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MALE ALLOCATION AND THE COST OF BIPARENTAL SEX IN A PARASITIC WORM

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Abstract. Digenetic trematodes have a complex life cycle that involves at least two hosts: a molluscan host, where the worms reproduce asexually, and a vertebrate host, where they reproduce sexually. In some species of the family Microphallidae, all (N = 500-1,000) of the asexual progeny produced in the molluscan host are consumed together by the vertebrate host, where they hatch and mature. This gives an opportunity for cross fertilization among genetically identical members of the same clone. In the present study, strategy models were used to determine the evolutionarily stable allocation to male function and the conditions for the spread of parthenogenetic egg production in these hermaphroditic worms. The results show that the ESS for male allocation increases asymptotically from near zero to 1/2 as the number of clonal families in the local mating population increases from one to infinity, which is directly parallel to Hamilton's (1967) result for local mate competition. The results also show that the minimum relative fitness of parthenogenetic offspring required for the spread of parthenogenetic egg production decreases asymptotically from one to 1/2 as the number of clonal families in the local mating population increases from 1 to infinity. Taken together, these results suggest that, where the levels of infection are low, the cost of cross-fertilization as well as the optimal allocation to male function in these worms should also be low.

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1. INTRODUCTION

The Red Queen hypothesis for the maintenance of biparental sex relies on the assumption that parasites will disproportionately infect the most common host phenotypes. This requires that the parasites are also sexual; if they become asexual, they should lose as a consequence the ability to track host phenotypes as they become common. It is therefore important to evaluate the cost of cross-fertilization in parasites as well as in hosts. In the present study, I present strategy models that were designed to evaluate the cost of biparental sex in a parasitic trematode (*Microphallus* sp), which has been implicated as a possible factor favoring sexual reproduction in its snail host, *Potamopyrgus antipodarum* (Lively, 1987).

Microphallus sp is a presently undescribed species of trematode that lives its adult life in the intestines of water fowl and wading birds in New Zealand; preliminary data suggest that adults probably live less than one week. The eggs produced by these simultaneously hermaphroditic worms are passed with the bird's feces and hatch following ingestion by *Potamopyrgus*. The resulting larva then attacks the snail, and successful infections result in the production of several hundred genetically identical cysts (Winterbourn, 1974). When the snail is ingested by the avian host, the cysts "hatch" and the juvenile worms migrate to the intestine, where they mature and reproduce sexually. This life cycle (summarized in Fig. 1) gives the opportunity for cross fertilization among members of the same clonal family, which is likely to affect the cost of biparental reproduction in the worm.

The "cost of sex" has proven to be a slippery concept. It refers to the disadvantage of biparental reproduction when uniparental reproduction is an option (Maynard Smith, 1971, 1978; Williams, 1971, 1975). Maynard Smith (1971, 1978) suggested that the investment of limited resources by dioecious females in their sons (and, similarly, the investment of such resources by hermaphrodites in male function) results in a reduction in fecundity, and that the cost of sex is proportional to this reduction (sometimes called the "ecological" cost of sex). Any factor that reduces the investment in males (or male function) would be expected to have the indirect effect of reducing the cost of sex. Williams (1971, 1975), in contrast, suggested that the cost of sex is due to the

reduction in relatedness between parents and their sexually derived offspring (the "genetic" cost of sex), and further that this cost is directly reduced by inbreeding (Williams, 1980; see also Uyenoyama, 1984, 1985, 1986). In a recent study of individual selection in panmictic populations, Lively and Lloyd (1990) showed that both costs are valid and that they apply to different situations. The present study relaxes the panmixia assumption and suggests that inbreeding: 1) directly reduces the genetic cost of sex (as suggested by Williams), and 2) indirectly reduces the ecological cost of sex when male allocation is unconstrained. The results also suggest that there may be an asymmetry in the host-parasite interaction, with a lower cost of sex for the parasite.

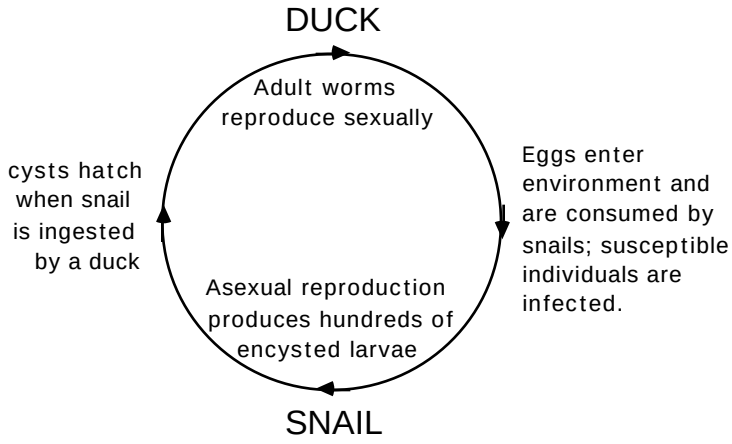

Figure 1. The life cycle of *Microphallus* sp. The present study is concerned with the number of infected snails ingested by the duck.

2. MALE ALLOCATION

In what follows, let a_i be the allocation to male function by individuals of the *i*th clonal family, and let $(1 - a_i)$ be the allocation to female function by the same individuals. The allocations to male and female functions by the members of the remaining families is a^* and $(1 - a^*)$, respectively.

Let F be the number of clonal families that coexist at the time of sexual reproduction in the avian host (which is the number of infected snails ingested by the bird, assuming that each snail was infected by a single egg). Assuming that the number of individuals, N, within a clonal family is constant and large

(i.e. N is approximately equal to N - 1), the probability of mating with a member of the same clone is 1/F, and the probability of mating with a member of a different clone is (1 - 1/F). Let d be a value between 0 and 1 that represents the relative fitness of inbred offspring resulting from matings between members of the same clonal family.

Assuming that all eggs are fertilized, the fitness of an individual in the ith family through female function, W_{if}, is the allocation to female function (1 - a_i) times the sum of the proportion of eggs fertilized by clone members (weighted by the inbreeding depression term, d), plus the proportion fertilized by non-clone members, which is

$$W_{if} = \frac{(1 - a_i)(d + F - 1)}{F}$$

The fitness of the same individual through male function, W_{im}, is the reproductive value of male function, V, times the proportion of eggs fertilized that were produced by other clone members (weighted by inbreeding depression), plus the proportion of eggs fertilized that were produced by non-clone members, which is:

$$W_{im} = \frac{V[(1 - a_i)d + (1 - a^*)(F - 1)]}{F}$$

The reproductive value of male function in this equation (V) is calculated as

$$V = \frac{a_i F}{(F - 1)a^* + a_i}$$

Total individual fitness is the sum of the gains through female function plus male function,

$$W_i = W_{if} + W_{im},$$

and the total fitness of the clonal family containing N individuals is NW_i.

We wish to know the value of a^* such that mutations causing slight deviations away from a^* are selected against. Such a value of a^* represents an unbeatable strategy in the sense of Hamilton (1967) and an evolutionarily stable strategy (ESS) in the sense of Maynard Smith (1982). Using the standard methodology, the ESS is calculated by determining the condition(s) for which

$$\frac{\partial W_i}{\partial a_i}\bigg|_{a_i = a^*} = 0,$$

which is when,

$$(1)\quad a^* = \frac{(F - 1)(d + F - 1)}{d(3F - 1) + (F - 1)(2F - 1)}$$

The second derivative is negative, so fitness is maximal at a^*.

The effect of inbreeding depression on the ESS can be seen by setting d to its extreme values, 0 and 1. When there is no inbreeding depression ($d = 1$),

$$(2)\quad a^* = \frac{F - 1}{2F} = \frac{K}{2(K + 1)},$$

where K is equal to $F - 1$, and gives the number of families available for interclonal cross fertilization. This result is directly analogous to Hamilton's (1967, eq. 5) result for local mate competition in dioecious species that practice biparental inbreeding, and to Lloyd's (1984, eq. 7) result for local mate competition in self compatible, cosexual plants. When inbred offspring do not survive to reproduce ($d = 0$), eq. (1) reduces to:

$$(3)\quad a^* = \frac{F - 1}{2F - 1} = \frac{K}{2K + 1},$$

which is directly analogous to Fischer's (1981) result for local mate competition in an outcrossing dioecious fish, and to Charnov's (1980, 1982) result for local mate competition in obligately outcrossing hermaphrodites (barnacles). The main conceptual difference between the present model and those of Charnov, Fischer, and Lloyd is that the number of mates is not limited. This is because each clone produces a large number of individuals; and, even if there were only one family, there would not be restricted access to mates of the kind that drives selection for reduced male allocation in their models. What is limited is the

number of different clonal families that co-occur at the time of reproduction, which is more similar to Hamilton's original model. Selection for reduced male allocation in this latter case of local family competition is due to the increased probability of cross-fertilization among members of the same clone as the number of clonal families decreases.

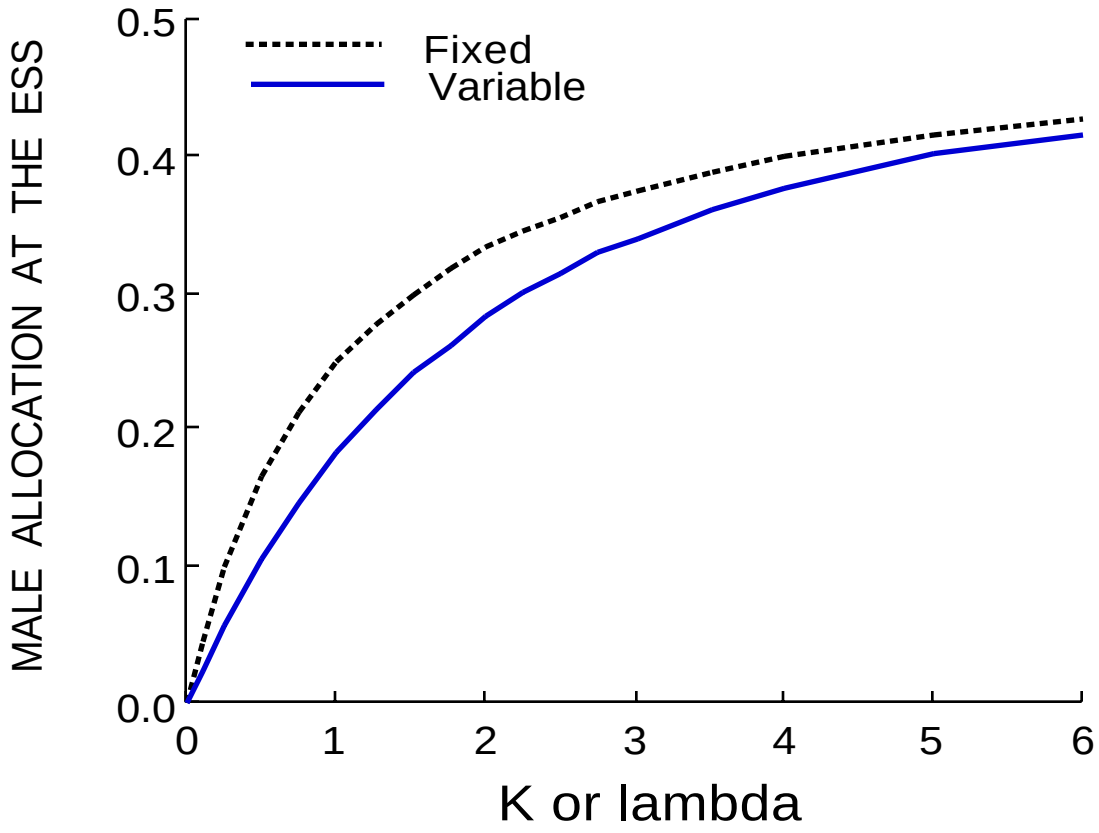

Figure 2. Male allocation at the ESS: (1) as a function of the number of clonal families available for interclonal cross fertilization (K), dashed line (from eq. 2), and (2) as a function of the mean number of families available for interclonal cross fertilization (λ) where K is determined by the Poisson distribution, solid line (from eq. 5). Both lines were plotted under the assumption that there is no inbreeding depression.

These results assume that family number is fixed. It is reasonable to suspect, however, that the number of clonal families available for interclonal sex would be variable. In this case, the fitness of individuals in the ith family is estimated as

$$W_i = \sum_{K=0}^{\infty} \Pr(K)(W_{if} + W_{im})$$

where Pr(K) is the probability of K. Working as above, the ESS is at

$$(4)\quad a^* = \sum_{K=0}^{\infty} \Pr(K)\frac{K(d + K)}{d(3K + 2) + K(2K + 1)}.$$

Assuming no inbreeding depression (d = 1) and that Pr(K) is given by the Poisson distribution,

$$(5)\quad a^* = \sum_{K=0}^{\infty} \frac{e^{-\lambda}\lambda^K}{K!} \times \frac{K}{2(K + 1)},$$

where λ is the mean value for K.

The effect of K being variable is difficult to see intuitively, so I have plotted a^* against K (for the fixed case, eq. 2) and lambda (for the variable case, eq. 5) in Fig. 2. Figure 2 shows that when the expected number of families is determined by the Poisson distribution, selection favors lower values of a^* for all lambda greater than zero. The difference between the two curves, however, is very slight for all lambda greater than about six.

3. COST OF BIPARENTAL SEX

Partial parthenogenesis.--The cost of cross-fertilization can be seen by considering a mutation that increases the fraction of eggs that develop parthenogenetically. I assume that parthenogenetic and cross-fertilized eggs have the same cost, and that the parthenogenetic eggs are drawn from the existing allocation to female function (1 - a_i); hence, a mutation that increases the number of parthenogenetic eggs by one also has the effect of decreasing the number of cross-fertilized eggs by the same amount. Let the fraction of eggs that develop by parthenogenesis in members of the ith clonal family be p_i. The remaining eggs, (1 - p_i), are fertilized by clone members with probability 1/F, and by non-clone members with probability (1 - 1/F). As previously, d is the fitness of offspring resulting from cross-fertilizations among members of the

same clonal family, relative to the fitness of offspring resulting from cross-fertilizations among member of different clones. Finally, let y be the fitness of offspring derived from parthenogenetic eggs relative to offspring resulting from cross-fertilizations among member of different clones, and let r_f be the fraction of the maternal genome transmitted to cross-fertilized eggs. (Note that y could be equal to d, depending on how parthenogenesis is achieved). The relative fitness gained through parthenogenetic eggs (W_{ip}) and cross-fertilized eggs (W_{if}) is, respectively:

$$W_{ip} = yp_i (1 - a_i),$$

$$W_{if} = \frac{r_f (1 - a_i)(1 - p_i)(d + F - 1)}{F}.$$

Similarly, the expected fitness through male function (W_{im}) is the reproductive value of males (V, as defined above) times the sum of clone-mated eggs plus non-clone-mated eggs, which is

$$W_{im} = \frac{Vr_m [(1 - a_i)(1 - p_i)d + (1 - a^*)(1 - p^*)(F - 1)]}{F},$$

where r_m is the fraction of the genome transmitted to the male gamete.

The proportion of resources allocated to parthenogenetic eggs will be selected to increase when this has a positive effect on W_i (equal to $W_{ip} + W_{if} + W_{im}$), which is when

$$\frac{\partial W_i}{\partial p_i} > 0.$$

Assuming that increasing p_i has no correlated effect on male allocation (i.e., $a_i = a^*$), the above inequality is satisfied when

$$(6)\quad y > \frac{d(r_f + r_m) + r_f (F - 1)}{F}$$

Because $r_f + r_m$ must equal one, this reduces to

$$(7)\quad y > \frac{d + r_f (F - 1)}{F}.$$

Hence, the minimum relative fitness of parthenogenetic offspring required for the spread of parthenogenetic egg production is independent of the allocation to male function (a_i), and inversely related to the number of clonal families (F) that coexist at the time of sexual reproduction. For a large number of families, parthenogenetic egg production is favored when $y > r_f$, which is when the relative fitness of parthenogenetically produced progeny is greater than the fraction of the maternal genome transmitted to sexual eggs (usually one-half, see also Lively and Lloyd, 1990). For a small number of families, the advantage of parthenogenesis is reduced, owing to the increase in cross fertilization among members of the same clone.

For the common case of diploidy ($r_f = 1/2$) and assuming no inbreeding depression ($d = 1$), eq. 7 reduces to

$$(8)\quad y > \frac{F+1}{2F} = \frac{K+2}{2(K+1)}$$

when family number is fixed, and to

$$(9)\quad y > \sum_{K=0}^{\infty} \Pr(K)\frac{K+2}{2(K+1)}$$

when the number of clonal families is determined by a probability distribution. Assuming, as previously, that the number of clonal families available for interclonal sex ($K = F - 1$) is given by the Poisson distribution for a mean of lambda, the minimum relative fitness of parthenogenetic offspring (y) required for the spread of parthenogenesis can be plotted as a function of lambda. This plot is given in Fig. 3, along with a plot of the minimum value of y required for the spread of parthenogenesis under the assumption that family number is fixed. Comparison of the two curves reveals that cross fertilization is more resistant to the spread of parthenogenetic egg production when the number of families available for interclonal sex is variable.

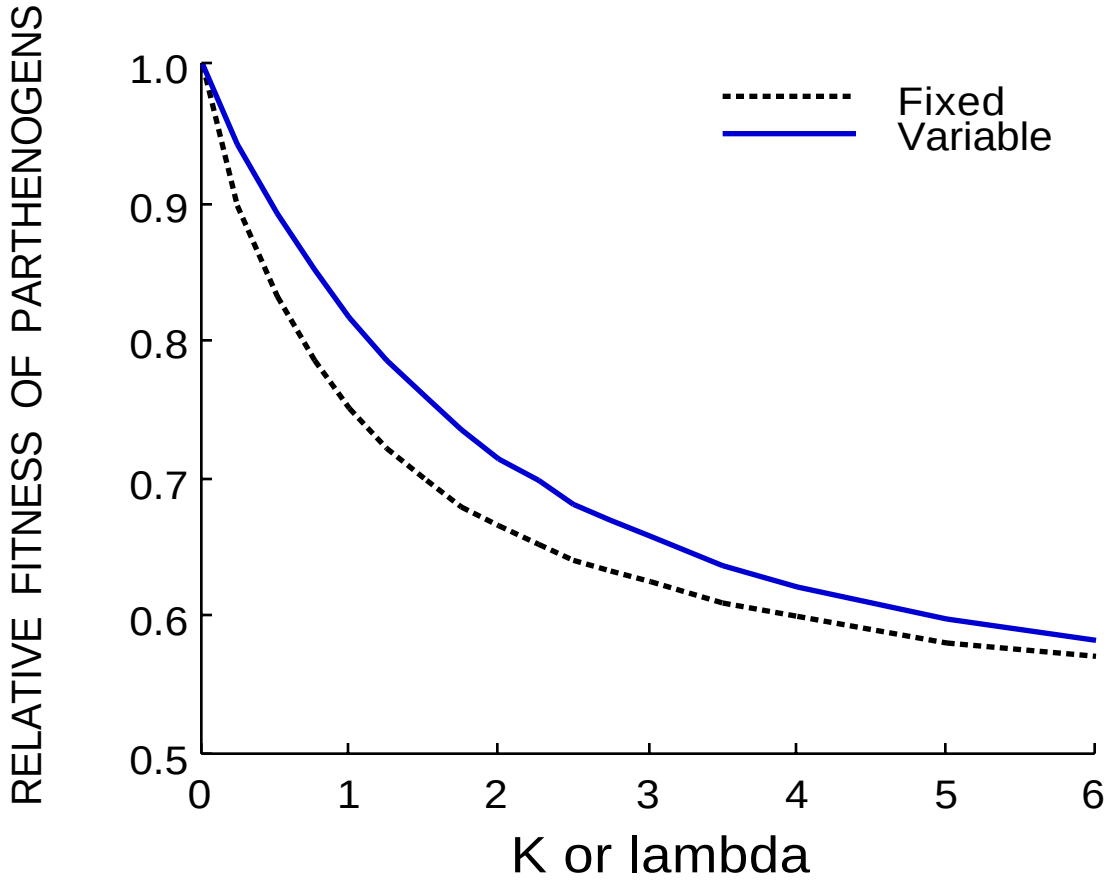

Figure 3. A graphical representation of the cost of sex: (1) as a function of the number of clonal families available for interclonal cross fertilization (K), dashed line (from eq. 8), and (2) as a function of the mean number of families available for interclonal cross fertilization (λ) where K is determined by the Poisson distribution, solid line (from eq. 9). Both lines were plotted under the assumption that there is no inbreeding depression. Biparental sex is stable to invasion by parthenogenetic egg production below the curves.

Complete parthenogenesis coupled with male sterility.--I now consider the spread of male-sterile, obligately parthenogenetic females in a population of obligately cross-fertilizing hermaphrodites. Because there is no exchange of alleles between the sexual and asexual subpopulations, it is appropriate to calculate the advantage of obligate parthenogenesis by its rate of increase (Bulmer, 1982).

The number of snails infected by obligately parthenogenetic worms at time t, A_t , can be used to calculate the expected number of snails infected by obligately parthenogenetic worms in the next generation, A_{t+1}:

$$(10)\quad A_{t+1} = \frac{A_t y k c}{A_t + (1 - a)S_t}$$

where S is the number of snails infected by sexual hermaphroditic worms, and a is the average allocation to male function by the hermaphrodites; y is the expected survivorship of asexual offspring relative to sexual offspring; and k is the carrying capacity of the population (following Charlesworth, 1980). The variable, c, represents the fraction of resources available to parthenogenetic individuals that are allocated to eggs. When A_t is very small relative to S_t, and S_t is approximately equal to k,

$$(11)\quad \frac{A_{t+1}}{A_t} = \frac{yc}{1 - a}.$$

The parthenogenetic subpopulation will, therefore, increase at the expense of the sexual subpopulation when $y > (1 - a)/c$. If the mutation to obligate parthenogenesis was not accompanied by a concomitant increase in the allocation to eggs [$c = (1 - a)$], there is no cost of sex (meaning that the relative fitness of parthenogenetic offspring, y, must be greater than 1 for parthenogenesis to spread). If, however, the parthenogens allocate all of their resources to eggs ($c = 1$), the condition for their spread when rare becomes $y > (1 - a)$, meaning that the cost of sex is proportional to the allocation to male function in the sexual population, as suggested by Maynard Smith (1971).

When the sexual population is at the ESS, $a = a^*$. Substituting a^* from eq. 2 above, eq. 11 becomes (for $c = 1$):

$$(12)\quad \frac{A_{t+1}}{A_t} = \frac{2Fy}{F + 1}.$$

Hence the parthenogenetic population will increase if $y > (F + 1)/2F$. This is the same condition as that for the spread of partial parthenogenesis (see eq. 8), except that it requires that female allocation in the sexual subpopulation is unconstrained and that it can evolve to the ESS. Female allocation may be

prevented from evolving to the ESS by structural constraints on brood size (see Heath, 1977, 1979; Charnov, 1982).

4. DISCUSSION

The number of clonal families in the local mating populations affects both the evolutionarily stable allocation to male function and the conditions for the spread of parthenogenesis. Male allocation is selected to increase with increasing family number, showing an asymptotic approach to one half (Fig. 2). For a fixed number of families, this result is directly parallel to the results of previous studies that have considered the effect of restricted access to mates on the optimal level of male allocation (e.g. Hamilton, 1967; Fischer, 1981; Charnov, 1980; Lloyd, 1984). The reason for this similarity can be seen intuitively by recognizing that each of the trematode families are derived from a single egg, and that their members are genetically identical. If each family is considered to be a single self-compatible individual, then the number of "mates" is simply the number of families.

While this may make the parallel nature of the results more clear by stretching the definition of "individual," it also obfuscates an important point: biparental inbreeding (in this case cross-fertilization among members of the same clone) selects for a reduced allocation to male function. This bears on the controversy as to whether Hamilton's original result for structured populations is due to restricted access to mates or to inbreeding (see Lloyd, 1984; Karlin and Lessard, 1986). Charnov (1980), Fischer (1981) and Lloyd (1984) have shown that restricted access to mates is sufficient to drive selection for reduced male allocation, but the number of mates in the present study is assumed to be large. What is restricted is the number of families in the breeding population. Hence the present study shows that biparental inbreeding is also sufficient to select for reduced allocation to male function, although under somewhat special conditions.

When the number of clonal families that co-occur during the sexual phase of the life cycle is variable and governed by the Poisson distribution (as might be expected in the wild), the optimal allocation to male function is lower

than it is when family number is fixed (Fig. 2). This is especially true when the mean number of families is low. As the mean increases, the Poisson distribution approaches that of the binomial, and the effect diminishes (see also Hamilton 1967; Karlin and Lessard, 1986).

The cost of sex under partial parthenogenesis was found to be due to Williams' (1975) "cost of meiosis". This cost, defined formally as $1 - r_f$, is the fraction of the genome not transmitted by the maternal parent to eggs (Lively and Lloyd, 1990). When the number of families in the mating population is small, the advantage of parthenogenesis is reduced due to the increased likelihood of biparental inbreeding, as suggested by Williams (1980). If family number is variable, the cost of sex is reduced even further (Fig. 3).

In contrast, the cost of sex under complete parthenogenesis was found to be due to Maynard Smith's (1978) "cost of males" or, in this case, the cost of male allocation. However, when male allocation can evolve to the ESS, the cost of sex is the same as under partial parthenogenesis for r_f equal to 1/2. That this is true may lie at the heart of the cost-of-sex controversy, but it is important to keep the two costs separate, primarily because male allocation (or more precisely, female allocation) may be constrained, and unable to reach the ESS.

Assuming that male allocation is not constrained, the cost of sex diminishes with a decrease in the number of families in the local mating population for both partial parthenogenesis (individual selection) and complete parthenogenesis (population selection). When infected snails are rare, as is the case in many of the *Potamopyrgus* populations in New Zealand (Lively, 1987), this means that the cost of sex may be very small. Hence, even though the snail hosts have become parthenogenetic in some populations, one would not necessarily expect for the trematodes to likewise give up sex.

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