

Ageing and typical survivorship curves result from optimal resource allocation

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ABSTRACT

Ageing is a general feature of higher organisms. Evolutionary theories of ageing rest on a gradual decline of fitness sensitivity to changes in survival and fecundity with age, so traits expressed late in life are less favoured by natural selection than those expressed early in life. From the life-history perspective, ageing may result from a scheme of optimal resource allocation in which investment in repair is lower than that required for removing all experienced damage. Here, we report the results of a dynamic programming model based on the disposable soma theory of ageing, which optimizes resource allocation to growth, reproduction and repair. The optimal allocation strategy responds to externally imposed mortality, and repair intensity varies with age, being highest early in life, diminishing later and stopping completely well before the end of the maximum expected life. Because the level of repair varies, the rate of ageing is highest under high extrinsic mortality and lowest under low mortality. The allocation strategy shapes the survivorship curve and maximum lifespan. The model results provide an explanation of the variety of survivorship curves and maximum lifespans observed in nature. The results are discussed alongside empirical data from studies using mainly comparative approaches.

Keywords: ageing, disposable soma theory, life history, longevity, mortality, resource allocation, survivorship curves.

INTRODUCTION

Ageing has attracted considerable attention from scientists in different disciplines. It is a process of decline in physiological functioning that results in increasing mortality and decreasing reproduction with age. From the life-history perspective, ageing means restriction of the life-history options open to an organism over time (Partridge and Barton, 1996). Because ageing appears non-adaptive, it is puzzling to evolutionary biologists. The commonly accepted explanation is that ageing occurs because natural selection acts less strongly on traits expressed late in life than on those expressed early in life (Medawar, 1952; Williams, 1957; Hamilton, 1966; Charlesworth, 1994). Two classes of mechanisms are based on this assumption (see Rose, 1991; Partridge and Barton, 1993): (1) the non-adaptive explanation, according to which late-acting deleterious mutations accumulate in

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populations over many generations; and (2) the adaptive explanation, according to which ageing results from optimization of the life history (high reproduction or survival rate early in life decreases performance late in life). The first mechanism is known as the mutation accumulation theory (Medawar, 1952). The second is known as the antagonistic pleiotropy theory (Williams, 1957), because it requires genes that have opposite effects on fitness when expressed early and late in life. Both theories were formulated in a population genetics framework and, as a result, their predictions deal with genetic properties of organisms (Rose, 1991). Although every biological phenomenon requires a genetic background, a phenotypic approach may help elucidate the evolution of ageing, since ageing is a phenotypic feature. Ageing can evolve as a part of optimal life history, and pleiotropy is not strictly necessary (Partridge and Barton, 1996; but see Blarer *et al.*, 1995). Any improvement in performance early in life is highly favoured even if it has negative effects later in life, but we do not need to invoke pleiotropic effects if the age-related changes in the state of an organism determine its performance. In both cases, later improvements are less beneficial because the organism has to survive to the age when they occur to gain anything. Accordingly, deleterious effects cost less if they appear late in life, since there is a good chance that the organism will die earlier for external reasons.

The life-history perspective on ageing is considered in the so-called disposable soma theory (Kirkwood, 1981). According to this theory, ageing results from the optimality of a low level of repair of somatic damage, which, left unrepaired, accumulates during life, leading to gradual deterioration of the organism's physiology. Kirkwood and co-workers (Kirkwood, 1985, 1990; Kirkwood and Rose, 1991) suggest that optimal investment in repair is lower than that required to repair all damage, thus ageing is inevitable. The theory is formulated within the so-called allocation principle: the amount of acquired energy is limited and should support different requirements in a way that maximizes fitness (Kozłowski, 1992; Perrin and Sibly, 1993). For example, energy invested in reproduction contributes directly to fitness, whereas the benefits from investment in soma are expected only in the future. Under unavoidable mortality risk, investment in soma may never be paid back before the organism dies. Clearly, the organism should stop investing in soma when the costs exceed the expected benefits (Perrin and Sibly, 1993). Following the allocation principle, the disposable soma theory assumes that organisms adjust their investment of resources between repair and reproduction in a way that maximizes fitness. The unavoidable hazard of accidental death makes the individual lifespan limited even in the absence of ageing. Thus it is non-optimal to repair soma if the expected life is short. Under high environmental mortality, organisms should concentrate on short-term benefits, investing in the immediate rewards assured by reproduction rather than undertaking risky investments in somatic maintenance.

It is widely believed that the death rate increases approximately exponentially with age in adult organisms (Finch, 1990). Therefore, for simplicity, previous models of the disposable soma theory (Kirkwood, 1985, 1990; Kirkwood and Rose, 1991) considered only the adult period of life, assuming the exponential relationship between mortality and age described by the Gompertz equation (Gompertz, 1825), and determined optimal values of the exponent (β) of this equation. As Abrams and Ludwig (1995) noted, those models contain a logical flaw because an exponential increase in mortality rate was assumed, whereas an increase in mortality should result from the model. Abrams and Ludwig (1995) argued that exponentially increasing death rates are expected to be rare under the disposable soma theory of ageing.

Abrams and Ludwig (1995) were interested in the relationship between repair–reproduction trade-offs and the form of the predicted age-dependence of mortality. They assumed specific functions for the trade-offs and followed the resulting mortality curves. Our approach here is different: we focus on the age-dependence of mortality that results from optimal allocation decisions, and specific trade-off functions are not explicitly assumed but result from optimization. In our model, growth, reproduction and repair compete for the same resources, but the mortality costs of growth and reproduction are indirect, as an increase in allocation to growth and reproduction necessarily decreases allocation to repair, thereby affecting the mortality rate. Recently, Teriokhin (1998) also considered mortality as the result of optimal resource allocation and argued that mortality affects the allocation strategy in that high mortality shifts energy expenditure primarily in favour of reproduction, then in favour of current survival, with repair having the lowest priority. However, only adult life and fixed lifespan were taken into account, and body size was not determined by resource allocation. According to our model, some unrepaired damage can accumulate even in juvenile life. Treating body size as independent of the resource allocation process introduces an additional degree of freedom that does not occur in nature (Kozłowski, 1992; Perrin and Sibly, 1993). Moreover, the assumption of a finite lifespan makes the assessment of allocation strategies impossible (Blarer *et al.*, 1995). In contrast with previous work (Abrams and Ludwig, 1995; Teriokhin, 1998), we base our work on somewhat different assumptions and extend our analysis to allocation patterns and the resulting shapes of survivorship curves and longevity. The only feature in the model that generates age-dependent mortality curves through optimal allocation decisions is extrinsic mortality. In previous work based on the same model (Cichoń, 1997), the focus was on the effect of external mortality – mediated by optimal allocation decisions – on age and size at maturity, growth rate and maximum longevity. Therefore, the present paper complements that work.

Variation in mortality rates is widely accepted to be the best predictor of variation in life-history traits, since mortality imposes the time horizon for optimization of life history (Kozłowski, 1992; Roff, 1992; Stearns, 1992; Charnov, 1993). For instance, mortality affects age and size at maturity, longevity and fecundity-related traits. One of the most important predictions of all evolutionary theories of ageing is that the rate at which the organism's state deteriorates (rate of ageing) depends to a great extent on external factors, such as mortality (Partridge and Barton, 1996). However, only mortality factors that are independent of age and condition should be considered, since a high rate of ageing by definition results in higher mortality rates (Abrams, 1993). Accordingly, we distinguish between age- and condition-independent mortality, referred to as the 'extrinsic mortality', and 'intrinsic mortality' associated with the accumulation of defects leading to an organism's deterioration and ageing.

THE MODEL

We assume that an organism living in a constant environment (no seasons, non-growing population) acquires resources at size-dependent rate P described by the function:

$$P(W) = aW^b - cW^d \quad (1)$$

where W is body size in energy units and a , b , c and d are constants. Thus, rate of production P is the difference between the assimilation and respiration rates (Sibly and Calow, 1986;

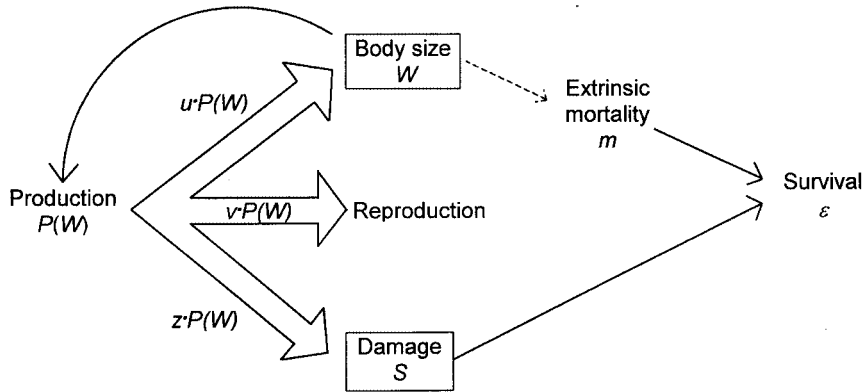


Fig. 1. The allocation model considered in this paper. Wide arrows represent energy flow, whereas thin arrows indicate the relationships between variables and parameters. The energy acquired at size-dependent rate P is distributed in varying proportions to growth (u), reproduction (v) and repair ($z = 1 - u - v$). The allocation pattern affects state variables; W represents body size and S the amount of accumulated defects (shown in boxes). The probability of survival ε results from extrinsic mortality m and from intrinsic mortality S (accumulated damage); in some cases, extrinsic mortality is size-dependent (indicated by broken line).

Reiss, 1989). For all the numerical examples presented here, the constants in the function P are the same: $a = 2.0$, $b = 0.5$, $c = 0.21$, $d = 0.83$. As long as we do not specify units, the relations between, rather than the absolute values of, the parameters are important. Moreover, the relative values of the parameters are not derived from any specific biological circumstances, but they do not affect the general pattern of results so long as the shape of the acquisition function P remains concave downward.

At each moment of life, the organism allocates surplus energy P in age-varying proportions to growth (u), reproduction (v) and repair ($z = 1 - u - v$) in a way that maximizes lifetime allocation of energy to reproduction (Fig. 1). Allocation to reproduction at each instant in time is vP , 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 and Diekmann, 1995). Under constant offspring size (here equal to 1), the present and expected future reproductive allocation at any age are equivalent to the reproductive value, which is indeed maximized in the model. The trajectory analysed to maximize the reproductive value involves only segments of life from a given age until the death of the cohort (Taylor *et al.*, 1974; Goodman, 1982). It allows the optimal life history to be searched for in short segments, starting from the oldest age and working backwards towards the youngest, using the dynamic programming method (Bellman, 1957; McNamara and Houston, 1996).

According to dynamic programming, the expected reproductive output (F) satisfies the recursion relation:

$$F(W, S, t, T) = \max_A \varepsilon(W, S, A, t) \{R(W, A, t) + F(W, S, t+1, T)\} \quad (2)$$

where ε denotes the probability of survival from age t to $t+1$, T is final age, A is the allocation strategy at age t , R is the current reproduction and S is the intrinsic mortality

caused by accumulation of unrepaired defects. Survivability over one time unit is given by $\varepsilon = e^{-(S(t)+m)}$, where m denotes the extrinsic mortality rate (Fig. 1). The expression \max_A means that we choose such an allocation strategy A at any time unit that maximizes $F(W, S, t, T)$. The residual reproductive output at the final age $F(W, S, t, T)$ is equal to zero. According to the backward procedure, residual reproductive output $F(W, S, t+1, T)$ is already known at current time unit t . The intuitive explanation of the above equation is that current reproduction R is determined by the allocation strategy A applied at each moment of life t . A myopic strategy would maximize the current success, but a foreseeable strategy should take into account the prospects of future survival and reproduction.

Extrinsic mortality m is constant (not dependent on body size, condition or age) or is size-dependent. A constant mortality rate may reflect non-selective predator pressure, whereas size-dependent mortality might mean selective predator pressure. In the latter case, mortality decreases with body size W according to the formula $m = eW^{-f}$, where e is initial mortality and f describes how fast mortality decreases with body size. In the cases presented here, e varies from 0.2 to 0.99 and f from 0.7 to 1. The total mortality rate is the sum of the extrinsic and intrinsic mortality rates.

Body size is a state variable and it increases by the amount of energy devoted to growth in every time unit according to the formula:

$$\frac{dW}{dx} = u(t) \cdot P(W(x)) \quad (3)$$

where x denotes time and $u(t)$ is the proportion of production rate P directed to growth in the period $t, t+1$. Differential equation (3) is solved for the time interval from t to $t+1$ numerically using the Runge-Kuta method (Press *et al.*, 1986).

Ageing affects the mortality rate (intrinsic mortality here) but it does not directly affect reproduction (the effect of ageing on reproduction will be discussed elsewhere). Since ageing is defined here in mortality terms, it is crucial to distinguish between two components of the total mortality rate: the extrinsic mortality caused by environmental factors and the intrinsic mortality caused by such intrinsic factors as tissue defects incurred during life. The organism is subject to damage from the beginning of life; the damage appears at a constant rate μ ($= 0.001$ throughout the paper) 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 alteration, oxidative damage caused by free radicals, cell damage, heavy metal effects, and so on (see Kirkwood, 1981, for a review). There are inevitable costs in terms of energy or other resources expended to remove the damage. Repair may include mechanisms like DNA screening and repair, proof-reading of macromolecule synthesis, protein turnover (mopping up abnormal proteins), cell turnover (mopping up and mitotic replacement of dead and dysfunctional cells), removing damage caused by highly reactive metabolites, heavy metal detoxification, removing bacteria or viruses, and so on. Damage is expressed in mortality units in the model. The intrinsic mortality rate S at age t is the second state variable and its dynamic is described by the formula:

$$S(t+1) = S(t) + \mu \cdot (1-z)^n \quad (4)$$

where z denotes the proportion of production directed to repair. The important assumptions underlying this function are as follows: (i) damage occurs at a constant rate

throughout an entire life; (ii) repair effects are non-linear with respect to the fraction of energy allocated to this process in accordance with the law of diminishing returns (a larger n means stronger non-linearity); (iii) only current defects can be repaired, which is a simplifying assumption that does not change the results if defects occur at a constant rate. Parameter n , called ‘efficiency of repair’ here, maps the amount of resources invested in repair into an intrinsic mortality rate. If the effect of defects on mortality is multiplicative rather than cumulative, we can expect $n > 1$. Under a given z , an increase in n enhances removal of damage, especially if z is small. Higher values of n make repair more efficient, since the same fraction of production devoted to repair results in relatively more defects being removed. Thus, high n promotes allocation to repair, slowing down ageing and enhancing longevity (Cichoń, 1997). Throughout this paper, n equals 4, which means a relatively high repair efficiency (Cichoń, 1997). Under $n = 1$ allocation to repair is never optimal, and under $n = 2$ the results are intermediate (Cichoń, 1997). Under intrinsic mortality described by equation (4), only damage that appears in a given time unit is potentially repairable. Thus, ageing must occur in the model, since it would require investing all acquired resources in repair to avoid ageing and such a strategy is inconceivable. It is realistic to assume that not all damage is repairable and that accumulation of some damage is unavoidable. If it is assumed that all damage would be repaired not for $z = 1$ as in equation (4) but for a lower value, such as 0.8, the results (not shown here) are qualitatively similar. This makes sense, since allocation in repair never approaches 0.8 under the parameter set studied here (see next section).

The recursive equation (2) allows construction of an algorithm in which the equation is solved iteratively from final age T backwards to $t = T - 1$, $t = T - 2$, . . . , $t = 1$ (Taylor *et al.*, 1974; Goodman, 1982). The optimal allocation strategy – the values of $u(t)$, $v(t)$ and $z(t)$ – yielding maximal F is searched for through the array of the combinations of state variables W and S . These optimal proportions of energy channelled to growth, reproduction and repair are stored at every time unit for that array, to later reconstruct the trajectory of growth and accumulation of intrinsic mortality proceeding forward, starting from $W = 1$ (offspring size) and $S = 0$ (no damage). Optimal proportions of resources and future reproductive values for state variables other than those stored in the array are estimated with linear interpolation. With this known, survivorship curves can be constructed. The life horizon is not determined beforehand. The model assumes that the backward procedure is interrupted if survival counted from current time unit t to the end of life T is less than 0.001 under optimal allocation, with such t being considered the starting point of life. In other words, maximum lifespan is defined as the age to which the probability of survival under a given strategy is less than 0.001. Thus, maximum lifespan results from the adopted allocation strategy.

RESULTS

The optimal share of available resources devoted to repair changes with an organism’s age. The allocation strategy is shaped by the extrinsic mortality rate (Fig. 2). First growth is favoured and reproduction begins when growth stops, so growth and reproduction do not overlap (age at maturity is indicated by arrows in Fig. 2). Repair, if any, competes for resources with growth and reproduction. The amount of resources allocated to repair is highest early in life; later it decreases slowly or quickly, and repair stops completely well before the end of life. Investment in repair is high and long-lasting when extrinsic mortality

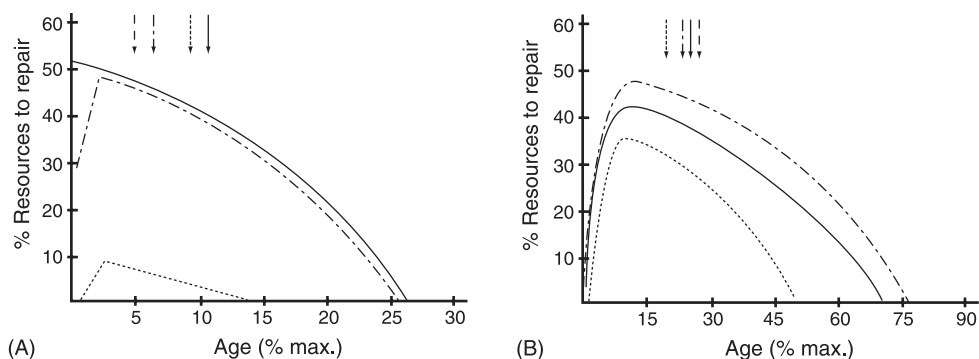


Fig. 2. Optimal share of resources allocated to repair as a function of age under different extrinsic mortality rates for (A) size-independent mortality (—, $m = 0.0001$; - - -, $m = 0.001$; ···, $m = 0.01$; - · - ·, $m = 0.05$) and (B) size-dependent mortality (—, $m = 0.2W^{-0.8}$; - - -, $m = 0.4W^{-1.0}$; ···, $m = 0.99W^{-0.7}$). Note: the last curve (- - -) is not shown because repair was not optimal in this case. The remaining share of resources is optimally allocated to either growth or reproduction, never to both processes simultaneously. Life-history strategies obtained from the model differ with respect of maximum lifespan; thus to make the strategies comparable, age was expressed as a percentage of maximum lifespan. Arrows indicate age at maturity.

is low, but appears not to be optimal when mortality is very high. For size-dependent mortality, there is low allocation to repair early in life (Fig. 2B). This is because the best strategy is to grow quickly to escape the small size range characterized by high mortality.

Intrinsic mortality, which reflects ageing in the model, increases with age at varying rates under different extrinsic mortality rates, either size-independent (Fig. 3A) or size-dependent (Fig. 3B). An increase in the intrinsic mortality rate is mediated by differential investments in repair that directly affect the accumulation of damage. Under the highest studied rate of extrinsic mortality, it is not optimal to invest anything in repair, so that the increase in intrinsic mortality is maximal and simply reflects the accumulation of all damage experienced. The total energy directed to repair increases with relaxing extrinsic mortality pressure, and allocation to repair changes dynamically with age, being maximal early in life (Fig. 2). Thus, low extrinsic mortality rates promote a slow increase in intrinsic mortality early in life due to the removal of defects, and an acceleration of intrinsic mortality in older age when repair ceases. This is especially pronounced under the lowest extrinsic mortality rate studied. The arrows in Fig. 3 denote the age at which repair ceases. It is clear that intrinsic mortality starts to increase quickly when repair is abandoned: repair of damage allows the organism to maintain good shape early in life, and it is optimal to lower the fraction of available resources channelled to repair later and to cease repair at some advanced age in favour of reproduction.

The dynamics of the intrinsic mortality rate are qualitatively similar under size-independent and size-dependent extrinsic mortality (compare Figs 3A and 3B). However, in the case of size-dependent mortality, there is a fast initial increase in intrinsic mortality due to a low level of repair when the growth rate is maximized to escape the risky body size range. Although intrinsic mortality increases slowly in the middle part of life, it is elevated early in life. The curves of total mortality (extrinsic plus intrinsic) differ much more between size-independent (Fig. 3C) and size-dependent (Fig. 3D) mortality. For size-independent

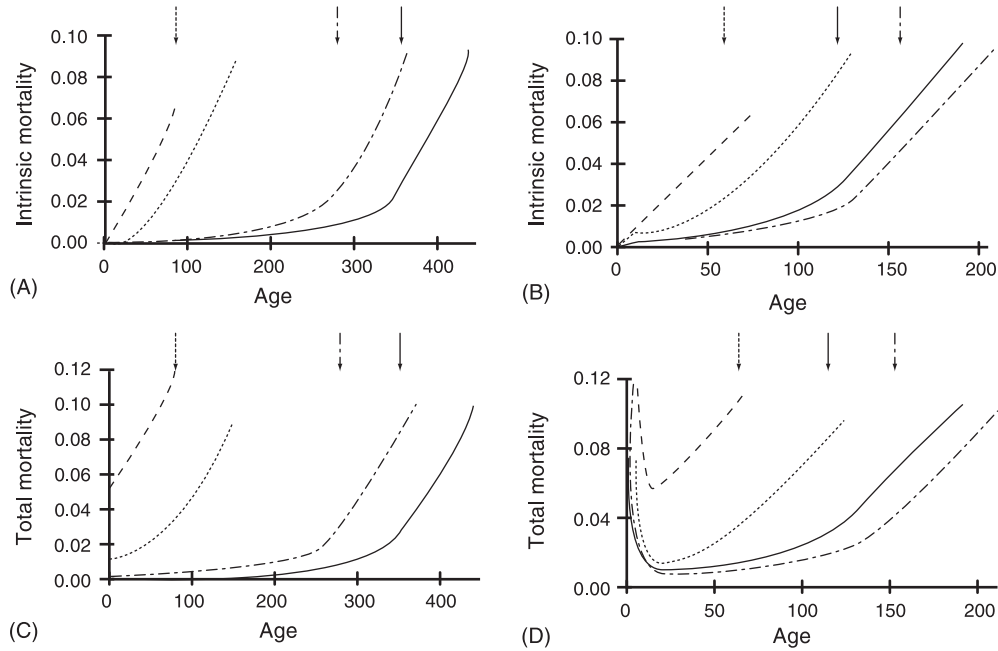


Fig. 3. Age-dependence of intrinsic mortality rate reflecting ageing (A, B) and age-dependence of the total mortality rate (C, D) for different levels of extrinsic mortality, constant during life (A, C) or size-dependent according to allometric equation, $m = eW^{-f}$ (B, D). The curves are for the optimal allocation of resources to repair, growth and reproduction. Arrows indicate the age at which repair stops completely. Extrinsic mortality: —, $m = 0.0001$; — · —, $m = 0.001$; ···, $m = 0.01$; - - -, $m = 0.05$ for constant mortality (A, B); —, $m = 0.2W^{-0.8}$, — · —, $m = 0.4W^{-1.0}$; ···, $m = 0.9W^{-1.0}$; - - -, $m = 0.99W^{-0.7}$ for size-dependent mortality (C, D).

extrinsic mortality, the total mortality rate curves are similar in shape to those for intrinsic mortality, but they are only shifted upward. Under extrinsic mortality decreasing with body size, the total mortality rate decreases sharply at the beginning of life because of fast initial growth due to a low level of repair, and starts to increase later as a result of ageing (acceleration of the intrinsic mortality rate).

The probability of surviving to a given age results from the mortality rate dynamic. Optimization of allocation to repair makes the model generate different mortality patterns. Investments in repair (dependent on extrinsic mortality) shape intrinsic mortality, which together with extrinsic mortality determines the survival rate. Thus it is possible to construct survivorship curves for the different extrinsic mortality rates (Fig. 4). In general, under a high extrinsic mortality rate, when repair is absent or very low, the survival rate declines with age; under a low extrinsic mortality rate, when high repair rate is favoured, the survival rate declines very slowly early in life but rapidly so at later ages. Size-independent mortality rates produce an array of different shapes of survivorship curves, from linear to concave downward on a logarithmic scale (Fig. 4A). Mortality rates decreasing with size generate survivorship curves with an inflection point, concave upward at the beginning and concave downward later (Fig. 4B).

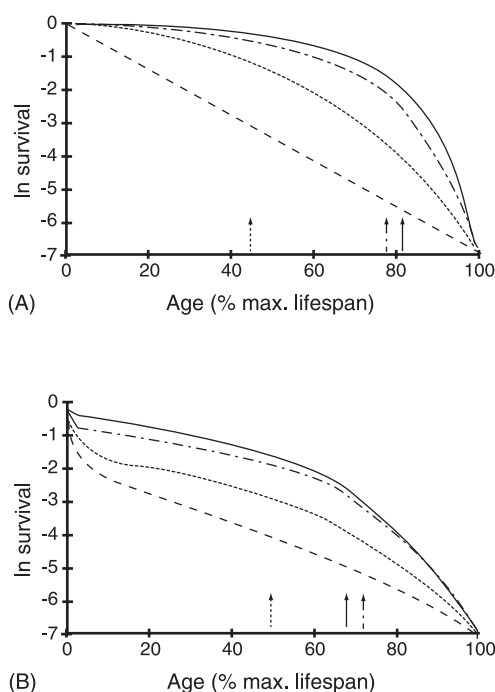


Fig. 4. Survivorship curves of optimal resource allocation to repair under different levels of extrinsic mortality, constant during life (A) or size-dependent according to the equation eW^{-f} (B). Arrows indicate the age at which repair stops completely; there is no allocation to repair for the highest mortality rates in (A) and (B). To make the survivorship curves comparable, age is expressed as a percentage of maximum lifespan. Extrinsic mortality: —, $m = 0.0001$; — · —, $m = 0.001$; ···, $m = 0.01$; — — —, $m = 0.05$ (A); —, $m = 0.2W^{-0.8}$; — · —, $m = 0.4W^{-1.0}$; ···, $m = 0.9W^{-1.0}$; — — —, $m = 0.99W^{-0.7}$ (B).

Because in the present model the final age T (maximum lifespan) is not fixed but is defined as the age to which survival probability equals 0.001, it is possible to study variation in maximum lifespan generated by optimal resource allocation under different mortality regimes. Maximum lifespan varies greatly under different levels of extrinsic mortality (Fig. 5, shaded bars), whereas without repair (hence at the maximum ageing) the effect of extrinsic mortality is not very pronounced (Fig. 5, unshaded bars). Although extrinsic mortality affects maximum lifespan directly, its indirect effect through optimal allocation strategies is much stronger. Both the direct and indirect effects of extrinsic mortality change maximum lifespan in the same direction. For low extrinsic mortality, repair extends life significantly (compare bars in Fig. 5).

DISCUSSION

We have shown allocation patterns to be strongly age-dependent: optimal investment in repair was highest early in life and decreased later, stopping completely before the end of life, if repair was optimal at all. This is because the return on investment in repair of somatic damage diminishes as an organism ages. Age-specificity of investments in repair has

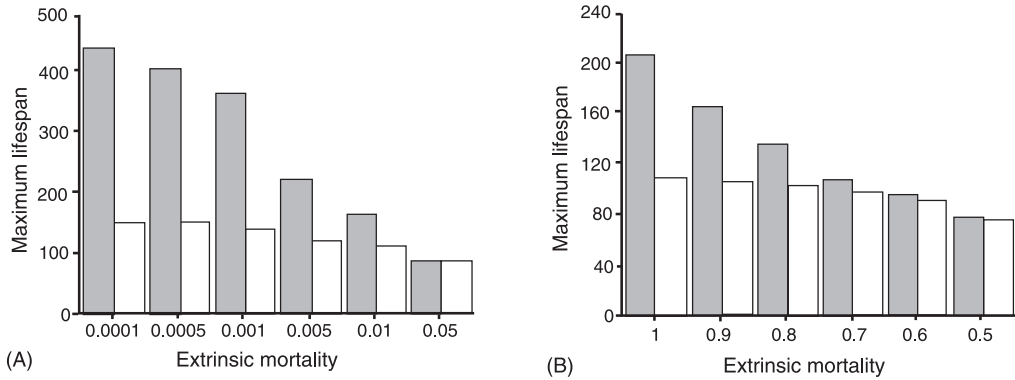


Fig. 5. Maximum lifespan under different extrinsic mortality rates, size-independent (A) or size-dependent according to the equation $m = eW^{-f}$ (B; extrinsic mortality presented on the x-axis reports different f values, while parameter $e = 0.4$ in all cases). The unshaded bars represent maximum lifespans without allocation to repair, and the shaded bars maximum lifespans resulting from optimal allocation to repair. Under constant mortality (A), the extrinsic mortality rate indirectly diversifies the maximum lifespan only slightly in comparison to the case in which ageing was postponed due to repair; note also that, at $m = 0.05$, repair proved not to be optimal and both bars are equal. Under size-dependent mortality (B), the differences between the unshaded and shaded bars are less pronounced; this is because fast growth at the cost of repair is optimal early in life, which causes accumulation of defects and results in a shorter lifespan.

obvious consequences for ageing patterns. The rate of ageing, defined here as an increase in the intrinsic mortality rate, is directly affected by repair, since damage that is not removed accumulates during life, leading to an elevation in the intrinsic mortality rate. Repair is optimal only early in life, so the optimal strategy cannot stop ageing but can only postpone it to a later age. Extrinsic mortality seems to be the main determinant of the allocation strategy. The higher the rate of extrinsic mortality, the lower the amount of resources channelled to repair and the faster the intrinsic mortality increases. Under low extrinsic mortality, a large proportion of resources is allocated to repair at early ages and nothing is invested later. As a result, intrinsic mortality increases slowly early in life and starts to increase rapidly later (Fig. 3). Teriokhin (1998) also argued that mortality affects the allocation strategy: under high external mortality, the priority in spending energy is shifted primarily in favour of reproduction and then in favour of current survival (as, for example, with vigilance), whereas repair of damage that accumulates if not removed has a lower priority.

In a previous paper based on the same model (Cichoń, 1997), the analysis focused on the effects of extrinsic mortality and repair efficiency on age and size at maturity and growth rate. Moreover, relations between resulting life-history traits were investigated. The rate of ageing as well as the rates of growth and reproduction resulted from the allocation strategy. Extrinsic mortality and repair efficiency were again responsible for diversification of life histories. High investments in repair under low extrinsic mortality resulted in delayed ageing and low growth rates, delayed maturity and larger body size. Slow growth led to large body size because of delayed maturation, while small body size was achieved under fast growth. This was because investments in repair competed with growth for resources. Cichoń (1997) showed that repair efficiency (n in equation 4) generated different allocation patterns. High

repair efficiency promoted investment in repair and therefore delayed ageing and enhanced longevity. However, the differences in life-history strategies were mainly quantitative unless the efficiency was so low that investment in repair did not occur at all. In the present paper, the results for only one repair efficiency are presented, but qualitatively they are representative of a broader range of efficiencies.

The present model generates a variety of ageing patterns, from a linear increase of intrinsic mortality to exponential-like curves. This is in line with the findings of Abrams and Ludwig (1995). They showed that an exponentially increasing mortality rate lacked any general evolutionary explanation and could be considered one of many possibilities under the disposable soma theory. The present model reaches comparable conclusions, although it is built upon assumptions different from theirs; in particular, it does not need to assume any specific shape of the reproduction–repair trade-off function. The only parameter in the model generating different intrinsic mortality curves is the rate of extrinsic mortality. High mortality rates result in linear increases in intrinsic mortality with age (maximal ageing rate), while low mortality rates produce non-linear curves. When the extrinsic mortality rate is size-independent, the curves describing the age-dependence of total mortality resemble in shape those for intrinsic mortality (Fig. 3C). The shapes of the curves for total mortality are much more complex when extrinsic mortality decreases with size (Fig. 3D). Escaping the most endangered size range through rapid growth (at the cost of repair) explains this shape. To conclude, age-specific mortality curves like those derived from the Gompertz equation should not be assumed when modelling life-history evolution and ageing in particular, since they result from life-history optimization. The Gompertz model also appears an inadequate predictor of changes in mortality rate with age in comparative (Sibly *et al.*, 1997) as well as in experimental (Promislow *et al.*, 1996) studies.

The survivorship curves derived from the present model resemble typical curves characteristic of organisms living under different environmental conditions (Fig. 4; see Odum, 1971, or other general ecology textbooks for comparison). Under low extrinsic mortality, the survivorship curves resemble those known for species living in safe environments or in protected conditions; human populations living in modern developed societies are usually used as an example. The survivorship curves obtained under high mortality resemble those observed for species living in harsh environments. Pearl (1928; see ecology textbooks) distinguished three general types of survivorship curves: (i) the highly convex curve (type I) typical of species with mortality rates low until near the end of the lifespan, (ii) the highly concave curve (type III) which results when mortality is very high during the young stages, and (iii) an intermediate diagonal straight line (type II) typical of organisms with mortality nearly constant throughout the whole lifespan. The present results for cases of extrinsic mortality constant throughout life confirm the type I and II curves (Fig. 4A). Although size-dependent mortality rates resulting in very high mortality very early in life (as suggested by Odum, 1971, to generate the type III curve) were assumed in the model, the survivorship curves obtained were of intermediate types, with an inflection point (Fig. 4B). This was because growth was fast and the risky body size range was escaped quickly; mortality was roughly constant after growth stopped at maturation. Then mortality started to increase again due to ageing. A pure type III curve requires age-dependent mortality diminishing with age throughout life. Odum (1971) and others have discussed the diversity of survivorship patterns arising as the result of the age-specificity of the mortality rate, but possible mechanisms underlying this diversity were not recognized. The present model provides an explanation for at least type I, type II and curves having an inflection point.

These diverse types of curves result from the optimal strategy of allocation to growth, reproduction and repair, generating age-specific mortality due to varying levels of repair.

The disposable soma theory states that because of the priority of reproduction, natural selection favours a strategy that devotes fewer resources to somatic maintenance than are necessary for indefinite survival, especially when future prospects of survival are low due to extrinsic factors. The theory predicts a negative correlation between environmental mortality and longevity, and suggests that this correlation evolved through selection to vary the investment in repair (Kirkwood, 1990). To check whether this prediction would hold under the assumptions of the present model, we analysed the variation in maximum lifespan generated by the model under different extrinsic mortality regimes (Fig. 5). The results are in accord with Kirkwood's (1990) findings and show a negative correlation between maximum longevity and extrinsic environmental mortality. Testing this prediction in nature is difficult. The only promising studies are comparative ones, but there is still a problem distinguishing between extrinsic and intrinsic sources of mortality. Most analyses of mortality components have been based on the Gompertz equation, and such analyses have been justly criticized (Abrams and Ludwig, 1995; this paper). A more satisfactory approach to the role of somatic damage accumulation and repair in ageing would consist in investigating the level of somatic repair and observing its effect on ageing rate and longevity. The prediction that the allocation strategy determines longevity, namely that long lifespan should be tied to a high rate of repair, can be tested in comparative studies that involve some measure of repair. In a series of comparative studies on vertebrates, several workers (for a review, see Bernstein and Bernstein, 1991) have made use of DNA repair rates (from empirical studies it is known that a typical mammalian cell experiences tens of thousands of instances of DNA damage per day). They have argued that DNA repair rates influence the evolution of maximum lifespan through their impact on the accumulation of DNA damage, which could have direct deleterious consequences for several aspects of cell functioning. This, in turn, shapes survival probability and maximum longevity. Indeed, several workers have confirmed that long-lived organisms have higher rates of DNA repair. However, Promislow (1994) re-analysed these existing data and suggested that the observed correlations might result from confounding effects of phylogeny. Another line of support may come from comparative studies using the rate of free radical production as an indirect measure of repair rate (free radicals are highly reactive destructive metabolic wastes, and energy-consuming mechanisms are required to prevent their production, neutralize them or remove their effects; thus a low level of free radicals can indicate a high repair rate). A comparative study of the brains of vertebrates showed that long-lived species had low levels of *in vitro* free radical production (low free radical turnover) in their tissues, which supports the hypothesis, but showed low antioxidant concentrations at the same time, against the predictions (Barja *et al.*, 1994). However, these analyses may suffer from the same problems of the confounding effects of phylogeny as previously described and there is no clear distinction between free-radicals production rate and removal. In recent studies of the asexual annelid *Paranais litoralis*, the lifespan of the worms was extended experimentally by subjecting them to repeated injury (Martinez, 1996). Martinez claimed that exposure to increased injuries enhanced the normal level of repair, resulting in rejuvenation of the soma.

The measure of ageing is central to this discussion. The maximum lifespan considered by many authors as a measure of ageing is not reliable, since it is a trait that selection cannot see (Medawar, 1952), although selection for lifespan may be indirect through, for example, resource allocation. Rather, studies on ageing should look at age-dependent changes in

fitness components. Recently, some theoretical studies also questioned the use of the age-dependence of mortality rate and fecundity as a measure of ageing, since changes in a single vital rate displayed by an organism may appear to run contrary to changes in its state (Partridge and Barton, 1996). Blarer *et al.* (1995) suggested that either an increase in mortality or a decrease in fecundity could occur without any deterioration in the organism's physiology. Since there is no state deterioration, the observed demographic effects reflect different strategies and not ageing.

In conclusion, the optimal allocation of resources to repair results from the extrinsic mortality hazard an organism is subject to. This allocation is not constant throughout the lifespan but changes dynamically with the individual's age. The optimal allocation strategy produces age-specific patterns of mortality (ageing rates). These patterns do not generally fit the Gompertz model of age-specific mortality. Survivorship and maximum longevity strongly depend on extrinsic mortality, but it is the optimal allocation strategy that underlies this relationship and explains the observed variation. The survivorship curves obtained here resemble those observed in nature and we suggest that allocation patterns may mould the diversification of those curves in nature. These results are consistent with previous theoretical investigations and fit the existing data fairly well, especially those based on comparative analyses.

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