Evolutionary Ecology Research, 2002, **4**: 1–25

The influence of life-history differences on the evolution of reaction norms

Gerdien de Jong* and Narayan Behera

Evolutionary Population Biology, University of Utrecht, Padualaan 8, 3584 CH Utrecht, The Netherlands

ABSTRACT

We analyse a general model for the evolution of reaction norms in a heterogeneous environment. In the life-cycle, zygotes disperse. Development to the adult phenotype is in one environment and selection in another environment after adult migration. Lack of adult migration always leads to the optimum reaction norm. Adult migration after development and unpredictability of selection are necessary for the evolved reaction norm to deviate from the optimum reaction norm. Zygote dispersal and adult migration, therefore, play very different roles in the evolution of reaction norms. The life-cycle includes three stages in which density-dependent number regulation can take place: in the environment of development and in the environment of selection before and after selection. Differences in productivity between patches – a source–sink structure – cause major deviations in phenotypic evolution. Weak optimizing selection leads to a compromise reaction norm heavily influenced by productivity differences. Strong optimizing selection leads to the phenotypic optima for the character states and might lead to a polymorphic reaction norm; the condition is that density-dependent number regulation is present after selection. Zygote dispersal around the parental patch causes density-dependent number regulation in the environment of development to influence the evolved reaction, as it is now effectively after selection. The influence of density-dependent number regulation, therefore, depends strongly upon the exact life-cycle.

Keywords: density dependence, dispersal stages, ESS phenotypic plasticity, heterogeneous environment, polymorphism.

INTRODUCTION

The natural world is heterogeneous, both in time and in space. To cope with this heterogeneity, many organisms are phenotypically plastic, minimizing fitness differences between environments by adaptively different developmental trajectories in phenotypic traits (Thompson, 1991). Phenotypic plasticity in a given trait evolves under natural selection. The major question in models of the evolution of phenotypic plasticity is to what ESS reaction norm different life histories and environmental conditions lead. The optimum

* Author to whom all correspondence should be addressed. e-mail: g.dejong@bio.uu.nl

reaction norm is defined as the collection of optimum phenotypes over environments. If separate populations existed for each environment, these optimum phenotypes would be reached under optimizing selection.

The life history of an organism strongly determines the outcome of selection on phenotypic plasticity. In the simplest quantitative genetic model (Via and Lande, 1985), zygotes disperse at random from a fully mixed zygote pool, and individuals are under optimizing selection in their patch of development. Under these simple conditions of total zygote dispersal and absence of adult migration, the evolved reaction norm equals the optimum reaction norm. Gavrilets and Scheiner (1993) introduced temporal variation next to spatial variation in their model of the evolution of reaction norms, and found that temporal variation between development and selection influenced the evolved reaction norm. An evolved linear reaction norm was shallower than the optimum reaction norm. De Jong (1999) showed that the evolved reaction norm deviates from the optimum reaction norm if adult migration leads to unpredictable selection. Unpredictability of selection leads to a fitness value that is an average of fitnesses in several environments. The averaging of fitness leads to an ESS reaction norm that is not the optimum reaction norm, but a compromise over the selected optimum phenotypes. In the simulation of Scheiner (1998), a life history of locally breeding parents and local development of juveniles was implicit; density regulation occurred in the juvenile stage. Again, the evolved phenotype in each environment proved a compromise between the selected optima.

Explicit demographic models of reaction norm evolution have been investigated by Houston and McNamara (1992), Kawecki and Stearns (1993) and Sasaki and de Jong (1999). Houston and McNamara (1992) used a model where the number of female descendents depended on the state of the individual. The ESS phenotypes over environments do not equal the optimum phenotypes, but some compromise between the states. Kawecki and Stearns (1993) explicitly used source–sink demographics in their model of selection on a plastic trait in an age-structured population. They showed that the evolved optimized reaction norm in a spatially heterogeneous environment deviates from the optimal trait values in isolated populations. Sasaki and de Jong (1999) included density-dependent number regulation in the model of de Jong (1999); again, the result depended upon adult migration. Unpredictability of selection led to an evolutionarily stable bet-hedging reaction norm, constituting a compromise between the phenotypic optima in the different patches, if selection is weak, but led to the phenotypic optima and might lead to polymorphism, if selection is strong. The influence of density-dependent number regulation on the evolved reaction norm is strong if number regulation is after selection but absent if it occurs in the environment of development.

Quantitative genetic models and demographic models reach a similar result, an evolved compromise reaction norm. In the models of Houston and McNamara (1992) and of Kawecki and Stearns (1993), offspring disperse. However, in quantitative genetics models, adult migration is necessary to evolve the compromise reaction norm. The dispersing life-history stage differs between the models. We want to spell out what migration stage in the life-cycle is crucial for the evolved ESS reaction norm to deviate from the optimum reaction norm.

In the present model, both zygote dispersal and adult migration are present. We first ask whether zygote dispersal and adult migration play a fundamentally different role in the evolution of reaction norms. Second, we wish to determine how the evolved ESS

phenotypes differ from the optimum phenotypes for different models of zygote dispersal and number regulation over the life-cycle.

We plan to study the effect of number regulation in different stages of the life history in more detail than in Sasaki and de Jong (1999). Sasaki and de Jong (1999) distinguished between number regulation before selection in the environment of development and after selection in the environment of selection. We add the possibility of number regulation before selection but in the environment of selection. This is to address the question of whether it is the relative timing of selection and number regulation or the occurrence in the environment of development or the environment of selection that influences the evolution of reaction norms. Finally, we investigate the interaction between selection intensity and density dependence in the appearance of polymorphism in reaction norms.

THE MODEL

Life-cycle

We consider the evolution of the reaction norm of a quantitative trait in a heterogeneous environment. The population is haploid and asexual. The phenotype of an individual depends on its genotype and the environment of development. The reaction norm of a genotype consists of all phenotypes developed in patches with different values of an environmental variable.

Zygotes arrive in a patch in the environment of development. Density-dependent number regulation occurs in each patch. After development in one patch and completion of the individuals' phenotype, surviving adult individuals migrate to another patch. In this patch, adults are subject to density-dependent number regulation, selection and again density-dependent number regulation. Viability selection is optimizing; the optimum phenotype varies with an environmental variable. Surviving individuals reproduce and die; asexual offspring disperse to a patch in the environment of development. At this point there are two options in the model: a 'zygote pool' (Fig. 1A) or 'zygote dispersal' (Fig. 1B). In the first case, all zygotes are assembled in a common pool from which they enter the different patches of development with a fixed probability. In the second case, the probability for a zygote to enter a given development patch depends upon the patch in which the parent reproduces. In the zygote pool model, the correlation between parental patch and development patch of the offspring equals zero; in the zygote dispersal model, this correlation is positive, up to 1 if all offspring remain in their parent's patch.

Model specification

In the environment of development and the environment of selection, there are n values of an environmental variable, different between patches. A juvenile develops in patch x and obtains the genotypic value g_x according to the wild-type reaction norm $\mathbf{g} = [g_x]$. In generation t, the $N_x(t)$ juveniles that arrived in patch x are subject to density-dependent number regulation according to a probability of survival of

$$u_x \equiv U_x[N_x(t)] = \exp\{-\alpha_x N_x\} \tag{1}$$

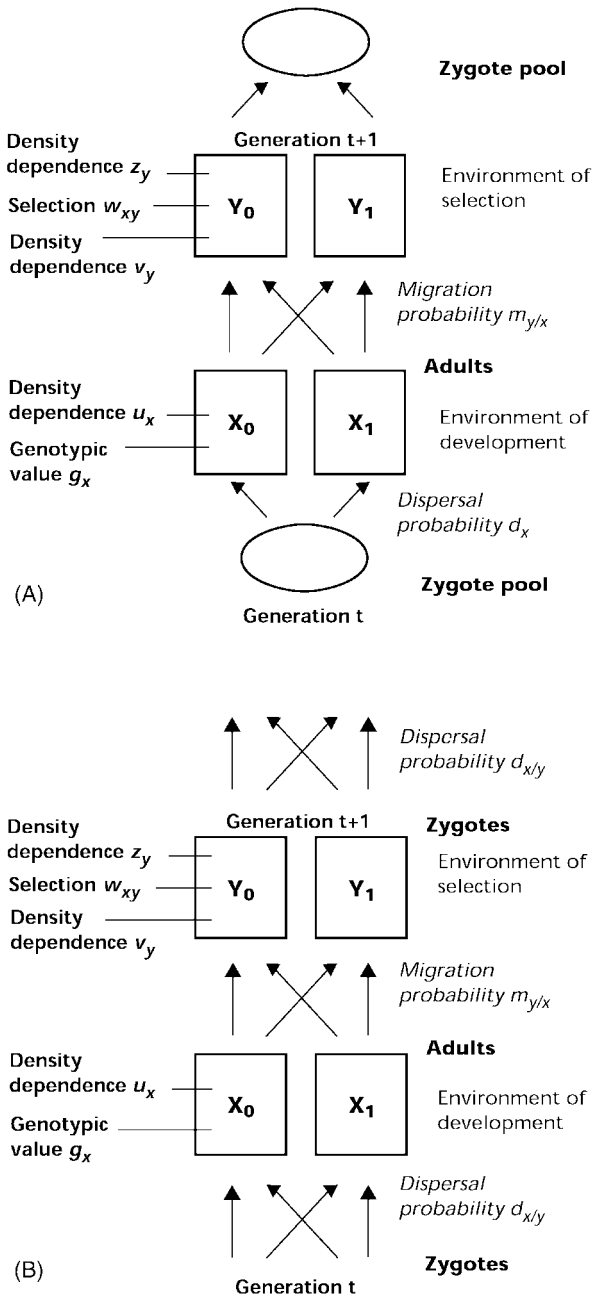


Fig. 1. Life-cycle showing development, selection and density-dependent number regulation stages: (A) zygote pool model, (B) zygote dispersal model.

where α_x is the sensitivity to density regulation in patch x. A surviving individual migrates from its development patch x to a selection patch y with a conditional probability $m_{y|x}$, where

$$\sum_{y=1}^{n} m_{y|x} = 1$$

for each x. The number of individuals in patch y after migration and before selection equals

$$N_{1,y}(t) = \sum_{x=1}^{n} N_x u_x m_{y|x} \tag{2}$$

Density-dependent number regulation in patch y before selection leads to a probability of survival of

$$v_y \equiv V_y[N_{1,y}(t)] = \exp\{-\beta_y N_{1,y}\} \tag{3}$$

where β_y denotes the sensitivity to density regulation before selection in patch y. In patch y, Gaussian optimizing selection on the phenotypic trait g_x occurs. The viability of an individual that developed in patch x (with phenotype g_x) and moved to selection patch y is given by

$$w_{xy}^{g} \equiv W_y[g_x] = \exp\{-s(\theta_y - g_x)^2\} \tag{4}$$

where θ_y is the optimum trait value in patch y and $s = 1/(2\sigma^2)$ measures the intensity of optimizing selection in Gaussian selection of width σ^2. After selection, the number of individuals in selection patch y is given by

$$N_{2,y}(t) = N_{1,y}(t)\ v_y w_{xy}^{g} \tag{5}$$

Density-dependent number regulation after selection leads to a probability of survival of

$$z_y \equiv Z_y[N_{2,y}(t)] = \exp\{-\gamma_y N_{2,y}\} \tag{6}$$

where γ_y is the sensitivity in patch y to density regulation after selection. Each surviving individual reproduces with fecundity F. Zygote offspring disperse from patch y to one of the development patches x with a conditional probability $d_{x|y}$, where

$$\sum_{x=1}^{n} d_{x|y} = 1$$

No genotypic differences exist in sensitivity to density. All genetic differences are contained in the reaction norm $\mathbf{g} = [g_x]$.

Fitness

In the zygote pool model, the population of zygotes is fully mixed before dispersing to the development patches x. The dispersal probability d_x to a patch x is, therefore, equal for each patch y and equal to the frequency of occurrence of the patches x. As the population is fully mixed in the zygote pool, recurrence equations of the numbers of each genotype can be written down from zygote pool to zygote pool. At equilibrium number, the expectation of fitness W_g for a particular genotype $\mathbf{g} = [g_x]$ can be found from the recurrence equations (Sasaki and de Jong, 1999) as

$$\mathrm{E}[W_g] = \textstyle\sum_x \sum_y d_x u_x^* m_{y|x} v_y^* w_{xy}^{g} z_y^* F \tag{7}$$

Weak selection at stable equilibrium number leads to a compromise ESS reaction norm defined by

$$g_x^* = \sum_y m_{y|x} v_y^* w_{xy}^{g*} z_y^* \theta_y / \sum_y m_{y|x} v_y^* w_{xy}^{g*} z_y^* \tag{8}$$

The environment of development plays no role in the evolved genotypic value in the zygote pool model. The evolved compromise reaction norm is weighted towards the optimum of the selection environment with least stringent density regulation and highest immigration.

In the zygote dispersal model, no stage of the life-cycle has a fully mixed population. Therefore, no obvious choice is present for the life-stage on which to base a recurrence equation. We count adults leaving their patch of development and count the total number of each genotype after a one-generation interval. We define fitness as the expected number of adult offspring of adult individuals leaving their patch of development, for each genotype. Therefore, we use the population growth rate of each population of one genotype. This is an adequate measure of fitness, as we are dealing with asexuals.

The expected number of adult offspring leaving any development patch k in generation $t + 1$, of a single adult individual of genotype **g** in generation t leaving development patch x going to selection patch y equals:

$$K_{kyx}^g = K_{kyx} w_{xy}^g \tag{9}$$

where

$$K_{kyx} = m_{y|x} v_y z_y F\, d_{k|y} u_k \tag{10}$$

The total number of adults of a genotype in generation $t + 1$ equals

$$N^g(t+1) = \sum_k \sum_y \sum_x N_x^g(t) K_{kyx} w_{xy}^g \tag{11}$$

Genotypic fitness is defined as the expected contribution to the next generation averaged over all individuals of a genotype in the present generation. The number of individuals of the genotype with strategy $\mathbf{g} = [g_x]$ leaving any development patch x in generation t is $N_x^g(t)$. The expected contribution to the next generation of individuals with genotype **g**, therefore, equals the growth rate λ^g of the population of genotype **g:**

$$\mathrm{E}[W^g] = N^g(t+1)/N^g(t)$$

$$\mathrm{E}[W^g] = \sum_k \sum_y \sum_x N_x^g(t) K_{kyx} w_{xy}^g / \sum_x N_x^g(t) \tag{12}$$

Simplifying yields (Appendix 1):

$$W^g = \sum_k \sum_x \frac{p_x^g}{p^g(t)} f_x K_{kyx} w_{xy}^g \tag{13}$$

where f_x is the frequency of individuals in patch x in generation t, p_x^g is the frequency of genotype **g** in patch x, and $p^g(t)$ is the frequency of genotype **g** in the total population. This expression for fitness can be used in a quantitative genetics approach (Appendix 1). However, finding the expected mean genotypic value at the end of selection involves taking the derivative of fitness (Lande, 1979; de Jong, 1999), which results in complications if equation (13) is used (Appendix 3).

An ESS approach is possible (cf. Sasaki and de Jong, 1999). At stable equilibrium number in a population with one genotype $\mathbf{g}^{\mathbf{r}}$, the expected number of descendants leaving patch k in generation $t + 1$ of an individual leaving patch x in generation t becomes

$$a^*_{kx} = \sum_y K^*_{kyx} w^g_{xy} \tag{14}$$

Averaging over all patch types, fitness (and population growth rate) in the population becomes

$$\mathrm{E}[W^g] = \sum_k \sum_y \sum_x N^*_x K^*_{kyx} w^r_{xy} / N^* = \lambda^* \tag{15}$$

The condition giving the ESS reaction norm is that no rare mutant can invade in a population in stable number: fitness of the resident genotype is at a maximum and equal to 1 (Appendix 2). Mutation is supposed to occur with equal probability in all individuals and over all patches and to be rare. Population size is not affected on invasion, nor are all density-dependent viabilities. Fitness of the resident genotype $\mathbf{g^r}$ is given by equation (15). Fitness of a mutant genotype $\mathbf{g^m}$ is given by

$$\mathrm{E}[W^m] = \sum_k \sum_y \sum_x N^*_x K^*_{kyx} w^m_{xy} / N^* \tag{16}$$

The condition for the resident strategy of genotypic values $\mathbf{g^r}$ to be a globally stable ESS is that for all mutant strategies $\mathbf{g^m}$ their fitness is less than the fitness of 1 of the resident strategy $\mathbf{g^r}$ (Appendix 2). Therefore, for all $\mathbf{g^m} \neq \mathbf{g^r}$,

$$W(\mathbf{g^m} \mid \mathbf{g^r}) = \sum \sum \sum f^*_x K^*_{kyx} w^m_{xy} < 1 \tag{17}$$

To find the fitness maximum indicating the ESS strategy for genotypic value in all environments, the derivatives of fitness towards the genotypic values in all patches of development have to be found. This can be done by using the derivative of the eigenvalue of the matrix defined by the elements a^*_{kx}, as given in equation (14) (Caswell, 1989). This eigenvalue represents the growth rate λ^g of an equilibrium population of genotype $\mathbf{g}$, and is identical to the fitness of genotype $\mathbf{g}$. The derivative of λ^g with respect to an element a^*_{kx} is specified in Appendix 3. This derivative involves both the relative frequencies of individuals arriving in a patch (the relative numbers f^*_x) and the relative frequencies of individuals contributing from a patch (the relative reproductive values r^*_k). The derivative of λ^g with respect to the genotypic value in any patch x given specific fitness functions is specified in Appendix 4.

The ESS genotypic value again involves a compromise between different optima. It is convenient to define the 'flow', for Gaussian selection equal to

$$q_{yx} = \frac{\sum_k r^*_k K^*_{kyx} w^{g*}_{xy}}{\sum_y \sum_k r^*_k K^*_{kyx} w^{g*}_{xy}} \tag{18}$$

Using these q_{yx} as probabilities for a path from x to k yields the implicit solution for the compromise genotypic value in environment x after Gaussian selection:

$$g^*_x = \frac{\sum_k \sum_y r^*_k K^*_{kyx} w^{g*}_{xy} \theta_y}{\sum_k \sum_y r^*_k K^*_{kyx} w^{g*}_{xy}} = \sum_y q_{yx} \theta_y = \mathrm{E}[\theta_y \mid x] \tag{19}$$

With weak Gaussian selection, fitness has a maximum at g^*_x; g^*_x is an ESS under weak selection. Density-dependent number regulation u^*_x in the environment of development now influences the evolved reaction norm, in contrast to the situation in the zygote pool model (equation 8). Reproductive value r^*_k appears, representing the equilibrium contribution from a patch. The evolved compromise reaction norm is additionally weighted by the conditional expectation of the productivity in the environments of development. That is, high

productivity due to less stringent density regulation in x_0 strengthens selection of g_0 towards θ_0 if offspring only incidentally disperse from their parents' patch. Moreover, low dispersal from the parental patch implies that the density-dependent viability u_x^* depends upon the number of adults surviving selection in that patch.

Dispersal and migration

Equation (19) predicts an ESS reaction norm g_x^* that clearly depends upon the zygote dispersal probabilities and adult migration probabilities. From equation (19), several special cases can be formulated, corresponding to different life-cycles.

Zygotes disperse at random, adults migrate

The prediction of the compromise ESS reaction norm g_x^* under weak selection can be compared between the cases of zygote dispersal (equation 19) and zygote pool (equation 8). The zygote pool model is equivalent to a dispersal probability to patch x of d_x, independent of patch y of the parent. Everything involving the index k disappears, and the expected value of g_x simplifies to

$$g_x^* = \sum_y m_{y|x} v_y^* w_{xy}^{g*} z_y^* \theta_y / \sum_y m_{y|x} v_y^* w_{xy}^{g*} z_y^* \tag{20}$$

Equation (20) equals equation (8), despite the reference to a different point in the life-cycle in the two models. The density dependence u_k in the environment of development has no influence on the ESS reaction norm. Reproductive value r_k^* plays no role. In fact, here we show why reproductive value can be ignored in standard population genetics models of selection in patchy situations.

Zygotes do not disperse, adults migrate

If all offspring develop in their parents' patch of selection, dispersal probability $d_{k|y} = 1$ for $k = y$ and $d_{k|y} = 0$ for $k \neq y$. Density dependence u_k refers to environment y of the parents: $u_{k(t+1)}^* = u_{y(t)}^*$. The expected value of g_x^* simplifies to:

$$g_x^* = \sum_y m_{y|x} v_y^* w_{xy}^{g*} z_y^* u_y^* r_y^* \theta_y / \sum_y m_{y|x} v_y^* w_{xy}^{g*} z_y^* u_y^* r_y^* \tag{21}$$

In this case, density dependence u_x and density dependence z_y play a similar role, both depending on the number of animals after selection. Reproductive value plays a role.

Adults do not migrate, zygotes disperse

If adults do not migrate, $m_{y|x} = 1$ for $y = x$ and $m_{y|x} = 0$ for $y \neq x$; selection is predictable. In equation (19), the summation over patches y disappears. The summation over patches k is as a consequence identical in numerator and denominator to equation (19). The equation simplifies to

$$g_x^* = \theta_x \tag{22}$$

for all distributions of zygotes. That is, the optimum reaction norm evolves not only in the case of a zygote pool (Via and Lande, 1985), but also for all dispersal patterns of zygotes, if selection is fully predictable from development. The evolved reaction norm

shows no influence of any other source of mortality, for instance density-dependent number regulation.

Density-dependent number regulation

Density-dependent viability depends upon the number in a patch and upon the values of the coefficients α_x, β_y and γ_y that rule the sensitivity to density in the viabilities u_x, v_y and z_y. If the sensitivity to density is the same over all patches, lower α_x would lead to higher productivity and higher equilibrium number, and higher α_x would lead to lower productivity and lower equilibrium number. Any difference between patches in, for instance, α_x, causes a productivity difference between patches, such that a patch with low α_x has higher productivity and a patch with high α_x has lower productivity than it would have in an environment with all α_x identical. The presence of another patch with lower α_x causes a patch with any particular value of α_x to be a 'sink' patch that contributes less to the population than it would have done if all patches had their own α_x value. The presence of another patch with higher α_x causes a patch with any particular value of α_x to be a 'source' patch that contributes more to the population than it would have done if all patches had their own α_x value. A large difference between patches in sensitivity coefficients at any particular density-dependent stage will cause a very low viability in the patches with high values of the sensitivity coefficients. The more productive patches will dominate the numbers in the population.

The presence of 'source' and 'sink' environments has a significant influence on the evolved reaction norm. To appreciate the effect of source and sink environments, it is useful to use the 'flow' of individuals through the patches of environments of development and of selection. The flow summarizes the relative contribution to the equilibrium population of organisms meeting each sequence of patches x, y and k, according to

$$q_{yx} = \frac{\sum_k r_k^* K_{kyx}^* w_{xy}^{g*}}{\sum_y \sum_k r_k^* K_{kyx}^* w_{xy}^{g*}} \tag{18}$$

The flow is counted from just before migration of adults in generation t to just before migration of adults in generation $t + 1$. Flow acts as if it is the biological frequency of the environment combinations. In general, the flow includes the reproductive value of the patches k. In the zygote pool model, the flow simplifies to

$$q_{yx} = m_{y|x} v_y^* w_{xy}^{g*} z_y^* / \sum_y m_{y|x} v_y^* w_{xy}^{g*} z_y^* \tag{23}$$

Given the definition of flow,

$$g_x^* = \sum_y q_{yx} \theta_y = \mathrm{E}[\theta_y | x] \tag{24}$$

in all models with equal selection intensity s in all environments y. Equation (24) applies equally to the zygote pool model and the zygote dispersal model. The difference between the zygote pool model and zygote dispersal model is in the flow.

Equal viability due to density-dependent number regulation clearly extinguishes the effect of density dependence on selection. If $v_y^* = v^*$ and $z_y^* = z^*$ in all the patches of the environment of selection, while $u_k^* = u^*$ in all patches of the environment of development, the flow becomes for Gaussian selection:

$$q_{yx} = m_{y|x} w_{xy}^{g*} / (\sum_y m_{y|x} w_{xy}^{g*}) \tag{25}$$

The evolved phenotype g_x is the same as for a pure genotypic frequency model, given we are using Gaussian optimizing selection. If we used quadratic optimizing selection, the flow would be identical to the migration probabilities from x to y.

Viability differs over patches given differences in density-dependent number regulation. Both environments x and y need to possess at least one source patch, with a low density dependence sensitivity coefficient, for the population to exist. Any large difference between density dependence sensitivity coefficients causes the patch with highest value of the sensitivity coefficient to be a sink patch. High values of β_1 or γ_1 in patch y_1 (much higher than in the other patches) imply $v_1^* \to 0$ or $z_1^* \to 0$ and, therefore, $q_{yx} \to 0$. Such a deep sink patch y_1 does not contribute much to selection. This is easily seen for two patches, y_0 and y_1. If y_1 is a deep sink patch, almost all animals will pass through patch y_0 and be selected towards its local optimum trait value θ_0. The effect of differences in the sensitivity coefficients need not be that extreme, but are present for all $v_1^* < v_{y\neq 1}^*$ or $z_1^* < z_{y\neq 1}^*$. Any productive patch y with higher viability due to less stringent density-dependent number regulation will have relatively more animals passing through it than a sink patch and will pull the balance of selection towards the patch's phenotypic optimum θ_y.

In the zygote pool model, a deep sink patch x_1 in the environment of development does not contribute any animals to the population. Selection towards the ESS phenotype in non-sink patches is not affected, but no ESS phenotype for the sink patch x_1 exists: the evolved phenotype will be totally due to genetic drift (Sasaki and de Jong, 1999). However, in the zygote dispersal model, a sink patch $x_1 = k_1$ tends towards the role of sink patches y_1. In such a sink patch, density-dependent viability is lower than in the other patches; for some patches y with a high probability $d_{k|y}$ for a zygote to develop in the sink patch, this will imply that $\sum_k d_{k|y} u_k^*$ might be considerably lower than for the other patches y. Such patches y that entail a high probability for their zygotes to disperse to a sink patch k_1 are relatively ineffective in selecting towards their phenotypic optimum.

Differential productivity of patches, therefore, strongly affects the evolution of reaction norms through redirecting the flow of animals through the patches.

Strong selection and stability of the ESS reaction norm

Selection strength in any patch y depends on the parameter $s = 1/(2\sigma^2)$ in the Gaussian optimizing selection function w_{xy}^g. The width of the Gaussian optimizing selection function is given by $\sigma^2 = 1/(2s)$; σ^2 is comparable to a variance. In patch y, optimizing selection is towards the local optimum θ_y. The deviation of the phenotypic values subject to selection in patch y from θ_y can be large relative to σ, or small. If the deviation of the phenotypic values is large relative to σ, selection on the phenotypes is relatively strong. If all deviations of phenotypic values from the local optimum are less than σ, by definition selection is weak. The second derivative of the Gaussian selection function is negative for $\theta_y - \sigma < g_x < \theta_y + \sigma$ and positive for $\theta_y - \sigma > g_x > \theta_y + \sigma$ in the tails. Weak selection, therefore, corresponds to a negative second derivation of the selection function.

The compromise evolved reaction norm is found when fitness has a maximum at g_x^*. The second derivative of fitness towards genotype decides whether fitness is at a maximum or at a minimum (Appendix 5). Under weak Gaussian selection, the second derivative of the Gaussian selection function is negative at g_x^*, which ensures a fitness maximum and stability of g_x^*. Strong Gaussian selection implies that the second derivative of the Gaussian selection function is positive at g_x^* and a fitness minimum is expected.

The evolved compromise phenotype $g_x^* = \mathrm{E}[\theta_y | x]$ in environment x is evolutionarily stable in a population of stable number if the conditional variance in optimum phenotypic values, as encountered in that development patch, is less than the selection width:

$$\mathrm{var}(\theta | x) = \sum_y q_{yx} (\theta_y - g_x^*)^2 < \sigma^2 \tag{26}$$

(Appendix 5; Geritz *et al.*, 1998; Sasaki and de Jong, 1999; Geritz and Kisdi, 2000). The definition of q_{yx} depends on the mode of zygote dispersal. Equation (26) is valid for weak selection. Under strong selection, $\mathrm{var}(\theta | x) > \sigma^2$, and the compromise reaction norm is not evolutionarily stable. The evolved reaction norm under strong selection might be monomorphic or polymorphic. A monomorphic reaction norm has the phenotypic optima $g_x^* = \theta_{y=x}$; for two patches, the optima $g_0^* = \theta_0$ and $g_1^* = \theta_1$ appear. However, under strong selection, it is also possible that at the end of selection two phenotypes develop in one patch; if this is the case, the reaction norm is polymorphic (Sasaki and de Jong, 1999). For two patches, this polymorphism in the reaction norm implies that, at evolutionary equilibrium, two genotypes are present. The first genotype has, for instance, $g_0^* = \theta_0$ and $g_1^* = \theta_1$, and the second genotype has $g_0^* = \theta_0$ and $g_1^* = \theta_0$; the presence of two different phenotypes developing in patch x_1 applies when patch y_1 is a sink patch and patch y_0 is a source patch. The monomorphic optimum reaction norm always evolves when density dependence is present before selection. The frequency dependence implicit in density dependence after selection opens the way for polymorphism.

The value of the selection intensity s that implies stability and a compromise reaction norm depends upon the sensitivity to density in the successive density-dependent stages. The sensitivities α_x, β_y and γ_y in part determine the flow through the environments and, therefore, the variance in optimum phenotypic values as encountered from a development patch. The exact value of the selection intensity s at which stability breaks down differs between the zygote dispersal model and the zygote pool model for identical density dependence, as the flow through the environments differs between the models. The flow is more balanced between the patches in the zygote dispersal model, which easily leads to a higher variance in optimum phenotypic values and more polymorphism.

ITERATIONS

We performed numerical iterations in a two-patch version of the model to show clearly the influence of density-dependent number regulation, to demonstrate the difference between the zygote dispersal model and the zygote pool model and to document the change from weak selection to strong selection. We assumed haploid, single-locus inheritance with many alleles coding for different reaction norms. The allele i will cause the phenotype of an individual to be $g_0(i)$ if it develops in patch $x = 0$ and to be $g_1(i)$ if the individual develops in patch $x = 1$. Each phenotype g_0 takes one of 21 equally spaced values between -1 and $+1$; each phenotype g_1 takes one of 21 equally spaced values between 0 and 2. Each of the 21 phenotypes in patch 0 is combined with each of 21 phenotypic values in patch 1. Therefore, there are $21 \times 21 = 441$ possible alleles, all of which segregate in the initial population at equal frequencies. Adult migration from patch x to patch y is with conditional probability $m_{y|x}$, where the probability of remaining in the same patch is higher than the probability of leaving; we only use symmetric probabilities $m_{0|0} = m_{1|1} > m_{0|1} = m_{1|0} = 1 - m_{1|1}$. Gaussian selection has the optimum $\theta_0 = 0$ in patch $y = 0$ and $\theta_1 = 1$ in patch $y = 1$. Zygote dispersal from patch y to patch x has conditional probability $d_{x|y}$, where the probability of the zygotes

remaining in the parental patch is higher than the probability of leaving: $d_{0|0} = d_{1|1} > d_{0|1} = d_{1|0} = 1 - d_{0|0}$. After iteration for 2000 generations, the phenotypic distributions in each patch x are scored.

Density dependence and weak selection

Weak selection implies that the compromise ESS reaction norm is associated with fitnesses in that part of the Gaussian fitness function where the second derivative of fitness towards the trait value is negative (Appendix 5). The negative second derivative implies that averaging over fitness values leads to a higher fitness than the fitness at the average genotypic value and, therefore, to fitness maximization (cf. Ruel and Ayres, 1999). The presence of density-dependent number regulation strongly influences the ESS reaction norm. The extent of the influence depends on the stage of the life-cycle in which density dependence occurs, on the mode of zygote dispersal and on the difference in density dependence between the two patches.

Density regulation in the patch of development influences the ESS reaction norm only if the patch of development is correlated with the parent's patch (Fig. 2A). Given weak selection, equal sensitivity to density ($\alpha_0 = \alpha_1$) and, therefore, equal productivity in both patches, and optimum phenotypes $\theta_0 = 0$ and $\theta_1 = 1$, the compromise ESS phenotypes in patch x_0 and patch x_1 will be numerically very near the migration probabilities: $g_0 m_{01}$ and $g_1 m_{11}$ (equation 19; Fig. 2A). In the zygote pool model, these same values for the ESS phenotypes are found whatever the density regulation in the patch of development is. In the zygote dispersal model, the shapes of the curves in Fig. 2A can be explained from the productivity differences between the patches x_0 and x_1. Given the positive correlation between parental patch and zygote patch, the effect of selection in patch y_1 is annihilated if patch x_1 is a very low productivity sink patch; the effect of selection in the high productivity patch y_0 pushes the character state g_0 strongly towards $\theta_0 = 0$. Phenotype g_1^* will be increasingly subject to genetic drift as the number of individuals surviving in patch x_1 decrease.

Density regulation after adult migration but before selection might lead to a marginally different ESS reaction norm than density regulation after selection. When the population faces density regulation after migration (before or after selection), the evolved reaction norm differs between the zygote pool model and the zygote dispersal model (Fig. 2B). The effect of a sink patch y_1 is stronger for the zygote dispersal model, as the flow of individuals within a generation is more localized to patches 0 or 1. Selection on individuals developing in patch x_0 leads to the phenotypic optimum $g_0 = \theta_0 = 0$ at wide differences in density dependence between patches, as migration to patch y_1 effectively becomes zero. An individual developing in the less promising patch x_1 is selected to a phenotype close to the optimum of the more productive patch y_0, as the flow through the high productivity patch y_0 is promoted over the flow through patch y_1. At wide differences in density dependence between patches y_0 and y_1, virtually no individuals make it through the line of '1' patches, and genetic drift takes over in determining the phenotype of those that do.

Strong selection and polymorphism

Strong selection implies that the ESS reaction norm associates with fitnesses in that part of the Gaussian fitness function where the second derivative of fitness towards the trait value is positive. The consequence is disruptive selection towards the optima $\theta_0 = 0$ and $\theta_1 = 1$, rather

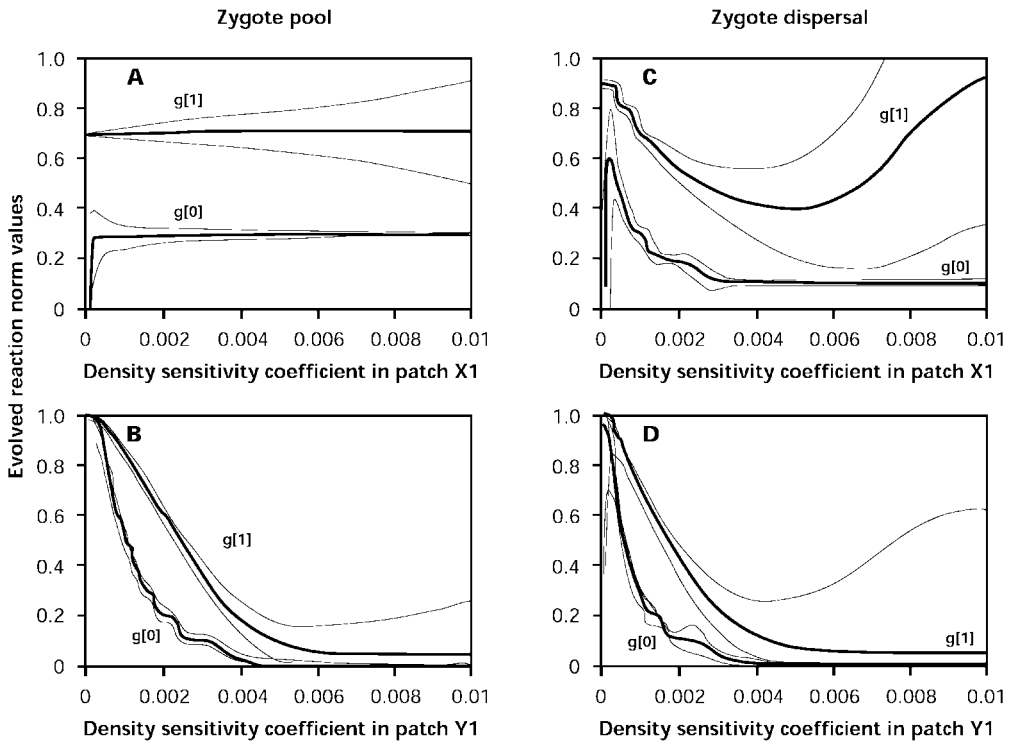
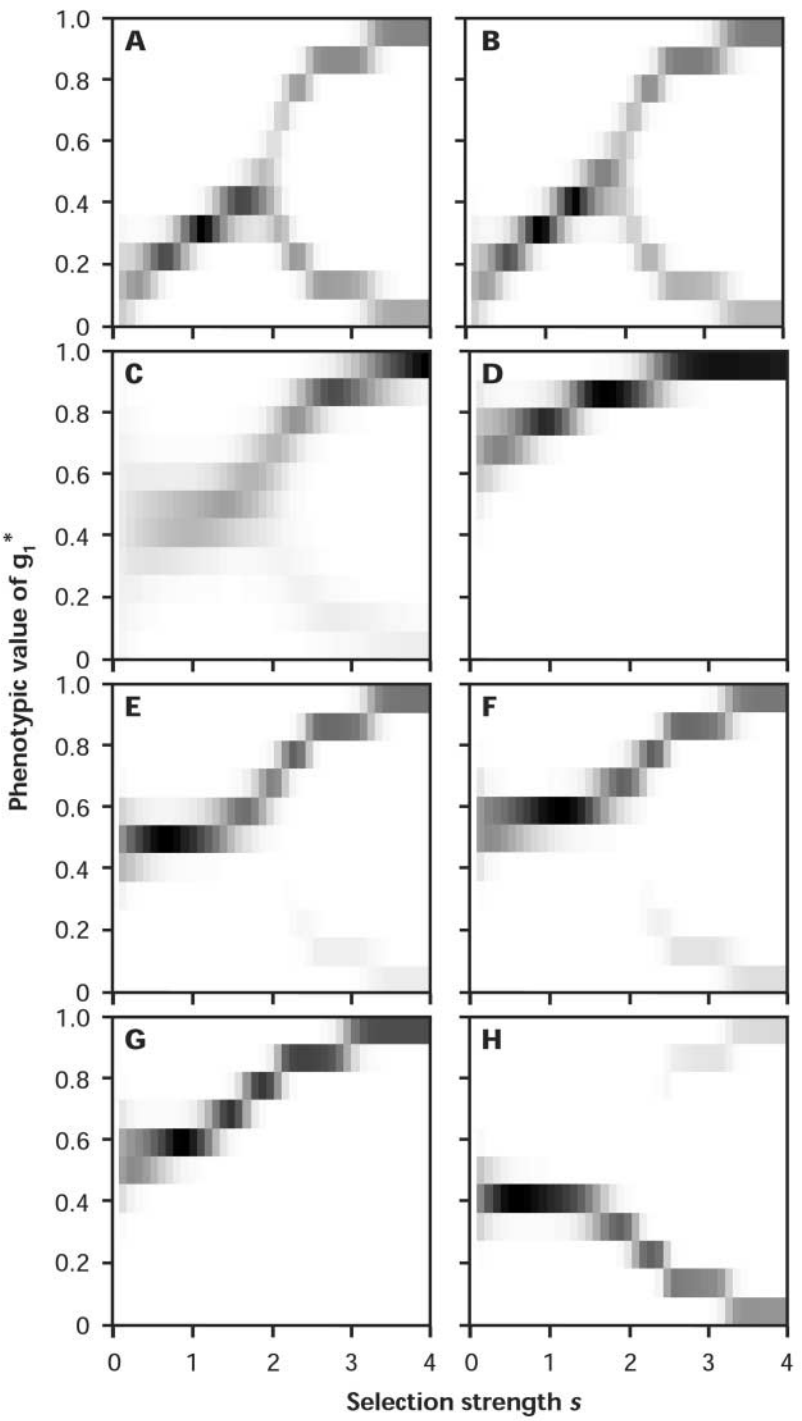


Fig. 2. The influence of density-dependent number regulation on the compromise reaction norm under weak selection. The average phenotypes g_0 and g_1 that evolve in patches x_0 and x_1 are indicated by the bold lines; the thin lines indicate one standard deviation. In all cases, $s = 0.125$, $d_{0|0} = d_{1|1} = 0.8$, $m_{0|0} = m_{1|1} = 0.7$. The optimum phenotypic values are $\theta_0 = 0$ and $\theta_1 = 1$. The expected values of g_0 and g_1 with no influence of density-dependent number regulation would be $g_0 = 0.3$ and $g_1 = 0.7$. These expected values are found if zygotes disperse at random and density dependence is in the environment of development (A), and if the density dependence sensitivities are identical in patches x_0 and x_1 (C) or patches y_0 and y_1 (B, D). (A, B) Zygote pool model; (C, D) zygote dispersal model.

(A, C) Density dependence α_1 in patch x_1 is variable (x-axis); density dependence in patch x_0 is fixed at $\alpha_0 = 0.001$; density dependence in the selection patches is absent: $\beta_0 = \beta_1 = 0$, $\gamma_0 = \gamma_1 = 0$. (B, D) Density dependence after selection γ_1 in patch y_1 is variable (x-axis); density dependence after selection in patch y_0 is fixed at $\gamma_0 = 0.001$; density dependence in the development patch and density dependence before selection in the selection patch are absent: $\alpha_0 = \alpha_1 = 0$, $\beta_0 = \beta_1 = 0$.

than stabilizing selection around a compromise reaction norm. Effectively, the fitness profile changes from convex to concave (Levins, 1968).

Often, strong selection leads to a monomorphic reaction norm with $g_0^* = \theta_0 = 0$ and $g_1^* = \theta_1 = 1$. However, the compromise reaction norm found under weak selection might branch into two optima if the conditional variance in the optima is larger than the width of the Gaussian selection function (equation 26). This conditional variance in optimum phenotypic values has a maximum value of 0.25, given that $\theta_0 = 0$ and $\theta_1 = 1$. The lowest selection intensity that allows polymorphism is, therefore, $s = 2$. A conditional variance of 0.25 requires that the flow through the two selection patches from a development patch


A
B
C
D
E
F
G
H
1.0
0.8
0.6
0.4
0.2
0
0 1 2 3 4
Phenotypic value of g_1^*
Selection strength s

is equal. Polymorphism at asymmetric flow starts at higher s. Different types of polymorphism in reaction norms are found. A polymorphic reaction norm can be due to polymorphism in g_1^*: ($g_0^* = \theta_0 = 0$, $g_1^* = \theta_0 = 0$) and ($g_0^* = \theta_0 = 0$, $g_1^* = \theta_1 = 1$). To a much lesser extent, polymorphism can be due to polymorphism in g_0^*: ($g_0^* = \theta_0 = 0$, $g_1^* = \theta_1 = 1$) and ($g_0^* = \theta_1 = 1$, $g_1^* = \theta_1 = 1$).

The two general conditions for the existence of polymorphism are density-dependent number regulation after selection and productivity differences between patches due to any of the density-dependent stages. A productivity difference due to density dependence after selection leads to polymorphism in the phenotype g_1 associated with the 'sink' patch y_1, for both the zygote dispersal and zygote pool models (Fig. 3A,B). If zygotes (mostly) stay on to develop in their parental patch, density dependence in the environment of development becomes effectively density-dependent after selection, and can promote polymorphism. Polymorphism due to a productivity difference caused by density dependence in the environment of development is, therefore, only possible in the zygote dispersal model (Fig. 3C,D). Density dependence after migration but before selection itself never causes polymorphism; however, if a productivity difference originates at this life-history stage and density-dependent number regulation is present after selection too, again polymorphism is present in the phenotype associated with the 'sink' patch (Fig. 3E,F).

Fig. 3. The influence of selection strength s (x-axis) on the evolved phenotype g_1^* (y-axis). The number of individuals with a given evolved phenotypic value g_1^* is indicated by the density of print. Maximum numbers differ between cases. A compromise evolved phenotypic value is present at lower selection strengths. Selection strength $s = 2.0$ is the lowest possible value for polymorphism to occur. In most cases, patch y_1 is the 'sink' patch with more intense density-dependent number regulation and lower productivity. In all cases, adult migration is given by $m_{0|0} = m_{1|1} = 0.7$.

(A, B) Density-dependent number regulation is present after selection and not in the other two potentially density-dependent stages: $\alpha_0 = 0.0$, $\alpha_1 = 0.0$; $\beta_0 = 0.0$, $\beta_1 = 0.0$; $\gamma_0 = 0.001$, $\gamma_1 = 0.004$. In (A), $d_{0|0} = d_{1|1} = 0.6$ for the zygote dispersal model; in (B), $d_0 = 0.6$ for the zygote pool model. Polymorphism appears with both modes of zygote dispersal, differing in detail.

(C, D) Density-dependent number regulation is present in the environment of development and not in the other two potentially density-dependent stages: $\alpha_0 = 0.001$, $\alpha_1 = 0.004$; $\beta_0 = 0.0$, $\beta_1 = 0.0$; $\gamma_0 = 0.0$, $\gamma_1 = 0.0$. In (C), $d_{0|0} = d_{1|1} = 0.8$ for the zygote dispersal model; in (D), $d_0 = 0.8$ for the zygote pool model. Polymorphism appears only with the zygote dispersal model.

(E, F) Density-dependent number regulation is present both before and after selection in the environment of selection; productivity differences are caused by density dependence before selection. In (E), density dependence is weak: $\beta_0 = 0.001$, $\beta_1 = 0.002$; $\gamma_0 = 0.001$, $\gamma_1 = 0.001$; in (F), density dependence is strong: $\beta_0 = 0.001$, $\beta_1 = 0.012$; $\gamma_0 = 0.01$, $\gamma_1 = 0.01$. In both (E) and (F), $d_{0|0} = d_{1|1} = 0.6$ for the zygote dispersal model and $\alpha_0 = 0.0$, $\alpha_1 = 0.0$.

(G, H) Density-dependent number regulation is present both before and after selection in the environment of selection; productivity differences are caused by density dependence before selection: $\alpha_0 = 0.0$, $\alpha_1 = 0.0$; $\beta_0 = 0.001$, $\beta_1 = 0.002$; $\gamma_0 = 0.001$, $\gamma_1 = 0.001$. Both (G) and (H) represent the zygote pool model. In (G), the probability for a zygote to go to the source patch is $d_0 = 0.6$; in (H), the probability for a zygote to go to the sink patch is $d_1 = 0.6$. Polymorphism appears when the probability to reach a patch x_1 is higher, leading to a higher probability for the 'sink' patch y_1.

In all these cases, the biology implies that the probability to survive density dependence in the 'sink' patches of the environment of selection is so low that it is advantageous for an organism to take the chance of migrating to the 'wrong' patch (y_1 from x_0 and y_0 from x_1) and risking selection there. Remember, the model does not involve any habitat choice!

The extent of polymorphism varies widely between the zygote pool model and the zygote dispersal model (Fig. 4A,B). In both cases, the prevalence of polymorphism increases when density dependence in the 'sink' patch becomes more restrictive and fewer animals are able to make it through patch y_1. Dispersal and migration probabilities also influence the occurrence of polymorphism. More animals going to a stronger sink patch leads to polymorphism more easily.

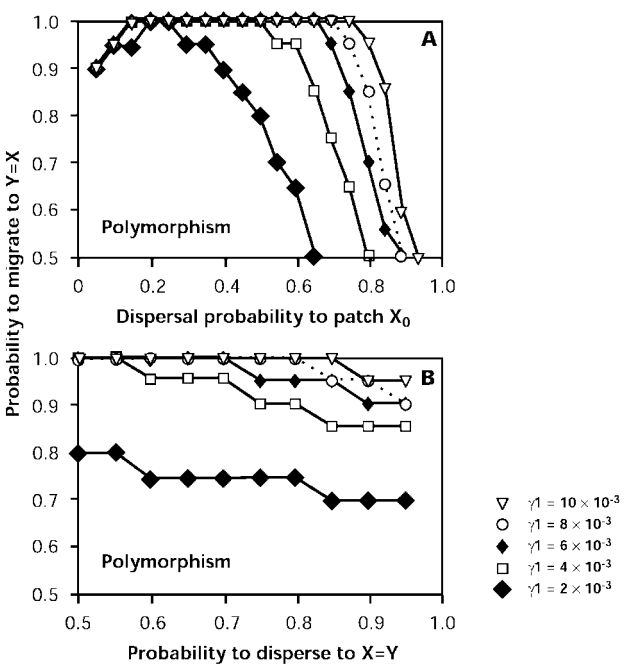


Fig. 4. Limiting conditions on polymorphism in the phenotype developed in patch x_1, if two development patches x_0 and x_1 and two selection patches y_0 and y_1 are present. The x-axis gives the probability of zygote dispersal from patch x_0 to patch y_0, and the identical probability of zygote dispersal from patch x_1 to patch y_1. The y-axis gives the probability of adult migration from patch y_0 to patch x_0, and the identical probability of adult migration from patch y_1 to patch x_1. The sink patch is patch y_1. The extent of polymorphism differs due to the probabilities of zygote dispersal and adult migration, and the strength of density-dependent number regulation after selection in patch y_1. The region of polymorphism is to the lower left, and increases with wider differences in density dependence between the patches. In all cases, $s = 3.5$, $\alpha_0 = \alpha_1 = 0$, $\beta_0 = \beta_1 = 0$, $\gamma_0 = 0.001$.

(A) Zygote pool model: dispersal probability d_0 to patch x_0 on x-axis, migration probability $m_{0|0} = m_{1|1}$ on y-axis; for different values of γ_1, the strength of density dependence after selection in patch y_1. (B) Zygote dispersal model: dispersal probability $d_{0|0} = d_{1|1}$ on x-axis, migration probability $m_{0|0} = m_{1|1}$ on y-axis; for different values of γ_1, the strength of density dependence after selection in patch y_1.

DISCUSSION

Life-history detail matters. The details of the life history determine which reaction norm will evolve: the optimum reaction norm, a compromise reaction norm or a polymorphic reaction norm. The model is written to capture maximum detail, given a discrete generation model: two dispersal stages are present, before development and after development; the stages are referred to as 'zygote dispersal' and 'adult migration'; three density-dependent stages are present, associated with development or with selection, and before and after selection. Therefore, it is possible to trace the conditions that underlie each result.

The first conclusion is that a compromise reaction norm requires migration between development and selection. A compromise reaction norm requires a probability distribution between the environment where the phenotype is determined and the environment in which its fitness is decided. Fitness is given by the expectation over several selective environments. The two-habitat model of Houston and McNamara (1992) states that in a 'good' patch a female decides to lay a clutch of size M_1, and in a 'bad' patch a female decides to lay a clutch of size M_2. Offspring disperse from a good patch with probability $(1 - \alpha_1)$ to a good patch and with probability α_1 to a bad patch. Houston and McNamara (1992) take V_1^* and V_2^* to be the reproductive values at equilibrium of animals in the good and bad patches, respectively. An adult in the good patch should maximize $(1 - \alpha_1)\ V_1^* + \alpha_1 V_2^*$. The model of Houston and McNamara (1992), therefore, involves a migration stage separating an animal's decision to lay a clutch of a certain size from selection on that clutch size. Overall fitness is arrived at as an expectation of future offspring over selection environments. The averaging over the selection environments causes a compromise reaction norm that does not correspond to the optimum reaction norm that would be reached if selection was separate in each environment (cf. Stearns and Koella, 1986). The model description (Houston and McNamara, 1992) states that offspring disperse before their phenotype is fixed. However, paying attention to the structure of the model shows that Houston and McNamara's offspring dispersal corresponds structurally to adult migration here. Their product of migration probability and reproductive values corresponds to the sum of the flow from a given development patch, and their compromise reaction norm in clutch size corresponds to the compromise genotypic value for patch x as found in equation (20) here. In Kawecki and Stearns (1993), offspring disperse: but fitness of an adult with a given life-history strategy is found as the expectation over environments of the reproductive value at birth of its offspring. This again means that selection on a phenotype is arrived at from an average over selective environments. It is not the name of the dispersing stage but fitness as an expectation over different environments after fixation of the phenotype that is crucial for the evolved reaction norm to be a compromise between phenotypic optima.

The second conclusion is the very different role of density-dependent number regulation in the different stages of the life history. Density-dependent number regulation after frequency-independent selection adds a frequency-dependent component to fitness (Prout, 1980). Density-dependent number regulation after selection facilitates the emergence of polymorphism if selection is strong (Sasaki and de Jong, 1999). Density-dependent number regulation in the environment of development is co-opted as density-dependent number regulation after selection if zygotes remain in the parental environment. In this case, density-dependent number regulation in the environment of development can lead to polymorphism, again if selection is strong. Biologically, having many animals with the optimum phenotype implies high mortality due to density-dependent number regulation after selec-

tion. A polymorphism arises in the following way. The probability to survive for an animal that develops in patch x_1 and has the 'wrong' phenotype 0 equals the probability to survive for an animal that develops in patch x_1 and has the 'right' phenotype 1. If the animal has phenotype 0, it bets on going to y_0 (with low probability), having the optimum phenotype there and encountering low mortality due to density dependence. If it has phenotype 1, the animal bets on going to y_1 (with high probability), having the local optimum phenotype and encountering high mortality due to density dependence.

The polymorphism found here in the 'sink' line of environments is comparable to evolutionary branching in a model of adaptive dynamics in a heterogeneous environment (Geritz *et al.*, 1998; Kisdi and Geritz, 1999; Geritz and Kisdi, 2000). Geritz *et al.* (1998) introduce a haploid version of Levene's (1953) soft selection model to demonstrate evolutionary dynamics and branching. In Kisdi and Geritz (1999) and Geritz and Kisdi (2000), two phenotypic optima are present over many patches. As here, the evolved phenotype is a compromise given by the weighted average of the optima, if the variance in the optima is less than the width of the Gaussian optimizing selection function. Evolutionary branching occurs if the variance in the optima is higher. Evolutionary branching requires frequency-dependent selection; the amount of frequency dependence introduced by setting a fixed ceiling to population number after the completion of density- and frequency-independent selection suffices. The fixed ceiling to the number emerging from a patch is the distinguishing feature in Levene's model. Levene's soft selection is a special case of density-dependent number regulation after selection (Holsinger and Pacala, 1990; Sasaki and de Jong, 1999).

The third conclusion is that weak and strong selection show very different adaptive patterns. Weak selection leads to a compromise reaction norm. The optimum phenotypes might not be found. Weak selection, therefore, implies ongoing selection and a genetic load. The observation will be of persistent stabilizing selection on the phenotypes over environments. Such stabilizing selection over environments has, for instance, been found in damselflies (Anholt, 1991). On the other hand, productivity differences between patches have a strong influence on the compromise reaction norm. So much so, that the evolved reaction norm approaches a specialist for the productive environment. In the non-productive 'sink' environment, selection might be ineffective and genetic drift might take over. The total situation would look much like adaptation to the common environment, at genetic variation under stress (Hoffmann and Merilä, 1998). Strong selection implies disruptive selection towards the phenotypic optima, even if some individuals always end up in a totally inappropriate selection environment. Only the optimum phenotypes evolve, even if polymorphism in the reaction norm occurs. Perhaps the cryptic mimicry of the caterpillars of *Nemoria arizonaria* provide a pertinent example (Greene, 1989).

The fourth conclusion concerns the role of 'reproductive value'. Reproductive value can be defined and found both in the zygote pool model and the zygote dispersal model, but it only influences the outcome of selection in the zygote dispersal model. Adult migration is decisive for the influence of reproductive value to appear. The life-cycle, therefore, decides crucially how to find the evolutionary outcome of selection in a heterogeneous environment.

ACKNOWLEDGEMENTS

We thank Carolien de Kovel for useful comments. N.B. thanks the Netherlands Organization for Scientific Research (NWO) for support (grant #805-36-188).

REFERENCES

Anholt, B.R. 1991. Measuring selection on a population of damselflies with a manipulated phenotype. *Evolution*, **45**: 1091–1106.

Caswell, H. 1989. *Matrix Population Models*. Sunderland, MA: Sinauer Associates.

de Jong, G. 1999. Unpredictable selection in a structured population leads to local genetic differentiation in evolved reaction norms. *J. Evol. Biol.*, **12**: 839–851.

Gavrilets, S.Y. and Scheiner, S.M. 1993. The genetics of phenotypic plasticity. V. Evolution of reaction norm shape. *J. Evol. Biol.*, **6**: 31–48.

Geritz, S.A.H. and Kisdi, E. 2000. Adaptive dynamics in diploid, sexual populations and the evolution of reproductive isolation. *Proc. R. Soc. Lond. B*, **267**: 1671–1678.

Geritz, S.A.H., Kisdi, E., Meszéna, G. and Metz, J.A.J. 1998. Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. *Evol. Ecol.*, **12**: 35–57.

Greene, E. 1989. A diet-induced developmental polymorphism in a caterpillar. *Science*, **243**: 643–646.

Hoffmann, A.A. and Merilä, J. 1999. Heritable variation and evolution under favourable and unfavourable conditions. *Trends Ecol. Evol.*, **14**: 96–101.

Holsinger, K.E. and Pacala, S.W. 1990. Multiple niche polymorphism in plant populations. *Am. Nat.*, **135**: 301–309.

Houston, A.I. and McNamara, J.M. 1992. Phenotypic plasticity as state-dependent life-history decision. *Evol. Ecol.*, **6**: 243–253.

Kawecki, T.J. and Stearns, S.C. 1993. The evolution of life histories in heterogeneous environments: optimum reaction norms revised. *Evol. Ecol.*, **7**: 155–174.

Kisdi, E. and Geritz, S.A.H. 1999. Adaptive dynamics in allele space: evolution of polymorphism by small mutations in a heterogeneous environment. *Evolution*, **53**: 993–1008.

Lande, R. 1979. Quantitative analysis of multivariate evolution, applied to brain size : body size allometry. *Evolution*, **33**: 402–416.

Levene, H. 1953. Genetic equilibrium when more than one niche is available. *Am. Nat.*, **87**: 331–333.

Levins, R. 1968. *Evolution in Changing Environments: Some Theoretical Explorations*. Princeton, NJ: Princeton University Press.

Prout, T. 1980. Some relationship between density-independent selection and density-dependent population growth. *Evol. Biol.*, **13**: 1–68.

Ruel, J.J. and Ayres, M.P. 1999. Jensen's inequality predicts effects of environmental variation. *Trends Ecol. Evol.*, **14**: 361–366.

Sasaki, A. and de Jong, G. 1999. Density-dependence and unpredictable selection in a heterogeneous environment: compromise and polymorphism in the ESS reaction norm. *Evolution*, **53**: 1329–1342.

Scheiner, S.M. 1998. The genetics of phenotypic plasticity. VII. Evolution in a spatially structured environment. *J. Evol. Biol.*, **11**: 303–320.

Stearns, S.C. and Koella, J.C. 1986. The evolution of phenotypic plasticity in life-history traits: predictions of reaction norms for age and size at maturity. *Evolution*, **40**: 893–913.

Thompson, J.D. 1991. Phenotypic plasticity as a component of evolutionary change. *Trends Ecol. Evol.*, **6**: 246–249.

Via, S. and Lande, R. 1985. Genotype–environment interaction and the evolution of phenotypic plasticity. *Evolution*, **39**: 505–522.

APPENDIX 1: GENOTYPIC FITNESS IN A MODEL WITH DISPERSAL BETWEEN PATCHES

In a population, two asexual genotypes A and B are present. The population is divided over a number of patches. The numbers of genotypes A and B completing their development in patch x in generation

t are given by $N_x^A(t)$ and $N_x^B(t)$. The total number of individuals completing development in patch x equals $N_x(t)$, the total number of individuals of genotype A over all patches $N^A(t)$ and the total number of individuals in the population $N(t)$. The frequency of individuals leaving patch x is, therefore, $f_x = N_x(t)/N(t)$, and the frequency of genotype A among these $N_x(t)$ individuals is $p_x = N_x^A(t)/N_x(t)$, leading to $N_x^A(t)/N(t) = p_x f_x$. The genotype frequency of genotype A over all patches in generation t equals

$$p(t) = N^A(t)/N(t) = \sum_x p_x f_x \tag{A1}$$

The genotype frequency of genotype B equals $q(t) = 1 - p(t)$.

Consider a single individual of genotype A leaving a development patch x in generation t. Take a_{kx}^A to be its number of descendents that one generation later leave development patch k. Similarly, a_{kx}^B is the number of descendents per individual of genotype B. The total number of genotype A leaving development patches k in generation $t + 1$ equals

$$N^A(t+1) = \sum_k \sum_x N_x^A(t) a_{kx}^A \tag{A2}$$

The genotypic fitness W^A of genotype A is defined by

$$W^A = N^A(t+1)/N^A(t)$$
$$W^A = \sum_k \sum_x N_x^A(t) a_{kx}^A / N^A(t) \tag{A3}$$

as given in equation (12), with $a_{kx}^A = \sum_y K_{kyx} w_{xy}^A = \sum_y m_{y|x} v_y w_{xy}^A z_y F d_{k|y} u_k$. The genotypic fitness equals the growth rate λ^A of the population of genotype A. Moreover,

$$W^A = \sum_k \sum_x \frac{p_x}{p(t)} f_x a_{kx}^A \tag{A4}$$

In general, genotypic fitness changes with the local frequency of the genotypes. In the present model, the local frequency of the genotypes depends upon the life history. If $d_{k|y} = d_k$ for all y in all generations, the genotype frequency is identical in all development patches. Therefore, if $d_{k|y} = d_k$ (which includes the zygote pool model),

$$W^A = F \sum_k \sum_y \sum_x f_x m_{y|x} v_y w_{xy}^A z_y d_k u_k \tag{A5}$$

Mean genotypic fitness equals $\bar{W} = p(t) W^A + q(t) W^B$.

A vector $\mathbf{g}$ of trait values over environments characterizes each genotype. Mean trait values, one for each environment of development, characterize the population. The vector of changes in mean trait values for each environment becomes

$$\Delta \bar{\mathbf{g}}_\mathbf{x} = \frac{1}{\bar{w}} \mathbf{G}\, \boldsymbol{\beta}_\mathbf{x} \tag{A6}$$

(Lande, 1979; Via and Lande, 1985). The selection gradient vector β_x contains elements $\partial \bar{W}/\partial \bar{g}_x$. Selection stops when the selection gradient vector equals zero (Lande, 1979). Equation (A6) can be used both when selection and development occur in the same patch and when they occur in different patches, with the appropriate selection gradient vector (de Jong, 1999). Equation (A6) does not require the population to grow at a constant rate and can, therefore, be quite generally used. The

selection gradient vector accommodates density-dependent selection. The requirement is, therefore, to find the derivative of fitness.

APPENDIX 2: INVASION FITNESS AND ESS

Suppose that a resident wild type population is in stable demographic equilibrium at a strategy of genotypic values in environments x given by the vector $\mathbf{g}^{\mathbf{r}}$. All density-dependent viabilities are at their equilibrium value, leading to

$$K^*_{kyx} = m_{y|x} v^*_y z^*_y F d_{k|y} u^*_k$$

$$K^{r}_{kyx}{}^* = K^*_{kyx} w^r_{xy} \tag{A7}$$

The resident strategy $\mathbf{g}^{\mathbf{r}}$ has a fitness of 1. Again, fitness and population growth rate are identical, as the population is asexual. The condition for the strategy of genotypic values $\mathbf{g}^{\mathbf{r}}$ to be a globally stable ESS is that for all mutant strategies $\mathbf{g}^{\mathbf{m}}$ their fitness is less than the fitness of 1 of the resident strategy. Therefore, for all $\mathbf{g}^{\mathbf{m}} \neq \mathbf{g}^{\mathbf{r}}$,

$$W(\mathbf{g}^{\mathbf{m}} | \mathbf{g}^{\mathbf{r}}) = \textstyle\sum_k \sum_y \sum_x f^*_x K^*_{kyx} w^M_{xy} < 1 \tag{A8}$$

For the resident strategy $\mathbf{g}^{\mathbf{r}}$ to be an ESS under density-dependent number regulation, fitness should be at a maximum and equal to 1 at strategy $\mathbf{g}^{\mathbf{r}}$. All first partial derivatives of $W(\mathbf{g}^{\mathbf{r}} | \mathbf{g}^{\mathbf{r}})$ towards the component genotypic values g^*_x of $\mathbf{g}^{\mathbf{r}}$ should, therefore, be zero. Given that the first derivatives are zero, the mixed second partial derivatives disappear, as each individual experiences only one environment of development. All direct second partial derivatives should be negative. The conditions for $\mathbf{g}^{\mathbf{r}}$ to be an ESS are, therefore, given by

$$\left. \frac{\partial W(\mathbf{g}^{\mathbf{m}} | \mathbf{g}^{\mathbf{r}})}{\partial g_x} \right|_{\mathbf{m}=\mathbf{r}} = 0 \tag{A9}$$

$$\left. \frac{\partial^2 W(\mathbf{g}^{\mathbf{m}} | \mathbf{g}^{\mathbf{r}})}{\partial g_x^2} \right|_{\mathbf{m}=\mathbf{r}} < 0 \tag{A10}$$

Again, the task becomes to find the derivative of fitness.

APPENDIX 3: THE DERIVATIVE OF FITNESS AND THE POPULATION MATRIX

Genotypic fitness at equilibrium density is given by

$$W^g = \textstyle\sum_k \sum_y \sum_x \dfrac{p^g_x}{p^g(t)} f^*_x K^*_{kyx} w^g_{xy} = \sum_k \sum_x \dfrac{p^g_x}{p^g(t)} f^*_x a^g_{kx} \tag{A4}$$

both in the quantitative genetics and ESS approach. The derivative of fitness of genotype $\mathbf{g}$ with respect to genotypic mean or genotypic value g_x as developed in environment x is, therefore, given by

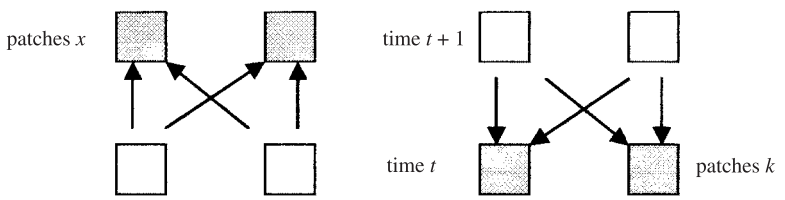
$$\frac{\partial W^g}{\partial g^g_x} = \textstyle\sum_k \sum_x \dfrac{p^g_x}{p(t)} f_x \dfrac{\partial a^g_{kx}}{\partial g^g_x} + \sum_k \sum_x \dfrac{1}{p(t)} f_x a^g_{kx} \dfrac{\partial p^g_x}{\partial g^g_x} \tag{A11}$$

In the zygote pool model, the second term disappears. Equation (A11) cannot readily be used to find an expression for the derivative of fitness. However, the derivative of fitness can be found from matrix methods, as the derivative of the eigenvalue of the matrix describing the equilibrium population.

Define the matrix **P** with k rows and x columns and the elements a_{kx}:

$$a_{kx} = \sum_y K_{kyx} w_{xy} = \sum_y m_{y|x} v_y w_{xy} z_y F d_{k|y} u_k$$

The matrix can be viewed in two directions. For this moment, writing x to indicate patches that are being filled and k to indicate patches from which individuals are spilling:



forward looking

proportion going to x

numbers

backward looking

proportion coming from k

reproductive values

At constant population growth rate λ and stable distribution of individuals over the patches, the proportion of individuals arriving in the patches x are given by the right eigenvector

$$\mathbf{Pn} = \lambda \mathbf{n} \tag{A12}$$

where **n** is the right eigenvector defining the proportion of the population that arrives at the patches x at constant population growth. At stable equilibrium, $\lambda = 1$ and $\mathbf{Pn}^* = \mathbf{n}^*$.

Transposition of the matrix reverses time. The eigenvector given by

$$\mathbf{P}^{\mathbf{T}} \mathbf{r}^{\mathbf{T}} = \lambda \mathbf{r}^{\mathbf{T}} \tag{A13}$$

represents the proportion of the population that contributes from the patches k at constant population growth. Transposing again converts the vector **r** to be the left eigenvector

$$\mathbf{rP} = \lambda \mathbf{r} \tag{A14}$$

At stable equilibrium, $\lambda = 1$ and $\mathbf{r}^*\mathbf{P} = \mathbf{r}^*$. The elements of this left eigenvector are called the 'reproductive values' belonging to the patches. Writing x to indicate patches in generation t and k to indicate patches in generation $t + 1$ as usual, the effective contribution of an individual in patch x to the future population is given by

$$r_x = \sum_k r_k^* a_{kx} \tag{A15}$$

This represents the total reproductive value of a single individual leaving development patch x.

Caswell (1989) gives the derivative of the eigenvalue, the population growth rate λ, with respect to one of the elements of the matrix as

$$\frac{\partial \lambda}{\partial a_{kx}} = \frac{r_k^* n_x^*}{< r^*, n^* >} \tag{A16}$$

Here $< r^*, n^* >$ represents a scalar product: the constant resulting from multiplying the row vector r^* with the column vector n^*. This constant is always positive and plays no role in our results (and is ignored). Genotypic value g_x in a given environment x influences the matrix elements a_{kx}. Therefore,

$$\frac{\partial \lambda}{\partial g_x} = \sum_k \frac{\partial \lambda}{\partial a_{kx}} \cdot \frac{\partial a_{kx}}{\partial g_x} = \sum_k r_k^* n_x^* \cdot \frac{\partial a_{kx}}{\partial g_x} \tag{A17}$$

We need to know the fitness function w_{xy}^g to determine the derivative of the matrix elements.

APPENDIX 4: EQUILIBRIUM GENOTYPIC VALUES

Let us look at two fitness functions for optimizing selection w_{xy}^g. In both fitness functions, the θ_y represent the optimum values in each environment y for the phenotypically plastic trait g_x. Trait value depends upon genotype **g**. The first fitness function is the quadratic fitness function: $w_{xy}^g = 1 - s(\theta_y - g_x^g)^2$, if $| \theta_y - g_x | < 1 | \sqrt{s}$, and $w_{xy}^g = 0$ otherwise. The second fitness function is the corresponding exponential fitness function $w_{xy}^g = \exp(- s(\theta_y - g_x^g)^2)$. In the exponential fitness function, the second derivative of the exponential function is negative if $| \theta_y - g_x | < 1/\sqrt{2s}$; outside this, corresponding to the tails outside one 'standard deviation', the second derivative of the exponential function is positive. With quadratic optimizing selection, and if all fitness values are larger than zero, mean fitness becomes:

$$\bar{W} = \sum_k \sum_y \sum_x f_x K_{kyx} (1 - s(\theta_y - \bar{g}_x)^2 - s \operatorname{var}_G(g_x)) \tag{A18}$$

where $\bar{g}_x$ is the mean genotypic value, and $\operatorname{var}_G(g_x)$ is the genotypic variance, in environment x. The derivative of fitness is found as the derivative of the population growth rate, according to equation (A17):

$$\frac{\partial \bar{W}}{\partial \bar{g}_x} = f_x^* \sum_y \sum_k r_k^* K_{kyx}^* \frac{\partial w_y(\bar{g}_x)}{\partial \bar{g}_x} = 2 s f_x^* \sum_y \sum_k r_k^* K_{kyx}^* (\theta_y - \bar{g}_x) \tag{A19}$$

Define $q_{yx} = (\sum_k r_k^* K_{kyx}^*)/(\sum_y \sum_k r_k^* K_{kyx}^*)$. The selection gradient vector with elements $\partial \bar{W}/\partial \bar{g}_x$ becomes zero if the mean trait value in any environment equals the expectation of the optima, using the q_{yx} as probabilities for a path from x to k:

$$\hat{\bar{g}}_x = \frac{\sum_k \sum_y r_k^* K_{kyx}^* \theta_y}{\sum_k \sum_y r_k^* K_{kyx}^*} = \sum_y q_{yx} \theta_y = \mathrm{E}[\theta_y \mid x] \tag{A20}$$

With exponential optimizing selection, mean fitness can be approximated by

$$\bar{W} = \sum_x \sum_y \sum_k f_x K_{kyx} \exp(- s(\theta_y - \bar{g}_x)^2) \tag{A21}$$

The derivative of fitness is again found as the derivative of the population growth rate, according to equation (A20):

$$\frac{\partial \overline{W}}{\partial \bar{g}_x} = f_x \sum_y \sum_k r_k^* K_{kyx}^* \frac{\partial w_y(\bar{g}_x)}{\partial \bar{g}_x} = 2s f_x \sum_y \sum_k r_k^* K_{kyx}^* w_y(\bar{g}_x)(\theta_y - \bar{g}_x) \tag{A22}$$

Define $q_{yx} = (\sum_k r_k^* K_{kyx}^* w_y(\bar{g}_x))/(\sum_y \sum_k r_k^* K_{kyx}^* w_y(\bar{g}_x))$. Using these q_{yx} as probabilities for a path from x to k yields the compromise genotypic value in environment x under Gaussian selection:

$$\hat{\bar{g}}_x = \frac{\sum_k \sum_y r_k^* K_{kyx}^* w_y(\bar{g}_x)\ \theta_y}{\sum_k \sum_y r_k^* K_{kyx}^* w_y(\bar{g}_x)} = \sum_y q_{yx} \theta_y = \mathrm{E}[\theta_y \mid x] \tag{A23}$$

The conditions for $\mathbf{g}^{\mathbf{r}}$ to be an ESS as given in equation (A9) again involve the first derivative, and lead to expressions identical to equations (A20) and (A23). The pertinent derivatives become:

$$\left. \frac{\partial W(\mathbf{g}^{\mathbf{m}} \mid \mathbf{g}^{\mathbf{r}})}{\partial g_x} \right|_{\mathbf{m}=\mathbf{r}} = f_x^* \sum_y \sum_k r_k^* K_{kyx}^* w_{xy}'^m = 0 \tag{A24}$$

$$\left. \frac{\partial^2 W(\mathbf{g}^{\mathbf{m}} \mid \mathbf{g}^{\mathbf{r}})}{\partial g_x^2} \right|_{\mathbf{m}=\mathbf{r}} = f_x^* \sum_y \sum_k r_k^* K_{kyx}^* w_{xy}''^m < 0 \tag{A25}$$

The ESS genotypic value becomes identical to the one given in equation (A23), if stable.

Neither the equilibrium numbers nor the equilibrium reproductive values can be derived analytically. An example referring to two patches is in order.

Density-dependent number regulation is only present after selection and differs between patches, with patch y_1 the sink patch: $\alpha_0 = \alpha_1 = 0$, $\beta_0 = \beta_1 = 0$, $\gamma_0 = 0.001$, $\gamma_1 = 0.002$. Migration is given by $m_{0|0} = m_{1|1} = 0.7$ and $m_{1|0} = m_{0|1} = 0.3$. Zygote dispersal is given by $d_{0|0} = d_{1|1} = 0.8$ and $d_{1|0} = d_{0|1} = 0.2$. The selection intensity $s = 0.125$ in both patches. Each individual has seven offspring on reproduction. The optimum phenotypic values are $\theta_0 = 0$ and $\theta_1 = 1$

At equilibrium, the total number of individuals equals 2962. The population matrix is given by:

$$\begin{bmatrix} 0.78605 & 0.39697 \\ 0.32036 & 0.40571 \end{bmatrix}$$

The equilibrium vector of relative numbers equals $[0.65\ 0.35]^T$ and the equilibrium vector of reproductive values equals [0.60 0.40]. The equilibrium genotypic values are given by $[0.12\ 0.45]^T$. The prediction from equations (19) and (A23) corresponds with the observed values from the iteration.

Two possibilities for the zygote pool model exist. First, if zygote dispersal is given by $d_0 = 0.8$ and $d_1 = 0.2$, the total number of individuals at equilibrium will be 2956. The population matrix is given by:

$$\begin{bmatrix} 0.82043 & 0.71846 \\ 0.20511 & 0.17961 \end{bmatrix}$$

The equilibrium vector of relative numbers arriving in the patches equals $[0.80\ 0.20]^T$ and the equilibrium vector of reproductive values equals [0.533 0.466]. The equilibrium genotypic values are given by $[0.22\ 0.64]^T$. Second, if zygote dispersal is given by $d_0 = 0.2$ and $d_1 = 0.8$, the total number of individuals at equilibrium will be 2963. The population matrix is given by:

$$\begin{bmatrix} 0.33170 & 0.16708 \\ 1.32681 & 0.66834 \end{bmatrix}$$

The equilibrium vector of relative numbers arriving in the patches equals $[0.20\ 0.80]^T$ and the equilibrium vector of reproductive values equals [0.665 0.335]. The equilibrium genotypic values are given by $[0.038\ 0.185]^T$. The reproductive values do not influence the equilibrium genotypic values in these two cases. Genotypic values predicted from equations (8) and (19) correspond to the observed genotypic values in the iterations.

APPENDIX 5: STABILITY

The selection gradient vector β_x might have all its elements $\partial \bar{W}/\partial \bar{g}_x = 0$ but not indicate a stable selective situation. The second derivatives $\partial^2 \bar{W}/\partial \bar{g}_x^2$ have to be negative to indicate a fitness maximum and stability of the compromise genotypic value. With the quadratic fitness function, these second derivatives are always negative if fitness is greater than zero. However, if fitness values of zero appear, due to deviations in trait values, a fitness maximum is not necessarily indicated. In fact, the first derivative might well indicate a fitness minimum. If so, the solution given in equation (A19) indicates an unstable equilibrium. The stable solution is given by $g_x = \theta_y$, most often with $x = y$. Clearly, this involves strong selection, as at least fitnesses of zero have to be involved.

In the Gaussian fitness function, an unstable equilibrium appears under less stringent fitness conditions. The second derivative for the Gaussian fitness function becomes

$$\frac{\partial^2 \bar{W}}{\partial \bar{g}_x^2} = 2s \cdot f_x \sum_y \sum_k r_k^* K_{kyx}^* w_y(\bar{g}_x) \cdot (2s(\theta_y - \hat{\bar{g}}_x)^2 - 1) \tag{A26}$$

The condition $\partial^2 \bar{W}/\partial \bar{g}_x^2 < 0$ leads to

$$\frac{\sum_y \sum_k r_k^* K_{kyx}^* w_y(\bar{g}_x)(\theta_y - \hat{\bar{g}}_x)^2}{\sum_y \sum_k r_k^* K_{kyx}^* w_y(\bar{g}_x)} < \frac{1}{2s} \tag{A27}$$

The selection strength s is related to the width σ_w^2 of the Gaussian fitness function by $1/2s = \sigma_w^2$. Equation (A26), therefore, indicates that, for a Gaussian fitness function, the stability criterion simplifies to the condition that the conditional variance in the optimum values θ_y, as seen from a particular patch x, is less than the width of the selection function. This condition relates directly to the second derivative of the selection function itself, as this second derivative is negative if $|\ \theta_y - g_x\ | < 1/\sqrt{2s}$. The condition is not general, but specific for Gaussian optimizing selection. The condition allows for, but does not necessarily lead to, polymorphism. In polymorphism, more than one phenotypic value g_x is found for a given patch x, corresponding to different θ_y.

The signs of the second derivatives that are of importance for stability are therefore related to the signs of the second derivatives of the fitness functions w_{xy}^g. Basically, the question is whether mean fitness with $g_x = \theta_y$ (potentially even different θ_y for genotypic values in one patch x) is less than mean fitness with $g_x = \hat{\bar{g}}_x$; if so, the compromise solution $g_x = \hat{\bar{g}}_x$ is stable. Therefore, we have to determine whether the expected value $\mathrm{E}[W(g_x = \theta_y)]$ is less than the expected value $\mathrm{E}[W(g_x = \hat{\bar{g}}_x)]$. We can easily see from the fitness functions that $\mathrm{E}[W(g_x = \hat{\bar{g}}_x)]$ is larger than $\mathrm{E}[W(g_x = \theta_y)]$ if the averaging is over that region of the fitness function where its second derivative is negative. On the other hand, $\mathrm{E}[W(g_x = \hat{\bar{g}}_x)]$ is less than $\mathrm{E}[W(g_x = \theta_y)]$ if the averaging is over that region of the fitness function where its second derivative is positive (for the Gaussian fitness function), or the fitness function equals zero (for the quadratic fitness function). This is an application of Jensen's inequality, which states that the expectation of the function values over a range of arguments is in general not equal to the function value at the expectation of the arguments: $\mathrm{E}[\hat{\mathrm{f}}(x)] \neq \hat{\mathrm{f}}(\mathrm{E}[x])$. Averaging over function values of nonlinear functions cannot be replaced by the function value of the average.