Population Density and Optimal Body Size

BY

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Abstract.–PERRIN N., 1990. Population density and optimal body size. *In*: Dynamical Models in Biology, R. Arditi (ed.). *Mém. Soc. vaud. Sc. nat. 18.3*: 169-179.

The population density of an organism is one of the main aspects of its environment, and should therefore strongly influence its adaptive strategy. The r/K theory, based on the logistic model, was developed to formalize this influence.

K-selection is classically thought to favour large body sizes. This prediction, however, cannot be directly derived from the logistic model: some auxiliary hypotheses are therefore implicit. These are to be made explicit if the theory is to be tested.

An alternative approach, based on the Euler-Lotka equation, shows that density itself is irrelevant, but that the *relative* effect of density on adult and juvenile features is crucial. For instance, increasing population will select for a *smaller body size* if the density affects mainly adult fecundity and/or survivorship.

Thus, an implicit assumption of the classical r/K predictions appears to be that density affects mainly juvenile growth and/or survival. In this case, density should indeed favour large body sizes. The theory appears nevertheless inconsistent, since a probable consequence of increasing body size will be a decrease in the carrying capacity.

Key-words: body size, density-dependent selection, optimization, life-history theory.

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La densité d'une population est l'un des facteurs majeurs de l'environnement des organismes qui la composent, et de ce fait doit influencer leur stratégie adaptative. La théorie r/K, fondée sur le modèle logistique, a été élaborée pour formaliser cette influence.

Il est classiquement admis que la sélection K favorise une grande taille corporelle.

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Cependant, cette prédiction ne peut être directement déduite du modèle logistique: certaines hypothèses auxiliaires sont donc implicites. Ces hypothèses doivent être explicitées pour pouvoir juger de la cohérence de la théorie.

Une approche différente, basée sur l'équation d'Euler-Lotka, montre que la densité en elle-même ne joue aucun rôle: c'est l'influence *relative* de la densité sur les caractères adultes et juvéniles qui importe. Notamment, une augmentation de la population entraînera une *diminution de la taille* optimale si la densité affecte principalement la fécondité et/ou la survie adulte.

Ainsi apparaît une hypothèse implicite aux prédictions classiques de la théorie r/K: la densité de population est supposée affecter principalement la croissance et/ou la survie juvéniles. Dans ce cas, l'accroissement de densité entraînera effectivement une augmentation de la taille corporelle. Une incohérence subsiste néanmoins dans la théorie, puisqu'une des conséquences probables de l'augmentation de taille sera une diminution de la capacité de soutien.

POPULATION DYNAMICS AND LIFE-HISTORY STRATEGIES

Studies in population ecology and demography are basically concerned with the dynamics resulting from the interaction between a population and its resources. A *life-history strategy* (LHS) can be defined as the interface in this interaction. The LHS consists both of the organism's *acquisition strategy* (i.e. the way it acquires resources from its environment), and *allocation strategy* (i.e. the way it allocates acquired resources, e.g. to maintenance, growth, and reproduction); this allocation strategy directly determines the organism's *per capita* rates of fecundity and mortality, and thereby its *per capita* rate of increase. Thus, on an *ecological time scale*, the LHS determines the rate at which resources are transformed into individuals and, so, it affects both resource and population dynamics.

On an *evolutionary time scale* conversely, resources contribute to shape the LHS by natural selection: the phenotypes which are the most able to transform a given resource into individuals, have thereby the highest *per capita* rate of increase, and are *ipso facto* those that make the highest genetic contribution to the future generations in this environment. Genetic evolution will progressively adapt populations to their particular environment, until some optimal, evolutionary stable, strategy is eventually reached. Thus, natural selection can be considered as an optimization process, the optimal LHS being defined as that strategy, among the set of all feasible ones, which confers the highest *per capita* rate of increase F in the environment under consideration.

Now, the population itself is a part of the organism's environment. So, the question can be asked: does the population density, or population dynamics, constitute a selective pressure of its own, does it therefore affect the optimal LHS?

THE r/K THEORY

The r/K theory (MacArthur and Wilson 1967) was probably the first formalized attempt to establish a causal link from population dynamics to phenotypic features. As indicated by its name, this theory relies on the logistic equation, which describes the *per capita* rate of increase $F = (1/N)dN/dt$ as a decreasing linear function of population density N

$$F = r(1 - N/K) \tag{1}$$

where r and K are environment- and genotype-specific parameters. If a negative genetic covariance exists between the two parameters r and K, then the optimal genotype (i.e. which confers the highest F-value) will vary according to population density: those genotypes which confer high r will be favoured in expanding populations, and those which confer high K in stable populations (fig. 1).
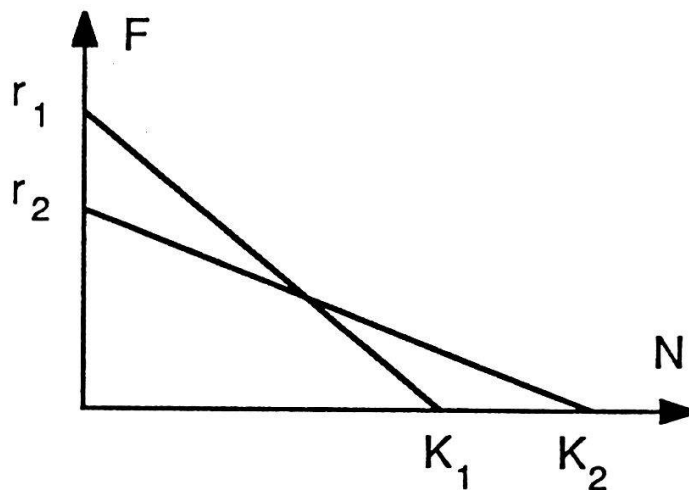
Figure 1.–The logistic equation expresses the *per capita* rate of increase F as a linear, decreasing function of population density N, so that the population dynamics is described by two parameters only, r and K. The r/K theory states that those genotypes which confer high r will be selected for in low density populations (here genotype 1), and those genotypes which confer high K in high density populations (here genotype 2).

Now, which phenotypic features are positively correlated with r, and which ones with K? Pianka (1970, 1972) enumerates a list of so-called «r/K correlates», from which it can be seen, for instance, that both large adult- and offspring-sizes are considered K-features.

Such statements are largely widespread in the present ecological literature. However, they are open to criticism, being based on some kind of

«ecological intuition» rather than on mathematical arguments. In fact, neither adult- nor offspring-size appears as explicit parameters in the logistic equation, so that these statements rely on some implicit assumptions.

Which ones? It is rather surprising to note that, under the simple and highly plausible assumption of a positive relationship between an organism size and the amount of resources it uses, the r/K theory generates quite opposite predictions. Indeed, the carrying capacity, or equilibrium population K, will thereby be negatively correlated with organism size; since, by definition, K-selection should result in an increase in the carrying capacity (see, however, the Appendix), it should actually favour smaller sizes (BOYCE 1984, PERRIN 1987).

ORGANISMS ARE DYNAMIC SYSTEMS

Though opposite to the classical ones, some predictions on optimal body size can thus be made from the r/K theory, by adding auxiliary hypotheses on the specific relationships between organism size and resource use. However, it does not make much sense to define an organism's unique optimal body size, for size is not a constant parameter, but varies through life (fig. 2).
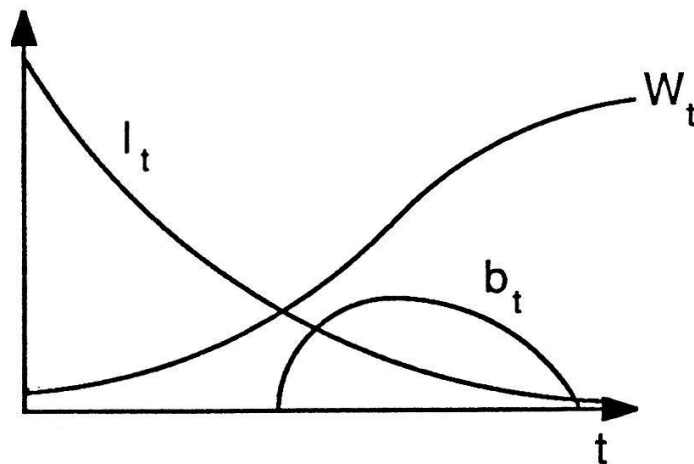
Figure 2.–The Euler-Lotka equation expresses the *per capita* rate of increase F as a function of the life-long schedules of survivorship l_t and fecundity b_t. Due to the physiological trade-offs in allocation strategy, the growth curve W_t also strongly depends on these schedules.

So, what is optimized by natural selection is not a size, but a growth process, resulting from a life-long strategy of allocating resources into growth, rather than into reproduction or maintenance.

Thus, when developing a theory of optimal growth, the first step will be to express the *per capita* rate of increase F as a function of this life-long strategy, then to find a maximum, e.g. by means of dynamic optimization.

The r/K approach considers organisms as sets of fixed parameters, so is unable to take account of the dynamic aspect of the problem.

From the Euler-Lotka equation

$$\int e^{-Ft}l_t b_t \, dt = 1 \, , \tag{2}$$

F is a function of the age-specific schedules of survivorship l_t and fecundity b_t (fig. 2). As can be seen, neither organism size nor population density appears in this equation. The size, however, depends on the energy allocation into growth, which, due to physiological trade-offs, also affects both survivorship and fecundity. Thus, knowing, or making assumptions about, the relationships between this life-long allocation strategy and the age-specific rates of mortality and fecundity, permits making predictions on the optimal growth process (SIBLY et al. 1985) or, equivalently, on the optimal size at a given age.

In a similar way, knowing, or making assumptions about, the relationship between population density N and dynamics F (e.g. using the logistic equation) will permit making predictions concerning the effect of N on the optimal growth process.

THE PROBLEM HAS NO ANALYTICAL SOLUTION

Equation (2) defines F as an implicit function of survival and fecundity, whereas the methods of dynamic optimization are designed to maximize an explicit functional (e.g. LEON 1976). This difficulty is partially circumvented by noting (TAYLOR et al. 1974) that the optimal LHS $(l, b)^*$ maximizes the function V

$$\max_{(l,b)\in L} V = \int e^{-F^* t}l_t b_t \, dt \, , \tag{3}$$

where L is the set of all feasible LHS and F^* is a constant, calculated from (2) for this particular life-history $(l, b)^*$. In other words, given this value of F^*, all other feasible life-histories will provide values for V which are equal to, or less than unity.

Such a shift from an implicit to an explicit functional renders part of the problem tractable by means of dynamic optimization (e.g. the Pontryagin Maximum Principle from optimal control theory: LEON 1976, SIBLY et al. 1985, PERRIN et al. 1987).

The problem remains, however, that F^* cannot be computed without knowing $(l, b)^*$, whereas, from eq. (3), F^* has to be known in order to derive $(l, b)^*$. Equation (3) therefore has no analytical solution, and an analysis can only provide necessary conditions for a given (l, b) to be the solution.

How Could Organisms Be Adapted to Rates of Increase
which Differ from Zero?

From eq. (3), organisms are predicted to maximize their life-long expectancy of reproduction, weighted by the age-specific factor e^{-F^*t}. This makes explicit the fact that it is the *actual* value of F that affects the optimal strategy: for example, the higher the value of F^*, the stronger should be the selective pressure for early maturity, since the more quickly this weighting factor decreases with age.

There seems therefore to be good *a priori* reasons to expect a causal link from population dynamics to LHS; such a relationship will be discussed more specifically thereafter, using the examples of optimal size at maturity and at birth. But the question must be asked first: how could actual organisms be adapted to rates of increase which differ from zero? Indeed, the argument can be made that, however variable a population is on a short (ecological) time scale, it is stable on an long (evolutionary) time scale. Since F averages to zero, the organism's LHS should be adapted to this null value of F, and be unaffected by population dynamics.

Were the organism nonplastic, i.e. were the phenotype univocally determined by the genotype, then this argument would hold. The organism's LHS should indeed be adapted to the average, null value of F; as shown by MATESSI and GATTO (1984), the profiles of K- and r-strategists should coincide exactly, were the r-strategists nonplastic.

A fluctuating environment, however, should select those genotypes that are able to produce different, locally adapted, phenotypes, and such a phenotypic plasticity should allow organisms to track fluctuating *per capita* rates of increase. Thus, phenotypic plasticity not only provides an attractive testbed for life-history theory (PERRIN 1988), but seems actually a necessary prerequisite for an organism to be optimal, when its rate of increase differs from zero.

Optimal Size at Maturity

Static vs Dynamic Approach

To introduce the problem, let us first assume a constant mortality rate μ, and a size-dependent, unimodal production rate p_W (fig. 3a).

The best size to reproduce could appear, on some «intuitive» grounds, to be that for which the production rate, i.e. the potential fecundity, is at a maximum (e.g. LYNCH 1980, SEBENS 1982; fig. 3a):

$$p'_W = 0 \, . \tag{4}$$

However, it can be shown (e.g. TAYLOR *et al.* 1974, LEON 1976) that, for

a maximum in V (eq. 3), the organism should in fact stop growing when (fig. 3a)

$$p'_W = F + \mu . \tag{5}$$

In order to contrast conditions (4) and (5), it should be noted first that, since F can be defined as $\beta - \mu$ (where β is the *per capita* rate of fecundity), the conditions (4) and (5) are equivalent only in a non-reproducing population. As a matter of fact, the «intuitive» approach leading to condition (4) fails to take account of the dynamic aspect of the problem.
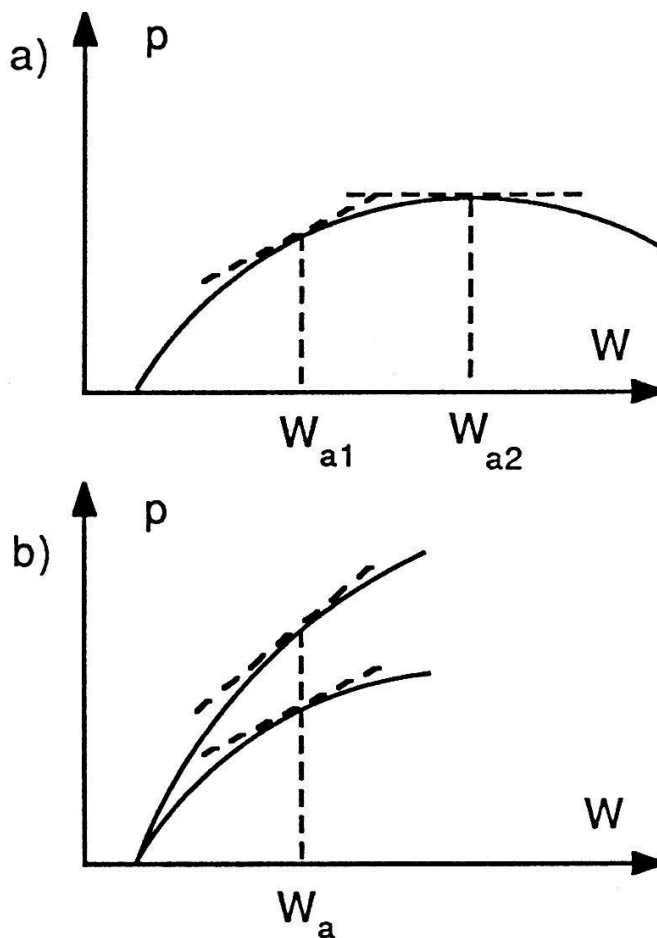

Figure 3.–(a) The organisms should not reproduce when the production rate p is at a maximum relative to size W (W_{a2}), but when $p'_W = F + \mu$ (W_{a1}), where F and μ are respectively the *per capita* rates of increase and of mortality. A decrease in F would shift the point of contact, and thereby the optimal adult size, to the right. (b) An increase in p_W results in an increase in F such that the optimal adult size W_a remains unchanged.

A biological interpretation of the dynamic approach leading to condition (5) is as follows: at each size W, an organism has to invest p_W into either growth or reproduction. As long as p'_W is positive, growing further is beneficial in terms of potential fecundity; delaying maturity however entails

two costs: (i) the probability to survive until maturity decreases at rate μ, and (ii) later produced offspring contribute less to the population dynamics F than do earlier produced ones (see comments on eq. 3). The eq. (5) just describes the condition where the costs of further growing offset the benefits. The «intuitive» approach can also be discarded on an empirical basis: for instance, laboratory observations on the cladoceran *S. vetulus* show that animals reproduce long before attaining the size at which production is at a maximum (PERRIN *et al.* 1987).

Age-Dependent Density-Dependence

Now, does the population density affect the optimal size at maturity, and how? From (5), and unlike (4), it can be seen that population dynamics F affects adult size. Assuming a negative relationship between F and N, the prediction appears that, by decreasing F, an increase in population density N would select for larger adult size (fig. 3a).

Does it mean, as suggested by LEON (1976), that the classical r/K prediction is thereby corroborated? Actually, it is not. The point must be made that F cannot change independently from production or mortality rates. Since $F = \beta - \mu$, any density-induced increase in mortality rate μ would decrease F in such a way that the sum $F + \mu$, and therefore the optimal adult size, will remain exactly the same. Similarly, a density-induced decrease in production rate p_W (and thereby in marginal production rate p'_W) would result in a parallel decrease in $F + \mu$, so that the optimal adult size will remain exactly the same (fig. 3b, and unpublished results).

So, the question arises: can the population dynamics, or population density, really affect the optimal adult size? The answer is: yes, it does. The point is that F is a function of both adult and juveniles features, which can vary independently of each other: for instance, F can decrease because of changes at the juvenile level only, whereas adult mortality μ and marginal production rates p'_W remain unchanged.

Thus, the condition (5) is to be interpreted as follows: if the density-dependence in F is mainly due to changes in juvenile growth or survival rates, then the optimal adult size should increase with population density. In contrast, if the density-dependence in F is mainly due to changes in adult fecundity or survivorship, then the adult size should decrease with increasing population density.

OPTIMAL OFFSPRING SIZE

A similar analysis can be made for offspring size (SIBLY and CALOW 1983, 1985, TAYLOR and WILLIAMS 1984, PERRIN 1988, 1989). A condition for

a maximum in the *per capita* rate of increase F relative to size at birth is (fig. 4)

$$g = F + \mu,$$ (6)

where $g = (1/W)dW/dt$ is the relative growth rate, and μ is the mortality rate.

Again, it can be seen that the optimal solution depends on the actual value of F. Relating F to N through the logistic equation shows that a decrease in F (that is, an increase in population density N) will select for smaller offspring. In this case, the prediction is quite opposite to the classical one, i.e. that offspring size should increase with increasing population density.

However, it is again worth noting, firstly, that F, g and μ are not independent from each other, so that any change in juvenile growth g or mortality μ will also affect F, secondly, that F is also a function of adult features. Thus, again, the condition (6) is to be interpreted as follows (SIBLY and CALOW 1985, PERRIN 1989): if the density-dependence in F is mainly due to changes in adult fecundity or survivorship, then the optimal size at birth should decrease with increasing population density. In contrast, if the density-dependence in F is mainly due to changes in juvenile growth or survival rates, then the optimal size at birth should increase with increasing population density.
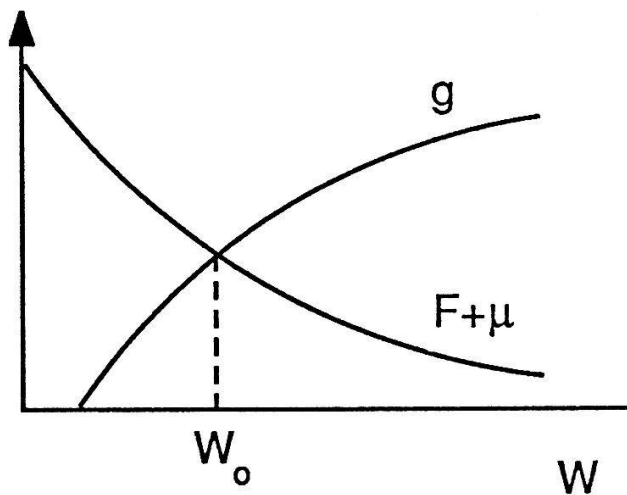

Figure 4.–A condition for the size at birth W_o to be optimal is $g = F + \mu$. $g = (1/W)dW/dt$ is the relative growth rate, F and μ are respectively the *per capita* rates of increase and of mortality. A decrease in F would shift the intersection point to the left, and thereby select for smaller offspring.

This theoretical result is again supported by empirical data: for instance, experiments with the cladoceran *S. vetulus* showed (i) that the density-dependence in F was mainly due to changes in offspring growth rate; (ii)

that offspring size was phenotypically plastic, and increased with density according to the predictions (PERRIN 1989).

CONCLUSIONS

(1) Both from the above analytical and experimental results, the population dynamics is expected to affect optimal body size, but not in the somewhat simplistic way proposed by the classical r/K theory .

(2) A dynamic approach that integrates the whole life cycle appears to be necessary since, what is optimized by natural selection is not a size, but a growth process. By considering organisms as sets of fixed parameters, the r/K theory fails to take account of the dynamics of this process.

(3) Contrary to classical r/K statements, no prediction on optimal adult or offspring size can be derived from population density or dynamics alone: the relative density-dependence of both juvenile and adult features must be known in order to make specific predictions.

(4) An implicit assumption of the classical r/K theory appears to be that the density-dependence in the *per capita* rate of increase F is mainly due to changes at the juvenile level. This assumption is plausible, but must be made explicit.

(5) Under this assumption, the prediction of increasing both offspring and adult sizes with increasing density is correct, but should not be considered as K-selection, since a predictable effect of increasing organism size is a decrease in the carrying capacity K.

APPENDIX

K-selection is sometimes identified to density-dependent selection (e.g. GREEN 1980, MATESSI and GATTO 1984); that is, referring to the prevailing ecological conditions, rather than to the outcome of selection. K-selection is thereby allowed to decrease the organism carrying capacity.

This in fact is a matter of definition; I think however that any reference to the logistic equation, and to MACARTHUR's formalization, would not make sense any more, were K-selection identified to density-dependent selection. So it is probably less confusing to define K-selection as a density-dependent process which results in increasing the carrying capacity (BOYCE 1984).

The point remains that a probable outcome of density-dependent selection could be in many cases a decrease in carrying capacity K (GREEN 1980, MATESSI and GATTO 1984, PERRIN 1987).

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