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Presence of specialist and generalist genotypes in a heterogeneous environment

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Abstract

Evolution of a phenotypically plastic trait is examined under weak optimizing selection in a heterogeneous environment. Selection operates towards optimum phenotype that depends upon the environment. Density dependent number regulation is caused by non-uniform food supply. The juveniles do not disperse but develop in the parental patch. An adult has a certain probability to migrate to a different patch of selection. Density dependent number regulation operates after selection. There is one gaussian food distribution over the patches. The model is individual based. A mutation model has been developed to find the ESS genotypic values. In the absence of phenotypic plasticity, multiple populations are observed at evolutionary equilibrium with clear niche separation. The genotypes are specialists. The splitting of the population occurs beyond a critical value of food width (defined as the food distance between patches where food supply is half of the maximum value) at fixed migration parameter. This critical value is reduced as peakedness of the migration distribution increases. The genotypes develop generalist tendency with the increase of the level of plasticity. Eventually genetic differentiation is removed and one population is found at equilibrium beyond a critical value of plasticity. The genotypes become generalists. Phenotypic plasticity increases niche width and tempers niche separation.

Key words: genotype, phenotype, evolution, plasticity, mutation model, ESS

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Introduction:

Many organisms live in heterogeneous environment. They display phenotypic plasticity in order to cope with this heterogeneity. It is believed that natural selection molds the level of phenotypic plasticity in a given trait. It is an important problem to study the evolution of phenotypic plasticity in a heterogeneous environment where unequal food supplies in different environments cause density dependent number regulation. The interrelationship between migration and population persistence were studied in demographic models (Okubo 1980). They came to the conclusion that a population can't survive in a suitable patch if individuals disperse too quickly into neighboring habitats that are unsuitable. Mayr (1963) has concluded that adaptation of a population at the periphery might be limited by gene flow from central populations. The interactions between gene flow and local selection pressure can limit the geographical range of a species (Kirkpatrick and Barton 1997). This model keeps track of the evolutionary and demographic changes in a continuously distributed population. A quantitative trait evolves under the influence of stabilizing selection and gene flow. One of the following consequences is possible. The species can go extinct, fill all of the available habitat or be restricted to a limited range where it is well adapted to allow population growth. The peripheral half of a species range can act as a demographic sink due to the mal-adaptation triggered by gene flow. The trait's genetic variance has small effect on species persistence when gene flow is very strong to keep population density far below the carrying capacity. In appropriate circumstances, a balance is struck between gene flow and local adaptation that constrains the species to a limited range. The important factor in the model is the inclusion of density-dependent population regulation so that limited-range equilibrium is possible. The basic tendency for gene flow from better-adapted regions to delimit local adaptation elsewhere may be compensated by phenotypic plasticity and by increase in genetic variation caused by the admixture (Grant and Grant 1994). The peripheral populations act as demographic sinks due to maladaptation caused by the gene flow. These regions are maintained by migration from better adapted populations. Gene flow acts as a mechanism that limits the geographical range of a species.

Christiansen and Loeschcke (1980) have studied the evolution of a species with respect to its environment by considering the evolution of characters that determine its niche. They have used a model of exploitative competition with a gaussian resource spectrum. Evolution is considered at a gene locus that influences the niche position. The allelic contributions to the genotypic values are additive. Intra-specific competition does not play an important role when the resource abundance spectrum imposes strong directional selection. So the genotypes that utilize the more abundant resources will have a higher selective value. But intra-specific competition becomes important when evolution due to this directional selection approaches the goal (i.e. the niche is at the resource optimum). Evolution makes the mode of the niche converge to the resource optimum when allele contributions are small in comparison to the distance between the mode of the niche and the resource optimum. The globally stable equilibrium will maintain at most two alleles in the population when the allele contributions are of the same order of magnitude as the above distance.

Intraspecific exploitative competition can be a dominant force for the maintenance of two-allele polymorphism.

The limitations on the evolution of adaptive plasticity and niche width have been examined (Whitlock, 1996). It is shown that species with narrower niche width fix beneficial alleles with high probability in less number of generations, develop lighter drift load and have a smaller number of deleterious alleles at mutation-selection equilibrium. The rate of evolutionary response is slow for species with larger niche width. The evolution of niche width and plasticity does not depend only on fitness in different environments, it is also related to the evolutionary process per se. The effective population size increases in proportion to niche width due to the availability of more resources to a species.

Scheiner (1998) has found evolved differences between genotypes in local habitats due to limited adult migration. Here unpredictability of selection causes the population structure to influence the evolution of reaction norms. In all locally differeentiated reaction norms, phenotypic plasticity was lower than the phenotypic plasticity selected for. Curvature appeared in evolved reaction norms even if the linear reaction norm was selected for. In addition to unpredictability of selection, edge effect due to finite size of the multipatch system also plays a role in creating local genotypic differentiation. The optimum reaction norm evolves when selection is totally predictable in a heterogeneous environment (Via and Lande 1985). As selection becomes unpredictable the slope of the evolved reaction norm becomes shallower than the slope of the optimum reaction norm (Gavrilets and Scheiner 1993; Sasaki and de Jong 1999). Sasaki and de Jong (1999) have simulated a two-patch model with the result that unpredictability of selection leads to evolutionarily stable bet-hedging reaction norm constituting a compromise between the phenotypic optima in the different patches. However, the polymorphic response in the evolutionarily stable population occurs in the presence of very strong selection. This model is basically a zygote pool model. De Jong and Behera (2002) have extended the above analysis by considering a zygote dispersal stage where there exists a correlation between the patch of selection and the patch of development. 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. In this model, the influence of density-dependent number regulation strongly depends on the exact life cycle.

We ask the following questions. First we ask how niche width evolves without phenotypic plasticity. We shall try to understand how the migration pattern and food width influence the niche width at evolutionary equilibrium. We shall see whether evolution of niche width is influenced by the level of phenotypic plasticity. We aim to study when phenotypic plasticity prevails over genetic variation. We also plan to explore how population number at equilibrium depends on the level of plasticity and migration pattern.

MODEL

Life cycle

We consider the evolution of the reaction norm of a quantitative trait and the consequent evolution of niche width of a haploid asexual population. The environment is heterogeneous. The phenotype of an individual is determined by its genotype and an environmental variable which depends on the environment of development. The population life cycle is described in Fig. 1. Zygotes arrive in a patch in the development environment. After the phenotype is determined in the patch of development, surviving adult individuals migrate to another patch. In the patch of selection, adults are subject to viability selection and then density-dependent number regulation. The density-dependent number regulation is caused by non-uniform food supply which is taken to be gaussian. Viability selection is optimizing. The optimum phenotype varies from patch to patch depending on the environmental variable. Surviving individuals reproduce and die. The offsprings disperse to the patch of development.

Model specification

There are n different values of an environmental variable in the environment of development and the environment of selection. A juvenile develops in patch x and obtains the genotypic value $g(x)$ according to the wild-type reaction norm g. In generation t, the $N_x(t)$ juveniles arrive 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

$$N_{1,y}(t) = \sum_{x=1}^{n} N_x . m_{y|x} \quad . \qquad (1)$$

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

$$w_{xy}^{g} \equiv W_y[g(x)] = \exp\{-s(\theta(y) - g(x))^2\} \qquad (2)$$

where $\theta(y)$ 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

$$N_{2,y}(t) = N_{1,y}\, w_{xy}^{g} \quad . \qquad (3)$$

Density-dependent number regulation after selection leads to the probability to survive of

$$z_y \equiv Z_y[N_{2,y}(t)] = \exp\{-\gamma_y N_{2,y}\} \quad (4)$$

where γ_y is the sensitivity in patch *y* to density regulation after selection. The number of individuals in patch y after selection equals

$$N_{3,y} = N_{2,y}\ z_y \quad . \quad (5)$$

Let $d_{x|y}$ be the dispersal probability of a zygote from selection patch y to development patch x. Each surviving individual reproduces with fecundity *F*. The number of zygotes arriving in development patch x in generation t+1 equals

$$N_x(t+1) = \sum_{y=1}^{n} N_{3,y}\ d_{x|y}\ F \quad . \quad (6)$$

Fitness

At equilibrium number, the expectation of fitness W_g for a particular genotype *g* can be found from the recurrence equations (Sasaki and de Jong 1999) as

$$\mathrm{E}[W_g] \ = \ \sum_x \sum_y \ m_{y|x}\ w_{xy}^{g}\ z_y^{*}\ d_{x|y}\ F \ , \quad (7)$$

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

$$g(x)^{*} \ = \ \sum_y \ m_{y|x}\ w_{xy}^{g*}\ z_y^{*}\ d_{x|y}\ \theta(y) \Big/ \sum_y m_{y|x}\ w_{xy}^{g*}\ z_y^{*}\ d_{x|y} \quad (8)$$

The evolved compromise reaction norm is weighted towards the optimum of the selection environment with least stringent density regulation, highest migration and dispersal.

The total number leaving any development patch *x* is $N_x(t)$ in generation *t*; in the next generation the total number leaving any development patch *k* and descendant of an individual leaving patch *x* in generation t equals:

$$N_x(t+1) \ = \ N_x(t) \sum_y \left(m_{y|x}\ w_{x,y}^{g}\ z_y\ F\right) d_{k|y} \quad . \quad (10)$$

At stable equilibrium, the total number becomes

$$N^* = \sum_x N_x^* \cdot \sum_k \sum_y \left(m_{y|x} \; w^g_{x,y} \; z_y^* \; F \; d_{k|y} \right). \qquad (11)$$

The condition giving the ESS reaction norm is that no mutant can invade: the growth rate of any mutant is less than 1. That is, that for

$$W_g = \sum_x \sum_y \sum_k \left(N_x^* m_{y|x} w_{xy}^{g^*} z_y^* F \; d_{k|y} \right) \Big/ \sum_x N_x^* \qquad (12)$$

fitness is at a maximum at $g(x)^*$. The implicit solution for the evolved reaction norm is given by

$$g(x)^* = \sum_y \sum_k m_{y|x} \; w_{xy}^{g^*} \; z_y^* \; d_{k|y} \; \theta(y) \Big/ \sum_y \sum_k m_{y|x} w_{xy}^{g^*} z_y^* d_{k|y} \qquad (13)$$

With weak Gaussian selection, fitness has a maximum at $g(x)^*$; $g(x)^*$ is an ESS under weak selection.

Density-dependent number regulation

A ‘source’ environment is a patch where food supply is high so that an individual survives with high probability. Conversely, food supply is low in a ‘sink’ environment so that density regulation is large leading to lower survival probability of a population. The presence of ' source' and 'sink' environments highly influences the evolved reaction norm. In order to appreciate the effect of source and sink environments, it is useful to define the “flow” of individuals through the patches of environments of development and of selection. The flow summarizes the relative frequency of organisms meeting each sequence of patches *y* and x, according to

$$f(y \mid x) = m_{y|x} \; w_{xy}^{g^*} \; z_y^* \; d_{x|y} \; / \left(\sum_y m_{y|x} \; w_{xy}^{g^*} \; z_y^* \; d_{x|y} \right). \qquad (14)$$

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.

Given the definition of flow

$$g(x)^* = \sum_y f(y \mid x) \; \theta_y = \mathrm{E}\left[\theta_y \mid x\right] \qquad (15)$$

in all models with equal selection intensity *s* in all environments *y*, the difference is in the definition of the flow.

Equal viability due to density-dependent number regulation extinguishes the effect of density-dependence on selection. If $z_y^* = z^*$ in all the patches of the environment of selection, the flow becomes, for gaussian selection:

$$f(y \mid x) = \; m_{y|x}\, w_{xy}^{g^*}\, d_{x|y} \; / \left(\sum_y m_{y|x}\, w_{xy}^{g^*}\, d_{x|y} \right) . \qquad (16)$$

The evolved phenotype $g(x)$ is the same as for a pure genotypic frequency model, given we are using Gaussian optimizing selection.

Evolved reaction norm

We define the linear reaction norm as $g(x) = g_0 + g_1 x$ where g_0 is the reaction norm height, g_1 is the reaction norm slope and x refers to the environment of development. The optimal reaction norm is defined as $c(y) = c_0 + c_1 y$ where c_0 is the optimal reaction norm height, c_1 is the reaction norm slope and y denotes the environment of selection. Let r(x,y) denote the migration correlation between the patch of development x and the patch of selection y. If selection is predictable, r(x,y) = 1 and the evolved reaction norm is given by $g_0 = c_0$ and $g_1 = c_1$. But, as the selection becomes unpredictable, $0 \le r(x,y) \le 1$

and the evolved reaction norm might differ from the optimal reaction norm. Weak selection leads to the bet-hedging of the reaction norm while strong selection gives polymorphism. When the heterogeneous environment is very wide with many patches and selection is weak, the evolved reaction norm is given by the following expressions.

$$g_0 = c_0 \qquad (17a)$$

and

$$g_1 = c_1 \, (\mathrm{Cov}\,(x,y)/\,\mathrm{Var}\,(x)) \, . \qquad (17b)$$

But now the density dependence influences the distribution of individuals over the patches. The optimal reaction norm is always obtained when the food supply is uniform

over the patches and migration is symmetric. The deviation from the optimal reaction norm occurs due to the asymmetry in the effective migration distribution. This is caused by two factors. One is the mirroring of migration due to the edges of the environment. This happens when the number of patches are finite. It can be better illustrated by a simple example. Suppose migration width is three and there is equal a priori probability of migration. If the straight migration occurs at the right boundary, there is no possibility of migration to one patch right of the boundary. This migration probability will be mirrored to the left patch of the boundary so that the probability of migration to this patch will be 0.666. This can cause asymmetry in the migration distribution. The other factor is the density dependent number regulation due to non-uniform food supply. The

non-uniform food supply will cause differential density regulation over the patches. This can create asymmetry in the migration distribution.

The predicted ESS g_0 and g_1 values depend on the number of individuals at evolutionary equlibrium. But this number can't be found analytically.

Furthermore, the small deviations of the evolved g_0 and g_1 values from the optimal c_0 and c_1 values are difficult to find as selection is weak and the model is individual-based.

So it seems best to develop a mutation model where deviations in g_0 and g_1 are introduced with low probability.

Simulation

We have performed numerical iterations in a fifteen-patch version of the model in order to bring out clearly the influence of density-dependent number regulation caused by non-uniform food supply. The one food distribution is considered as gaussian with a peak occurring in the middle patch. The food width is defined as the food distance between patches where food supply is half of the maximum value. The amount of food remains the same, thus food width only changes food distribution over the patches. The migration distribution is characterized by the width and peakedness. When the migration probabilities to one of the neighbouring patches as determined by the migration width are equal, the peakedness of migration becomes one. In general, the migration probability to successive neighbouring patches decreases geometrically by a factor whose value is given by the peakedness. When the peakedness of adult migration is high the probability that the individual will migrate to one of the neighbouring patches (determined by the migration width) is small. For example, when peakedness = 1 and migration width = 7, the individual has equal a priori probability (equal to 0.142) to stay in the parental patch or migrate to one of the three neighbouring patches in the left and three in the right. But, as the peakedness becomes five, the probability of the individual to stay in the parental patch is equal to 0.668 while migration probability to one of the three left neighbouring patches decreases geometrically by a factor of five. The same situation holds for migration to the right neighbouring patches. The juveniles do not disperse but stay in the same environment. After reproduction, a mutational deviation is introduced to the genotypic values of the offsprings. More genetic variation is created by mutation.

The program is individual-based and object-oriented. The individuals are considered as objects with associated properties. In every stage of the life cycle, each individual is tracked whether dead or alive. The optimum phenotypes of patch 0 is zero, patch 1 is one and so on. Initially the g_0 values of the individuals are chosen randomly within the interval of –1 and 15. Let $g_0[j]$ and $g_1[j]$ are the genotypes of the jth individual, and x be a uniformly distributed pseudo-random number in the interval of 0 and 1. The following expressions are used to find the initial g_0 and g_1 values of an individual.

$$g_0[j] = (p+1)x - 1 \qquad (15a)$$

and

$$g_1[j] = \{1/(p-1)\}\{(p+1)x - 1\} - \{1/(p-1)\}\, g_0[j] \qquad (15b)$$

where p denotes the total number of patches in the model. One can see that the values of $g_1[j]$ lie approximately within –1 and 1. The starting population is 150. The selection intensity is taken as 0.07. The reproduction occurs asexually with the fecundity equal to two. The mutation probability for an individual's genotype is 0.001. The simulation is iterated for 10000 generations. Then the ESS genotypic values are obtained and the distribution of phenotypes over the patches is found.

Results

The most important conclusion is that genotypes at evolutionary equilibrium become specialists with clear genotypic differentiation and niche separation in the absence of phenotypic plasticity. But they gradually develop generalistic tendency as the level of plasticity is increased and eventually become fully generalists at unrestricted plasticity.

Population differentiation without plasticity

There is clear niche separation without phenotypic plasticity. There is a threshold value of food width beyond which many populations are observed at evolutionary equilibrium (for fixed migration width). This threshold value increases for low peakedness (Fig. 2). The threshold value rises with high migration width. Niche separation is always possible if migration width is greater than food width. For fairly restricted food width, niche separation occurs below a critical value of migration width. For instance, when food width = 1.9 and peakedness = 1, the critical migration width = 9 (Fig. 2). Fig. 3A shows that a monomorphic population splits into five populations at evolutionary equilibrium with distinct niches. The niche width becomes narrower. When a large number of samples are analysed, five and six populations are observed at equilibrium with probabilities of 0.6 and 0.4 respectively. The clear gap in the phenotypic value between two neighbouring populations implies niche separation. The phenotypic gaps are equally placed. The genotypic values at equilibrium is given in Fig. 3B. When the peakedness is increased to five in the model corresponding to Fig. 3A, seven distinct populations are obtained at evolutionary equilibrium (data not shown). Without plasticity, low migration width and high peakedness give many populations while the reverse situation generates two populations (data not shown). When the food width is larger, the differential density regulation among patches is relatively smaller. This causes higher survival probability in the parental patch. Thus there is less necessity for an individual to migrate to a neighbouring patch in order to survive. Hence we find more specialist genotypes and many populations as the food width increases. The higher migration width increases the propensity of an individual to migrate to a neighbouring patch. So we need a greater food width with less

differential density regulation so that individuals will be restricted in the same patches producing more specialist genotypes. Similarly, when peakedness is lower,

an individual can migrate to other patches with greater probability. So the food width has to be higher in order to find many populations at equilibrium. The above argument also works for the fact that the splitting of the population at equilibrium occurs for a higher value of food width when the level of plasticity increases.

Allowing increasing level of plasticity

As the degree of plasticity is increased the number of distinct populations at equilibrium decreases leading to larger niche width. The number of distinct populations at evolutionary equilibrium are reduced to four with lower value of the degree of plasticity (Fig. 4A) and to two with high limited plasticity (Fig. 5A). The corresponding genotypic values are given in Fig. 4B and 5B. When g_1 varies from –0.5 to 0.5, the statistical probabilities of finding 1, 2, 3 or 4 populations at equilibrium are 0.12, 0.68, 0.13 and 0.07 respectively. When the degree of plasticity increases, the splitting of the population at equilibrium occurs for a higher value of the food width (data not shown). Some limited plasticity is required to remove genetic differentiation and get one population at equilibrium. Genotypes develop generalist tendency.

Phenotypic plasticity reduces genetic variation

With unrestricted plasticity only one population is found at equilibrium (Fig. 6). The niche width is the largest. The statistical probability of finding one population is always one when a large number of simulations are investigated. It should be mentioned that one population is already observed with very high value of the degree of plasticity (i.e. when g_1 varies from –0.9 to 0.9) (data not shown). One can never get niche separation with unrestricted plasticity even if the food width is very high (data not shown). Genotypes are always generalists.

Population number at evolutionary equilibrium

The population number at evolutionary equilibrium varies in the range of 1000 and 1500. This population number increases with the level of plasticity and migration peakedness but decreases with migration width (data not shown). With higher degree of plasticity, the individuals can adapt better in a variable environment. So the death of an individual will be less likely. The population number at equilibrium will increase. When the peakedness is greater, an individual is confined to the same patch with a higher probability and less likely to go extinct due to a hostile environment. The same argument applies to the case when the migration width is lower. So more number of population will survive at evolutionary equilibrium.

DISCUSSION

The present model attempts to investigate the relationships among gene flow, phenotypic plasticity, niche width and adaptation. Unrestricted phenotypic plasticity removes niche separation and genetic variation. The unequal food supply in different patches gives rise to differential density regulation in the patches. The adult migration makes the genotypes concentrated near the patches where there is local

abundance of food supply. Hence the genotypic differentiation arises at evolutionary equilibrium. The genotypic values in the groups of populations are separated by gaps. The local adaptability causes the genotypes to be specialists. They can adapt well only in the local environment.

Without plasticity, the niche widths at evolutionary equilibrium become narrower with the persistence of specialist genotypes. The genotypes specialize to local patches due to differential density regulation and adult migration. Related type of niche limitation occurs due to gene flow and local adaptation which constrain the species to a limited range (Kirkpatrick and Barton 1997). The larger the number of specialist genotypes, the shorter the niche widths. This is advantageous to the species. Because species with narrower niche widths can have a higher rate of evolutionary response, can fix beneficial alleles with high probability, can have lower frequency of deleterious alleles at mutation-selection equilibrium and can persist longer as species in the sense that they can increase mean fitness faster (Whitlock 1996). The gaussian food distribution plays an important role in the evolution of specialist genotypes which is a form of polymorphism. Similar type of two-allele polymorphism is observed in the presence of gaussian food supply and intraspecific competition (Christiansen and Loeschcke 1980).

Without phenotypic plasticity, low migration width and high peakedness give many specialist populations at equilibrium. This situation reduces the probability of migration of an individual to neighbouring patches. So the individuals are more likely to be confined in the local patches, thus making them more specialists. When the food width is larger, the differential density regulation among patches is relatively smaller. This causes higher survival probability in the parental patch. Thus there is less necessity for an individual to migrate to a neighbouring patch in order to survive. Hence we find more specialist genotypes and many populations as the food width increases.

When phenotypic plasticity is present, the genotypes can adapt in a wider range of environments without undergoing extinction. So the genotypes develop generalistic tendency as the degree of phenotypic plasticity is increased. Finally there remains only generalist genotypes of a single population beyond a critical value of the level of plasticity. Here phenotypic plasticity prevails over genetic variation. With unlimited plasticity, evolved generalist genotype gives rise to broader niche width for the species. Species with larger niche width have slower rate of evolutionary response (Whitlock 1996). The splitting of the population at equilibrium occurs for a higher value of food width when the level of plasticity increases but remains below the critical value. The population never splits when plasticity is unlimited even for very large value of the food width. The population number at evolutionary equilibrium increases with the level of plasticity as plastic individuals adapt better in a variable environment. Plasticity leads to the increase of niche width. As niche width increases, the effective population size will increase proportionately (Whitlock 1996).

When juveniles disperse with certain frequency, the specialist tendency of the genotypes is reduced. So there is less probability of niche separation. Here we have seen that phenotypic plasticity increases niche width and tempers niche separation. It is obviously an interesting problem to investigate the situation where phenotypic plasticity allows niche separation. This can happen in a heterogeneous

environment when two Gaussian food distributions are clearly separated (de Jong and Behera, manuscript). Another stimulating question is to investigate if niche separation and polymorphism can occur in a sexual population. This happens in a diploid sexual population with additive allelic effect in the presence of two separate Gaussian food distributions in a heterogeneous environment (Behera and de Jong, manuscript).

The importance of developmental selection with sexual display in the evolution of traits was highlighted (Higginson and Reader, 2009). Appearance of genotype environment interaction can be dependent on properties of the given measurement scale (Van den Berg and Schwabe, 2014). It was demonstrated that genotype environment interaction can be inferred from genetic variants associated with phenotypic variability without any reference to environmental factors.

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Figure legends

1. Population life cycle.
2. The critical value of food width beyond which many populations (specialist genotypes) are observed at evolutionary equilibrium. No plasticity, peakedness = 1 (solid line), peakedness = 5 (dashed line). The results are averaged over six simulations.

3-6: The phenotypes and genotypes of the population at evolutionary equilibrium. Model parameters: migration width = 3, migration peakedness = 1, food width = 4 and juveniles stay at home environment. Single simulation

3: no plasticity, 5 populations A. Phenotypes B. Genotypes

4: Limited plasticity (is g_1 –0.3 to 0.3), 4 populations A. Phenotypes B. Genotypes

5: Limited plasticity (g_1 is –0.8 to 0.8), 2 populations A. Phenotypes B. Genotypes

6: Unrestricted plasticity (g_1 is –1.0 to 1.0), 1 population, Phenotype. The genotype is $g_0 = 0$ and $g_1 = 0.98$.

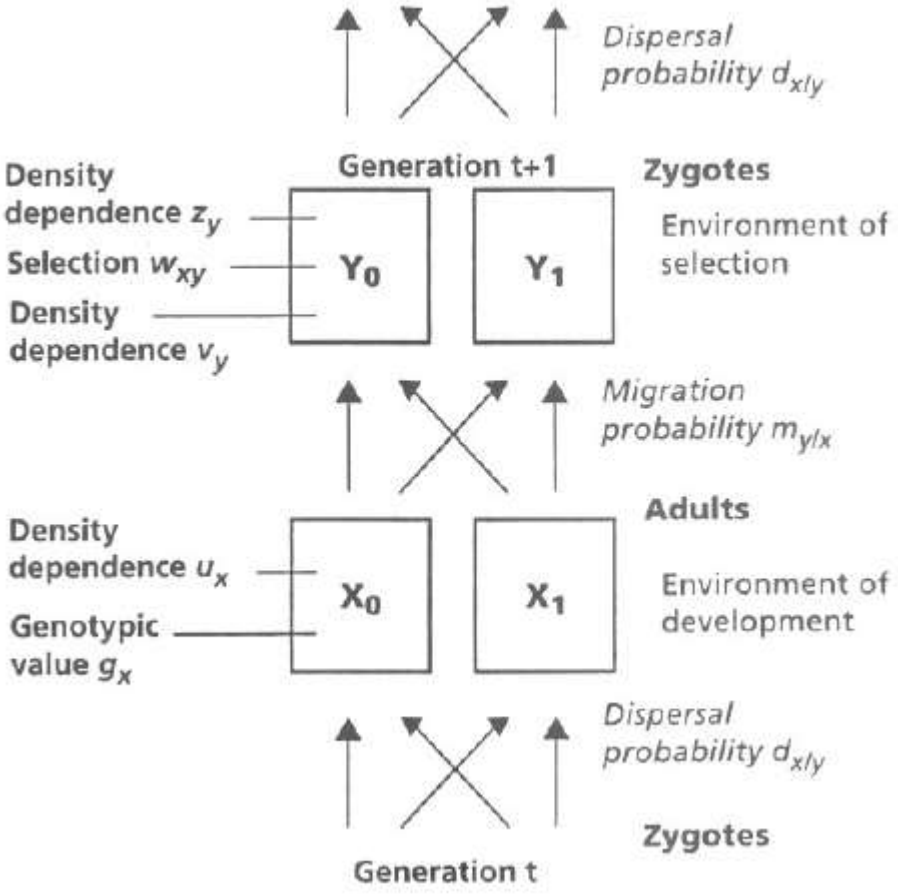

Dispersal probability $d_{x/y}$
Generation t+1
Zygotes
Density dependence z_y
Selection w_{xy}
Density dependence v_y
Y_0
Y_1
Environment of selection
Migration probability $m_{y/x}$
Adults
Density dependence u_x
Genotypic value g_x
X_0
X_1
Environment of development
Dispersal probability $d_{x/y}$
Zygotes
Generation t

Fig: 1

Fig. 2

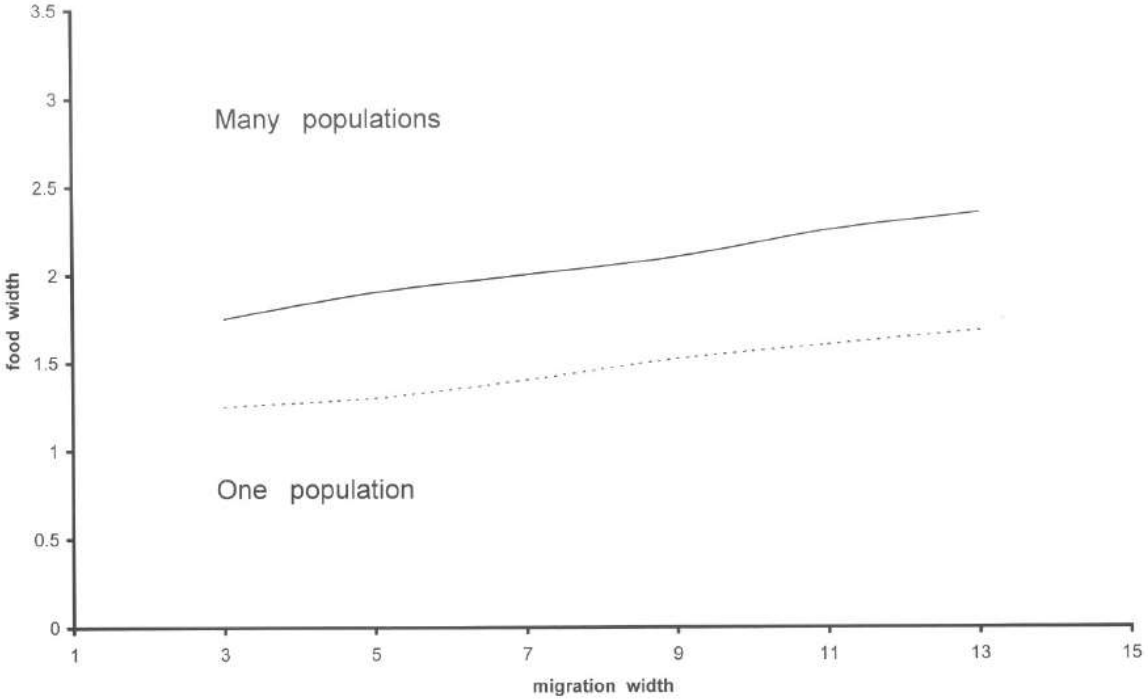

3.5
3
2.5
2
1.5
1
0.5
0
food width
Many populations
One population
1
3
5
7
9
11
13
15
migration width

Fig. 3A

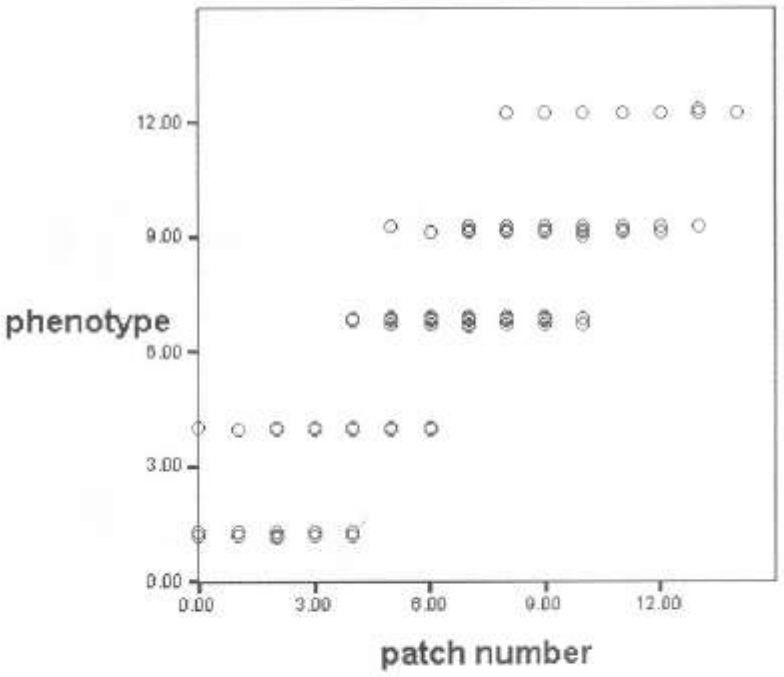

12.00
9.00
6.00
3.00
0.00
phenotype
0.00
3.00
6.00
9.00
12.00
patch number

Fig. 3B

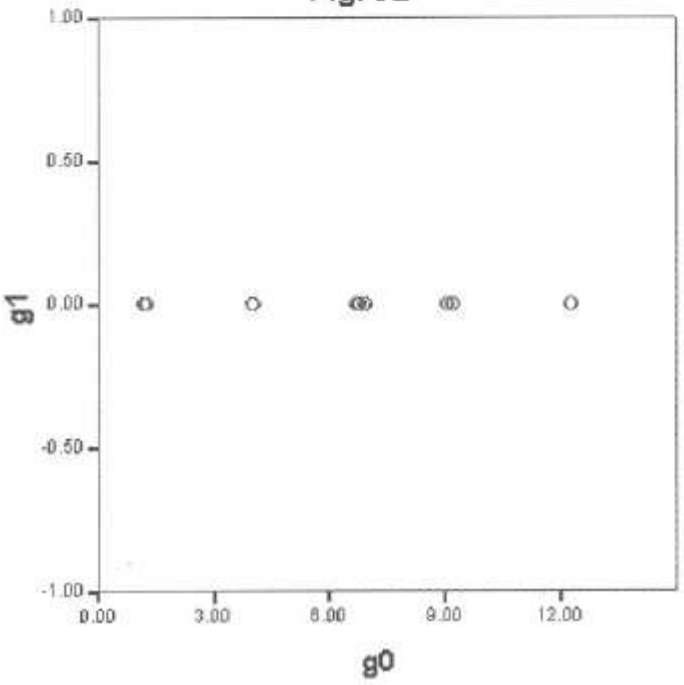

1.00
0.50
0.00
-0.50
-1.00
g1
0.00
3.00
6.00
9.00
12.00
g0

Fig. 4A

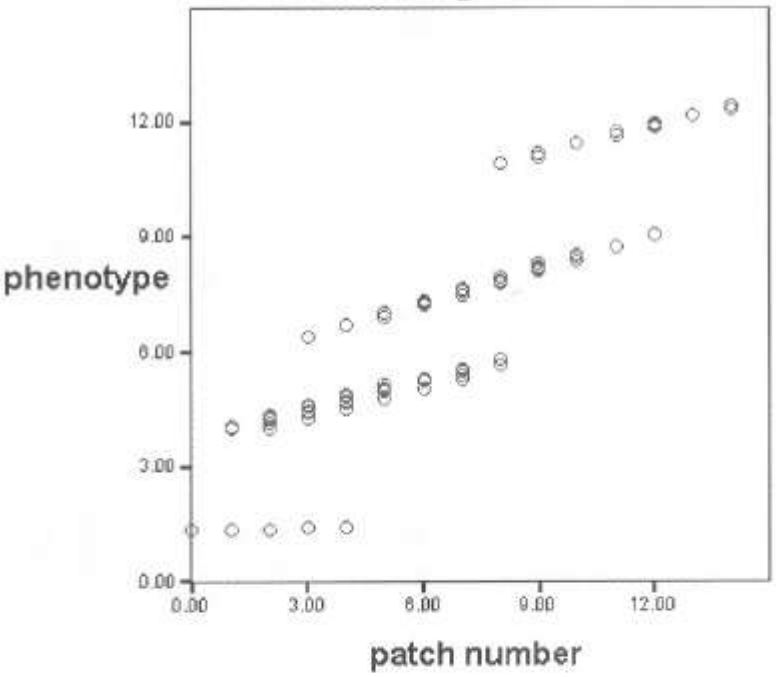
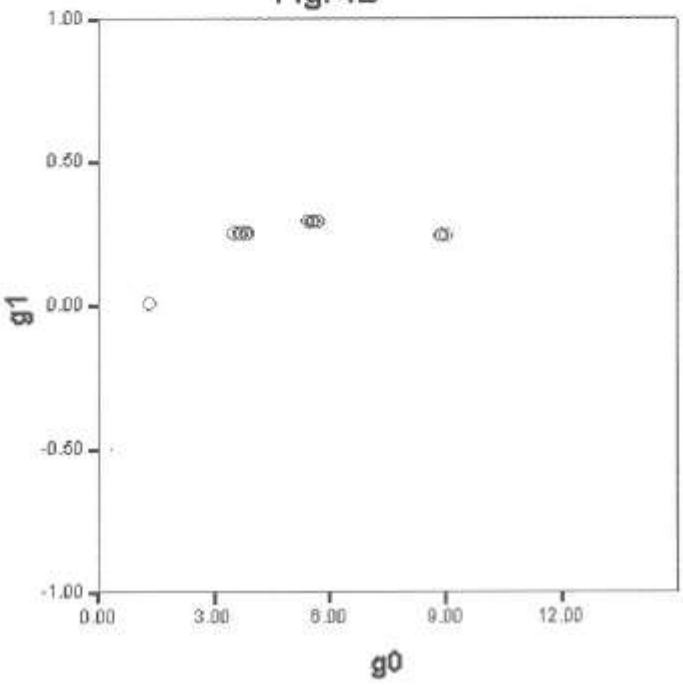
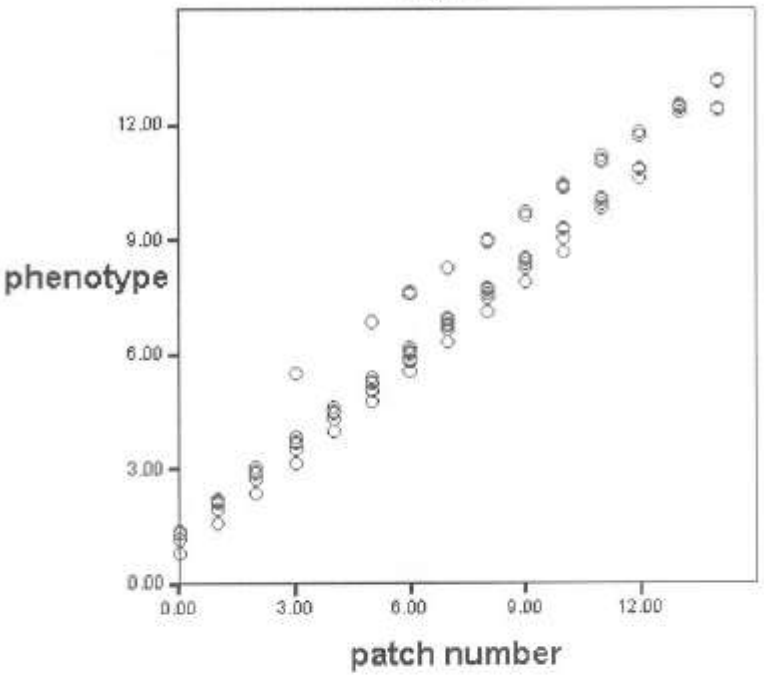


Fig. 4B



12

Fig. 5A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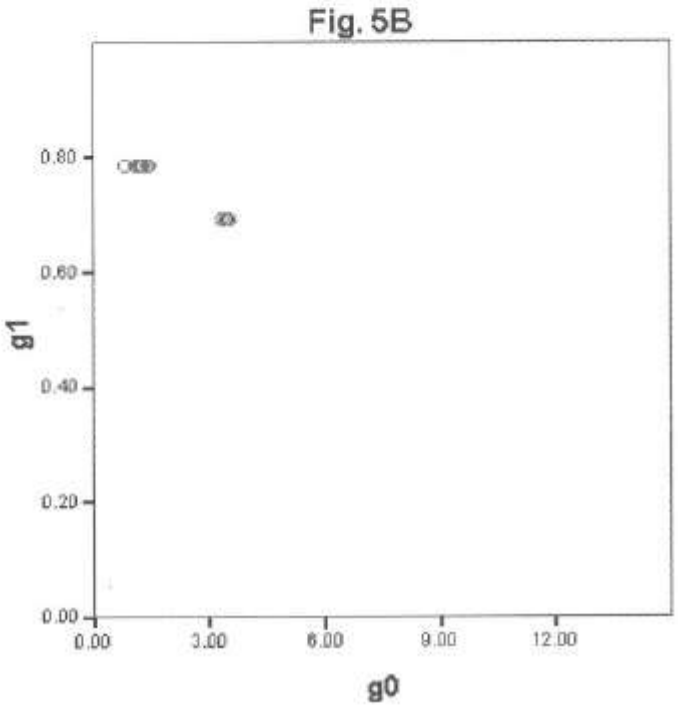
phenotype
0.00
3.00
6.00
9.00
12.00
patch number

Fig. 5B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0.20
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g1
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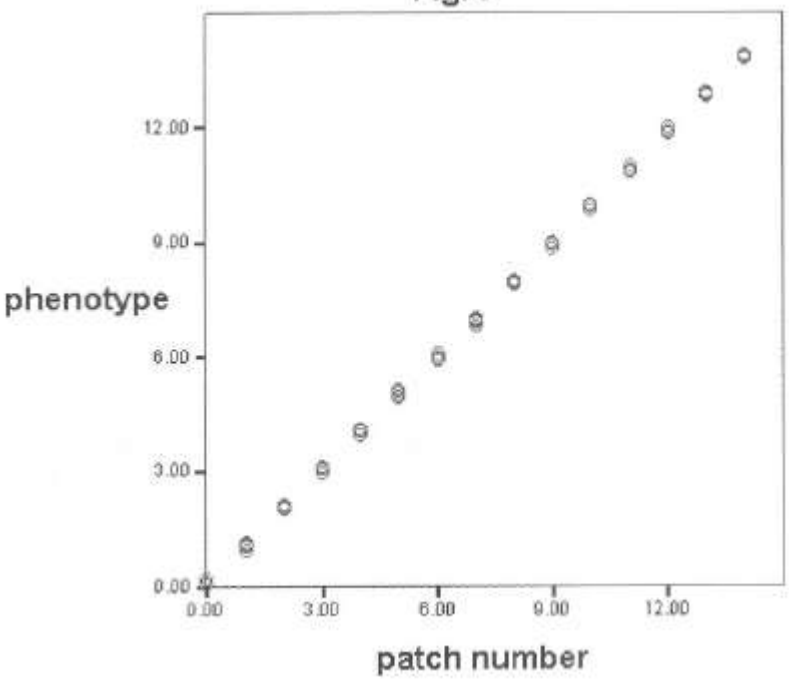
3.00
6.00
9.00
12.00
g0

Fig. 6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phenotype
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3.00
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9.00
12.00
patch number