Endosperm triploidy has a selective advantage during ongoing parental conflict by imprinting

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The endosperm of the flowering plant mediates the supply of maternal resources for embryogenesis. An endosperm formed in sexual reproduction between diploid parents is typically triploid, with a 2 : 1 ratio of maternal genetic material (denoted as 2m : 1p). Variation from this ratio affects endosperm size, indicating parent-specific expression of genes involved in endosperm growth and development. The presence of paternally or maternally imprinted genes can be explained by parental conflict over the transfer of nutrients from maternal to offspring tissue. Genomic imprinting can, for example, provide the male parent of an embryo in a mixed-paternity seed pod, with an opportunity for expressing its preference for a disproportionate allocation of resources to its embryo. It has been argued that a diploid 1m : 1p endosperm was ancestral and the 2m : 1p endosperm evolved after parental conflict, to improve maternal control over seed provisioning. We present a population genetic model, which instead places the origin of triploidy early in the parental conflict over resource allocation. We find that there is an advantage to having a triploid endosperm as the parental conflict continues. This advantage can help to explain why the 2m : 1p endosperm prevails among flowering plants.

Keywords: endosperm; triploidy; evolution; parental conflict; imprinting

1. INTRODUCTION

The triple fusion that results in the 2 maternal : 1 paternal (2m : 1p) primary endosperm nucleus was first reported in 1898 and was subsequently found in a large proportion of flowering plants (Vijayaraghavan & Prabhakar 1984). Because the 2m : 1p endosperm is widespread and disturbance of the genetic ratio upsets development, it has long been believed that an adaptive explanation should be sought for its unusual genetic constitution. Stebbins (1976) argued that all innovations that distinguish the reproductive process in flowering plants from other seed plants are associated with the shortening of time taken to mature seeds. In this context, triploidy is seen as an adaptation to encourage the rapid growth of the endosperm through heterozygote and polyploid vigour and the presence of more templates for protein synthesis. However, there is no reason why the 2m : 1p ratio should be crucial to accruing these advantages (Westoby & Rice 1982; Queller 1983; Haig & Westoby 1989).

What was missing from explanations of increased vigour was the careful consideration of inclusive fitness, which reveals the distinct and sometimes antagonistic interests of each genetically distinct tissue involved in seed provisioning (Charnov 1979; Queller 1983). Whenever the amount of parental investment in offspring is variable we should expect conflict between all genetically distinct tissue capable of influencing the level of investment. This expectation is an extension of ideas of parent-offspring conflict (Trivers 1974). In general (Queller 1989), disproportionate provisioning of the embryo inside a developing seed should be favoured by the associated female gametophyte, the endosperm, and the embryo itself. If there is at least some chance of mixed paternity among seeds raised

by the maternal plant, the male sporophyte should be added to this list. If reallocation of resources is inefficient, or the fitness of a seed depends nonlinearly on its provisioning, the female parent will favour uniform provisioning of all its offspring.

In the ancestors of flowering plants, the female gametophyte acted as the nurse tissue for the embryo. Explanations of endosperm evolution must address why the endosperm arose and displaced the gametophyte as nurse tissue and why the triploid 2m : 1p genetic configuration was favoured. Williams & Friedman (2002) have shown that diploid endosperms are common among early flowering plant lineages. Queller (1989) produced an inclusive fitness argument for how such a diploid endosperm may have succeeded the gametophyte. Suppose initially, both tissues shared the nursing role. Being genetically identical to the embryo the new diploid nurse tissue would be selected to increase acquisition of resources. The gametophyte's best interests would then be served by reducing its acquisitiveness. Eventually, the diploid endosperm would replace the gametophyte as the primary acquisitive tissue for the embryo. The subsequent doubling of the maternal contribution to the endosperm can be seen as a maternal adaptation to lower the acquisitiveness of the endosperm (Queller 1984). However, it is not clear why the triploid endosperm persists until lower acquisitiveness can be selected (Queller 1989). Two mechanisms have been proposed to explain the doubling of the maternal contribution, one driven by parent-offspring conflict, the other by parental conflict.

Hårdling & Nilsson (2001) present a parent-offspring argument. They suppose that endosperm amount (level of resource acquisition) is determined by a single allele; heterozygotes for this allele are assumed fittest, so an intermediately provisioned seed is most viable. They suppose that there is no dosage effect on doubling the maternal contribution to the endosperm. The allele for

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endosperm triploidy, therefore, has no effect on the fitness of seeds, but it is assumed to have an effect on the number of seeds set. Triploidy increases the number of low-provisioned homozygous seeds, but lowers the number of high-provisioned homozygous seeds. They argue that the former increases maternal fitness by more than the latter decreases it and hence triploidy is selectively advantageous. This argument has somewhat awkward assumptions. Optimum viability for intermediately provisioned seeds implies an unusual relationship between provisioning and fitness. The usual relationship is monotonically increasing with diminishing returns (Smith & Fretwell 1974; Parker & Macnair 1978). Härdling & Nilsson's (2001) assumptions are incompatible with Queller's (1989) argument for the diploid endosperm displacing the gametophyte, because increased aggression in the endosperm would not improve fitness.

Haig & Westoby (1989) present a parental conflict argument that makes use of parent-specific gene expression. If parents can modify the expression of genes responsible for endosperm acquisitiveness, then paternally derived genes would be selected for increased expression. This is particularly true if paternity amongst seeds developing on the maternal plant is very mixed. In any case, all seeds with increased acquisitiveness fathered by the same individual would have a more competitive endosperm. The costs would fall on embryos fathered by other individuals. Antagonistically, maternally derived genes for resource acquisition will be selected for decreased expression. The expectation over time, then, is that paternally derived genes will have high expression and maternally derived genes will be silenced. The advantage of triploidy consists of the presence of a second set of maternal genes reducing transcription resource availability for paternal genes. This would lower the acquisitiveness of the endosperm. The argument of Haig and Westoby can account for the presence of imprinted genes in the endosperm genome, and can also make successful predictions about the outcomes of some interploidy crosses (Haig & Westoby 1991; Vinkenoog *et al.* 2002). The assumption about restricted transcription resources is as yet untested, but can be circumvented by instead considering parental conflict enacted through the imprinting or silencing of both acquisition-promoting and acquisition-inhibiting genes active in the endosperm. Wilkins & Haig (2001) found that a model of this sort would predict that acquisition-promoting genes would be silent when maternally derived, and acquisition-inhibiting genes would be silent when paternally derived. Although their model was presented for a pair of imprinted genes in mammals, similar results should hold for imprinting in the endosperm.

Here, we present a model in which we consider the fate of a gene causing endosperm triploidy arising during ongoing parental conflict by imprinting of endosperm resource acquisition genes. The purpose of this modelling is to demonstrate that triploidy has a two-fold advantage. First, it is an alternative maternal response to additional paternal imprinting of acquisition-inhibiting genes. Second, once present in a population where triploidy is selectively neutral, the additional active maternal copies of acquisition-inhibiting genes prove an advantage during future invasions of paternally imprinted genes.

2. MODELLING

We will construct a population genetic model that enables us to consider the fate of paternally or maternally imprinted endosperm genes and genes causing endosperm triploidy. Our model requires assumptions about imprinting, population dynamics, competition for resources and the effects of polyploidy. These assumptions are discussed in detail here.

(a) *Characterization of imprinting*

Genomic imprinting is a mechanism that causes gene expression to vary according to its parental origin. Typically, an imprinted gene will be silent when inherited from one parent, but this is not always the case. We do not assume that imprinting can have only a downregulatory effect on expression. A maternally (paternally) imprinted gene is a gene that, when derived from the female (male) parent, carries an imprint or expression modifier. The precise mechanism for setting imprints in flowering plants is not known, although certain elements of the mechanism have been established (Adams *et al.* 2000; Vinkenoog *et al.* 2002). Barlow (1993) records a simple model for how parent-specific expression can be achieved. According to this model imprints are set during gametogenesis, when short sequences in the promoter region of a gene, imprinting boxes, attract an imprinting factor which modifies the gene and alters expression. The gene's imprinting boxes, attract an imprinting factor which modifies the gene and alters expression. The modification persists in somatic cells but is erased in early germ cells and reset according to the gender of the new individual. Imprinting boxes and factors must be different for maternally and paternally imprinted genes.

Our model will assume that new imprinted genes can arise when a gene acquires, through mutation, an imprinting box that attracts extant imprinting factors. A heterozygote for such a mutation will set the imprint in only those gametes that carry the mutation. An alternative hypothesis is that new imprints arise when mutant imprinting factors recognize new sections of promoter sequences. This can lead to a different pattern of inheritance, with all offspring of a heterozygote carrying imprints. This distinction is the same as that recognized by Spencer & Williams (1997) in their modifier locus models of genomic imprinting. This hypothesis was also explored by the authors with similar results to those presented here.

(b) *Life-history functions and basic model*

We will be considering a perennial hermaphrodite sexual ancestor species with no imprinting. We assume random mixing of gametes produces a fixed number of seeds per individual per reproductive season. The provisioning, s , of these seeds is normalized to 1. We introduce a function $f(s)$ that specifies the viability of a seed provisioned s . This function is usually assumed to be monotonically increasing and convex in the region of $s = 1$, that is increasing provisioning attracts diminishing returns. Convexity ensures that maternal fitness is optimized by uniform resource allocation. We suppose $f(0) = 0$, and for convenience that f is differentiable, so f is sigmoidal on $[0, 1]$. The form of f is shown in figure 1. Our species is iteroparous, so maternal fitness depends on a combination of

Figure 1. Illustrative functions: (a) seed viability; (b) parental mortality; (c) modification to provisioning, in this case an increase ($d_s \geq 0$) funded by contemporary siblings; and (d) modification to provisioning funded by maternal reserves. For paternal imprinting, d_s and d_m vary with frequency of the imprint in the population. For maternal imprinting, d_s and d_m vary with frequency of the imprint in the maternal genome.

present offspring viability and the likelihood of survival until future seasons. For this reason we must place conditions on the relationship of f and parental mortality.

Parental mortality will play a more prominent role here than is usual in population genetic models because we will be assuming that over-provisioned seeds may have access to maternal reserves not intended for reproduction this season. Because this reserve deficit affects all future offspring equally, it is equivalent to increased current mortality in the mother. We require a function, $m(\bar{s})$, that specifies the mortality rate per reproductive season of a plant producing seeds with average level of provisioning, $\bar{s}$. We suppose m is monotonic increasing and we want to impose conditions of m , so that $\bar{s} = 1$ gives an evolutionarily stable balance of offspring viability to parent mortality. To obtain these conditions we introduce a model for a diallelic locus affecting seed provisioning, with one allele dominant over the other. Suppose carriers of the dominant allele produce seeds provisioned s_1 and other individuals produce seeds provisioned s_0 . Let u , v and w be the frequency of homozygotes for the dominant allele, heterozygotes and homozygotes for the recessive allele, respectively. We assume weak selection so the rates of change of each type are given as follows:

$$\begin{aligned} \frac{du}{dt} &= \frac{M(u, v, w) f(s_1)(u^2 + uv + v^2/4)}{F(u, u, w)} - m(s_1)u, \\ \frac{dv}{dt} &= \frac{M(u, v, w) f(s_1)(uv + 2uw + v^2/2 + vw)}{F(u, u, w)} - m(s_1)v, \\ \frac{dw}{dt} &= \frac{M(u, v, w) f(s_0)(v^2/4 + vw + w^2)}{F(u, u, w)} - m(s_0)w, \end{aligned} \quad (2.1)$$

where

$$\begin{aligned} F(u, v, w) &= f(s_1)(1 - (1 - u - v/2)^2) + f(s_0)(1 - u - v/2)^2, \\ M(u, v, w) &= m(s_1)(u + v) + m(s_0)w. \end{aligned}$$

Implicit in the formulation of this model is the assumption that any change in mortality of individuals is not substantial enough to destabilize the population. This is consistent with individuals producing a large quantity of offspring, only a few of whom will reach maturity. The change in mortality simply accelerates or decelerates the turnover of individuals. This is why the average mortality, M , modifies the rate of growth for each type. Because mortality is usually assumed constant and unitary in population genetic models, this term rarely appears.

The variables u , v and w are not linearly independent and the model can be written in terms of variables $p = u + v/2$, the frequency of the dominant allele, and v . Hence we obtain

$$\begin{aligned} \frac{dp}{dt} &= p \left(\frac{M(p, v) f(s_1)}{F(p)} - m(s_1) \right), \\ \frac{dv}{dt} &= \frac{M(p, v) f(s_1) 2p(1 - p)}{F(p)} - m(s_1)v, \end{aligned} \quad (2.2)$$

where

$$\begin{aligned} F(p) &= f(s_1)(p^2 + 2p(1 - p)) + f(s_0)(1 - p)^2, \\ M(p, v) &= m(s_1)(p + v/2) + m(s_0)(1 - p - v/2). \end{aligned}$$

Now p and v are independent and our region of interest, Δ , in (p, v) -space is the triangle with vertices at $(0, 0)$, $(0.5, 1)$ and $(1, 0)$. The region Δ is invariant under the flow given above. Clearly $(0, 0)$ and $(1, 0)$ are equilibria. By linearizing about $(0, 0)$ we find eigenvalues $-m(s_1)$ and $m(s_0)f(s_1)/f(s_0) - m(s_1)$. The former indicates that the v -axis is always a stable manifold, so stability of $(0, 0)$ is determined by the sign of the latter eigenvalue. If $s_0 = 1$ the dominant allele cannot invade if $m(s_1)f(1) > m(1)f(s_1)$. The same condition is obtained if we insist recessive mutants cannot invade. So for $\bar{s} = 1$ to be evolutionarily stable we must have

$$m'(1) = \frac{f'(1)m(1)}{f(1)} \quad \text{and} \quad m''(1) > \frac{f''(1)m(1)}{f(1)}.$$

It is reasonable to adjust the time-scale in our models so that $m(1) = 1$. An appropriate m is shown in figure 1.

At this point we have developed a basic model that can describe the population genetics of non-imprinted genes that modify seed provisioning. To incorporate imprinted genes we must consider how imprinting can alter the balance of maternal provisioning of seeds on the mother plant.

(c) Provisioning functions

Endosperm can exercise a degree of control over their acquisitiveness, deploying haustoria which tunnel into maternal tissue to obtain nutrients (Johri & Ambegaokar 1984, pp. 23–28). Imprinting endosperm resource-acquisition genes will increase or decrease acquisitiveness in the carrier; this will either consume or free up additional resources. In the case of increased acquisition, consuming additional resources must either deprive contemporary siblings or the mother. The first case has been modelled by Law & Cannings (1984) and the second by Queller (1984).

For a general model we intend to incorporate both these components of endosperm over-consumption.

We consider first, the modified provisioning of seed with a paternally imprinted endosperm acquisitiveness gene (henceforth paternally imprinted seed) when over-consuming seeds deprive only contemporary siblings of resources. Such a seed is formed from pollen carrying an imprinted endosperm acquisitiveness gene (henceforth imprinted pollen). We assume that before the imprint arising, all seeds in the pod are provisioned s_0 . We let $s_0 + d_s(x)$ be the provisioning of a paternally imprinted seed, where d_s is small and dependent on the proportion of pollen that is imprinted, x . The bearers of the imprint will be over-consumers if $d_s(x) > 0$, and under-consumers if $d_s(x) < 0$. We fix $d_s(0) = \delta$ and $d_s(1) = 0$, because when $x = 1$, all seeds compete equally for a fixed amount of resources. We suppose d_s is monotonic. If no resources are lost by reallocation then we expect average provisioning of seeds to remain constant for $x \in [0, 1]$. The provisioning of a seed that is not paternally imprinted is therefore $s_0 - xd_s(x)/(1-x)$. d_s is likely to have $(1-x)$ as a factor, for example, a simple linear model requires $d_s(x) = \delta(1-x)$. If we assume that resources are acquired by the endosperm at a constant rate over a period of time, we obtain a concave d_s ; an example for $d_s \geq 0$ is illustrated in figure 1. We can improve the flexibility of this model by adding a parameter, e , describing the efficiency with which resources are reallocated to or away from paternally imprinted seeds. The provisioning of a seed without the paternal imprint is then

$$s_0 - \frac{xd_s(x)}{(1-x)e}.$$

We suppose that reallocation can incur only losses and not gains for total seed provisioning. Thus if $d_s > 0$, then $e \leq 1$, but if $d_s < 0$, then $e \geq 1$. Law & Cannings (1984) chose to have completely independent fitness functions for over-consuming and under-consuming seeds. We can obtain this level of flexibility by allowing e to vary with x , although our conditions of e constitute a restriction that they did not impose.

We now add a component derived from additional maternal resources. We define $d_m(x)$ which describes the modification of the provisioning of a paternally imprinted seed when the proportion of imprinted pollen is x . We fix $d_m(0) = \delta_0$ and $d_m(1) = \delta_1$, and assume d_m is monotone. In the case of over-consumption ($d_m > 0$) we assume also that

$$d'_m(x) > \frac{-d_m(x)}{x} \text{ for all } x \in [0, 1].$$

This ensures that average seed provisioning increases with x . For under-consumption this inequality must be reversed, so that average seed provisioning decreases with x . An appropriate over-consumption function is illustrated in figure 1. Combining the two components, we give the provisioning of a paternally imprinted seed as

$$s_0 + d_s(x) + d_m(x).$$

We can split the modified provisioning of seed with a maternally imprinted endosperm acquisitiveness gene (henceforth maternally imprinted seed) in exactly the same way. By our assumptions, a newly arising maternal imprint should be passed on to half of the offspring on the maternal plant. So a heterozygote for the mutation causing the imprint will provision maternally imprinted seeds $s_0 + d_s(1/2) + d_m(1/2)$ and other seeds $s_0 - d_s(1/2)/e$. Homozygotes for the

mutation will pass it to all offspring; their provisioning will be $s_0 + d_m(1)$.

At this point we have completed the preparation that enables us to model the fate of newly arising paternally or maternally imprinted endosperm acquisitiveness genes. These models are provided in electronic Appendix A. The models produce results that corroborate those of Wilkins & Haig (2001): that paternal imprints will be selected only if they increase endosperm acquisitiveness, and maternal imprints will be selected only if they bring provisioning closer to the maternal optimum. If we equate imprinting with silencing, genes that are paternally imprinted must be genes whose activity inhibits endosperm acquisitiveness. Similarly, genes that are maternally imprinted must be genes whose activity promotes endosperm acquisitiveness. Henceforth, we do not consider imprints that would not be selected.

(d) *Triploidy and dosage*

The basic model (equation (2.2)) is sufficient to demonstrate that the addition of a second haploid maternal genetic complement to the endosperm would be selected for if it could bring average seed provisioning closer to the maternal optimum. In the early stages of parental conflict by imprinting, the haploid genomes of embryo sac nuclei should feature a few maternally imprinted genes that promote endosperm acquisitiveness, and the full complement of active genes that inhibit endosperm acquisitiveness. If some or all of these inhibition genes have a cumulative effect with increased dosage, then we would expect an additional nucleus to engender a surfeit of gene products that inhibit endosperm acquisitiveness. It is not unreasonable to suppose that the imprinted genes in the endosperm have cumulative effects with dosage, because if they did not, the 2m:1p ratio would not be so critical. If average seed provisioning is initially above the maternal optimum, additional nuclei could prove advantageous by helping to reduce provisioning. In most species of flowering plants today the addition or loss of an entire haploid genetic complement is disastrous for endosperm development (Scott *et al.* 1998). For this reason triploidy must arise at an early stage in the parental conflict.

Suppose triploid endosperm individuals from a population where $\bar{s} \geq 1$ arrive in a population where a diploid endosperm prevails, and similarly $\bar{s} \geq 1$. In this case there will be no immediate selective advantage to triploidy. If a novel paternal imprint arises, triploid endosperm carriers of the new imprint have two active maternal copies of the imprinted acquisition-inhibiting gene. Seeds with diploid endosperm carry only one. As long as these genes have a cumulative effect with dosage, the acquisitiveness of the triploid endosperm will be less enhanced than that of the diploid endosperm. In the following model, we consider carriers of a novel paternal imprint with diploid endosperm are provisioned $s_0 + d_{2,s}(x) + d_{2,m}(x)$ and carriers with triploid endosperm are provisioned $s_0 + d_{3,s}(x) + d_{3,m}(x)$. Because we expect the triploid endosperm to be less acquisitive we assume

$$d_{2,s} \geq d_{3,s} \geq 0 \text{ and } d_{2,m} \geq d_{3,m} \geq 0.$$

(e) *Endosperm triploidy model*

We model the effect the arrival of a novel paternal imprint has on the frequency, y , of a dominant allele for endosperm triploidy in a population mixed for endosperm ploidy. The frequency of the mutation that causes the novel paternal

Table 1. Life-history parameters and abbreviations for individuals with diploid ($i = 2$) and triploid ($i = 3$) endosperm. (PI, seeds carry the novel paternal imprint; NPI, seeds do not.)

PI-seed provisioning	—	$s_0 + d_{i,s}(x) + d_{i,m}(x)$
NPI-seed provisioning	—	$s_0 - x d_{i,s}(x)/(1-x)e$
viability of PI-seed	$f_i^+(x)$	$f(s_0 + d_{i,s}(x) + d_{i,m}(x))$
viability of NPI-seed	$f_i^-(x)$	$f(s_0 - x d_{i,s}(x)/(1-x)e)$
parental mortality	$m_i(x)$	$m(s_0 + d_{i,m}(x)x)$

imprint to be set is given by x . The life-history parameters of the model are specified in table 1.

To describe the population dynamics of the dominant allele for endosperm triploidy we reprise the basic model (equation (2.2)). As with the basic model, we require the variable, v , describing heterozygote frequency for the triploidy allele. The only difference from the basic model is that average mortality, M , average viability, F , and the viabilities of offspring of carriers of the triploidy allele must now depend on the frequency of the paternal imprint. In place of the offspring viability ($f(s_1)$ in the basic model) we now require the average offspring viability, F_3 , of triploidy carriers with and without the paternal imprint. Similarly, we define F_2 , the average offspring viability of individuals not carrying the triploidy allele. $F_3(x) = f_3^+(x)x + f_3^-(x)(1-x)$, $F_2(x) = f_2^+(x)x + f_2^-(x)(1-x)$, $F(x, y) = F_3(x)y(2-y) + F_2(x)(1-y)^2$, $M(x, y, v) = m_3(x)(y + v/2) + m_2(x)(1-y - v/2)$. (2.3)

We now construct the population dynamics for the paternal imprint. Consider the diploid and triploid populations separately. Seeds homozygous for the mutation must have a paternal imprint and so have viability $f^+(x)$; these seeds occur with frequency x^2 . Seeds heterozygous for the mutation have a 50% chance of carrying the paternal imprint and so viability is, on average, $(f^+(x) + f^-(x))/2$. Heterozygotes occur with frequency $2x(1-x)$. Consequently, the average viability experienced by the mutation is

$$f^+(x)x^2 + \left(\frac{f^+(x) + f^-(x)}{2}\right)x(1-x) = \frac{x}{2}(f^+(x) + f^+(x)x + f^-(x)(1-x)).$$

Now combining both diploid and triploid populations, the average mutant viability is

$$\frac{x}{2}(f_3^+(x)y(2-y) + f_2^+(x)(1-y)^2 + F(x, y)).$$

We define

$$F^+(x, y) = f_3^+(x)y(2-y) + f_2^+(x)(1-y)^2.$$

The same frequency of paternal imprints is found in all individuals in the population and so the mutation experiences the average mortality, M . These considerations allow the introduction of paternal imprinting into the basic model as follows:

$$\begin{aligned} \frac{dx}{dt} &= \frac{M(x, y, v)x}{2} \left(\frac{F^+(x, y)}{F(x, y)} - 1 \right), \\ \frac{dy}{dt} &= y \left(\frac{M(x, y, v)F_3(x)}{F(x, y)} - m_3(x) \right), \\ \frac{dv}{dt} &= \frac{2M(x, y, v)F_3(x)y(1-y)}{F(x, y)} - m_3(x)v. \end{aligned} \quad (2.4)$$

This model has equilibria $E_0 = \{(0, y, 2y(1-y)) | y \in [0, 1]\}$, which is consistent with triploidy having no advantage before the paternal imprint arising. We can show the following inequality:

$$\frac{f_2^+(x)}{F_2(x)} \geq \frac{F^+(x, y)}{F(x, y)} \geq \frac{f_3^+(x)}{F_3(x)} \geq 1 \quad \text{for all } y \in [0, 1].$$

This indicates that the imprinting mutation would be selected more strongly in a diploid population than a triploid population, and for a mixed population the strength of selection will be intermediate. Thus we know $\dot{x} > 0$ on $[0, 1]$, so the paternal imprint must fix.

If $s_0 \geq 1$, the dynamics of the triploidy allele are straightforward, because the relationship of m and f requires

$$M(x, y, v)F_3(x) \geq m_3(x)F(x, y),$$

thus y is monotonically increasing on any trajectory from $x = 0$ to $x = 1$. The triploidy allele will fix only if $d_{3,m} > 0$, otherwise

$$M(1, y, v) = m_3(1) = m(s_0) \quad \text{and} \quad F(1, y) = F_3(1) = f(s_0) \quad \text{for all } y, v \in [0, 1].$$

In which case $E_1 = \{(1, y, 2y(1-y)) | y \in [0, 1]\}$ are equilibria. If $s_0 < 1$, the triploidy allele may be selected against for some or all of a trajectory from $x = 0$ to $x = 1$.

3. DISCUSSION

We have shown that an episode of paternal imprinting of endosperm acquisitiveness genes will favour an allele causing endosperm triploidy, as long as seed provisioning is initially at or above the maternal optimum. A selective advantage is available regardless of whether resources for overgrowth come predominantly from contemporary siblings or from maternal reserves. The triploidy allele will only fix, however, if some overgrowth depends on maternal reserves; this distinction is illustrated in the plots in figure 2. Haig & Westoby (1989) developed the original argument that genomic imprinting in the endosperm is the result of parental conflict, and that the second female contribution constitutes an escalation of this conflict. This theory allows endosperm triploidy a selective advantage as a mechanism of retaliation against paternal imprinting.

Figure 2. The selective advantage of endosperm triploidy during the fixing of a paternal imprint. Individuals carrying the paternal imprint may derive additional resources from contemporary siblings or from maternal reserves. Dashed lines, paternal imprint; solid lines, triploidy allele. (a) The short-lived advantage of triploidy when all additional resources are obtained from siblings. (b) Triploidy fixes when some additional resources are obtained from maternal reserves.

Our argument demonstrates that even if endosperm triploidy is initially selectively neutral it will attract an advantage during the fixing of future paternal imprints. This latent advantage can contribute to explaining why endosperm triploidy became widespread when it arose and why it has rarely been displaced by alternative endosperm types. The argument depends explicitly on paternal and maternal imprints affecting antagonistic endosperm development genes, and so is not derivable from a model that considers either acquisitiveness-promoting or acquisitiveness-inhibiting genes alone. The advantage to the mother of producing 2m : 1p endosperm over 1m : 1p endosperm is readily predicted by inclusive fitness theory (Queller 1989; Haig 1997). The additional maternal genetic material increases the endosperm's relatedness to siblings and so brings its interests into closer alignment with maternal interests. Effectively this improves maternal control over endosperm acquisitiveness, and in our model this proves advantageous when paternal imprints arise. Improved maternal control over seed provisioning is also responsible for the selective advantage to endosperm triploidy that Härdling & Nilsson (2001) found. Our model, however, is more general because it does not depend on the optimum endosperm acquisitiveness being maintained by heterozygote advantage.

The story of endosperm evolution is not complete and may yet require components that parental conflict arguments cannot provide. In particular, parental conflict is unlikely to be able to explain why additional paternal nuclei have not been introduced to the endosperm. This question cannot be resolved within the scope of the models presented here. Experimental 2x × 4x crosses yield a 2m : 2p endosperm whose seed is unviable in most species (Vinkenoog *et al.* 2002), suggesting that currently it is too late for a male nucleus to be added. In the end it may be that physiological constraints prevented a tetraploid endosperm arising before imprinting strength rose above a prohibitive threshold.

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