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Evolutionary effects of density-dependent selection in plants

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Summary

The evolution of traits that affect genotypic responses to density regulated resources can be strongly affected by population dynamics in ways that are unpredictable from individual viability or reproduction potentials. Genotypes that are most efficient in utilizing energy may not always displace less efficient ones, and the evolution of energy allocation strategies may not always favour reproductive fitness because of their effects on destabilizing population growth rates. Furthermore, genetic polymorphisms in single loci that affect such traits can be maintained in populations with stable, periodic changes in population size and gene frequencies in the absence of heterozygote superiority. In fact, in the models investigated in this paper, the polymorphism is maintained, even in the absence of equilibrium genotypic frequencies.

1. Introduction

Heritable variations in life history parameters that influence the allocation of energy to vegetative and reproductive organs also affect population size dynamics. The feedback effects of population dynamics on the evolution of allocation strategies are not well known. In particular, it is not clear how a population might evolve if all genotypes must compete for a density regulated resource, and each genotype optimizes its own resource allocation for survival or reproduction. We investigate that question with a dynamic model of density-dependent selection and present examples in which mutations in genes that control the model parameters lead to a stable system that cannot be invaded by new genotypes that are only moderately different from existing alleles. These specific examples suggest that complex evolutionary behaviour of plant systems can be generated from simple gene effects, and that plant population dynamics can affect the evolution of energy allocation.

2. Background

Density and competition for scarce resources influence acquisition and allocation of energy to various plant organs, often in complex ways. Species evolve means of coping with less than optimum resource supplies, in some instances developing life history strategies that maximize some measure of fitness (Pugliese, 1988; Clegg et al. 1978; Kozlowski & Wiegert, 1986; Vincent & Pulliam, 1980; Doust, 1989; Namkoong & Rodriguez, 1990). Genetic variation in response to density and competition is common (See Dingle & Hegmann, 1982), and it can affect life history evolution (Charlesworth, 1980; Caswell, 1989). Because genetic variation exists in many of the component traits that affect fitness (Kalisz, 1986; Barker, 1988; Atchley, 1984), the evolution of life histories is not easily predictable (Lande, 1982; Lande & Arnold, 1983; Lande, 1988; Barker, 1988).

The reverse is true as well. Evolution of growth and reproductive strategies has substantial consequences for the dynamics of the populations in which it occurs (Tilman, 1985; Caswell, 1982). For example, increases in physiological efficiency can lead to population explosions and unstable population growth. Allocation of increased amounts of energy to reproduction may cause survival to decline, with unpredictable effects on population sizes. Organisms often do not react passively to environmental density changes but instead adjust their reproductive strategies to gain optimal benefit from their allocation of energy. Thus, predicting the outcome of allocation processes on fitness and population growth is difficult.

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If fitness can be modelled as an explicit function of density, then analysis of resulting sets of difference or differential equations generates predictions for evolutionary effects of genetic variation in population density. For competition among non-intermating, or haploid systems, Cressman (1990) derives stability conditions for equilibria under density dependence in differential equations. For competition with random mating in continuous-time processes with gene effects that are density- but not frequency-dependent, genetic polymorphisms in density response can be maintained only if overdominance exists in viability fitness or, in the case of Lotka–Volterra models, in carrying capacity (Selgrade & Namkoong, 1984; Namkoong & Selgrade, 1986). In the absence of overdominance, these models suggest that no genetic polymorphisms would persist. However, in discrete-time models with difference equations, qualitative behaviour is more difficult to predict. Periodic behaviour can develop, and genetic polymorphisms can be maintained even in the absence of overdominance in carrying capacity (Asmussen, 1979; Asmussen & Feldman, 1977), or in multiple niche selection (Getz & Kaitala, 1989).

Analysis of population models without genetic variation indicates that parameters can singly or collectively affect population sizes at equilibria and the stability of equilibria (Bishir & Namkoong, 1992). The situation is even more complex when two populations compete. Even without intermating, the evolutionary outcome is not easily predictable if one of the populations is unstable. In a simple model having exponential growth rates that are determined by the weighted sum of the competing population densities, Franke & Yakubu (1991) find that instability of one of two populations in the absence of the other can generate unexpected coexistences. In their model, there can be no coexistence if both species are stable when acting alone and no interior equilibrium exists. Coexistence is possible, however, if: (1) at least one of the boundary equilibria is unstable, (2) each unstable species has a stable periodic orbit, and (3) the range of population sizes in the periodic orbit of one unstable species includes its equilibrium point at all sizes of the other species if the second species is stable, or includes its orbit if the second species is unstable.

The dynamics of genetic variation in intermating populations in which genotypes may exhibit unstable growth dynamics have not been explored. In this paper we investigate some of the interrelationships between genetic and density variation and the ultimate consequence of genetic variation in traits that influence energy acquisition and allocation. We explore the joint evolutionary dynamics of individual growth and reproduction and density-dependent evolution. We show that it may not be generally possible to infer the populational consequences of gene effects.

3. The model

Full details of the model we use can be found in Bishir and Namkoong (1992). In brief, the model assumes a plant's acquisition of energy is affected by the density (number of plants per unit area) of the community in which the plant exists. Further, at each density encountered, the plant allocates its acquired energy among reproduction, maintenance, and growth in such a way as to maximize fitness, broadly defined as the average number of members of next year's population per individual in this year's population. For the examples presented here, we simplify the general model by assuming that all individuals are of the same size, so no growth takes place. That is, we assume that all energy goes to maintenance (survival) and to reproduction. Further, we assume that individuals respond to density only through the total numbers of plants, of all genotypes (species), in the community. In the presence of total density X, the fitness of a particular genotype is defined as

$$\phi(X) = np + s,$$

where

n = number of seeds produced by an individual plant, where one seed matures per reproductive bud, and is a function of the energy invested in reproduction;

p = probability of a seed surviving to become a full-grown plant 'next year', and is a function of plant density, and

s = probability of an existing plant surviving to remain in the population next year and is a function of the energy invested in maintenance.

The model requires that at each density X encountered, plants allocate acquired energy so as to maximize fitness and the quantities n, p and s all depend directly or indirectly on X. The allocation is subject to the restrictions:

(1) $L + n \leqslant b,$

and

(2) $E = u(L) + v(n) + m,$

where b = total number of bud positions available to the plant that could be allocated to either leaves or seeds; L = number of bud positions devoted to leaves; E = total amount of energy acquired; $u(L)$ = total amount of energy required to produce L leaves; $v(n)$ = total amount of energy needed to produce n seeds and m = total energy allotted to maintenance.

We use linear forms,

$$u(L) = \mu L \quad \text{and} \quad v(n) = \beta n,$$

for energy requirements for leaf and seed production. In addition, we take

$$s(m) = 1 - e^{qm}, \quad p = k e^{-x/c},$$

and

$$E = \epsilon(1 - e^{-aL})[r/(X+d)],$$

where μ, β, q, k, c, ϵ, a, r, and d are positive constants. Thus, survival probability, s, depends on maintenance energy, m, which depends on acquired energy and its allocation. Acquired energy E reflects the level of incident energy per unit area, ϵ, multiplied by crown area, $r/(X+d)$, and an 'absorption proportion', $1 - e^{-aL}$. Using these functions it can be shown that fitness expressed as a sum of adult survival and progeny production rates, is a monotonic declining function of density (Bishir & Namkoong, 1992).

4. Examples

We examine three particular parameters that influence individual viability and reproduction, the maximum number of buds (b) an individual can develop, the energy invested in an individual seed (β), and the seedling survival parameter (k). We investigate two phenomena that represent evolutionarily feasible events. The first is simply that mutations in relevant alleles may lead to increased size or energy gathering efficiency, thus allowing the plant to increase b and β, in stepped increments. An increased number of buds allows each plant to produce more leaves, more seeds, or both. As the number of leaves L increases, so does energy acquisition, E. Hence, such changes may enable the species to more rapidly increase population size without necessarily decreasing its energy allocation to maintenance.

The second case is that mutations permit changes in the allocation of energy between leaves and seeds. An increased amount of energy in seeds causes a higher seedling survival rate but a decrease in the survival and fecundity of parent plants. We thus allow the plant to increase b at the expense of decreasing β and seedling survival.

To illustrate these effects, we set the following parameters to establish an arbitrary scale:

$$a = 0.005, \quad d = 100, \quad \epsilon = 100\,000, \quad q = 0.0001,$$
$$\mu = 0.1 \quad \text{and} \quad r = c = 1000.$$

We begin with a base case in which $k = 0.1$, $b = 1200$ and $\beta = 2$. Computer simulation shows that a population composed only of this one genotype (AA) moves toward a stable equilibrium density of 5705. At that density the allocation of each plant's 1200 buds is divided between 415 leaves and 785 seeds. Each adult plant uses 13400 units of energy for maintenance and has a survival rate of 74%. The juveniles survive at a rate of 0.033%. Numerical results indicate that genotypes with lower energy efficiencies than the base genotype, in that they produce fewer buds and allocate less energy per seed at any level of energy acquisition (b and β both decline), have stable but lower

equilibrium population sizes. If heterozygotes (AA') have b and β parameters intermediate between those of the base genotype and any hybrid type with fewer b and lower β, the base genotype will exclude any invaders in an intermating population.

(i) *Example 1*

If an alternative homozygous genotype ($A'A'$) has a higher energy efficiency such that its b and β parameters are 1400, and 3 respectively, and if it were to grow with no other genotypes, the equilibrium population size would be unstable. The population would have a stable period 2 orbit alternating between 5368 and 6377 individuals. These plants allocate their 1400 buds between 384 leaves and 1016 seeds at the lower density and 647 leaves and 753 seeds at the higher density. Their adult survival rates are approximately 71.4% at both densities, but seedling survival varies from 0.047% at the lower density to 0.017% at the higher.

In contrast, in a population composed only of AA homozygotes, the equilibrium population size is stable at 5705, and if the heterozygote AA' has intermediate energy efficiency with $b = 1300$ and $\beta = 2.5$ its equilibrium population size is stable at 5751 if grown as a population of only AA' individuals.

In a randomly intermating population of all three genotypes, the population would exhibit period 2 behaviour with a total of 5547 plants (of all types) at the lower density, while the larger density totals 6030 plants. Respective frequencies of the A' allele are 0.725 and 0.726, respectively. Heterozygote frequencies exceed Hardy–Weinberg expectations at either density. Not only are the b and β parameters of the heterozygote intermediate between those of the homozygotes, but adult survival rates and the expected seedling survival per plant are also intermediate at both points. For the AA, AA', and $A'A'$ genotypes, the adult survival rates at both densities are approximately 0.738, 0.727 and 0.725, respectively. Seedling survival rates per parent are 0.32, 0.35 and 0.38, respectively, at the low density, and are 0.17, 0.19 and 0.21 at the high density. If fitness is taken as the sum of the genotype's per capita survival and progeny contributions to the next generation, the ordering of genotypic fitness at both densities is $A'A' > AA' > AA$. Therefore, there is no heterozygote superiority in the parameters, in fitness, or in any phenotypic measure of survival. We summarize these results in Table 1.

If an even more highly efficient allele, A'', can produce $A''A''$ genotypes that have $b = 1500$ and $\beta = 3.5$, a population of only these homozygotes would ultimately grow to an even wider stable period 2 orbit of 5240 and 6766 individuals. If this allele was in a random mating population with either of the A or A' allele as defined above, and their heterozygote was

Table 1. *Period 2 polymorphism among alleles affecting energy efficiency*

	Genotypes		
	AA	$A'A$	$A'A'$
Parameters			
b	1200	1300	1400
β	2	2·5	3
Behaviour without competition	Stable equilibrium	Stable equilibrium	Period 2
Limiting population size (X)	5705	5751	5368
			6377
Behaviour with competition			
Limiting population sizes			
Lower: Total $X = 5547$	417	2213	2917
$s(X)$	0·32	0·35	0·38
$p(X)$	0·738	0·727	0·725
$\phi(X)$	1·058	1·077	1·105
Upper: Total $X = 6030$	451	2402	3177
$s(X)$	0·17	0·19	0·21
$p(X)$	0·738	0·727	0·725
$\phi(X)$	0·908	0·917	0·935

exactly intermediate in b and β, a biallelic period 2 coexistence would exist. However, if there is random mating in a population with alleles that generate homozygotes with $b = 1300$ and $\beta = 2·5$, and their heterozygote is intermediate with $b = 1400$ and $\beta = 3·0$, the A'' allele is eliminated. In all of these cases, the heterozygote, if grown as a population of only that heterozygote, would exhibit period 2 behaviour. We can therefore discern no general rules for which allelic or genotypic behaviours would predict coexistence.

A stable period 2 allelic polymorphism with competing but intermating genotypes differs from the results for competition without any mating. If one competitor has the parameters of the $A''A''$ genotype above, $(b = 1500$ and $\beta = 3·5)$, it will exclude competitors of single genotype populations that have either $b = 1400$ and $\beta = 3·0$, or $\beta = 1300$ and $\beta = 2·5$, or $b = 1200$, and $\beta = 2·0$ in the absence of any heterozygotes. Thus analyses of ESS-type models that ignore intermating cannot predict the outcome of evolution in those situations.

(ii) *Example 2*

If alleles affect allocation of energy, rather than efficiency, any increases in plant size or energy gathering efficiency may be offset by decreased investments in reproduction and seedling survival. For these cases, we assume that decreases in b are accompanied by increases in β and in the k parameter of seedling survival. We narrow the range of variation from that of example 1 but otherwise, the model is as above. Consider a homozygous genotype (AA) with b, β and k parameters of 1400, 1·9 and 0·1, respectively. If grown in a pure stand, it would attain a stable equilibrium density of 5871, and would allocate its

1400 buds to 451 leaves and 949 seeds. This allocation produces an adult survival rate of 73·2% and a juvenile survival rate of 0·03%. If an alternative homozygote $(A'A')$ were to devote less resources to plant size and more to seeds such that its parameters were 1300, 2·0 and 0·125, respectively, in a pure stand, it would converge to a stable period 2 cycle alternating between population sizes of 5572 and 6314, and would allocate its buds to 354 or 557 leaves, and 946 or 743 seeds at the lower and higher densities, respectively. If a heterozygote exists with parameters of 1350, 1·95 and 0·12, it would, if grown as a population of only heterozygotes, converge to a stable period 2 orbit of 5535 and 6054. In a random mating population with the above homozygotes the total population would converge to an attracting cycle of period 2, with total densities of 5535 and 6507. Allele A frequencies are slightly different at the two densities, both being just over 11%. At both densities, the fitness of the heterozygote exceeds that of the homozygotes, but there is a slight deficiency of heterozygotes below Hardy–Weinberg expectations. The order of the surival rates for the adult genotypes is $AA > A'A > A'A'$, but order of the survival rate of seedlings produced is $A'A > AA > A'A'$ at both densities. Small changes in the parameter levels chosen and in the level of dominance in the parameters of the heterozygote can generate different genotypic fitnesses. For example, using the same b and β parameters for all genotypes but using $k = 0·1$, 0·1135 and 0·12, for AA, $A'A$ and $A'A'$, respectively, generates a slightly different cycle of period 2 without heterozygote fitness superiority and with a slight deficiency of heterozygotes below Hardy–Weinberg expectations. In this case, the ordering of genotypes is $AA > A'A > A'A'$ for adult survival, $A'A' > A'A > AA$ for seedling

Table 2. *Period 2 polymorphism among alleles affecting energy allocation*

	Genotypes		
	AA	*A'A*	*A'A'*
Parameters			
b	1400	1350	1300
β	1·9	1·95	2·0
k	0·1	0·12	0·125
Limiting population sizes			
Lower *X* = 5575	71	1103	4401
Upper *X* = 6507	83	1288	5136
Parameters			
b	1400	1350	1300
β	1·9	1·95	2·0
k	0·1	0·1135	0·12
Limiting population sizes			
Lower *X* = 5617	1	178	5438
Upper *X* = 6339	2	201	6136

survival, and $A'A' > A'A > AA$ for total fitness. These results are summarized in Table 2.

5. Conclusions

The phenotypic effect of alleles on plants may have effects on population dynamics that allow unexpected polymorphisms to persist. Alleles that increase physiological efficiency may not always displace alleles with lesser efficiency in average effect due to their destabilization of population size equilibria. Thus, physiological efficiency may not be maximized by natural selection and more allelic variations may persist in life history traits than anticipated from simple density dependent selection. The examples we examine indicate that allelic polymorphisms in random mating populations may persist even if: (1) frequency-dependent selection is absent, (2) fitness is a monotonic declining function of total density, and (3) competition between non-intermating biotypes would result in the exclusion of one biotype by the other. Polymorphisms can persist in the absence of heterozygote superiority in the genotypic reaction parameters, in juvenile and adult survival, and in some measures of inclusive fitness. Without any traditional measure of overdominance or underdominance and hence, no expectation of polymorphic equilibria, an attracting stable period 2 behaviour can evolve. Furthermore, the existence of both excesses and deficiencies of heterozygotes from Hardy–Weinberg expectations occur in spite of random intermating among adults. The examples also indicate that in some cases an evolutionary stability is achievable in which an allele endowing higher levels of energy efficiency and higher fitnesses at high densities may not invade populations having an allele endowing lower states of efficiency. Thus, population density effects can override what may seem to be the physiologically ordered evolution of fitness, permitting polymorphisms to exist by population regulation instead of by individual level effects.

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