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Adaptive strategies in size-structured populations: Optimal patterns and perturbation analysis

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ABSTRACT

In some species of plants, fish and invertebrates, life histories are best classified according to size. The probability of surviving, and of growing or staying in the same stage, are size-dependent. So is fecundity for adult females. Trade-offs exist between these traits due to finite energy availability; these constrain the maximization of fitness. The optimization model developed here predicts the occurrence of life cycles of individuals with determinate and indeterminate growth, based on its actual pattern of size-specific reproductive values. A perturbation analysis is performed to evaluate the direction of change in optimal phenotypes when mortality factors, specific to a given size class, increase. The model predicts that natural selection favours an extra investment of energy in growth and fecundity during the affected stage if the mortality factor perturbed is unavoidable, or an extra investment in protection if it is avoidable. For all other stages, the response depends on patterns of energy allocation before perturbation. Strategies for sizes smaller than the one where perturbation occurs are opposite to strategies in the larger ones.

Keywords: avoidable and unavoidable mortality, constrained optimization, life-history evolution, perturbation analysis, reproductive values, size-structured populations.

INTRODUCTION

The optimization approach to study the evolution of life histories assumes that some measure of fitness is maximized, subject to constraints and trade-offs between life-history traits. If sufficient genetic variation exists, optimal phenotypes are expected to occur. Perturbation analysis allows us to infer the direction of change of optimal values when the environment varies. This theoretical framework has been used widely in non-structured and age-structured life histories (Schaffer, 1974; Law, 1979; Michod, 1979; León, 1983, 1988; Caswell and Real, 1987; Hernandez and León, 1995).

The structure in a life history refers to the categorization of its vital rates according to a variable such as age, size, instar, and so on. Here, we consider life histories with a size structure. After birth, an individual grows from one stage to another. Each size class is characterized by specific values of fecundity, survivorship and growth rate, regardless of

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age. As is widely accepted, such a classification is more appropriate for many species of plants and fish, and some invertebrates, than an age-structured one (Caswell, 1989; Roff, 1992; Takada and Caswell, 1997).

This paper has two main parts. First, an optimization model was developed assuming that population growth is maximized subject to trade-offs between size-specific traits. Comparisons of natural and theoretical life-cycle patterns are discussed. Two important conceptions are moulded here. One is the relevance of considering the juvenile and the adult phases separately, each being size-classified. The other is the crucial role played by the pattern of size-specific reproductive values in characterizing them. Second, a perturbation analysis was performed with a comparative statistics approach: the direction of change of optimal phenotypes was evaluated, but no specific values were computed (Henderson and Quandt, 1971; Michod, 1979; León, in press). We found that adaptive strategies depend considerably on allocation of energy among vital functions before perturbation.

Throughout, we present analogies and comparisons between age and stage classifications. This is to emphasize the subtleties involved in defining either of them as fitted for a given life cycle. The biased development of life-history theories towards age structure sometimes makes it difficult to think in terms of any other stage unit, such as size, instar, and so on. Hence, models sometimes force age structures when it is not appropriate. The classification of a structured population must follow the natural facts, that is, the homogeneity of the vital functions according to a given trait.

OPTIMAL PATTERNS AND THE ROLE OF REPRODUCTIVE VALUE

The model

Consider a density-independent, size-structured population with stages $x = 1, 2, \ldots, n$. Stages, or size classes, are defined by size ranges within which the set of vital rates is homogeneous for all individuals, and distinct for individuals in other stages. It is important to bear in mind that size classes may contain individuals of different ages. At each stage, energy is allocated to protection, reproduction and growth. If this is finite, an optimal configuration that maximizes fitness is expected to evolve. The existence of trade-offs between these functions has been demonstrated empirically and their genetic basis corroborated (Roff, 1992: 150 ff.). The life-history parameters specific to each size class are considered concave increasing functions of their respective energy allocations. These are: f_x, fecundity of a female size x; g_x, growth increment per unit size, expressed as the probability of individual size x of growing to size $x + 1$; and s_x, survivorship of individuals from stage x to $x + 1$. The vital parameters f_x, g_x and s_x are connected via the finite energy budget. For the formal analysis, we will consider that s_x is a concave decreasing function of independent variables g_x, f_x. That is, $s_x \equiv s_x(g_x, f_x)$, where $(\partial s_x/\partial g_x) < 0$, $(\partial s_x/\partial f_x) < 0$, $(\partial^2 s_x/\partial g_x{}^2) < 0$ and $(\partial^2 s_x/\partial f_x{}^2) < 0$. For pre-reproductive stages, $s_x \equiv s_x(g_x)$ only.

The probability of individuals in stage x of growing and surviving to stage $x + 1$ is $P_x = s_x \cdot g_x$, and the probability of surviving but remaining in stage x is $Q_x = s_x(1 - g_x)$. Both events are measured every unit of time. For every size class x, there is a contribution of f_x newborns per individual (all females) of stage x to the first stage ($x = 1$) per time unit. Stage 1 is always a non-reproductive class ($f_1 = 0$). Note that more non-reproductive classes could follow if size-groups can be recognized before the onset of maturity. The dynamics of the population, in a constant and density-independent environment, are given

by $\boldsymbol{n}(t+1) = \boldsymbol{A} \cdot \boldsymbol{n}(t)$, where $\boldsymbol{n}(t)$ is the vector whose elements are the number of individuals in stage x at time t, and $\boldsymbol{A}$ is the population projection matrix:

$$\boldsymbol{A} = \begin{bmatrix} Q_1 & f_2 & \cdots & f_{n-1} & f_n \\ P_1 & Q_2 & \cdots & 0 & 0 \\ 0 & P_2 & \cdots & \vdots & \vdots \\ \vdots & \vdots & \cdots & Q_{n-1} & 0 \\ 0 & 0 & \cdots & P_{n-1} & Q_n \end{bmatrix} = \begin{bmatrix} s_1(1-g_1) & f_2 & \cdots & f_{n-1} & f_n \\ s_1 g_1 & s_2(1-g_2) & \cdots & 0 & 0 \\ 0 & s_2 g_2 & \cdots & \vdots & \vdots \\ \vdots & \vdots & \cdots & s_{n-1}(1-g_{n-1}) & 0 \\ 0 & 0 & \cdots & s_{n-1} g_{n-1} & s_n \end{bmatrix} \quad (1)$$

$\boldsymbol{A}$ is non-negative, primitive and irreducible, so the dominant eigenvalue λ is real and non-negative, and gives the asymptotic rate of population growth. λ is the measure of fitness that is maximized by an optimal combination of s_x, g_x and f_x, under the constraint imposed by $s_x(g_x, f_x)$.

The dominant right and left eigenvectors of matrix $\boldsymbol{A}$ are:

$$\boldsymbol{A}\boldsymbol{u} = \lambda \boldsymbol{u} \quad (2)$$

$$\boldsymbol{v}'\boldsymbol{A} = \lambda \boldsymbol{v}' \quad (3)$$

where $\boldsymbol{u} = (u_1, \ldots, u_n)$ is the stable stage distribution, and $\boldsymbol{v} = (v_1, \ldots, v_n)$ is the vector of stage-specific reproductive values v_x. Taking the differential of both sides of equation (2) and then the scalar product with $\boldsymbol{v}$, we get a general expression for the sensitivity of λ to changes in life-history parameters (Caswell, 1978, 1989):

$$\mathrm{d}\lambda = \frac{\langle (\mathrm{d}\boldsymbol{A})\boldsymbol{u}, \boldsymbol{v} \rangle}{\langle \boldsymbol{u}, \boldsymbol{v} \rangle} \quad (4)$$

where $(\mathrm{d}\boldsymbol{A})$ is the matrix differential of all elements of $\boldsymbol{A}$.

If we apply this to our particular size-classified matrix $\boldsymbol{A}$ in (1), we obtain the net effect on λ of changes in all its elements:

$$\mathrm{d}\lambda = \sum_{i=1}^{n} u_i (\mathrm{d}f_i + v_i \, \mathrm{d}Q_i + v_{i+1} \, \mathrm{d}P_i) \quad (5)$$

We assume here that the eigenvectors may be scaled so that $\langle \boldsymbol{u}, \boldsymbol{v} \rangle = 1$ and that the elements of $\boldsymbol{u}$ and $\boldsymbol{v}$ are given as relative values with respect to stage 1 (i.e. $u_1 = v_1 = 1$). Templeton (1980) obtained an equivalent result for $\mathrm{d}\lambda$ in age-classified models.

Using equation (5) and the explicit definitions of Q_x and P_x given above, we evaluate the partial effects on λ when g_x and f_x change, and equate to zero (conditions for maximum λ). We get:

$$\frac{\partial \lambda}{\partial g_x} = (v_{x+1} - v_x) s_x + [v_x(1-g_x) + v_{x+1} g_x] \left(\frac{\partial s_x}{\partial g_x} \right) = 0 \quad (6a)$$

$$\frac{\partial \lambda}{\partial f_x} = 1 + [v_x(1-g_x) + v_{x+1} g_x] \left(\frac{\partial s_x}{\partial f_x} \right) = 0 \quad (6b)$$

Optimal values of g_x, f_x are simultaneous solutions to these equations. As $(\partial^2 \lambda / \partial g_x \partial g_y)$ and $(\partial^2 \lambda / \partial f_x \partial f_y) = 0$ for $x \neq y$ and < 0 for $x = y$, the corresponding Hessian matrices are negative definite, which ensures a maximum λ. An optimal phenotype is given by $\boldsymbol{s}^*$, $\boldsymbol{g}^*$, $\boldsymbol{f}^*$. These are vectors whose elements are the corresponding optimal stage-specific values.

We assume that general conditions exist to ensure that the optimal configuration is ultimately established by natural selection; namely, sufficient time and genetic variability, and a constant environment.

The conditions for optimality in equations (6a,b) can be rewritten as:

$$\frac{\partial \lambda}{\partial g_x} = (v_{x+1} - v_x)s_x + W_x\left(\frac{\partial s_x}{\partial g_x}\right) = 0 \tag{7a}$$

$$\frac{\partial \lambda}{\partial f_x} = 1 + W_x\left(\frac{\partial s_x}{\partial f_x}\right) = 0 \tag{7b}$$

where

$$W_x \equiv [v_x(1 - g_x) + v_{x+1}g_x] \tag{8}$$

W_x is an average of the reproductive values of two consecutive stages weighted by the corresponding probabilities of staying or moving to the next stage. Equation (7b) is analogous to the optimality conditions in age-structured populations (Charlesworth and León, 1976). The age-classified situation is met if $g_x = 1$, and thus $W_x \equiv v_{x+1}$. That is, all individuals move to the next stage provided they survive; none can remain behind. This is a crucial difference underlying age and size classifications.

Reproductive values in size-classified models

Note that as $(\partial s_x/\partial g_x) < 0$, then the condition $(\partial \lambda/\partial g_x) = 0$ in equation (7a) cannot be satisfied if $v_{x+1} < v_x$, since $(\partial \lambda/\partial g_x)$ would always be negative. In this case, maximum λ corresponds to the minimum g_x value; that is, $g_x^* = 0$ and therefore we would expect individuals to stop growing at stage x. From a size-classification point of view, this means that this stage would be the last of the life cycle.

Thus, $v_{x+1} > v_x$ is a necessary condition for an optimal value $0 < g_x^* < 1$; that is, for further growth and therefore for the existence of a subsequent size class $x + 1$. The sufficient condition, though, involves the magnitudes of all other elements in equation (7a).

The question then is: What is the pattern of $\boldsymbol{v}$ along stages in a size-structured population? It is well known that, for age-structured populations, v_{x+1} is usually greater than v_x for ages before first reproduction, declining afterwards. Nonetheless, this is not necessarily so for size-structured populations.

Using life-cycle graphs theory, Caswell (1980, 1989) derived simple algebraic formulas for the characteristic equation and the right and left eigenvectors of any stage-classified projection matrix. We are interested in his result for reproductive values in size-classified models. The reproductive value specific to a stage x is obtained by summing all the contributions of that stage back to stage 1 (newborns). These contributions take into account the reproductive outputs and probabilities of survival not only at stage x, but along all future stages of the life cycle (i.e. from x to n), and the time required to accomplish each step. Applied to our notation, his formula becomes:

$$v_1 = 1 \qquad v_x = \frac{f_x}{\lambda - Q_x} + \sum_{j=x+1}^{n} \frac{f_j}{\lambda - Q_j} \prod_{i=x}^{j-1} \frac{P_i}{\lambda - Q_i} \qquad x = 2, 3, \ldots, n \tag{9a}$$

Using our explicit definitions of P_x and Q_x we get:

$$v_1 = 1 \qquad v_x = \frac{f_x}{\lambda - s_x(1-g_x)} + \sum_{j=x+1}^{n} \frac{f_j}{\lambda - s_j(1-g_j)} \prod_{i=x}^{j-1} \frac{s_i g_i}{\lambda - s_i(1-g_i)} \qquad x = 2,3,\ldots,n \quad (9b)$$

which can also be rewritten in a recursive form as:

$$v_1 = 1 \qquad v_x = \frac{f_x}{\lambda - s_x(1-g_x)} + \frac{s_x g_x}{\lambda - s_x(1-g_x)} v_{x+1} \qquad x = 2,3,\ldots,n \quad (9c)$$

Observe that equation (9c) expresses the reproductive value of any stage x as the sum of two elements: current stage reproduction (via f_x) and the expected contribution from future stages (via v_{x+1}, and hence f_{x+1}, ..., etc.). This follows the concept first introduced by Williams (1957, 1966) for age-specific reproductive value. In the size-classified case, these terms are weighted by the probabilities of surviving within, as well as between, stages accordingly.

Equation (9c) is useful to obtain an expression for the difference between reproductive values of consecutive stages. Rearranging we get:

$$v_{x+1} - v_x = \frac{[(\lambda - s_x)v_{x+1} - f_x]}{\lambda - s_x(1-g_x)} \quad (10)$$

For simplicity, we define

$$\alpha_x = [\lambda - s_x(1-g_x)]^{-1} \quad (11)$$

and equation (10) becomes

$$v_{x+1} - v_x = [(\lambda - s_x)v_{x+1} - f_x]\alpha_x \quad (12)$$

Note that α_x measures the time-lag involved in remaining within a given stage, and the effect that this has upon the net rate of population growth. The longer an individual remains at a given size class, the more relevant the reproductive contribution of that stage becomes. $\lambda \geq 1$ is assumed, otherwise the population would be vanishing. Thus, α_x is always positive.

For stages prior to reproduction ($f_x = 0$), equation (12) is positive, so $v_{x+1} > v_x$ always. Therefore, the condition $(\partial\lambda/\partial g_x) = 0$ required in equation (7a) can be met. Note that only this equation is needed in the juvenile stages to specify an optimal phenotype $\boldsymbol{s}^*, \boldsymbol{g}^*$. Once the first adult stage is attained ($f_x > 0$), equation (12) can be of either sign; now both $v_{x+1} > v_x$ and $v_{x+1} < v_x$ are feasible. As stated above, the last size class x of the life cycle is that for which $(\partial\lambda/\partial g_x) < 0$. This occurs when $(v_{x+1} - v_x) < -(W_x/s_x)(\partial s_x/\partial g_x)$, as can be deduced from equation (7a). Observe that this includes the condition $(v_{x+1} - v_x) < 0$.

Discussion: Predicted patterns in size-classified life cycles

Based on the arguments above, in a size-structured population, we expect life cycles where reproductive values keep climbing with size, and individuals grow, until an adult stage is reached in which either reproductive values start decreasing with size, or still increase but insufficiently so. Then growth stops.

A pattern of ever-increasing reproductive values in pre-reproductive individuals seems very reasonable: at any juvenile stage, the expected net future reproductive outcome is the

same, since it has not yet started. However, the nearer the stage is to adulthood, the greater the reproductive value is, since it has already overcome the mortality factors threatening earlier stages.

The moment of maturity represents a major breakthrough; it is the trait connecting both parts of the life cycle. Juveniles assign energy to protection and growth only. After maturity, some must also go to production. The choices are (1) to stop growing and keep the trade-off between two functions only, or (2) start sharing among the three.

Both of these patterns are observed in nature. Stearns (1992: 93) characterized these as organisms with 'determinate growth' (most insects, birds, mammals and annual plants) and 'indeterminate growth' (most perennial plants, fish, amphibians, reptiles, crustaceans, worms and molluscs), respectively. It is difficult, however, to locate in the literature data to support that these patterns, whenever a size-structure applies, correspond to reproductive values that either decrease or increase with size. Obviously, it would be desirable to compare natural facts with theoretical tenets. So far, the model hints at what to look for.

The optimization of the onset of maturity has been a classical problem in the evolution of life histories, particularly when trying to explain the occurrence of delayed maturity. However, there has been a strong bias towards the age-classified structure (e.g. Cole, 1954; Kozlowski and Weigert, 1987; Stearns, 1992). Sometimes, the influence of size has been misinterpreted as a 'variation' in age at maturity and not the contrary. Nonetheless, it is now recognized that, for populations where a size-structure is appropriate, the threshold for maturity might be determined by size and not by age (e.g. Harper, 1977; Somerton and MacIntosh, 1983; Roff, 1992). A clear distinction must be made, though. If growth rate is constant, then delayed maturity means that the organism reaches a bigger size, which might confer greater fecundity and survival. This has been one of the classical explanations for the evolution of delayed maturity. However, if the growth rate is allowed to change by reallocation of energies, time and size are not necessarily connected in this way. Then, in this context, the term 'delayed maturity' is ambiguous. A distinct concept arises: in a size-classified population, delayed maturity might mean becoming adult at a bigger size, but not necessarily later in time.

Attaining a bigger size during the juvenile phase appears to be relevant, especially for life cycles of non-growing adults. After maturity, reproductive values are expected to decrease, probably with age. This is certainly accentuated in semelparous species. For species of indeterminate growth, whatever the benefits of big size, these are still options offered through the rest of the life cycle. Reproductive values keep climbing. This contrast of sudden change of pattern at maturity in non-growers compared to a more subtle transit in growing species is closely related to the work of Takada and Caswell (1997). They used a size-structured model, based on a McKendrick–von Foerster equation, to study optimal size at maturity. They proposed that an abrupt decrease in survival or growth rate after maturation can be a selective force favouring delayed maturation.

A final word regarding the classification of structured populations. From a size-structured point of view, organisms that stop growing at a given size are lumped together into a final size-class. At this stage, organisms can either reproduce and die, or still survive and keep on reproducing for a number of time intervals. In the latter case, we expect survivorship and fecundity – and hence reproductive values – to start declining at some point due to senescence. Then, for this last stage, an age-structured classification is appropriate, where trade-offs exist between fecundity and survivorship functions only.

In the literature, there are models which use mixed classifications for structured populations (e.g. Law, 1983; Nisbet, 1997).

Geometrical interpretation of the optimization model

Figure 1 is a graphical representation of the model. In Fig. 1a – juvenile stages – the concave curve represents the trade-off $s_x(g_x)$, that is, the possible combinations of s_x and g_x allowed by the constraint. The optimal phenotype is that which maximizes λ. According to equation (7a), the slope of this curve evaluated at the optimum is $(\partial s_x/\partial g_x)^* = -s_x(v_{x+1} - v_x)/W_x$. This is the slope of the tangent straight line shown in the figure.

The family of convex curves in Fig. 1a was drawn from equation (10) (with $f_x = 0$); they reflect the geometric relationship between reproductive values and life-history traits s_x and g_x. Each of these lines has an equal λ value (iso-fitness lines). Clearly, the point where one of these isoclines is tangent to the trade-off curve corresponds to the optimum phenotype.

Observe that if $(\partial s_x/\partial g_x)$ is computed directly from equation (10), and evaluated at the optimum, the same result $(\partial s_x/\partial g_x)^* = -s_x(v_{x+1} - v_x)/W_x$ is obtained; the slope for both is the same. The curve tangent to the trade-off curve is the one with the maximum λ and v_x values. This means that both the reproductive value and λ are maximized simultaneously.

Correspondingly, Fig. 1b displays the case for adult stages. The result is analogous to the juvenile case, one dimension added accordingly. The slope $(\partial s_x/\partial f_x)^*$, evaluated at the optimum computed from both equations (7b) and (10), is $-1/W_x$. The tangent iso-fitness and trade-off surfaces determine the optimal configuration.

ADAPTIVE RESPONSES TO PERTURBATION

Assuming a homogeneous population where phenotypes of individuals are optimal, we investigate how energy is reallocated after perturbation. This consists of an increase in the intensity of avoidable and unavoidable mortality factors specific to a stage k. In mechanistic terms, we ask what mutant, endowed with a modified life history, would preferentially invade under the altered circumstances.

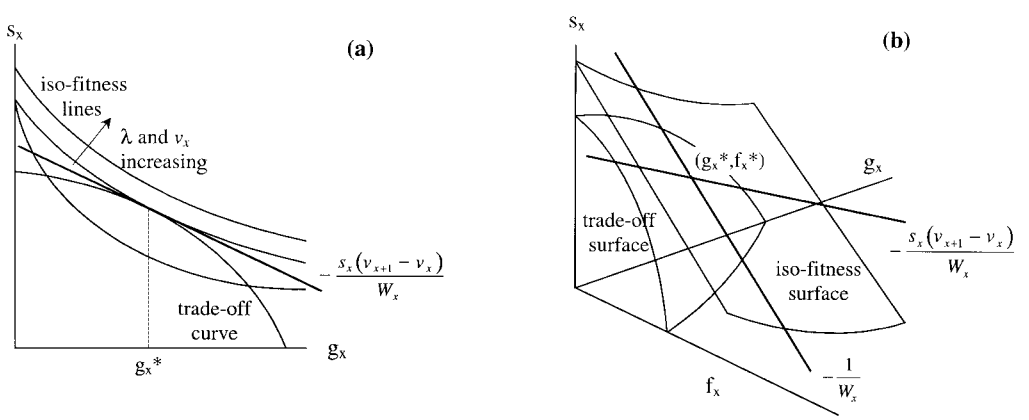

Fig. 1. Graphical representation of optimization model. Tangent trade-off and iso-fitness curves in (a), and surfaces in (b), define optimum.

Avoidable and unavoidable mortality factors

Unavoidable mortality factors affect all phenotypes with equal intensity. Avoidance refers to phenotypes being less affected if more energy is devoted to protection (León, 1983, 1988). The survivorship specific to stage k is expressed in terms of these as:

$$s_k(g_k, f_k; \mu_k, m_k) = \gamma_k(\mu_k) \cdot \sigma_k(g_k, f_k; m_k) \tag{13}$$

γ_k and σ_k are survivorship of individuals in stage k for unavoidable and avoidable mortality, respectively. γ_k is the same for all individuals of size k, whereas σ_k is determined by the phenotype; it is a concave decreasing function of g_k and f_k. The μ_k and m_k parameters measure the intensities of the unavoidable and avoidable factors, respectively.

In previous studies in which mortality factors were distinguished in this way (Hernandez and León 1995; León and De Nóbrega, in press), it was established that some results depend on the degree of avoidance. An unavoidable mortality factor can range from 'easy' to 'hard-to-avoid'. This is determined by the mixed partial derivatives $(\partial^2\sigma_k/\partial m_k \partial g_k)$ and $(\partial^2\sigma_k/\partial m_k \partial f_k)$. These measure second-order effects of how changes in the intensity of the mortality factor m_k affect $(\partial\sigma_k/\partial g_k)$ or $(\partial\sigma_k/\partial f_k)$; that is, how it affects the sensitivity of survivorship to phenotypic values. Note that $(\partial\sigma_k/\partial g_k)$ and $(\partial\sigma_k/\partial f_k)$ also represent the trade-offs defined above but at a level of the avoidable mortality factors only, so these are negative. Hence, a negative mixed partial derivative means that, as m_k increases, the absolute value of $(\partial\sigma_k/\partial g_k)$ – or $(\partial\sigma_k/\partial f_k)$ – also increases (is more negative). This is the 'easy-to-avoid' situation, as the avoidable quality is enhanced by higher intensities. Accordingly, a positive mixed partial derivative proposes decreases in the trade-off values as m_k increases. This corresponds to the 'hard-to-avoid' case, suggesting that, at higher intensities, the avoidance character of the mortality factor is weak, so that conceptually it becomes unavoidable.

Perturbation analysis

We wish to evaluate the direction of change of the optimum phenotype when the intensity of a mortality factor specific to size k increases. Thus, we need to calculate expressions for $(\mathrm{d}g_x^*/\mathrm{d}M_k)$ and $(\mathrm{d}f_x^*/\mathrm{d}M_k)$, where $M_k = \mu_k, m_k$. The signs of these provide information on whether optimal g_x and f_x are expected to increase or decrease when the intensities of the hostilities change. We wish to obtain solutions for both juvenile and adult stages, and for both unavoidable and avoidable mortality factors.

We start by differentiating implicitly equations (7a) and (7b). We get:

$$\frac{\mathrm{d}g_x^*}{\mathrm{d}M_k} = -\left(\frac{\partial^2\lambda}{\partial M_k \partial g_x}\right) \Big/ \left(\frac{\partial^2\lambda}{\partial {g_x}^2}\right) \tag{14a}$$

and

$$\frac{\mathrm{d}f_x^*}{\mathrm{d}M_k} = -\left(\frac{\partial^2\lambda}{\partial M_k \partial f_x}\right) \Big/ \left(\frac{\partial^2\lambda}{\partial {f_x}^2}\right) \tag{14b}$$

We must make clear that, throughout the perturbation analysis, we consider that $v_{x+1} > v_x$ always. As discussed above, this is a condition for a size-classification of structured populations.

For clarification, the final computed expressions for equations (14a,b) are displayed in Table 1 as formal results of the model. However, in the remainder of this subsection, we present a step-by-step derivation of these expressions. This provides insight about the mechanisms involved. In a following subsection, the signs of these results are inspected.

Taking the appropriate derivatives from equations (7a) and (7b), we obtain expressions for the denominator and the numerator in (14a,b) separately. First, from equation (9b), we work out that $\partial v_x/\partial g_z = 0$, for all z, when evaluated at the optimum. Using this result, we find that the second derivatives in the denominators are always negative:

$$\frac{\partial^2 \lambda}{\partial {g_x}^2} = 2(v_{x+1} - v_x)\frac{\partial s_x}{\partial g_x} + W_x \frac{\partial^2 s_x}{\partial {g_x}^2} \tag{15a}$$

and

$$\frac{\partial^2 \lambda}{\partial {f_x}^2} = W_x \frac{\partial^2 s_x}{\partial {f_x}^2} \tag{15b}$$

The definite signs in equations (14a,b) are therefore the signs of the mixed partials at the numerators. For these, we obtain:

$$\frac{\partial^2 \lambda}{\partial M_k \partial g_x} = s_x \frac{\partial (v_{x+1} - v_x)}{\partial M_k} + \frac{\partial W_x}{\partial M_k}\left(\frac{\partial s_x}{\partial g_x}\right) + (v_{k+1} - v_k)\left(\frac{\partial s_k}{\partial M_k}\right)\underset{x=k}{\delta} + W_k\left(\frac{\partial^2 s_k}{\partial M_k \partial g_k}\right)\underset{x=k}{\delta} \tag{16a}$$

$$\frac{\partial^2 \lambda}{\partial M_k \partial f_x} = \frac{\partial W_x}{\partial M_k}\left(\frac{\partial s_x}{\partial f_x}\right) + W_k\left(\frac{\partial^2 s_k}{\partial M_k \partial f_k}\right)\underset{x=k}{\delta} \tag{16b}$$

where

$$\underset{x=j}{\delta} = \begin{cases} 1, & x = j \\ 0, & x \neq j \end{cases} \tag{17}$$

Observe that these expressions provide differential effects of the perturbation ($\partial/\partial M_k$) on elements that affect all stages – via $(v_{x+1} - v_x)$ and W_x – the first two terms in equation (16a), first in equation (16b); and on those that act only upon the perturbed stage – via s_k and trade-off curves ($\partial s_k/\partial g_k$) and ($\partial s_x/\partial f_x$) – the last two terms in equation (16a), last in equation (16b). The latter exploit the fact that changes in M_k act upon s_k only; all other s_x are not affected (see equation 13).

Now, observe that $(v_{x+1} - v_x)$ and W_x are both expressions involving reproductive values of consecutive stages. From equation (9b), we can compute the partial effect of the perturbation on a stage-specific reproductive value as:

$$\frac{\partial v_x}{\partial M_k} = -\psi_k\left(\frac{\partial s_k}{\partial M_k}\right)\frac{v_x}{R_x}\left(\frac{\beta_x}{\beta_1} - \underset{x \leq k}{\delta}\right) \tag{18}$$

where ψ_k, R_x and β_x distinguish between juvenile (J) and adult (A) stages:

$$\psi_k^{(\mathrm{J})} = \frac{\lambda \alpha_k}{s_k} \qquad R_x^{(\mathrm{J})} = 1 \qquad \beta_x^{(\mathrm{J})} = \sum_{j=x}^{n} \alpha_j \tag{19a}$$

$$\psi_k^{(\mathrm{A})} = \frac{R_{k+1} W_k}{s_k g_k v_{k+1}} \qquad R_x^{(\mathrm{A})} = \sum_{j=x}^{n} f_j \alpha_j \prod_{i=1}^{j-1} s_i g_i \alpha_i \qquad \beta_x^{(\mathrm{A})} = \sum_{j=x}^{n} f_j \alpha_j \prod_{i=1}^{j-1} s_i g_i \alpha_i \sum_{k=x}^{j} \alpha_k \tag{19b}$$

Table 1. Equations for the change of optimum according to the type of mortality factor perturbed

Unavoidable

$$\frac{\mathrm{d}g_x^*}{\mathrm{d}\mu_k} = \frac{\left(\frac{\partial\gamma_k}{\partial\mu_k}\right)\psi_k\left[\left(g_x\frac{\partial s_x}{\partial g_x}+s_x\right)\frac{v_{x+1}}{R_{x+1}}\left(\frac{\beta_{x+1}}{\beta_1}-\underset{x<k}{\delta}-\frac{R_{k+1}}{\psi_k s_k}\underset{x=k}{\delta}\right)+\left((1-g_x)\frac{\partial s_x}{\partial g_x}-s_x\right)\frac{v_x}{R_x}\left(\frac{\beta_x}{\beta_1}-\underset{x\le k}{\delta}-\frac{R_k}{\psi_k s_k}\underset{x=k}{\delta}\right)\right]}{\frac{\gamma_x}{s_k}\left[2(v_{x+1}-v_x)\frac{\partial s_x}{\partial g_x}+W_x\frac{\partial^2 s_x}{\partial g_x^2}\right]} \qquad (\mathrm{G_U})$$

$$\frac{\mathrm{d}f_x^*}{\mathrm{d}\mu_k} = -\frac{s_k\psi_k}{\gamma_k s_x\psi_x}\left(\frac{\partial\gamma_k}{\partial\mu_k}\right)\left(\frac{\partial s_x}{\partial f_x}\right)\left(\frac{\partial^2 s_x}{\partial f_x^2}\right)^{-1}\left[\left(\frac{\beta_{x+1}}{\beta_1}-\underset{x<k}{\delta}+\underset{x=k}{\delta}\right)-s_x(1-g_x)\,\alpha_x\left(\frac{\beta_x}{\beta_1}-\underset{x\le k}{\delta}\right)\right] \qquad (\mathrm{F_U})$$

Avoidable

$$\frac{\mathrm{d}g_x^*}{\mathrm{d}m_k} = \frac{\psi_k\left(\frac{\partial\sigma_k}{\partial m_k}\right)\left[\left(g_x\frac{\partial s_x}{\partial g_x}+s_x\right)\frac{v_{x+1}}{R_{x+1}}\left(\frac{\beta_{x+1}}{\beta_1}-\underset{x<k}{\delta}\right)+\left((1-g_x)\frac{\partial s_x}{\partial g_x}-s_x\right)\frac{v_x}{R_x}\left(\frac{\beta_x}{\beta_1}-\underset{x\le k}{\delta}\right)\right]-\left\{(v_{k+1}-v_k)\left(\frac{\partial\sigma_k}{\partial m_k}\right)+W_k\frac{\partial^2\sigma_k}{\partial m_k\partial g_k}\right\}\underset{x=k}{\delta}}{\frac{\sigma_x}{s_k}\left[2(v_{x+1}-v_x)\frac{\partial s_x}{\partial g_x}+W_x\frac{\partial^2 s_x}{\partial g_x{}^2}\right]} \qquad (\mathrm{G_A})$$

$$\frac{\mathrm{d}f_x^*}{\mathrm{d}m_k} = -\frac{s_k}{\sigma_k}\left(\frac{\partial^2 s_x}{\partial f_x^2}\right)^{-1}\left\{\frac{\psi_k}{\psi_x s_x}\left(\frac{\partial\sigma_k}{\partial m_k}\right)\left(\frac{\partial s_x}{\partial f_x}\right)\left[\left(\frac{\beta_{x+1}}{\beta_1}-\underset{x<k}{\delta}\right)-s_x(1-g_x)\alpha_x\left(\frac{\beta_x}{\beta_1}-\underset{x\le k}{\delta}\right)\right]+\frac{\partial^2\sigma_k}{\partial m_k\partial f_k}\underset{x=k}{\delta}\right\} \qquad (\mathrm{F_A})$$

$\underset{x\le j}{\delta}$, ψ_k, R_x, β_x as defined in text; see equations (17) and (19).

Note also that the following relationships hold (these will be useful later):

$$\beta_x = \alpha_x R_x + \beta_{x+1} \qquad \frac{v_x}{R_x} = s_x g_x \alpha_x \frac{v_{x+1}}{R_{x+1}} \qquad R_x \equiv v_x \prod_{i=1}^{x-1} s_i g_i \alpha_i \qquad \beta_x \equiv \sum_{i=x}^{n} \alpha_i R_i \tag{20}$$

After some algebra, the effects of the perturbation involving all stages are given by:
(I) first two terms in equation (16a):

$$-\psi_k \left(\frac{\partial s_k}{\partial M_k}\right)\left\{\left(g_x \frac{\partial s_x}{\partial g_x} + s_x\right)\frac{v_{x+1}}{R_{x+1}}\left(\frac{\beta_{x+1}}{\beta_1} - \underset{x<k}{\delta}\right) + \left[(1-g_x)\frac{\partial s_x}{\partial g_x} - s_x\right]\frac{v_x}{R_x}\left(\frac{\beta_x}{\beta_1} - \underset{x\le k}{\delta}\right)\right\} \tag{21a}$$

(II) first term in equation (16b) – adults only:

$$-\psi_k \left(\frac{\partial s_k}{\partial M_k}\right)\frac{v_{x+1} g_x}{R_{x+1}}\left(\frac{\partial s_x}{\partial f_x}\right)\left[s_x(1-g_x)\alpha_x\left(\frac{\beta_x}{\beta_1} - \underset{x\le k}{\delta}\right) - \left(\frac{\beta_{x+1}}{\beta_1} - \underset{x<k}{\delta}\right)\right] \tag{21b}$$

The significance of these intermediate results is that they give the definite signs for equations (14) for stages $x \neq k$. For the perturbed stage $x = k$, all other terms in equations (16) must also be considered.

Insert equations (21a,b) into (16a,b) to complete expressions for the numerators in equations (14). With these and equations (15), we obtain general equations for (dg_x^*/dM_k) and (df_x^*/dM_k), for both juvenile and adult stages. One of the main objectives of this work is to distinguish evolutionary outcomes for perturbations to avoidable and unavoidable mortality factors; thus, equations are computed for $M_k \equiv \mu_k$ and $M_k \equiv m_k$. Using equation (13), we get:

$$\frac{\partial s_k}{\partial \mu_k} = \frac{s_k}{\gamma_k}\frac{\partial \gamma_k}{\partial \mu_k} \qquad \frac{\partial^2 s_k}{\partial \mu_k \partial g_k} = \frac{1}{\gamma_k}\frac{\partial s_k}{\partial g_k}\frac{\partial \gamma_k}{\partial \mu_k} \qquad \frac{\partial^2 s_k}{\partial \mu_k \partial f_k} = \frac{1}{\gamma_k}\frac{\partial s_k}{\partial f_k}\frac{\partial \gamma_k}{\partial \mu_k} \tag{22a}$$

$$\frac{\partial s_k}{\partial m_k} = \frac{s_k}{\sigma_k}\frac{\partial \sigma_k}{\partial m_k} \qquad \frac{\partial^2 s_k}{\partial m_k \partial g_k} = \frac{s_k}{\sigma_k}\frac{\partial^2 \sigma_k}{\partial m_k \partial g_k} \qquad \frac{\partial^2 s_k}{\partial m_k \partial f_k} = \frac{s_k}{\sigma_k}\frac{\partial^2 \sigma_k}{\partial m_k \partial f_k} \tag{22b}$$

These provide the last step that leads to the final expressions presented in Table 1.

Inspection of signs in the results

Positive signs for equations G_i or F_i in Table 1 imply that the predicted strategy after perturbation is to increase the amount of energy allocated to growth g_x^*, or to fecundity, f_x^*, respectively. The inverse occurs for negative G_i or F_i.

In the equations presented in Table 1, none of the signs are conclusive. However, inspection of its elements provides relevant information about the leading strategies expected after perturbation. In the first place, we observe that the avoidable or unavoidable character of the mortality factor perturbed does not affect the results for the non-perturbed stages (i.e. for $x \neq k$, equations $G_U \equiv G_A$ and $F_U \equiv F_A$). Thus, we will inspect unperturbed and perturbed stages in turn. First, we examine only the signs of the equations; the biological interpretation will follow.

Signs in equations for unperturbed stages ($x \neq k$)

For simplicity, we use $G \equiv G_U \equiv G_A$ and $F \equiv F_U \equiv F_A$ for stages $x \neq k$. Because $v_{x+1} > v_x$, $\beta_1 > \beta_x > \beta_{x+1}$, and all partial derivatives in the equations are negative, we conclude that the terms inside the square brackets in the numerators decide the signs. These, in turn, ultimately depend on the relative magnitudes of the basic vital rates: s_x, g_x and f_x.

We can infer some tendencies by analysing the general behaviour of the elements inside the square brackets under the light of extreme scenarios of energy allocation before perturbation. As will become clear later, these terms are very sensitive to changes in the distribution of energy between s_x and g_x; in particular, to the magnitudes of the trade-off of $|\partial s_x/\partial g_x|$ and α_x. Thus, consider optimal phenotypes with energy allocations biased towards g_x to the detriment of s_x (I) and vice versa (II), for any level of f_x. We expect the magnitude $|\partial s_x/\partial g_x|$ to be higher under scenario (I) and lower under scenario (II) (see Fig. 2). The behaviour of α_x is the opposite (see equation 11): it is smaller the higher g_x and the lower s_x are, as in (I). Note also that if λ is high enough (specifically ≥ 2), α_x is always a fraction; however, if λ is of the order of 1, it can take high integer values for high s_x and low g_x, as could be the case in scenario (II).

Under scenario (I), the phenotype is in the range of higher g_x and lower s_x, and $|\partial s_x/\partial g_x|$ is high. This promotes a negative $[g_x(\partial s_x/\partial g_x) + s_x]$; G is then definitely > 0 for $x < k$, and < 0 for $x > k$. However, when $|\partial s_x/\partial g_x|$ is low – plus lower g_x and higher s_x (scenario II) – the term $[g_x(\partial s_x/\partial g_x) + s_x]$ goes positive and hence the responses might be reversed. The sign of the whole term in square brackets in G depends on the relative values of the two components inside. Comparing the relative contributions of all counterpart elements on both sides, we conclude that $G < 0$ for $x < k$ as long as $|\partial s_x/\partial g_x|$ is low, which is the case; and $G > 0$ for $x > k$, unless $v_{x+1} \gg v_x$.

On the other hand, the term in square brackets in F is strongly influenced by the magnitude of $s_x(1 - g_x)\alpha_x$. All three components within this product change in a concordant manner. Thus, it is very low in scenario (I), which definitely favours a negative F for $x < k$. For $x > k$, we expect F to be positive, since $\beta_x \approx \beta_{x+1}$ for a small α_x (see equations 20). Higher $s_x(1 - g_x)\alpha_x$ values, as in (II), definitely favour a negative F for $x > k$; which is reinforced by a large f_x (it makes $\beta_x \gg \beta_{x+1}$). For $x < k$, we expect a positive F, unless λ is very high, in which case it is also negative.

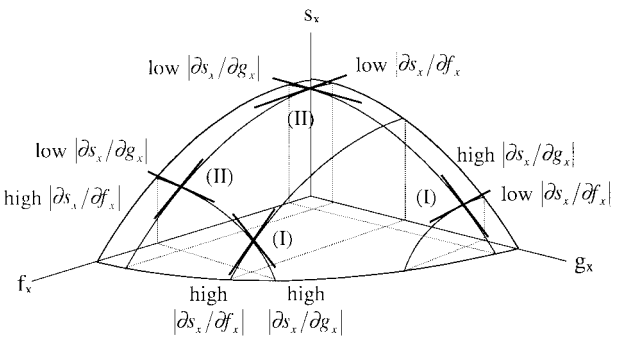

Fig. 2. Regions on the trade-off surface $s_x(g_x, f_x)$ showing 'scenarios': allocation of energy biased towards g_x (I) or towards s_x (II) for two distinct levels of fecundity. Relative magnitudes of slopes $|\partial s_x/\partial g_x|$ and $|\partial s_x/\partial f_x|$ change with f_x.

Table 2 presents a summary of the main tendencies analysed here. To emphasize the wider range of possibilities, we show predominantly the extreme responses.

Ultimate strategies include s_x* behaviour, too. For juvenile stages, this is unequivocal: any alteration in g_x* definitely implies an opposite change in s_x*. Yet, in the case of adults, the variation in s_x* depends on the directions of change of both g_x* and f_x*: whenever these two vary in the same direction, the outcome for s_x* is obviously the opposite; however, if g_x* and f_x* change in opposite directions, s_x*could formally be driven either way. In this case, the sensitivities of s_x to changes in the independent variables compete. And these are measured by the slopes of the partial trade-offs $|\partial s_x/\partial g_x|$ and $|\partial s_x/\partial f_x|$. In other words, due to the concavity of the relationships, we expect changes in s_x* to be mainly driven by – and to be opposite to – the vital function with the highest assignation of resources. Figure 2 shows how for either high or low $|\partial s_x/\partial g_x|$, the slope $|\partial s_x/\partial f_x|$ becomes higher with f_x. Thus, for high f_x ranges, we expect s_x* variations to go opposite to f_x* changes; this means that, in this case, s_x* and g_x* vary concurrently. Different configurations may be thought of, and in fact the actual outcome depends on how each slope changes with respect to the other variable; that is, we would need to have some information on $(\partial^2 s_x/\partial f_x \partial g_x)$.

Signs in equations for the perturbed stage ($x = k$)

Unlike other stages, the signs of equations G and F for the perturbed stage do differ according to the mortality factor concerned.

We analyse the unavoidable case first, that is, G_U and F_U in Table 1. The term $(R_{k+1}/\psi_k s_k)$ in G_U is equal to $(1/\lambda\alpha_k)$ for juveniles, and to $(g_k v_{k+1}/W_k)$ for adult stages (see equations 19 and 20). Under scenario (I), both the $[g_k(\partial s_k/\partial g_k) + s_k]$ and $[(\beta_{k+1}/\beta_1) - (R_{k+1}/\psi_k s_k)]$ terms in G_U are negative; under scenario (II), they are both positive. In all cases, this makes for a positive G_U. Note that the magnitude of the ratio (β_{k+1}/β_1) has no significant influence: it is a fraction, closer to 1 if the perturbation occurs at an early stage ($\beta_{k+1} \approx \beta_1$), but closer to 0 if it is at a later stage ($\beta_{k+1} \ll \beta_1$). If its magnitude is comparable to that of $(R_{k+1}/\psi_k s_k)$, then the whole of the left-hand term inside the square brackets in G_U is negligible. Conversely, if the magnitudes of both ratios are opposite (any combination), the outcome supports the conclusion already drawn of a positive G_U.

Table 2. Main tendencies under extreme scenarios

		Changes favoured (↑ higher or ↓ lower g_x* or f_x*)			
			$x = k$		
		$x < k$	Unavoidable	Avoidable	$x > k$
Scenario I	g_x*	↑	↑	↓	↓
	f_x*	↓	↑	↓	↑
Scenario II	g_x*	↓	↑	↓	↑(b)
	f_x*	↑(a)	↑	↓	↓

Scenarios: (I) g_x* and $|\partial s_x/\partial g_x|$ high, s_x* low; (II) g_x* and $|\partial s_x/\partial g_x|$ low, s_x* high.
Unavoidable: includes 'hard-to-avoid'. *Avoidable*: 'easy-to-avoid' only.
(a) unless $\lambda \gg 2$, (b) unless $v_{x+1} \gg v_x$.

Inspection of equation F_U gives a straightforward positive sign always. Thus, unquestionably, for either a juvenile or an adult stage, under any circumstances, the expected response after an unavoidable perturbation is to increase both g_k* and f_k*, and hence diminish s_k*.

For the avoidable mortality situation – G_A and F_A – the signs and magnitudes of the mixed partial derivatives $(\partial^2\sigma_k/\partial m_k\partial g_k)$ and $(\partial^2\sigma_k/\partial m_k\partial f_k)$ are crucial. As discussed above, these depend on the degree of avoidance of the mortality factor. The 'easy-to-avoid' case contributes a negative term in the equations, whereas the 'hard-to-avoid' contribution is positive. Not surprisingly, the latter coincides with the unavoidable situation.

The fundamental point here is that G_A and F_A can yield opposite responses to those expected for the unavoidable situation; this is possible due to the presence of the negative mixed partials. If they are high enough to counterbalance all other terms, we expect both negative G_A and F_A.

Under scenario (I), a negative G_A is more easily reached because some of the terms already contribute negatively to the outcome, for example, high $|\partial s_k/\partial g_k|$ and $v_{x+1} \gg v_x$. This is not so in the case of scenario (II), where the whole term inside the square brackets in G_A is positive. The latter is also the case with F_A.

A graphical illustration helps to visualize the perturbation analysis performed. As stated previously, optimal configurations are represented graphically by the slopes $(\partial s_x/\partial g_x)$* and $(\partial s_x/\partial f_x)$*; that is, tangent straight lines to the trade-off curves evaluated at the optimum (Fig. 1). The perturbation affects the slopes of all stages, whereas the actual shape of the trade-offs is only affected in the perturbed stage (see equations 16a,b). For unperturbed stages, the slopes 'slide' on the trade-off surface as g_x and f_x vary after perturbation. However, for the perturbed stage, the trade-off surface itself also changes, and therefore the slopes 'slide' and 'jump'. Depending on the avoidable character of the perturbation, the trade-off surface changes in disproportionate ways (Fig. 3). Bear in mind that the 'sliding' is driven by the sign of the term inside the square brackets in the equations, which affects all stages x; and for stage k other terms were considered in addition – these provide the 'jump'. In the unavoidable, or hard-to-avoid, situation (Fig. 3b), both movements make for a decreased slope (more negative) as g_x* or f_x* increases. In the easy-to-avoid case (Fig. 3d), changes in slope when sliding and jumping may be opposite. That is, the positive term in square brackets is counterbalanced by the negative mixed partial, if it is large enough.

The main results are summarized in Table 2. The unavoidable and hard-to-avoid situations have been merged under 'unavoidable', because biologically these can be considered equivalent. The 'avoidable' results refer specifically to the easy-to-avoid situation. In the following discussion, these categories will be considered.

DISCUSSION OF ADAPTIVE STRATEGIES PREDICTED BY THE MODEL

The results of the model indicate that the degree of avoidance of the increased mortality threat determines strategies in the perturbed stages only; opposite responses are expected for the avoidable and unavoidable cases. In unperturbed stages, the responses are mainly determined by how energy was allocated before perturbation, and by its location with respect to the size class perturbed.

For the perturbed size classes, in both juveniles and adults, the strategies are similar. When the increased mortality factor is unavoidable, the response is a compensatory one

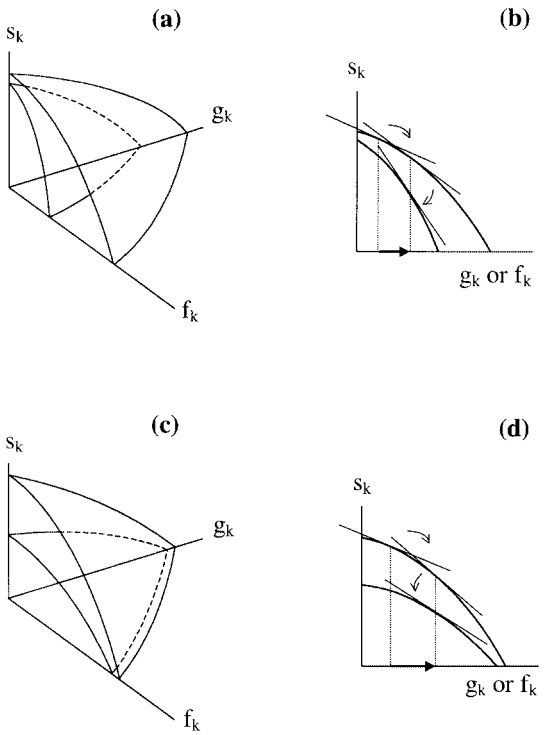

Fig. 3. Graphical representation of perturbation analysis. Change of trade-off surface in stage k due to increased mortality factors: (a) unavoidable or hard-to-avoid, (c) easy-to-avoid; (b) and (d) are sections showing 'sliding' and 'jumping' of slopes in (a) and (c), respectively.

(*sensu* León, 1983, 1988). Dedicating more energy to protection does not render more fitness if it is against an unavoidable threat, whereas an increased growth rate provides the opportunity of attaining a bigger size sooner and moving to the next stage. For adult stages, this means not only growing faster, but also increasing fecundity. Conversely, if the affected mortality factor is avoidable, the strategy is to divert energy towards protection, to the detriment of growth and fecundity. That is, a direct defence against the new threat, slowing down growth and reproduction. Survival is assured even if it has to be at an unhurried and less productive pace.

In stages not directly affected by perturbation, the adaptive responses appear to be governed by the former pattern of energy allocations. Populations of fast growers (scenario I) will adopt a strategy of growing even faster until the affected size is reached. The extra energy required is taken from that devoted to protection and reproduction (if adults). Once the perturbed stage is passed, the strategy reverses: growth rates become slower to allow increased survivorship – and fecundity in adults. Likewise, the strategy of slow growers (scenario II) is to diminish growth even further with a consequent enhanced survivorship and reproductive activity, until the perturbed size stage is attained. The inverse strategy is then adopted.

This shows a clear general pattern of response: any bias in resource allocation among vital functions is accentuated in those size classes smaller than the perturbed one; in contrast, it is counterbalanced in the larger stages. This occurs whether the affected size is pre- or post-reproduction.

A comment about life cycles with 'determinate' and 'indeterminate' adult growth is worthy here. According to the predictions above, we can infer that an increase of mortality threats in late size classes of adults with indeterminate growth provokes a reinforcement of the pattern in earlier stages; that is, even faster growth to the detriment of fecundity. However, consider that the perturbation occurs at an early stage of the adult phase, say the size of first reproduction. Then, the effect is the opposite, and in the long term this could lead to a 'determinate' growth pattern: stop growing to allow increased reproduction. In addition, this trend is strengthened by the fact that, as fecundity increases, the difference between consecutive reproductive values diminishes; this also favours life cycles with determinate growth. Analogous reasoning applies to slow growing adults. Thus, a general mechanism for the evolution of patterns by reinforcement or relaxation of previous ones is foreseen. These occur according to the size-specific perturbations affecting early or late size classes in the adult phase.

Verifying evolutionary predictions with natural situations is not a straightforward task. Circumstantial evidence, then, is examined. Comparing how related species are adapted to different environments, or the distinct ways in which the same species respond via developmental plasticity, can provide us with clues.

A clear indication of evolution of growth rate in response to size-selective predation was reported by Edley and Law (1988) in *Daphnia magna* populations. The aim of their study was to investigate the evolutionary consequences of size-specific culling, particularly important in the management of exploited species. For several generations, two culling patterns were performed on different sets of *Daphnia* populations: removing individuals from the first seven, and from the last five, of the 12 size classes recognized. Their results indicate that, after harvesting large sizes, the selected individuals in early stages show lower growth rates and higher reproductive rates; they also start to reproduce, and die, at smaller sizes. When culling smaller sizes, individuals in early stages grow faster and reproduction is strikingly lower. It is impossible to match these results with the predictions of our model, since the premises are not exactly the same. However, there is a clear pattern of opposite responses when culling is performed at earlier (smaller) or late (larger) size classes, showing a trade-off between growth and reproductive functions. These findings correspond roughly to our results in scenario II.

Barrera and Medialdea (1996) found a positive correlation between development time and resistance to starvation in mosquito larvae. Rapidly growing larvae are associated with high predation and dry environments, but sufficient food resources; their resistance to starvation is very low. The opposite strategy, slow growth, is identified with permanent habitats and low predation. More energy is invested in nutrient storage, which permits the larvae to survive at the expense of slower development.

These results illustrate how, under contrasting environmental pressures, distinct and opposite responses occur, according to how organisms invest in growth and protection. Testing the specific predictions of the model would require the design of experiments (or natural observations) that took into account the distinction between avoidable and unavoidable mortality threats, and the relevance of energy assignments among vital functions.

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REFERENCES

Barrera, R. and Medialdea, V. 1996. Development time and resistance to starvation of mosquito larvae. *J. Nat. Hist.*, **30**: 447–458.

Caswell, H. 1978. A general formula for the sensitivity of population growth rate to changes in life history parameters. *Theor. Pop. Biol.*, **14**: 215–230.

Caswell, H. 1980. On the equivalence of maximizing reproductive value and fitness. *Ecology*, **61**: 19–24.

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

Caswell, H. and Real, L.A. 1987. An approach to the perturbation analysis of optimal life histories. *Ecology*, **68**: 1045–1050.

Charlesworth, B. and León, J.A. 1976. The relation of reproductive effort to age. *Am. Nat.*, **110**: 449–459.

Cole, L.C. 1954. The population consequences of life history phenomena. *Q. Rev. Biol.*, **29**: 103–137.

Edley, M.T. and Law, R. 1988. Evolution of life histories and yields in experimental populations of *Daphnia magna*. *Biol. J. Linn. Soc.*, **34**: 309–326.

Harper, J.L. 1977. *Population Biology of Plants*. London: Academic Press.

Henderson, J.M. and Quandt, R.E. 1971. *Microeconomic Theory: A Mathematical Approach*. New York: McGraw-Hill.

Hernandez, M.J. and León, J.A. 1995. Evolutionary perturbations of optimal life histories. *Evol. Ecol.*, **9**: 478–494.

Kozlowski, J. and Weigert, R.G. 1987. Optimal age and size at maturity in annuals and perennials with determinate growth. *Evol. Ecol.*, **1**: 231–244.

Law, R. 1979. Optimal life histories under age-specific predation. *Am. Nat.*, **114**: 399–417.

Law, R. 1983. A model for the dynamics of a plant population containing individuals classified by age and size. *Ecology*, **64**: 224–230.

León, J.A. 1983. Compensatory strategies of energy investment in uncertain environments. In *Population Biology* (H.E. Freedman and C. Strobeck, eds), Lecture Notes in Biomathematics, Vol. 52, pp. 85–90. Berlin: Springer-Verlag.

León, J.A. 1988. Avoidable mortality in life history theory. In *Mathematical Ecology*, *2nd Research Seminar* (T. Hallam, L. Gross and S. Levin, eds), pp. 85–98. Singapore: World Scientific.

León, J.A. in press. Comparative statics of life history evolution. *Theor. Pop. Biol.*

León, J.A. and De Nóbrega, R. in press. Comparative statics of joint reproductive allocation. *J. Theor. Biol.*

Michod, R.E. 1979. Evolution of life histories in response to age-specific mortality factors. *Am. Nat.*, **113**: 531–550.

Nisbet, R.M. 1997. Delay-differential equations for structured populations. In *Structured Population Models in Marine*, *Terrestrial*, *and Freshwater Systems* (S. Tuljapurkar and H. Caswell, eds), pp. 89–118. New York: Chapman & Hall.

Roff, D.A. 1992. *The Evolution of Life Histories: Theory and Analysis*. New York: Chapman & Hall.

Schaffer, W.M. 1974. Selection of optimal life histories: The effects of age structure. *Ecology*, **55**: 291–303.

Somerton, D.A. and MacIntosh, R.A. 1983. The size at sexual maturity of blue king crab, *Paralithodes platypus*, in Alaska. *Fishery Bull.*, **81**: 621–628.

Stearns, S.C. 1992. *The Evolution of Life Histories*. Oxford: Oxford University Press.

Takada, T. and Caswell, H. 1997. Optimal size at maturity in size-structured populations. *J. Theor. Biol.*, **187**: 81–93.

Templeton, A.R. 1980. The evolution of life histories under pleiotropic constraints and r-selection. *Theor. Pop. Biol.*, **18**: 279–289.

Williams, G.C. 1957. Pleiotropy, natural selection and the evolution of senescence. *Evolution*, **11**: 398–411.

Williams, G.C. 1966. Natural selection, the costs of reproduction and a refinement of Lack's principle. *Am. Nat.*, **100**: 687–690.