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Parental sex discrimination and intralocus sexual conflict

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Intralocus sexual conflict occurs when populations segregate for alleles with opposing fitness consequences in the two sexes. This form of selection is known to be capable of maintaining genetic and fitness variation in nature, the extent of which is sensitive to the underlying genetics. We present a one-locus model of a haploid maternal effect that has sexually antagonistic consequences for offspring. The evolutionary dynamics of these maternal effects are distinct from those of haploid direct effects under sexual antagonism because the relevant genes are expressed only in females. Despite this, we find the same opportunity for sexually antagonistic polymorphism at the maternal effect locus as at a direct effect locus. Thus, sexually antagonistic maternal effects may underlie some natural genetic variation. The model we present permits alternative interpretations of how the genes are expressed and how the fitness variation is assigned, which invites a theoretical comparison to models of both imprinted genes and sex allocation.

Keywords: maternal effect; sexual conflict; sex allocation

1. INTRODUCTION

Mothers sometimes treat sons and daughters differently: *Blastophaga* fig wasps produce more daughters than sons so that each son has more offspring than his sisters (Hamilton 1979); a female antechinus may eat the sons attached to her nipples but continue to suckle her daughters (Cockburn 1994); and Dominican women wean their sons before their daughters (Quinlan *et al.* 2005). It may be the case that variation among mothers in the differential treatment of sons and daughters results from phenotypic plasticity of a common genotype, but it is also possible that loci segregate for alleles with differential effects on sons and daughters. The possibility that alleles can have differential fitness effects on male and female carriers in nature is well appreciated (Rice & Chippindale 2001) and has even been demonstrated empirically (Foerster *et al.* 2007).

In this paper, we present a simple model in which alleles expressed in haploid individuals have sex-specific effects on their fitness (direct haploid effects) and compare this to a model in which alleles expressed in haploid mothers have sex-specific effects on offspring fitness ('madumnal' effects). We use *madumnal effect* to refer to an effect of the genotype of a haploid mother on the offspring to distinguish such effects from a *maternal effect*, an effect of a diploid mother's genotype on offspring. There are two main motivations for understanding the evolutionary dynamics and outcomes of sexual antagonism for these different modes of gene action: first, because the underlying genetics are known to influence the evolutionary dynamics and outcomes under sex-specific selection (e.g. Rice 1984); and second, because empirical measures of naturally occurring variation due to sexual antagonism are informed by these predictions (e.g. Foerster *et al.* 2007). We consider models of haploid inheritance because many organisms have life cycles with a dominant haploid phase (Haig & Wilczek 2006).

2. DIRECT HAPLOID EFFECTS

In the diplohaplontic life cycle of plants, gametes are produced by haploid gametophytes, eggs and sperm unite to produce a diploid sporophyte that produces spores by meiosis, and spores germinate to produce gametophytes (figure 1). Consider a locus with two alleles A_1 and A_2. Let p_e and p_s be the frequency of A_1 in successful eggs and successful sperm and, since there are no fitness differences among diploid genotypes, let $p = (p_e + p_s)/2$ be the frequency of A_1 in the resulting sporophytes, spores and newly germinated gametophytes of the subsequent generation before selection. The A_1 gametophytes have male fitness w_{1m} and female fitness w_{1f}. A_2 gametophytes have male fitness w_{2m} and female fitness w_{2f}. After selection, the frequencies of A_1 among successful eggs and sperm of the next generation are

$$p'_e = p\frac{w_{1f}}{\bar{W}_f} = \left(\frac{p_e + p_s}{2}\right)\frac{w_{1f}}{\bar{W}_f}, \tag{2.1a}$$

$$p'_s = p\frac{w_{1m}}{\bar{W}_m} = \left(\frac{p_e + p_s}{2}\right)\frac{w_{1m}}{\bar{W}_m}, \tag{2.1b}$$

$$\begin{aligned}\bar{W}_f &= pw_{1f} + (1-p)w_{2f},\\ \bar{W}_m &= pw_{1m} + (1-p)w_{2m}.\end{aligned} \tag{2.1c}$$

The frequency of A_1 among spores of the next generation is

$$p' = \frac{1}{2}\left(p'_e + p'_s\right), \tag{2.2a}$$

$$\frac{p'}{p} = \frac{1}{2}\left(\frac{w_{1f}}{\bar{W}_f} + \frac{w_{1m}}{\bar{W}_m}\right). \tag{2.2b}$$

A_1 can invade a population fixed for A_2 if $p'/p > 1$ for p close to zero. That is,

$$\frac{w_{1f}}{w_{2f}} + \frac{w_{1m}}{w_{2m}} > 2. \tag{2.3}$$

Similarly, A_2 can invade a population fixed for A_1 if

$$\frac{w_{2f}}{w_{1f}} + \frac{w_{2m}}{w_{1m}} > 2. \tag{2.4}$$

We care about the stability (or instability) of these two monomorphic equilibria because genetic polymorphism exists if equations (2.3) and (2.4) are both satisfied.

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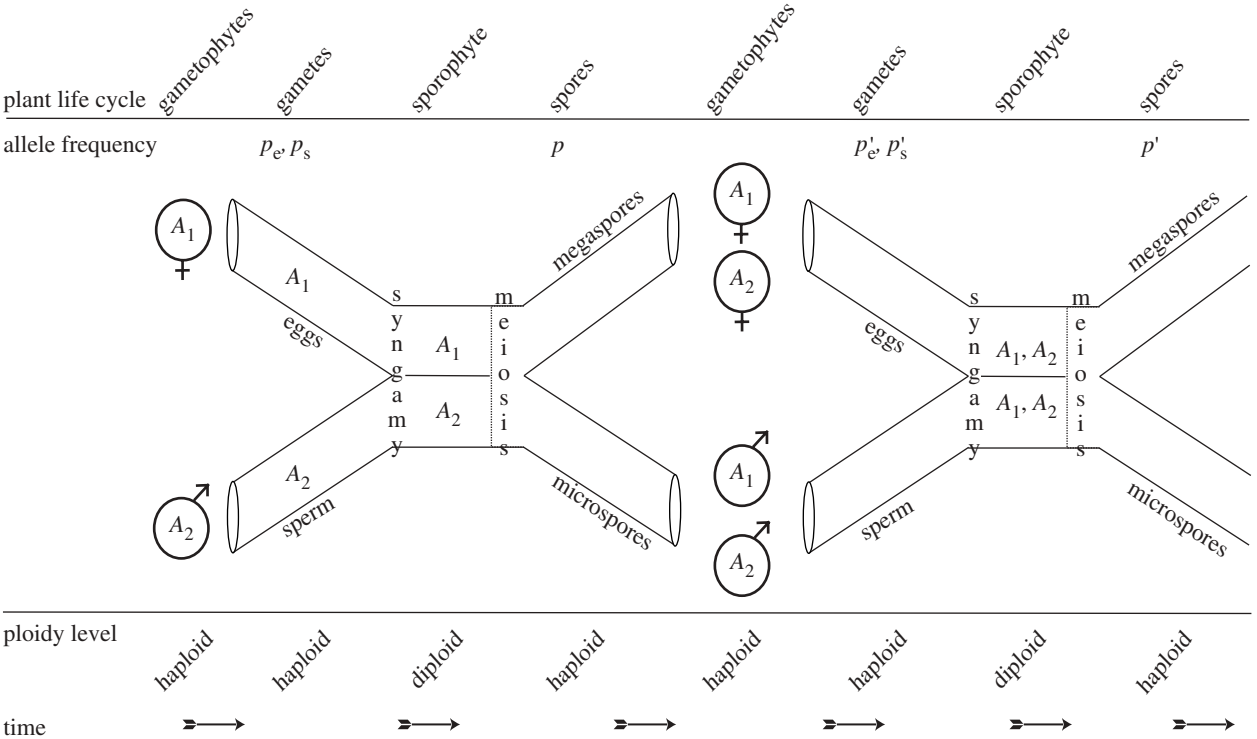

Figure 1. Plant life cycle and genetic transmission. The parental gametophytes have genotypes A_1 and A_2. Therefore, the sporophyte has an A_1A_2 genotype (madumnal allele written first) and offspring gametophytes will have either the A_1 or A_2 genotype. In the sexually antagonistic direct effect model, the fitness of an offspring depends on whether it has an A_1 or A_2 genotype and its sex. In the sexually antagonistic madumnal effect model, the genotype of the maternal gametophyte dictates the fitness of offspring gametophytes, regardless of the genotype of the offspring but still in a sex-specific fashion. In the direct effect model, the two male offspring gametophytes above would have different fitness, whereas in the madumnal effect model, they would have the same fitness by virtue of experiencing the same madumnal effect. Sexual antagonism for madumnal effects can be manifested in two manners: the madumnal genotype can differentially affect the viability/fertility of descendant gametophytes and it can cause the sporophyte to make differential investment in microspores and megaspores. Either way, the madumnal genotype causes varying fitness returns via male reproduction and female reproduction in the offspring gametophyte generation. Further still, the madumnal genotype can exert this control in two physiologically distinct manners: gene expression from the madumnal gametophyte can give rise to the downstream effects in sporophytes or offspring gametophytes mentioned before and imprinted expression in the diploid sporophyte of the maternally inherited allele can give rise to these same downstream effects.

3. MADUMNAL EFFECTS

Suppose instead that differential fitness of male and female gametophytes is determined by the madumnal genotype, i.e. the genotype of the female gametophyte in the preceding generation. A madumnal effect is an indirect effect of a gene expressed in the madumnal gametophyte on the viability or fertility of son and daughter gametophytes (figure 1). This model is general, permitting alternative interpretations of both the mode of gene action and how sexual antagonism for fitness is achieved, but we put off any discussion of equivalent formulations until later.

A_1 has frequency p_e among eggs and p_s among sperm of the parental gametophyte generation. After fertilization, a proportion (p_e) of sporophytes carry madumnal A_1 alleles; the remaining proportion of sporophytes ($1 - p_e$) carry madumnal A_2 alleles. The former class of sporophytes is associated with zygote-to-gametophyte-to-sperm fitness v_{1m} and zygote-to-gametophyte-to-egg fitness v_{1f}, whereas the latter class of sporophytes is associated with zygote-to-gametophyte-to-sperm fitness v_{2m} and zygote-to-gametophyte-to-egg fitness v_{2f}. After selection, the allele frequencies among the gametes of the offspring generation are

$$p'_e = \frac{p_e}{2}\left(\frac{v_{1f}}{\bar{V}_f}\right) + \frac{p_s}{2}, \tag{3.1a}$$

$$p'_s = \frac{p_e}{2}\left(\frac{v_{1m}}{\bar{V}_m}\right) + \frac{p_s}{2}, \tag{3.1b}$$

$$\bar{V}_f = p_e v_{1f} + (1 - p_e)v_{2f},$$
$$\bar{V}_m = p_e v_{1m} + (1 - p_e)v_{2m}. \tag{3.1c}$$

Comparisons of equations (2.1) and (3.1) reveal the fundamental difference between the madumnal effect model and the direct effect model. Half of the alleles in gametophytes are egg-derived and half are sperm-derived. Under the direct effect model, a gametophyte's fitness is determined by its own haploid genotype regardless of whether the allele is egg- or sperm-derived. In the madumnal effect model, only the 50 per cent of gametophytes with egg-derived alleles are subject to selection on the lingering effects of the allele they inherit, whereas the rest are subject to selection on the effects of an allele they do not inherit.

If gametes unite at random, these non-inherited alleles are a random sample from the egg pool. Therefore, there is no selective change in allele frequency among gametophytes that inherit sperm-derived alleles and the efficacy of selection is therefore diluted by half.

Formal analysis of the recursion equations (see electronic supplementary material) shows that A_1 can invade a population fixed for A_2 if

$$\frac{v_{1f}}{v_{2f}} + \frac{v_{1m}}{v_{2m}} > 2, \tag{3.2}$$

and A_2 can invade a population fixed for A_1 if

$$\frac{v_{2f}}{v_{1f}} + \frac{v_{2m}}{v_{1m}} > 2. \tag{3.3}$$

Therefore, the conditions for obtaining polymorphism are essentially the same under the haploid direct effect model and the madumnal effect model.

4. DISCUSSION

In the models presented above, a locus segregates for alleles with higher than average fitness for one sex and lower than average fitness for the other. Therefore, these are models of intralocus sexual conflict, also known as 'sexual antagonism' or 'intersexual ontogenetic conflict' (Rice & Chippindale 2001). Consistent with previous work, we find that such intralocus conflict can maintain genetic variation, especially when the sexual antagonism is strong (Prout 2000). Our two models are qualitatively similar except that selection is attenuated in the madumnal effect model because half of the gene pool (padumnal alleles) is shielded from selection. This is similar to the dilution of the force of selection for genes with maternal effects, the so-called 'relaxed constraint' (Barker *et al.* 2005).

Our madumnal effect model applies not only to organisms with dominant haploid life cycles but also to diploid organisms with imprinted gene expression. Though the site of gene expression differs for an imprinted gene of a sporophyte and a madumnal effect gene of a female gametophyte, the consequences of the two effects are genetically and evolutionarily indistinguishable. It is perhaps not surprising then that our madumnal effect model should show similar dynamics to the model by Pearce & Spencer (1992) of imprinted genes with sex-specific viabilities (see the electronic supplementary material).

Further, if the sex-specific fitness parameters are interpreted as fitness returns on investment in sons or daughters instead of as effects on the viability of haploid offspring, our madumnal effect model is relevant to the evolution of a gametophytic madumnal effect on sex allocation or of an imprinted sporophytic sex allocation locus. Imprinting of loci that influence sex allocation is a possibility if madumnal and padumnal alleles in the sporophyte have different optimal sex allocations (Haig 1992; Wild & West 2009). To give one example, pollen is often dispersed over greater distances than seeds. Therefore, the padumnal alleles of diploid sporophytes are more likely to be recent immigrants to the local population and are predicted to favour greater allocation to female function (seeds) than madumnal alleles, which are predicted to favour greater allocation to male function (pollen) to reduce local resource competition among related seedlings.

Polymorphism is possible at a madumnal effect locus under sexual antagonism (inequalities 3.2 and 3.3) but we do not expect such polymorphism to persist indefinitely in nature. Previous theory has examined how additional factors not considered in our simple model can lead to the erasure of genetic variation at sexually antagonistic direct effect loci. For instance, Rice (1986) shows that a mutant dominant sex-determining allele at one locus can spread by virtue of its linkage to a sexually antagonistic allele held in equilibrium at a second locus. This sex-determining allele is at an advantage because it is only found in the sex for which the linked allele is beneficial; the alternate allele at the sex-determining locus experiences the positive fitness effects of the linked allele in one sex just as often as it experiences the negative fitness effects in the other. This highlights a general theme that emerges from examination of intralocus sexual conflict resolutions. Sexual antagonism selects for non-random associations between the effects of genes and the sex of the bearer of those effects. This thinking has been applied to an examination of maternal effects, as well. Both Z- and W-linkage offer an avenue by which sexually antagonistic maternal effect genes can avoid their negative effects and experience their positive effects exclusively (Miller *et al.* 2006; Rice *et al.* 2008).

For a madumnal effect locus with sexually antagonistic viability effects in offspring gametophytes, we predict that a resolution will entail mothers acquiring the ability to bias sex allocation in favour of the sex for which their indirect effect is beneficial. For instance, if the sons of A_1 mothers have higher fitness than daughters, there will be selection for these mothers to have male biased broods. Any allele at a second locus that biases sex allocation to sons will be selected to come into linkage with the A_1 allele. Trivers & Willard (1973) were the first to discuss the logic behind sex allocation biases evolving in response to maternal properties. In their original formulation, maternal condition selects for biased allocation. We suggest extending the Trivers–Willard logic to the genetic properties of the mother and her indirect effects on offspring fitness.

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