Koopman, B.O. (1980). *Search and Screening: General Principles with Historical Applications*. New York: Pergamon Press.
Lessells, C.M., Snook, R.R., and Hosken, D.J. (2009). The evolutionary origin and maintenance of sperm: selection for a small, motile gamete mating type. In T.R. Birkhead, D.J. Hosken, and S. Pitnick (editors), *Sperm Biology: An Evolutionary Perspective*. Academic Press, Amsterdam, pp. 43–67.
Maire, N., Ackermann, M., and Doebeli, M. (2001). Evolutionary branching and the evolution of anisogamy, *Selection*, **2**, 119–131.
Margulis, L. and Sagan, D. (1986). *Origins of Sex*. New Haven, CT: Yale University Press, p. 195.
Maynard Smith, J. (1978). *The Evolution of Sex*. Cambridge: Cambridge University Press.
Nozaki, H. (2008). A new male-specific gene "*OTOKOGI*" in *Pleodorina starrii* (Volvocaceae, Chlorophyta) unveils the origin of male and female. *Biologia*, **63**, 772–777.
Parker, G.A., Baker, R.R., and Smith, V.G.F. (1972). The origin and evolution of gamete dimorphism and the male-female phenomenon. *Journal of Theoretical Biology*, **36**, 529–553.
Peierls, R.E. (1960). Wolfgang Ernst Pauli. 1900–1958. *Biographical Memoirs of Fellows of the Royal Society*, **5**, 175–192.
Roney, H.C., Booth, G.M., and Cox, P.A. (2009). Competitive exclusion of cyanobacterial species in the Great Salt Lake. *Extremophiles*, **13**, 355–361.
Scudo, F.M. (1967). The adaptive value of sexual dimorphism. I. Anisogamy. *Evolution*, **21**, 285–291.
Strasburger, E., Jost, L., Schenck, H., and Karsten, G. (1921). *A Text-book of Botany*, 4th edn. London: Macmillan and Co.
Togashi, T., Bartelt, J.L., and Cox, P.A. (2004). Simulation of gamete behaviors and the evolution of anisogamy: reproductive strategies of marine green algae. *Ecological Research*, **19**, 563–569.
Togashi, T. and Cox, P.A. (2004). Phototaxis and the evolution of isogamy and "slight anisogamy" in marine green algae: insights from laboratory observations and numerical experiments. *Botanical Journal of the Linnean Society*, **144**, 321–327.
Togashi, T., Nagisa, M., Miyazaki, T., Yoshimura, J., Bartelt, J.L., and Cox, P.A. (2006). Gamete behaviours and the evolution of "marked anisogamy": reproductive strategies and sexual dimorphism in Bryopsidales marine green algae. *Evolutionary Ecology Research*, **8**, 617–628.
Williams, G.C. (1975). *Sex and Evolution*. Princeton, N.J.: Princeton University Press, p. 113.
Willson, M.F. (1994). Sexual selection in plants: perspective and overview, *The American Naturalist*, **144**, S13–S39.
Wright, S. (1977). *Evolution of the Genetics of Populations: Experimental Results and Evolutionary Deductions*, Vol. 3. Chicago, IL: University of Chicago Press.
Yang, J.-N. (2010). Cooperation and the evolution of anisogamy. *Journal of Theoretical Biology*, **264**, 24–36.

GEOFF A. PARKER

1

The origin and maintenance of two sexes (anisogamy), and their gamete sizes by gamete competition

1.1 INTRODUCTION

It is generally assumed (e.g. Maynard Smith, 1978; 1982) that ancestrally, gametes were small and isogamous (monomorphic). The evolution of anisogamy (gamete dimorphism) is a crucial transition in evolution (Maynard Smith and Szathmáry, 1995): it represents the evolution of the two sexes, males and females. Following Parker *et al.* (1972), I favor defining a sex in relation to the type of gamete a sexual phenotype carries. A sex is thus an adult phenotype defined in terms of the size of (haploid) gamete it produces: in an anisogamous population, males produce microgametes and females produce macrogametes. A simultaneous hermaphrodite is thus both male and female simultaneously, and a sequential hermaphrodite transforms sequentially from male to female (or vice versa). This definition of a sex differs from one that defines a sex in terms of gamete mating types (e.g. Wiese, 1981; Hoekstra, 1990). Under the Parker *et al.* definition of a sex in terms of gamete size, a mating type is not considered to be a sex, but simply a gametic type (that may or may not be related to gamete size) that shows a preference for fusion with certain other gamete types. In isogamous populations, there is thus one sex (though there may be several mating types). Retaining the definition of a sex for an adult phenotype that produces a given gamete size, and a mating type for a gamete phenotype that has a given characteristic for selective fusion may serve to remove some of the confusions that have arisen in the literature.

The Evolution of Anisogamy: A Fundamental Phenomenon Underlying Sexual Selection, ed. Tatsuya Togashi and Paul Alan Cox. Published by Cambridge University Press.

This initial sexual asymmetry in gamete size, anisogamy, plays the central role in driving all the dramatic pre- and postcopulatory processes of Darwinian sexual selection. This divergence in the two different gamete size-producing morphs, males and females, results in the skewing of parental investment (Trivers, 1972), operational sex ratios (Emlen and Oring, 1977), potential reproductive rates (Clutton-Brock and Vincent, 1991), or whatever index is proposed to account for the intensity of sexual selection.

The present review considers the origin, maintenance, and development of anisogamy and gamete size through the selective pressures of competition between gametes for gametic fusion, and focuses mainly on the work of my colleagues and myself over the past 3–4 decades. The initial selective force of gamete competition, coupled with the evolution of increasing vegetative complexity, usually in the form of complex multicellular organization (another major transition in evolution; Maynard Smith and Szathmáry, 1995) is argued to offer the most plausible origin of anisogamy from an ancestral isogamous, externally fertilizing state (Parker *et al.*, 1972; Bulmer and Parker, 2002). The subsequent development of internal fertilization greatly reduced the potential for sperm competition, which resulted in a reduction in ejaculate expenditure, allowing sexual selection to become intense and generating high anisogamy ratios (*A* = ovum cell mass/sperm cell mass, Parker, 1982; for a different definition of anisogamy ratio, see Togashi *et al.*, 2007).

An excellent survey of all theories relating to the evolution of anisogamy (including gamete competition) is given in the recent review of Lessells *et al.* (2009); theories unrelated to gamete competition are also reviewed in the present volume.

1.2 THE ORIGIN OF ANISOGAMY BY DISRUPTIVE SELECTION ON GAMETE SIZE THROUGH GAMETE COMPETITION (PBS THEORY)

Parker, Baker, and Smith (1972) proposed a theory (often referred to as the PBS theory or model, e.g. Bell, 1978; Lessells *et al.*, 2009) for the evolution of anisogamy and two sexes by gamete competition, based on disruptive selection on individuals varying in the size of gamete they produced. The essence of our theory, summarized early in the 1972 paper, relates to two simple assumptions:

> Two very fundamental pressures immediately appear obvious; both would be related to gamete size and would act in opposition. These are *numerical productivity* (i.e. the number of gametes produced in unit time by a given parent) and *zygote fitness* (i.e. a measure of the probability that a zygote will survive to reach adulthood and reproduce, and in the shortest time).

We pointed out that, all else equal, in an ancestral population where gametes are shed into the sea, adults would have maximum fitness by producing the maximum number of gametes capable of carrying the haploid chromosomes and surviving to fuse with another gamete, but that there would not be a drive to produce maximum gamete numbers "if parents producing fewer but larger gametes experienced a compensating advantage because of the greater fitness of their offspring" (PBS).

To investigate the implications of these two very simple assumptions about gamete productivity and zygote provisioning, PBS examined a computer simulation with the following more detailed assumptions: (1) there is an ancestral isogamous population with wide variation in the mass, m, of each gamete produced, (2) each individual has the same fixed resources, M, for allocation to gametes, so that there is a direct trade-off between gamete size and number, n, with $n = M/m$, (3) gamete size is controlled by alleles at a single locus, with either diploid control ("small-producing" dominant over "large-producing," or vice versa in different simulations) or haploid control (by the allele carried by the gamete, or by the haploid parent), (4) gametes are released simultaneously by individuals into an external medium (envisaged to be the sea), and fusion between all gametes is random, (5) the size (mass), S, of a zygote is the sum of the masses of the two fusing gametes, i.e. $S_{ij} = m_i + m_j$, and (6) the "fitness", f, of a zygote is an increasing function of zygote mass (f is most simply viewed as survival to reproduction, but includes all aspects related to reproductive success).

The PBS simulations used an explicit function for zygote fitness, namely that $f(S) = aS_{ij}^{x}$, where a is a proportionality constant. Our results, confirmed by further simulations (Parker, 1978) to correct an error in the original computations, were as follows. Whatever the genetic system (haploid or diploid control), low values for exponent x caused fixation of the allele for producing the smallest gametes (isogamy with microgametes). When x became sufficiently high, there was a stable polymorphism of alleles for producing the smallest (microgametes) and the largest (macrogametes); all other alleles became lost from the population. At even higher x, the result was fixation of the allele for producing the largest gametes (isogamy with macrogametes). The range of exponent x over which anisogamy is stable depended on the range of gamete sizes

present in the population; as x increased, the range of x allowing anisogamy increased (examples of the simulations are shown in Figures 1.1a, 1.1b). Further, if (i) there is a vast range of gamete sizes in the ancestral population, (ii) $x > 1$, and (iii) $f(2\delta) \rightarrow 0$, where δ = the minimum size for a gamete to survive, the surviving adults are approximately equal numbers of two genotypes, one that produces microgametes (proto-sperm producers) and one that produces macrogametes (proto-ovum producers).

The interplay between exponent x (which defines how steeply zygote fitness increases with zygote size) and the ratio of maximum to minimum gamete sizes is thus critical in determining which of the three solutions (i.e. microgamete isogamy, anisogamy, or macrogamete isogamy) is achieved in the ancestral population. The PBS simulations (Parker, 1978) suggested that with a very wide range of gamete sizes, the critical exponent x value was 1.0 (i.e. linearity) for the threshold between microgamete isogamy ($x < 1$) and anisogamy ($x > 1$).

Analytical treatments using population genetics approaches (Bell, 1978; Charlesworth, 1978) supported the PBS results. For haploid control of gamete size (the dominant phase of the life cycle for many green algae is the gametophyte which is haploid) they showed that, where θ = the ratio of minimum/maximum gamete size ($\theta = m_{\min}/m_{\max} = n_{\max}/n_{\min}, 0 < \theta < 1$), the range of x for stable anisogamy is obtainable from:

$$2^{-x}\theta^{1-x}(1+\theta)^{x} > 1, \tag{1.1a}$$

$$2^{-x}\theta^{-x}(1+\theta)^{x} > 1 \tag{1.1b}$$

(Figure 1.1c). They also derived the equilibrium allele or genotype frequencies, confirming for both haploid and diploid cases that the ratio of microgamete producers to macrogamete producers approaches equality (a unity sex ratio) as the disparity in size between the two gamete-producing morphs becomes very great.

PBS also discussed their general assumptions about gamete productivity and zygote provisioning in terms of a general and more plausible form for $f(S)$ than the power function $f(S) = aS_{ij}{}^{x}$, which was used for heuristic purposes in their simulations. They argued that the most plausible form for $f(S)$ would be one in which $f(S) = 0$ up to some minimum S, after which $f(S)$ would rise steeply with decreasing slope. They argued that in complex multicellular organisms, whatever the starting conditions, selection would drive towards conditions favoring anisogamy, and that simple unicellular organisms were, however, more likely to remain isogamous (see Section 1.2.5).

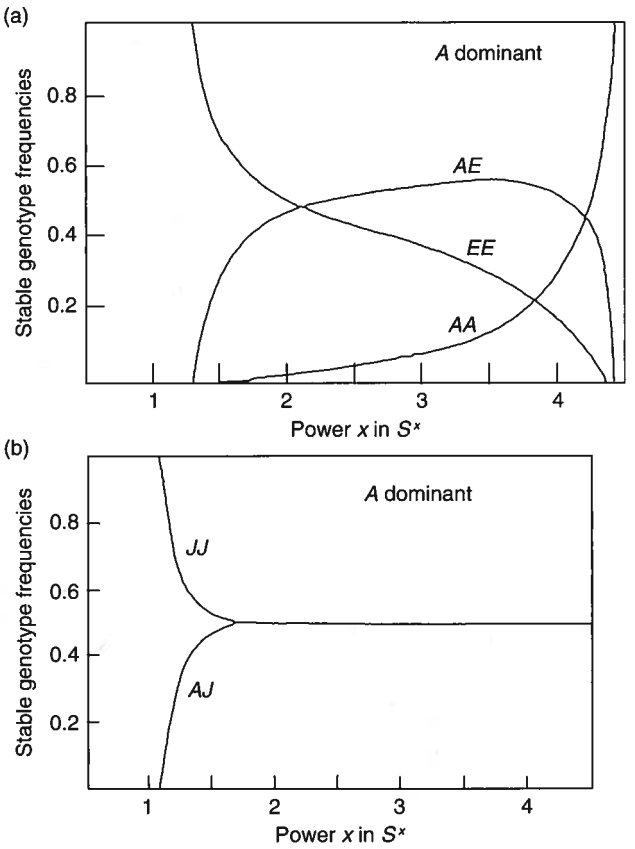

Figure 1.1 Isogamy and anisogamy in the PBS model. (a) and (b) (from Parker, 1978) show the stable genotype frequencies attained in the PBS simulations in relation to power x in the zygote fitness function, $f(S) \propto S^{x}$, starting with three alleles for number of cell divisions, v, that determine gamete productivity, n, where $n = 2^{v}$. Alleles for low productivity (large gametes) are dominant over those for high productivity (small gametes). In simulation (a) the three alleles are A, C, E, with $v = 1, 3, 5$, giving $n = 2, 8, 32$ (dominance series A: C: E). In (b) the three alleles are A, E, J, with $v = 1, 5, 10$, giving $n = 2, 32, 1024$ (dominance series A: E: J). (c) Charlesworth's (1978) haploid conditions for isogamy and anisogamy in the PBS model. Anisogamy is stable between the upper and lower curves, which show the critical x values from conditions (1a) and (1b); θ = the ratio of minimum/maximum gamete size. Fixation of the smallest gamete size occurs below the lower curve (condition (1a)), and fixation of the largest gametes size above the upper curve (condition 1b)). (Redrawn with permission from B. Charlesworth).

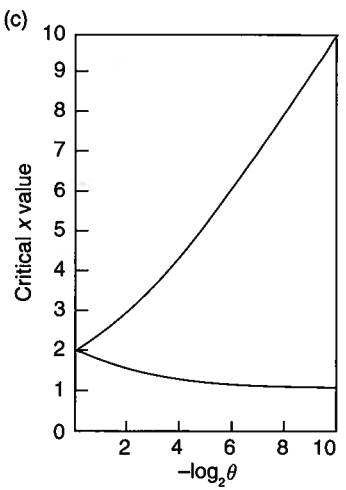

Figure 1.1 (cont.)

Maynard Smith (1978, 1982) used a graphical analysis based on game theory to investigate the basic PBS model, generating conditions for the two types of isogamy or anisogamy. However, caution is required regarding Maynard Smith's isogamous optimum, m^*, which occurs at a zygote size of $S = 2m^*$, where $m^* > \delta$, where δ is the minimum possible gamete size for survival before fusion. This solution, at which $f'(S) = 2f(S)/S$, was shown subsequently to be continuously unstable by Matsuda and Abrams (1999, see also Bulmer and Parker 2002), and also by McNamara *et al.* (2003) in the context of Parker's (1985) analogous ESS solution for biparental care. Thus although an ESS, selection will not converge to it. Maynard Smith's isogamous gamete size ESS, m^*, is nevertheless important in determining how the system behaves in relation to δ (Matsuda and Abrams, 1999; Bulmer and Parker, 2002): we expect isogamy or anisogamy depending on whether $\delta > m^*$ or $\delta < m^*$. If $0 < \delta < m^*$, the system is expected to move towards an anisogamous ESS with $m_1^* = \delta$ and $m_2^* = S^* - \delta$, where $S^* - \delta$ is solved from $f'(S) = f(S)/(S - \delta)$. But when $\delta > m^*$, the system will move to an isogamous ESS with gametes of size δ.

1.2.1 Mating types and the PBS theory

The PBS model was based on random fusion between all gametes. A central problem discussed by PBS and later investigated in detail by Parker (1978), again by computer simulation, was why disassortative fusion for gamete size evolved, in which microgametes fuse only with

macrogametes: essentially, why should proto-sperm and proto-ova become mating types, rather than fuse randomly? This question assumes that disassortative fusion arises under what has been defined as *pseudoanisogamy* (see Scudo, 1967; Bell, 1982; Lessells, 2009), i.e. a state where there is gamete dimorphism but no mating types – or if there are mating types, these fuse independently of size. Several other authors have modeled the evolution of anisogamy starting from ancestral populations without mating types (e.g. Bell, 1978; Charlesworth, 1978; Cox and Sethian, 1985). An alternative possibility is that the prior existence of mating types in the ancestral population played a seminal role in the evolution of anisogamy, a possibility that was first investigated by Charlesworth (1978).

1.2.1.1 *Evolution of disassortative fusion from anisogamy without mating types*

PBS (see also Parker, 1978) used the following argument to account for the evolution of disassortative fusions. Assuming that there is no risk of remaining unfused, sperm producers would be favored if they produced sperm (mass $m_1 = \delta$) that avoided sperm–sperm fusions and fused selectively with ova (mass $m_1 = \delta$). However, ovum producers would also do better by producing gametes that fuse selectively with other ova. There thus arises a "primordial sexual conflict." PBS argued that sperm producers were likely to "win" this conflict for two reasons. First, there will be a higher adaptation rate in sperm. The mutation rate is likely to be proportional to the number of gametes produced, and will therefore differ for the two sexes. Sperm producers would have experienced a higher incidence of mutants favoring sperm–ovum fusions than ovum producers experienced to prevent such fusions. Second, sperm-producers were likely be under stronger selection to avoid sperm–sperm fusions and to favor sperm–ovum fusions, than ovum-producers are to avoid sperm–ovum fusions, especially if sperm–sperm fusions are almost lethal ($f(2\delta) << f(m_2 + \delta)$) and the fitness function $f(S)$ is increasing with decreasing slope (ovum–ovum fusions thus have less than the twice the fitness of sperm–ovum fusions even if $\delta \to 0$; $f(2m_2) < 2f(m_2 + \delta)$).

PBS argued that even if assortatively fusing ova "won" so that the existing sperm producers became extinct, the system would not be stable: the resulting isogamous population would experience new drives of sperm producers. In the absence of sperm, the advantage in maintaining antisperm selectivity would be lost and may erode. A mutation for gamete size reduction in one of the existing ovum producers could probably invade with or without this erosion, because it

would possess surface characteristics of the ovum mating type. PBS likened the sperm–ovum relationship to that between parasite and host, with parasitic sperm producers dependent upon and propagating at the expense of the host ovum producers.

Parker (1978) investigated the fate of various mutant mating types with size-selective fusion in an anisogamous population with random fusion to examine a further possibility: that mutant assortatively fusing ova (i.e. ova that fuse only with other ova) may fail to encounter other ova, especially when there is: (i) sperm competition and (ii) a time limit for fusion, after which the gamete dies, or cannot fuse for some other reason (for an alternative perspective on size-selective fusion relating to gamete encounters, see Cox and Sethian, 1985). A phenotypic (non-genetic) model considered the fitness of various mutant strategies for selective fusion, each occurring in males and females with equal probability, in a randomly fusing population. Defining the gamete mating-type behavior as A = assortative (i.e. fusion with the same gamete size), D = disassortative (i.e. fusion with the other gamete size), and R = random, and using subscripts O for ova and S for sperm, the various strategies become, for example, A_OA_S (the parent produces ova or sperm that fuse assortatively), A_OD_S (the parent produces ova that fuse assortatively and sperm that fuse disassortatively), etc. The population strategy is R_OR_S (random fusion by both gamete types). If the fitness of a zygote arising from an ovum–ovum fusion is standardized as 1, then following the original PBS formulation for $f(S) \propto S^x$, the products of ovum–sperm fusion have fitness $b = (0.5 + 0.5/n)^x$, and sperm–sperm fusions $c = (1/n)^x$. If all gametes can fuse without difficulty, A_OD_S will obviously be the best strategy ($1 > b > c$). A_OR_S will always invade R_OR_S, and A_OA_S and D_OD_S will sometimes invade, depending on n and x. Strategies D_OR_S, D_OA_S, and R_OA_S can be discounted – their fitness can never exceed that of the parental population, R_OR_S.

Whether selection favors selective fusion, and if so, what form of selective fusion, depends on the risk of a gamete's failure to achieve fusion before the gamete dies. In Parker's (1978) model, fertilization of mutant gametes occurred successively during P fertilization time intervals or steps; P can be regarded as a gamete survival time. If the probability of fusion in a given step is T (a measure of the "aptitude for fusion"; Scudo, 1967), the probability of not being fused by step P is $(1 - T)^P$. The best strategy depended on T and the number of steps (Figure 1.2 shows some results with $T = 0.1$ and $x = 2$). If gametes can survive only one step, random fusion (R_OR_S) was always best, because any mutant selective fusion strategy lost all gametes that "refused" to fuse. But if gametes

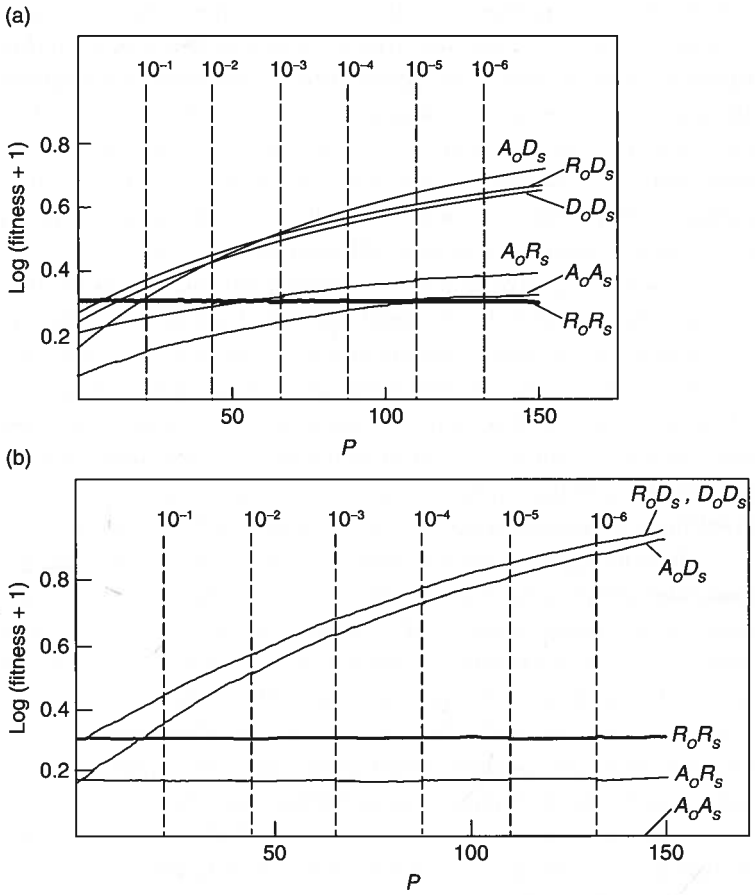

Figure 1.2 Comparison of fitnesses of rare mutant selective fusion strategies after P steps in a fertilization process with the fitness of the population strategy in which both ova and sperm show random fusion (R_OR_S). The broken vertical lines indicate the proportion of R_OR_S gametes that remain unfused at step P; different P values can be regarded as different gamete lifetimes. At low P, random fusion (R_OR_S) always has the highest fitness. A = assortative (i.e. fusion with the same gamete size), D = disassortative (i.e. fusion with the other gamete size), and R = random; subscript O = ova and S = sperm. The proportion of gametes fusing per step is $T = 0.1$, and power $x = 2$. (a) Low anisogamy ratio, with number of sperm per ovum = $n = 10$. (b) High anisogamy ratio, with number of sperm per ovum = $n = 10^5$. With permission from Parker (1978).

could survive several steps, mutant selective fusion strategies were able to invade (Figure 1.2); the most advantageous selective fusion strategy depended on the degree of anisogamy. Thus at low levels of anisogamy (10^1, Figure 1.2a), the first strategy to beat R_OR_S was R_OR_D, followed by D_OD_S, and then A_OD_S (by step P = 19). With T = 0.1, by step P = 22, proportion 10^{-1} of the gamete pool remained unfertilized. As P increased, the fitnesses of the other viable selectively fusing strategies A_OD_S, A_OR_S, and eventually A_OA_S had all overtaken that of R_OR_S.

At high anisogamy ratios (10^5, Figure 1.2b), the fitness of strategies lacking D_S sperm is very small because of the low viability and high probability of sperm–sperm unions. Thus R_OD_S and D_OD_S are favored if they survive only two steps, and A_OD_S beats R_OR_S by P = 17. A_OD_S eventually beats R_OD_S and D_OD_S, but only when almost all of them have fused, requiring a $P > 1020$. Thus if there is simultaneous spawning, to be successful at high anisogamy ratios, A_OD_S mutants must rely on selfing of A_O ova. They are outcompeted for ova by R_S sperm.

Thus the apparent advantage of A_OD_S over A_RD_S (representing the primordial sexual conflict) can be lost if there is a high anisogamy ratio before the A_O mutant arises from A_R, and if there is a sufficient risk of gamete death before fusion. If randomly fusing gametes are used up quickly in the gamete pool relative to mutant selectively fusing gametes, A_O ova may die before being able to fuse, or be forced to fuse with other mutant A_O ova from the same parent, producing inbred zygotes comparable to the products of apomixis or parthenogenesis. However, disassortatively fusing sperm, D_S, generally have a high advantage when anisogamy would be favored under random fusion. Genetic simulations involving diploid control two loci, one with alleles for fusion behavior and the other with alleles for gamete size, with T=0.1 and P = 100, were used to confirm these conclusions. Without sex limitation, fusion genes code for selective or random fusion independently of gamete size produced (i.e. for strategies D_{OS}, A_{OS}, or R_{OS}). This simulation yielded near fixation of disassortative fusion genes (for D_{OS}) in conjunction with stable anisogamy, provided the anisogamy ratio was reasonably high. But because of the polymorphism, random-fusion genes (for R_{OS}) were not entirely eliminated unless n was very high. Simulations with various sex-limited fusion genes showed that genes for the non-limited disassortative fusion strategy, i.e. D_{OS}, won at high anisogamy ratios against all strategies but the one which played the sex-limited strategy, R_OD_S, but at high anisogamy ratios it has only a very small advantage over non-limited disassortative fusion (D_{OS}). I argued that the reasons for the establishment of non-limited disassortative fusion are probably related to avoiding

selfing, and to the cost of maintaining random fusion in ova (in terms of motility, etc.) outweighing the benefits of becoming obligatorily disassortative, e.g. non-motile (Parker, 1978). With asynchrony of spawning, disassortative fusion alleles may do even better than with perfect synchrony, because the probability of remaining unfertilized decreases.

The view of the evolution of anisogamy and disassortative fusion as being the primordial sexual conflict has been attacked by Iyer and Roughgarden (2008), following the line of the "social-selection program," purporting that Darwinian sexual selection is fallacious and that sexual cooperation operates rather than sexual conflict (Roughgarden *et al.*, 2006). They state: "... that contradictory to Parker *et al.*'s claim (1972), proto-ova may actually gain from fusing with proto-sperm as compared to selectively fusing with other proto-ova." The purpose of my 1978 paper was exactly to demonstrate that assortatively fusing ova can be selected against if the risk of gamete death before fusion is sufficiently high (Figure 1.2); Iyer and Roughgarden fail to mention that if the risks of gamete death before fusion are *not* sufficiently high, there is sexual conflict because assortatively fusing ova will indeed do best, as my paper showed (Parker, 1978). (In the original PBS model, all gametes fused, so the PBS model was correct to stress that our scenario would favor assortatively fusing ova; Parker (1978) set out to examine what happened when gametes do *not* always manage to fuse.)

Sexual conflict implies a difference between optima for the sexes (see Parker, 1979; 2006 and other articles in the same volume; Arnqvist and Rowe, 2005); conditions for conflict cannot be deduced by comparison of two separate and different hypothetical populations. For reasons that are therefore entirely unclear, Iyer and Roughgarden (2008) attempt to show that anisogamy does not involve conflict by comparing the fitness of a "female" individual in an isogamous "male" population all producing "egg-size sperm," with that of a similar female in a true male population producing microgametes. The important point for conflict is simply that in a given population, an ovum will do best to fuse randomly if fusion rates are sufficiently low, or to reject fusions with sperm and to accept those with ova if fusion rates are sufficiently high, depending on the risk that an ovum may fail to fuse before it dies (Parker, 1978). Iyer and Roughgarden appear to have misunderstood that primordial sexual conflict is *not* in the evolution of (pseudo-) anisogamy per se; it is the conflict arising in an anisogamous population during the evolution of size-disassortative fusion, relating to how much investment proto-sperm and proto-ova receive from the gametes they fuse with.

1.2.1.2 *Evolution of disassortative fusion and anisogamy from ancestral mating types*

However, did disassortative fusion arise as a result of anisogamy (Parker, 1978), or did mating types evolve before the evolution of anisogamy, and act as the initial asymmetry under which anisogamy arises (Charlesworth, 1978)? It seems very likely that mating types arose before gamete dimorphism, since they appear to occur in all forms, isogamous or anisogamous (Wiese *et al.*, 1979; Wiese, 1981; Maynard Smith, 1982; Czárán and Hoekstra, 2004). The evolution of mating types involves reduction in the number of suitable gametes available for fusion (Iwasa and Sasaki, 1987), so this cost must be more than offset by the fitness advantage to a mutant with some form of selective fusion. This cost to the mutant is initially small in isogamous populations (because it can fuse with any other gamete), but the cost increases as the mutation spreads (Iwasa and Sasaki, 1987; Hurst, 1996). In isogamous populations, several mating types may thus spread, and indeed are often found (Hurst, 1996). However, in anisogamous populations, a third gamete size mating type is unable to spread or persist, and a mating type that fuses independently of gamete size is likely to face problems for various reasons (e.g. Parker, 1978; Hoekstra, 1987). A review of the evolution of mating types is given by Lessells *et al.* (2009).

Many approaches (e.g. Charlesworth, 1978; Maynard Smith, 1978; Bulmer, 1994; Bulmer and Parker, 2002) assume that mating types act as the initial asymmetry for the evolution of anisogamy. Using a haploid model (the normal mode of genetic control of gamete size in the Volvocales, Wiese, 1976), Charlesworth (1978) investigated population genetics models with two alleles, A_1 and A_2, at the gamete size locus and two mating-type alleles, M_1 and M_2. He showed that if linkage is sufficiently tight, linkage disequilibrium tends to build up, so that the gamete-size alleles become associated with different mating types, leading to a situation in which there is disassortative fusion with respect to gamete size. The pre-existence of mating types did not affect the conditions for the evolution of anisogamy. Further, selection favors closer linkage between the two loci, even when linkage is initially so loose that the mating-type alleles are equally frequent in microgametes and macrogametes (see also Hoekstra, 1984). Charlesworth plausibly argues that genes coding for size-assortative fusion (as in Parker's 1978 model) are a less likely origin of disassortative fusion than an origin via mating-type loci, which are frequently found in isogamous green algae (Lewin, 1976), although there is the initial problem as to how genes on separate

chromosomes could become linked on the same chromosome, if this were not the case originally. Subsequent models of anisogamy have tended to assume close linkage between the mating-type locus and the locus controlling gamete size, or a tight association between gamete size and mating type if the model is phenotypic rather than genetic.

While it seems likely that mating types preceded the evolution of anisogamy and could become closely linked with a gamete-size locus, the problem of explaining conflict concerning selective fusions still remains. Maynard Smith (1982) makes the point that were linkages between the mating-type locus and the gamete-size locus to be selected against, e.g. in the case of sexual conflict outlined by Parker (1978), in which ova may do better to fuse with other ova than with sperm, the fact that mating types may have arisen before anisogamy does not mean that this initial mating-type behavior must be maintained, ensuring disassortative fusion. Any mutant breaking the + and – mating-type rule could spread if favored by selection; an example could be an ovum producer with a mutation that allowed ova to fuse with other ova, despite being of the same mating type. Thus consideration of the evolution of selective fusion in hypothetical conditions of pseudo-anisogamy (random fusions for gamete size with or without mating types) seems important, even if the ancestral population involved mating types. Also, as Charlesworth (1978) points out, in an ancestral species where gamete size is determined non-genetically (see e.g. Wiese, 1981), size disassortative fusion obviously could not evolve by associations between mating-type alleles and gamete-size alleles, but could arise by a mechanism similar to that proposed by Parker (1978).

1.2.2 What determines whether the ESS is isogamy or anisogamy?

An important study by Matsuda and Abrams (1999) discussed the conditions favoring the evolution of the size of isogametes in a population with two mating types, and why isogamy should be relatively rare. Their model followed that of PBS but also included a "mating success" function defining the success of a gamete at fusing with other gametes. They showed that if size is not closely linked to mating type, isogamy can be stable under a wide range of conditions. However, when size is linked to mating type, isogamy is stable only if there are significant direct effects of size on gamete survival and fusion success; even then, isogamy may only be locally stable. They concluded that isogametes larger than the minimum possible gamete size are likely to be

explained by direct effects of size on gamete survival before fusion, rather than by particular forms of the zygote fitness function.

In their computer simulations, PBS used a very simple relation between zygote fitness, f, and zygote size, S, i.e. $f(S) = aS_{ij}^{x}$, though they discussed more general forms.

Randerson and Hurst (2001a) criticized PBS, claiming: (i) that disruptive selection does not lead to anisogamy unless the slope of $f(S)$ is accelerating at the origin (i.e. $x > 1$) and (ii) that this is an unusual assumption for which there is no empirical evidence. In fact, both these claims are incorrect (Bulmer *et al.*, 2002). They incorrectly developed the model of Levitan (2000; see also Vance, 1973) used empirically for zygote survival in echinoids, leading to the erroneous conclusion that $f(S)$ under this model is zero below some finite size, above which it is decelerating. They then claimed that anisogamy could not evolve in this case by disruptive selection following PBS. Analyzed correctly, Levitan's (2000) model actually generates a form for $f(S)$ that is accelerating at the origin. However, Randerson and Hurst's claim is incorrect even if $f(S)$ is zero below some finite size, above which it is decelerating, since this form is very similar to a sigmoidal function, and both can give rise to anisogamy (Bulmer *et al.*, 2002).

In response to Bulmer *et al.* (2002), Randerson and Hurst (2002) abandoned their earlier conclusion (Randerson and Hurst, 2001a) that disruptive selection never gives rise to anisogamy under plausible assumptions about the form of $f(S)$, and instead claimed the opposite: that, since a biologically plausible model must assume some finite size below which zygote fitness is zero, all logically consistent conditions appear to lead to anisogamy; PBS could not therefore account for the stable maintenance of isogamy. The flaw in Randerson and Hurst's (2002) argument is that it ignores the fact that if there is a size limit below which zygote fitness is zero, there must also be a size limit below which gamete fitness is zero (Bulmer and Parker, 2002). They nevertheless stimulated an important development in PBS theory, relating to the evolution of isogamy versus anisogamy.

Bulmer and Parker (2002) responded to Randerson and Hurst (2001b) by re-examining the PBS theory, starting from an isogamous population with two mating types (+ with gamete size m_1, and - with size m_2) obeying the PBS size–number trade-off (each individual produces a number of gametes, $n = M/m$). They used an evolutionary game-theory approach to determine the existence and continuous stability of isogamous and anisogamous strategies for the two mating types. In addition to the PBS relationship, $f(S)$, between the fitness of a

zygote and its size, $S = m_1 + m_2$, they added the relationship, $g(m)$, between the reproductive fitness of a gamete and its size, m, which is a simpler version of Matsuda and Abrams (1999) "mating success" function. The fitness of + individuals is therefore

$$w_1(m_1, m_2) = \frac{Mg_1(m_1)}{m_1} f(m_1 + m_2),$$

and the reproductive fitness $w_2(m_1, m_2)$ is analogous for - individuals.

Bulmer and Parker (2002) analyzed various forms for $g(m)$ and $f(S)$, including a sigmoidal form based on Vance (1973) and Levitan (2000), and an exponentially diminishing-returns function which is zero below some finite size, above which it is decelerating continuously. The general conclusions for the evolution of isogamy and anisogamy were rather similar for these forms, but the details differ.

For the sigmoidal form (based on Vance, 1973), the gamete and zygote survival (or other aspects of success) functions are

$$g(m) = \exp\left(-\frac{\alpha}{m}\right), \tag{1.2a}$$

$$f(S) = \exp\left(-\frac{\beta}{S}\right), \tag{1.2b}$$

where α, β, are positive parameters. This has an isogamous ESS at

$$m^* = \alpha + \beta/4, \tag{1.2c}$$

but to test whether this is continuously stable (i.e. it converges back to the ESS after a small perturbation; see Eshel, 1983) requires calculation of the best response (m_1) of an individual of the + mating type to the strategy m_2 played by the - mating type. As m_2 varies, so will the best response, m_1, and we can call the function describing the best response $R(m_2)$. Since the best response to a high m_2 is a low m_1, and vice versa, $R(m_2)$ has a negative slope. If this slope, $R'(m)$, is between -1 and 0, there is continuous stability; but if $R'(m)$ is less than -1, the ESS is continuously unstable. This can be demonstrated in Figure 1.3a–c, which gives results for the Vance functions (1.2a) and (1.2b), by using the "cobweb" technique (Sandefur, 1990). This technique can be explained as follows. Taking a given m value on the broken line in each of Figure 1.3a–c, the intersect on the curve (above or below it) gives the best reply, $R(m)$. This m value can be extrapolated to its equivalent new m value by finding its intersect, horizontally left or right, on the broken line. For this new m, we can obtain a new $R(m)$, and

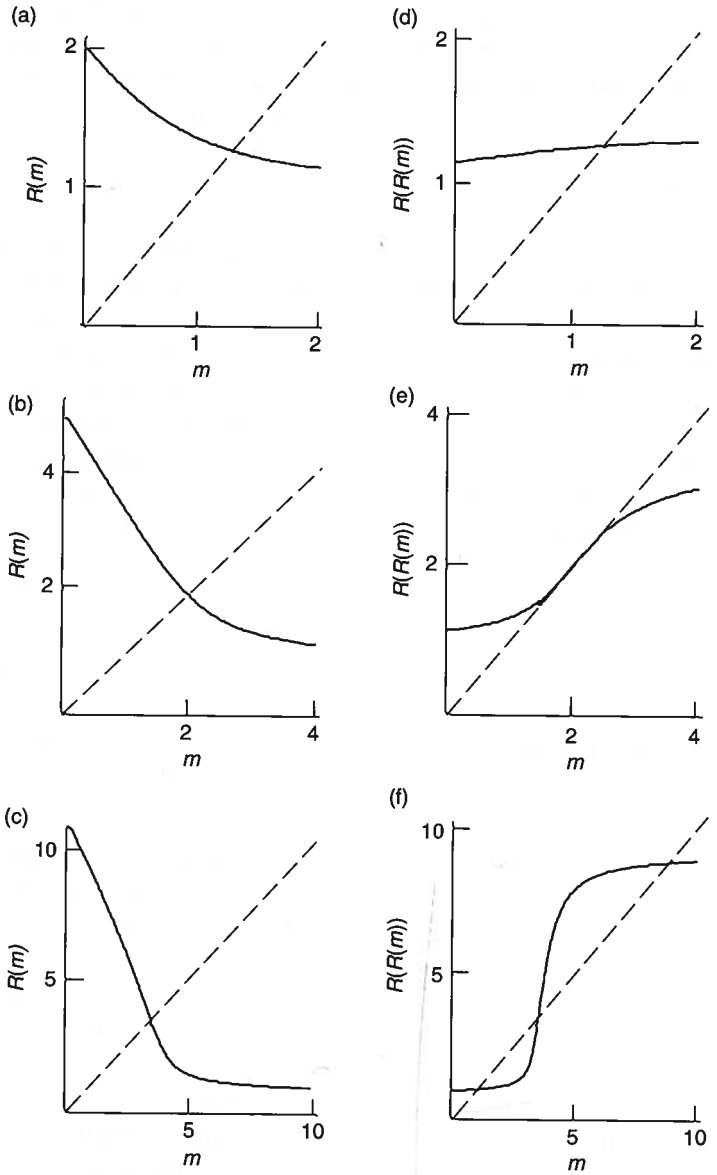

Figure 1.3 Isogamy and anisogamy in the Bulmer and Parker (2002) version of PBS. (a–c) The best response function, $R(m)$, and (d–e) the best response to the best response, $R(R(m))$, using functions (2a) with $\alpha = 1$, and (2b). In (a) and (d), $\beta = 1$. (a) There is a continuously stable isogamous ESS at (1.25, 1.25) because the slope $R'(m) > -1$ at this point; (d) shows that there is no anisogamous ESS. In (b) and (e), $\beta = 4$. (b) There is an

so on to produce a "cobweb." If $R'(m) > -1$ at the ESS (under-compensation; Figure 1.3a), the cobweb converges towards the intersection of the curve and the broken line, which is therefore a stable equilibrium. If $R'(m) > -1$ (over-compensation; Figure 1.3c), the cobweb diverges away from the intersection, which is an unstable equilibrium.

At the isogamous ESS in equation (1.2c), the slope is

$$R'(m) = -\beta/4\alpha. \tag{1.2d}$$

This is greater than -1 when $\beta < 4\alpha$, as in Figure 1.3a. However, when $\beta > 4\alpha$, the slope is less than -1 (Figure 1.3c), and the isogamous ESS in equation (1.2c) is not continuously stable. This leads to an anisogamous ESS, which can be found by plotting the "iterated best response function" or "best response to the best response," $R(R(m))$, i.e. the function describing the best responses of an individual of the + mating type to the best responses of the - mating type (Figure 1.3d–f). The anisogamous ESS is a pair of strategies, m_1^* for + and m_2^* for - which can be found by the points, other than the isogamous ESS, at which $R(R(m))$ intersects the 45° line through the origin (Figure 1.3d–f). There is no anisogamous ESS when the isogamous ESS is continuously stable (Figure 1.3d), but an anisogamous ESS arises when the isogamous ESS is continuously unstable (Figure 1.3f). Figure 1.3b (for $R(m)$) and Figure 1.3e (for $R(R(m))$) relate to the case where $\beta = 4$, so that $R'(m) = -1$, i.e. intermediate between isogamy and anisogamy.

In unicellular organisms, Bulmer and Parker (2002) argued that the two survival functions, (1.2a) and (1.2b), are rather similar, so that $\alpha \approx \beta$, resulting in isogamy. As multicellularity began to evolve, α probably remained roughly constant, but β would have increased with the need to provision the embryo. At the stage where β had increased more than fourfold, anisogamy should have replaced the ancestral isogamous state. Thus we would expect a sudden transition from isogamy during the evolution of increasing vegetative complexity (usually multicellularity) as the gamete survival function $g(m)$ and the zygote survival function $f(S)$ separated along the provisioning axis (Figure 1.4).

Caption for Figure 1.3 (cont.)
isogamous ESS at (2, 2) which verges on continuous instability since at this point $R'(m) = -1$; (e) an anisogamous ESS is incipient. In (c) and (f), $\beta = 10$. There is a continuously unstable isogamous ESS at (3.5, 3.5) where the slope $R'(m) < -1$; (f) there is an anisogamous ESS at (1.13, 8.87). With permission from Bulmer and Parker (2002).

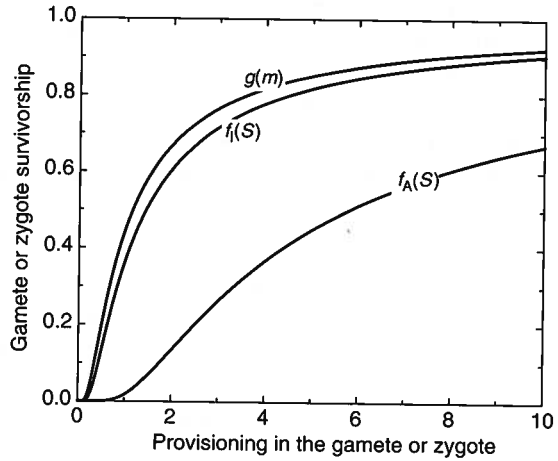

Figure 1.4 Isogamy and anisogamy when there are separate gamete survival and zygote survival functions, $g(m)$ and $f(S)$, in the Bulmer and Parker (2002) model following the Vance function (Equations (1.2a) and (1.2b)). In the ancestral state, the curves for $g(m)$ and $f_I(S)$ are close together and isogamy is stable (shown here with $\alpha = 0.8$ and $\beta = 1.0$). As multicellularity develops, the gamete function $g(m)$ probably remains relatively unchanged in its position on the provisioning axis, but the need to provision the zygote increases, pushing $f(S)$to the right, leading to anisogamy if $\beta > 4\alpha$. In the case shown, $f_A(S)$ has $\beta = 5.0$, which would generate anisogamy.

Bulmer and Parker (2002) also investigated cases (including a modified version of the Vance function) where there is a critical minimum gamete size, δ, below which the gamete dies before fertilization (e.g. Maynard Smith, 1978; Bulmer, 1994), and concave functions where $g(m)$ and $f(S)$ are initially zero, e.g. up to δ, and then increase from zero with decreasing slope (δ could be seen as the component due to the chromosomes, and $m - \delta$ the energy reserves that increase survival). These all behaved qualitatively rather similarly with regard to the stability of isogamy and anisogamy, and in each case it was the relationship between the gamete and zygote survival functions that determined whether or not anisogamy evolves.

Thus Bulmer and Parker (2002) have shown that in the ancestral unicellular state, isogamy is likely to be stable because the gamete and zygote survival functions ($g(m)$ and $f(S)$) are likely to have been similar; this leads to isogamy whether these functions are sigmoidal or concave, though in the latter case allowance must be made for a minimal

gamete size. The transition to multicellularity probably left $g(m)$ relatively unchanged while $f(S)$ moved to the right along the provisioning axis, leading to the evolution of anisogamy. The origin of anisogamy is tightly coupled with the evolution of the gamete and zygote survival functions, $g_1(m_1)$ for sperm, $g_2(m_2)$ for ova, and $f(S)$ for zygotes, during their separation during this transition. The theoretical biology of these functions becomes the key to understanding the evolution of the sexes, and would greatly merit further study. The specialization of micro- and macrogamete sizes after the development of anisogamy are discussed in Section 1.6.

Bonsall (2006) has extended Bulmer and Parker's approach (2002) by developing Equations (1.2a) and (1.2b) to include variability in survival through differential mortality or metabolic damage of gametes or zygotes as a function of their size, and by including a distribution in the number of gametes (or zygotes), $n(T)$, produced by a parent due to mortality over lifespan T. In addition to the classic PBS mechanism, Bonsall concludes that anisogamy can evolve through the effects of variability in mortality or distributed metabolic costs acting on isogamous gametes, by inducing disruptive selection on isogamous gametes favoring individuals that produce smaller gametes to compensate for the costs associated with metabolic damage. Thus in Bonsall's rather complex models, different mechanisms associated with survival disrupt the isogamous state and also affect the evolution of anisogamy; he concludes that the persistence of both the isogamous and anisogamous ESS points is not always assured and calls for a more pluralistic approach.

1.2.3 The ancestral isogamous state - which came first, smaller or larger gametes?

In the early studies of PBS, Charlesworth (1978) and Bell (1978) suggested that anisogamy can evolve only if there is sufficient size variation in the ancestral population to generate it (see Figure 1.1), or if gamete size mutations have sufficiently large effect (but see Maire *et al.*, 2002). PBS argued that whatever gamete size the ancestral isogamous population showed, selection may generally tend to push gamete size towards a zone of the zygote fitness:size relationship, $f(S)$,where anisogamy will occur, depending on the $f(S)$ relation for the ancestral state. PBS envisaged two scenarios: in the first, there is a general reduction in gamete size starting from isogamous macrogametes that then allows invasion by microgametes (PBS, Figure 9), and in the second, there is invasion by

macrogametes into an ancestral population of isogamous microgametes, followed by a subsequent reduction in the size of the microgametes (PBS, Figure 10). The latter scenario is probably more likely and was proposed by Maynard Smith (1978), who argued that maleness was ancestral, with macrogametes invading an isogamous microgamete (male) population.

Bulmer and Parker's (2002) analysis also suggests that the ancestral stage would have been isogamous unicells (microgametes). As the size of the adult increased (e.g. through the evolution of muticellularity), Bulmer and Parker predicted that $f(S)$ would increase away from its early position near $g(m)$, and during this stage, the isogamous microgamete size would initially have increased. At some threshold in this shift towards larger isogametes, isogamy becomes unstable, and is suddenly replaced by anisogamy through invasion of macrogametes (see Figures 1.3 and 1.4). As increasing adult complexity causes $f(S)$ to move further away from $g(m)$, macrogamete size increases and microgamete size decreases (compare Figures 1.3d, 1.3e, 1.3f) in a manner very similar to that depicted in Figure 10 of Parker *et al.* (1972).

1.2.4 Requirements for PBS theory

The original PBS simulation model made specific assumptions, listed as (1) to (6) on p. 19. However, it is clear that the basic PBS theory, that isogamy and anisogamy can be explained by gamete competition under the two general selective forces of gamete productivity and zygote provisioning, is correct even when most of their detailed assumptions (see p. 19) are relaxed. The initial studies with random gamete fusion (PBS, Charlesworth, 1978) suggested that sufficient genetic variation must exist in the size of gametes produced in the ancestral isogamous population (i.e. requiring mutations of large effect). However, Maire *et al.* (2002) have shown using individual based dynamic simulations that PBS requires neither mutations of large effect nor the pre-existence of mating types; gamete dimorphism can evolve through arbitrarily small mutations in a population without mating types through a mechanism based on evolutionary branching in allele space. It is unlikely that an exact trade-off between gamete size and number must apply, though there must be a sufficiently significant negative relation between size and number. The exact nature of the genetic determination of size and number need not affect the ESS, which can be derived by haploid or diploid models or by game theoretic, phenotypic models. Though the PBS assumption was that

ancestrally, isogametes with a wide range of size variation fused at random to generate anisogamy without mating types (pseudo-anisogamy), the PBS model is not affected if the ancestral state is initially from mating types (e.g. Charlesworth, 1978). Nor is it greatly affected by the fact that encounter rates will be functions of gamete size, though this does increase the range of zygote fitness functions that generate anisogamy (Cox and Sethian, 1984). Simultaneous shedding of gametes within local populations is also not necessary, provided that gametes from different parents compete sufficiently to generate some gamete competition. The theory remains robust even when gamete competition is reduced because of such biological features as internal fertilization, and gamete competition through sperm or pollen competition can help to explain why anisogamy is maintained so ubiquitously in present-day multicells (Parker, 1982). Given that there must logically be some gamete size–number trade-off in an ancestral species in which reproductive expenditure relates mainly to gamete production, the important assumption in PBS relating to the isogamy/anisogamy dichotomy is that zygote fitness increases sigmoidally with zygote mass, or, equivalently increases with diminishing returns from a positive intercept (more detailed models of gamete collisions suggest that an even broader range of functions may allow the switch between isogamy and anisogamy, e.g. Cox and Sethian, 1984). This assumption, the basis of the Smith–Fretwell (1974) model, has considerable empirical support (see e.g. Lessells *et al.*, 2009).

PBS theory therefore appears to be very robust, mainly reliant only on the general assumptions about productivity versus provisioning, which can generate disruptive selection through gamete competition. It has been shown to be challenged only when certain special conditions apply, such as when variability in mortality or distributed metabolic costs acting on gametes are so significant that they can interfere with the basic PBS disruptive selection mechanism involving gamete competition across the productivity–provisioning axis (Bonsall, 2006), or when the success of large gametes increases sufficiently as a result of sperm limitation to offset this mechanism (Dusenbery, 2000; 2002; but see Lessells *et al.*, 2009).

1.2.5 Evidence for PBS theory

The claim that a switch from isogamy to anisogamy is likely in the transition from the ancestral unicellular state to the increased complexity of multicellular forms (PBS) as the gamete and zygote survival

functions (Bulmer and Parker, 2002) become sufficiently separated (see Figure 1.4) has some support from comparative studies, particularly of volvocine algae. Knowlton (1974) was the first to note an association between increased complexity and a switch from isogamy to anisogamy in the Volvocales, and this has been supported using modern comparative methods (Randerson and Hurst, 2001a; 2001b). In other chlorophyte algae the same correlation occurs, but is less clear (Bell, 1978). The most extensive analysis (Bell, 1982) involved many algal and protozoan groups, and demonstrated a clear correlation between increasing vegetative organization and increasing gamete dimorphism.

Randerson and Hurst's (2001b) study of the Volvocales controlled for phylogenetic effects, and showed that: (i) the anisogamy ratio (A = macro-/microgamete volume), and (ii) the macrogamete size (see also Bell, 1985) increased with adult size, though these results were sensitive to the mode of analysis and the phylogeny used. They proposed an alternative explanation of these results to PBS, based on the present mode of reproduction in volvocine algae (which may not relate to their ancestral mode of reproduction under which anisogamy evolved). In many colonial forms, male and female colonies clump together and male colonies release sperm in sperm packets; each packet swims as a unit until it reaches a female colony and the individual sperm are released. Randerson and Hurst (2001b) explain the positive correlation between anisogamy ratio and vegetative complexity in terms of the need to produce more (and hence smaller) sperm to fertilize the higher number of eggs in larger female colonies. It remains to be tested whether this is a better explanation of the association between anisogamy ratio and increased multicellularity in Volvocales; although this mode of reproduction does not involve release of separate gametes and random fertilization in the external medium (as assumed by PBS), there may well be sufficient gamete competition under this mode of reproduction to allow the PBS mechanism to operate.

In marine green algae, gametes are typically released individually in one event into the surrounding medium (Togashi *et al.*, 1998; 1999), exactly as the PBS model assumes. However, support for the PBS prediction is controversial, since isogamous species are multicellular, though often with low "vegetative complexity" (*sensu* Bell, 1978; 1982). Generally, isogamous species and slightly anisogamous species produce their gametes through synchronized cell divisions during gametogenesis, so their gametes cannot be larger than their cell size. In contrast, strongly anisogamous species often produce giant female gametes. This is possible because they are typically unicellular and

multinucleate. In certain marine green algal groups, while an increased anisogamy ratio may loosely correlate with increased morphological complexity, it does not correlate with multicellularity, and some forms are large and vegetatively complex, but retain isogamy (Bell, 1978). As with volvocine algae, brown algae generally tend to show the expected PBS correlation between vegetative size and complexity and increased anisogamy and oogamy (Bell, 1978).

Thus although increased morphological complexity is loosely associated with increased gamete dimorphism in algae (Knowlton, 1974; Bell, 1978; 1982; 1985; Randerson and Hurst, 2001b; Iyer and Roughgarden, 2008), it does not accurately predict whether a given species will show gamete monomorphism or dimorphism (Bell, 1978; Madsen and Waller, 1983). There may be a number of reasons for this, relating to species ecology and other special conditions. For example, although examined in the context of the evolution of anisogamy in relation to increasing collision probability, Togashi and Cox (2004) have shown that phototaxic responses will be important, and fertilization in shallow water versus deep water can exert a major effect in explaining the variation between isogamy and anisogamy in marine algae (Togashi *et al.*, 2007).

While plants and animals appear generally to match PBS predictions, fungi are less accommodating. Fungal modes of reproduction are summarized in various texts (e.g. Alexopoulos, 1962), and though fungi do not contradict the predictions, concurrence with PBS is evident only in certain groups (Bulmer and Parker, 2002). Forms showing the fusion of two naked gametes (planogametes) are closest to the assumptions of the PBS model. The simplest Chytridiomycetes are unicellular, aquatic and the entire thallus is used to produce gametes, and often have isogamous planogametes. Some *Allomyces* species have a simple thallus with specialized reproductive organs, and reproduction involves fusion of motile anisogametes (Cox and Sethian, 1985). The most complex Chytridiomycetes such as *Monoblepharis* have developed more complex thalli, and have non-motile female gametes. This trend appears to follow rather closely with the PBS prediction that anisogamy should correlate with vegetative complexity.

In higher fungi, motile free-swimming gametes are absent; instead, there are various forms of transfer of gametic nuclei, some which could possibly be analogous to, or even an extension of, anisogamy under the PBS theory. For example, in spermatization, numerous minute, uninucleate male spermatia are carried by insects, wind, water, etc. to female gametangia or to unspecialized somatic hyphae,

to which they attach and transfer their contents. Other forms are not analogous to PBS, probably because the mode of reproduction has transformed from an ancestral state involving gametes to one in which there is direct transfer of nuclei between hyphae. For example, the haploid spores (basidiospores) resulting from meiosis of Basidiomycetes germinate on moist substrates to form haploid, monokaryotic hyphae, which usually exist as multiple mating types. When two such hyphae of different mating types meet, they fuse, pass nuclei into each, and typically both mycelia become "dikaryotized" (the donor nuclei divide and migrate from cell to cell forming a dikaryon in the recipient mycelium). The present selective forces are clearly quite different from those envisaged in the PBS theory, but may have arisen from a system similar to that in Chytridiomycetes.

1.3 THE LOSS OF MOTILITY BY FEMALE GAMETES (OOGAMY)

In isogamous populations, the isogametes are typically motile to ensure encounter and fusion. PBS (see also Parker, 1979) argued that once anisogamy has arisen, the evolution of disassortative fusions of ova with sperm may be related to the advantages of loss of motility by ova. Due to the vast numerical predominance of sperm arising through the size–number trade-off at production, and through selection quickly favoring avoidance of sperm–sperm fusions, ova may fuse very quickly with sperm. Thus although loss of motility in ova may ultimately interfere with the ability of ova to fuse with other ova, a mutant that loses ovum motility may suffer little reduction in the probability of fusion of its ova, allowing energy spent on motility to be channeled into increased productivity. Thus as the anisogamy ratio increases and ovum–ovum encounters become rare, selection against disassortative fusion by ova decreases, or actively favors it (Parker, 1978), so that the best strategy for ovum producers may be total commitment to fusions with sperm, with a resulting reallocation of motility expenditure into productivity.

An analysis of gamete motility dimorphism is given by Hoekstra and his co-workers. Hoekstra *et al.* (1984) analyzed the relation between swimming speed of gametes in an isogamous population starting with equal motilities (isomotility) and gametic abilities to locate, and to be located by, other gametes. Gametes produce attractant pheromones and the volume searched by a gamete is proportional to its speed of movement. Motility dimorphism may arise in this isogamous

population via disruptive selection on swimming speed, and no more than two different swimming speeds can coexist in a stable polymorphism. An initial difference in swimming speed of at least twofold leads to a stable motility dimorphism (anisomotility), and loss of motility of one of the gamete types is likely. Hoekstra (1984) extended this model by adding the effect of anisogamy on gamete motility within the PBS framework, but assuming two initial mating types. He concluded that conditions for stable anisogamy were broadened only when the gamete size and mating-type loci are closely linked. Anisogamy has no effect on the evolution of anisomotility, and anisomotility no effect on the evolution of anisogamy. Hoekstra (1984) also concluded that anisogamy can also evolve solely as a consequence of its effect on gamete motility (i.e. omitting the basic productivity-provisioning assumptions of PBS), if the size difference between the gamete types is sufficiently large. His assumption that gamete speed is inversely proportional to the gamete cross-sectional area has, however, been disputed (Dusenbery, 2000; 2002; Randerson and Hurst, 2001a).

Cox and Sethian (1985) considered the evolution of motility dimorphism within the framework of the evolution of gamete dimorphism; further models have been constructed by Dusenbery (2000, 2002); see Lessells *et al.* (2009) for a review.

Thus although gamete motility dimorphism may have arisen even before anisogamy evolved, and/or had an important influence on the evolution of anisogamy, it seems likely that macrogametes would anyhow lose their motility as the anisogamy ratio increased, generating oogamy, since the probability of fusion due to the vast numbers of tiny sperm rendered the cost of maintaining motility of ova unnecessary (PBS, Parker, 1978). For sperm producers, however, the maintenance or increase of motility would be favored through sperm competition by virtue of increased encounter and ovum penetration possibilities. Many of the adaptations of sperm (e.g. involving vigorous motility, hyaluronidase secretion by the acrosome, and fast migration of the sperm pronucleus towards the ovum pronucleus) can be interpreted in terms of competition between the sperm of several parental variants (PBS).

1.4 OTHER THEORIES FOR THE ORIGIN OF ANISOGAMY AND THEIR RELATION TO GAMETE COMPETITION

An excellent review of theories for the evolution of anisogamy, and their roles in the differentiation of gamete dimorphism is given by

Lessells *et al.* (2009); for an overview of the inter-relationships between the various theories see their Figure 2.1. Readers are referred to this source, and to other chapters in the present volume for alternative theories for the origin of anisogamy; I discuss them here only very briefly in relation to their impact on the evolution of anisogamy by gamete competition.

1.4.1 Classical views

The earliest analyses of the evolution of anisogamy (Kalmus, 1932; Kalmus and Smith, 1960; Scudo, 1967) were based on maximization of gamete fusions in a population, which can be achieved by division of the total resource for reproduction in a population anisogamously. Though unstated, such a fitness maximization implies group or population selection, which may operate in certain circumstances; it is nevertheless clear that the selective pressure of gamete encounter rate plays a role in the evolution of anisogamy (e.g. Cox and Sethian, 1985; Dusenbery, 2000; 2006; Togashi *et al.*, 2007; Iyer and Roughgarden, 2008) and is partly implicit in PBS through the fact that productivity (microgamete production) causes microgametes to dominate the fusions of macrogametes (see also Parker, 1978). However, in the original PBS model all gametes were assumed to fuse (or equivalently, gamete survival up to fusion was independent of gamete size), while with the classical theory (and the sperm limitation theory below) there is strong selective pressure to ensure gamete fusion.

1.4.2 Sperm limitation

The "sperm limitation" theory began as a modification of PBS by Cox and Sethian (1984, 1985) and generates an individual selection basis for the classical view that anisogamy relates to increasing gamete encounter rates. In broadcast spawners, infertility resulting from failure to meet another gamete can sometimes be significant (Levitan, 1993; 1996a; 1998) and is likely to select for increased encounter rates, and can be seen both as increasing the "target size" of macrogametes and a driver of anisogamy (e.g. Cox and Sethian, 1985; Levitan, 1996b; 1998; 2000; 2006; Dusenbery, 2000; 2002; 2006; Togashi and Cox, 2004). Assumptions in these models vary, for example, Dusenbery's model (2006) does not include a zygote fitness function, $f(S)$, but instead assumes that a gamete's "fertile period" (longevity) increases with its size, and encounter rates may be increased by attractant pheromones,

phototaxis, and other methods. There is empirical evidence that larger-sized eggs can increase fertilization rates (Levitan, 1998; 2006). Togashi *et al.* (2007) introduced the concept of gametic investment per unit volume of the space in which gametes searched for fusion partners, an effect which may explain the prevalence of isogamous species in shallow water and anisogamous species in deep water in marine green algae. Computations show that positive phototaxis is favored, particularly in shallow water, and may increase gamete density to a level where sperm limitation might not be the dominant selective force in the evolution of isogamous or slightly anisogamous marine green algae (Togashi *et al.*, 2008). It remains unclear how ubiquitous sperm limitation is as a selective pressure; while sperm limitation may or may not have been a significant selective force ancestrally, fertilization rates in most external fertilizers are currently high (Yund, 2000).

It is important to stress that gamete competition in the form of sperm competition is still operative under sperm limitation, through the size-productivity trade-off (Lessells *et al.*, 2009). There are often cheaper ways to increase encounter rates than by increasing ovum size, and Lessells *et al.* (2009) conclude that sperm limitation alone may be insufficient to explain the evolution of anisogamy, though it may well contribute (see also Randerson and Hurst, 2001a).

1.4.3 Conflicts with cytoplasmic elements

This theory originated with Cosmides and Tooby (1981), who proposed that competition between cytotypes with different compositions of cytoplasmic elements would result in selection for increased gamete size, allowing the invasion of nuclear genes coding for microgametes because of the productivity benefits arising through the size–number trade-off. An early population genetic model of this process (Hoekstra, 1987) failed to generate anisogamy, and instead generated a single cytoplasmic allele for gamete size. However, there have been many subsequent modifications of the original intracellular conflict theory for anisogamy; these are reviewed by Lessells *et al.* (2009) who conclude that this theory is unlikely to be the sole explanation for anisogamy, though it may help to maintain gamete dimorphism once this has evolved. Randerson and Hurst's models (1999) suggest that intracellular conflict is less likely to lead to gamete dimorphism in multicellular than in unicellular organisms, contrary to the empirical evidence.

It must also be noted that the two basic PBS assumptions relating to gamete productivity and zygote provisioning cannot be ignored in any theory for the evolution of gamete size; inescapably, there must be a size–number trade-off at gamete production since an adult's reproductive budget must be constrained, and zygote size must affect zygote survival/fitness. Thus although the parasitic organelle theory may be a part of the explanation, it cannot exclude it, unless it can explain anisogamy in situations where PBS would predict isogamy (or vice versa).

1.5 STABILITY OF ANISOGAMY UNDER INTERNAL FERTILIZATION – WHY IS ANISOGAMY NOT LOST WHEN SPERM COMPETITION IS REDUCED?

Under PBS conditions, anisogamy arises by gamete competition, through disruptive selection against intermediate-sized gamete-producing genotypes, because of the advantages of provisioning on the one hand, and productivity on the other. Proto-ovum producers have gametes that survive well as zygotes. Proto-sperm producers are able to produce so many proto-sperm that they gain most fusions with the proto-ova from proto-ovum producers: gamete competition thus generates anisogamy in ancestral externally fertilizing organisms. Gamete competition – in the sense of competition for fusions – is still present, even if there is severe gamete limitation so that the fusion probability of gametes is low.

We also need to know how anisogamy is maintained despite the many evolutionary changes subsequent to the evolution of early multicellular organisms: for example, how is anisogamy maintained when gamete competition is dramatically reduced due to the evolution of internal fertilization?

1.5.1 Sperm competition and a direct sperm size–number trade-off

The PBS model for the evolution of anisogamy relies on external fertilization – fusions occur in a gametic pool, most plausible in the sea. With the onset of internal fertilization (coupled with the much earlier evolution of disassortative fusion) came a dramatic reduction in gamete competition – or more specifically, sperm competition. While PBS argued that gamete competition was responsible for the

evolutionary *origin* of anisogamy, gamete competition in the form of sperm competition (competition between the sperm of different males over the fertilization of a given set of ova; Parker, 1970) has also been claimed to be important for the *maintenance* of anisogamy (Parker, 1982).

Consider a sessile multicellular animal or plant in which groups of individuals spawn simultaneously, and all eggs get fertilized. Suppose that sperm have size δ, defined as the minimum size to carry the chromosomes and to survive until all ova are fertilized (essentially, investment δ includes no investment for zygote provisioning). If a mutant male arises that increases sperm investment to $m > \delta$, it raises the fitness f of any zygote it produces by $b(m)$, from F to $F + b(m)$, where F is the zygote fitness due only to ovum size. Suppose that the direct size–number trade-off applies – a male that increases provisioning in each sperm decreases sperm numbers, so non-mutant males produce (relatively) δ^{-1} sperm, and the mutant produces m^{-1} sperm. The fertilization success of the mutant obeys the "raffle principle" where fertilization success is proportional to proportionate representation in the pool of competing sperm (see Parker, 1998). Thus if there are N competing males, the proportion of eggs gained by the mutant male will be his contribution divided by the total sperm, i.e. $m^{-1}/[m^{-1} + (N-1)\delta^{-1}]$. For sperm to stay small, mutants that increase sperm size must not invade: mutant male fitness must decrease if m deviates above the minimum sperm size δ. Thus for δ to be locally stable, the gradient of male fitness at $m = \delta$ must be negative.

Remembering that $b(\delta) = 0$, Parker (1982) used this technique to show that δ is locally stable, if

$$b'(\delta) < F(N-1)/\delta N, \tag{1.3a}$$

where N = the number of males in the spawning group. If males do not contribute to the zygote, the optimal provisioning for females to invest per egg is given by the following interpretation of the Smith and Fretwell (1974) solution

$$b'(m^*_{\text{ovum}}) = F/m^*_{\text{ovum}}. \tag{1.3b}$$

Assuming that zygote provisioning via sperm has the same benefit as provisioning through ova (i.e. both affect zygote survival equally), then $b'(m^*_{\text{ovum}}) = b'(\delta)$ and we obtain a simple condition in terms of the anisogamy ratio A (= ovum size/sperm size):

$$A = m^*_{\text{ovum}}/\delta > N/(N-1). \tag{1.3c}$$

If this is satisfied, males should not increase their zygote provisioning, hence anisogamy is stable. Condition (1.3c) is very robust: the anisogamy ratio A must only exceed $N/(N-1)$. With external fertilization, there will generally be high sperm competition (high N) if spawning tends to be synchronous. Thus in large groups, sperm can be almost as big as ova before a mutant with extra provisioning will spread, and even when only two males compete ($N = 2$), sperm size remains at δ unless A is extremely weak (below 2).

Both selection for increased fertilization probability and sexual selection to outcompete other males by releasing sperm closer to eggs (Parker, 1970) may have driven the evolution of internal fertilization. With internal fertilization, sperm competition is likely to become much reduced, but probably in most groups never entirely absent (see reviews in Smith, 1984; Birkhead and Møller, 1998).

Imagine that sperm competition is rare, and when it occurs, it involves only two males. Such situations are common in internal fertilizers, but also apply to external fertilizers with rare sperm competition, e.g. species in which spawnings occasionally involve both a sneak male as well as the guarding male. Applying the same technique, if a given male faces sperm competition from one other male with low probability p, the condition to keep sperm minimal becomes

$$b'(\delta) < pF/[\delta(4-2p)] \tag{1.4a}$$

and from the Smith–Fretwell equation (1.3b),

$$A = m^*_{\text{ovum}}/\delta > (4-2p)/p, \tag{1.4b}$$

which, with $p = 1$, is the same as (1.3c) with $N = 2$ (if $p = 1$, two males always compete). The probability p that a male faces sperm competition at a given copulation can be related to the expected probability q that a given female in the population mates twice: $p = 2q/(1+q)$ (Parker *et al.*, 1997). Thus condition (1.4b) can be written as

$$q > 2/A. \tag{1.4c}$$

Thus anisogamy is stable provided that the probability that a female mates twice (or releases her eggs with two competing ejaculates) is more frequent than 2/anisogamy ratio.

Again, condition (1.4c) is a remarkably robust one. Anisogamy ratios of vertebrates commonly exceed 10^6; even with $A = 10^3$, anisogamy is stable provided two ejaculates compete in at least 0.2% of clutches.

Increased sperm provisioning is disadvantageous in these two models because, at high anisogamy ratios, a unit increase in investment in each sperm causes significant cost, but trivial benefits. In the "internal fertilization" model a mutant that has double the sperm size has half the sperm number, which reduces its gains under sperm competition from 0.5 to 0.333. Assuming that the original sperm size is due to chromosomes rather than provisioning, it increases its zygote's provisioning only from A to $A + 1$. At high anisogamy ratios, an extra unit of provisioning yields a trivial increase in zygote viability, but a large cost in terms of gamete competition.

This analysis suggests two conclusions. First, provided that a sufficiently high anisogamy ratio had already evolved, the reduction in sperm competition through internal fertilization would be generally insufficient to threaten the stability of anisogamy. Second, apart from the haploid chromosomes, typically no component of sperm mass should be for investment in the zygote (for similar conclusions for isogamy, see Matsuda and Abrams, 1999); all sperm characteristics should relate solely to ensuring fusion with ova. Section 1.6.3 outlines how sperm size may be optimized to maximize fertilization gains under sperm competition.

The external fertilization model (for N competing ejaculates) and the internal fertilization model (where females mate twice with probability q) have in the literature become known as the "intensity" and "risk" models in sperm competition games, i.e. games that seek the ESS sperm allocations under a variety of sperm competition scenarios (see Parker, 1998; Parker and Pizzari, 2010) where competition for fertilization follows the raffle principle.

Sperm competition does not always comply with the raffle principle. In many internal fertilizers such as insects with fixed-volume sperm stores, there is sperm displacement, so that as new sperm are input to the stores, previously stored sperm are displaced. I investigated the case where proportion z of the sperm store is displaced volumetrically by the ejaculate of the last male to mate (Parker, 1978). Again sperm competition is rare and a given male faces sperm competition from another male with probability p. The assumption was that when the mutant male (with sperm size $m > \delta$) mates last, he will have zm^{-1} sperm in competition with $(1-z)\delta^{-1}$ non-mutant sperm, and when he mates first, he has $(1-z)m^{-1}$ sperm remaining to compete with the $z\delta^{-1}$ sperm from the first male. This implicitly assumes either that there is little or no seminal fluid (all displacement is by sperm mass), or that the seminal fluid volume is proportional to the sperm

mass. The condition for maintaining sperm at the minimum size, δ, now becomes

$$A = m^*_{\text{ovum}}/\delta > (2-p)/pz(1-z) \quad (1.5a)$$

and substituting $p = 2q/(1+q)$, condition (1.4a) becomes

$$q > 1/Az(1-z). \quad (1.5b)$$

Note that if $z = 0.5$ (each male's ejaculate is equally represented volumetrically in the sperm stores), condition (1.4b) becomes $q > 4/A$: at intermediate levels of sperm displacement, anisogamy is again robust, but less so than the raffle (cf. condition (1.4c)). However, it is much less robust than the raffle when displacement is either very high ($z \rightarrow 1.0$) or very low ($z \rightarrow 0$).

In the most comprehensive review of insect sperm competition to date, Simmons (2001, Table 2.3) lists 133 species of non-social insects for which there is data on the mean paternity of the last male to mate (P_2), which is related to z. The majority of species have $0.5 < P_2 < 0.95$, i.e. the last male predominates at fertilization. The lowest P_2 value is that of 0.02 for *Anopheles gambiae* (Bryan, 1968), and there are 12 species in the range $0.95 \leq P_2 \leq 1$, with five listed as 1.0 (with standard deviation recorded as 0.00, or not given). Whether this implies that anisogamy could be threatened remains uncertain, since records of 1.00 may represent low sample sizes; there appears to be no sign of large sperm size and small sperm number in these species. P_2 can be very close to 1.0 and yet be sufficient to maintain anisogamy. Parker (1982) cites the example of the ferocious water bug *Abedus herberti* with $P_2 = 0.997$ (Smith, 1979). Even at this exceptionally high P_2 level, the minimal sperm strategy, δ, appears relatively safe since $A >> z(1-z)$.

So variations in the mechanism of sperm competition from a fair raffle may not greatly affect the stability of anisogamy through sperm competition, under a sperm size–number trade-off.

1.5.2 No sperm competition: sperm size trade off against mate acquisition or paternal care

In a hypothetical sessile ancestral organism there is every reason that the gamete size–number trade-off should apply; there may be little else on which to expend the resources available for reproduction. The direct size–number trade-off need not apply, however, when other reproductive strategies arise, for example, with the evolution of mobility and

mate searching. I examined the case where increasing sperm size reduces a male's number of matings (Parker, 1984). Suppose that in a population there is no sperm competition, and k is the optimal proportion of the reproductive budget spent on minimal sperm (size δ) by males in relation to fertility (sperm numbers increase fertility, following the next section). Then $(1-k)$ is spent on gaining matings. The number of matings achieved by a mutant that deviates by producing sperm of size $m > \delta$ is therefore reduced, because only $(1 - km/\delta)$ of the reproductive budget can be spent on gaining matings. With a sex ratio of unity, Parker (1984) showed that the minimal sperm strategy is stable if

$$k > A^{-1}, \quad (1.6)$$

which is a very easy condition to satisfy in vertebrates. A rough measure of k can be taken as the gonadosomatic index (proportion of body mass devoted to testes, see Parker and Ball, 2005). The seahorses (Sygnathidae) have probably close to zero sperm competition risk (Stockley *et al.*, 1997; Van Look *et al.*, 2007), and produce remarkably few tiny sperm with high fertilization efficiency (e.g. *Hippocampus kuda*, Van Look *et al.*, 2007). They have a very small gonadosomatic index for fishes (0.15 for *Sygnathus typhle* is the smallest value in Table 1 of Stockley *et al.*, 1997); their anisogamy ratio is such that A^{-1} is many orders of magnitude less than 0.15.

A similar argument may be made for a trade-off of sperm size against paternal care, when this occurs. For example, under near zero sperm competition risk, seahorses have retained minimal sperm, but have responded by producing the tiniest of ejaculates, with a ratio of numbers of sperm ejaculated to eggs fertilized probably less than 2.5, which is comparable to insects and many orders of magnitude smaller than that of most fishes (Van Look *et al.*, 2007). They also show considerable paternal care: increased paternal care appears to have been more favorable than investment in the zygote via increased sperm size.

1.5.3 No sperm competition: sperm numbers increase fertility (sperm limitation)

Sperm limitation can also assist in the maintenance of anisogamy (Parker, 1982). The probability of fertilization, P, is typically an increasing function of sperm numbers surrounding the ovum, or the set of ova. The classical theory for large numbers of tiny sperm is that this is necessary to increase fertility.

First, consider the case where the sperm size–number trade-off applies, so that the maximum sperm number in an ejaculate is proportional to δ^{-1}, and the fitness of a mutant with sperm size $m > \delta$ is $P(m)[F + b(m)]$, where F is the contribution to zygote viability of the ovum, and $b(m)$ the contribution from the mutant male. Remembering that we are now considering a state without sperm competition (one ejaculate fertilizes one set of eggs), applying the techniques of former sections we can see that δ is stable against m if the anisogamy ratio

$$A > P(\delta^{-1})/[P'(\delta^{-1}) \cdot \delta^{-1}] \tag{1.7}$$

(Parker, 1982). Thus, if fertility is high ($P \rightarrow 1.0$), the anisogamy ratio must exceed the reciprocal of the product of sperm number and the gradient of fertilization probability. Applying data on cattle to Equation (1.7), I was unable to conclude whether or not the classical theory (here, that minimal-sized sperm are stable to maximize sperm numbers for fertilization) is plausible (Parker, 1982). It certainly seems possible that if sperm size trades off against sperm number, selection to increase fertilization probability may contribute to the stability of minimal-sized sperm.

Can the classical theory also explain why minimal sperm are produced in such large numbers? For this, we need to know the optimal value for k, the proportion of a male's reproductive budget spent on sperm, under the selective pressure relating to increasing the probability of fertilization; call this k_P^*. I first concluded that the value of k_P^*, which is set by $P'(k_P^*) = P(k_P^*)/1$ (Equation (17) in Parker, 1984; see also Parker and Pizzari, 2010), could not readily account for sperm numbers (and hence the proportionate expenditure on sperm, k_P^*) seen in cattle. However, this conclusion was based on interpreting sperm *expenditure* as equivalent to sperm *number* in the published relationship between conception probability and sperm numbers, which is not correct. To derive the result in terms of sperm numbers, let s = the sperm number and D = the energetic cost of each sperm unit. If the costs of gaining a mating are C and the male has an energy budget for reproduction of R, his number of matings is $R/(C + Ds)$, and at each mating he gains a fertility probability of $P(s)$. His fitness is the product $w = P(s)R/(C + Ds)$, which is maximized when $dw/ds = 0$. Remembering that $k = Ds/(C + Ds)$, i.e. the proportion of total reproductive effort spent on the ejaculate when C is spent on gaining matings, this gives the result that

$$k_P^* = sP'(s)/P(s). \tag{1.8a}$$

For cattle (see Parker, 1982), the number of sperm ejaculated (s) is of the order of 10^{10}, and the probability of conception (P) around 0.75. The gradient of the probability of conception (P') with sperm numbers around the normal ejaculate size could not be evaluated, but from artificial insemination sperm dilution data, it could be deduced to be $<< 10^{-9}$. Until more accurate data can be analyzed, it is therefore not possible to deduce whether (1.8a) suggests a value that is too low to be plausible.

To compare (1.8a) with the ESS proportion of reproductive effort to spend on the ejaculate when there is sperm competition, consider a population where females have a low probability q of mating twice. Fitness can be calculated as $w = v(s)R/(C + Ds)$, where v is the expected value of a mating of a mutant with $s \neq s^*$, where s^* is the ESS sperm number. Thus $v(s) = [(1 - q) + 2qs/(s + s^*)]/(1 + q)$, and we obtain

$$k_{SC}^* = q/2 \tag{1.8b}$$

(Parker and Ball, 2005). Expressed in term of the probability p that a given males faces sperm competition this is $k_{SC}^* = p/(4 - 2p)$ (Parker, 1982; 1984). This is a plausible value for relative testes size (Parker, 1982). There is also considerable evidence suggesting that relative testes size increases with sperm competition risk in comparative studies across species (e.g. Parker and Pizzari, 2010). Thus sperm competition can readily explain why there are so many sperm produced in internal fertilizers.

Other theories for why so many tiny sperm are produced by internal fertilizers are reviewed in Pizzari and Parker (2009).

1.5.4 Summary of maintenance of anisogamy

As multicellularity increases, zygote size becomes increasingly advantageous, driving ovum size up. Simultaneously, sperm competition tends to maintain sperm at a minimal size, δ, constrained only by success in fertilization. The anisogamy ratio A (ovum size/sperm size) therefore becomes very large. This high A value generates the fundamental reason why sperm are expected to remain "minimal," i.e. adapted solely to the function of success in fertilization without making any contribution to zygote provisioning. Effectively, any small increase in sperm size to enhance zygote provisioning has trivial benefit, but since this increase occurs in all sperm of a mutant male, the cost in terms of

other selective forces that maintain sperm numbers at a high level is very significant. Thus anisogamy generally remains stable in multicellular plants and animals.

The main agent maintaining "minimal sperm" (or "minimal pollen") is probably sperm (or pollen) competition. Despite reductions in sperm competition with the evolution of internal fertilization in animals (or insect pollination in plants), under a sperm size–number trade-off, with $A >> 1$, sperm competition alone may still be generally sufficient to maintain anisogamy. Further, Parker (1984) has argued that in a species with zero male parental care, but with maternal care, the important index for stability of minimal sperm is not the anisogamy ratio A, but rather the investment ratio, I, which is: total energetic investment in the zygote by the female divided by the energetic cost of a sperm of size δ. For species with female parental care, this makes anisogamy highly robust by ensuring that minimal sperm are stable unless the risk of sperm competition is infinitesimal or zero.

There are also other selective pressures that will militate against increasing sperm size; these include the classical argument that sperm numbers increase fertilization probability, and the fact that sperm size may be traded off against other reproductive activities such as mate searching and paternal care.

1.6 Optimal sizes of the male and female gametes: anisogamy ratios

What determines the anisogamy ratio (A = ovum mass/sperm mass) in a species? These vary by many orders of magnitude, and though giant sperm do exist where A is low, but greater than unity (e.g. *Drosophila bifurca*, Bjork and Pitnick, 2006), A is usually a very large number.

Following the evolution of internal fertilization, the evolution of the anisogamy ratio, A, becomes complex, with largely independent selective pressures shaping ovum size and sperm size. Ovum size becomes specialized towards provisioning of the zygote, and sperm size is shaped by survival prospect up to fusion and competitive advantages of size under sperm competition.

1.6.1 Gamete size dimorphism from PBS theory

In extending PBS theory to include gamete survival in relation to gamete size (i.e. $g(m)$), as well as zygote survival in relation to zygote size ($f(S)$), Bulmer and Parker (2002) argued that once gamete

dimorphism arises by disruptive selection on an isogamous ancestral population with the same $g(m)$ function for the + and – mating types, the $g(m)$ function differentiates into two forms, $g_1(m_1)$ for microgametes and $g_2(m_2)$ for macrogametes. When a high anisogamy ratio has become established, sperm (size m_1) contributes insignificantly to the size of the zygote, which effectively becomes the ovum size (m_2), i.e. $S = m_1 + m_2 \approx m_2$ (see also Parker, 1982). Sperm are selected to maximize $g_1(m_1)/m_1$, so that the optimal sperm size can be found by the Smith and Fretwell (1974) equation

$$[g_1(m_1)]' = g_1(m_1)/m_1. \tag{1.9a}$$

In contrast, ova are selected to maximize $g_2(m_2)f(m_2)/m_2$, where $g_2(m_2)$ is the ovum's survival probability in relation to its size, up to the time of fertilization, and $f(m_2) = f(S)$ is the zygote survival probability.

As multicellularity and complexity increases, function $f(m_2)$ ensures that egg size is typically large in higher organisms. This high provisioning, coupled with the high sperm densities typical when the ova are available for fertilization, make it inevitable that ova have high survival prospects before fusion. Where good data exist (fish, insect, birds, mammals), large changes in sperm numbers have little effect on fertilization probability, which typically approaches 1.0 (Ball and Parker, 2000). Thus in most higher organisms, the probability of ovum survival to fusion, $g_2(m_2)$, is likely to be independent of quite large changes in m_2, at least in the general region of typical ovum size, so that ova are selected to maximize $f(m_2)/m_2$, i.e. the optimal ovum size also satisfies the Smith and Fretwell (1974) equation

$$f'(m_2) = f(m_2)/m_2. \tag{1.9b}$$

Effectively, ovum size becomes determined entirely by $f(m_2)$, the principle used to consider conditions for the stability of anisogamy in higher organisms (Parker, 1982; see Section 1.5.1).

However, an exception to this principle occurs in species that are often sperm limited, such as certain marine external fertilizers, where fusion probability increases with ovum size (Levitan 1993), so that $g_2(m_2)$ is a significantly increasing function of m_2 in the region of the optimal ovum size. Here the appropriate Smith–Fretwell formulation to derive the optimal egg size is

$$[g_2(m_2)f(m_2)]' = g_2(m_2)f(m_2)/m_2 \tag{1.9c}$$

(Bulmer and Parker, 2002), which gives a larger ovum size than equation (1.9b). Levitan (1993; 1996a) gives evidence for such deviations in marine invertebrates.

Once anisogamy develops, other selective forces shape the development of micro- and macrogametes: sperm become or remain very small and retain their motility (Parker, 1982; Randerson and Hurst 2001a) and ova become oogamous (non-motile). I next review briefly models for the subsequent specialization of the gamete sizes.

1.6.2 Optimization of ovum size

The fact that sperm contribute little or nothing to the future survival/fitness of the zygote means, as we have seen, that optimal ovum size can be established from the Smith and Fretwell (1974) model. The topic is too large to be reviewed here, but most analyses of optimal ovum size use variants of the Smith–Fretwell approach. This is a non-competitive optimum, which suggests that when resources for reproduction in a species vary, number rather than size of ova, should vary (a prediction often approximated, see Maynard Smith, 1978). Competitive effects between ova are rarely modeled. Under sperm limitation theory, "target gamete" increases in egg size (e.g. Levitan, 1993) arise through egg competition for sperm only if fertilizations remove ova fast enough from the sperm pool that they decrease the fertilization rate of other ova (Levitan, 1996; Lessells *et al.*, 2009). Within-clutch sib-competition between developing zygotes can also increase egg size (e.g. Parker and Begon, 1986).

The range of ovum size in vertebrates is vast, with diameters ranging from a tiny 50 μm in the field vole, *Microtus agrestis*, to a massive 15 cm (with volume 1.77 liters) in the shark, *Chlamydoselachus auguineus* (Lombardi, 1998). Examples of two distinct seed sizes in certain plants probably relate to differences in dormancy, dispersal, or ecological niches (see Maynard Smith, 1978).

1.6.3 Sperm competition and the evolution of sperm size

Section 1.5.1 outlined reasons why sperm mass should relate only: (i) to carrying the haploid chromosomes, (ii) to optimizing survival before fusion, or (iii) to increasing competitiveness at fertilization. On this prediction, sperm should not include any component for provisioning the zygote, unless this arises as material recovered from expenditure

on sperm survival or fertilization competitiveness. However, although sperm are small in total mass compared to ova, like ova, their size variation is vast. For example, a typical bird sperm may be around 150 μm in length (Birkhead and Møller, 1998), but the largest sperm is that of *Drosophila bifurca* which is 58.3 mm long (Pitnick *et al.*, 1995). It is nevertheless well below the mass of the ovum (i.e. $A > 1.0$); Bjork and Pitnick (2006) calculate a sperm/egg production rate ratio of 5.8/1. How can we explain these large variations in sperm size?

Parker (1993; see also Snook, 2005; Pizzari and Parker, 2009) argued that increased sperm size may either: (i) unilaterally increase energy reserves, and hence increase sperm survival to fertilization (see also Bulmer and Parker, 2002), or (ii) unilaterally increase sperm tail and motility machinery, which would decrease survival, since energy reserves would be used up faster (Gomendio and Roldan, 1991), or (iii) increase both. Thus if sperm are not fully provisioned by the female, increased sperm size may "buy" either increased survival, or faster motility (i.e. higher competitive loading through increasing swimming speed) or some other competitive advantage under sperm competition, or both simultaneously.

The models in Sections 1.6.3.1 to 1.6.3.3 below assume that the sperm characteristics are determined by the parent that produces them; evolutionary conflict between parent and gamete can occur when sperm traits are determined by the haploid gametes of a diploid parent (Section 1.6.3.4).

1.6.3.1 Sperm size and number under sperm competition risk: competitive loadings

Most models for the evolution of sperm size generally (like those for ovum size) assume that gamete size is determined by the genotype of the diploid "parent" (Parker, 1993; Parker *et al.*, 2010); obvious exceptions are animals with haplodiploidy, and plants that produce gametes in the haploid phase. Very different conclusions from those given below are generated if sperm size is determined by the genes of the haploid sperm under diploidy (Parker and Begon, 1993).

Much theoretical work has centered on the topic of sperm competition and ejaculate economics (Parker and Pizzari, 2010) and on how sperm competition may influence sperm size and number variation (Pizzari and Parker, 2009). A general approach to size–number variation, using the sperm competition risk model where females mate

twice with probability q and once with probability $(1 - q)$, has been developed by Parker (1993) and Parker *et al.* (2010).

Calling sperm size or mass m, and sperm number s, a male's total investment in the ejaculate will be proportional to the product, ms. Parker *et al.* (2010) showed the ESS sperm size and number (m^*, s^*) at a given level of sperm competition risk (q = the probability that a female mates twice) in a population are

$$m^* = \beta G'(s^*, \mathrm{I}), \tag{1.10a}$$

$$s^* = \beta G'(m^*, \mathrm{I}), \tag{1.10b}$$

where

$$\beta \equiv \left(\frac{R}{D}\right)\left(\frac{2q}{1+q}\right), \tag{1.10c}$$

R is the resource available to a male for reproduction (sperm production and acquiring matings), and D is the cost of one unit of ejaculate (each male spends units of his reproductive budget R per mating on his ejaculate). Hence we obtain the general rule

$$s^* G'(s^*) = m^* G'(m^*) \tag{1.11}$$

(Parker, 1993; Parker *et al.*, 2010), i.e. the product of sperm number and the marginal gains through increasing sperm numbers, $G'(s^*)$, must equal the product of sperm size times the marginal gains through increasing sperm size, $G'(m^*)$. Though rule (1.11) above indicates how the balance between sperm size and number is attained at the ESS, to deduce how sperm competition shapes sperm size and number, explicit functions for $G(s)$ and $G(m)$ are required.

Parker *et al.* (2010) investigated three different mechanisms of sperm competition (Figure 1.5). In the simplest mechanism, sperm compete in a fertilization raffle with no constraint on the space available for competing ejaculates, such as may occur in many external fertilizers. This commonly differs from internal fertilizers, where there may be some space constraint on storage of ejaculates due to limitations imposed by the female tract. This constraint is most severe in species with small, fixed-volume sperm stores, as in many insects; in such systems the most recent ejaculate often displaces some of the previously stored ejaculate(s) from a female's sperm stores. Two forms of displacement were studied: (i) displacement mainly by seminal fluid, so that any small changes in sperm mass do not affect the amount of

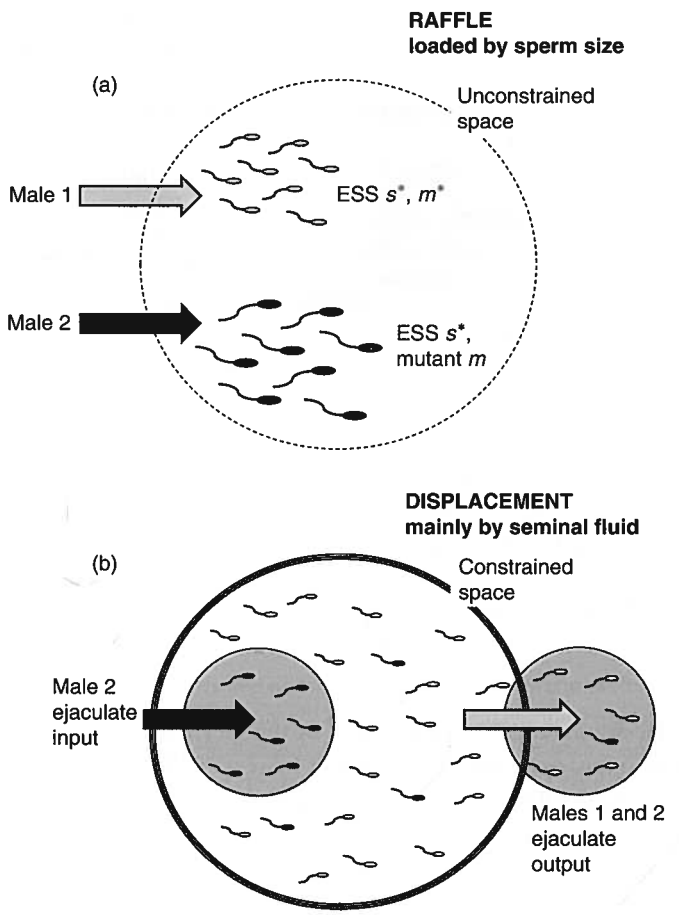

Figure 1.5 Mechanisms of sperm competition investigated in the Parker *et al.* (2010) models for sperm size and number. (a) Raffle. Males ejaculate into an unconstrained space from which sperm are drawn for fertilization; there is no displacement of previous sperm before fertilization. The ESS sperm mass is m^* and sperm number is s^*. Mutant male 2 has larger sperm ($m > m^*$). Fertilizations are drawn from the sperm available after both males have ejaculated, after the loaded raffle principle in which the raffle is loaded by sperm size through function $r(m)$ (see Equation (1.12)). (b) Displacement mainly by seminal fluid. During ejaculation of the second male there is continuous displacement of previously stored sperm from the constrained sperm storage space in the female tract following the model of direct displacement with instant mixing (Parker and Simmons, 1991). Each sperm is transferred in a fixed volume of seminal fluid that is much larger than the sperm mass, so that deviations in sperm size have a negligible effect on displacement.

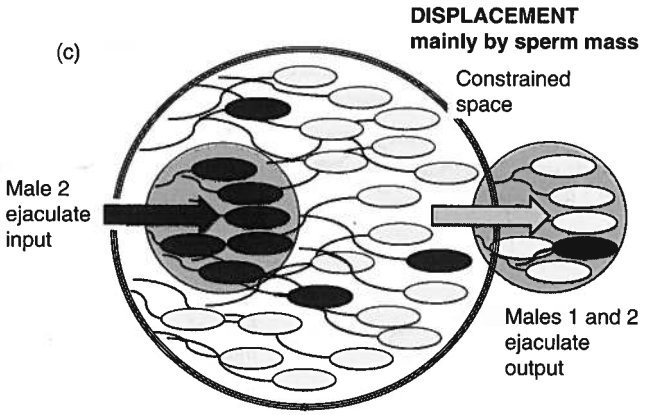

Figure 1.5 (cont.) (c) Displacement mainly by sperm mass. Displacement is as in (b), except that the seminal fluid volume per sperm is negligible, so that for a fixed number of sperm, displacement increases with sperm mass. In both (b) and (c), once ejaculation of the second (last) male is complete, the fertilization follows a raffle loaded by sperm size as in (a). With permission from Parker *et al.* (2010).

displacement, and (ii) displacement by the sperm mass itself, so that as sperm increase in size, displacement increases. These different mechanisms have different forms for the gain through increases in sperm numbers, $G(s)$, in (1.11) above.

Parker *et al.* (2010) showed that the product, m^*s^*, was either constant (in the raffle or the sperm mass-displacement models) or highly invariant (the seminal-fluid-displacement model) at a given value for β (which defines the resources available and the sperm competition risk level, q, see (1.10c)), and that m^*s^* increases with the risk of sperm competition, q (Figure 1.6a). Thus, effectively, there is a direct sperm size–number trade-off within the ejaculate allocation m^*s^* at a given level of β.

How sperm size and number are allocated as m^*s^* increases across populations with the risk of sperm competition depends on the marginal gains through increasing sperm numbers, $G'(s^*)$, or sperm size, $G'(m^*)$; see Equation (1.11). If the sperm competition mechanism is a raffle (i.e. no constraint on space for competing ejaculates) then the sperm numbers in the set of sperm that compete for fertilizations (the "fertilization set"; Parker *et al.*, 1990) are directly proportional to the sperm numbers ejaculated. However, the composition of

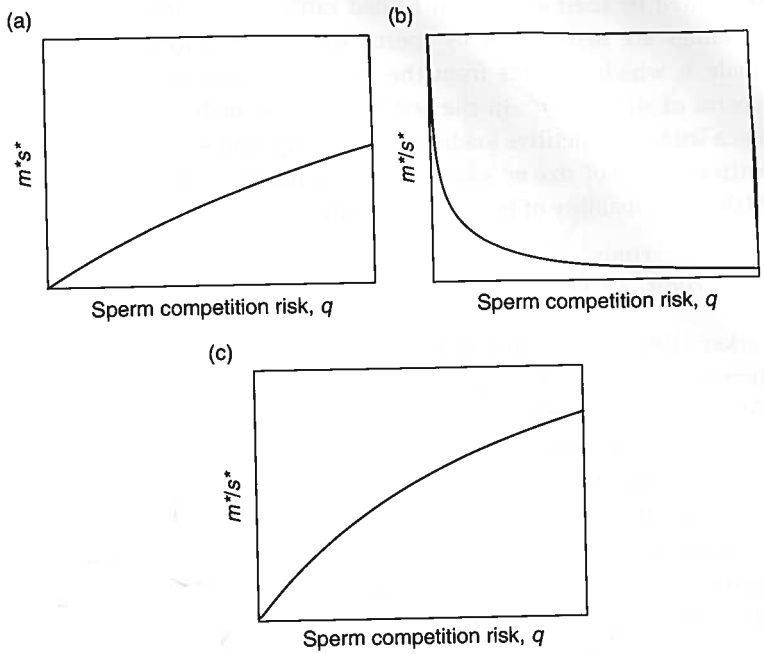

Figure 1.6 Sperm size and number in relation to sperm competition risk, q, in the models of Parker *et al.* (2010). (a) The total ejaculate expenditure (the product of sperm size and number, m^*s^*, increases with q, and is influenced only by the constants, R, D, and q, in β (see (1.10c)). As m^*s^* increases with q, m^* remains constant if the competitive loading for sperm, r, is a function only of m. Thus the increase in m^*s^* is due entirely to an increase in sperm numbers, s^*, leading to a declining relation between m^*/s^* and q as in (b) (the "default expectation"). This declining relation (b) was also common when the competitive loading due to sperm size, function $r(m)$, was distorted by sperm density, as in Figure 1.7 (c to e). (c) An increasing relation between m^*/s^* and q can occur only if there is greater investment in sperm size than number as risk increases. In Parker *et al.* (2010), this required sperm density to decrease the slope of $r(m)$, or to increase the intercept of $r(m)$ (see text). With permission from Parker *et al.* (2010) and Immler *et al.* (2011).

the fertilization set in the displacement models depends on how much of the first male's sperm has been displaced by the second male to mate. In all three mechanisms studied (Figure 1.5), Parker *et al.* (2010) assumed that once the sperm numbers from the competing males in the fertilization set have been determined (i.e. after displacement has occurred), sperm then compete in proportion to their numbers

weighted by their size (i.e. a loaded raffle, where the competitive loadings are determined by sperm size). Thus consider a mutant male 1, which deviates from the ESS sperm size, m^*, by having a sperm of size $m \neq m^*$. In the fertilization set, male 1 has s_1 sperm, each with a competitive loading of $r(m)$. They compete against male 2 with s_2 sperm of size m^* each having a competitive loading of $r(m^*)$. Male 1's probability of fertilization is thus

$$\frac{r(m)s_1}{r(m)s_1 + r(m^*)s_2}. \tag{1.12}$$

Parker (1993) and Parker *et al.* (2010) found that certain forms for increasing $r(m)$ yield a maximum or minimum (Figure 1.7a); for the ESS to be an intermediate sperm size, $r(m)$ must be of a form such that a tangent can be drawn from the origin (Figure 1.7b) following the marginal value theorem principle (Charnov, 1976; Parker and Stuart, 1976) and the Smith-Fretwell (1974) model for optimal ovum size. However, if r is solely a function of sperm mass, m, then the ESS sperm size m^* is constant and independent of sperm competition risk, q (Parker, 1993; Parker *et al.*, 2010). If m^* is constant with q, and m^*s^* increases with q (Figure 1.6a), then as q increases, the ratio sperm size/number (m^*/s^*) decreases with q (Figure 1.6b). This could be defined as the "default expectation" (Immler *et al.*, 2011), i.e. as sperm competition risk increases, relatively more is invested in sperm numbers than in sperm size. But some taxa show increases in both sperm number and sperm size as risk, q, increases, and *Drosophila* species show evidence (see Immler *et al.* (2011) that this involves relatively more being invested in sperm size than in sperm numbers, since m^*/s^* increases with q (Figure 1.6c). How can this be explained?

One possibility is that increases in sperm density generated by increased sperm competition risk cause changes in the form of $r(m)$. Discounting forms for $r(m)$ that yield maxima or minima (Figure 1.7a), consider forms that yield an intermediate ESS sperm size, m^* (Figure 1.7b). If r is a function of sperm density in the fertilization set, as well as a function of sperm size, m^* can vary with sperm competition risk (Parker, 1993; Parker *et al.*, 2010). How such distortions in the form of r through increases in sperm density change the allocation between sperm size and number as sperm competition risk increases is a complex problem. Parker *et al.* (2010) analyzed how ESS values for sperm size and number (m^*, s^*) should vary with sperm competition risk q, using an exponentially diminishing returns form for $r(m,s)$ that changes with the sperm density in the fertilization set in three ways

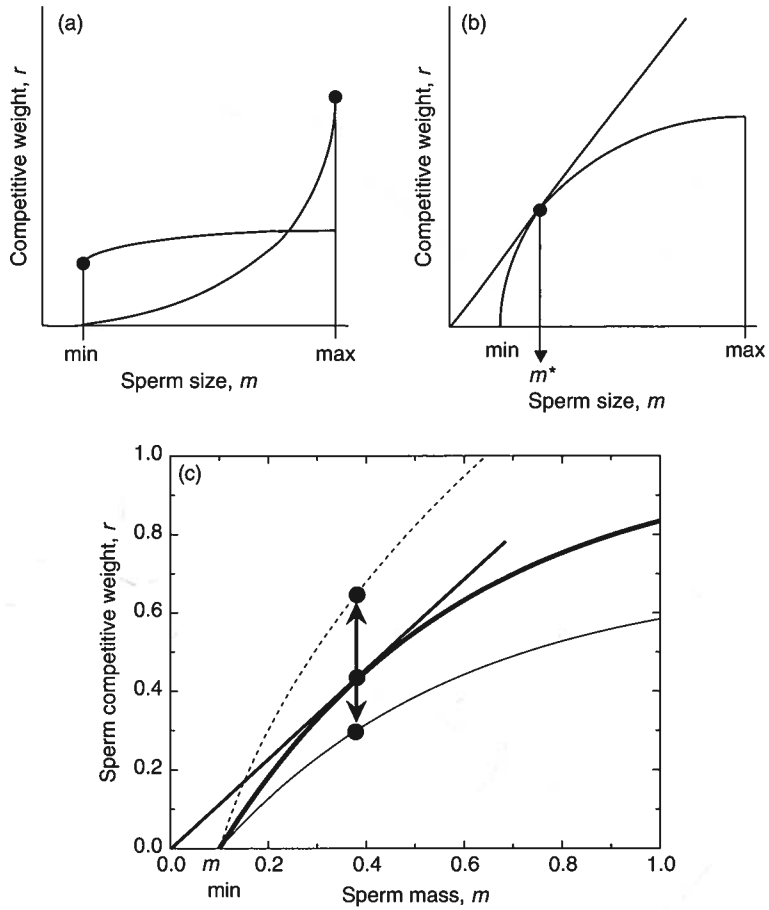

Figure 1.7 A sperm's competitive loading, r, in the fertilization raffle is set by its size, m, and we assume that $r(m)$ is monotonic increasing; (a) shows two forms for $r(m)$ that will not yield an intermediate ESS sperm size, m^*; instead they give a minimum or maximum size shown by the filled circles. (b) An intermediate ESS sperm size is attained if a tangent can be drawn from the origin to $r(m)$; the filled circle shows the ESS Graphs. (c–e) show how the form of $r(m)$ may be distorted, changing the ESS (open circles) as sperm density increases due to increasing risk, q. (c) Increasing sperm density may increase (dotted curve) or decrease (thin continuous curve) the asymptote of $r(m)$. As shown here, this does not affect the ESS m^* in the sperm competition mechanisms in Figures 1.5a and 1.5b, but it does change the ESS m^* in the mechanism in Figure 1.5c.

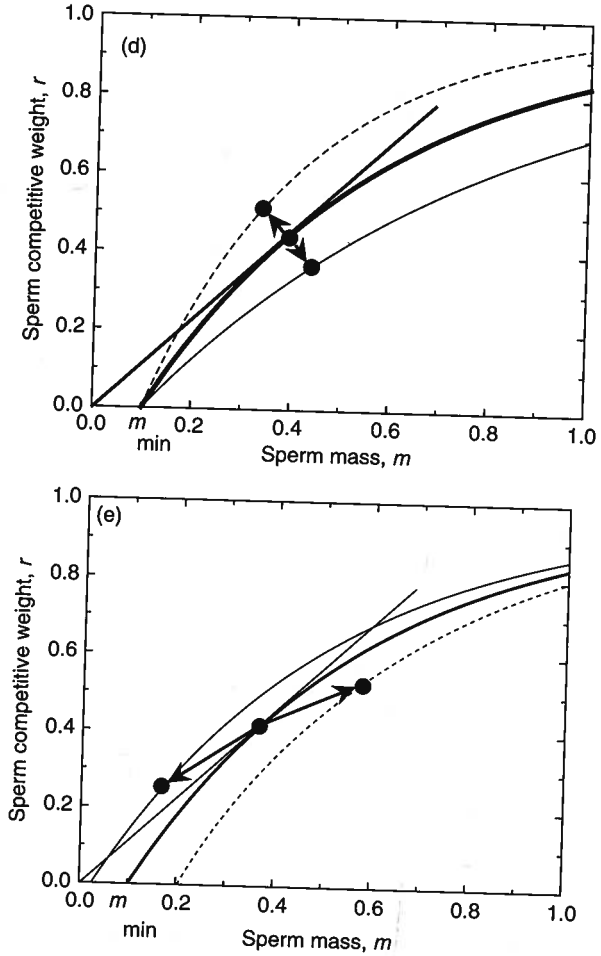

Figure 1.7 (cont.) (d) Increasing sperm density may increase (dotted curve) or decrease (thin continuous curve) the slope of $r(m)$. If the slope increases, m^* decreases, and if the slope decreases, m^* increases. (e). Increasing sperm density may increase (dotted curve) or decrease (thin continuous curve) the intercept of $r(m)$. If the intercept increases, m^* increases, and if the intercept decreases, m^* decreases. Graph (a) and (b) from Parker (1993); (c–e) modified from Parker *et al.* (submitted).

(Figure 1.7c–e). Increases in sperm density were used to change: (i) the asymptote value of $r(m)$ (Figure 1.7c), (ii) the rate at which $r(m)$ rises to its asymptote (the slope of $r(m)$; Figure 1.7d), or (iii) the minimum competitive sperm size (the intercept of $r(m)$; Figure 1.7e).

The results can be summarized as follows. In all models, s^* and m^*s^* increase monotonically with q, but the effect of sperm numbers on sperm size depends critically on how sperm density changes $r(m)$. If sperm density changes the asymptote of $r(m)$, sperm size varies with q only when the displacement mechanism is by sperm mass (Figure 1.5c). Here, if sperm density *increases* the asymptotic value of r, m^* increases with q, and if increasing sperm density *decreases* the asymptotic value of r, m^* decreases with q. In the other two models (Figure 1.5a, b) sperm size is unaffected by a change in the asymptote of $r(m)$ (as shown in Figure 1.7a).

However, in all three models in Figure 1.5, changes in the slope or intercept of $r(m)$ with increasing sperm density cause m^* to change with q (see Figure 1.7b, c): m^* increases with q if increased sperm density increases the intercept or decreases the slope of $r(m)$, and m^* decreases with q if increased sperm density decreases the intercept or increases the slope of $r(m)$.

Most effects of sperm density were found to generate the default relation in which relatively more is spent on sperm numbers than on sperm size as risk increases (i.e. a negative relationship between m^*/s^* and risk, q, as in Figure 1.6b). In the raffle (Figure 1.5a) and displacement by seminal fluid (Figure 1.5b) models, very steeply increasing effects of sperm density on the slope or intercept of $r(m)$ are needed to generate a positive relationship between m^*/s^* and risk, q (Figure 1.6c). However, in the displacement by sperm mass model (Figure 1.5c), this positive relationship could be generated even with linear increasing effects of sperm density.

These effects of sperm density on the competitive value of sperm size have different biological implications. If sperm density decreases the slope of $r(m)$, this suggests that the advantage of sperm size decreases as density increases. If the intercept of $r(m)$ is increased by sperm density, this suggests that sperm size becomes increasingly important in competition as sperm density increases, since the minimum size for a sperm to have any chance of fertilization increases as sperm density increases.

Thus theoretically there are reasons why sperm size may increase, remain constant, or decrease with sperm competition risk across species, and all such patterns have been reported for different groups (Pizzari and Parker, 2009). The ratio of sperm size/number has rarely been studied, but is important since it informs how the trade-off between sperm size and number shifts as the overall ejaculate expenditure, ms, increases with sperm competition risk. The work of Parker

et al. (2010) suggests that m^*/s^* would most commonly decrease with risk across species or populations (following the default expectation; Figure 1.6b). An increasing relationship between m^*/s^* and risk (Figure 1.6c) suggests that, counter to the default expectation, relatively more is spent on sperm size than on sperm number. This seems most likely to be found only when there are extreme competitive interactions between sperm so that the minimum size for successful sperm increases with sperm density.

Some evidence for these predictions was found in passerine birds and drosophilid flies (Immler *et al.*, 2011). As predicted, across both groups, the product ms increased with risk (as does testis size in many species, Parker and Pizzari, 2010). In passerines, both sperm size, m^*, and number, s^*, increased with risk, but m^*/s^* decreased (following the default expectation, Figure 1.6b). In drosophilids, sperm size increased with risk, but at an increasing rate – a decreasing rate is predicted by Parker *et al.* (2010). Further, in drosophilids, sperm number initially increases then decreases with risk (continuous increases in sperm number are predicted by Parker *et al.*, 2010). However, the ratio m^*/s^* increased with risk (Figure 1.6c), suggesting that competition becomes more intense with sperm competition density with the minimum sperm size increasing with sperm density.

1.6.3.2 *Sperm size and sperm survival under sperm competition risk*

Using a similar sperm competition risk approach, Parker (1993) calculated ESS sperm size and number when two males mate sequentially with a female before fertilization occurs: assuming that size increases sperm survival, the ESS sperm size increases (and ESS sperm number decreases) with the time delay between matings and the time from the last mating to fertilization.

1.6.3.3 *Sperm size and number under sperm competition intensity*

Ball and Parker (1996) developed a model of a continuous fertilization process relating to external fertilizers, such as many fish species, in which eggs and sperm are shed simultaneously. Eggs are fertilized at a rate proportional to sperm density surrounding the egg mass and to the "aptitude for fusion," α; increasing α increases the rate of fertilization (and the proportion of eggs fertilized) at a given sperm density. The

model investigated both the risk range (females mate once with probability $(1-q)$, and twice with probability q) and intensity range (where N ejaculates compete) of sperm competition, and predicted how ESS sperm size and number, and the ESS level of infertility (the eggs remaining unfertilized after all sperm have died) should vary across species or populations. Increased sperm mass, m, was assumed to increase sperm competitive ability, for example by increasing swimming speed, r, and could affect sperm longevity either positively or negatively through a survival function $\tau(m)$, which defined the length of time a sperm survives if it has mass m. As in other sperm competition games, ejaculate expenditure is traded off against expenditure on obtaining further matings (here spawnings).

This model predicted that, across species, the ESS ejaculate expenditure (m^*s^*) increases (as with the previous risk models) with sperm competition intensity, N. The balance between sperm size and number shifts between two extremes, with one optimum at $N = 1$ (zero competition), and the other at maximum sperm competition ($N \to \infty$). The non-competitive optimum (at $N = 1$) maximizes the total distance traveled by the entire ejaculate in its lifetime, i.e. it conserves sperm longevity to gain the most possible fertilizations from the ejaculate, and has

$$m^* = \left[\frac{r'(m^*)}{r(m^*)} + \frac{\tau'(m^*)}{\tau(m^*)}\right]^{-1}, \tag{1.13a}$$

(Ball and Parker, 1996).

In contrast, the optimum for maximum competition (at $N \to \infty$) maximizes the product of sperm speed and sperm number, i.e. it "snatches" the biggest share of fertilizations by focusing on the first instant of the fertilization process. It has the familiar marginal value form found for risk models by Parker (1993) and Parker *et al.* (2010):

$$m^* = \frac{r(m^*)}{r'(m^*)}. \tag{1.13b}$$

The ESS sperm size and number are shown in relation to sperm competition intensity, N, in Figure 1.8. Suppose that longevity decreases with sperm size (i.e. $\tau'(m)$ is negative), because the main increase is in tail length, which increases sperm energy expenditure. Then sperm size should increase with N, between the non-competitive and the competitive optima (Figure 1.8a, broken curve). However, if longevity increases with sperm size (i.e. $\tau'(m)$ is positive), then the non-competitive optimal

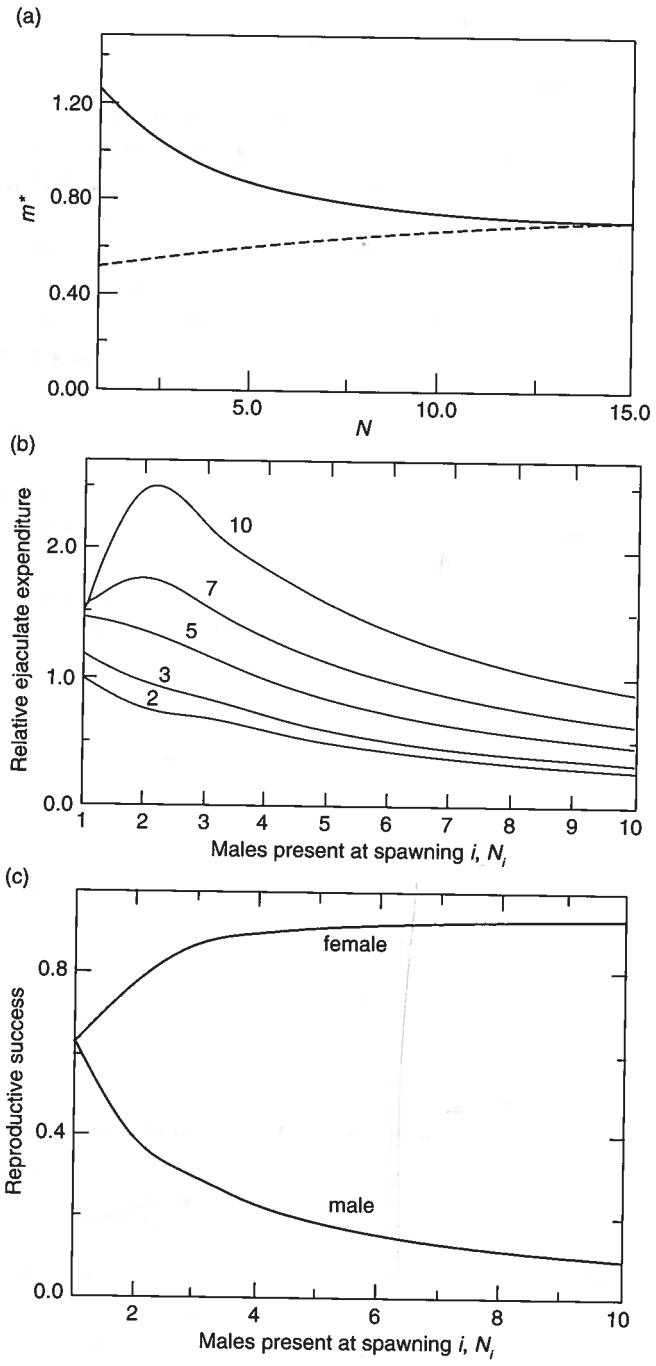

Figure 1.8 (a) Results from the model of Ball and Parker (1996), showing the relation between sperm size, m^*, and sperm competition intensity

sperm size is greater than that for maximum competition, and sperm size decreases with N (Figure 1.8a, continuous curve). In this continuous fertilization model, sperm numbers, s^*, and the total ejaculate investment, m^*s^*, always increase with N across high sperm competition intensities, whether $\tau'(m)$ is positive or negative. Decreases in s^* are possible only over a range of low sperm competition risk if sperm longevity decreases with sperm size ($\tau'(m)$ is negative), and if infertility is high.

Ball and Parker (1997) extended this continuous fertilization model to investigate how the ESS sperm number, s^*, and the ejaculate expenditure (m^*s^*) should vary within a species or population when males can assess the number of competitors present at a given spawning. We assumed that the sperm are manufactured and stored before ejaculation, so that the ESS sperm mass m^* is constant and shaped only by the mean number, N, of males present in a species at a spawning, but the ESS sperm number, s^*, can be varied strategically at a given spawning in relation to the number of males present, N_i. In this model, we assumed that increased sperm mass mainly caused increased tail length and hence reduced sperm longevity because of faster use of the sperm's energy reserves. The results for the average expenditure (m^*s^*) in relation to the average intensity, N, are similar to those of the previous model (Ball and Parker, 1996) where males cannot assess the number of competitors present at a spawning. However, expenditure in relation

Caption for Figure 1.8 (cont.)
across species that vary in the number N of males' ejaculates at spawnings. The lifespan, τ, of a sperm may increase or decrease with sperm mass, m; the upper curve shows that if $\tau(m)$ is increasing, sperm size reduces across species with N; the lower curve for $\tau(m)$ decreasing causes sperm size to increase across species with N. At $N=1$ there is no sperm competition and the non-competitive optimum (Equation (1.13a)); as sperm competition intensities become very high, $\tau(m)$ has no effect on m^* (equation (1.13b)), modified from Ball and Parker (1996). (b) Results from the model of Ball and Parker (1997) showing the relation between ejaculate expenditure, m^*s^*, and sperm competition intensity in species where males can assess N_i, the number of males present at a given spawning, i. Each separate curve represents a species; the average number N of males at spawnings in that species is shown against the curve. (c) Reproductive success achieved by males and females in the model of Ball and Parker (1997) in relation to the number of competing males, N_i, present at spawning i. (b) and (c) With permission from Ball and Parker (1997).

to the local sperm competition level varies with N_i, due to strategic adjustments of sperm numbers (Figure 1.8b). Depending on the population average levels of competition, N, two patterns emerge for the relation between the number of competitors present at a spawning, N_i. If the average N is low, ejaculate expenditure, m^*s^*, declines with N_i; males expend most sperm when on their own ($N_i = 1$) and m^*s^* decreases as N_i increases. At higher average N, m^*s^* increases between $N_i = 1$ and $N_i = 2$, and then decreases as N_i increases (see also Parker *et al.*, 1996 for a similar result with instantaneous fertilization). For any given level of average competition, N, and local competition, N_i, reducing α (the aptitude for fusion) increases m^*s^* and reduces the fertility of the spawning.

An interesting effect of the strategic allocation by males, s_i^* sperm when there are N_i males present at a spawning, is that fertility increases with N_i. Thus there is sexual conflict over the number of sperm ejaculated; a given female does best if she spawns when N_i is high, and a given male gains most when N_i is low (Figure 1.8c). Thus females may be selected to prefer to spawn in large groups of males (Shapiro and Giraldeau, 1996) while males should act aggressively to attempt to dispel competitors.

1.6.3.4 *Haploid control of sperm size and number under diploidy*

A given allele in a diploid parent will be present in only half of the haploid sperm that the parent produces. Though the classical view is that sperm characteristics are determined by the diploid parental genotype, intraejaculate competition can occur if the characteristics of the sperm are determined by the sperm haplotype. Coupled with interejaculate (= sperm) competition, this generates "gamete–parent conflict" because the ESS balance between size and number of sperm in an ejaculate is quite different under haploid control and diploid control (Parker and Begon, 1993). Conflict between haploid and diploid expression is reduced, but is not lost, as interejaculate competition increases, and may have a variety of consequences relating to allocation of sperm size and number, and their variation in natural populations (see Parker and Begon, 1993).

1.7 ANISOGAMY AS A STAGE IN THE EVOLUTIONARY CHAIN OF SEXUALITY

The evolution of sexuality consists of a series of sequential steps, each one catalyzed by its predecessor. Selection for sexual recombination favored the evolution of fusion and gametes, probably in isogamous

populations that released gametes into an external medium (the sea). The evolution of increasing vegetative complexity, usually in the form of complex multicellular organization, generated increasing importance of zygote size for zygote fitness, hailing the origin of anisogamy coupled with the evolution of disassortative fusion from existing linkage of genes for gamete size with those for mating types, if mating types were already present in the ancestral isogamous population (Charlesworth, 1978). If mating types had not already evolved in the ancestral population, early anisogamy without mating types (pseudo-anisogamy) would be followed by selection for disassortative fusions (Parker, 1978). Both pathways would quickly generate the unity sex ratio from Fisher's (1930) principle. This in turn results in sexual selection, both pre- and postcopulatory, and evolution of internal fertilization and reduced sperm competition, with the subsequent specialization of the sperm cell. The theory of disruptive selection by gamete competition still remains a candidate as the most powerful explanation of the origin of anisogamy (Lessells *et al.*, 2009), and hence of male and female phenotypes, which in turn resulted in sexual selection and sexual conflict, leading ultimately to the vast diversity of adaptations that we associate with the two sexes.

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REFERENCES

Alexopoulos, C. J. (1962). *Introductory Mycology*. New York: John Wiley & Sons, Inc.

Arnqvist, G. and Rowe, L. (2005). *Sexual Conflict*. Princeton, NJ: Princeton University Press.

Ball, M. A. and Parker, G. A. (1996). Sperm competition games: external fertilization and "adaptive" infertility. *Journal of Theoretical Biology*, **180**, 141–150.

Ball, M. A. and Parker, G. A. (1997). Sperm competition games: inter- and intraspecies results of a continuous external fertilization model. *Journal of Theoretical Biology*, **186**, 459–466.

Ball, M. A. and Parker, G. A. (2000). Sperm competition games: a comparison of loaded raffle models and their biological implications. *Journal of Theoretical Biology*, **206**, 487–506.

Bell, G. (1978). The evolution of anisogamy. *Journal of Theoretical Biology*, **73**, 247–270.

Bell, G. (1982). *The Masterpiece of Nature*. London: Croom Helm.
Bell, G. (1985). The origin and evolution of germ cells as illustrated by the Volvocales. In H. O. Halvorson and A. Monroy (editors), *Orgin and Evolution of Sex*. New York: Allan R. Liss, pp. 221–256.
Birkhead, T. R. and Møller, A. P. (editors) (1998). *Sperm Competition and Sexual Selection*. London: Academic Press.
Bjork, A. and Pitnick, S. (2006). Intensity of sexual selection along the anisogamy-isogamy continuum. *Nature*, **441**, 742–745.
Bonsall, M. B. (2006). The evolution of anisogamy: the adaptive significance of damage, repair and mortality. *Journal of Theoretical Biology*, **238**, 198–210.
Bryan J. H. (1968). Results of consecutive matings of female *Anopheles gambiae* species *B* with fertile and sterile males. *Nature*, **218**, 489.
Bulmer, M. G. (1994). *Theoretical Evolutionary Ecology*. Sunderland, MA: Sinauer Associates Inc.
Bulmer, M. G. and Parker, G. A. (2002). The evolution of anisogamy: a game-theoretic approach. *Proceedings of the Royal Society of London B*, **269**, 2381–2388.
Bulmer, M. G., Luttikhuizen, P. C., and Parker, G. A. (2002). Survival and anisogamy. *Trends in Ecology and Evolution*, **17**, 357–358.
Charlesworth, B. (1978). The population genetics of anisogamy. *Journal of Theoretical Biology*, **73**, 347–357.
Charnov, E. L. (1976). Optimal foraging: the marginal value theorem. *Theoretical Population Biology*, **9**, 129–136.
Clutton-Brock, T. H. and Vincent, A. C. J. (1991). Sexual selection and the potential reproductive rates of males and females. *Nature*, **351**, 487–489.
Cosmides, L. M. and Tooby, J. (1981). Cytoplasmic inheritance and intragenomic conflict. *Journal of Theoretical Biology*, **89**, 83–129.
Cox, P. A. and Sethian, J. A. (1984). Search, encounter rates, and the evolution of anisogamy. *Proceedings of the National Academy of Sciences of the United States of America*, **81**, 6078–6079.
Cox, P. A. and Sethian, J. A. (1985). Gamete motion, search and the evolution of anisogamy, oogamy, and chemotaxis. *American Naturalist*, **125**, 74–101.
Czárán, T. L. and Hoekstra, R. F. (2004). Evolution of sexual symmetry. *BMC Evolutionary Biology*, **4**, 34.
Dusenbery, D. B. (2000). Selection for high gamete encounter rates explains the success of male and female mating types. *Journal of Theoretical Biology*, **202**, 1–10.
Dusenbery, D. B. (2002). Ecological models explaining the success of distinctive sperm and eggs (oogamy). *Journal of Theoretical Biology*, **219**, 1–7.
Dusenbery, D. B. (2006). Selection for high gamete encounter rates explains the evolution of anisogamy using plausible assumptions about size relationships of swimming speed and duration. *Journal of Theoretical Biology*, **241**, 33–38.
Emlen, S. T. and Oring, L. W. (1977). Ecology, sexual selection, and the evolution of mating systems. *Science*, **197**, 215–223.
Eshel, I. (1983). Evolutionary and continuous stability. *Journal of Theoretical Biology*, **103**, 99–111.
Fisher, R. A. (1930). *The Genetical Theory of Natural Selection*. Oxford: Clarendon Press.
Gomendio, M. and Roldan, E. R. S. (1991). Sperm competition influences sperm size in mammals. *Proceedings of the Royal Society of London B*, **243**, 181–185.
Hoekstra, R. F. (1984). Evolution of gamete motility differences. II. Interaction with the evolution of anisogamy. *Journal of Theoretical Biology*, **107**, 71–83.

Hoekstra, R. F. (1987). The evolution of sexes. In S. C. Stearns (editor), *The Evolution of Sex and its Consequences*. Basel: Birkhäuser Verlag, pp. 59–91.
Hoekstra, R. F. (1990). The evolution of male-female dimorphism – older than sex. *Journal of Genetics*, **69**, 11–15.
Hurst, L. D. (1996). Why are there only two sexes? *Proceedings of the Royal Society of London B*, **263**, 415–422.
Kalmus, H. (1932). Über den Erhaltungswet den phenotypishen (morphologishen) Anisogamie und die Entstehung der ersten Geshlectsuntershiede. *Biologische Zentralblatt*, **52**, 716.
Kalmus, H. and Smith, C. A. B. (1960). Evolutionary origin of sexual differentiation and the sex-ratio. *Nature*, **186**, 1004–1006.
Knowlton, N. (1974). A note on the evolution of gamete dimorphism. *Journal of Theoretical Biology*, **46**, 283–285.
Hoekstra, R. F., Janz, R. F., and Schilstra, A. J. (1984). Evolution of gamete motility differences I. Relation between swimming speed and pheromonal attraction. *Journal of Theoretical Biology*, **107**, 57–70.
Immler, S., Pitnick, S., Parker, G. A., *et al.* (2011). Resolving variation in reproductive trade-offs: sperm size versus number. *Proceedings of the National Academy of Sciences* (in press).
Iwasa, Y. and Sasaki, A. (1987). Evolution of the number of sexes. *Evolution*, **41**, 49–64.
Iyer, P. and Roughgarden, J. (2008). Gametic conflict versus contact in the evolution of anisogamy. *Theoretical Population Biology*, **73**, 461–72.
Lessells, C. M., Snook, R. R., and Hosken, D. J. (2009). The evolutionary origin and maintenance of sperm: selection for a small, motile gamete mating type. In T. R. Birkhead, D. Hosken, and S. Pitnick (editors), *Sperm Biology: An Evolutionary Perspective*. London: Academic Press, pp. 43–67.
Levitan, D. R. (1993). The importance of sperm limitation to the evolution of egg size in marine invertebrates. *American Naturalist*, **141**, 517–536.
Levitan, D. R. (1996a). Predicting optimal and unique egg sizes in free-spawning marine invertebrates. *American Naturalist*, **148**, 174–188.
Levitan, D. R. (1996b). Effects of gamete traits on fertilization in the sea and the evolution of sexual dimorphism. *Nature*, **382**, 153–155.
Levitan, D. R. (1998). Sperm limitation, gamete competition, and sexual selection in external fertilizers. In T. R. Birkhead and A. P. Møller (editors), *Sperm Competition and Sexual Selection*. London: Academic Press, pp. 175–218.
Levitan, D. R. (2000). Optimal egg size in marine invertebrates: theory and phylogenetic analysis of the critical relationship between egg size and development time in Echinoids. *American Naturalist*, **156**, 175–192.
Levitan, D. R. (2006). The relationship between egg size and fertilization success in broadcast-spawning macine invertebrates. *Ingetratise and Comparative Biology*, **46**, 298–311.
Lewin, R. A. (editor) (1976). *The Genetics of Algae*. Oxford: Blackwell.
Lombardi, J. (1998). *Comparative Vertebrate Reproduction*. Boston, MA: Kluwer Academic Publishers.
Madsen, J. D. and Waller, D. M. (1983). A note on the evolution of gamete dimorphism in algae. *American Naturalist*, **121**, 443–447.
Maire, N., Ackermann, M., and Doebeli, M. (2002). Evolutionary branching and the evolution of anisogamy. *Selection*, **2**, 119–131.
Matsuda, H. and Abrams, P. A. (1999). Why are equally sized gametes so rare? The instability of isogamy and the cost of anisogamy. *Evolutionary Ecology Research*, **1**, 769–784.
Maynard Smith, J. (1978). *The Evolution of Sex*. Cambridge: Cambridge University Press.

Maynard Smith, J. (1982). *Evolution and the Theory of Games*. Cambridge: Cambridge University Press.
Maynard Smith, J. and Szathmáry, E. (1995). *The Major Transitions in Evolution*. San Francisco, CA: W. J. Freeman.
McNamara, J. M., Houston, A. I., Barta, Z., and Osorno, J.-L. (2003). Should young ever be better off with one parent than with two? *Behavioral Ecology*, **14**, 301–310.
Parker, G. A. (1970). Sperm competition and its evolutionary consequences in the insects. *Biological Reviews*, **45**, 525–567.
Parker, G. A. (1978). Selection on non-random fusion of gametes during the evolution of anisogamy. *Journal of Theoretical Biology*, **73**, 1–28.
Parker, G. A. (1979) Sexual selection and sexual conflict. In M. S. Blum and N. A. Blum (editors), *Sexual Selection and Reproductive Competition in Insects*. New York: Academic Press, pp. 123–166.
Parker, G. A. (1982). Why are there so many tiny sperm? Sperm competition and the maintenance of two sexes. *Journal of Theoretical Biology*, **96**, 281–294 and 98, 707.
Parker, G. A. (1984). The producer/scrounger model and its relevance to sexuality. In C. J. Barnard (editor), *Producers and Scroungers: Strategies of Exploitation and Parasitism*. London: Croom Helm, pp. 127–153.
Parker, G. A. (1985). Models of parent–offspring conflict. V. Effects of the behaviour of the two parents. *Animal Behaviour*, **33**, 519–533.
Parker, G. A. (1993). Sperm competition games: sperm size and sperm number under adult control. *Proceedings of the Royal Society of London B*, **253**, 245–254.
Parker, G. A. (1998). Sperm competition and the evolution of ejaculates: towards a theory base. In T. R. Birkhead and A. P. Møller (editors), *Sperm Competition and Sexual Selection*. London: Academic Press, pp. 3–54.
Parker, G. A. (2006). Sexual conflict over mating and fertilization – an overview. *Philosophical Transactions of the Royal Society of London B*, **361**, 235–259.
Parker, G. A. and Ball, M. A. (2005). Sperm competition, mating rate and the evolution of testis and ejaculate sizes: a population model. *Biology Letters*, **1**, 235–238.
Parker, G. A. and Begon, M. (1986). Optimal egg size and clutch size: effects of environment and maternal phenotype. *American Naturalist*, **128**, 573–592.
Parker, G. A. and Begon, M. E. (1993). Sperm competition games: sperm size and sperm number under gametic control. *Proceedings of the Royal Society of London B*, **253**, 255–262.
Parker, G. A. and Pizzari, T. (2010). Sperm competition and ejaculate economics. *Biological Reviews*, doi: 10.1111/j.1469-185x.2010.00140.x.
Parker, G. A. and Simmons, L. W. (1991). A model of constant random sperm displacement during mating: evidence from *Scatophaga*. *Biological Sciences*, **246**, 107–115.
Parker, G. A. and Stuart, R. A. (1976). Animal behaviour as a strategy optimizer: evolution of resource assessment strategies and optimal emigration thresholds. *American Naturalist*, **110**, 1055–1076.
Parker, G. A., Baker, R. R., and Smith, V. G. F. (1972). The origin and evolution of gamete dimorphism and the male–female phenomenon. *Journal of Theoretical Biology*, **36**, 529–553.
Parker, G. A., Ball, M. A., Stockley, P., and Gage, M. J. G. (1996). Sperm competition games: individual assessment of sperm competition intensity by group spawners. *Proceedings of the Royal Society of London B*, **263**, 1291–1297.

Parker, G. A., Ball, M. A., Stockley, P., and Gage, M. J. G. (1997). Sperm competition games: a prospective analysis of risk assessment. *Proceedings of the Royal Society of London B*, **264**, 1793–1802.
Parker, G. A., Immler, S., Pitnick, S., *et al.* (2010). Sperm competition games: sperm size and number under raffle and displacement, and the evolution of P_2. *Journal of Theoretical Biology*, **264**, 1003–1023.
Parker, G. A., Simmons, L. W. and Kirk, H. (1990). Analysing sperm competition data: simple models for predicting mechanisms. *Behavioral Ecology and Sociobiology*, **27**, 55–65.
Pitnick, S., Spicer, G. S., and Markow, T. A. (1995). How long is a giant sperm? *Nature*, **375**, 109.
Randerson, J. P. and Hurst, L. D. (1999). Small sperm, uniparental inheritance and selfish cytoplasmic elements: a comparison of two models. *Journal of Evolutionary Biology*, **12**, 1110–1124.
Randerson, J. P. and Hurst, L. D. (2001a). The uncertain evolution of the sexes. *Trends in Ecology and Evolution*, **16**, 571–579.
Randerson, J. P. and Hurst, L. D. (2001b). A comparative test of a theory for the evolution of anisogamy. *Proceedings of the Royal Society of London B*, **268**, 879–884.
Randerson, J. P. and Hurst, L. D. (2002), Survival and anisogamy: response. *Trends in Ecology and Evolution*, **17**, 358.
Roughgarden, J., Oishi, M., and Akcay, E. (2006). Reproductive social behavior: cooperative games to replace sexual selection. *Science*, **311**, 965–969.
Sandefur, J. T. (1990). *Discrete Dynamical Systems: Theory and Applications*. Oxford: Clarendon Press.
Scudo, F. M. (1967). The adaptive value of sexual dimorphism: I. Anisogamy. *Evolution*, **21**, 285–291.
Shapiro, D. and Giraldeau, L. A. (1996). Mating tactics in external fertilizers when sperm is limited. *Behavioral Ecology*, **7**, 19–23.
Simmons, L. W. (2001). *Sperm Competition and its Evolutionary Consequences in the Insects*. Princeton, NJ: Princeton University Press.
Smith, C. C. and Fretwell, S. D. (1974). The optimal balance between size and number of offspring. *American Naturalist*, **108**, 499–506.
Smith, R. L. (1979). Repeated copulation and sperm precedence: paternity assurance for a male brooding water bug. *Science*, **205**, 1029–1031.
Smith, R. L. (editor) (1984). *Sperm Competition and the Evolution of Animal Mating Systems*. London: Academic Press.
Snook, R. R. (2005). Sperm in competition: not playing by the numbers. *Trends in Ecology and Evolution*, **20**, 46–53.
Stockley, P., Gage, M. J. G., Parker, G. A., and Møller, A. P. (1997). Sperm competition in fishes: the evolution of testis size and ejaculate characteristics. *American Naturalist*, **149**, 933–954.
Togashi, T. and Cox, P. A. (2004). Phototaxis and the evolution of isogamy and "slight anisogamy" in marine green algae: insights from laboratory observations and numerical experiments. *Botanical Journal of the Linnean Society*, **144**, 321–327.
Togashi, T., Cox, P. A., and Bartelt, J. (2007). Underwater fertilization dynamics of marine green algae. *Mathematical Biosciences*, **208**, 205–221.
Togashi, T., Motomura, T., and Ichimura, T. (1998). Gamete dimorphism in *Bryopsis plumosa*. Phototaxis, gamete motility and pheromonal attraction. *Botanica Marina*, **41**, 257–264.

Togashi, T., Motomura, T., Ichimura, T., and Cox, P.A. (1999). Gametic behavior in a marine green alga, *Monostroma angicava*: an effect of phototaxis on mating efficiency. *Sexual Plant Reproduction*, **12**, 158–63.

Togashi, T., Nagisa, M., Miyazaki, T., *et al.* (2008). Effects of gamete behavior and density on fertilization success in marine green algae: insights from three-dimensional numerical simulations. *Aquatic Ecology*, **42**, 355–362.

Trivers, R.L. (1972). Parental investment and sexual selection. In B. Campbell (editor), *Sexual Selection and the Descent of Man 1871–1971*. Chicago, IL: Aldine-Atherton, pp. 136–172.

Van Look, K.J., Dzyuba, B., Cliffe, A., Koldewey, H.J., and Holt, W.V. (2007). Dimorphic sperm and the unlikely route to fertilisation in the yellow seahorse. *Journal of Experimental Biology*, **210**, 432–437.

Vance, R.R. (1973). On reproductive strategies in marine bottom invertebrates. *American Naturalist*, **107**, 339–352.

Wiese, L. (1976). Genetic aspects of sexuality in Volvocales. In R.A. Lewin (editor), *The Genetics of Algae*. Oxford: Blackwell Scientific, pp. 174–197.

Wiese, L. (1981). On the evolution of anisogamy from isogamous monoecy and on the origin of sex. *Journal of Theoretical Biology*, **89**, 573–580.

Wiese, L., Wiese, W., and Edwards, D.A. (1979). Inducible anisogamy and the evolution of oogamy from isogamy. *Annals of Botany*, **44**, 131–139.

Yund, P.O. (2000). How severe is sperm limitation in natural populations of marine freespawners? *Trends in Ecology and Evolution*, **15**, 10–13.

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2

The evolutionary instability of isogamy

2.1 INTRODUCTION

Multicellular organisms are usually anisogamous with different mean sizes of gametes of different sexes or mating types. It is generally believed that isogamy (equal gamete sizes of different mating types) is the most primitive mating system (e.g. Maynard Smith, 1978; 1982; Hoekstra, 1987; Maynard Smith and Szathmáry, 1995). Although we observe many isogamous species in algae, fungi, and protozoans (for review see Bell, 1982; Hoekstra, 1987), anisogamy has a much wider taxonomic distribution. Attempts to use evolutionary theory to understand this restricted distribution have met with limited success, as have attempts to understand the distribution of isogamy within groups that include both isogamous and anisogamous species, as observed in the freshwater green algal order Volvocales (Randerson and Hurst, 2001). However, there are numerous exceptions to fully accept the synthetic theory (Bell, 1978; Hoekstra, 1987). We use a series of mathematical models to help understand what might account for the comparative scarcity of isogamy. The models also produce some new hypotheses that may help explain some of the anomalies in the comparative data.

It is generally believed that the evolution of mating types preceded the evolution of gamete size dimorphism (Hoekstra, 1987; Maynard Smith and Szathmáry, 1995). For example, Hoekstra (1982, 1987) has proposed several possible mechanisms for the evolution of mating types in a randomly mating population. In the simplest mechanism, it is assumed that gamete adhesion was initially brought about

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