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THE EVOLUTIONARILY STABLE PHENOTYPE DISTRIBUTION IN A RANDOM ENVIRONMENT

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Abstract.—In an unpredictably changing environment, phenotypic variability may evolve as a “bet-hedging” strategy. We examine here two models for evolutionarily stable phenotype distributions resulting from stabilizing selection with a randomly fluctuating optimum. Both models include overlapping generations, either survival of adults or a dormant propagule pool. In the first model (mixed-strategies model) we assume that individuals can produce offspring with a distribution of phenotypes, in which case, the evolutionarily stable population always consists of a single genotype. We show that there is a unique evolutionarily stable strategy (ESS) distribution that does not depend on the amount of generational overlap, and that the ESS distribution generically is discrete rather than continuous; that is, there are distinct classes of offspring rather than a continuous distribution of offspring phenotypes. If the probability of extreme fluctuations in the optimum is sufficiently small, then the ESS distribution is monomorphic: a single type fitted to the mean environment. At higher levels of variability, the ESS distribution is polymorphic, and we find stability conditions for dimorphic distributions. For an exponential or similarly broad-tailed distribution of the optimum phenotype, the ESS consists of an infinite number of distinct phenotypes. In the second model we assume that an individual produces offspring with a single, genetically determined phenotype (pure-strategies model). The ESS population then contains multiple genotypes when the environmental variance is sufficiently high. However the phenotype distributions are similar to those in the mixed-strategies model: discrete, with an increasing number of distinct phenotypes as the environmental variance increases.

Key words.—Bet-hedging, evolutionarily stable strategy (ESS), genetic polymorphism, phenotypic variation, random environment.

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Organisms typically face unpredictable temporal fluctuations in their environment, and many theoretical studies have examined the potential effects of environmental fluctuations on evolution. Specific issues include the maintenance of genetic variability within species; the rate of evolution at genetic and phenotypic levels; the coexistence of competing species; and the evolution of sexual reproduction, recombination, “bet-hedging,” and phenotypic variability as adaptations to changing environments.

Here, we examine the effect of temporally fluctuating selection on the evolution of a quantitative trait. We assume that the trait is subject to Gaussian stabilizing selection with a randomly varying optimum, and we allow generations to overlap so that an evolutionarily stable population can have genetic variance maintained by selection (Ellner and Hairston 1994). Two extreme models are considered. In the first, we assume that each adult female can produce phenotypically heterogeneous offspring and that the distribution of offspring phenotypes is determined by the mother’s genotype. The mother can then use phenotypic variability in her offspring as a bet-hedging strategy against unpredictable selection acting on her offspring. In the second, we assume that each individual experiencing selection has a phenotype determined (nonrandomly) by its own genotype. In this model, fluctuating selection can maintain genetic variability (Ellner and Hairston 1994), and the phenotype distribution is a function of the allele frequencies in the population.

For both models, we ask what distribution of phenotypes is present (in each generation or on average) when the population has reached an evolutionarily stable state, defined by

the property that selection acts against any new mutations (alleles or phenotypes not present in the population). Specifically, we examine the questions (1) Which environmental structures favor the evolution of a single phenotype versus phenotypically heterogeneous populations? (2) What array of phenotypes is expected in the evolutionarily stable population? (3) Under what conditions is the evolutionarily stable state genetically polymorphic? (4) Is there any testable relationship between the fitness of different phenotypes in an evolutionarily stable population?

In this paper, we are interested in polymorphism, either genetic or phenotypic, which is evolutionarily stable. Evolutionary stability means that any mutant allele affecting the phenotype distribution for the trait of interest, which is not present in the wild-type population, will have lower fitness than wild-type alleles. In the population genetics literature, this has been formalized as “evolutionary genetic stability” (Eshel and Feldman 1982), which is the population genetical version of the evolutionarily stable strategy (ESS) stability concept of evolutionary game theory (Maynard Smith 1982). The underlying biological assumption is that the alleles maintained in nature would be those that resist invasion by new mutants.

The ESS stability criterion is more stringent than the usual criteria for “protected” polymorphism or “stable” coexistence among a fixed set of alleles or competing species. For example, a two-allele polymorphism is protected if neither allele drives the other to extinction. However, the same polymorphism would be evolutionarily unstable if there is a feasible allele that could invade the diallelic population (“fea-

sible" means that it is within the constraints on possible phenotypes; in our models, any distribution of offspring is assumed to be feasible).

Thus, our results will differ from previous studies of the conditions under which fluctuating selection can maintain protected genetic polymorphisms (e.g., Gillespie 1972, 1973, 1991; Karlin and Lieberman 1974; Seger and Brockmann 1987; Frank and Slatkin 1990). One important example is the predicted relationship between environmental variability and the maintenance of genetic variance. By the storage effect of generation overlap (Chesson and Warner 1981), any non-zero level of variability is enough to maintain two suitably chosen alleles in a protected polymorphism, in either of our models. In contrast, we show below that polymorphism is never an ESS in the mixed-strategies model, and polymorphism is an ESS in the pure-strategies model only when the environmental variability is sufficiently large (Ellner and Hairston 1994).

Life-history strategies in changing environments often have been discussed in terms of Levins' graphical method of "fitness sets" and "adaptive function" (Levins 1962, 1968). No doubt Levins' theory is one of the most successful in evolutionary ecology, and has yielded a number of fruitful applications (e.g., Schaffer 1974; Venable 1985). However, Levins' methods are applicable only when the environment fluctuates between two values (e.g., good years and bad years). As a result, Levins' theory does not predict the evolutionary outcome for more general patterns of environmental fluctuations. Models with more general environmental fluctuations have been analyzed, but with a priori restrictions on the possible phenotype distributions in order to make the analysis tractable. For example it is commonly assumed that the phenotype distribution for any single genotype is discrete, consisting of a finite number of distinct types, rather than continuous (e.g., Venable 1985, Brown and Venable 1986, Yoshimura and Clark 1991). Slatkin and Lande (1976), however, assumed a Gaussian distribution with genetically determined variance and obtained the condition for the evolution of increased variance. Bull (1987) extended Slatkin and Lande's results to obtain ESS phenotypic variance, assuming also a Gaussian distribution. Our models allow very general patterns of environmental fluctuations, and arbitrary phenotype distributions. Not surprisingly, we find that the ESS phenotype distribution may be very different from what Levins' theory predicts. However, our results provide some justification for the assumption that the distribution of phenotypes is discrete.

Genotype-Frequency Dynamics

We consider a model with density-dependent selection resulting from intraspecific competition in a phenotypically heterogeneous population. Two different population models give rise to exactly the same selection equations, which we then analyze. For simplicity, we first describe the population models assuming haploid inheritance.

The first is the "lottery model" of competition for living sites (Chesson and Warner 1981). Let M be the total number of sites, and let $x_i(t)$ be the fraction of sites occupied by type- i adults in year t (age structure of adults is ignored).

Mortality of adults leaves $(1 - \gamma)M$ sites open for occupation by new recruits, where γ is the annual survivorship of adults. Selection affects the number of open sites occupied by type- i recruits. Specifically, let $W_i(t)$ be the relative viability of type- i juveniles, so that the number of type- i juveniles succeeding to occupy a site is

$$(1 - \gamma)Mx_i(t)W_i(t) / \sum_j x_j(t)W_j(t),$$

the sum running over all types in the population. The proportions x_i , therefore, change according to

$$x_i(t + 1) = x_i(t) \cdot \left\{ (1 - \gamma) \frac{W_i(t)}{\sum_j x_j(t)W_j(t)} + \gamma \right\}. \quad (1)$$

With diploid inheritance, the offspring of adults with a given genotype are distributed among several genotypes in Mendelian proportions, and (1) is modified accordingly.

The second model is a density-dependent version of the often-used "annual plant with seed bank" model introduced by Cohen (1966). Adults in this model produce dormant propagules (e.g., eggs or seeds), which survive until the next year and either hatch or remain in dormancy. Individuals hatched in each year then experience fluctuating selection, and the total propagule production is regulated to a carrying capacity. After initial transients, the total dormant propagule population converges to a constant, and the changes in the genotype frequencies in the seed bank are again given by equation (1), in which the generation overlap γ is the product of annual survivorship and the probability of staying dormant (see Ellner and Hairston 1994).

The reason equation (1) applies to both models is that once initial transients have died out, a constant fraction of the population at the time of census each year is derived from offspring produced the previous year. The current frequencies are consequently a fixed weighted average of past frequencies and the most recent relative contributions of new offspring. This feature of our model is certainly only a rough approximation to actual patterns of generational overlap (e.g., Philippi [1993] has shown that seeds of desert annuals do not have a constant germination fraction, even under constant environmental conditions). However, we expect similar results to hold so long as the amount of generational overlap has a positive lower bound.

To give the relationship between phenotype and reproductive success, we adopt a standard model from quantitative genetics. An individual's phenotype is assumed to be given by some scalar quantity that we denote z . Individuals reproducing in year t experience stabilizing selection with optimum phenotype θ_t . We assume Gaussian selection, so that the fecundity of an individual of phenotype z is proportional to

$$w_\theta(z) \stackrel{\text{def}}{=} e^{-(z-\theta)^2/2\sigma_w^2}, \quad (2)$$

where σ_w^2 is the "selection variance." We assume that the optimum phenotype θ_t varies independently between generations, following a probability density function $q(\theta)$ with mean zero and variance σ_θ^2 which is symmetric around its mean. We will call σ_θ^2 the "environmental variance". We

assume, except when noted otherwise, that the distribution of θ_t is bounded.

To simplify the notation, we rescale units for the phenotype value z and the optimum phenotype θ so that $\sigma_w = 1$. The fitness function (2) in the rescaled variables is

$$w_\theta(z) = e^{-(z-\theta)^2/2}. \quad (2')$$

We use prime ('') to denote variables in unscaled units (e.g. θ' , $\sigma_{\theta'}$).

MIXED-STRATEGIES MODEL

We first consider mixed-strategist dynamics, in which an individual can produce a variety of phenotypes with a genetically determined distribution. Let $p_i(z)$ be the phenotype probability distribution for offspring produced by an individual of genotype i . Then, the fitness of genotype i in year t is given by taking expectation of fitnesses over the various phenotypes:

$$W_i(t) = \int_{-\infty}^{\infty} w_\theta(z)p_i(z) dz. \quad (3)$$

(Note that in equation (3) and elsewhere, the phenotype distribution may be any probability distribution on the real line, and need not have a density function. Thus, $p(z)$ should be regarded as a generalized function, or more precisely $p_i[z]dz$ in (3) should be taken to stand for Lebesgue integration with respect to a measure $p_i[dz]$).

We first show that a distribution $p(z)$ is an ESS for the mixed-strategies model if and only if it maximizes the geometric mean of genotype fitness (3), and hence does not depend on the amount of generational overlap. We also show that the ESS is characterized by a stochastic version of the “ideal free distribution,” generalizing a result of Slatkin (1978) for distributions with exactly two phenotypes.

Evolutionary stability is defined by noninvasibility, that is, that any new type introduced into the population at low numbers will tend to decrease in frequency. From (1) we expect that a genotype with distribution $p_i(z)$ will increase in the population when rare if its boundary growth rate, given by

$$E_t \log \left\{ (1 - \gamma) \frac{W_i(t)}{\sum_j x_j(t) W_j(t)} + \gamma \right\}, \quad (4)$$

is positive. In (4), E_t denotes the long-term average over the years, and j in the summation runs over all the genotypes other than i in the population. However, invasion fails if (4) is negative. This is the standard “invasibility” criterion for competition models with random parameters. Our results here are all based on using invasibility criteria to determine evolutionary stability. The validity of the invasibility criterion has been proved rigorously under conditions satisfied by the models considered here (Chesson and Ellner 1989; Ellner 1989) for competition between two types. However, it should be kept in mind that, as Yoshimura and Clark (1991) noted, chance events may temporarily favor an “inferior” strategy, so that the approach to an evolutionarily stable state will be imperfect in nature, and is likely to involve frequent detours and backsliding.

In the mixed-strategies model, an evolutionarily stable population must consist of a single genotype (Ellner and Hairston 1994): If the evolutionarily stable population contains more than two coexisting alleles with different mixed strategies, then a mutant allele with a mixture of these two mixed strategies would enjoy the advantage of bet-hedging (in mathematical terms, by Jensen’s inequality) and can invade the population. Hence, a genetically polymorphic population is never evolutionarily stable in the mixed-strategies model. Therefore, it follows from (3) and (4) that a phenotype distribution p^* is an ESS phenotype distribution if

$$\rho(p | p^*) = E_\theta \log \left\{ (1 - \gamma) \frac{\int_{-\infty}^{\infty} w_\theta(z)p(z) dz}{\int_{-\infty}^{\infty} w_\theta(y)p^*(y) dy} + \gamma \right\} < 0, \quad (5)$$

for all $p \neq p^*$.

The long-term average over time in (4) is replaced by expectation over the distribution of the optimal phenotype θ in (5), because θ_t is the only random quantity in (4) when the population being invaded consists of a single genotype. Using (5), we establish the following result in Appendix A and B:

There is a single ESS $p^(z)$, which maximizes mean log fitness.*

$$\Phi(p) = E_\theta \log \left\{ \int_{-\infty}^{\infty} w_\theta(z)p(z) dz \right\}. \quad (6)$$

Moreover p^ is the only local maximum of Φ , and is symmetric [$p^*(z) = p^*(-z)$].*

Note that maximizing mean log fitness is equivalent to maximizing the geometric mean fitness, a commonly used criterion for life-history evolution in temporally changing environments, implying that the identical criterion applies to the mixed-strategy ESS in overlapping generation models.

Equation (6) implies that the ESS does not depend on the amount of generational overlap, γ . This result is strictly a characteristic of the mixed-strategies model, and does not remain true if only pure strategies are allowed (see below). Overlapping generations enables a clutch of offspring having a single phenotype to experience various environmental states. This has a similar effect to a mixed strategy, because, in the latter, an individual has offspring of various phenotypes in an environmental state. Conversely, if any mixed strategy is feasible without any cost, the amount of generational overlap becomes irrelevant to the ESS.

The ESS Is an Ideal Free Distribution

A more intuitive characterization of the ESS can be obtained from equation (6). If the mutant distribution p is slightly different from p^* , that is, $p - p^* = O(\epsilon)$ in some suitable norm (e.g., total variation), then $\Phi(p) - \Phi(p^*)$ can be expanded in a power series in ϵ , as shown in Appendix C. This gives necessary and sufficient conditions for p^* to be an ESS, which we summarize as follows. Let $\varphi(z)$ be the mean relative fitness of a z -type individual in the wild-type population:

$$\varphi(z) = E_\theta \left\{ \frac{w_\theta(z)}{\bar{w}_\theta} \right\}, \quad (7a)$$

where

$$\bar{w}_\theta = \int_{-\infty}^{\infty} w_\theta(z) p^*(z) dz, \quad (7b)$$

is the mean fitness of the population in environment θ . Then $p^*(z)$ is the ESS if and only if

$$\begin{aligned} \varphi(z) &= 1, & \text{for all } z \in \text{support}(p^*) \\ \varphi(z) &\leq 1, & \text{for all } z \notin \text{support}(p^*) \end{aligned} \quad (8)$$

(the support of a probability distribution is the smallest closed set having probability 1). At any isolated points z_i in the support of p^* , (8) will be satisfied if and only if

$$\varphi(z_i) = 1, \quad \text{and} \quad (9a)$$

$$\varphi(z) \text{ has a maximum at } z = z_i. \quad (9b)$$

Condition (8) states that all phenotypes found in the evolutionarily stable population enjoy the same relative fitness when averaged over the environments, whereas any phenotype missing from the population would have lower average relative fitness. This is a kind of ideal free distribution (IFD) for evolutionarily stable mixed strategies (Fretwell 1972; Maynard Smith 1982), generalized to a stochastically changing environment. For the special case in which only two phenotypes are possible (rather than a continuum), a similar result was derived by Slatkin (1978). The standard IFD says that all strategies used in the ESS should have equal fitness (i.e., equal payoff averaged over all individuals using a given strategy); condition (8) requires that all types have equal mean relative fitness.

Using the criteria (8) and (9), we now examine which kind of phenotype distributions become evolutionarily stable for different types of environmental fluctuations.

Stability of Monomorphic Distribution

The simplest possible strategy is to make all progeny having the mean optimum phenotype:

$$\text{Prob}\{z = 0\} = 1. \quad (10)$$

This is clearly the best strategy when the environment does not vary at all, and it remains stable so long as φ attains its global maximum at $z = 0$. For p^* given by (10),

$$\begin{aligned} \varphi(z) &= E_\theta \left[\frac{w_\theta(z)}{w_\theta(0)} \right] = e^{-z^2/2} E_\theta [e^{z\theta}] \\ &= e^{-z^2/2} \int_{-\infty}^{\infty} e^{z\theta} q(\theta) d\theta. \end{aligned} \quad (11)$$

Stability of (10) therefore depends on how rapidly the tails of q decay.

(i) For θ having a uniform distribution on an interval $[-\alpha, \alpha]$, where $\alpha = (3\sigma_\theta^2)^{1/2}$, the relative fitness of z individuals is

$$\varphi(z) = e^{-z^2/2} \frac{\exp(\alpha z) - \exp(-\alpha z)}{2\alpha z}. \quad (12)$$

If the (scaled) environmental variance σ_θ^2 is smaller than 1, then $\varphi(z)$ has a unique maximum at $z = 0$, and hence the monomorphic equilibrium is stable. If $\sigma_\theta^2 > 1$, on the other hand, $\varphi(z)$ is bimodal with a symmetric pair of peaks, and hence a monomorphic population can be invaded. Since the phenotype θ has been rescaled relative to σ_w , this result can be restated as follows: the monomorphic distribution at $z = 0$ is evolutionarily stable if and only if the environmental variance is smaller than the selection variance, $\sigma_\theta^2 < \sigma_w^2(\theta)$ indicating the optimum phenotype measured in unscaled units.

(ii) For a Gaussian distribution of θ , the relative fitness of z individuals becomes

$$\varphi(z) = e^{(\sigma_\theta^2 - 1)z^2/2}. \quad (13)$$

Therefore, the monomorphic distribution (10) is again evolutionarily stable if and only if $\sigma_\theta^2 < 1$.

These two examples suggest that monomorphism would be evolutionarily stable if the environmental variance were sufficiently small. This conjecture is valid for many classes of distributions, but not always. Expanding (11) in a Taylor series around $z = 0$, we have

$$\varphi(z) = 1 + \frac{1}{2} (\sigma_\theta^2 - 1) z^2 + O(z^4), \quad (14)$$

for any symmetric probability density $q(\theta)$. This implies that when $\sigma_\theta^2 < 1$, $\varphi(z)$ attains a local maximum at $z = 0$. Monomorphism thus has a kind of local evolutionary stability whenever $\sigma_\theta^2 < 1$, for any distribution of θ . However, phenotypes far from zero may have higher relative fitness than the wild type, so condition (8) may fail, and monomorphism therefore is not an ESS. This will occur for distributions of θ having slowly decaying tails, as shown in the next example.

(iii) For bi-exponentially distributed θ ,

$$q(\theta) = \frac{\alpha}{2} e^{-\alpha|\theta|}, \quad (15)$$

where $\alpha = (2/\sigma_\theta^2)^{1/2}$, the mean relative fitness of z individuals is

$$\begin{aligned} \varphi(z) &= \frac{\alpha^2}{\alpha^2 - z^2} e^{-z^2/2}, & \text{if } |z| < \alpha \\ &= +\infty, & \text{if } |z| \geq \alpha. \end{aligned} \quad (16)$$

(Note that, in [16], it is the long-term average of relative fitness which diverges to infinity (for $|z| > \alpha$), not the relative fitness in each year, which always remains finite.) The monomorphic population always can be invaded by an extreme phenotype ($|z| > \alpha$), and hence monomorphism is never the ESS for any environmental variance. Indeed, no phenotype distribution with bounded support can be an ESS if θ has the distribution (15) or any other distribution with tails decreasing slower than the Gaussian (proof given in Appendix D). Thus, for this case, there are at least two “weakly stable” phenotype distributions: monomorphism and the true ESS. Numerical results suggest that the true ESS distribution consists of an infinite, unbounded sequence of discrete phenotypes $z = 0, \pm z^1, \pm z^2, \dots$.

To summarize, the monomorphic strategy will be the ESS if the distribution of θ is concentrated near the mean in two

senses: the environmental variance is smaller than the fitness variance, and the tails of θ 's distribution decrease rapidly enough (the exact condition is eq. [11] being everywhere less than 1).

Dimorphic ESS

What, then, is the form of the ESS after the monomorphic distribution becomes unstable? One common possibility, discussed in this section, is bifurcation to a dimorphism ESS when the environmental variance σ_θ^2 increases past 1, in a family of distributions (such as the uniform) for which the ESS is monomorphic when $\sigma_\theta^2 < 1$. The stability of this type of ESS again depends on the tails of the environment distribution $q(\theta)$.

The ESS distribution is symmetric; thus, a dimorphic ESS must be evenly divided between phenotypes x and $-x$ ($x > 0$):

$$\text{Prob}\{z = x\} = \text{Prob}\{z = -x\} = \frac{1}{2}. \quad (17)$$

The conditions (9a,b) for the evolutionary stability of (17) are then satisfied if

$$\varphi(\pm x) = E_\theta \left[\frac{w_\theta(\pm x)}{\bar{w}_\theta} \right] = 1, \quad (18a)$$

$$\frac{\partial \varphi}{\partial z}(\pm x) = E_\theta \left[\frac{1}{\bar{w}_\theta} \frac{\partial w_\theta}{\partial z}(\pm x) \right] = 0, \quad (18b)$$

$$\frac{\partial^2 \varphi}{\partial z^2}(\pm x) = E_\theta \left[\frac{1}{\bar{w}_\theta} \frac{\partial^2 w_\theta}{\partial z^2}(\pm x) \right] < 0, \quad (18c)$$

where

$$\bar{w}_\theta = \frac{1}{2} [w_\theta(x) + w_\theta(-x)]. \quad (19)$$

Condition (18a) is automatically satisfied, because of the symmetry of θ . Substituting (2') into (18b) and (18c) and using (18a), we have

$$\frac{\partial \varphi}{\partial z}(x) = E_\theta \left[\theta \frac{w_\theta(x)}{\bar{w}_\theta} \right] - x = 0, \quad (20)$$

and

$$\frac{\partial^2 \varphi}{\partial z^2}(x) = E_\theta \left[(x - \theta)^2 \frac{w_\theta(x)}{\bar{w}_\theta} \right] - 1 = (\sigma_\theta^2 - 1) - x^2. \quad (21)$$

For the environmental variance slightly larger than the fitness variance, we can set

$$\sigma_\theta^2 = 1 + \varepsilon, \quad (22)$$

for small $\varepsilon > 0$ and seek an ESS distribution in which the phenotypes $\pm x$ are close to zero. Assuming (22), Taylor expansion around $x = 0$ gives

$$\frac{w_\theta(x)}{\bar{w}_\theta} = \frac{2}{1 + e^{-2x\theta}} = 1 + x\theta - \frac{1}{3}(x\theta)^3 + O(x^5). \quad (23)$$

Substituting this into (20) and noting that all odd moments of θ are zero, we find

$$x = \pm \sqrt{\varepsilon \frac{3\sigma_\theta^4}{E(\theta^4)}} + O(\varepsilon). \quad (24)$$

Similarly, applying (23) and (24) to (21),

$$\frac{\partial^2 \varphi}{\partial z^2}(x) = \left(1 - \frac{3\sigma_\theta^4}{E(\theta^4)} \right) \varepsilon + O(\varepsilon^2). \quad (25)$$

From this we conclude that the dimorphic equilibrium is stable for small positive ε , if

$$Q \stackrel{\text{def}}{=} \frac{E(\theta^4)}{3\sigma_\theta^4} < 1 \quad (26)$$

and is unstable if $Q > 1$. $3Q = E(\theta^4)/\sigma_\theta^4$ is the kurtosis of θ . It is interesting to note that the ESS phenotypes $\pm x$ are $O[\varepsilon^{1/2}] = O[(\sigma_\theta^2 - \sigma_w^2)^{1/2}]$ analogous to a standard bifurcation from a fixed point to a periodic solution in a difference or differential equation.

Figures 1a and 1b illustrate the bifurcation to dimorphism for a uniform distribution of θ , which has $Q = 3/5$. For $\sigma_\theta = 0.9$, the monomorphic distribution is stable and $\varphi(z)$ has a unique maximum at $z = 0$, whereas for $\sigma_\theta = 1.5$, the ESS is dimorphic and $\varphi(z)$ has a symmetric pair of local maxima at the two phenotypes in the ESS. Figure 1c, on the other hand, demonstrates a numerical example of a trimorphic ESS for a truncated bi-exponential distribution of θ with $Q > 1$. For the distribution (15) with unbounded exponential tails in which $Q = 2$, the ESS is never dimorphic, as noted above.

The Gaussian distribution, which has $Q = 1$, therefore, is exactly on the dividing line between two qualitatively different types of ESSs. Tails decreasing faster than Gaussian would tend to give $Q < 1$, so a dimorphic distribution is stable, whereas tails decreasing slower than Gaussian give $Q > 1$, and the dimorphic equilibrium is unstable. When θ exactly follows the Gaussian distribution, the results are very different. Suppose that $q(\theta)$ is Gaussian and $p^*(z)$ is also Gaussian with mean 0 and variance σ_p^2 . Then, by choosing $\sigma_p^2 = \sigma_\theta^2 - 1$, we can easily verify by direct calculation that $\varphi(z) \equiv 1$ for all z . This implies that the Gaussian distribution with mean 0 and variance $\sigma_\theta^2 - 1$ satisfies condition (8) and therefore is the ESS whenever $\sigma_\theta^2 > 1$. (This is the ESS phenotypic variance obtained by Bull [1987]. Note, however, that the Gaussian distribution is an ESS only when $q(\theta)$ is exactly Gaussian.)

Figure 2 is the result of numerical simulation of evolutionary dynamics for uniformly distributed θ , showing that a mutant allele with a dimorphic phenotype distribution invades and replaces the wild-type allele with the "best" Gaussian phenotype distribution (having the optimal phenotypic variance $\sigma_p^2 = \sigma_\theta^2 - 1$). Advantage of dimorphism over Gaussian $p(z)$ is substantial, and the replacement will take place in relatively short time.

Polymorphic ESSs Are Generically Discrete

When the environmental variance is increased to well above the selection variance, numerical calculations of the ESS phenotype distribution (obtained by numerically optimizing the objective function Φ) tend to show an increasing number of distinct phenotypes. For the uniformly distributed θ , the ESS phenotype distribution goes from 2 to 3, 4, 5 . . .

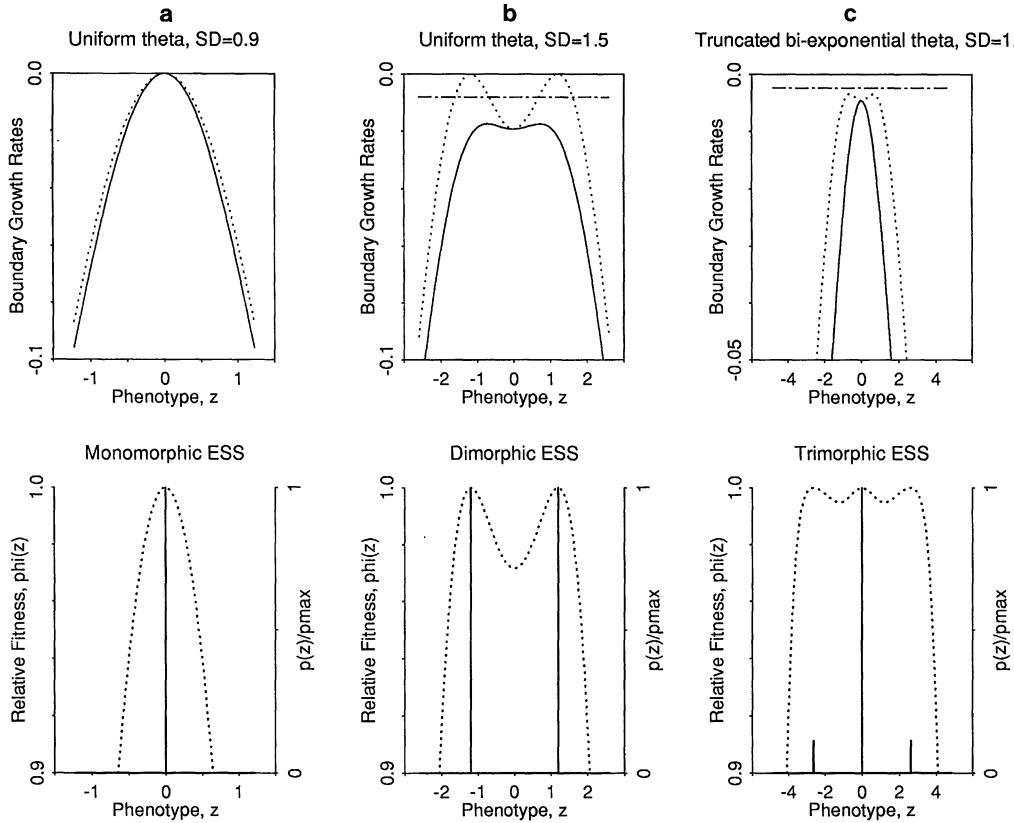


FIG. 1. Evolutionarily stable strategy (ESS) phenotype distribution and mean relative fitness in the mixed-strategy model. Upper panels show the boundary growth rates, defined as (5), of monomorphism at z (solid curves), dimorphism at z and $-z$ (dotted curves), and Gaussian with the variance $\sigma_\theta^2 - 1$ (hatched curves), in the population of the ESS phenotype distributions p^* (shown in lower panels). Generation overlap $\gamma = 0.75$. Phenotypes are scaled in units of σ_w . Lower panels show that the mean relative fitness [$\phi(z)$: dotted curves] satisfies an IFD condition [equation (9) in the text]; that is, the mean relative fitness is maximized at phenotypes in the ESS $p^*(z)$ (solid spikes). $p^*(z)$ was obtained by a numerical optimization of the objective function Φ . a. Uniform distribution of θ_t with $\sigma_\theta/\sigma_w = 0.9$; $Q = 3/5$. The ESS $p^*(z)$ is monomorphic at $z = 0$. b. Uniform distribution of θ_t with $\sigma_\theta/\sigma_w = 1.5$. The ESS is dimorphic. c. Truncated biexponential distribution of θ_t : $q(\theta) = Ce^{-5|\theta|/\alpha}$ for $|\theta| < \alpha$, and $q(\theta) = 0$ for $|\theta| > \alpha$, where C is a normalizing constant. For this distribution $Q \approx 1.26$. α is chosen so that $\sigma_\theta/\sigma_w = 1.5$. The ESS is trimorphic. Boundary growth rates of any monomorphic, dimorphic, or Gaussian strategies are strictly negative, as expected, except when these coincide with the ESS [Boundary growth rate of optimal Gaussian distribution is omitted in a. because the optimal Gaussian is monomorphic at 0].

phenotypes as σ_θ^2 increases (fig. 3a). For Q slightly above 1, the dimorphic distribution is not stable, and we conjecture that, instead, the ESS is trimorphic for σ_θ^2 just above 1, followed by bifurcations to 5, 7, ... phenotypes in the ESS; figure 3b shows an example of this pattern of bifurcations.

It would be difficult to give even approximate general conditions for the ESS to include a given number of distinct phenotypes. However, we obtain in Appendix E a strong qualitative result: *the ESS distribution is discrete, rather than continuous, for all distributions $q(\theta)$ with bounded support.*

However, if we (temporarily) consider $q(\theta)$ with unbounded support, the situation is a bit more complicated. For any continuous density $p(z)$, (E3) in Appendix E gives a distribution $q(\theta)$ such that $p(z)$ is the ESS; hence, any continuous density can be an ESS phenotype distribution. This seems to say that our model predicts everything and therefore predicts nothing. However, the actual situation is that continuous $p(z)$ s are highly exceptional. Specifically, we show in Proposition 2 of Appendix E that the ESS is discrete rather than continuous for a “generic” set of distributions $q(\theta)$. The intuitive meaning of “generic” is that exceptions exist but are very

rare; for example, in the set of real numbers, the property of not being an integer is generic (the precise result is that the ESS is discrete for all $q(\theta)$ in an open, dense set). Thus, the general prediction of the model is that continuous phenotype distributions are not optimally adaptive as a bet-hedging strategy against fluctuating selection.

Non-Gaussian Fitness

We assumed a Gaussian fitness function (2) to derive ESS phenotype distributions for given $q(\theta)$. However, our general assertion on the rarity of continuous ESSs is valid for much wider classes of fitness functions. For example, the ESS is generically discrete for any fitness function of the form $\exp[-f(z - \theta_t)]$, where $f(x)$ is a polynomial of degree greater than one. (More precisely, the ESS is generically discrete for any analytic fitness function with tails decreasing faster than exponential: see Appendix E.)

We believe that it is reasonable to assume that the fitness function is analytic, based on the following argument: Many random processes intervene between the genetically deter-

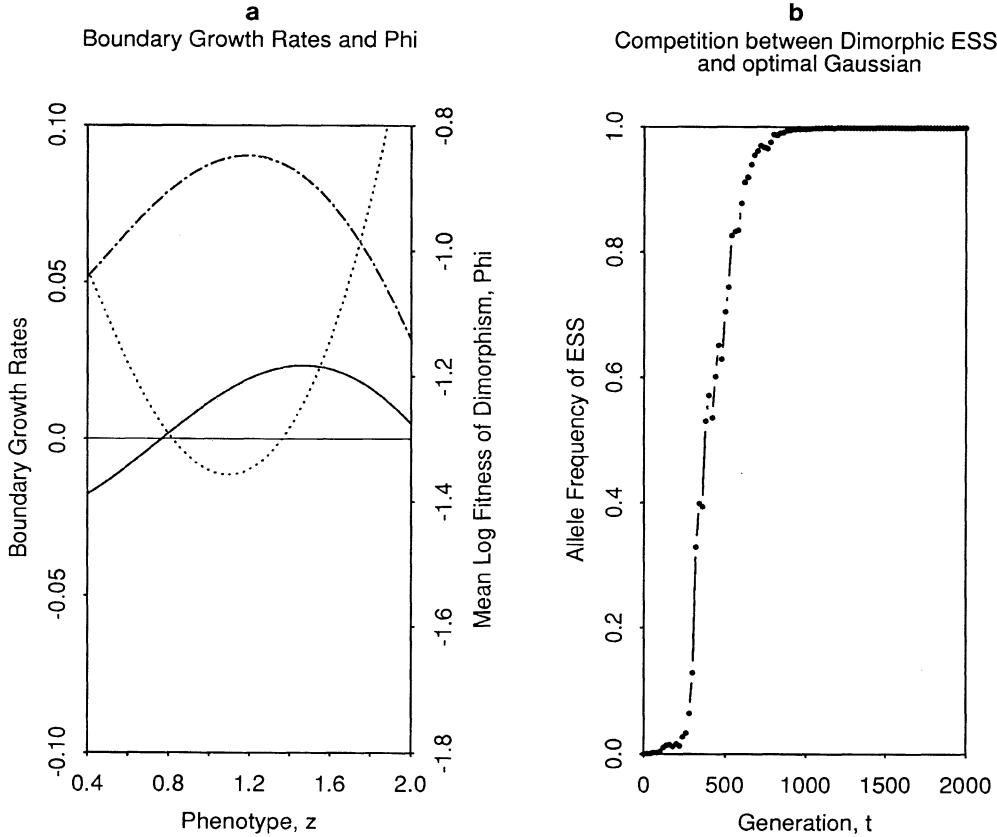


FIG. 2. Competition between mixed strategies: a dimorphism beats the “optimal” Gaussian. Uniformly distributed θ_t with $\sigma_\theta/\sigma_w = 1.5$. Generation overlap $\gamma = 0.75$. a. Solid curve, the boundary growth rates $p(\pm z|N)$ of alleles with dimorphic phenotype distributions at phenotypes $\pm z$, plotted as a function of its positions $|z|$ (horizontal axis), when the wild type is a Gaussian phenotype distribution N with the optimal variance [i.e., $N(0, \sigma_\theta^2 - \sigma_w^2)$]. Dotted curve, the boundary growth rate $p(N|\pm z)$ of the Gaussian N when the wild type is dimorphism $\pm z$. Hatched curve, mean log fitness Φ of dimorphism $\pm z$. The ESS is dimorphism at $z \approx 1.2$ at which Φ is maximized. Around $z = 1.2$, that is, close to the ESS, $p(\pm z|N)$ is positive and $p(N|\pm z)$ is negative, indicating that dimorphism can invade the “optimal” Gaussian distribution, but the “optimal” Gaussian cannot invade dimorphism. If the dimorphism is far enough from the ESS (e.g., $|z| > 1.4$), then Gaussian and dimorphic strategies will coexist. b. A simulation result of the competition between Gaussian with the optimal variance, and the ESS dimorphism at $z \approx \pm 1.2$. Initial frequency of the ESS was 0.001. The ESS invades optimal Gaussian distribution and finally replaces it.

mined trait and the realized individual phenotypes, a common model being $P = G + E$ (phenotype = genotypic effect + environmental effect). If the random environmental effect is Gaussian, it would smooth out any roughness in the phenotypic fitness functions, to give a genotypic fitness function that is analytic (Nagylaki 1989). If this is the case, then the ESS is generically discrete.

When the fitness function is not Gaussian, the bifurcation diagram for ESSs changes accordingly. For example, if the fitness function is slightly deviated from Gaussian as $w_\theta(z) = k(z - \theta)$ with

$$k(z) = e^{-x^2/2 - (\alpha x^4/4 + \beta x^3/3)} \quad [\alpha, \beta = O(\varepsilon)].$$

then the monomorphic ESS is given by $x^* = -\beta\sigma_\theta^2 + O(\varepsilon^2)$. This monomorphic ESS loses stability when σ_θ exceeds the threshold: $\sigma_\theta = 1 + (3\alpha/2)(1 - 2Q) + O(\varepsilon^2)$. We numerically verified that a slightly asymmetric dimorphic ESS appears for σ_θ above the threshold, if the kurtosis Q is smaller than one (which is necessary for the stability of dimorphic ESS for Gaussian fitness).

Our theory does not apply in general if the fitness function is not smooth. For example, if we assume a discontinuous fitness function: $w_\theta(z) = k_0(z - \theta)$ with

$$\begin{aligned} k_0(x) &= e^x && \text{for } x \leq 0 \\ &= 0 && \text{for } x > 0 \end{aligned}$$

then the ESS is continuous if $q(\theta)$ has a smooth density with unbounded support (this example was suggested to us by Yoh Iwasa; the ESS obtained is equivalent to the ESS male butterfly emergence pattern for a given female emergence pattern $q(\theta)$ [Iwasa et al. 1983]).

PURE-STRATEGIES MODEL

So far we have assumed that a genotype can produce any distribution of phenotypes with no constraints or cost. In this section, we briefly jump to the opposite extreme and assume that each genotype produces a single phenotype z rather than a distribution of phenotypes. Our purpose is to note that the

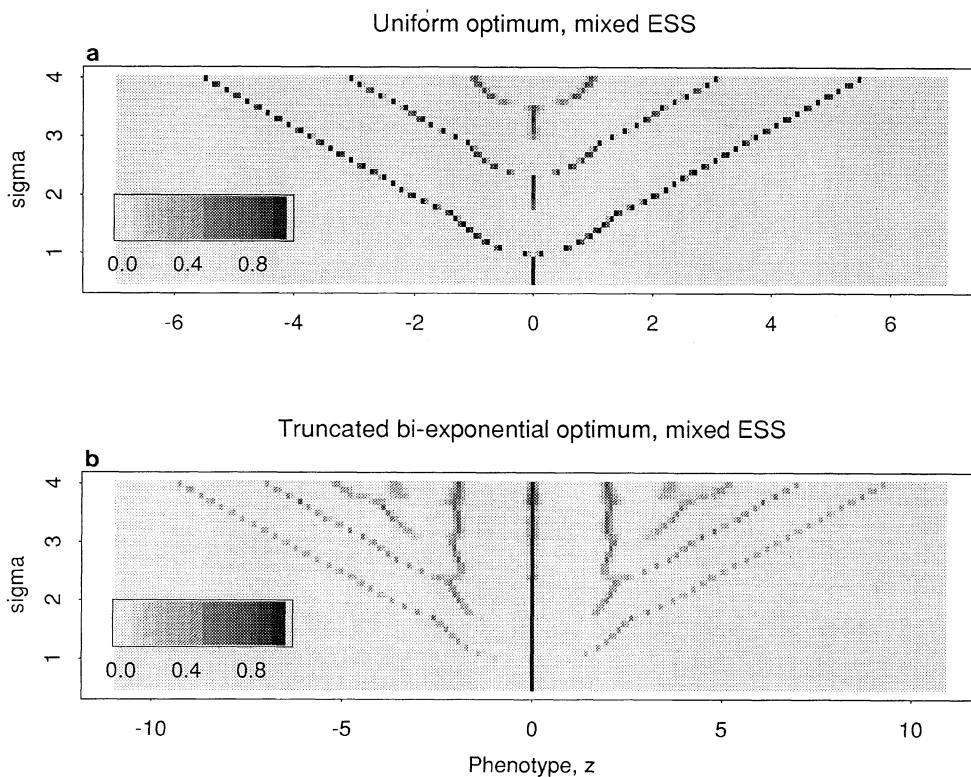


FIG. 3. Bifurcation of evolutionarily stable strategy (ESS) phenotype distributions: mixed-strategy model. a. Uniform distribution of θ_t , $Q = 3/5$. b. Truncated biexponential distribution of θ_t as defined in figure 1c: $q(\theta) = Ce^{-3|\theta|/\alpha}$ for $|\theta| < \alpha$, and $q(\theta) = 0$ for $|\theta| > \alpha$. In this distribution, $Q \approx 1.058$, independent of α . The variance was increased by increasing the value of α . In each plot, the horizontal axis is the phenotype z (scaled relative to σ_w), and the vertical axis is σ_0 . Each horizontal slice represents the relative frequency distribution of phenotypes in the ESS distribution, with darkness indicating frequency. To make the smaller modes visible, a square-root scale was used [darkness is proportional to $(\text{frequency})^{0.5}$]. ESS phenotype distribution was obtained by numerically maximizing the objective function Φ using a discrete approximation with 201 evenly spaced phenotypes in the range -6 to 6 .

major qualitative results for the mixed-strategy model continue to hold in this pure-strategies model (although the quantitative predictions are, of course, different). This similarity suggests that the qualitative conclusions are robust and should continue to apply in realistic intermediate situations, for example, if the production of heterogeneous offspring is possible but carries some cost or is subject to some constraints. As above, we assume Gaussian selection so that an individual with phenotype z has fitness proportional to $\exp[-(\theta_t - z)^2/2]$.

One difference between the two models is that the phenotype distribution may vary over time in the pure-strategies model. When generations overlap, an evolutionarily stable population must contain multiple genotypes if $\text{Var}(\theta_t)$ is sufficiently high in the pure-strategies model (Ellner and Hairston 1994). Multiple genotypes are maintained when $\text{Var}(\theta_t)$ is high enough that a single “bet hedger” can be invaded by more risk-prone strategies that have a high relative fitness under some conditions. Fluctuations in the selection regime will then cause the allele frequencies to fluctuate from generation to generation, so that the phenotype distribution also fluctuates.

A second difference is the location of the threshold for local instability of a monomorphic population: $\sigma_0^2 > 1$ for mixed strategies, $\sigma_0^2 > 1/\gamma$ for the pure-strategies model.

Allowing mixed strategies increases the suite of possible invaders and therefore enlarges the range of conditions under which a monomorphic population can be invaded. Thus, the pure- and mixed-strategy models need not be quantitatively similar at a given set of parameters.

However, the pure- and mixed-strategy models have very similar patterns of bifurcations as σ_0^2 increases. Figure 4 shows typical simulation results for the pure-strategy model with the trait controlled by a single locus. For both haploid and diploid inheritance, we see phenotype distributions with several distinct modes, the number of modes increasing with increases in σ_0^2 , just as in the mixed-strategy model. As predicted by the local stability analysis, the average phenotype distribution is monomorphic when $\sigma_0^2 < 1/\gamma$, and becomes polymorphic when the threshold is crossed.

With diploid inheritance, the phenotype distribution need not be the same as the distribution of alleles. The allele frequency distributions show bifurcations analogous to those of the phenotype distributions for haploid inheritance, but the resulting phenotype distributions are more complicated. For example, 3 modes in the allele frequency distribution will produce 5 modes in the phenotype distribution.

Ellner and Sasaki (MS) have shown that the results seen in figure 4 hold very generally for the pure-strategies model. In particular, the ESS population generically consists of a

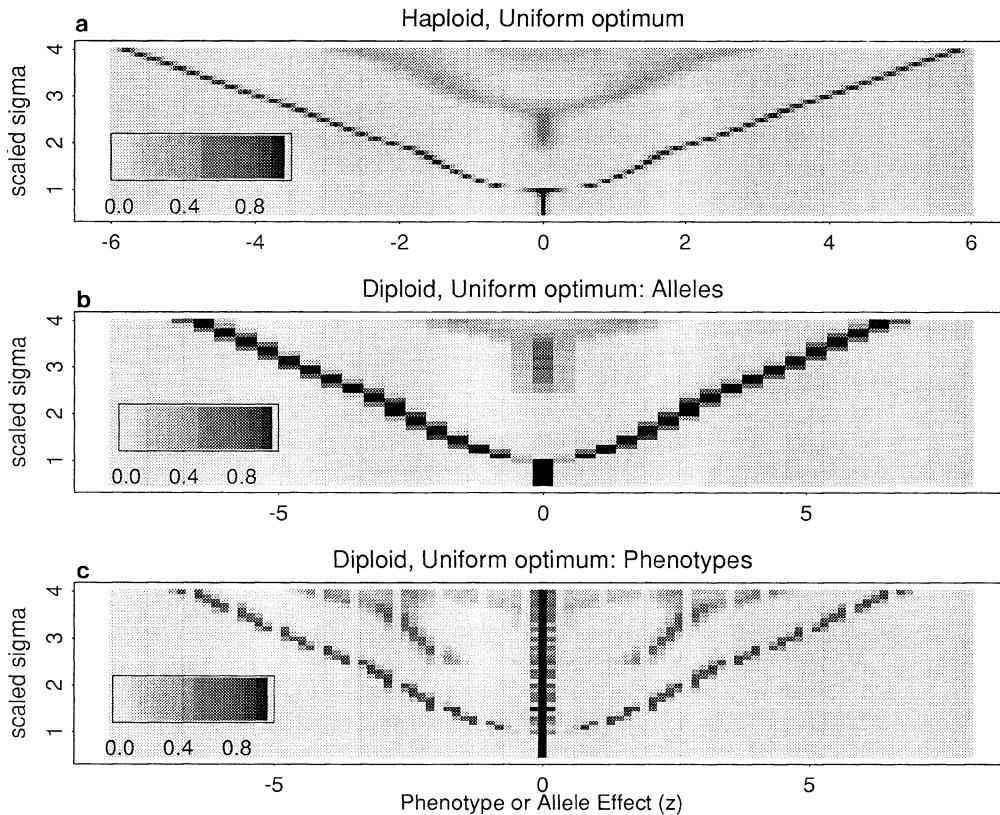


FIG. 4. Bifurcation of evolutionarily stable strategy (ESS) phenotype distributions: pure-strategy model with haploid/diploid, single-locus inheritances. Uniform distribution of θ_t is assumed. The display format is the same as figure 3 except that the vertical axis is $\sigma_0/\sigma_{\text{crit}}$, where $\sigma_{\text{crit}} = 1/\gamma^{\alpha}$ is the level at which the monomorphic distribution loses local stability. a. Haploid inheritance. Each horizontal slice represents the relative average frequency of phenotypes in the population. Average frequency was estimated by running the model for 25,000 generations and averaging frequencies after the first 5000 generations, with 201 evenly spaced phenotypes in the range -6 to 6 . b, c. Diploid inheritance. In b the horizontal axis is allele effects on phenotype, and the average frequency distribution of alleles is plotted. In c, the horizontal axis is the phenotype z and the average frequency histogram of phenotypes is plotted. The model had 41 alleles with effects ranging from -8 to $+8$, and the $(41)^2/2$ diploid genotypes were assigned to 101 evenly spaced phenotype categories in the range -8 to $+8$. Average frequency estimated as in a.

discrete set of alleles, and hence there is a discrete phenotype distribution. Local stability analysis confirms that for $Q < 1$ and γ near 1 (as in fig. 4) the initial bifurcation is to an ESS with two alleles having equal but opposite phenotypic effects; hence, the population is dimorphic for haploid inheritance and trimorphic for diploid inheritance.

For larger values of Q , or for traits controlled by several loci, the pattern of bifurcations in the pure-strategies model may be more complicated than that shown in figure 4. However, the essential qualitative result is preserved: there is a discrete distribution of alleles (and phenotypes) in the ESS, with the number of distinct types increasing as the variance of fluctuations in the selection regime is increased (Ellner and Sasaki MS). The apparently nondiscrete distributions in figure 4 are due to the finite length of the simulations: as time progresses, selection will eliminate all but a few alleles.

DISCUSSION

The present model revealed that a variety of polyphenotypic strategies should evolve in a temporally fluctuating, or coarse-grained, environment. Dividing progeny into a variety

of phenotypes pays because it reduces the variance over time of the within-generation mean fitness of a genotype. This does not take effect if the arithmetic mean genotype fitness determines the fate of the mutant, that is, if the environment is fine-grained or spatially heterogeneous. In the “mixed-strategies model,” in which an individual can produce offspring with a distribution of phenotypes, the evolutionarily stable population always consists of a single genotype. There is a unique ESS distribution that does not depend on generational overlap, and the ESS distribution generically is discrete rather than continuous. The ESS distribution is monomorphic when environmental variance is below the threshold. Beyond the threshold, there is bifurcation to the increased number of distinct phenotypes. In the pure-strategies model, in which an individual produces offspring with a single, genetically determined phenotype, the ESS population contains multiple genotypes when the environmental variance is sufficiently high. However, the phenotype distributions are similar to those in the mixed-strategies model: discrete, with an increasing number of distinct phenotypes as the environmental variance increases.

The most striking result of the mixed-strategy model is that a continuously varying selection regime favors a set of distinct morphs instead of a continuous range of phenotypes. In other words, the purpose of risk-spreading is best achieved by dividing progeny into a few blocks, and minor differences within blocks do not contribute to risk-spreading. This may occur because each phenotype keeps a high fitness in a range of environmental states around the one in which it is most fit. Therefore, two phenotypes must be sufficiently separated, otherwise a risk-spreading genotype will not get maximum performance from both of them.

Within-plant variations in seed size and shape partially conform to the discrete ESS phenotype distribution predicted by our model. The agreement is that high phenotypic variation among seeds often takes the form of “somatic seed polymorphism,” in which a single plant produces several distinct types of seeds differing in size, shape, or color (Harper 1977; Silvertown 1984). Dimorphisms are common in annual *Compositae*, but three or more distinct seed morphs also occur (e.g., in *Reichardia* and *Calendula*, Feinbrun-Dothan 1978). Dimorphisms and polymorphisms in germination behavior, not related to obvious physical differences between seed types, also occur (Silvertown 1984). The disagreement is that seed size and shape are not absolutely constant within morphs (Michaels et al. 1988). According to our model, continuous variation within a seed morph in should be opposed by selection, i.e. parental fitness would be higher if the variation could be eliminated. (Note, however, that this only refers to a set of offspring produced at a single time; seasonal trends in seed size [Cavers and Steel 1984] caused by changes in the parent or seasonal trends in parameters affecting seed success are not considered in our model). We are not aware of any experimental data bearing on this hypothesis but would be pleased to learn otherwise.

The fact that the ESS distribution is discrete in the mixed-strategies model depends on the fact that our model does not contain any intrinsic advantage to being different from other phenotypes. We have assumed a “game against the field,” such that each individual competes with all others rather than primarily competing with similar-phenotype individuals. However, in cases where intraspecific competition is strongest between similar phenotypes, an additional frequency-dependent component of fitness should be considered. We have numerically confirmed that adding this kind of frequency dependence alters the ESS phenotype distribution as expected: the discrete set of offspring types is smoothed out, that is, each discrete type is replaced by a continuous distribution in a small interval, which tends to widen as the intensity of like-with-like competition is increased.

In Seger and Brockmann’s (1987) classification of bet-hedging, our mixed-strategy model falls in the category of “don’t put all your eggs in one basket.” The defining feature is that high within-genotype (or within-clutch) variance in fitness at any one time is used to decrease the between-year fluctuation in the average fitness, at the expense of not maximizing the average fitness in any given year. There is a large literature on this type of bet-hedging, which would be impossible to review here (and redundant: see Seger and Brockmann 1987; Philippi and Seger 1989). However, two studies especially close to ours should be mentioned. Slatkin and

Lande (1976) considered a model based on quantitative genetics for evolution of the level nonheritable phenotypic variance under fluctuating selection. In their model, an offspring’s phenotype was the sum of a nonrandom mean determined its genotype, and a Gaussian random “error” with genetically determined variance, V_e . Their main finding was the existence of a threshold value of σ_θ^2 for $V_e > 0$ to evolve. Bull (1987) extended Slatkin and Lande’s results and obtained the ESS phenotypic variance as $V_e = \max\{0, \sigma_\theta^2 - \sigma_w^2\}$ where σ_w^2 is the fitness variance. Their models were much more constrained than ours—the phenotype distribution could only be a mixture of Gaussians—but the similarity of their findings to ours suggests that our results would be generalizable to multilocus quantitative traits. Indeed, analyses of a multi-locus diploid version of our pure-strategies model reveals that there is a cascade of bifurcations from monomorphism to an increased number of alleles and polymorphic loci, and increased genetic variance, as the environmental variability increases, although the genotype distribution is no longer Gaussian but discrete at least under a broad class of selection regimes (Ellner and Sasaki MS).

In general, mixed-strategy bet-hedging is superior to a genetic polymorphism of monophenotypic genotypes in that the genetically polymorphic population can always be invaded by a bet-hedger that mimics the phenotype distribution in the population. Thus, genetic polymorphism is expected only when it is costly to make different phenotypes. We also expect that mixed strategies should be more common in organisms or traits with less developmental constraints on offspring, for example, more common in plants than in vertebrates. Seger and Brockmann (1987) suggested that bet-hedging should be more common and genetic polymorphism less common in relatively short-lived species, all else being the same. This accords with our conclusion that the lower the generation overlap (γ), the greater the difference between the mixed- and pure-strategy ESSs.

We assumed that the fitness of an individual depends only on the deviation between the individual’s phenotype, z , and the optimum phenotype θ , with θ varying over time but the shape of the fitness function remaining constant. A more general analysis in which the shape of the fitness function also varies in time would be very complicated. However, regarding our finding of discrete ESSs for a wide class of fitness functions and environmental distributions, we believe that this result should hold in more general selection regimes.

We also assumed that there is no temporal autocorrelation in the environment. In general, we would expect optimal bet-hedging strategies to be affected by temporal autocorrelation (e.g., optimal dispersal rate; Cohen and Levin 1991). Here, autocorrelation would affect the ESS phenotype distribution in the pure-strategies model, because the invasion success of a mutant depends on the genotype frequencies of wild-type alleles, which are influenced by autocorrelation. However, in the mixed strategy model, autocorrelation does not affect the result of Ellner and Hairston (1994) that the ESS population cannot contain multiple genotypes. The criterion for the ESS phenotype distribution (eq. 8 above) is then not affected by autocorrelation; hence, autocorrelation has no effect whatsoever on the ESS in the mixed-strategies model.

The pure-strategy model was motivated by the high levels

of genetic variance for reproductive timing in a freshwater copepod with diapausing eggs, *Diaptomus sanguineus*. The relevance of the model to the maintenance of genetic variation in species with extended diapause or overlapping generations of adults is discussed elsewhere (Ellner and Hairston 1994). Another potential application is to the evolution of recombination. Recombination is one genetic device for producing variance in offspring phenotypes. Previous two-locus genetic models of temporally fluctuating environments fail to demonstrate a significant advantage for positive recombination (Maynard Smith 1978), except when assuming very special correlations between genotypic fitness fluctuations (Sturtevant and Mather 1938; Charlesworth 1976; Sasaki and Iwasa 1987). In a quantitative-genetic model with a fluctuating optimum (Maynard Smith 1988), high recombination would be favored in some selection regimes, but there is an implicit assumption that enough genetic variance is maintained by some other mechanism. Recombination fails to produce a robust advantage in these models because the genetic variance is depleted under fluctuating selection, which occurs because the models all assume nonoverlapping generations. If overlapping generations are assumed, genetic polymorphism at multiple loci can be maintained under fluctuating selection, and recombination might then work as a bet-hedging strategy for changing environments (Sasaki in prep.).

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APPENDIX A

In this appendix, we prove that a phenotype distribution p^* is an evolutionarily stable strategy (ESS) if and only if it maximizes the log mean growth rate $\Phi(p) = E_\theta \log \left\{ \int_{-\infty}^{\infty} w_\theta(z)p(z) dz \right\}$ and hence the ESS phenotype distribution is independent of the degree of generation overlap. We also will show that ESS phenotype distribution is symmetric around the mean optimum.

Proof: Because $w_\theta(z) \leq 1$, the integral in (6) is well defined (though it may equal $-\infty$). Suppose that p^* maximizes (6), that is, $\Phi(p) < \Phi(p^*)$ for all $p \neq p^*$. Then the boundary growth rate of p invading p^* is

$$\begin{aligned} \rho(p | p^*) &= E_\theta \log \left\{ (1 - \gamma) \frac{\int_{-\infty}^{\infty} w_\theta(z)p(z) dz}{\int_{-\infty}^{\infty} w_\theta(z)p^*(z) dz} + \gamma \right\} \\ &= E_\theta \log \left\{ \frac{(1 - \gamma) \int_{-\infty}^{\infty} w_\theta(z)p(z) dz + \gamma \int_{-\infty}^{\infty} w_\theta(z)p^*(z) dz}{\int_{-\infty}^{\infty} w_\theta(z)p^*(z) dz} \right\} \\ &= E_\theta \log \left\{ \int_{-\infty}^{\infty} w_\theta(z)\bar{p}(z) dz \right\} - E_\theta \log \left\{ \int_{-\infty}^{\infty} w_\theta(z)p^*(z) dz \right\} \\ &= \Phi(\bar{p}) - \Phi(p^*) < 0, \end{aligned}$$

where $\bar{p}(z) = (1 - \gamma)p(z) + \gamma p^*(z)$. Therefore, p^* is an ESS. However, suppose that p^* does not maximize Φ , that is, there is a $\tilde{p} \neq p^*$ such that $\Phi(\tilde{p}) \geq \Phi(p^*)$. By Jensen's inequality applied to the log function,

$$\Phi[\alpha p + (1 - \alpha)q] \geq \alpha\Phi(p) + (1 - \alpha)\Phi(q), \quad (\text{A1})$$

for all probability distributions p and q , and any $0 < \alpha < 1$, with equality only if $p = q$. Then, as above,

$$\begin{aligned} \rho(\tilde{p} | p^*) &= E_\theta \log \left\{ \frac{(1 - \gamma) \int_{-\infty}^{\infty} w_\theta(z)\tilde{p}(z) dz + \gamma \int_{-\infty}^{\infty} w_\theta(z)p^*(z) dz}{\int_{-\infty}^{\infty} w_\theta(z)p^*(z) dz} \right\} \\ &= \Phi[(1 - \gamma)\tilde{p} + \gamma p^*] - \Phi(p^*) \\ &> (1 - \gamma)\Phi(\tilde{p}) + \gamma\Phi(p^*) - \Phi(p^*) \\ &= (1 - \gamma)[\Phi(\tilde{p}) - \Phi(p^*)] \geq 0. \end{aligned}$$

and therefore p^* is not an ESS.

Because the probability distribution of θ is assumed to be symmetric around zero, we expect that the ESS phenotype distribution is also symmetric, and this is indeed the case. The symmetry of θ implies that $p(z)$ and $p(-z)$ have the same value of Φ , and then, by (A1), the symmetrized distribution $[p(z) + p(-z)]/2$ has a higher value of Φ (implying that p cannot be an ESS) unless p is symmetric.

The result that p^* is symmetric depends on our assumptions that the selection function w and the distribution of θ are both symmetric. If either of these assumptions is dropped, it may be asymmetric.

APPENDIX B

In this appendix, we prove the existence and uniqueness of an ESS phenotype distribution for the mixed-strategy model. We revert to standard real-analysis notation to emphasize that phenotype distributions may be any probability measure on the real line.

Lemma B1. Define $W_\theta(\mu) = \int w_\theta(z) d\mu(z)$, for signed measures μ with finite total variation and Gaussian selection. If $W_\theta(\mu_1) = W_\theta(\mu_2)$ at a sequence of points $\{\theta_n\}$ having a point of accumulation, then $\mu_1 = \mu_2$.

Proof: $W_\theta(\mu_1 - \mu_2)$ is an analytic function of θ ; thus, by the Identity

Theorem for analytic functions the zeros at $\{\theta_n\}$ imply that it is identically 0. Because W_θ is a convolution, taking Fourier transforms, we have $\hat{w} \cdot (\hat{\mu}_1 - \hat{\mu}_2) = 0$, where \hat{w} is the Fourier transform of the Gaussian kernel. Because $\hat{w}(t)$ is everywhere positive (Akhiezer 1988), we have $\hat{\mu}_1 = \hat{\mu}_2$ and therefore $\mu_1 = \mu_2$.

Proposition B1. Assume that (1) the support of θ is contained in some finite interval $[-A, A]$; (2) the relative fitness function $w_\theta(z)$ is positive and continuous on $[-A, A] \times [-A, A]$; and (3) it pays to be close to 0 in the sense that $w_\theta(z_1) > w_\theta(z_2)$ whenever $|z_1 - 0| < |z_2 - 0|$. Then there exists a probability measure μ^* at which $\Phi(\mu)$ is globally maximized, where

$$\Phi(\mu) = E_\theta \log \int w_\theta(z) d\mu(z).$$

Proof: We can restrict attention to μ in M_A , the set of probability measures on the interval $[-A, A]$ because assumptions (1) and (3) imply that it never pays to put probability mass outside that interval. That is, if μ is not in M_A then $\Phi(\mu) < \Phi(\mu^+)$ where

$$\mu^+(S) = \mu(S \cap [-A, A]) + \mu(A, \infty) * 1_{\{A \in S\}} + \mu(-\infty, A) * 1_{\{-A \in S\}}.$$

M_A is sequentially compact in the topology of weak convergence (by the Helly-Bray Theorem). Suppose (to show that Φ is continuous) that the sequence μ_n in M_A converges weakly to $\mu \in M_A$. For any $\theta \in [-A, A]$, $w_\theta(z)$ is a bounded, continuous function of z , therefore

$$\int w_\theta(z) d\mu_n(z) \rightarrow \int w_\theta(z) d\mu(z). \quad (\text{B1})$$

From assumption (2), there exists a constant $C > 0$ such that $1/C < w_\theta(z) < C$ for all $(\theta, z) \in [-A, A]$. Therefore, all the integrals in (B1) have logarithms bounded in absolute value by $\log(C)$; thus, by dominated convergence we have that $\Phi(\mu_n) \rightarrow \Phi(\mu)$. Thus, Φ is continuous on M_A in the topology of weak convergence. The set $\{\Phi(\mu), \mu \in M_A\}$ is bounded above by $\log(C)$, so it has a supremum Φ^* . Choose a sequence μ_n such that $\Phi(\mu_n) \rightarrow \Phi^*$. Sequential compactness implies that μ_n has a subsequence converging to a limit μ^* , and the continuity of Φ implies that $\Phi(\mu^*) = \Phi^*$.

Proposition B2. For Gaussian selection, μ^* is unique and is the only local maximum of Φ .

Proof: To show that μ^* is unique, suppose that $\Phi(\mu) = \Phi(\mu^*)$ but $\mu \neq \mu^*$. By the lemma, the support of θ contains at least one point θ_0 at which $W_\theta(\mu) \neq W_\theta(\mu^*)$; hence (by continuity), $W_\theta(\mu) \neq W_\theta(\mu^*)$ for all θ in some interval around θ_0 . For θ in that interval, we have, by Jensen's inequality,

$$\log W_\theta(\{\mu + \mu^*\}/2) > \{\log W_\theta(\mu) + \log W_\theta(\mu^*)\}/2,$$

and integrating with respect to $q(\theta)$ gives $\Phi(\{\mu + \mu^*\}/2) > \Phi(\mu^*)$. This contradicts the definition of μ^* .

Let μ be any probability measure other than μ^* . By the last proposition, $\Phi(\mu^*) > \Phi(\mu)$; thus, once again, by Jensen's inequality, we have that

$$\Phi[(1 - \varepsilon)\mu + \varepsilon\mu^*] > (1 - \varepsilon)\Phi(\mu) + \varepsilon\Phi(\mu^*) > \Phi(\mu),$$

for any $0 < \varepsilon < 1$; hence, μ is not a local maximum of Φ .

APPENDIX C

We derive here the conditions (8) for $p^*(z)$ to be globally evolutionary stable in the mixed-strategy model; that is, for $p^*(z)$ to refuse invasion by any rare mutant. Because the ESS in the mixed-strategy model is the only local maximum of the objective function Φ , the problem to be solved is to find a phenotype distribution p that locally maximizes

$$\Phi(p) = E_\theta \log \left[\int_{-\infty}^{\infty} w_\theta(z)p(z) dz \right], \quad (\text{C1})$$

under the constraints

$$\int_{-\infty}^{\infty} p(z) dz = 1 \quad (\text{C2a})$$

$$p(z) \geq 0, \quad \forall z \in R. \quad (\text{C2b})$$

This constrained maximization problem is solved by the method of Lagrange multipliers. Let λ be the Lagrange multiplier and define the augmented objective function

$$L(p) = \Phi(p) - \lambda \int_{-\infty}^{\infty} p(z) dz. \quad (\text{C3})$$

Then a phenotype distribution p^* gives the local optimal distribution, if

$$\Delta L \equiv L(p^* + \Delta p) - L(p^*) < 0 \quad (\text{C4})$$

holds for arbitrary deviation function $\Delta p(z)$, which satisfies $|\Delta p(z)| < \varepsilon$, and

$$\Delta p(z) \geq 0 \quad \text{for } z \text{ at which } p^*(z) = 0. \quad (\text{C5})$$

The latter comes from (C2B) for $p^* + \Delta p$. Because it follows from (C1) that

$$\Phi(p^* + \Delta p) - \Phi(p^*) = E_\theta \log \left\{ 1 + \int_{-\infty}^{\infty} \left(\frac{w_\theta(z)}{\bar{w}_\theta} \right) \Delta p(z) dz \right\}, \quad (\text{C6})$$

where $\bar{w}_\theta = \int_{-\infty}^{\infty} w_\theta(z) p^*(z) dz$, we have

$$\Delta L = E_\theta \log \left\{ 1 + \int_{-\infty}^{\infty} \frac{w_\theta(z)}{\bar{w}_\theta} \Delta p(z) dz \right\} - \lambda \int_{-\infty}^{\infty} \Delta p(z) dz. \quad (\text{C7})$$

Expanding the RHS of (C7) in a Taylor series with respect to Δp , we then have

$$\begin{aligned} \Delta L &= \int_{-\infty}^{\infty} \left\{ E_\theta \left[\frac{w_\theta(z)}{\bar{w}_\theta} \right] - \lambda \right\} \Delta p(z) dz \\ &\quad - \frac{1}{2} \left(\int_{-\infty}^{\infty} E_\theta \left[\frac{w_\theta(z)}{\bar{w}_\theta} \right] \Delta p(z) dz \right)^2 + O(\varepsilon^3). \end{aligned} \quad (\text{C8})$$

In order that $\Delta L < 0$ holds for any deviation function $\Delta p(z)$ satisfying (C5), it is necessary that

$$\begin{aligned} E_\theta \left[\frac{w_\theta(z)}{\bar{w}_\theta} \right] &= \lambda, & \text{if } p^*(z) > 0, \\ &\leq \lambda, & \text{if } p^*(z) = 0. \end{aligned} \quad (\text{C9})$$

Conversely, if p^* satisfies (C9), the first term in the RHS of (C8) is 0 or negative and the second term is negative, which guarantees $\Delta L < 0$ for any $\Delta p(z)$ satisfying (C5). Finally, we can verify that $\lambda = 1$ by taking the average of both sides of (C9) over the distribution $p^*(z)$.

APPENDIX D

Proposition: If the tail of the environmental distribution $q(\theta)$ decays slower than Gaussian, that is, if there are some $c > 0$, $s > 1$, and $T > 0$, such that

$$q(\theta) \geq ce^{-\theta^2/2s^2} \quad \text{for all } \theta > T, \quad (\text{D1})$$

then the ESS phenotype distribution has unbounded support.

Proof: Suppose that p has support contained in an interval $[-A, A]$, $A > 0$. Without loss of generality, we can assume that $A > T$. We will show that $\varphi(z)$, the relative fitness of z -individual, diverges to infinity as $z \rightarrow \infty$, implying that p is not an ESS.

When $\theta > A$, individuals with the extreme phenotype $z = A$ enjoy an advantage over individuals with intermediate phenotype; that is, $w_\theta(y) < w_\theta(A)$ for any y in $(-A, A)$. The mean fitness of the population is then smaller than that of an individual with phenotype A :

$$\bar{w}_\theta < w_\theta(A) \quad \text{if } A < \theta. \quad (\text{D2})$$

Therefore,

$$\begin{aligned} \varphi(z) &= E_\theta \left[\frac{w_\theta(z)}{\bar{w}_\theta} \right] > \int_A^{\infty} \frac{w_\theta(z)}{\bar{w}_\theta} q(\theta) d\theta > \int_A^{\infty} \frac{w_\theta(z)}{w_\theta(A)} q(\theta) d\theta \\ &= \exp\{(A^2 - z^2)/2\} \int_A^{\infty} \exp\{(z - A)\theta\} q(\theta) d\theta. \end{aligned} \quad (\text{D3})$$

Using the inequality (D1), we have

$$\varphi(z) > \exp\{(s^2 - 1)z^2/2 - s^2Az + (s^2 - 1)A^2/2\}$$

$$\int_A^{\infty} c \exp[-\{\theta - s^2(z - A)\}^2/2s^2] d\theta. \quad (\text{D4})$$

As $z \rightarrow \infty$, the second factor in the right-hand side of (D4) converges to a finite constant, whereas the first factor diverges to $+\infty$ because $s > 1$.

APPENDIX E. RARITY OF CONTINUOUS ESSs

We here show that in the mixed-strategies model, (i) the ESS distribution is discrete, rather than continuous, for all distributions $q(\theta)$ with bounded support and that (ii) the ESS distribution is generically discrete in the sense that it holds for all $q(\theta)$ in a generic (open and dense) set of L_1 probability densities. We will prove these for a wider class of fitness functions than that assumed in the text (i.e., Gaussian). We assume that the fitness function is given by a kernel function $k(x)$ as $w_\theta(z) = ck(z - \theta)$ where (1) $k(x)$ is analytic; (2) $k(x)e^{cx}$ is absolutely integrable for any $\alpha > 0$; (3) $k(x) > 0$. c is a normalizing factor such that $\int_{-\infty}^{\infty} k(x) dx = 1$. Note that $k(x)$ is not necessarily symmetric or unimodal.

Proposition 1. The ESS phenotype distribution is discrete for all distributions $q(\theta)$ with bounded support.

Proof: Suppose that the support of an ESS phenotype distribution $p(z)$ includes an interval (or a convergent sequence), which we denote C . Then the relative fitness of z individuals must be the same for all z in C :

$$\varphi(z) = \int_{-\infty}^{\infty} \frac{w_\theta(z)}{\bar{w}_\theta} q(\theta) d\theta = 1, \quad \text{for all } z \in C \quad (\text{E1})$$

with $\bar{w}_\theta = \int_{-\infty}^{\infty} w_\theta(z) p(z) dz$. Recall that the fitness function is of the form $w_\theta(z) = c \cdot k(z - \theta)$, where k satisfies the properties (1)–(3) above. Letting $h = q/(k^* p)$ or

$$h(\theta) = q(\theta) / \int_{-\infty}^{\infty} k(\theta - z) p(z) dz,$$

and noting $\int_{-\infty}^{\infty} k(z - \theta) d\theta = 1$, the condition (E1) is rewritten as

$$\int_{-\infty}^{\infty} k(z - \theta)[h(\theta) - 1] d\theta = 0, \quad \text{for all } z \in C. \quad (\text{E2})$$

This integral defines an analytic function of z ; hence, by the Identity Theorem for analytic functions, (E2) must be zero for all z . We then take Fourier transforms in (E2)

$$\hat{k} \cdot \hat{g} = 0, \quad (\text{E3})$$

where $g(\theta) = h(\theta) - 1 \cdot \hat{k}(\omega) = \int_{-\infty}^{\infty} k(x)e^{-i\omega x} dx$ is an analytic function of ω as shown in the following. Let us extend the domain of \hat{k} to the complex plane and regard ω as a complex variable. Such \hat{k} is well defined by the assumption (2). Integrating \hat{k} along a closed contour L surrounding ω gives

$$\oint_L \hat{k}(\zeta) d\zeta = \oint_L \int_{-\infty}^{\infty} k(x)e^{-i\zeta x} dx d\zeta = \int_{-\infty}^{\infty} k(x) \oint_L e^{-i\zeta x} d\zeta dx.$$

The inner integral in the right hand-side vanishes by Cauchy's theorem because an exponential function is regular (analytic). Thus, $\oint_L \hat{k}(\zeta) d\zeta = 0$ for all ω , and hence \hat{k} is analytic (and also when restricted to the real axis). That \hat{k} is analytic means it has only isolated zeros, for otherwise it is identically zero (by the Identity Theorem). Hence we conclude from (E3) that $h(\theta) = 1$ except isolated points. Thus, we have

$$q(\theta) = \int_{-\infty}^{\infty} k(\theta - z) p(z) dz, \quad (\text{E4})$$

pointwise, except possibly at an isolated set of points. Consequently (E4) holds in L_1 (i.e., for both sides regarded as elements of L_1). Any such q clearly has unbounded support because k has unbounded support; hence, for any q with bounded support p^* must be discrete.

Proposition 2. Any ESS phenotype distribution is discrete for a set of probability densities $q(\theta)$, which is generic (contains an open, dense set) in the set $F = \{q \in L_1(-\infty, \infty) : q \geq 0, \|q\|_1 = 1\}$ with the L_1 topology.

Proof: Define $R = \{k^* p : p \text{ is a Borel probability measure on the real line}\} \subset F$. We have shown above that if an ESS distribution exists that is not discrete, then $q \in R$. Any element of R is Lipschitz (in fact

differentiable); thus, the complement of R contains all step functions and therefore is dense in F . To complete the proof, it is therefore sufficient to show that R is closed. Thus, let q_n be a sequence in R , that is, $q_n = k^*p_n$ for probability measures p_n , and suppose that $q_n \rightarrow q$ in L_1 . We need to show that $q \in R$.

It is easy to see that the Lipschitz constants of q_n and q are both no larger than C , the Lipschitz constant of k . Let y be any point at which $q_n(y) \neq q(y)$, and let $d = |q_n(y) - q(y)|$. For x in the interval $(y - d/2C, y + d/2C)$, we have $|q_n(x) - q(x)| \geq d - 2C|y - x|$, and, therefore, $\|q_n - q\|_1 \geq d^2/2C$. Thus, q_n must converge to q pointwise as well as in L_1 .

Let χ_n denote the probability measure corresponding to q_n . The set $\{q_n\}$ is clearly mass preserving (in the sense of Breiman 1992, ch. 8) because it is L_1 convergent. The relationship $q_n = k^*p_n$ can be restated

in terms of random variables as follows: χ_n is the distribution of $K + P_n$, where K and P_n are independent random variables with K having density function $k(z)$ and P having probability measure p_n . Because $\Pr\{K \geq 0\} = \alpha > 0$, this implies that

$$\begin{aligned}\chi_n(T, \infty) &= \Pr\{K + P_n > T\} \geq \Pr\{K \geq 0 \text{ and } P_n > T\} \\ &= \alpha \cdot p_n(T, \infty).\end{aligned}$$

Consequently, the set $\{p_n\}$ also is mass preserving and therefore has a weak limit point; that is, there is a probability measure p such that some subsequence p_{n_j} converges in distribution to p . Let $\tilde{q} = k^*p$. For any fixed θ , weak convergence implies that $q_{n_j}(0) \rightarrow \tilde{q}(\theta)$. Because $q_{n_j} \rightarrow q$ pointwise we must have $q = \tilde{q}$ and therefore $q \in R$.

Note that if $q(\theta)$ has unbounded support, the results of Appendix B may not hold: an ESS may fail to exist, or may be nonunique.