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POPULATION REGULATION AND OPTIMAL LIFE-HISTORY STRATEGIES

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1 Introduction

Today one of the most exciting problems of evolutionary biology is the identification of the constraints determining the directions of evolutionary processes. These constraints arise both at individual and populational levels of organization. Maximization principles of population biology make it possible to give the direction of changes in a wide set of individual characters if the actual constraints are known. Maximization of the Malthusian fitness is the soundest optimization criterion, as the maximization of the population growth rate is very close to the original concept of Darwinian fitness. The connection of the Malthusian fitness to the main fitness components can be formulated by the Euler-Lotka equation. The theory of life history evolution tries to determine the changes in the fitness components under different environmental conditions. In order to do this one must identify the constraints leading to trade-offs between the demographic parameters and their dependence on the environment.

The idea that density-dependent selection determines the direction of life history evolution has been a basic idea in evolutionary ecology for 20 years. The theory of r- and K-selection was based on this idea. Concerning density dependence the following three problems are discussed in this paper:

(i) How can the mode of population regulation constrain life history evolution?

(ii) How does population dynamics change in consequence of life history evolution?

(iii) What is the significance of the mode of population regulation in evolution?

At first we have two general remarks on a traditional way of discussing these problems, that is, on the theory of r- and K-selection. Then we present the principles of our approach and show the results of a simple model. Finally we summarize our answers to the questions raised above.

2 Two remarks on the theory of r- and K-selection

2.1 *Distinction between the r-selecting and K-selecting environments is not well-defined*

In the theory of life history evolution inspired partly by the density-dependent selection models [1,2,10] two sorts of environments are distinguished: an r-selecting one where the genotype with the highest r_0 value (rate of increase at zero density) has the highest fitness and a K-selecting one where the genotype with the highest equilibrium density is favoured. In the case of r-selection it is supposed that the population density is maintained at a smaller density than the equilibrium density for a long time. It is impossible to exist at a density lower than the equilibrium density for a long period. Local populations cannot grow in an "ecological vacuum" for ever. This period must be rather short in the evolutionary time scale. Roughgarden [10] introduced a simple discrete model of density-dependent selection, where a mortality factor - which was not taken into consideration when the genotypic equilibrium densities were defined - simulated the r-selecting environment. The population density is decreased to a low value from time to time due to this mortality factor. This value was considered to be much lower than the "equilibrium density". So not all sorts of mortalities were taken into account when the equlibrium densities (K_{ij}) of the genotypes were defined. There is not any general rule to decide which mortality should and which one should not be considered when the genotypic equilibrium densities are defined in a given environment. The consequence of this uncertainty is that different K_{ij} values may be calculated for the same genotypes in the same environments. As the definition of the "r_0 selecting environment" is based on the above-discussed arbitrary exclusion of some mortality factors at the definition of the genotypic K_{ij} values, the concept of the "r_0-selecting environment" is rough. If one wants to describe population dynamics and density-dependent selection, the simplest and the only general way for defining K is to involve every mortality in the definition of the genotypic K_{ij} values. However, as it was proved several times, using this definition maximizing r (at the given density) is equivalent to maximizing K, so in this sense every population is K-selected under a wide range of conditions [3,12]. Though in the case of varying environments there are very difficult averaging problems, which have not been resolved generally yet [4], the general way of defining equilibrium density must remain the same.

2.2 *Any theory of life history evolution must involve demography*

Even if a clear distinction were made between the r_0-selecting and K-selecting environments one could predict nothing concerning life history evolution if only models of the above mentioned sort were used. It is evident that if you want to have a theory that can

predict life history parameters in different schemes of density-regulation in various environments, these parameters must be involved in the theory explicitly. The theory of r,K-**strategy** predicts the expected values of such traits like fertility, longevity, individual growth rate, size, etc. under various environmental conditions. These traits are not involved in the models of density-dependent selection and their relations to the parameters involved in the model are not straightforward at all. Both r_0 and K can be increased by increasing the fertility as well as by decreasing the mortality. On the other hand models involving these traits, i.e. demographic life history optimization models, very often ignore density dependence. The "general life history problem" was defined without even referring to population density [11] and the authors of the "integrated approach to life-cycle evolution" [12] also forgot that **the optimization problem can be altered by the difference between the equilibrium densities in different environments.** The difference between the interests of field biologists and model makers is even more obvious if we notice that such problems like finding the conditions under which organisms can survive only with short life, small size, and many offsprings has never been considered explicitly in general life history models involving density dependence.

3 Some principles

3.1 We regard the life history strategy as a vector of the demographic parameters. Only those differences in the environment can change the optimal life history strategy which change the admissible set of the life history strategies (Fig. 1).

This principle also means that some of the fitness components and in most of the cases the absolute selective value of a genotype will differ in different environments.

3.2 The optimal stragegy may differ in different environments as a joint result of direct and indirect environmental effects on the option set.

Environmental differences (e.g. the presence or the absence of a predator) may change the demographic parameters directly (e.g. juvenile survival decreases in case of nest predation) and also indirectly via the density-dependent demographic parameters by changing the equilibrium population density (e.g. in the previous case competition between the juveniles can be reduced at the same time due to lower population density). Assuming small changes these environmental effects on the optimal strategy can be traited as they were additive and independent of each other.

3.3 Density dependence of the optimal life history strategy can be ignored only if each element of the considered strategy is independent of the population density.

Population density must be regulated. "...no population can be driven entirely by density-independent factors all the time." [6]. Species without density-regulation quickly become extinct. In turn at least one of the demographic parameters must be density-dependent. If the investigated life history traits depend

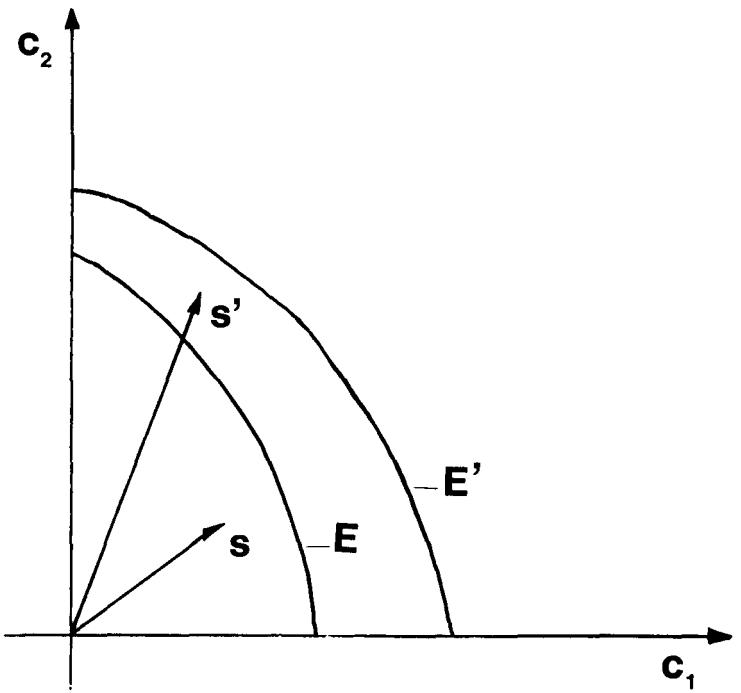

Figure 1. c_1 and c_2 are two demographic parameters. The admissible sets of strategies in the environments E and E' are represented by the areas below the appropriate curve. The strategy s' is admissible only in the environment E', but s is admissible in both environments.

on the density-dependent parameters then the admissible set of the life history strategies is changed by the changed equilibrium density. Thus the optimal strategy may be changed due to density dependence (see Priciple 1).

3.4 Fertility can increase above a certain threshold only at the cost of juvenile or parental survival [13].

3.5 If this cost is density-dependent then it is lower at low population density than at high population density [5]. Consequently optimal fertility will be higher in populations with low critical density than in populations with high population density in this type of species.

Introducing the concept of r-, K-selection Macarthur & Wilson [5] write: "**In an environment with no crowding** (r-selection), **genotypes which harvest the most food** (even if wastefully) **will**

rear the largest families and be most fit. Evolution here favors productivity. At the other extreme, **in a crowded area,** (K-selection), **genotypes** which can at least replace themselves **with a small family** at the lowest food level **will win,** the food density being lowered so that **large families cannot be fed.** Evolution here favors efficiency of conservation of food into offspring - there must be no waste."

This argumentation supposes that large families are more sensitive to crowding than small ones (as high fertility is coupled with low efficiency and vice versa). Principle 5 is a generalization of this widely accepted assumption. It may be called MacArthur principle.

The principle seems to be applicable whenever sib competition occurs besides competition among the juveniles and/or their parents. This wide applicability explains why the theory of r-, K-strategy works in many situations.

One of those rare models where the effect of density dependence on the optimal strategy is considered was developed by R.E. Michod [8]. He dealt with the effect of increased age-specific mortality on the age-specific reproductive effort. In his model mortality increased only in one age group. He proved that the optimal reproductive effort increases before and decreases after the afflicted age group as a response to the direct effect. The effect of density increases the optimal reproductive effort in every age group, because the MacArthur principle was involved in the assumptions of the model (Michod, p. 532).

4 A model for an example

Here we give a simple model to present an application of the principles. Let us see a population breeding seasonally. Let the population density be N_1 at the beginning and let it be N_2 at the end of the breeding season. It is supposed that juveniles - if they survive - become adults by the end of the breeding season. (This is the definition of the breeding season.) It is also supposed, that every adult is equivalent, so neither N_1 nor N_2 have to be decomposed into age groups. For simplicity, we will refer to the interbreeding period as "winter" and population densities before and after the breeding period as spring and autumn densities.

The annual recruitment rate, λ is decomposed into two parts: λ' is the recruitment rate within the breeding season (e.g. the value N_2/N_1), and ℓ is the interbreeding survival of an individual.

There are genotypes with different fertilities denoted by n in the population. Obviously λ' depends on fertility. It is supposed that winter survival is independent of fertility. So the dynamics of a population of females with n female offspring can be given by the next simple equations:

$$\lambda = \lambda'(n) \cdot \ell \quad (1)$$

$$N_2 = N_1 \cdot \lambda'(n) \quad (2)$$

$$N_1 = \ell \cdot N_2 \quad (3)$$

Obviously in an ecological equilibrium the annual recruitment rate is one.

$$\lambda = \lambda' \cdot \ell = 1 \qquad (4)$$

Trivially the ratio of densities before and after the breeding season are determined by both ℓ and λ'. From (2) and (4):

$$N_2/N_1 = \lambda' = 1/\ell \qquad (5)$$

Genotypes with higher fertilities have lower juvenile or adult survivals (Principle 4). Thus the optimal fertility is not the largest possible one, but the one which maximizes λ'.

As the population must be regulated (Principle 3) the mode of this regulation should be defined.

Here we have two variables describing population dynamics. At least one of them must be density dependent. Simplifying the teachings of the classic discussion about population-regulation one important difference between the opinions can be discovered. Birch's rejection of "density-dependent regulation" was based on his experience that the advantageous periods for population growth are not long enough for leading to competition. On the other hand D. Lack argued that many populations can be regulated by competition for food or by predation etc. in the advantageous period. Therefore we call the regulation "Lack-type regulation" when density dependence is present in the breeding (the advantageous) season, but not in winter. The meaning of "Birch-type regulation" is just the opposite: only winter survival depends on density.

Let us compare the optimal strategies (characterized here only by fertility) and the population dynamics of a single species in two environments. Let us suppose that one of the environments is more favourable than the other.

In the simplest situation two cases can be distinguished: one where the difference in the external conditions has some effects only on the breeding season parameters, and the other case where it affects only the winter survival.

As we consider two sorts of population-regulation we have four different cases to study (Tables 1 and 2).

What will be the difference in the population dynamics in the four situations? There are two cases where the difference between the environments concerns density-dependent parameters. Let us consider the case where the direct effect of the unfavourable environment decreases the winter survival but does not affect the breeding season parameters so does not have any selective effect on the fertility. If nothing changed the annual recruitment rate would become lower than one in the worse environment. As the population density in winter decreases density-dependent mortality decreases as well until the winter survival reaches its original value again. Thus the environmental effect may be compensated by a decrease in the rate of density-dependent mortality at a lower population density in winter. In this new equilibrium both the spring and the autumn density is lower than before but their ratio

Table 1:
Changes of the equilibrium population densities

Density-dependence	Decrease in the interbreeding season (ℓ)	Decrease in the breeding season (λ')
ℓ	N_1/N_2 = const N_1,N_2 decreases	N_1/N_2 increases N_2 decreases N_1 decreases (?)
λ'	N_1/N_2 decreases N_1 decreases N_2 decreases (?)	N_1/N_2 = const N_1,N_2 decreases

Notations:

n = fertility
λ' = recruitment rate during the breeding season
ℓ = survival in the interbreeding season (independent of n)
E = environmental variable (the demographic variables are decreasing functions of E)
N_1 = population density at the beginning of the breeding season
N_2 = population density at the beginning of the interbreeding season (when ℓ is density-dependent)

does not change. As λ' is density-independent and the difference between the environments does not lead to selection, there is no other way for compensation. If the population density becomes zero before ℓ increases to its original value the population extincts. In that case where λ' is density dependent and the environmental effect acts in the breeding season the difference between the environments leads to similar changes in the population dynamics (Table 1).

In the other two cases the direct and indirect effects act in different seasons. In these cases higher recruitment rate in the breeding season compensates for the higher winter mortality (when the winter conditions become more severe) or a lower winter mortality compensates for the less successful breeding (when the breeding season becomes less advantageous). Consequently the ratios of the autumn and spring densities change. However, the equilibrium population density before the density dependent season must be lower in the worse environment because this is the reason

Table 2:
Conditions for increasing optimal fertility
with increasing mortality (i.e. conditions for $dn/dE > 0$)

Density-dependence	Decrease in the interbreeding season (λ)	Decrease in the breeding season (λ')
λ	$n = \text{const.}$	$\frac{\partial^2 \lambda'}{\partial E \partial n} > 0$
λ'	$\frac{\partial^2 \lambda'}{\partial N \partial n} < 0$	$n = \text{const.}$

Notations: see Table 1.

of the compensating effect. It is very probable that the population density is also lower after the density-depdendent season although it is not necessary. For instance if density dependence in winter is very strong, the lower autumn density can cause higher population density in spring than that in the better environment.

Of course, if density dependence is not strong enough to compensate for the deterioration of the environment the population extincts.

What is the difference between the optimal fertilities (Table 2)? The question can be answered if we notice that the optimal value is implicitly defined by the optimality condition:

$$\frac{\partial \lambda'}{\partial n} = 0$$

and that λ must be one everywhere. Using the implicit function theorem the condition of increasing fertility with harshening environment can be given [7,9]. The result is given in Table 2. There is only one case where the condition is generally satisfied in nature. The condition to be satisfied is just the MacArthur principle (Principle 5), the case where the difference between the environments acts on winter survival and the breeding season parameters depend on density. So increased winter mortality and consequently decreased spring density results in increased optimal fertility in populations with Lack-type regulation.

In the case of Birch-type regulation the MacArthur principle is not applicable. If the direct effect of the environment decreases the fertility-related survivorships in the breeding season it is probable that genotypes with higher fertility will be affected

more so the optimal fertility will tend to decrease in these populations. Obviously if both the direct environmental effect and the change in the critical density act only on winter survival, the optimal fertility does not change.

In the last case maximization of λ' is affected by both the direct and indirect effects of the differences between the environments. These effects tend to change the optimal fertility in opposite directions and can compensate for each other. If only the resource level is lower in the worse environment and only the per capita resource level affects the demographic parameters the population density decreases to a level at which the per capita resource level is the same as before. In that case deterioration of the environment is compensated simply by some decrease in population density and optimal fertility does not change [7].

Let us summarize what we can say about the joint changes in the optimal strategy and the population density!

To maintain the balance between birth and death rates the population has to increase its fertility or decrease its mortality in an environment with a new source of mortality. This increase/decrease can be a consequence of decreasing population density or a consequence of spreading of a new phenotpye by direct selection. The effect of decreasing population density can be selective as well (e.g. MacArthur principle). If these factors cannot compensate for the increased mortality, the population extincts.

Our simple example shows that it is possible, at least in principle, to calculate both the optimal strategy and the corresponding equilibrium population density. The model can be generalized for involving a finite number of quantitative traits (Géza Meszéna manuscript). Moreover the optimal strategy with zero equilibrium density can also be calculated. The population can adapt to the environmental change if the optimal λ is greater than one. Giving the species specific constraints of the individual organization makes it possible to judge if the species can adapt to certain conditions characterized by their effects on the fitness components. We are still far from the practical applications.

Concerning the optimal phenotype we can ask, when a new phenotype with higher fertility can spread as a consequence of a new mortality factor. The following conditions together are sufficient:

(i) Environmental change decreases the density of the critical age-group.

(ii) At least one of the demographic parameters which are in trade-off with fertility is density-dependent.

(iii) Disadvantage of higher fertility decreases with lower density (MacArthur principle).

(iv) There is no direct environmental effect (or it is small) which could increase the disadvantage of higher fertility.

Of course, it might happen that the direct environmental effect decreases the disadvantage of higher fertility and increases the optimal fertility. However, in that case we have to suppose that the new mortality factor has a stronger effect on the phenotype with lower fertility than on the one with higher fertility.

5 Conclusions

Let us see the questions to be answered! How can density dependence influence life history evolution? Are we right to say, e.g. that density-dependent selection leads to high fertility, short life span and quick growth at relatively low equilibrium population density? Even if populations of a single species are compared, the differences between these traits can be explained most easily by some combinations of the direct and indirect effects of the differences between the compared environments. If a species is widely distributed for instance, its winter mortality is certainly higher in the northern populations than in the southern ones. If it is a species with Lack-type regulation then both fertility and individual growth rate can increase due to the density-dependent selection, as not only juvenile survival but individual growth rate can depend on fertility and density. However, shorter life-span can be the direct result of increased mortality and the selection for quicker growth can be the direct result of the shorter advantageous period. The mode of population regulation has even smaller role in interspecific or in other intertaxonomic comparisons where such taxon specific constraints like the differences in body size explain most of the differences between the life-history traits and their covariations (see e.g. Stearns [14], as the first of a series of papers with the same conclusion). Thus one can presuppose a strong density effect on genecological variations and much smaller effect on evolutionary variations. Consequently the evolutionary significance of density-dependence is certainly less than its ecological role.

The consequences of life histroy evolution in the dynamics of populations are the results of the direct and indirect environmental effects and all the congruent selective processes must be considered in the calculations.

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