The Population Dynamics of Maternal-Effect Selfish Genes

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ABSTRACT

We use population genetic methods to describe the expected population dynamics of the selfish-gene chromosomal factor, *Medea* (maternal-effect dominant embryonic arrest), recently discovered in flour beetles, genus *Tribolium*. In the absence of deleterious effects on gross fecundity, *Medea* factors spread to fixation for all degrees of maternal-effect lethality greater than zero and the rate of spread is proportional to the strength of the maternal-effect. The rate of spread when rare is very slow, on the order of the frequency squared p^2, but this can be accelerated to order p when there is density regulation at the level of families as is known to occur for some genetic strains of flour beetles. When there are general deleterious effects of *Medea* on fecundity, affecting all offspring genotypes in addition to the genotype-specific maternal effect, then a stable interior polymorphism is possible. The location of the interior equilibrium and the probability of loss or fixation are sensitive to the degree of dominance of these fecundity effects.

BEEMAN *et al.* (1992) reported the discovery of a unique class of maternal-effect, dominant lethal genetic factors widespread in natural populations of the flour beetle, *Tribolium castaneum*. They called the factor *Medea*, an acronym for Maternal-effect dominant embryonic arrest. These factors are aptly labelled "selfish genes" because they self-select by maternal-effect lethality of all offspring not inheriting either a paternal or maternal copy of the factor. The purpose of this report is to describe the theoretically expected population dynamics for the spread of *Medea* factors through a population in light of the known biology of flour beetles and the Mendelian transmission of *Medea* factors (BEEMAN *et al.* 1992).

We will investigate two possible phenotypic effects of *Medea*: (1) *Medea* females have a lowered fecundity *independent of the maternal-effect lethality*; and, (2) variable degrees of the maternal-effect lethality. Our results show that *Medea* must be nearly completely recessive in its effects on maternal fecundity (effect 1) in order to spread through a population. In addition, when homozygous *Medea* females have a lowered fecundity, a stable polymorphism is possible. We further show that the rate of increase in the frequency, p, of *Medea* when rare, is very slow, on the order of p^2. However, our model shows that the subdivision of a population into density-regulated families enhances the spread of *Medea* considerably and changes the rate of spread when rare to order p. By "density regulation," we mean the kind of population structure often referred to as "soft selection" (*e.g.*, WADE 1985; KELLY 1992). In some genetic strains of *T. castaneum*, this kind of local density regulation has been documented in laboratory populations (*cf.* Figure 3 in MCCAULEY and WADE 1980). We illustrate the dynamics of the spread by partitioning the net change in *Medea* frequency into within and between family components (WADE 1979, 1980, 1982, 1985; BREDEN and WADE 1991; KELLY 1992).

The population dynamics of *Medea* are unlike those of the so-called "extreme selfish" genetic elements, such as the supernumerary B chromosome, "paternal sex ratio" (PSR), in the parasitoid wasp *Nasonia vitripennis* (WERREN *et al.* 1987; BEUKEBOOM and WERREN 1992; WERREN and BEUKEBOOM 1993). Population size does not diminish continually with the spread of *Medea* as it does with PSR. In addition, local density regulation retards the spread of PSR (BEUKEBOOM and WERREN 1992; WERREN and BEUKEBOOM 1993) whereas it accelerates the spread of *Medea* (see below). The population dynamics are also unlike those of the maternally inherited cytoplasmic organisms, *Wolbachia pipiens*, that cause partial reproductive isolation in a number of arthropods (STEVENS and WADE 1992; WADE and STEVENS 1994).

THE MODEL

Let G_{MM}, G_{M+}, and G_{++} be the frequencies of the three *Medea* genotypes so that $(G_{MM} + G_{M+} + G_{++})$ equals 1. Let D_{MM}, D_{M+}, and D_{++}, and S_{MM}, S_{M+}, and S_{++} be the frequencies of the three *Medea* genotypes in dams (D) and sires (S), respectively. The frequency of *Medea* factor within females is $p_D = (D_{MM} + [D_{M+}]/2)$ and similarly for p_S, the frequency within males. Although phenotypically *Medea* is a maternal-effect dominant, it is inherited as an autosomal Mendelian gene by both sexes (BEEMAN *et al.* 1992). We will assume in much of our analysis that the genotype frequencies in males and females are equal because, in the absence of sex-specific selection, deviations from equality are erased by a single

TABLE 1

Mating-types, family fitnesses, family frequencies, offspring genotypes, and offspring fitnesses

Family	Mating types		Frequency	Female fecundity	Offspring genotypes		
	Sire	Dam			*MM*	*M+*	*++*
1	*MM*	*MM*	$S_{MM}D_{MM}$	$(1 - s)$	1.0		
2	*M+*	*MM*	$S_{M+}D_{MM}$	$(1 - s)$	0.5	0.5	
3	*++*	*MM*	$S_{++}D_{MM}$	$(1 - s)$		1.0	
4	*MM*	*M+*	$S_{MM}D_{M+}$	$(1 - hs)$	0.5	0.5	
5	*M+*	*M+*	$S_{M+}D_{M+}$	$(1 - hs)$	0.25	0.5	$0.25(1 - t)$
6	*++*	*M+*	$S_{++}D_{M+}$	$(1 - hs)$		0.5	$0.5(1 - t)$
7	*MM*	*++*	$S_{MM}D_{++}$	1		1.0	
8	*M+*	*++*	$S_{M+}D_{++}$	1		0.5	0.5
9	*++*	*++*	$S_{++}D_{++}$	1			1.0

The mating-type frequencies are the product of the genotypic frequencies of sires (S) and dams (D). The parameter s specifies the degree that *Medea* might reduce female fecundity *independent of the maternal-effect lethality*. Dominance of *Medea* in its effect on female fecundity is defined by h, where $h = 0$ indicates completely recessive and $h = 1$ is completely dominant. The extent of the maternal-effect lethality is modeled by the parameter t. When $t = 1$, there is complete lethality and partial lethality is given by $0 < t < 1$.

episode of reproduction and the frequencies become equal in the two sexes (*cf.* Equation 2 below). However, we will derive first the more general transition equations with the sex-specific frequencies.

The frequencies of the different mating-types in the population and the genotypes of the offspring produced by each mating type are presented in Table 1. The family-specific fecundities, $(1 - s)$, $(1 - hs)$, and 1, are associated with the respective maternal genotypes, D_{MM}, D_{M+}, and D_{++}. This permits us to investigate the role of fitness costs ($0 < s < 1$) and benefits ($s < 0$) to the *Medea* factor on female fecundity or family size. The parameter h ($0 < h < 1$) permits us to modify the degree of dominance of the *Medea* factor in its effect on female fecundity. The parameter t lets us vary the degree of the maternal-effect lethality to normal (wild-type) homozygous offspring from complete lethality at $t = 1$, to normal survival at $t = 0$.

With this model, we find that the genotype frequencies in the offspring generation after selection (represented by a superscript prime) are

$$\begin{aligned} D'_{MM} &= S'_{MM} = (p_S)(p_D - sX)/W \\ D'_{M+} &= S'_{M+} = \{(1 - p_S)(p_D - sX) \\ &\qquad + (p_S)([1 - p_D] - [shD_{M+}/2])\}/W \\ D'_{++} &= S'_{++} = \{(1 - p_S)([1 - p_D] \\ &\qquad - [sh + t - tsh]D_{M+}/2)\}/W \end{aligned} \tag{1}$$

where X equals $(D_{MM} + [hD_{M+}/2])$ and mean fitness, W, equals

$$\begin{aligned} W = \{1 - sX - sh(D_{M+}/2) \\ - (D_{M+}/2)t[1 - sh][1 - p_S]\}. \end{aligned} \tag{2}$$

It is clear from Equation 1 that any gene frequency difference between males and females is eliminated after one generation. Hence, we set p_S and p_D equal to p, $(1 - p)$ equal to q, and use G_{MM}, G_{M+}, and G_{++} for the genotype frequencies.

The frequency of *Medea* in the offspring can be obtained using the weighted genotype frequencies from Equation 1. The change in the frequency of *Medea* from parent to offspring is given by $(p' - p)$ or

$$\Delta p = \{pG_{M+}(sh + 2qt[1 - sh]) - 2qsX\}/4W, \tag{3}$$

where W is equal to $\{1 - sp - [G_{M+}/2][tq(1 - sh) - s(1 - 2h)]\}$. Equations 1 and 3 with W give the exact theoretical dynamics of *Medea* across generations.

We can partition the change in gene frequency (Equation 3) into components of within-family and among-family selection (Wade 1979, 1980, 1982, 1985; Breden and Wade 1991; Kelly 1992). The genotype-specific lethality effects of *Medea* represent within-family selection against the non-*Medea* allele. This selection occurs only within families 5 and 6 of Table 1; there is no selection within the other seven families for or against *Medea*. The change in gene frequency caused by the within-family component of selection is

$$\Delta p_I = (t/8W)(1 - hs)(G_{M+})(q + [G_{M+}/2]). \tag{4}$$

Note that, whenever $t > 0$, this expression is greater than or equal to zero. Hence, within-family selection always favors the evolution of *Medea*. Note also that the rate of change by within-family selection is diminished by the factor $(1 - hs)$ so that *Medea* alleles with recessive effects on gross fecundity will spread faster than alleles with dominant effects.

The among-family component of selection is given by

$$\begin{aligned} \Delta p_A = &-(s/4W)\{2qG_{MM} + (q - p)hG_{M+}\} \\ &- (t/8W)(1 - hs)(G_{M+})\{q + [G_{M+}/2] - 4pq\}. \end{aligned} \tag{5}$$

Equations 4 and 5 sum to give Equation 3. The among-family component of selection is negative for two reasons: (1) whenever *Medea* reduces female fecundity

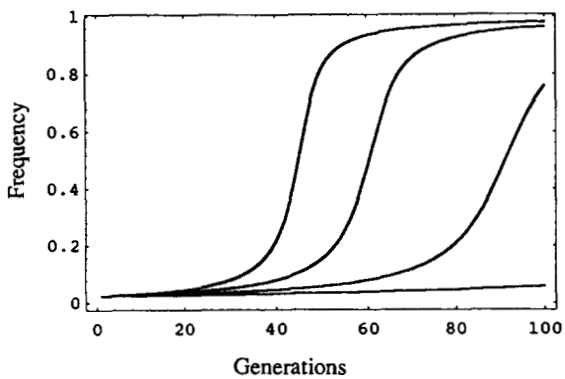


FIGURE 1.—Variation in the degree of maternal-effect lethality affects the change in the frequency of *Medea* factor ($h = 0$) through time when there is no fecundity effect ($s = 0$) other than that owing to maternal effect lethality. From left to right, the different curves correspond to t values of 1.00, 0.75, 0.50 and 0.00, respectively.

($s > 0$), families of *Medea* females are smaller than those of non-*Medea* females (*cf.* Table 1); and, (2) the maternal-effect of *Medea* reduces the size of families 5 and 6 (*cf.* Table 1) and, consequently, the average family size of *Medea* females. Because *Medea* reduces family size by embryonic lethality ($t > 0$), it is always opposed by among-family selection.

When there is no *Medea* effect (*i.e.*, $t = 0$) and the fecundity effective is completely recessive (*i.e.*, $h = 0$), then Equation 4 equals 0 and Equation 3 reduces to

$$\Delta p = -sp^2q/2W,$$

which is one-half the rate of gene frequency change in the standard population genetic model for selection against a recessive deleterious allele (CROW and KIMURA 1970, p. 182). The rate of gene frequency change is halved in Equation 3 relative to the standard rate because selection is among families rather than among genotypes or, equivalently, selection is acting in only one sex, the females.

We first examine several special cases of Equations 3, 4 and 5 in light of the known biology of *Medea*.

Normal fecundity of *Medea* females ($s = 0$): With normal fecundity of *Medea* females (BEEMAN *et al.* 1992), we set s equal to 0 in Equation 3 to obtain

$$\Delta p = \{pqG_{M+}t\}/2W, \quad (6)$$

where W becomes equal to $\{1 - qt(G_{M+}/2)\}$. It is clear from Equation 6 and Figure 1, that the rate of spread of *Medea* proportional to the size of t, the degree of maternal-effect lethality. More selfish *Medea* factors (*i.e.*, t close to 1) spread more rapidly than less selfish factors (*i.e.*, t close to 0). However, any degree of selfishness, no matter how small (*i.e.*, $t > 0$), will ensure ultimate fixation of *Medea* at least in large populations (Figure 1). Although among-family selection opposes the spread of *Medea* even in the absence of fecundity effects ($s = 0$), the strength of among-family selection is always less than that of within-family selection (compare Equations 4 and 5).

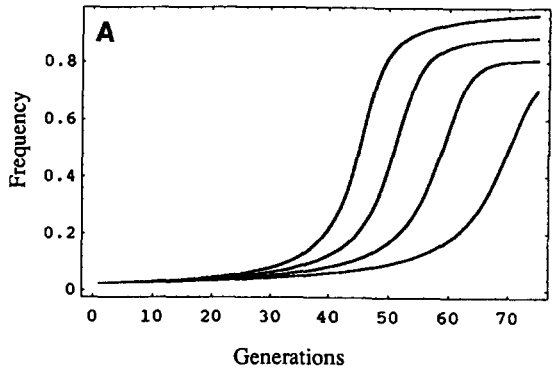
When *Medea* is rare as it might be upon first invading a population by mutation or migration, then homozygotes will be absent or nearly so ($G_{MM} = 0$). In this instance, ($G_{M+}/2$) is equal to p and Equation 6 becomes simply

$$\Delta p = \{p^2qt\}/W. \quad (7)$$

Thus, the initial spread of *Medea* factors will be very slow and of the order of p^2. At this rate, there is a fair chance that *Medea* will be lost in a finite population in the early stages of invasion by random genetic drift similar to selection for advantageous but recessive alleles (ROBERTSON 1978). With local density regulation of families, the rate of spread when rare is accelerated as we show in *Local density regulation within families* below.

Impaired fecundity of *Medea* females ($s > 0$): When *Medea* reduces fecundity an interior equilibrium frequency ($0 < p < 1$) is possible (Figure 2A). Under the assumptions of weak selection (small t and s) and recessive fecundity effect ($h = 0$), we can show analytically that the interior equilibrium is stable. Under these assumptions, W reduces to $\{1 - sp - (pq)(qt - s)\}$ and has a first derivative with respect to p equal to $\{-t + 2(2t - s)p - 3tp^2\}$. Setting the first derivative equal to 0, we can find two solutions for the equilibrium value of $\hat{p}$: (1) $3(2t - s)/6t$ and (2) $(2t - s)/6t$. Since the second derivative of W with respect to p is negative when $\hat{p} > 2(2t - s)/6t$, the first solution corresponds to a *stable* interior polymorphism. Although we assumed weak selection in order to substitute $(2pq)$ for $(G_{M+}/2)$ in the equation for W, we can see from Figure 2A that the approximate solution gives nearly the same values of the interior equilibria for stronger selection (t and s near 1). The equilibrium value for the frequency of *Medea* is fairly high even with a large fecundity effect, *i.e.*, s in the range of 0.50 to 0.75 as shown in Figure 2A. Comparing Figures 1 and 2A, it is clear that changes in t over the range between 0.25 and 0.75 have a greater effect on the rate of spread than recessive deleterious fecundity effects, s when $h = 0$, over the same range.

Partial dominance of the fecundity effects ($h = 0.2$) slows the rate of spread of *Medea* (Figure 2B) considerably. The degree of dominance can also affect whether *Medea* increases or is lost even with small effects on fecundity (Figure 3, $s = 0.25$). With a 25% loss in fecundity ($s = 0.25$), *Medea* increases whenever h is less than 0.46 but is lost when the degree of partial dominance exceeds 0.47. The critical value for dominance of fecundity effects, above which *Medea* is lost rather than is spread, depends in a complicated way on the value of several parameters in Equation 3, including t. When t is set equal to 0.75 and s equal to 0.50, a completely recessive *Medea* factor will spread but one with weak partial dominance of the fecundity effects ($h = 0.15$) will be lost (Figure 4).



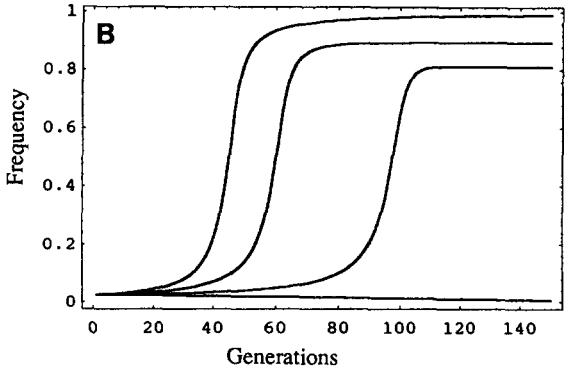


FIGURE 2.—(A) A recessive effect ($h = 0$) on overall fecundity changes the rate of spread of *Medea* ($t = 1.0$) through a population. From left to right, the different curves correspond to s values of 0.00, 0.25, 0.50 and 0.75, respectively. Note that changes in t (Figure 1) have a greater effect on the population dynamics than changes in s. (B) A partially dominant effect ($h = 0.05$) on overall fecundity changes the rate of spread of *Medea* ($t = 1.0$) through a population. From left to right, the different curves correspond to s values of 0.00, 0.25, 0.50, and 0.75, respectively. Note that changing h from 0.00 in (A) to 0.05, slows the rate of spread and, in the case of a strong fecundity effect ($s = 0.75$), leads to the loss of *Medea* instead of fixation. See text for further discussion.

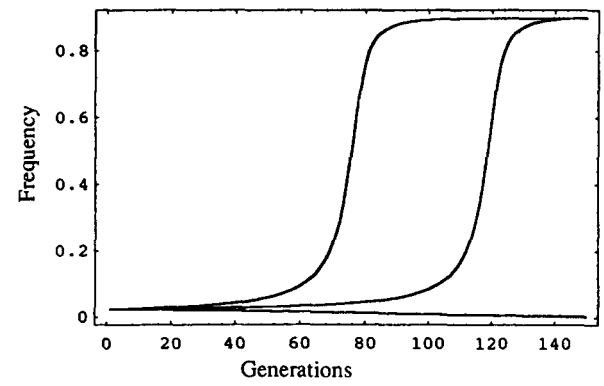


FIGURE 3.—Changes in the dominance of a reduction in fecundity ($s = 0.25$) for a *Medea* factor with strong maternal-effect lethality ($t = 1.0$), affect the rate of spread and the probability of fixation or loss. From left to right, the different curves correspond to h values of 0.10, 0.15 and 0.20, respectively.

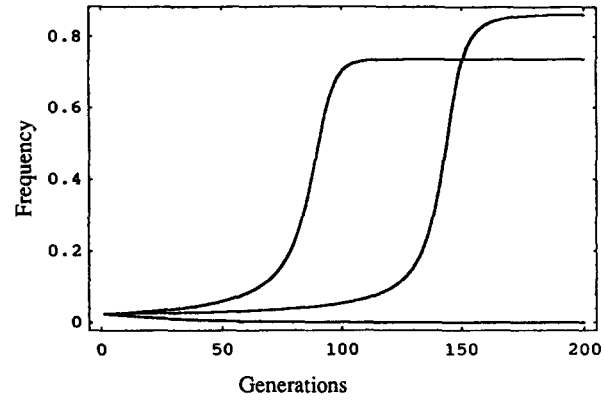


FIGURE 4.—Simultaneous changes in the magnitude of the fecundity effect, s, and its partial dominance change the rate of spread, the location of the stable interior equilibrium, and the probability of loss. From left to right, the different curves correspond to $\{s = 0.50, h = 0.0\}$, $\{s = 0.25, h = 0.10\}$ and $\{s = 0.50, h = 0.15\}$, respectively.

Local density regulation within families: We introduce local density regulation within families by postulating that the homozygous ++ genotypes in families 5 and 6 of Table 1 are replaced upon death by *MM* and *M*+ genotypes. If we divide the Mendelian expectations for the surviving genotypes by $(1 - G_{++}t)$, where G_{++} is 0.25 in family-type 5 and 0.50 in family-type 6, then we have introduced a form of parental compensation for the deaths of some offspring owing to *Medea* (Table 2). This is equivalent to family-level soft selection (WADE 1985; KELLY 1992), an ecological situation in which each family produces a very large number of offspring and a large fraction of them die for reasons of local density dependent competition. Differently put, despite the deaths owing to maternal-effect lethality, *Medea* females produce as many total surviving offspring as other females as a result of a reduction in "competitive" deaths owing to the preceeding "genetic" deaths. MCCAULEY and WADE (1980) have shown that, under controlled laboratory conditions, some genetic strains of *T. castaneum* exhibit this kind of local density regulation and that there is genetic variation for these density effects as well.

The net effect of these assumptions regarding local density dependence is to maintain constant fitness for all families (Table 2). Instead of *Medea* families 5 and 6 having a lower average fitness than other families (Table 1), they now have a fitness equivalent to any other non-*Medea* family. This enhances the rate of spread of the *Medea* factors, especially when rare, because the opposing effects of among-family selection are diminished with local density regulation.

To investigate the effects of this kind of density regulation on the initial rate of spread, we further assume that *Medea* is rare so that we can set $D_{MM} = S_{MM} = G_{MM} = 0$ as we did above. These assumptions change the model to the form illustrated in Table 2. Setting s equal to 0 and t equal 1, we have

$$\Delta p = p(3 - 2p)/6 \qquad (8)$$

TABLE 2

Mating-types, family fitnesses, family frequencies, offspring genotypes, and offspring fitnesses with local density regulation

Family	Mating types		Frequency	Female fecundity	Offspring genotypes		
	Sire	Dam			*MM*	*M+*	*++*
5	*M+*	*M+*	$S_{M+}D_{M+}$	1	0.25/*A*	0.5/*A*	0.25(1 − *t*)/*A*
6	*++*	*M+*	$S_{++}D_{M+}$	1		0.5/*B*	0.5(1 − *t*)/*B*
8	*M+*	*++*	$S_{M+}D_{++}$	1		0.5	0.5
9	*++*	*++*	$S_{++}D_{++}$	1			1.0

The mating-type frequencies are the product of the genotypic frequencies of sires (S) and dams (D). The extent of the maternal-effect lethality is modelled by the parameter t. When $t = 1$, there is complete lethality and partial lethality is given by $0 < t < 1$. Because of local density regulation, the genetic deaths of ++ homozygous offspring in families 5 and 6 change the mortality pattern due to density and family size remains constant; hence, A is equal to $(1 - 0.25t)$ and B is equal to $(1 - 0.5t)$. See text for further discussion.

which is approximately (to order p^2) equal to

$$\Delta p = 0.5p. \tag{9}$$

Thus, soft selection at the level of families increases the initial rate of spread of *Medea* from order p^2 to order p, accelerating the rate of spread when *Medea* is rare.

DISCUSSION

We have shown that the population dynamics of *Medea*, a Mendelian inherited selfish genetic element, depend upon four factors: (1) the degree of maternal-effect lethality $(1 - t)$; (2) the fecundity fitness cost (s) to females carrying *Medea*; (3) the degree of dominance (h) of the fecundity fitness cost in females; and, (4) the mode of population regulation (hard or soft family selection). In the absence of a fecundity cost ($s = 0$), any degree of the maternal-effect lethality ($0 < t < 1$) permits *Medea* to spread and the rate of spread is directly proportional to t (Equation 3); *i.e.*, *Medea* factors conferring partial lethality will spread more slowly than factors which result in complete lethality of homozygous wild-type offspring genotypes.

We also examined the effects of a cost to *Medea* females in terms of lowered fecundity ($s > 0$). The existence of other fitness costs or fecundity effects of *Medea* has not yet been demonstrated in flour beetles (BEEMAN *et al.* 1992). However, we have included such fitness effects in the model because they have been hypothesized or shown to characterize other selfish genes (*e.g.*, transposable elements) and other maternally transmitted cytoplasmic agents (*e.g.*, *W. pipiens*; WADE and STEVENS 1985; HOFFMAN and TURELLI 1988; STEVENS and WADE 1990). There is also much theoretical interest in the problem of the evolution of selfish genes when the intra-host effects favor their evolution but the inter-host effects oppose it. We find that, for *Medea*, the fecundity fitness effects to mothers must be recessive or nearly so in order to permit *Medea* to spread. It is clear that, if a female heterozygous for *Medea* lays a reduced number of eggs ($s > 0$) and then, in addition, kills a large fraction of her offspring ($t > 0$), her net contribution to the next generation will be greatly reduced. Despite the genotypic bias caused by killing only homozygous non-*Medea* ++ offspring, *Medea* will not spread if it severely affects the fecundity of heterozygous *M+* females in whose families the selfish gene advantage of maternal-effect lethality operates. This is owing to the opposing effects of among-family selection. In this case, the numbers of *MM* and *M+* offspring are reduced within these families as a result of additional (and as yet undiscovered) effects of the maternal *Medea* genotype. As a result, the strength of the selection against the normal + allele and in favor of the *M* allele is reduced because a further component of selection (selection between families) that affects *all* genotypes within the *Medea* families (family-types 5 and 6 of Table 1 and 2), is introduced.

When the fecundity effect is recessive ($h = 0$), however, *Medea* can spread even when s is near 1 for homozygous *Medea* females (Figure 2A). The maternal-effect lethality in the families of heterozygous females, *M+*, more than offsets the decrease in fecundity of homozygous *Medea* females, *MM*. (Note that there is no maternal-effect lethality operating in the families of *MM* females because every offspring receives at least one *M* allele from the mother.) Thus, the degree of dominance of any additional effects of *Medea* on maternal fecundity, beyond those affecting the ++ offspring, is important to the evolutionary fate of the *Medea* factor because dominance changes the balance between the opposing levels of within-family (favorable) and between-family (unfavorable) selection.

There is a stable interior equilibrium when there are additional and recessive fecundity effects of *Medea* (Figures 2–4). This may contribute to explaining the limited geographic distribution of the *Medea* factors described in BEEMAN *et al.* (1992). On the other hand, once a *Medea* factor has swept through a population and become fixed, there would be no way to detect it by test crosses because, by definition, it affects only the families of heterozygous *M+* females (Tables 1 and 2). Thus, the current geographic distribution may not reflect a stable equilibrium but rather a transient polymorphism on its way to complete fixation.

The initial rate of spread of *Medea* is very slow, on the order of p^2, without local density regulation at the level of families [*i.e.*, with hard selection (WADE 1985; KELLY

1992)]. However, if there is soft selection at the level of families, the rate of spread is increased to order p from p^2. Soft selection amounts to setting the mean fitness of all families equal to 1 despite the within-family selection by maternal-effect lethality of + + homozygous offspring. Soft selection means that all families produce equal numbers of offspring despite the genotypically biased mortality occurring within some families. The ecological model represented by soft selection is considered to be one of local density regulation at the level of the family (KELLY 1992). Differently put, it represents the case where each female lays a sufficiently large number of eggs that there is severe competition among her progeny. The numbers of offspring surviving this competition are independent of maternal genotype and other within-family selection events [*cf.* WADE (1985) and KELLY (1992) for further discussion]. The deaths owing to maternal-effect lethality within families of *M*+ females serve to reduce, but not eliminate, the ecological competition. The large geographic area spanned by *Medea* in natural populations of flour beetles indicates a rapid or efficient method of spread and lends credence to this kind of ecological model of local density regulation.

The evolutionary effects of within-family density regulation or soft selection on *Medea* factors can again be understood in terms of within and between family selection. Local density regulation eliminates the among-family variance in fitness that arises owing to the deaths of + + offspring in family-types 5 and 6. Without the among-family variance in fitness, the among-family selection which opposes the spread of *Medea* cannot operate. Hence, the overall rate of spread is accelerated.

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LITERATURE CITED

BEEMAN, R. W., K. S. FRIESEN and R. E. DENELL, 1992 Maternal-effect selfish genes in flour beetles. Science **256:** 89–92.

BEUKEBOOM, L. W., and J. H. WERREN, 1992 Population genetics of a parasitic chromosome: experimental analysis of PSR in subdivided populations. Evolution **46:** 1257–1268.

BREDEN, F., and M. J. WADE, 1991 "Runaway" social evolution: reinforcing selection for inbreeding and altruism. J. Theor. Biol. **153:** 323–337.

CROW, J. F., and M. KIMURA, 1970 *Introduction to Population Genetic Theory.* Harper & Row, New York.

HOFFMANN, A. A., and M. TURELLI, 1988 Unidirectional incompatibility in *Drosophila simulans*: inheritance, geographic variation and fitness effects. Genetics **119:** 435–444.

KELLY, J. K., 1992 Kin selection in density regulated populations. J. Theor. Biol. **157:** 447–461.

MCCAULEY, D. E., and M. J. WADE, 1980 Group selection: the genetic and demographic basis for the phenotypic differentiation of small populations. Evolution **34:** 813–821.

ROBERTSON, A., 1978 The time of detection of recessive visible genes in small populations. Genet. Res. **31:** 255–264.

STEVENS, L., and M. J. WADE, 1990 Cytoplasmically inherited reproductive incompatibility in Triboloium flour beetles: the rate of spread and effect on population size. Genetics **124:** 367–372.

WADE, M. J., 1979 The evolution of social interactions by family selection. Am. Nat. **113:** 399–417.

WADE, M. J., 1980 Kin selection: its components. Science **210:** 665–667.

WADE, M. J., 1982 Evolution of interference competition by individual, family, and group selection. Proc. Natl. Acad. Sci. USA **79:** 3575–3578.

WADE, M. J., 1985 Hard selection, soft selection, kin selection, and group selection. Am. Nat. **125:** 61–73.

WADE, M. J., and L. STEVENS, 1985 Microorganism mediated reproductive isolation in flour beetles (genus *Tribolium*). Science **227:** 527–528.

WADE, M. J., and L. STEVENS, 1994 The effect of population subdivision on the rate of spread of parasite-mediated cytoplasmic incompatibility. J. Theor. Biol. **167:** 81–88.

WERREN, J. H., and L. W. BEUKEBOOM, 1993 Population genetics of a parasitic chromosome: theoretical analysis of PSR in subdivided populations. Am. Nat. **142:** 224–241.

WERREN, J. H., U. NUR and D. EICKBUSH, 1987 Extrachromosomal factor causing loss of paternal chromosomes. Nature **327:** 75–76.

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