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Oedipal mating as a factor in sex allocation in haplodiploids

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SUMMARY

Most theoretical work on brood sex ratio bias is based on life histories involving potential sibmating, where inseminated females colonize a habitat producing progeny that mate randomly among themselves. However, another type of life history can favour female biased broods; it involves mother–son matings and is uniquely accessible to haplodiploids. Colonization is accomplished by immature stages (mating is postdispersal) and female bias is favoured at low colonization densities by the fact that, unlike isolated males, isolated females are not lost to the gene pool because they can mate with their parthenogenetically produced sons. We present a mathematical model of the life history including parameters describing colonization density, degree of aggregation, the penalty incurred when a female must wait to mate with her parthenogenetically produced son, and inbreeding. Low colonization density favours female bias as does increased aggregation; a high penalty associated with waiting for maturation of a son with which to mate means that some proportion of males among progeny will be favoured even at very low colonization densities. Life histories that fit the model are known in nematodes and mites.

1. INTRODUCTION

Under most circumstances, gonochores are expected to produce approximately equal numbers of each sex (Fisher 1930). However, there are instances where females produce a strong bias of female progeny with the result that the operational sex ratio is skewed. Hamilton (1967) reviewed many such cases and provided a mathematical model to explain them. The model was based on a life history where inseminated females colonize a habitat and give rise to offspring that remain in the habitat mating randomly among themselves; females inseminated by such matings would then leave to search for new habitats. Under such conditions, when the number of colonizing females per habitat is low, these females typically produce female biased broods. The results can be explained in two ways: the female's male progeny are competing locally for mates and it is in the female's interest to produce only as many males as are needed to inseminate her female progeny. Alternatively, it can be argued that the level of inbreeding in these life histories is such that there is kin selection; thus, female biased groups generate more progeny and therefore result in more future colonizations. The two effects are usually impossible to separate but there is evidence that both effects may contribute in some groups (Yamaguchi 1985; May & Seger 1985). Hamilton's model seems to be applicable to a wide variety of organisms, most of which (as was recognized by Hamilton) are haplodiploid (males haploid and produced parthenogenetically, females diploid and derived from fertilized eggs). See Yamaguchi (1985) for a diplodiploid example.

Although theoretical work on skewed sex ratios has concentrated on the effect of sib-matings and local mate competition, some haplodiploids have another sort of life history that is expected to encourage skewed sex ratios. This life history involves matings between mothers and sons. Under this model, uninseminated females (or immature stages) search for new habitats. If males are unavailable, females produce them parthenogenetically; thus, females have a higher value than males as colonists because they can mate with their parthenogenetically produced sons. Borgia (1980) suggested that mother–son mating, far from being anomalous, was common among haplodiploids. However, the element that makes mother–son mating important is the dispersal of uninseminated females or immature stages. If there is a non-zero possibility that a colonizer will be isolated in a habitat and if the sex ratio is not skewed, then female colonizers will do better than males. Gould (1983) advanced the notion that the possibility of colonizing a habitat on the basis of isolated females could have been a key advantage favouring fixation of haplodiploidy. However, it is unclear how common such colonizing strategies are among haplodiploids.

This article presents a mathematical model describing how the sex ratio in haplodiploids is expected to behave under various colonization scenarios, given that mating occurs after dispersal. In particular we shall examine the effects of the level of aggregation and the colonization rate upon the optimum brood

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sex ratio. Finally we assess the commonness of such life histories in nature.

2. THE MODEL

(*a*) *Assumptions and procedure*

We consider a parasite or any species similarly dependent on colonization of discrete habitats or hosts. No density dependence is considered in the life history of the parasites. This assumption is not generally valid, but our attention is focused upon conditions of low parasite density, and hence this assumption is not too restrictive. It is assumed that no movement is possible between hosts. Sex determination is assumed to be haplodiploid: females are able to control the proportion of the progeny that is male: sons are produced parthenogenetically, but females require insemination to produce daughters. Production of sons and daughters incurs the same physiological cost to the female.

The reproductive output of a female depends upon whether or not she arrives with at least one male, because if no male is present she must wait to mate with a parthenogenetically produced son. We represent this delay in sexual reproduction as a cost, or devaluation of her fecundity. The probability of this cost being exacted is a function of the dispersion pattern of males and females. The relative fitness of males and females also depends on their respective dispersion patterns. In particular, female progeny have greater value than male progeny since they can colonize on the basis of isolated individuals by mating with parthenogenetically produced sons. Males, similarly isolated, are lost to the gene pool. Thus, at low rates of colonization (where there is an appreciable chance of males being isolated in hosts) one expects selection to favour female biased broods. Therefore the optimal sex ratio is sensitive to the dispersion pattern in the host population.

In presenting the model we first develop functions that describe dispersion patterns of males and females in the host population as functions of prevailing male and female densities. This allows us to calculate the probability of parasites locating potential mates under different rates of colonization.

Second, we examine the reproductive output of a female as a function of the probability of her colonizing a host with at least one male, bearing in mind that she must wait to mate with a parthenogenetically produced son before she can produce female progeny if she finds herself in a host with no male. Third, we calculate the expected numbers of the two sexes in the ensuing colonization generation as a function of the brood sex ratio produced by the female.

Fourth, we assign values to males and females. Males are assumed to compete with other males for mates, and their fitness is assumed to be inversely proportional to the number of competitors present. Males produce no male progeny: their fitness is entirely through production of daughters. The fitness of female offspring is the sum of two components: one that describes fitness in hosts where no male is present, and the other that describes her fitness in hosts with at least one male. We include a parameter that describes the cost of outbreeding, because the male that a female mates with may be unrelated to her.

Finally we calculate the optimal sex ratio of colonists as a function of the expected number of parasites that colonize a given host. The optimal sex ratio of the colonists is characterized by the condition that there is no advantage for a given female in having the sex ratio of her offspring deviate from the sex ratio of the colonists.

(*b*) *Dispersion Pattern*

To determine the probability of finding a mate under different colonization rates, we treat male and female propagules as independently distributed in the host population according to negative binomial distributions with a common clumping parameter K, but with means y and x respectively. Although our notation differs slightly, the present analysis is similar to that developed by May (1977) to explore mating probabilities in *Schistosoma* (Digenea) and other parasites under differing degrees of aggregation. In particular, our case corresponds to May's case II (sexes distributed independently) and Appendix C, where complete promiscuity is assumed.

Propagules may be passively dispersed stages or actively dispersing immatures. To develop these distributions, we assume that hosts are of differing susceptibilities to infection. Variation in susceptibility may arise from factors inherent to the organisms (e.g. genetics) or from interactions of the organism with its environment (e.g. behaviour, local microhabitat differences, etc.). We assume that the susceptibility types are distributed according to the Gamma distribution with parameter K. Among hosts of a given susceptibility type t, we assume that the distribution of male and female parasites is Poisson with means ty/K and tx/K respectively. By compounding the gamma distribution of host types with the Poisson distribution of parasites within hosts of a given type, we arrive at negative binomial distributions for males and females with respective means y and x, and common clumping parameter K.

The density of the gamma distribution of host types is given by

$$d\gamma(t,K) = \frac{e^{-t}t^{K-1}dt}{\Gamma(K)}, \qquad 0 \leqslant t < \infty . \tag{1}$$

According to the Poisson model, the probability that a host of type t is infected with exactly m males is expressed as

$$p_m(y,t,K) = \mathrm{e}^{-ty/K}\frac{1}{m!}(ty/K)^m. \tag{2}$$

It is essential that we distinguish between the distribution of parasite numbers when they are confined to hosts of a given type t, and when they are averaged over all possible hosts, weighted according to the distribution of possible host types. It is the latter

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distribution that gives us the probability that parasites will encounter possible mates. We want to calculate the probability that a parasite will be isolated in a host with no members of the opposite sex because, under these circumstances, females enjoy a higher value. The probability that a host has no male parasites, averaged over all host types (i.e. irrespective of its type) is given by

$$P_0(y,K) = \int_0^\infty p_0(y,t,K)\mathrm{d}\gamma(t,K) = \int_0^\infty \mathrm{e}^{-ty/K}\mathrm{d}\gamma(t,K). \quad (3)$$

This integral may be evaluated to yield

$$P_0(y,K) = \begin{cases} (1+y/K)^{-K} & \text{if } K < \infty \\ \mathrm{e}^{-y} & \text{if } K = \infty . \end{cases} \quad (4)$$

The number of offspring produced by each female that survive to colonize in the next host colonization event is assumed to be N. If no males colonize the host in which a female finds herself, the female experiences a delay in reproduction since she must produce males parthenogenetically in order to produce sexually. This results in some lesser number of progeny W_rN $(0 < W_r < 1)$. The expected number of progeny in a host of type t is denoted by $Nh(t,y,K)$. The quantity $h(t,y,K)$ is computed by considering two cases: either no males colonize the host and the output of the female is W_rN, or at least one male colonizes the host and the output is N. The result is

$$\begin{aligned} h(t,y,K) &= p_0(y,t,K)W_r + (1 - p_0(y,t,K)) \\ &= 1 - p_0(y,t,K)(1 - W_r). \end{aligned} \quad (5)$$

Generalizing over all host types, we calculate the expected numbers of males and females in this next colonization event, denoted by x' and y' respectively:

$$x' = \int_0^\infty N(1-\phi)\frac{xt}{K}h(t,y,K)\mathrm{d}\gamma(t,K), \quad (6)$$

$$y' = \int_0^\infty N\phi\frac{xt}{K}h(t,y,K)\mathrm{d}\gamma(t,K), \quad (7)$$

where ϕ denotes the fraction of male offspring produced by each female. This fraction is assumed to be determined by the female. Integrals (6) and (7) may be evaluated to yield

$$x' = N(1-\phi)xH(y,K), \quad (8)$$

$$y' = N\phi xH(y,K). \quad (9)$$

Here

$$H(y,K) = 1 - (1 - W_r)(1 + y/K)^{-(K+1)}, \quad (10)$$

$NH(y,K)$ being the expected number of offspring per female averaged over all host types. $H(y,K)$ varies depending on the probability that the penalty $1 - W_r$, will be exacted under a particular colonization rate. As y approaches ∞, the probability of this penalty approaches zero, and $H(y,K)$ approaches 1. As y approaches 0, $H(y,K)$ approaches W_r.

(c) *Values of males and females*

We define V_m and V_f as the values of males and females, respectively. We assume that colonizations of a given host by different individuals are independent events. Thus, if a male colonizes a given host, the distribution of the remaining males will be Poisson as above. If a host of type t is colonized by $1+m$ males, the value of offspring produced attributable to each male is

$$\frac{xt}{K}\frac{1}{1+m}N(1-\phi)\frac{V_f}{2}. \quad (11)$$

Here xt/K is the mean number of females in a host of susceptibility type t. Males share the progeny of the females in the host equally among themselves. One of the assumptions of the model is haplodiploidy; males are impaternate and there is no term relating to male progeny. The factor $1/2$ is due to the fact that males contribute half of the genes to female progeny.

The factor $1/(1+m)$ in equation (11) is an expression of competition among males for mates. Competition for mates has encouraged evolution of highly pugnacious males in many haplodiploid arthropods (Hamilton 1967, 1979). There is increasing evidence of intense intraspecific interference competition among males in haplodiploid nematodes such that regardless of infective dose the mature population of worms in a host consists of single males and a few females (Zervos 1988; Adamson *et al.* 1989; Adamson & Noble 1992). It should be noted that equation (11) holds even if only a fixed number (say r) males can survive in a given host. In this case, a given male will contribute only if he is among the first r to arrive at the host. The probability of arriving first (or in any particular order) is $1/(1+m)$. Thus the contribution of a given male is $r/r(1+m) = 1/(1+m)$, as in equation (11). The expected value of a male colonizer in a host irrespective of type is

$$V_m' = N(1-\phi)\frac{V_f}{2}\int_0^\infty \frac{xt}{K}\sum_{m\geqslant 0}\frac{p_m(t,y,K)}{1+m}\mathrm{d}\gamma(t,K). \quad (12)$$

This awkward looking integral can be evaluated using generating functions. The generating function of the Poisson distribution for the number of males colonizing a host of type t is defined by

$$f(s,t,y,K) = \sum_{m\geqslant 0} p_m(t,y,K)s^m \quad (13)$$

After substitution of equation (2), the result is

$$f(s,t,y,K) = \sum_{m\geqslant 0}\mathrm{e}^{-ty/K}\frac{1}{m!}(tsy/K)^m = \mathrm{e}^{ty(s-1)/K}. \quad (14)$$

It follows by integration of equation (13) and from equation (14) that

$$\sum_{m\geqslant 0}\frac{p_m(t,y,K)}{1+m} = \int_0^1 f(s,t,y,K)\mathrm{d}s = \frac{K}{ty}(1 - \mathrm{e}^{-ty/K}). \quad (15)$$

After substitution of (15), the integration in (12) may be performed to yield

$$V_m' = xN(1-\phi)\frac{V_f}{2}G(y,K), \tag{16}$$

where

$$G(y,K) = \int_0^\infty \frac{t}{K}\frac{K}{ty}(1-e^{-ty/K})d\gamma(t,K) = \frac{1}{y}[1-(1+y/K)^{-K}]. \tag{17}$$

Note that as the integrand of equation (17) is derived from equation (15), the value of $G(y,K)$ reflects the possibility that a male will have to compete with other males for mates. It may be regarded as an index of the effect of male competition on male reproductive output. It follows from (17) that

$$G(y,K) \approx \begin{cases} 1 & \text{if } y \text{ is small,} \\ 1/y & \text{if } y \text{ is large.} \end{cases} \tag{18}$$

Figure 1 shows $G(y,K)$ vs y for various values of K. The decrease in G as y increases is sharpest for small values of K, as the distribution of males is highly clumped for small K: if a given host is infected by a male, the host is likely to be infected by more than one male.

The expected value of a female colonizer who selects a fraction ϕ' (possibly different from ϕ, which is the sex ratio for the population as a whole) is given by

$$V_f' = NP_0(y,K)W_r[(1-\phi')V_f + \phi' V_m] + N[1-P_0(y,K)][(1-\phi')iV_f + \phi' V_m]. \tag{19}$$

Here $i=(1+I)/2$, where I is the inbreeding coefficient. The first term on the right hand side results from the possibility that no males colonize the host. The product iV_f appears in the second term because the female contributes only a fraction i of the genes of her female offspring if she is inseminated by a male who is not one of her offspring.

(*d*) *Equilibrium assumptions*

We assume the population size is at equilibrium, with fixed ϕ, x and y, i.e. $V_m' = V_m$, $V_f' = V_f$. If so, equation (8) implies that

$$N(1-\phi) = 1/H(y,K). \tag{20}$$

This result may be substituted into equation (16) to obtain

$$\frac{V_m}{V_f} = \frac{x}{2H(y,K)}G(y,K). \tag{21}$$

A sex ratio ϕ' will not be stable unless it maximizes the right hand side of (19). If fact ϕ' appears linearly there, so the necessary condition for such stability (no improvement if ϕ' differs from ϕ) is that

$$P_0(y,K)W_r(V_f - V_m) + (1-P_0(y,K))(iV_f - V_m) = 0. \tag{22}$$

Equation (22) may be solved for V_m/V_f in the form

$$\frac{V_m}{V_f} = \frac{i(1-P_0(y,K)) + W_r P_0(y,K)}{1-P_0(y,K)+W_r P_0(y,K)}. \tag{23}$$

When (21) and (23) are combined, the result is

$$x = X(y) = \frac{i(1-P_0(y,K)) + W_r P_0(y,K)}{1-P_0(y,K)+W_r P_0(y,K)} \times \frac{2H(y,K)}{G(y,K)}. \tag{24}$$

This equation expresses x, the mean number of female colonists per host, as a function of y, the mean number of male colonists per host, and therefore allows us to calculate the stable brood sex ratio given by

$$\phi = \Phi(y) = \frac{y}{X(y)+y}. \tag{25}$$

(*e*) *The effect of colonization density*

Although equation (25) is complicated, we can obtain simple information in some limiting cases. A high rate of colonization corresponds to high values of x and y. If y is large, in view of equation (18) we have

$$\frac{1}{G(y,K)} \approx y, \tag{26}$$

and from equation (4)

$$P_0(y,K) \approx 0. \tag{27}$$

It follows from equation (10) that

$$H(y,K) \approx 1. \tag{28}$$

Finally, under such conditions we can reasonably expect inbreeding to be negligible and i therefore becomes 1/2. Hence equation (24) becomes

$$x \approx 2iy, \tag{29}$$

and equation (25) becomes

$$\phi \approx \frac{1}{2i+1}. \tag{30}$$

Thus as colonization density increases, the optimum brood sex ratio approaches 1/2.

On the other hand if y is small, then from equation

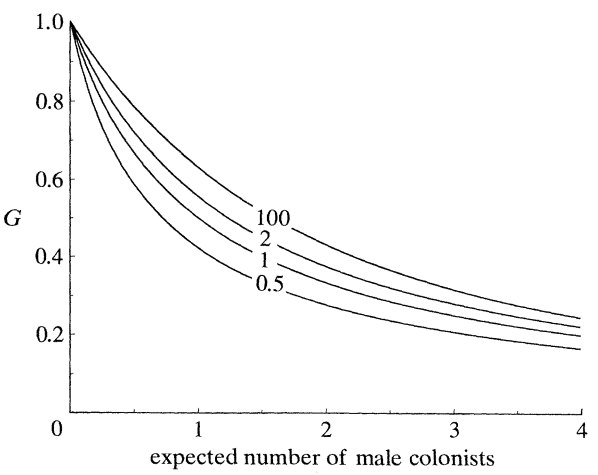

Figure 1. The function $G=\frac{1}{y}[1-(1+y/K)^{-K}$ as a function of male colonists, y, for several values of K, the aggregation parameter. $G(y,K)$ has a maximum value of 1, and decreases to 0 as y approaches ∞; it can be regarded as an index of the effect of male competition for mates on male reproductive output. For any given density of colonization, y, the effect is more pronounced as aggregation increases (lower K).

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(4), $P_0(y,K) \approx 1$, and equation (10) implies that

$$H(y,K) \approx W_r. \quad (31)$$

In view of equation (18), (24) becomes

$$x \approx 2W_r. \quad (32)$$

That is, as y approaches 0, x approaches $2W_r$. The quantity, $1 - W_r$, describes the penalty a female experiences as a result of having to produce and mate with her son. A zero penalty corresponds to $W_r = 1$, and under these conditions purely female broods are predicted unless colonization densities $(x+y)$ exceed a mean of 2 per host. The effect of increasing the penalty is to shift the curve to the left. Figure 2 shows the optimum ratio of males to females as a function of the expected total number of parasites, for various values of K. The response of the ESS sex ratio under Hamilton's (1979) model involving local mate competition is shown as a dotted curve, for the purpose of comparison.

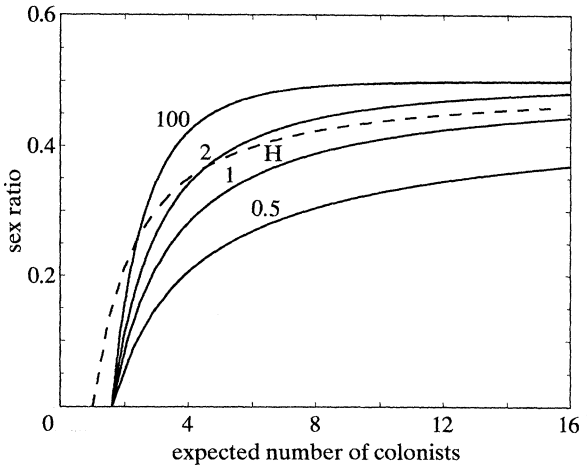

Figure 2. Optimum sex ratio (proportion of male progeny) for haplodiploid females as a function of colonization density for various levels of aggregation ($K = 0.5$, 1.0, 2.0 or 100) (solid lines) under a model involving mother–son mating (W_r is set at 0.8; see text for details). At low colonization densities, female progeny are favoured because even when isolated they can mate and produce progeny with parthenogenetically produced sons. The dashed line indicates the optimum sex ratio under Hamilton's (1979) sib-mating scheme. Here, all colonists are inseminated females.

3. DISCUSSION

(*a*) *Dynamics of colonization*

In our model, the risk colonists encounter of not locating a mate is calculated with the assumption that colonists arrive simultaneously. In many situations, however, colonists arrive over a protracted period. Thus, the number of colonists in a host at the end of this time may not be an accurate measure of the risk. For a given density of colonization, rapid development to reproductive age or a protracted colonization period (which corresponds to a lower rate of colonization) render the system more sensitive to problems relating to mate location, and would encourage more reliance on female colonists. A further element added by introducing a dynamic to colonization is that late arriving colonists are more likely to encounter mates than are those that arrive early; further, these mates may often be progeny of parasites that arrived earlier.

(*b*) *Effect of aggregation*

K describes the degree of aggregation of the parasites; the lower its value, the higher the level of aggregation. When K is infinite the distribution is random and Poisson. The effect of increasing aggregation (decreasing K) in the model is to decrease the optimum brood sex ratio for any given rate of colonization.

This result seems counterintuitive at first, because increased aggregation should decrease the chances that parasites will be isolated from members of the opposite sex. This should decrease the higher value female progeny enjoy by virtue of their ability to produce mates parthenogenetically. However, the value of males in our model is proportional to $G(y,K)$, the value of which decreases with increasing y. This decrease is more pronounced for lower K (figure 1). Aggregation increases the probability that males will occur together in the same host and compete with other males for mates. Competition for mates is not experienced by females and this asymmetrical aspect of the model presumably drives the optimum brood sex ratio towards female bias.

Throughout the analysis we have ignored potential effects of density dependence, although such effects are well documented in host–parasite systems (e.g. Keymer 1982; Schad & Anderson 1985; Anderson & May 1985). Our justification has been that the effects we are examining occur at very low mean colonization densities where density dependence is expected to be less important. However, this justification is invalid where parasites are highly aggregated because, under such circumstances, high densities can be experienced by most parasites even when mean densities are low. Exactly how this affects our conclusions depends not only on the strength of density dependence but on how it operates; for example, does it affect parasite survival or fecundity, and is it experienced equally by the sexes? It is outside of the scope of this article to examine these aspects here, but it should be realized that aggregation is an almost ubiquitous aspect of parasite populations (Anderson & Gordon 1982) that can strongly enhance the importance of density dependence in the real world.

(*c*) *Mother–son matings as a colonization strategy in nature*

The nature of the above life history strategy demands that isolated females can produce males parthenogenetically. This criterion is satisfied by haplodiploidy, which is the characteristic form of sexual reproduction in a number of animal groups: pinworm nematodes, monogonant rotifers, most mites (but not ticks), thysanopterans, hymenopterans, and a few scale insects and beetles. Males need not be

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haploid as long as they are impaternate. However, parahaploid organisms, in which males are haploid but derive from fertilized eggs whose paternal chromosome complement disappears, could not avail themselves of the strategy discussed here. The further requirement is that mating occur post dispersal. That is, colonization must be accomplished by immature stages, or virgin females.

Many of the examples discussed by Hamilton (1976) do not fit the model because inseminated females colonize habitats, often laying eggs in clusters. Mother–son matings do occur in some of these species (Balfour-Browne 1922; van Embden 1930; Entwhistle 1964), but it is not clear such matings form part of a colonization strategy. Rather, they result from the occasional failure of a mating system that normally involves potential sibs: i.e. inseminated females colonize and mating occurs among their progeny. Uninseminated haplodiploid females can still produce male progeny and are therefore not a complete genetic loss. In fact, Godfray (1990) notes that as long as the population is in equilibrium with respect to sex ratio, virgin haplodiploid females suffer little disadvantage. However, in our model, colonization occurs before mating and male progeny have no fitness unless they colonize a host that contains a female. The ability of a female to produce sons with which to mate is potentially central to the colonization process.

The case that first called our attention to this phenomenon was that of *Gyrinicola batrachiensis*, a nematode parasite of tadpoles. This species belongs to the order Oxyurida, whose members, known as pinworms, have recently been shown to be haplodiploid (Adamson 1989). Pinworms live as adults in the posterior gut of their hosts and are transmitted by larvae in eggs that are passed in faeces and contaminate the host's environment. Mother–son matings are not expected to occur commonly in such life cycles since a female's progeny must leave the host. However, in *Gyrinicola* spp. and a few species in other genera, a second mode of reproduction has developed that makes mother–son matings possible. In these forms, females produce two types of eggs. Thin-shelled eggs contain well developed larvae that hatch at deposition and develop in the same host as the mother; thick-shelled eggs are deposited in the two to four cell stage of cleavage and must pass to the external environment before they are infective.

In the two species that have been studied, *G. batrachiensis* and *Tachygonetria vivipara*, there appears to be an alternation of generations such that females that hatch from thick-shelled eggs produce predominantly thin-shelled eggs, and vice versa (Adamson 1981*a*,*b*; Adamson & Petter 1983). Furthermore, most embryos in thick-shelled eggs, the colonizing generation, are diploid and will develop as females. Hosts are colonized when they accidentally ingest thick-shelled eggs contaminating their environment; these hatch releasing larvae that develop to adulthood in the gut. Females of this generation produce thin-shelled eggs containing larvae that will develop in the same host as the mother. If they are isolated from males, they can produce thin-shelled eggs parthenogenetically and mate with their sons. Females derived from thin-shelled eggs produce thick-shelled eggs that pass out of the host and disperse.

The colonizing period in *G. batrachiensis* lasts about 3 months (July through September) in Canada. During a three year study, the number of adult worms in hosts in late fall varied between 3 and 12 (Adamson 1981*b*). If we assume colonization is a random process then the mean interval between colonizations varied between 9 and 35 days with a mean of 17. This corresponds to about the period a female requires to reach reproductive age. Thus, early colonists may often reach reproductive maturity in isolation from others of the opposite sex even though, by the end of the colonization period, hosts typically contain a dozen worms. Males mature about twice as fast as females (Adamson 1981*a*) and this lessens the delay in reproduction associated with mating with a son.

Brood sex ratio does seem to be skewed in *G. batrachiensis*. Thus in a sample of 234 females, 205 of 296 (69%) embryos in thick-shelled eggs were diploid and would develop as females. Most worms that develop from experimental infections using thick-shelled eggs, and most worms recovered from young of the year hosts were female.

Many mites are haplodiploid and several groups have life histories that could accommodate mother–son matings. Most astigmatic mites are free living bacterial or fungal feeders that occur in damp situations. However, dispersal is often accomplished by a hypopus that has a phoretic association with particular insects, birds or mammals (Hughes 1961; Houck & O'Connor 1991). The hypopus is a late nymphal stage that typically both sexes would pass through. However, we would expect a bias towards females in these colonizing stages unless large numbers colonize the same phoretic host. Sex ratios among dispersing hypopi are unknown but in many species males are rare and, when found, occur in clusters. This may indicate that uninseminated females are forced to produce mates parthenogenetically after colonization.

A simple life history to which the model applies was described by Oliver (1962) studying the astigmatid mite *Histiostoma murchei*. This species colonizes coccoons, or egg cases, of earthworms. Female development includes an motile hypopodal stage, and it is this stage that colonizes the host. Thus, only females colonize, and these produce a small number of haploid eggs that develop as males and inseminate females in the coccoon. Inseminated females then produce a larger number of fertilized eggs that hatch and develop within the coccoon to the hypopodal stage.

In the model presented, strict reliance on female colonizing stages is expected to occur wherever the colonization rate is less than $2Wr$. Since Wr cannot be greater than 1 by definition, colonization rates in excess of 2 individuals per host should favour production of at least some dispersing males. Oliver (1962) estimated intensity of infection in *H. murchei* at 2.3 adult females per egg case. Depending on the dynamics of colonization this may or may not be an accurate estimate of colonization rate. However, Oliver (1962) suggests that colonization occurs while

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egg cases are being formed and is therefore virtually simultaneous. Under the model we would expect some fraction, however small, of male colonizers to exist. In *H. murchei*, however, development through a hypopodal, or colonizing, stage occurs only in females.

Many mesostigmatid mites disperse as deutonymphs and would make good potential candidates for the type of life history we describe. For example, *Macrocheles muscaedomesticae* (Scopoli) is a predator of early stages of development of house flies and uninseminated females disperse on adult flies according to Pereira & Castro (1947).

Members of the family Tetranychidae, spider mites, are another group where mother–son matings may be frequent. de Boer (1985) suggested that most spider mite (Tetranychidae) populations begin when virgin females mate with their parthenogenetically produced sons. However, most long distance dispersal is accomplished by mated females (Mitchell 1970; Potter 1979). Kennedy & Smitley (1985) note that, although dispersal to new host plants is typically effected by mated females, these produce largely female progeny that disperse by crawling to new leaves where they frequently will not find mates. Thus, there are two levels of dispersal of which the more local level involves a female bias. To our knowledge this suggestion has not been documented in any detail.

Most Thysanoptera mate predispersal but the dynamics of dispersal are complicated by the fact that females and males move from one plant to another throughout their lifetime (Lewis 1973). There is some evidence, however, that many female western flower thrips are virgin early in the season; thus, male biased sex ratios are common early in the season and this is difficult to explain except as the result of virgin females producing males in the absence of mates (Higgins & Myers 1992; C. J. Higgins, personal communication). If these thrips winter as pupae or virgins, and are likely to experience difficulty locating mates in the spring, we would predict a female bias in wintering stages.

In monogonant rotifers and some Hymenoptera (notably, Cynipidae) males are seasonally rare or absent. One therefore expects some level of mother–son matings to be obligatory. In fact, this is not the case, because both groups can produce females as well as males parthenogenetically. Matings may occur between mothers and sons but these are key to any colonization strategy. At some point in the season, rotifers give rise to mictic females, which produce unfertilized eggs that develop as males, and, after insemination, fertilized eggs that diapause. These latter give rise to amictic females of the next season (Ruttner-Kolisko 1971; Gilbert 1974). Cynipidae undergo two generations a year in temperate regions. Amictic females produce haploid and diploid eggs parthenogenetically, whereas inseminated mictic females produce amictic female progeny.

The model presented here demonstrates that female-biased sex ratios can be favoured in haplodiploid species that colonize discrete habitats prior to mating. Furthermore, there is evidence that such life histories occur in some nematodes and mites. More work is needed before the distribution of this type of life history can be appreciated fully, but it is clearly not the rule even within haplodiploids. The extremely spotty distribution of such life histories does not support Gould's (1983) suggestion that these sorts of life histories may have been ancestral in haplodiploids.

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REFERENCES

Adamson, M.L. 1981*a* Development and transmission of *Gyrinicola batrachiensis* (Walton, 1929) Adamson, 1981 (Pharyngodonidae; Oxyuroidea). *Can. J. Zool.* **59**, 1351–1367.

Adamson, M.L. 1981*b* Seasonal changes in populations of *Gyrinicola batrachiensis* (Walton, 1929) in wild tadpoles. *Can. J. Zool.* **59**, 1377–1386.

Adamson, M.L. 1989 Evolutionary biology of the Oxyurida (Nematoda): Biofacies of a haplodiploid taxon. *Adv. Parasitol.* **28**, 175–228.

Adamson, M.L. & Noble, S. 1992 Structure of the pinworm (Oxyurida; Nematoda) guild in the hindgut of the American cockroach, *Periplaneta americana*. *Parasitology* **104**, 497–507.

Adamson, M.L. & Petter, A.J. 1983 Studies on gametogenesis in *Tachygonetria vivipara* Wedl, 1862 and *Thelandros alatus* Wedl, 1862 (Oxyuroid; Nematoda) from *Uromastix acanthinurus* in Morocco. *Can. J. Zool.* **61**, 2357–2360.

Adamson, M.L., Buck, A. & Noble, S.J. 1992 Transmission pattern and intraspecific competition as determinants of population structure in pinworms (Oxyurida; Nematoda). *J. Parasitol.* **78**, 420–426.

Anderson, R.M. & Gordon, D.M. 1982 Processes influencing the distribution of parasite numbers within host populations with special emphasis on parasite induced host mortalities. *Parasitology* **85**, 373–391.

Anderson, R.M. & May, R.M. 1985 Helminth infections in humans: mathematical models, population dynamics and control. *Adv. Parasitol.* **24**, 1–101.

Balfour-Browne, F. 1992 On the life history of *Mellitobia acasta* Walker, a chalcid parasite of bees and wasps. *Parasitology* **14**, 349–371.

Boer, R. de 1985 Reproductive barriers. In *Spider mites: their biology, natural enemies and control* (ed. W. Helle & M. W. Sabelis), vol. 1A, pp. 193–200, Amsterdam: Elsevier.

Borgia, G. 1980 Evolution of haplodiploidy: models for inbred and outbred systems. *Theor. Pop. Biol.* **17**, 103–128.

Endem, F. van 1931 Zur Kenntnis der Morphologie und Okologie des BrotkaferParasiten *Cephalonomia quadridentata* Duchaussoy. *Z. Morphol. Okol. Tiere* **3**, 425–574.

Entwhistle, P.F. 1964 Inbreeding and arrhenotoky in ambrosia beetles *Xyloborus compactus* (Eichh.) (Coleoptera; Scolytidae). *Proc. R. ent. Soc. Lond.* A **39**, 401–439.

Fisher, R.A. 1930 *The Genetical theory of natural selection.* (272 pages.) Oxford University Press.

Gilbert, J.H. 1974 Dormancy in rotifers. *Trans. Am. Microsc. Soc.* **93**, 490–513.

Godfray, H.C.J. 1990 The causes and consequences of constrained sex allocation in haplodiploid animals. *J. evol. Biol.* **3**, 3–17.

Gould, S.J. 1983 *Hen's teeth and horse's toes.* (413 pages.) New York and London: W. W. Norton & Company.

Hamilton, W.D. 1967 Extraordinary sex ratios. *Science, Wash.* **156**, 477–488.

Hamilton, W.D. 1979 Wingless and fighting males in fig

wasps and other insects. In *Sexual selection and reproductive competition in insects* (M. Blum & A. Blum), pp. 167–220. New York: Academic Press.

Higgins, C.J. & Myers, J.H. 1992 Sex ratio patterns and population dynamics of western flower thrips (Thysanoptera; Thripidae). *Environ. Ent.* **21**, 322–330.

Houck, M.A. & O'Connor, B.M. 1991 Ecological and evolutionary significance of phoresy in the Astigmata. *A. Rev. Ent.* **36**, 611–636.

Hughes, A.M. 1961 The mites of stored food. *Min. Agric. Fish. Food Tech. Bull.* **9**, 287 p.

Kennedy, C.G. & Smitley, D.R. 1985 Dispersal. In *Spider mites, their biology, natural enemies and control* (ed. W. Helle & M. W. Sabelis), vol. 1A, pp. 233–242. Amsterdam: Elsevier.

Keymer, A. 1982 Density dependent mechanisms in the regulation of intestinal helminth populations. *Parasitology* **84**, 573–587.

Lewis, T. 1973 Thrips, their biology, ecology and economic importance. New York: Academic Press.

May, R.M. 1977 Togetherness among Schistosomes: its effects on the dynamics of the infection. *Math. Biosci.* **35**, 301–343.

May, R.M. & Seger, J. 1985 Sex ratios in wasps and aphids. *Nature, Lond.* **318**, 408.

Mitchell, R. 1970 An analysis of dispersal in mites. *Am. Nat.* **104**, 425–431.

Oliver, J.H. 1962 A mite parasitic in the coccoons of earthworms. *J. Parasitol.* **48**, 120–123.

Pereira, C. & de Castro, M.P. 1947 Forese e partenogenese arrenotoca em *Macrocheles muscaedomesticae* (Scopoli) (Acarina; Macrochelidae) e sua significacão ecologica. *Arq. Inst. Biol. São Paulo* **18**, 71–89.

Potter, D.A. 1979 Reproductive behaviour and sexual selection in Tetranychine mites. *Rec. Adv. Acarol.* **1**, 137–145.

Ruttner-Kolisko, A. 1971 Rotatoria. *Die Binnengewasser* **26**, 99–234.

Schad, G.A. & Anderson, R.M. 1985 Predisposition to hookworm in man. *Science*, **228**, 1537–1540.

Yamaguchi, Y. 1985 Sex ratios of an aphid subject to local mate competition with variable maternal condition. *Nature, Lond.* **318**, 460.

Zervos, S. 1988 Evidence for population regulation, reproductive competition and arrhenotoky in a thelastomatid nematode of cockroaches. *Parasitology* **96**, 369–379.

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