THE COST OF BIPARENTAL SEX UNDER INDIVIDUAL SELECTION

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Submitted June 2, 1988; Revised November 16, 1988, and April 24, 1989; Accepted June 12, 1989

In the 1970s, Williams (1971, 1975) and Maynard Smith (1971, 1978) ignited the interest of population biologists in the question of why there is sex, especially in species that could reproduce without it. They were united in the view that sex should have an explanation at the individual level, but they differed in their portrayals of the disadvantage (or "cost") that sex entails. Maynard Smith (1971, 1978) viewed the cost of sex as being one of producing males in dioecious species and, similarly, as one of allocation of limited resources to male function in hermaphrodites. The idea here is that the advantage to asexual reproduction stems from eliminating males (or male gametes), thereby resulting in a more efficient production of offspring. Williams (1971, 1975), by contrast, viewed the cost of sex as being one of reducing the genetic contribution to offspring (his "cost of meiosis") regardless of whether the sexes are separate or combined (see also Maynard Smith 1974), and he reasoned that the genetic cost of meiosis would be reduced by biparental inbreeding (Williams 1979, 1980).

Recent analytic models have shown that these two costs of sex are not interchangeable (although commonly treated as such) and that they apply to different situations (B. Charlesworth 1980; Lloyd 1980*a*; Uyenoyama 1984). B. Charlesworth (1980), de Jong (1980), and Lloyd (1980*a*) showed that the cost of sex in an obligately sexual dioecious population depends only on the sex ratio. Their models also demonstrated that the advantage of an obligately asexual clone increases as an increasing function of the proportion of males in the sexual population, becoming twofold when this proportion is a half (as first shown in Maynard Smith 1971). This result supports Maynard Smith's cost of producing males, and it is consistent with the conclusion reached by Treisman and Dawkins (1976).

It is important to note, however, that selection for an obligately asexual clone operates at the group level (at least once the clone becomes established), because there is no gene flow between the sexual and asexual subpopulations (Uyenoyama 1984; Williams 1988). Uyenoyama (1984, 1985) analyzed an individual-selection model for a dioecious species by calculating the conditions for increase in the

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Am. Nat. 1990. Vol. 135, pp. 489–500.

fraction of parthenogenetic offspring produced by individual females, and she showed that under the assumption of panmixia, the cost of sex depends only on male frequency. This is consistent with the above result for competition between sexual and asexual subpopulations, and it indicates that the cost of producing males applies to both group and individual selection in dioecious species (contrary to Williams 1988). However, although biparental inbreeding does not affect the cost of sex under group selection (B. Charlesworth 1980; Lloyd 1980*a*), it can affect the conditions for the spread of alleles that increase the fraction of apomictic offspring in partially parthenogenetic females (Uyenoyama 1984, 1985).

Selection for asexual reproduction in hermaphrodites is conceptually similar to that of partial parthenogenesis in dioecious females in that it also requires an analysis of selection at the individual level, but it is further complicated by the potential for self-fertilization. B. Charlesworth (1980) and Lloyd (1980*a*) showed that selfing makes invasion by mutations increasing apomixis more difficult (see also Bell 1982; Uyenoyama 1986). This result may be seen intuitively by recognizing that selfing is a form of uniparental reproduction, and it makes clear the value of keeping separate the cost of sex, which includes mixis within and between parents, and the cost of biparental reproduction per se. In addition, both authors argued that the cost of biparental sex in hermaphrodites is due to gene sharing (see also Marshall and Brown 1981; Uyenoyama 1984) and that it is independent of the allocation to male gametes (but see Harper 1982).

This result for hermaphrodites is directly the opposite of that obtained for dioecious populations, suggesting that the cost of biparental sex in dioecious species is due to the cost of producing males, whereas the same cost in hermaphrodites is due to the cost of meiosis. Unfortunately, this simple generalization is rejected by recent theoretical work on selection for cross-fertilization in cleistogamous plants (such plants are hermaphroditic species in which individuals produce both open, potentially outcrossed flowers [chasmogamous] and closed, obligately selfed flowers [cleistogamous]). The cost of biparental sex in cleistogamous plants is due to male allocation (Schoen and Lloyd 1984; Lloyd 1988).

Two related questions arise from this compendium of results. Why should the cost of biparental sex in hermaphroditic plants depend on whether they have cleistogamous flowers, and why should the same cost for cleistogamous hermaphrodites be more similar to dioecious species than it is to non-cleistogamous hermaphrodites? Another question that has not been addressed is also of interest: what is the cost of cross-fertilization in hermaphrodites that have the capacity for somatic uniparental reproduction? The present study addresses these questions; it also suggests general conditions under which male allocation and gene sharing enter into the cost of biparental sexuality.

THE MODELS

In this paper, *parthenogenetic* refers to uniparental reproduction through ova; this includes both apomixis and the various forms of automixis (see Bell 1982). Cross-fertilization, biparental sex, and amphimixis are used interchangeably to mean sexual reproduction involving two parents, which may or may not be related.

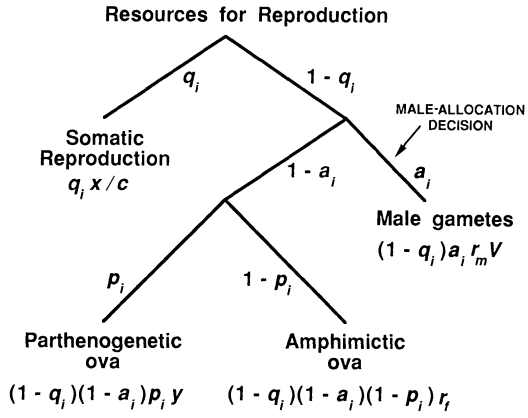

FIG. 1.—Flowchart for the resource-allocation decisions in a cosexual individual. Note that the allocation to somatic reproduction precedes the male-allocation decision and that the allocation to parthenogenetic ova follows the male-allocation decision. Variables are defined in the text in the subsection "*Cosexes*."

Sex refers to mixis and syngamy and may be either uniparental or biparental. *Male allocation* is used to mean both the investment of resources by dioecious females in sons and the investment of resources in male gametes by cosexual individuals. Finally, *cosex* is used (in the sense of Lloyd 1980*b*) to mean an individual that produces both male and female gametes (hermaphroditic, monoecious, etc.) and which, on the average, has equal expected gains in fitness through male and female functions. The present models are concerned primarily with cosexes and do not address the effects of variation in functional gender on the cost of cross-fertilization (as do the models of Hoekstra and van Loo 1986). The models also assume random mating in a large population.

Cosexes

Consider a cosexual species in which reproduction is limited by resources. Let q_i be the proportion of resources allocated by the ith individual to vegetative reproduction or asexual budding (somatic reproduction in general). Of the remaining resource base $(1 - q_i)$, let a_i be the fraction of resources allocated to male gametes, and let $(1 - a_i)$ be the fraction of resources allocated to potentially amphimictic ova. Finally, let p_i be the fraction of these latter ova that are parthenogenetic, and let $(1 - p_i)$ be the fraction that are amphimictic; a flowchart of these resource-allocation decisions is given in figure 1.

The expected fitness of the ith individual gained through somatic reproduction is $q_i x/c$, where x is the expected survival rate of vegetatively produced progeny relative to sexually produced progeny, and c is the relative cost to the parent of a vegetatively produced individual. Similarly, the expected gain through resources allocated to parthenogenetic ova is $(1 - q_i)(1 - a_i)p_i y$, and the expected gain through resources allocated to amphimictic ova is $(1 - q_i)(1 - a_i)(1 - p_i)r_f$ where y is the fitness of offspring derived from parthenogenetic ova relative to that of offspring derived from amphimictic ova, and r_f is the fraction of the genome

transmitted to amphimictic ova during meiosis ("genome dilution" in the sense of Lewis 1987). Finally, assuming a large population, the expected gain in fitness through male gametes is closely approximated by $(1 - q_i)a_i r_m V$; r_m is the fraction of the genome transmitted to male gametes, and V, the reproductive value of male function, equals $(1 - a^*)(1 - p^*)/a^*$, where p^* and a^* represent the population means for the allocation to parthenogenetic ova and male function, respectively. The total expected genetic fitness of the ith individual, W_i, is the sum of these four different ways of reproducing, which reduces to

$$W_i = (q_i x/c) + (1 - q_i)(1 - a_i)[p_i y + (1 - p_i)r_f] + (1 - q_i)a_i r_m V. \quad (1)$$

This derivation of W_i assumes that any sperm used in the production of uniparental zygotes through self-fertilization does not affect the total number of outcrossed progeny gained through male function. This is a reasonable assumption for prior self-fertilization and extreme anisogamy (the cost of amphimixis as a function of the degree of anisogamy is derived in the Appendix). The model also assumes that all amphimictic ova are fertilized, keeping in mind that this is unlikely to be true when the average allocation to male function in the population is low or when pollinators are scarce.

The fecundity of the ith individual, N_i, can be calculated from equation (1) as

$$N_i = (q_i x/c) + (1 - q_i)(1 - a_i)[p_i y + (1 - p_i)]. \quad (2)$$

This equation is used to contrast the genetic effects of selection with the effects on fecundity.

Mutations switching amphimictic ova to parthenogenetic ova.—Consider a dominant mutation that decreases the fraction of amphimictic ova by increasing the fraction of ova that develop parthenogenetically (i.e., an increase in p_i). The mutation is expected to increase when its effect on individual fitness is positive, that is, when

$$\partial W_i/\partial p_i = (1 - q_i)(1 - a_i)(y - r_f) > 0, \quad (3)$$

which (for all a_i and $q_i < 1$) is when

$$y > r_f. \quad (4)$$

Hence, the cost of biparental sex is calculated in terms of the relative fitness of parthenogenetic offspring (y) required for the spread of mutations increasing the investment in such offspring (see also Bulmer 1982; Uyenoyama 1984); cross-fertilization is considered costly when this value is less than one.

Note that genome dilution through male function (r_m) has no effect on the cost of biparental sex. Note, too, that the mutant's allocation to male gametes (a_i) also has no effect and that the same is true for the mean allocation to male gametes in the population (a^*) (see also B. Charlesworth 1980; Lloyd 1980*a*). The cost of biparental sex in this case is due to genome dilution through amphimictic ova (r_f). This result supports Williams' (1975) basic view that the cost of crossing is due to gene sharing, with the refinement that the cost is paid only through female function; in the present study, the cost of meiosis is used to mean $1 - r_f$.

The above result shows that a mutation increasing the fraction of partheno-

genetic ova will increase for $y > \frac{1}{2}$. Fecundity also increases when

$$\partial N_i/\partial p_i = (1 - q_i)(1 - a_i)(y - 1) > 0, \tag{5}$$

that is, when $y > 1$. Conversely, fecundity decreases when $y < 1$. Hence, for $\frac{1}{2} < y < 1$, the mutation for increased parthenogenesis will spread even though fewer offspring are produced.

Mutations for increased somatic reproduction.—Consider a mutation that increases the allocation to vegetative progeny or asexual budding (q_i). The mutation is expected to spread in a large population when

$$\begin{aligned}\partial W_i/\partial q_i = (x/c) - (1 - a_i)p_i y - (1 - a_i)(1 - p_i)r_f \\ - [a_i r_m(1 - p^*)(1 - a^*)/a^*] > 0.\end{aligned} \tag{6}$$

Assuming that the mutant's allocations to male gametes and parthenogenetic ova are near the population means (i.e., $a^* = a_i$, and $p^* = p_i$), the condition for the spread of the mutation becomes

$$x > c(1 - a_i)[p_i y + (1 - p_i)(r_f + r_m)]. \tag{7a}$$

If it is also assumed that there is no recovery of the cost of crossing through parthenogenetic ova (i.e., $p_i = 0$), this condition reduces to

$$x > c(1 - a_i)(r_f + r_m). \tag{7b}$$

Note that in inequality (7b) the cost of meiosis is recovered through male function ($r_f + r_m = 1$), and the condition for spread of the mutant simplifies to

$$x > c(1 - a_i). \tag{7c}$$

Subsequent mutations of the same kind also increase until $q_i = q^* = 1$.

Note that, in contrast to the preceding case, the cost of crossing is due to the allocation of resources to male function, and it is independent of gene sharing (inequality 7c). There is also an additional cost of sex, which is due to any additional direct and indirect costs (c) associated with the production of cross-fertilized progeny. Hence, factors that decrease either male allocation (e.g., restricted access to mates; see Charnov 1980, 1982; Lloyd 1984) or investment in indirect costs (e.g., pollinator rewards; see Lloyd 1987) have the added effect of decreasing the cost of crossing. Inequality (7c) was also derived in considerations of selection for cleistogamous flower production (Schoen and Lloyd 1984; Lloyd 1988). Hence, the cost of crossing in the face of mutations that increase somatic reproduction is the same as for mutations that increase the production of cleistogamous flowers.

Finally, selection for increased somatic reproduction increases fecundity when

$$\partial N_i/\partial q_i = (x/c) - (1 - a_i)[p_i y + (1 - p_i)] > 0. \tag{8}$$

In the absence of parthenogenetic ova (i.e., $p_i = 0$), this condition becomes $x > c(1 - a_i)$, which is the same as the condition for the spread of a mutation increasing somatic reproduction. Hence, fecundity is increased by selection that increases somatic reproduction.

Male allocation.—As just demonstrated (inequality 7c), male allocation affects the conditions for the spread of mutants increasing somatic reproduction (and cleistogamous flower production). It is of interest, therefore, to ascertain the reverse effect of somatic reproduction on selection for male allocation to determine whether there could be a feedback effect of each upon the other. A mutation that alters male allocation in a single individual (a_i) cannot increase in a large population (i.e., a phenotype at a^* is an evolutionarily stable strategy [ESS] in the sense of Maynard Smith 1982) when

$$\begin{aligned} \partial W_i/\partial a_i = & -p_i(1-q_i)y - r_f(1-p_i)(1-q_i) \\ & + [r_m(1-q_i)(1-a^*)(1-p^*)/a^*] = 0. \end{aligned} \tag{9}$$

Assuming that the mutation does not affect the proportion of parthenogenetic ova (i.e., $p_i = p^*$), the ESS is reached when

$$(1-a^*)/a^* = [yp^*/r_m(1-p^*)] + r_f/r_m. \tag{10}$$

The absence of q_i and q^* from equation (10) suggests that the evolutionarily stable allocation to male gametes is not affected by somatic reproduction and that there is no feedback of the type just postulated. The proportion of parthenogenetic (p_i) ova, however, does affect the ESS (see also D. Charlesworth and Charlesworth 1981; Lloyd 1987). Note that in the absence of parthenogenetic ova (i.e., $p_i = p^* = 0$), the well-known result of selection for equal investment in male and female functions is reached for $r_f = r_m$ (review in Charnov 1982).

Partial Parthenogenesis in Dioecious Females

Mutations switching amphimictic ova to parthenogenetic ova.—It is of value to consider briefly a conceptually similar situation in dioecious species: partial parthenogenesis in the broods of dioecious diploid females, where females are the homogametic sex. This situation is similar in the sense that it requires an evaluation of selection at the individual level. The fitness of an individual in a large population is

$$W_i = p_i y + (1-a_i)r_f(1-p_i) + a_i r_m(1-p_i)V, \tag{11}$$

where V equals $(1-a^*)/a^*$ and gives the reproductive value (in the sense of Fisher 1958) of males as a function of the ratio of sexual females to males in the population (following Uyenoyama 1984). Note that a is used here to represent the allocation to male offspring, rather than male function; all other variables are as defined for cosexes, above. A flowchart of the allocation decisions is given in figure 2. As previously, let there be a mutation that increases the allocation to parthenogenetic eggs (p_i) in a single individual. The mutation tends to increase when it increases individual fitness, that is, when

$$\partial W_i/\partial p_i = y - (1-a_i)r_f + [a_i r_m(1-a^*)/a^*] > 0. \tag{12}$$

Assuming as before that the mutant's male allocation is near the population mean ($a_i = a^*$), the inequality is satisfied when

$$y > (1-a_i)(r_f + r_m), \tag{13a}$$

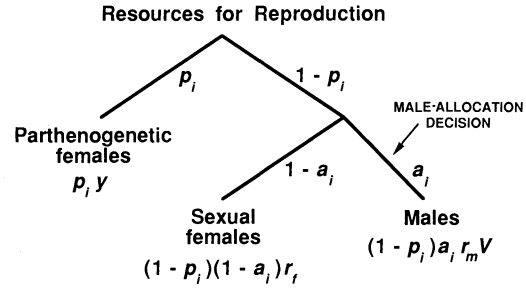

FIG. 2.—Flowchart for the resource decisions under partial parthenogenesis in a dioecious population. Variables are defined in the subsection "*Partial Parthenogenesis in Dioecious Females.*"

which, because $r_f + r_m = 1$, reduces to

$$y > (1 - a_i). \tag{13b}$$

Note that, as in the case for mutations that increase somatic reproduction in cosexes, in which the cost of meiosis is recovered through male function (inequality 7b), the cost of meiosis in partially parthenogenetic dioecious females $(1 - r_f)$ is recovered through the expected reproductive contribution of sons (r_m; see inequality 13a). Hence, under panmixia, the cost of biparental sex in partially parthenogenetic females is due to the allocation to sons (see also Uyenoyama 1984). Curiously, the cost of crossing in dioecious females is more like that for somatic reproduction in cosexes than is the more directly analogous situation of parthenogenesis in previously amphimictic ova (in which the cost of crossing is due to gene sharing).

Male allocation.—The effect of selection increasing the allocation to parthenogenetic daughters on selection for the optimum ratio of males to sexual females is derived as for cosexes by considering a mutation that alters the investment in sons (a_i), without affecting the investment in parthenogenetic daughters $(p_i = p^*)$. The mutation cannot increase in the population (i.e., a phenotype at a^* is an ESS) when

$$\partial W_i/\partial a_i = -r_f(1 - p_i) + [r_m(1 - p_i)(1 - a^*)/a^*] = 0, \tag{14}$$

that is, when

$$(1 - a^*)/a^* = r_f/r_m. \tag{15}$$

Hence, the allocation to parthenogenetic daughters p_i has no effect on selection for male allocation, and (for $r_f = r_m$) there is selection for equal investment in males and sexual females (Fisher 1958).

DISCUSSION

Resolution of the evolutionary paradox concerning the maintenance of biparental sex (Williams 1975; Maynard Smith 1978; Bell 1982; Shields 1982; Stearns 1987; Michod and Levin 1988) requires a thorough understanding of its costs and

the situations under which they apply. The purpose of the present study was to compare the two major explanations for the cost of biparental reproduction that stem directly from the evolution of anisogamy: the cost of male allocation (Maynard Smith 1971, 1978) and the cost of meiosis (Williams 1971, 1975). The results show that both are valid concepts, which apply to the advantages gained by increasing the allocation of resources to different kinds of uniparental offspring (see also Lloyd 1980*a*; Uyenoyama 1984). They also suggest that the two costs are probably mutually exclusive for any particular situation.

The cost of meiosis was found to be the sole cost of biparental sex for cosexual species, given mutations that increase the proportion of non-cleistogamous ova that develop parthenogenetically. (This result was first formally derived by B. Charlesworth [1980] and Lloyd [1980*a*].) The present model for this situation also shows that the cost of gene sharing is paid only through female function (see inequality 4). We have used the cost of meiosis in the present study to mean the reduction in the genome that occurs during the production of amphimictic eggs, usually to one-half. (We have not included a term for biparental inbreeding, but we feel that it would not have an effect under the assumptions of the present model—unless there was inbreeding or outbreeding depression—because r_f plus r_m cannot be greater than one. Assigning values of greater than 0.5 to these variables would amount to a practice of counting the contribution of alleles through kin matings more than once [Lloyd 1989].)

The cost of male allocation, by contrast, was found to be the cost of biparental sex in cosexes, given mutations that increase vegetative reproduction and asexual budding. This is consistent with Maynard Smith's (1971, 1978) portrayal of the cost of cross-fertilization in hermaphrodites as being due to the reduction in fecundity that results from allocating limited resources to male gametes. There is also an additional cost of crossing in this situation, which is due to any direct and indirect costs (c) associated with the production of cross-fertilized progeny (e.g., male genitalia in animals, nectar and petals in plants; see Lloyd 1987).

A cost of male allocation has also been observed in models on the selective advantage of cleistogamous (closed, autogamous) flowers in hermaphroditic plants (Schoen and Lloyd 1984; Lloyd 1988). This result seems, at first, to be inconsistent with the present study: why should autogamy in closed flowers be more similar to vegetative reproduction (with respect to the cost of crossing) than it is to autogamy in open flowers? It seems reasonable to suggest that the answer to this question lies in the order in which the allocation decisions are made. The decision to allocate q_i resources to cleistogamous flowers and to vegetative reproduction both preempt resources that would otherwise be allocated to gametes for potential cross-fertilization ($1 - q_i$). In an important sense, then, the allocation of resources to the production of uniparental progeny through both somatic means and cleistogamous flowers precedes the decision to allocate resources to male gametes (the male-allocation decision, a_i) and to non-cleistogamous ova ($1 - a_i$), as is shown in figure 1. This contrasts with the decision to make p_i uniparental progeny through non-cleistogamous parthenogenetic ova. In this decision, which follows the male-allocation decision, the cost of crossing is

due to gene sharing. Hence, we suggest that, in general, the cost of crossing is due to male allocation when mutations that increase the allocation to uniparental progeny precede the male-allocation decision, and that the cost of crossing is due to gene sharing in the face of similar mutations that follow the male-allocation decision.

The biological reasons for this dichotomy may be seen as follows. Mutations that occur after the male-allocation decision and that switch amphimictic ova to parthenogenetic ova are favored (for $y > r_f$), because they increase the genetic contribution to the next generation; there is, however, no increase in the number of offspring produced by the mutant (unless $y > 1$). Mutations that occur before the male-allocation decision and that shunt resources into vegetative reproduction, asexual budding, or cleistogamous flower production are favored (for $x > c[1 - a_i]$) because they increase the number of offspring produced. Although it is true that such offspring are more related to the mutant than are those produced through amphimictic ova, this reduction in relatedness is recovered through cross-fertilized male gametes (inequality 7b). The advantage of somatic reproduction stems from recovering part of the allocation to male gametes, which is largely "wasted" (in the sense that most male gametes do not contribute directly to the production of progeny; see Maynard Smith 1971). This wastage is set up by overriding selection to invest equally in male and female allocations (review in Charnov 1982), independent of the investment in vegetative reproduction, asexual budding, or cleistogamous flower production (eq. 10).

Finally, for dioecious females capable of partial parthenogenesis, the cost of biparental sex is due to the cost of producing males (see also Uyenoyama 1984). This result seems counterintuitive at first, because partial parthenogenesis in dioecious females is more directly analogous to partial parthenogenesis in cosexes (in which the cost of crossing is due to gene sharing) than it is to vegetative reproduction and cleistogamous flower production in cosexes (in which the cost of crossing is also due to male allocation). However, like vegetative reproduction and cleistogamous flower production in cosexes, the allocation by dioecious mothers to parthenogenetic ova preempts resources from use in the production of outcrossed gametes (cf. figs. 1 and 2). Hence, it is the order of allocation decisions, and not the type of uniparental offspring, that is important in determining the cost of biparental sex.

In conclusion, whether the cost of biparental sex is due to gene sharing or to male allocation depends on the phenotypic effect of mutations increasing uniparental reproduction. Mutations that increase the allocation to uniparental progeny by preempting resources to potentially outcrossed gametes have an advantage that is proportional to male allocation. Such phenotypic effects include increased vegetative reproduction, asexual budding, and cleistogamous flower production in cosexual individuals, and partial parthenogenesis in dioecious individuals. By contrast, mutations that increase the allocation to uniparental progeny without affecting the allocation to pollen or sperm in cosexual individuals have an advantage that depends only on the reduction in relatedness between the maternal parent and her offspring.

SUMMARY

Models of evolutionarily stable strategies are presented, which were designed to determine whether the disadvantage of anisogamous biparental sex is due to the cost of male allocation or to the cost of meiosis. The results show that (1) the cost of biparental sex is due to gene sharing given mutations increasing the proportion of non-cleistogamous parthenogenetic ova in cosexual individuals and (2) the cost of biparental sex is due to male allocation given mutations increasing somatic reproduction in cosexual individuals and mutations increasing partial pathenogenesis in dioecious females. It is suggested that, in general, the cost of biparental sex is due to male allocation when mutations that increase uniparental reproduction affect events before the male-allocation decision, and that the cost of biparental sex is due to gene sharing when mutations that increase uniparental reproduction affect events that come after the male-allocation decision.

ACKNOWLEDGMENTS

We thank L. Delph, M. McKone, T. Meagher, P. Raimondi, C. Thomas, M. Uyenoyama, R. Vrijenhoek, S. Weeks, and an anonymous reviewer for helpful comments on the manuscript. This study was supported by National Science Foundation grant INT-8702961 (to C.M.L.).

APPENDIX

In obligately sexual, dioecious populations, the degree of asymmetry between gametes has no effect on the cost of crossing; all that matters is that males produce no offspring directly. In cosexes, however, the situation is different; for example, there is no cost of crossing when the gametes are of equal size or are isogamous (Manning 1975; Maynard Smith 1978; B. Charlesworth 1980; Uyenoyama 1984). The question that arises is how the cost of crossing in cosexes is related to the degree of anisogamy. We seem to know the end points (no cost of crossing under isogamy, and a twofold cost of crossing under extreme anisogamy) but not what happens in the middle. This is an important question, especially if we consider a possible evolutionary race between the evolution of sex and the evolution of anisogamy.

Stated more formally, we wish to know the shape of the curve that relates the minimum advantage required by amphimictic progeny that is required to maintain outcrossing as a function of the size of amphimictic eggs relative to the size of the male gametes. Consider a cosexual individual that makes three types of gametes: apomictic eggs at cost C_1, amphimictic eggs at cost C_2, and male gametes at cost C_3. Assuming that the male-allocation decision comes after the decision to divide resources between apomictic eggs and sexual gametes, the fitness of the ith individual in a large population is

$$W_i = \left[\frac{p_i y}{C_1} + \frac{(1 - a_i)r_f(1 - p_i)}{C_2} + \frac{a_i(1 - p_i)}{C_3}\right]\frac{r_m(1 - a^*)/C_2}{a^*/C_3}.$$

Cross-fertilization is evolutionarily stable when

$$\partial W_i/\partial p_i = (y/C_1) - [(1 - a_i)r_f/C_2] - [a_i r_m(1 - a^*)/a^* C_2] < 0,$$

which for $a_i = a^*$ occurs when

$$y < C_1(1 - a_i)(r_f + r_m)/C_2.$$

Note that male allocation (a_i) affects the conditions for the spread of the mutant and that the cost of meiosis is recovered through male function. Assuming that the size of a zygote following syngamy of an amphimictic egg equals the size of an apomictic egg ($C_1 = C_2 + C_3$), the condition under which biparental sex is evolutionarily stable becomes

$$y < (C_2 + C_3)(1 - a_i)(r_f + r_m)/C_2 .$$

Hence, the minimum advantage required by amphimictic progeny to maintain outcrossing is a linear function of the size of amphimictic eggs and is proportional to $(C_2 + C_3)(1 - a_i)/C_2$.

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