Diversity of age-specific reproductive rates may result from ageing and optimal resource allocation

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Abstract

This paper reports the results of a dynamic programming model which optimizes resource allocation to growth, reproduction and repair of somatic damage, based on the disposable soma theory of ageing. Here it is shown that different age-dependent patterns of reproductive rates are products of optimal lifetime strategies of resource partitioning. The array of different reproductive patterns generated by the model includes those in which reproduction begins at the maximum rate at maturity and then declines to the end of life, or increases up to a certain age and then drops. The observed patterns reflect optimal resource allocation shaped by the level of extrinsic mortality. A continuous decline in the reproductive rate from the start of reproduction is associated with high extrinsic mortality, and an early increase in the reproductive rate occurs under low extrinsic mortality. A long-lived organism shows a low reproductive rate early in life, and short-lived organisms start reproduction at the maximum rate.

Introduction

In iteroparous organisms, fecundity may increase with age following reproductive maturity, usually declining at later ages, or it may be maximal at maturity and gradually decrease to the end of life (Roff, 1992). Selection acts on the product of survival and fecundity; thus the age-specificity of reproduction and survival is critical to an understanding of life history evolution (Charlesworth, 1994).

Although the age-specificity of survival rates is well explained by ageing or condition-independent mortality differing among age classes, for example, size- or age-selective predation (Cichoń & Kozłowski, 2000), the age-specificity of reproduction is still not fully explained. The reproductive rate rises with age when more resources are devoted to reproduction with increasing age. This may occur when other resource-demanding activities accompany reproduction (Roff, 1992). In indeterminate growers, the growth rate declines after maturity and the freed resources are directed to reproduction, which increases fecundity (Kitahara *et al.*, 1987; Kozłowski, 1991). However, an increase in fecundity with age is also common in organisms that do not grow after maturity (e.g. Adams, 1985; Forslund & Pärt, 1995). There are three main hypotheses explaining this pattern for determinate growers. According to the so-called selection hypothesis, weaker individuals are constantly eliminated from the population because they have poorer survival by definition (Newton, 1989). As a consequence, at the population level, average fecundity will increase with age, because only high reproductive performers survive to older age classes. The second explanation assumes that the increase in fecundity with age reflects a gradual improvement in the competence of older and more experienced individuals (Clutton-Brock, 1988). Older individuals can obtain better territories, mates and other resources. The third hypothesis explains age-specific reproductive patterns in terms of optimization of reproductive investments: the fecundity of individuals increases with age because life expectancy is decreasing and it no longer pays to save resources for future reproduction (Williams, 1966; Gadgil & Bossert, 1970; Schaffer, 1974; Charlesworth & Léon, 1976). Energy invested in current

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reproduction contributes directly to fitness, whereas the benefits from all other investments are only expected ones which may never be realized if the organism dies.

The above hypotheses may explain an increase in fecundity with age. In many cases, though, fecundity constantly decreases with age or improves up to a certain age and then declines. This decline in fecundity at older ages is typically attributed to ageing. Thus two separate hypotheses have to be invoked to describe lifetime reproductive patterns. Here I suggest a single hypothesis to explain both the increase in fecundity with age in determinate growers and its decrease at older ages. I present a model that generates different reproductive patterns resulting from optimal resource allocations to growth, reproduction and self-maintenance (repair), shaped by the level of environmentally caused mortality. As reproduction competes for resources with growth and repair, reproductive allocations depend on the optimal allocation strategy as well as on the resource acquisition rate, a rate which deteriorates with age as a result of physiological ageing. The ageing rate reflects the level of investment in repair of somatic damage, which otherwise accumulates during life, leading to physiological deterioration of the organism. This assumption is consistent with the disposable soma theory of ageing (e.g. Kirkwood, 1985), which states that ageing is inevitable because the level of repair of somatic damage is lower than that required to repair all damage. In contrast to the other modelling approaches to ageing (e.g. Kirkwood, 1985; Abrams & Ludwig, 1995), the present model allows studying age-specificity of resource allocation. In addition to reproduction-repair trade-off considered in the previous models, here optimal procedure involves growth allocation. Moreover, the shapes of trade-offs between allocation targets are not explicitly defined but result from optimal age-specific strategy. The present model extends previous work on optimal resource allocation and ageing (Cichoń, 1997; Cichoń & Kozłowski, 2000). Here ageing not only affects the survival rate, as in the previous model, but also the resource acquisition rate. Whereas in the previous work I focused on ageing patterns and life history consequences of ageing (Cichoń, 1997; Cichoń & Kozłowski, 2000), the present paper studies consequences of ageing for age-specific reproductive schedules. Here I suggest that diverse curves of age-specific reproductive rates may result from ageing and optimal resource allocation.

Materials and methods

An organism living in a constant environment (no seasons, stable population) acquires resources at size-dependent rate P, being the difference between assimilation and respiration (Sibly & Calow, 1986; Reiss, 1989):

$$P(W) = a\,W^{b} - c\,W^{d} \qquad (1)$$

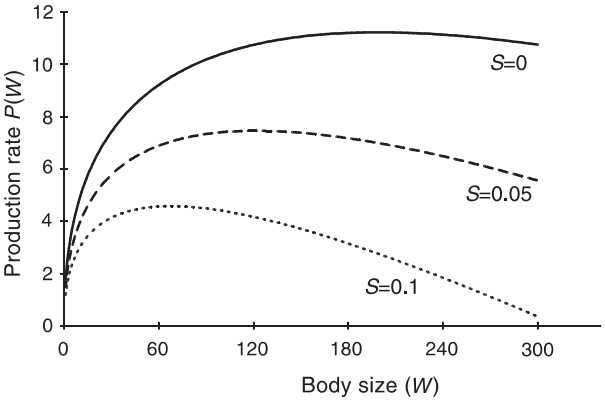

Fig. 1 Production rate is calculated according to the formula $P(W) = ((1-gS) \times 2\ W^{0.5}) - (0.21\ W^{0.83})$, so that the assimilation rate is lowered by damage S. As damage is expressed in mortality units, g is a conversion coefficient, which equals 3 throughout the paper. The lines represent size-dependent production rates for three different sample levels of damage that an organism experiences during its life.

where W is body size in energy units and b, c and d are constants ($b = 0.5$, $c = 0.21$, $d = 0.83$ throughout the paper). Parameter a is not constant as in the previous model (Cichoń, 1997) but is a function of damage, as explained later (see also Fig. 1). The relative values of the parameters are arbitrary, but they do not affect the general pattern so long as the shape of function P remains concave downward. As the units are not specified, the absolute values of the parameters are not of key importance.

At each moment of life the organism allocates excess energy P in age-varying proportions to growth (u), reproduction (v) and repair ($1-u-v$) in a way that maximizes the expected lifetime allocation of energy to reproduction. Allocation to reproduction at each time is $v \times P$, and reproductive output is released at the end of each time unit. The lifetime allocation converted to offspring number is a proper measure of fitness for populations at equilibrium regulated by density-dependence early in life (Mylius & Diekmann, 1995). Under constant offspring size, the expected present and future reproductive allocation at any age is equivalent to the reproductive value, which is in fact maximized in the model. The lifetime optimal allocation was assessed using a dynamic programming technique (Bellman, 1957; McNamara & Houston, 1996). In brief, the dynamic programming is based on the assumption that for an organism of a given age, only future decisions account for maximizing fitness. Thus, to know the optimal decision at a given age it is only necessary to know all optimal decisions from that age until the end of life. An organism which is about to die in the next time unit has no further prospects (its reproductive value equals 0), so at the end of life fitness is determined only by current reproductive

decision, which should in fact maximize reproduction. Moving back in life by one time unit, the future reproductive value and the optimal decision leading to this value is already known, as we calculated it above, and it can be used to find out the optimal decision at that particular age. Proceeding backward from the final to initial age, one can find out the whole lifetime optimal strategy in this way. The procedure requires defining the so-called control variables, which affect the so-called state variables and defining the dynamic of state variables. In the model there are two control variables determining the share of resources invested to growth (u) and reproduction (v), and two state variables, body size and the damage state of an organism. Body size affects production rate (see Eqn 1) and is shaped by growth allocation. Damage affects production rate and chances of survival, but can be removed at the cost of growth and/or reproduction. The state variables change as a function of control variables.

Body size dynamic is described by the formula:

$$\mathrm{d}W/\mathrm{d}x = u(t)\,P\,(W(x)) \qquad (2)$$

solved numerically by the Runga–Kuta method for time x from the interval $t-1$ to t (Press *et al.*, 1986). The term $u(t)$ denotes the fraction of production P directed to growth in the period t, $t + 1$. Thus, body size increases by the amount of energy allocated to growth (u) in every time unit.

Damage affects the mortality rate and assimilation rate in such a way that increasing the amount of damage accumulated increases mortality and decreases assimilation (the first term in Eqn 1). Thus, the damage state can be considered as an ageing state. Damage affects only the component of mortality caused by intrinsic factors such as tissue defects, whereas the organism is also subject to extrinsic, environmentally caused mortality. The model organism incurs damage from the beginning of life; it appears at constant rate μ per time unit ($\mu = 0.001$) and is potentially repairable at some cost. Damage can include any repairable defects appearing in somatic tissues at any level, from a change in a single molecule to the loss of whole organs and structures: DNA damage, protein turnover, oxidative damage caused by free radicals, cell turnover, heavy metal detoxification, etc. (see Kirkwood, 1981 for review). The dynamic of damage is described by the formula:

$$S(t) = S(t-1) + \mu(1-u-v)^n \qquad (3)$$

where $(1-u-v)$ denotes the proportion of production directed to repair. The term $(1-u-v)^n$ reflects the proportion of damage repaired at a given time unit. The effect of repair investments is nonlinear and determined by n, which describes the shape of the relationship between allocation to repair and accumulation of damage and, under $n > 1$ makes low values of allocation more profitable in terms of damage removal. Under $n = 1$, repair is not optimal and never occurs. Throughout the paper, $n = 4$ (see Cichoń, 1997 for more details). According to Eqn (3), the only damage that appears in a given time unit is potentially repairable. This makes ageing inevitable because to avoid ageing, all energy would have to be spent on repair. However this assumption is not crucial for the observed general patterns. Damage affects the production rate such that, in the first term of Eqn (1), describing the assimilation rate, parameter a is a function of accumulated damage ($a = (1-gS) \times \alpha$) where g is a conversion coefficient, as damage S is expressed in mortality units and α is an assimilation constant defining maximum assimilation ($g = 3$ and $\alpha = 2$ throughout the paper; see Fig. 1).

With the dynamic of state variables known, the quantity maximized in the model can be defined. As already mentioned above, the lifetime allocation converted to offspring number is maximized in the model. According to dynamic programming, expected reproductive output (F) satisfies the recursive relation

$$F(W,S,t,T) = \max_{u,v} \varepsilon(S,t)\{R(W,v,t) + F(W,S,t+1,T)\} \qquad (4)$$

where ε denotes the probability of survival from t to $t + 1$, T the final age, R the current reproduction and S the intrinsic mortality caused by accumulation of unrepaired defects. Survivability over one time unit is given by $\varepsilon = \mathrm{e}^{-(S(t)\,+\,m)}$, where m denotes the extrinsic mortality rate. The reproductive value at final age $F(W, S, t, T)$ is equal to 0. According to the backward procedure, future survival and reproduction $F(W,S,t + 1,T)$ is already known at current time unit t.

Equation 4 is solved iteratively from final age T backwards to $t = T - 1$, $t = T - 2$, …, $t = 1$ (Taylor *et al.*, 1974; Goodman, 1982). The allocation strategy (the values of $u(t)$ and $v(t)$) yielding maximal F is considered optimal. With the optimal allocation known, it is possible to reconstruct the lifetime optimal life history proceeding forward, starting from $t = 1$, $W = 1$ and $S = 0$. As the state variables cannot be continuous in the dynamic programming, the optimal allocation for intermediate values of the state variables are approximated by means of linear interpolation (Mangel & Clark, 1988). Maximum lifespan results from the adopted allocation strategy, and is defined as the age at which the probability of survival under a given strategy counted from time unit t to T (proceeding forward) is less than defined 0.001. This assumption does not conform to the classic dynamic programming in which the time horizon is set *a priori* (Bellman, 1957; Mangel & Clark, 1988). When modelling life history strategies final age cannot be known *a priori* as it is usually affected by optimal strategy. In the present model final age depends on mortality, which in turn depends on allocation decisions. This implies that an iterative procedure must be employed, in which backward steps must be followed by forward procedure to

find out when the optimal strategy gives survival probability equal to or less than 0.001, and when the backward procedure should be interrupted. Thus, backward procedure searches for optimal strategy assuming last backward step as the initial stage of life and the forward procedure finds out whether the whole lifespan was already covered by the backward procedure.

Results

In response to different levels of external mortality, the present model generates an array of qualitatively different curves describing age-specific reproduction (Fig. 2). As an optimal strategy, growth does not overlap with reproduction and an initial increase in reproductive rate is because of an age-specific trade-off between reproductive and repair allocations. High extrinsic mortality favours early maturity and high reproductive investments early in life. High reproductive investments are possible because repair is not beneficial when life expectancy is already low for reasons other than damage. Thus, under high mortality, the reproductive allocation is always maximal but fecundity diminishes because of physiological ageing. Low extrinsic mortality makes repair beneficial because the chance of dying as a result of internal sources of mortality (damage) is high, relative to the external sources of it. High investments in repair reduce the share of resources available for reproduction; consequently the reproductive rate is low in early adult life. Some damage, however, accumulates throughout life regardless of repair, making repair less beneficial later in life because of shortening life expectancy. More and more resources thus become available for reproduction later in life, but the reproductive rate starts to decrease after a certain age, although repair stops and all resources become available for reproduction at that time. This is because of physiological deterioration caused by increasing levels of damage. Low reproductive investments early in life are compensated by postponed ageing and prolonged life. Hence, the different curves of age-specific reproduction are coupled with differences in longevity. An initial increase of the reproductive rate is characteristic of long-lived organisms and a constant decline in the reproductive rate characterizes short-lived organisms (Fig. 3).

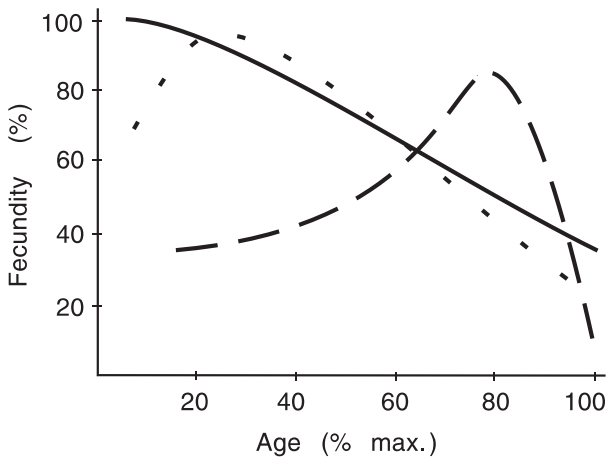

Fig. 2 Patterns of age-specific fecundity under three different extrinsic mortality rates (m). To make the different strategies comparable, both age and fecundity are expressed as percentages of the maximum value for a given strategy. Note that maximum longevity varies greatly between strategies with 395, 155 and 80 time units for low, medium and high extrinsic mortality rates, respectively. (– –) $m = 0.001$; (- - -) $m = 0.01$; (—) $m = 0.05$.

Discussion

The different age-specific patterns of reproduction obtained from the model resemble those observed in nature (e.g. Adams, 1985; Newton, 1989; Rose, 1991; Tatar *et al*., 1993; Forslund & Pärt, 1995; Miyatake, 1997), and here they are products of optimal strategies of resource allocation to growth, reproduction and repair. In fact, repair is a key determinant of age-specific fecundity curves, as growth and reproduction never overlap in an optimal strategy. High investment in repair makes fewer resources available to reproduction, but as an optimal strategy, less and less is spent on repair later in life, and thus reproductive rate increases with age. However, because of physiological ageing, reproductive rate deteriorates in advanced ages. To my knowledge, the model is the first to use one mechanism to explain both the increase and the following decrease in fecundity observed in some organisms. It also suggests that different patterns of fecundity can be related to the level of extrinsic mortality shaping allocation patterns. To some extent, this is corroborated by empirical data showing that organisms living under high mortality pressures tend to have high reproductive outputs. For instance, guppies (*Poecilia reticulata*) experiencing heavy predation allocate higher proportion of their body reserves to reproduction early in life, and despite being smaller, produced more offspring in early litters than their counterparts living under low predation risk (Reznick, 1996). In a very elegant evolutionary experiment Stearns *et al*. (2000) showed that fruit flies (*Drosophila melanogaster*) living under high mortality regime matured earlier, had shorter lifespan and had their peak of fecundity shifted early in life than flies from low mortality regime. This exactly matches the patterns described in the present paper. Some lines of evidence come also from comparative studies. For instance, among mammals, those with high levels of natural mortality, mature earlier and produce larger litters after shorter gestation (Promislow & Harvey, 1990).

The possibility that the increase in fecundity with age results from increasing reproductive allocation has already been considered in theoretical studies (see Roff,

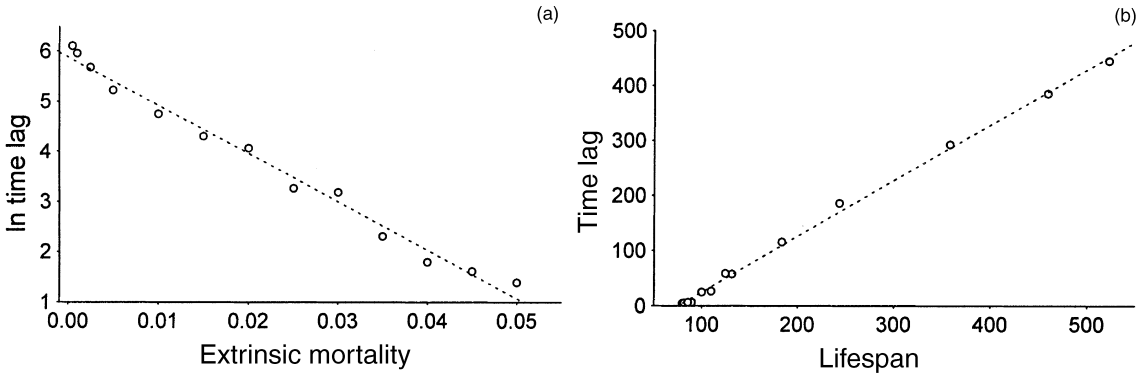

Fig. 3 The length of period in which reproductive rate increases (time lag) vs. external mortality rate (a) and maximum longevity (b). Time lag is a difference between age at maximum reproduction and age at maturity.

1992 for review). Life history theory predicts that more and more resources should be devoted to reproduction with increasing age, as life expectancy and the chances for future reproduction decrease (Schaffer, 1974). To date, theoretical analysis predicts increased fecundity with age as a result of optimal allocation to growth and reproduction – if there is growth after maturity, the growth rate declines with age, freeing resources for reproduction (Roff, 1992; Stearns, 1992). Kitahara *et al.* (1987) analysed allocation to growth and reproduction and obtained a good fit of their model to the data on cod and herring. The reproductive allocation increased with age in a model considering growth and reproduction in perennial plants (Kozłowski & Uchmański, 1987). Kozlowski's (1991) model considering additional allocation into storage fitted the data on growth and reproduction in the Iceland scallop (Vahl, 1981). The present model shows that an increase in the reproductive allocation is also possible in organisms that do not grow after maturity. (Note, however, that cessation of growth after maturity is not assumed in the model but results from optimization of life history strategy.) Here reproduction competes for resources with repair; repair diminishes with age and makes more resources available for reproduction. Repair allocation declines with age because life expectancy and chances for future reproduction decrease, as predicted from general life history theory (Schaffer, 1974).

The present model's assumption that the assimilation rate decreases because of age-related accumulation of damage has inevitable life history consequences. Its results are qualitatively different from those of the model in which only the survival rate was affected by ageing (Cichoń, 1997); now ageing has more severe effects, so it is more profitable to invest in repair. Generally, higher repair slows the accumulation of damage, thus improving survival and prolonging life. Under the same parameter set, the growth rate is also lower in the present model because resources are used for repair. As a result, life is longer, maturity postponed and body size is smaller.

In the present paper, I suggest that the different lifetime reproductive patterns observed in nature may result from optimal resource allocation. This explanation does not exclude other possible mechanisms mentioned in the introduction, and further experimental studies are needed to assess their relative effects. To date, the empirical studies of reproductive patterns have been mainly observational, concentrated mostly on describing the age-specificity of reproduction but not on understanding the underlying mechanisms (see Forslund & Pärt, 1995). Further studies should focus on identifying the factors responsible for shaping age-related differences in reproductive rates and revealing the extent to which resource allocation can explain them. The appropriate experiments are rather difficult to perform. Comparative studies would seem very promising, especially in regard to whether the rates of externally caused mortality can mould the diversity of reproductive patterns and, in consequence, whether long-lived organisms actually increase their reproduction in early adult life more often than do short-lived ones.

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