 2\) and under this function for survival is
\[
s^* = \frac{\beta}{4} + \frac{\alpha}{4}(1 - \exp(-\upsilon q)),
\]
where \(\upsilon\) is the shape parameter and \(q\) is the length of the chain. When distributed mortality acts on zygotes, the log change in gamete fitness with respect to size is given by
\[
\frac{d \ln(w_i)}{ds_i} = \frac{1}{s_i} - \frac{\alpha}{s_i} + \ln \left( f'(\sum_j s_j) \right),
\]
where \(f'(\sum_j s_j)\) is the derivative of the zygote survival function, and the isogamous ESS is then
\[
s^* = \frac{\beta(1 - \exp(-\upsilon q))}{4} + \alpha.
\]
Similarly, \(\upsilon\) is the shape parameter and \(q\) is the length of the linear chain.
In contrast to the fixed survival model, distributed mortality allows gametes (Fig. 2) and zygotes (Fig. 3) to achieve higher fitness. This arises as the probability of mortality is distributed over the lifespan of the gamete and/or zygote and discounts the instantaneous effects (hazard) of mortality. Higher fitness compared to the baseline model is possible if chain length increases (gametes: Fig. 2B, zygotes: Fig. 3B) or the shape parameter declines (gametes: Fig. 2C, zygotes: Fig. 3C).

Stability of the isogamous ESS with distributed mortality acting on gametes is ensured if the slope of the second derivative of the fitness function (evaluated at the ESS) is negative and greater than $-1$. Under distributed mortality the disruptive selection away from the isogamous ESS is possible. However, long linear chains or small shape parameters affect the stability of the isogamous state. For example, from Fig. 2A, the slope of the second derivative of the fitness function at the ESS is on the boundary of stability (slope = $-1$). In contrast, under low shape parameters (Fig. 2B), the slope of the second derivative is less than $-1$. Continuous stability can not be assured and disruptive selection would favour the evolution of an anisogamous condition. However, longer chains (more physiological complexity) favour the persistence of the isogamous state (the slope of the second derivative is greater than $-1$ at the isogamous ESS). Generally, the isogamous state is not a global equilibrium and depends on the precise conditions leading to the fitness optimum. Perturbations away from this state can favour the evolution of anisogamy.

In contrast, stability of the isogamous ESS under distributed zygote mortality follows a different pattern. Under this form of mortality (Fig. 3A), slow rates of
Fig. 2. The effects of distributed gamete mortality on the evolution of anisogamy. (A) Distributed mortality with short chain length ($q = 1$) and shape parameter $\nu = 1$. (B) Distributed mortality with short chain length ($q = 1$) and small shape parameter $\nu = 0.1$. (C) Distributed mortality with long chain length ($q = 10$) and shape parameter $\nu = 1$. Insets show how the fitness functions for the baseline model (dashed line: Eqs. (1)–(3)) and the distributed mortality model (solid line: Eqs. (1) and (10)) change with respect to gamete size. (Other parameter values were $x = 1$, $\beta = 1$).

Fig. 3. The effects of distributed zygote mortality on the evolution of anisogamy. (A) Distributed mortality with short chain length ($q = 1$) and shape parameter $\nu = 1$. (B) Distributed mortality with short chain length ($q = 1$) and small shape parameter $\nu = 0.1$. (C) Distributed mortality with long chain length ($q = 12$) and shape parameter $\nu = 1$. Insets show how the fitness functions for the baseline model (dashed line: Eqs. (1)–(3)) and the distributed mortality model (solid line: Eqs. (1) and (10)) change with respect to gamete size. (Other parameter values were $x = 1$, $\beta = 1$).
reaction (Fig. 3B) and complex physiology (long chains: Fig. 3C) promote the continuous stability of the isogamous ESS as the slope of the second derivatives is greater than −1. Other things being equal, distributed mortality of gametes is more disruptive than mortality acting on zygotes and as such changes in gamete survival properties are more likely to favour the evolution of anisogamy.

5.1.3. Metabolic damage

Under metabolic damage, the log change in gamete fitness (Eqs. (1) and 17)) with respect to size is given by

\[ \frac{d \ln(w_i)}{ds_i} = \frac{1}{s_i} + \ln\left(g'(s_i)\right) + \ln\left(f\left(\sum_j s_j\right)\right), \quad (26) \]

where \( g'(s_i) \) and \( f\left(\sum_j s_j\right) \) are the derivatives of the gamete and zygote survival functions, respectively (from Eq. (17)). Under the survival function given by Eq. (17) acting solely on gamete survival (and \( i = 1, 2 \)), the solution for the isogamous ESS under metabolic damage is evaluated from

\[ -(1 - \exp(-\omega))^k \left(\frac{0.75k}{s_i^{0.25}} - p_0\right) + \frac{x}{s_i} \]

\[ - \frac{1}{s_i} + \frac{\beta}{(s_i + s_j)^2} = 0. \quad (27) \]

Similarly, the solution for the isogamous ESS under metabolic damage acting only on zygotes is evaluated from:

\[ -(1 - \exp(-\omega))^k \left(\frac{0.75k}{s_i^{0.25}} - p_0\right) + \frac{x}{s_i} \]

\[ - \frac{1}{s_i} + \frac{\beta}{(s_i + s_j)^2} = 0. \quad (28) \]

In contrast to the baseline model, metabolic damage acting on gametes (Fig. 4) or zygotes (Fig. 5) leads to lower fitness. As might be expected, metabolic damage is costly in terms of reproduction. Fitness optima are determined by the strength of metabolic damage (\( k \)) and the rate at which this damage can be repaired (\( p_0 \)).

Rather than qualitatively changing the stability properties, metabolic damage acting on gametes scales the ESS conditions. In fact under complex physiologies (long chains) the stability properties are almost identical to the baseline model. Long chains allow the effects of metabolic damage to be dissipated. Similar patterns are observed under slow rates of reaction (small shape parameters). Continuous stability of the isogamous ESS is assured as the slopes of the second derivatives of the fitness function with respect to changes in size are negative and greater than −1.

The isogamous ESS points are only locally stable and again the evolution of anisogamy is possible under complex physiologies and/or biochemical complexities. However, under this scenario of gamete or zygote survival, there is also the possibility of runaway selection. That is, the formation of large isogamous gametes is entirely plausible. The isogamous ESS is unstable and although the fitness function declines around this local unstable point (Fig. 4A), there is a second fitness minima beyond which, as gamete size increases, fitness increases (Figs. 4A and 5A). This uncontrolled increase is countered by the costs imposed through damage either in the length of the chain (increased physiological costs through innovation) or in changes in the rates of mortality propagating through the chain (shape parameters) (Figs. 4B and C). Under metabolic damage acting on zygotes (Fig. 5), reducing the rates of the biochemical processes (Fig. 5B) or allowing physiological innovations (Fig. 5C) favours the persistence of the isogamous ESS (continuous stability of the fitness function is negative and greater than −1). Similar to the distributed mortality model, damage to zygotes is less disruptive than metabolic damage on gametes for the evolution of anisogamy. In contrast to the distributed mortality model, there remains the possibility that metabolic damage on isogamous gametes can lead to runaway selection on gamete size.

5.1.4. Can there be more than two sexes?

The possibility that more than two sexes can evolve can also be explored under the framework presented. Here, the evolution of more than two types of gamete is considered under the metabolic damage model (Eq. (17)). Proposing this question on the evolution of more than two sexes necessitates a consideration of the mating structure under scrutiny. At least three plausible scenarios exist; two of these involve the differential fusion of different mating types (e.g. Iwasa and Sasaki, 1987; Hurst, 1996; Bull and Pease, 1989) while the third involves the fusion of multiple gametes. It is this latter scenario that is explored here. Zygote fitness is now a function of multiple (more than two) types of gamete, and under the metabolic damage framework (Eq. (17)), the fitness function (Eq. (1)) is replaced with

\[ w_i = \frac{R_i}{s_i} \exp\left[-\frac{x}{s_i}\right] \frac{\beta}{\sum_j s_j} \left(1 - \exp(-\omega)^k\right) \]

\[ \times \left(\frac{k}{\sum_j s_j^{0.25}} - p_0 \sum_j s_j\right). \quad (29) \]

Given that isogamy and diploidy are likely to be the ancestral traits, comparisons are made between fitness optima and stability of the isogamous condition where there are two (\( i = 1, 2 \)) or three (\( i = 1, 2, 3 \)) different types of gamete. Results show that the possibility for more than two sexes is restricted but not impossible. Under different scenarios of metabolic damage, diploid zygotes have higher fitness optima than triploid zygotes.
Fig. 4. Metabolic damage acting on gametes and the evolution of anisogamy. (A) Metabolic damage under a short chain ($q = 1$) and constant shape parameter ($\omega = 1.0$). (B) Metabolic damage under a short chain ($q = 1$) and low shape parameter ($\omega = 0.1$). (C) Metabolic damage under a long chain ($q = 10$) and constant shape parameter ($\omega = 1.0$). Insets show how the fitness functions for the baseline model (dashed line: Eqs. (1)–(3)) and the metabolic damage model (solid line: Eqs. (1) and (17)) change with respect to gamete size. (Other parameter values were $\alpha = 1$, $\beta = 10$, $k = 2$, $p_0 = 1$).

Fig. 5. Metabolic damage acting on zygotes and the evolution of anisogamy. (A) Metabolic damage under a short chain ($q = 1$) and constant shape parameter ($\omega = 1.0$). (B) Metabolic damage under a short chain ($q = 1$) and low shape parameter ($\omega = 0.1$). (C) Metabolic damage under a long chain ($q = 10$) and constant shape parameter ($\omega = 1.0$). Insets show how the fitness functions for the baseline model (dashed line: Eqs. (1)–(3)) and the metabolic damage model (solid line: Eqs. (1) and (17)) change with respect to gamete size. (Other parameter values were $\alpha = 1$, $\beta = 1$, $k = 2$, $p_0 = 1$).
However, under complex physiological innovations (long chains) diploid and triploid zygotes have equivalent fitness optima (Fig. 6C): the evolution of more than two sexes can occur given sufficient physiological innovations. Comparing the stability of the isogamous ESS with different types of gamete illustrates that the presence of an additional gamete is no more likely to induce the evolution of anisogamy. Continuous stability conditions remain relatively unaffected (slope of the second derivative is negative and greater than \( \frac{C_1}{C_0} \)) by the evolution of additional gametes (Fig. 6).

5.1.5. Dynamics of the evolution of anisogamy

The effects of gamete and zygote survival on the evolution of anisogamy can be investigated in more detail by examining the dynamics around the neighborhood of the known ESS points. In order to do this it is necessary to consider the rate at which each gamete is likely to increase. By considering the rate of change of gamete size as a product of heritability and the change in fitness with change in gamete size (\( \frac{\partial w_1}{\partial s_1} \)) at a particular ESS point (\( s^* \))

\[
\frac{ds_1}{dr} = h_1^2 \frac{\partial w_1}{\partial s_1} |_{s_1=s_2=s^*}, \tag{30}
\]

where \( h_1^2 \) is the heritability (ratio of the additive genetic variance to the total phenotypic variance) of gamete type \( s_1 \), then the evolutionary dynamics of anisogamy can be explored. Given this is a coupled system (zygotes are fused from (at least) two gametes), the rate of change of a second mating type \( s_2 \) also needs to be considered and is described by

\[
\frac{ds_2}{dr} = h_2^2 \frac{\partial w_2}{\partial s_2} \bigg|_{s_1=s_2=s^*}. \tag{31}
\]

Similarly, \( h_2^2 \) is the heritability of gamete type \( s_2 \) and \( \frac{\partial w_2}{\partial s_2} \) is the partial derivative of the fitness function for mating type \( s_2 \) evaluated at a particular ESS point \( s^* \). Analysis of the dynamics around the neighborhood of an ESS proceeds by evaluating the trace and determinant of the linearized versions of these equations (Eqs. (30)–(31)), and determining the characteristics of the associated eigenvalues.

Under variable mortality on gametes (Eq. (4)) the evolutionary dynamics around the ESS are unstable as the isogamous ESS is an unstable saddle point (the eigenvalues are real but have opposite signs). In contrast, variable mortality on zygotes (Eq. (5)) is stabilizing as the isogamous ESS (Fig. 1B) is a stable node (both eigenvalues are real and negative) over a wide range of variability (\( \sigma_z \)) in zygote size.
Under distributed mortality acting on gametes (Eq. (10)) the dynamics around the isogamous state are unstable as the isogamous ESS (Fig. 2) is an unstable saddle point. Similarly, the anisogamous ESS is also an unstable saddle point (Fig. 2). Distributed mortality on zygotes is globally unstable as the isogamous ESS is either an unstable saddle point (Fig. 3A) or an unstable node (both eigenvalues are positive)(Fig. 3B and C). Similar dynamics are observed under damage accumulation mortality acting on zygotes and gametes (Figs. 4 and 5): both isogamous and anisogamous ESS points are locally unstable saddle points.

6. Discussion

Here, a series of models have been presented to explore the conditions under which anisogamy might evolve. A number of results emerge from these analyses. First, it has been shown that variability in gamete and zygote survival can induce disruptive selection. Second, metabolic damage (appropriately described through a series of linear ODEs) leads to nonlinear consequences on the evolution of anisogamy. Third, evolutionary stable strategies are at best only locally stable and a series of bifurcations are expected around both the isogamous and anisogamous ESS.

Principally, the evolution of anisogamy was thought to be driven by mechanisms associated with post-zygotic survival (Maynard Smith, 1978; Matsuda and Abrams, 1999). In contrast, it is shown here that anisogamy can evolve through the effects of variability in mortality or distributed metabolic costs acting principally on morphologically identical, isogamous gametes. Under this hypothesis, disruptive selection on isogamous gametes would favour individuals that produce smaller gametes to compensate for the costs associated with metabolic damage. Dealing with metabolic damage on gametes effectively involves the innovation of novel physiologies (increases in chain length) or reductions in biochemical reaction (chain shape parameters) in order to minimize the fitness costs of metabolic damage. One possible physiological innovation is to evolve disproportionately smaller gametes. A second (physiological innovation) is to reduce mitochondria leakage and the impact of free radicals on cellular function (e.g. Allen, 1996). Either way, it is clear that metabolic damage and DNA repair mechanisms might be a major driver not only on the evolution of recombination (Berstein et al., 1988; Michod et al., 1988) but also on the evolution of anisogamy. Appreciating the difference between gamete competition (high $n_i$) and gamete survival (limitation) is instrumental in understanding the evolution of anisogamy. Investment in many small gametes over a single large, well-provisioned gamete has focused attention on the post-zygotic mechanisms for the evolution of anisogamy.

Appreciating that gamete survival (limitation) can also influence the evolution of anisogamy (Levitan, 1996) through the processes associated with metabolic damage is a clear novel prediction of the work presented here.

The evolution of more than two sexes has attracted some interest (Weinshall, 1986; Iwasa and Sasaki, 1987; Roughgarden, 1991; Hurst, 1996; Iwanaga and Sasaki, 2004; Parker, 2004). Weinshall (1986) argued that a simple two-state environment was insufficient to explain the evolution of sex while Roughgarden (1991) suggested that the costs of recombination could be offset in a fluctuating environment. Hurst (1996) and Iwanaga and Sasaki (2004) examined the role of cytoplasmic conflict and costs of mating to explained why two sex systems have evolved. Most recently, it has been argued that the organization of genetic information with complex social groups such as the social Hymenoptera might provide evidence of an evolutionary transition to more than two sexes (Parker, 2004). This argument, however, seems to depend on the detailed complexities associated with a haplo-diploid mating system. In the current study, it has clearly been shown that there are no substantial fitness benefits to the evolution of more than two mating strategies. In fact, under metabolic damage, having more that two mating types can have fitness impediments. The evolutionary transition to sex as a mechanism for dealing with metabolic stress (Allen, 1996) probably has more profound effects on the evolution of anisogamy than any transition to more than two sexes. Notwithstanding, the evolutionary innovations underpinning these transitions are only starting to be appreciated and refinements (e.g. knowledge of genetic architecture, dominance and epistasis) of the theory presented here are required to comprehend more fully the conditions as to whether more than two sexes might evolve.

6.1. Persistence of anisogamy

As noted by Matsuda and Abrams (1999), it is interesting to explore why isogamous gametes are so rare. Disruptive selection and post-zygotic survival are the key processes allowing the evolution of anisogamy. However, it is equally important to examine the conditions that allow the persistence of anisogamy. Here, it has been shown that an anisogamous ESS may also be a locally unstable state. This suggests that the instability of both the isogamous and anisogamous statistics will leads to evolutionary tension between two disruptive selective process. It is predicted that there is no fixed end point and once selection acts to differentiate gametes (based on size), it will also prevent an unique end state to the process of the evolution of anisogamy.

Understanding why anisogamy is the ubiquitous condition is likely to require the consideration of other evolutionary biological processes. For instance, variation in mating success can promote the evolution of
anisogamy (Levitan, 1996) and greater variance in the mating success of one strategy will affect not only reproductive behaviour but also other aspects of the evolutionary biology of sex (Williams, 1975; Boudry et al., 2002). Variation in female mate success in the sea urchin Strongylocentrotus franciscanus can influence the evolution of gamete dimorphisms. Intense selection on gametes for increased fertilization success occurs in both sexes and gamete (sperm) limitation can lead to divergent selection on isogamous gametes (Levitan, 1996). Differential survival of mating types is also likely to favour the evolution and persistence of anisogamy. Variance in reproductive success in Pacific oyster (Crassostrea gigas) has been linked to three main factors: gamete quality, sperm-egg interactions and differential zygote viability (Boudry et al., 2002). Difference in gamete maturation, sperm competition and unbalanced parental contributions might all skew reproductive success and bias the evolution of anisogamy. Similar variance in mate success might be afforded through differential provisioning of zygotes: anisogamy might be favoured if there is more equitable investment in the zygote. This can occur at the moment of fertilization (Bressac et al., 1994) through to continued parental investment (Clutton-Brock, 1991).

Appreciating how anisogamy has evolved requires a broader class of processes to be explored. Here, it has been shown that the dynamics of anisogamy are continuously unstable and we might not expect either condition to persist under a productivity-provisioning conflict. It has been suggested that different mechanisms associated with survival disrupt the isogamous state and affect the evolution of anisogamy. However, it is also likely that the incorporation of additional life-history traits such as polyandry, parental investment and sexual selection are important determinants of the evolution of different-sized gametes (Andersson, 2004). More consideration needs to be given to incorporating these processes with the mechanisms of variable gamete and zygote survival: understanding the evolution of anisogamy and its implications for the ‘evolution of sex’ clearly needs more pluralistic approaches.

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