# Note

## Further Properties of Gavrilets' One-Locus Two-Allele Model of Maternal Selection

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ABSTRACT

I derive several properties of the model proposed by Gavrilets for maternal selection at a single diallelic locus. Most notably, (i) stable oscillations of genotype frequencies (*i.e.*, cycling) can occur and (ii) in the special case in which maternal effects and standard viability selection act multiplicatively, maternal selection effectively acts on maternally derived alleles only.

THE maternal environment is of crucial importance to the developing organism. This realization is long-standing for groups such as mammals, but more recently it has become clear that maternal effects profoundly influence embryonic development in many animals not exhibiting such a close relationship between mother and offspring, even those in which maternal care is completely absent (Rossiter 1996; Mousseau and Fox 1998; Wolf 2000). In a DNA microarray analysis of 4028 *Drosophila melanogaster* genes, for example, 1212 of the RNA transcripts found in the first hours of development were maternally deposited during oogenesis (Arbeitman *et al.* 2002). Nevertheless, all but 27 of these same genes were subsequently expressed from the embryo's own copies, and so much of their maternal effect is compounded by standard genetic expression (Arbeitman *et al.* 2002). Gavrilets (1998; see also Wade 1998) proposed a two-allele single-locus model that describes the population-genetic consequences of this pattern of gene expression when fitness differences exist among both the maternal and zygote's own gene products. In this note I derive several properties of this model and compare it with other population-genetic models of selection at a single locus.

**General model:** Gavrilets (1998) considered a single locus with two autosomal alleles, *A* and *a*, in a randomly mating, dioecious population, in which the effects of mutation and genetic drift are negligible. Let $i = 1$, 2, and 3 correspond to the genotypes *AA*, *Aa*, and *aa*, respectively, and suppose $w_{ij}$ is the fitness of individuals of genotype $i$ with genotype $j$ mothers. If $x$, $y$, and $z$ are the postselection frequencies of adults with respective genotypes *AA*, *Aa*, and *aa*, then the recursion equations for these frequencies in the following generation are

$$\begin{aligned}\overline{w}x' &= w_{11}px + \tfrac{1}{2}w_{12}py,\\ \overline{w}y' &= w_{23}pz + \tfrac{1}{2}w_{22}y + w_{21}qx,\\ \overline{w}z' &= \tfrac{1}{2}w_{32}qy + w_{33}qz,\end{aligned} \tag{1}$$

in which

$$\begin{aligned}p &= x + y/2,\\ q &= 1 - p,\end{aligned} \tag{2}$$

and $\overline{w}$, the population's mean fitness, is the sum of the right-hand sides of (1) so that the iterated frequencies ($x'$, $y'$, and $z'$) add to one. This normalization means that only the relative values of the fitness parameters, rather than their absolute values, need to be specified. This property, shared with most models of selection, means that there are just six independent parameters. Gavrilets (1998) wrote Equations 1 solely in terms of $x$, $y$, and $z$ (*i.e.*, without $p$ and $q$), but the above form reveals the paternal contribution (the $p$'s and $q$'s), which is useful when considering the special multiplicative case below. Equations 1 are not formally equivalent to those previously used to describe any other population genetic system. They are not, for example, equivalent to those describing fertility selection (Bodmer 1965; Hadeler and Liberman 1975), which has five independent parameters once the sex-symmetry property (Feldman *et al.* 1983) is noted, even though both systems are determined by iterations of genotype frequencies.

The model exhibits a number of interesting properties. As shown by Gavrilets (1998), distinct polymorphic equilibria can be simultaneously locally stable (see,

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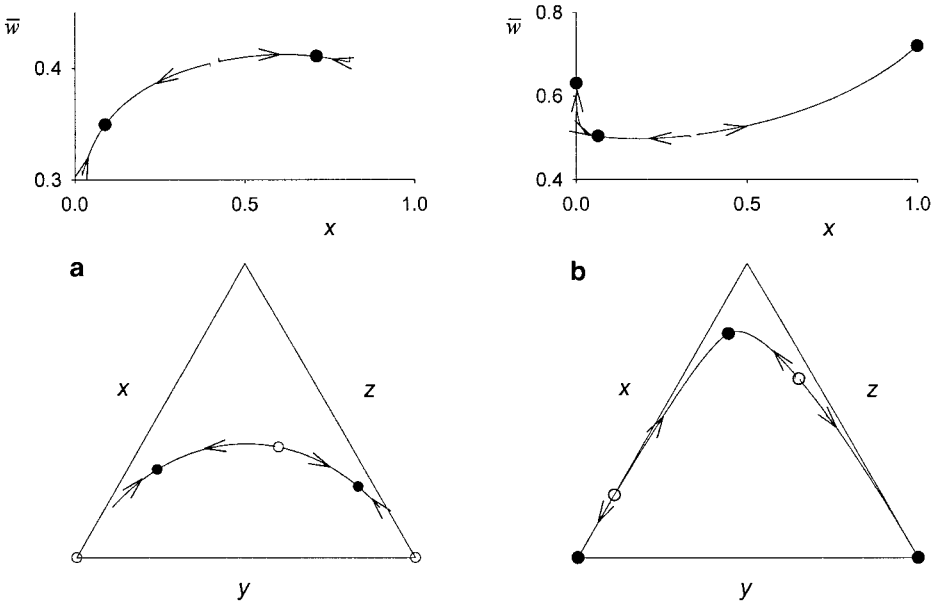


FIGURE 1.—Mean fitness (top graphs) and genotype-frequency dynamics (bottom graphs) for numerical examples of the general model: (a) $w_{11} = 0.385$, $w_{12} = 0.642$, $w_{21} = 0.306$, $w_{22} = 0.494$, $w_{23} = 0.068$, $w_{32} = 0.875$, and $w_{33} = 0.245$, which has two simultaneously locally stable polymorphic equilibria; (b) $w_{11} = 0.719$, $w_{12} = 0.070$, $w_{21} = 0.109$, $w_{22} = 0.943$, $w_{23} = 0.264$, $w_{32} = 0.128$, and $w_{33} = 0.630$, which has simultaneously stable fixations and a stable polymorphism. The genotype frequencies are plotted on ternary graphs, in which the frequency of a genotype increases the farther a point is from the axis labeled with that genotype. (The frequency of the genotype, therefore, is one at the apex opposite the labeled axis.) Mean fitness, $\bar{w}$, is plotted as a function of $x$ for the trajectories shown in the bottom graphs. Locally stable equilibria are shown as solid circles; unstable equilibria as open circles. Arrows indicate the direction of temporal change.

*e.g.*, Figure 1a). Nevertheless, these systems are rare in parameter space. I found <10 cases in $10^5$ sets of $w_{ij}$ values independently and uniformly sampled over the unit interval [0, 1]. Each of these sets was investigated for simultaneously stable equilibria by iterating from 100 randomly chosen initial genotype frequencies. The example shown in Figure 1a also shows that mean fitness, $\bar{w}$, need not increase over time and hence need not be at a local maximum at equilibrium. It is also possible, as noted by GAVRILETS (1998), for a parameter set to give rise to a locally stable polymorphic equilibrium while both fixations are also locally stable (see Figure 1b).

Perhaps the most surprising behavior is genotype-frequency cycling, although it is even rarer in parameter space than two stable polymorphisms: I found just 2 cases in $10^5$ parameter sets, with fitnesses chosen from a uniform distribution with the proviso that both fixations are unstable. In the example shown in Figure 2, $(x, y, z)$ alternates between (0.2769, 0.6315, 0.0916) and (0.5608, 0.1956, 0.3306), with $\bar{w}$ between 0.2620 and 0.3306, respectively.

GAVRILETS (1998) found cycling when paternal as well as maternal effects contributed to fitnesses and argued that the interaction of these two sorts of parental effects were what gave rise to the oscillations. He noted also that GINZBURG and TANEYHILL (1994) had found that ecological factors could give cycling in models of population size with maternal effects. Thus, the example above demonstrates that cycling can occur in models with just maternal selection arising from purely genetic effects.

Cycling has been found previously in a number of other population genetic models: constant fertility selection at a diallelic locus (HADELER and LIBERMAN 1975; see also DOEBELI and DE JONG 1998); frequency-dependent viability selection at a diallelic locus (MAY 1979; ALTENBERG 1991); density-dependent selection at a diallelic locus (ASMUSSEN 1979); constant viability selection in the two-locus, two-allele model (HASTINGS 1981; AKIN 1982); and mutation with constant viability selection (HOFBAUER 1985; see also BÜRGER 2000).

**Multiplicative model:** This special case further assumes that the selective pressures of the maternal effects and ordinary viability selection are independent, as, for example, when selection occurs at two separate stages in the life cycle of each individual, the first the result of its mother's phenotype and the second the result of its own phenotype. These effects thus act multiplicatively, and so

$$w_{ij} = v_i m_j \tag{3}$$

for $i = 1, 2, 3$. Equations 1 thus become

$$\begin{aligned} \bar{w}x' &= v_1 p(m_1 x + m_2 y/2), \\ \bar{w}y' &= v_2(m_3 pz + m_2 y/2 + m_1 qx), \\ \bar{w}z' &= v_3 q(m_2 y/2 + m_3 z). \end{aligned} \tag{4}$$

This model is formally equivalent to BODMER's (1965) model of multiplicative fertility selection (GAVRILETS 1998). But as BODMER (1965) noted, his model is also the same as OWEN's (1953) model of separate viability selection on males and females. In the case of maternal selection, this last equivalence allows a natural reinterpretation of how maternal selection acts.

Because mating is at random, the maternal environment affects all paternally derived alleles equally, which amounts to no selection on males at this stage. Thus if

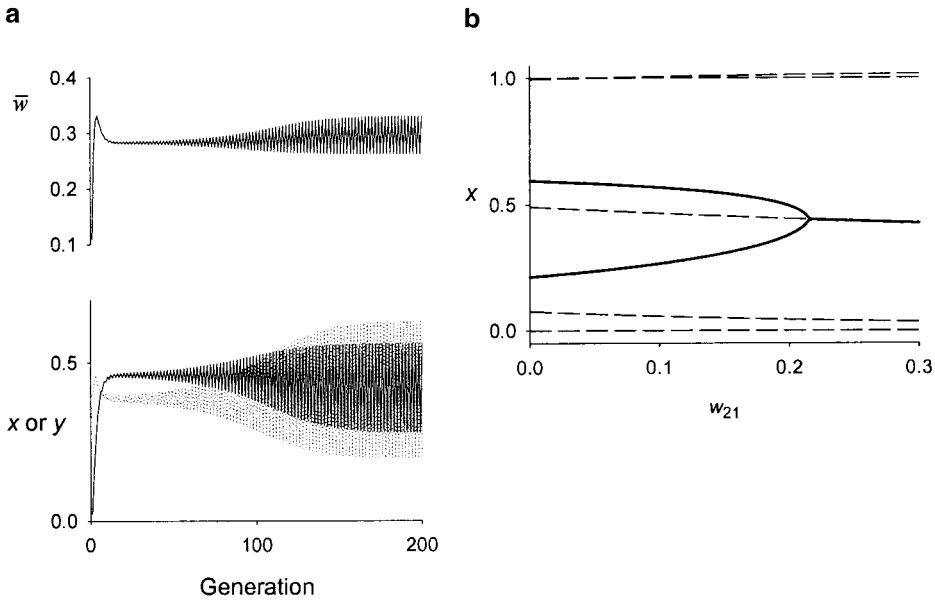


FIGURE 2.—(a) Genotype frequency and mean-fitness dynamics for a numerical case of the general model exhibiting cycling: $w_{11} = 0.028$, $w_{12} = 0.966$, $w_{21} = 0.116$, $w_{22} = 0.011$, $w_{23} = 0.886$, $w_{32} = 0.614$, and $w_{33} = 0.042$. The solid line in the bottom graph shows $x$; the dotted line, $y$. (b) Bifurcation diagram for *AA* homozygote frequency, $x$, showing also equilibrium values as a function of $w_{21}$. For $w_{21} > \sim 0.214$ only one equilibrium (heavy solid line) is stable; all other equilibria are unstable (dashed lines). As $w_{21}$ decreases below $\sim 0.214$, a stable two-cycle bifurcates from this equilibrium, which becomes unstable. Note that a sixth unstable and unfeasible equilibrium that ranges from $x \approx 1.36$ to $x \approx 3.23$ is not shown.

the frequency of maternally derived *A* alleles in zygotes after maternal-effect selection is $p_f$ and that of paternally derived *A* alleles in zygotes after maternal-effect selection (which is also that in sperm) is $p_m$, we have

$$p_f = \frac{m_1 x + \tfrac{1}{2} m_2 y}{m_1 x + m_2 y + m_3 z} \tag{5}$$

and

$$p_m = x + \tfrac{1}{2} y \quad (= p). \tag{6}$$

Thus, a little algebra shows that

$$p_f' = \frac{m_1 x' + \tfrac{1}{2} m_2 y'}{m_1 x' + m_2 y' + m_3 z'} = \frac{m_1 v_1 p_f p_m + \tfrac{1}{2} m_2 v_2 (p_f q_m + q_f p_m)}{m_1 v_1 p_f p_m + m_2 v_2 (p_f q_m + q_f p_m) + m_3 v_3 q_f q_m} \tag{7}$$

and

$$p_m' = x' + \tfrac{1}{2} y' = \frac{v_1 p_f p_m + \tfrac{1}{2} v_2 (p_f q_m + q_f p_m)}{v_1 p_f p_m + v_2 (p_f q_m + q_f p_m) + v_3 q_f q_m}, \tag{8}$$

which are the equations of differential viability selection on the two sexes, with the fitness of female genotype $i$ being $m_i v_i$, and that in males, $v_i$. In this multiplicative model, therefore, maternal selection is effectively acting only on females.

At least six equilibria may exist for certain parameter values, but some of these may be unfeasible or even complex, and others are unstable. The greatest number of simultaneously locally stable equilibria is two (OWEN 1953; MANDEL 1971), but both of these may be internal (*i.e.*, polymorphic; GAVRILETS 1998). A numerical example is shown in Figure 3. For $m_2 = 0$, the fixation of *A* and an internal equilibrium are simultaneously locally stable, whereas the fixation of the *a* allele and a second internal equilibrium, at $p \approx 0.585$, both are unstable. Two further equilibria are unfeasible, although the second misleadingly looks feasible in Figure 3, since at $m_2 = 0.05$, for instance, the frequency of *AA* genotypes is negative, yet $\hat{p} = \hat{x} + \hat{y}/2 \approx -0.079 + 1.111/2 \approx 0.476$. As the value of $m_2$ is increased past $\sim 0.43$, the first of these unfeasible equilibria breaks into feasible biological space and steals the local stability from the *A* fixation. Further increases in $m_2$ lead to this internal equilibrium merging with the unstable internal equilibrium, at which point they become complex conjugates and biologically irrelevant. Various other properties (*e.g.*, monotonic convergence near equilibria) can be derived by application of MANDEL's (1971) results.

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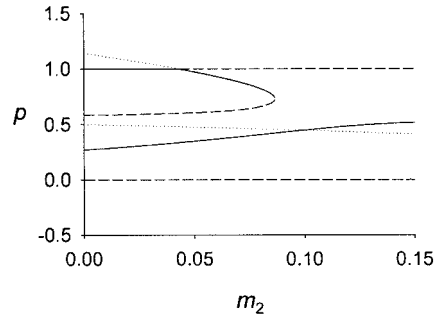


FIGURE 3.—Equilibrium values for $p$ as a function of $m_2$ in the multiplicative model. All other fitness parameters are constant: $m_1 = 0.35$, $m_3 = 0.88$, $v_1 = 0.41$, $v_2 = 0.73$, and $v_3 = 0.23$. Solid lines indicate locally stable equilibria; dashed lines, unstable but feasible equilibria; and dotted lines, unstable unfeasible equilibria.

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