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A life history model of somatic damage associated with resource acquisition: damage protection or prevention?

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Abstract

A resource acquisition–allocation model is developed to examine the trade-off between reproduction and somatic protection. Unlike previous studies, resource intake is not assumed to be constrained: instead, resource intake is free to vary, with increased intake being associated with an increased risk of somatic damage. This gives rise to an optimal resource intake as well as an optimal allocation strategy. This paper studies the relative importance of acquisition and allocation strategies in regulating acquisition-related mortality. Under the optimal allocation strategy mortality rate increases with age, in accordance with the disposable soma theory of aging. Contrary to the usual interpretation of the disposable soma theory, this increase in mortality can arise from an increase in the resource acquisition effort rather than a decrease in the resources allocated to protection. At early ages resource acquisition is found to be the primary path for regulating life history costs, whilst allocating resources to protection becomes more important later in life. Models for targeted and non-targeted damage repair are considered and the robustness of our results to the structure and parameterization of the model is discussed. The results from our models are discussed in light of published data. Resource acquisition is shown to be a potentially important mechanism for controlling somatic damage which deserves further study.

Keywords: Lifehistory; Acquisition–allocation model; Aging; Somatic damage; Resource intake

1. Introduction

Damage seems to be an unavoidable consequence of living, and can range from the cellular level, with damage occurring to DNA and cellular proteins, to the loss of organs and structures (Kirkwood, 1981). Even for an organism to maintain itself it must run some risk of damage and a corresponding increase in mortality, whilst the demands of reproduction and growth carry additional dangers (Bell, 1980; Metcalfe and Monaghan, 2001). Because somatic damage is thought to play an important rôle in the aging process it has been a topic of considerable research, and is a central component of the wear and tear hypothesis of aging as well as the disposable soma theory of aging (Rose, 1991; Kirkwood and Austad, 2000). A large part of the interest in somatic damage is due to the fact that organisms frequently have the capacity to either protect themselves against damage or to repair structures which are already damaged. For example, organism's maintain immune responses against pathogens (e.g. Coop and Kyriazakis, 1999), energy and materials are devoted to

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detoxification processes (Illius and Jessop, 1995), cell damage can be repaired (Stadtman, 1992; Kapahi et al., 1999), the concentration of damaging free-radicals can be controlled with anti-oxidants (Finkel and Holbrook, 2000) and predation risks are lessened by vigilance behaviour (for a review see Treves, 2000). Despite these physiological and behavioural strategies for damage protection and their ability to sustain life, perhaps indefinitely, organisms still tolerate damage accumulation, and aging as a consequence.

Models of resource allocation to somatic protection, growth and reproduction are the main tool for understanding damage limitation strategies in an evolutionary setting. Models are used to study the allocation strategy which maximizes an organism's lifetime reproductive effort (e.g. Perrin, 1992; Stearns, 1992), and have led to considerable insight into the evolutionary forces operating on life history allocation strategies by allowing a quantitative formulation of the current theories of aging. Four recent studies have modelled resource allocation between reproduction and somatic protection (Abrams and Ludwig, 1995; Cichoń, 1997; Teriokhin, 1998; Shanley and Kirkwood, 2000), from which three general results emerge:

1. A certain amount of damage should be tolerated even if this damage could be repaired. This result comes from the fact that allocating resources to protection does not give an immediate fitness benefit. Since there is a probability of mortality, it is possible that the fitness benefits from damage repair will never be realized before death occurs. Therefore, allocating resources to damage protection is only beneficial if the future reproductive benefits are sufficiently certain, and it is often better to tolerate some damage in return for an immediate fitness gain.
2. The resources allocated to damage protection should decrease as the organism ages, producing an acceleration of somatic damage and senescence. Since future reproductive benefits are likely to decrease as an organism ages, damage repair becomes progressively less favourable.
3. There should be a negative correlation between resource allocation to protection and reproduction. This prediction is due to the general assumption that resources are allocated from a fixed budget, such that an increase in allocation to repair requires resources to be diverted away from reproduction. Since fitness can only be increased through resource allocation to reproduction, allocation to repair must be sacrificed at some point in order for an organism to reproduce.

In obtaining these general predictions the models have primarily concentrated upon allocation strategies where resources are supplied from a fixed budget. The assumption of a fixed resource budget stems from the hypothesis that resource intake is often constrained by factors such as resource availability, gut capacity, or foraging time (Allen, 1996). This hypothesis concurs with the common view of optimal foraging theory, that food intake is wholly beneficial, and should be maximized as far as the physical constraints allow (Stephens and Krebs, 1986). Although evidence for intake rate maximization does exist, especially for short-time-scales (Kenny and Black, 1984; van Wieren, 1996; Illius et al., 1999), and evidence for time minimization has been found on longer time-scales (Bergman et al., 2001), other data suggest that it is not a general mechanism. For example, organisms are seen to regulate their intake in response to changes in resource uncertainty (Forkman, 1993), as well as changes in the time available for foraging (Iason et al., 1999), supposed constraints on intake are observed to be elastic (Owen-Smith, 1994), reproductive provisioning in birds is restrained in relation to the predation risk (Lambrechts et al., 2000; Ghalambor and Martin, 2001), mice also appear to restrain their energy intake (Johnson and Speakman, 2001), whilst intake strategies amongst insects are seen to be either grow fast and risk a high mortality, or grow slowly and have a longer reproductive life (McPeek et al., 2001). These adaptive intake strategies do not appear to be maximizing intake, and may be balancing the benefits of resource intake against the possible costs. If this is the case what cost could resource intake incur?

Many different foraging costs have been proposed; for example, predation (Lima, 1998), parasitic infection (Coop and Kyriazakis, 1999; Hutchings et al., 2002), costs of energy expenditure (Deerenberg and Overkamp, 1999), costs of respiration (Ketelaars and Tolkamp, 1992a, b; Tolkamp and Ketelaars, 1992), free-radical damage (Finkel and Holbrook, 2000), ingestion of toxins (Duncan and Gordon, 1999), production of toxins by gut bacteria (Gibson et al., 1988), costs of resource storage (Witter and Cuthill, 1993) and excessive growth (Metcalfe and Monaghan, 2001). Studies that manipulate the brood size of birds find that resource acquisition is increased in birds with larger broods (Nilsson, 2002) and that increased brood size also increases susceptibility to oxidative stress (Alonso-Alvarez et al., 2004). Calorie restriction has been shown to affect age-regulated gene expression (Lee et al., 2002). Evidence such as this opens up the possibility of a link between resource intake and fitness costs. Arguably all of these costs are eventually associated with some form of somatic damage. Resource acquisition is, therefore, potentially an important source of somatic damage which can be controlled by regulating resource intake as well as by allocating resources to damage prevention and repair. Recognizing this link between resource acquisition and somatic damage emphazises the important connection between foraging behaviour and life

history (Boggs, 1992). Resource acquisition and resource allocation are interdependent, because they both affect the life history of an organism. Abrams and Ludwig (1995) noted that regulating resource acquisition could be an alternative to allocating resources to somatic protection, and suggested that future work could adapt their formulation to investigate acquisition–allocation models, but they did not develop their suggestion. The interdependency between resource intake and allocation, which is rarely addressed in theoretical studies, raises the question of when intake should be regulated, and how this should be complemented by the resource allocation strategy? This question can be answered by constructing a model which incorporates both resource acquisition and allocation.

In this paper, we directly address this question by studying an acquisition–allocation model where resource intake need not be constrained and carries with it a cost. We examine the three general conclusions from previous somatic protection models mentioned above and discuss their generality when resource intake is costly and unconstrained, and when damage accumulation is either dependent or independent of the current damage state of an organism. We determine the optimal resource intake and allocation strategy against somatic damage and investigate how this strategy is affected by changes in the model's parameters, such as the probability of damage occurring, the efficacy of damage protection and repair and the possibility of reproductive success in the future. The robustness of our results to the structure of the model is also investigated. Since resource allocation affects the life history of an organism over its entire lifetime, the model is applied to look at the optimal acquisition–allocation strategy over an organism's reproductively active lifetime.

2. The model

We develop a state-dependent model which investigates an organism's average resource intake and the optimal resource allocation towards reproduction and protection against damage, given an organism's current state of "damage" (where damage is associated with an organism's mortality risk). For the sake of clarity we take an organism's state of damage to be a physical somatic damage (e.g. cell damage due to free radicals), although the same model formulation could be used for more abstract forms of damage (e.g. an increased probability of being predated). For simplicity the model does not consider an organism's allocation to growth, and considers a constant physiological and external environment. We define an optimal strategy as one that maximizes an organism's lifetime reproductive effort. This measure of fitness is commonly used because of its simplicity (Stearns, 1992), although in general the measure of fitness will depend upon the density-dependence in the system (Mylius and Dickmann, 1995). Lifetime reproductive effort is a true measure of fitness if density-dependent population regulation affects an individual's expected lifetime reproductive effort (Mylius and Dickmann, 1995), although in many other situations lifetime reproductive success is an acceptably good proxy for fitness (Benton and Grant, 2000).

All resource intake is measured in units of an organism's basic maintenance requirements. In our model basic maintenance requirements do not include the resources used for protective mechanisms. Of an organism's total resource intake, i_T, one unit is used for maintenance, and the remainder is free to be allocated towards reproductive output, i_R, or towards protective mechanisms, i_P, such that $i_T = i_P + i_R + 1$. Our model considers both optimal and constrained resource intake. Under constrained intake, i_T is constrained to take a particular value, whilst under optimal intake (Yearsley et al., 2002) i_T is taken to be the value which maximizes fitness (optimal intake can vary through an organism's lifetime whilst constrained intake cannot).

2.1. An organism's state of damage

Our model assumes that food acquisition leads to the generation of somatic damage, which in turn increases the mortality risk to an organism. This mortality risk can be reduced by increasing the allocation of resources to somatic protection. In this paper, we will assume that somatic protection refers to damage repair mechanisms, although the same model formalism could be used for other protection mechanisms. Oxidative and toxic stresses are examples of somatic damage to cells and organs which can be associated with resource acquisition or energy expenditure (Alonso-Alvarez et al., 2004).

In the model the accumulated damage is a state variable of an organism. The somatic damage associated with resource acquisition can accumulate over an organism's lifetime and can only be reduced by allocating sufficient resources towards somatic repair. We denote the state of damage at the end of a breeding cycle as $D(D_0, i_T, i_P)$ where D_0 is an organism's state of damage at the start of the breeding cycle. Dependent upon the resource intake and the allocation to repair, it is possible for the somatic damage to either increase or decrease during a breeding cycle.

Since the relationship between resource use and somatic damage is unclear we study two contrasting processes of damage repair, which we call process 1 and 2. Both processes assume that the rate of damage accumulation is an increasing, accelerating function of the total resource intake, i_T and a decreasing, decelerating function of the resources allocated to repair, i_P. The

difference between the processes lies in the distribution of the repair effort. Process 1 assumes that resources allocated to repair are targeted specifically at the somatic damage, so that the rate of change of somatic damage can be written as

$$\frac{\mathrm{d}D}{\mathrm{d}\tau} = ai_T^{\alpha} - bi_P^{\beta}, \tag{1a}$$

where D is the level of damage, a is the rate of damage accumulation from unit resource intake and b is the rate of damage removal from unit allocation to repair. The parameters α and β represent the nonlinearity in the processes of damage accumulation and repair, respectively (where realistic bounds on the parameters α and β are $\alpha>1$ and $0<\beta<1$). Eq. (1a) implicitly assumes that resources allocated to repair are targeted at the somatic damage because the level of damage does not enter into the right-hand side of Eq. (1a). Therefore, one unit of resources allocated to repair will reduce somatic damage by the same amount irrespective of how common or rare somatic damage is within an organism.

Process 2 assumes that the resources allocated to repair are not specifically targeted at the damage, but are instead randomly distributed, which implies that the probability of effective repair per unit of resource allocated to repair is proportional to the amount of damage present. This process is written as

$$\frac{\mathrm{d}D}{\mathrm{d}\tau} = ai_T^{\alpha} - Bi_P^{\beta}D, \tag{1b}$$

where B has the same interpretation as b when there is one unit of damage (a generalization of this process which unifies Eqs. (1a) and (1b) is discussed when we consider the robustness of the model's results). By rescaling $D \to aD/B$ and $\tau \to \tau/B$, we can set $a = 1$ and $B = 1$ without loss of generality.

The power law relationships in Eqs. (1a) and (1b) were chosen because of their ability to capture the essence of a nonlinear relationship in a simple fashion. If $\alpha>1$ then damage accumulates proportionally faster as resource intake increases, so that a resource intake of two units is more than twice as harmful as a resource intake of one unit. If $\beta<1$ then as more resources are allocated to protection, their effect becomes increasingly inefficient.

Eqs. (1a) and (1b) can be solved to give the amount of damage after a time τ as

$$D(D_0, i_T, i_P) = D_0 + \tau(i_T^{\alpha} - bi_P^{\beta}), \tag{2a}$$

$$D(D_0, i_T, i_P) = D_{\infty} + (D_0 - D_{\infty})\,\mathrm{e}^{-\tau i_P^{\beta}}, \tag{2b}$$

where D_0 is the initial damage and $D_{\infty} = i_T^{\alpha}/i_P^{\beta}$ is the amount of damage under process 2 as τ becomes large. If τ is the length of a breeding cycle, then Eqs. (2a) and (2b) give the level of somatic damage at the end of the breeding cycle.

2.2. Reproduction and survival

We assume that the probability of an organism surviving until the end of a breeding cycle is related to the somatic damage by the equation

$$S(D_0, i_T, i_P) = \exp[-\mu - \sigma D(D_0, i_T, i_P)], \tag{3}$$

where μ is the extrinsic mortality rate and σ represents the mortality associated with each unit of damage. This survival function (Eq. (3)) assumes that survival is a random Poisson process. The expected reproductive effort can then be written as

$$E(\text{reproductive effort}) = (\gamma i_R + V)S(i_T, i_P), \tag{4}$$

where γ is the resource conversion efficiency and V is the expected future reproductive effort. An example of the survival function (Eq. (3)) and the reproductive effort (Eq. (4)) for process 2 (non-targeted repair) is shown in Fig. 1 (process 1, targeted repair, is qualitatively similar). Survival continuously decreases as resource intake for reproduction is increased, because damage is increased due to resource acquisition (Fig. 1a). Survival probability is increased by allocating resources to repair up to a critical threshold, above which the benefits of allocating resources to repair are outweighed by the risks associated with acquiring the resources in the first place. The reproductive effort attains a maximum at an optimal intake for reproduction, $i_R^{\star}$, and repair, $i_P^{\star}$ (Fig. 1b).

As the model is intended to be generic the formulation of damage accumulation could be applied to various scenarios. If $\alpha = 1$ then the amount of damage is proportional to the resource intake, which would likely be the case if predation risk were proportional to the time spent foraging. As a greater proportion of an organism's foraging effort is spent upon being vigilant then i_P increases and the overall predation risk decreases. In the case of parasitic infection, the amount of damage is associated with the parasite burden carried by an organism, where σ is the virulence of the parasites and α is the nonlinear effect of increasing parasite burden upon the host's survival probability. Applied to toxins, or free-radical damage, the amount of damage can be interpreted as the level of oxidative or toxic stress. This stress is a balance between the source of the stress (ultimately resource intake) and the protective mechanisms which try to limit this stress. In this case, σ describes the importance of stress in determining an organism's survival probability.

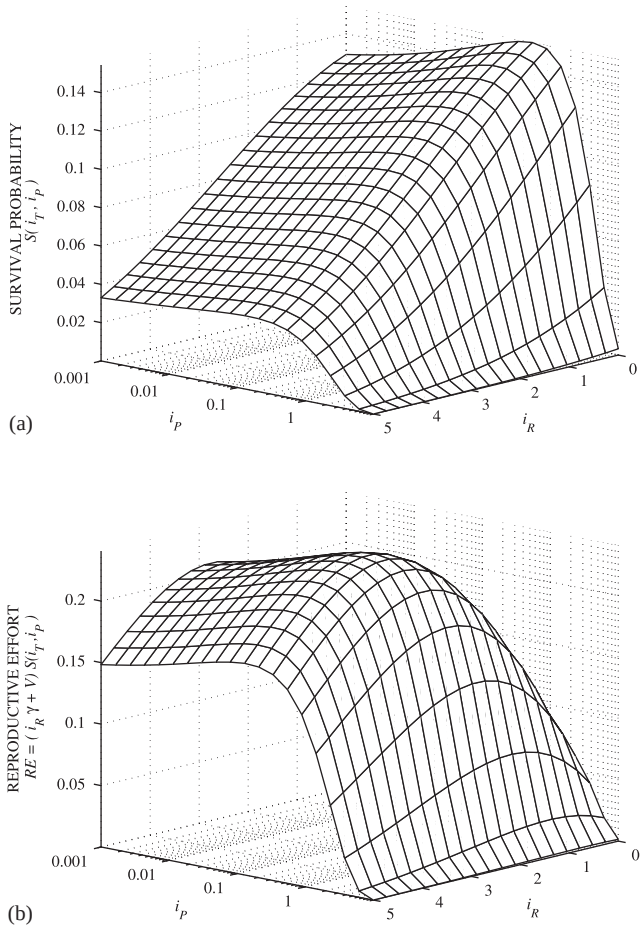

Fig. 1. (a) The survival probability (Eq. (3)) and (b) the reproductive effort (Eq. (4)) as a function of the resource intake allocated to reproduction, i_R, and the resource intake allocated to protection, i_P, for the process when damage accumulation given by Eq. (1b), and with the default parameter values shown in Table 1. The optimal allocation strategy, $(i_R^\star, i_P^\star) = (2.6, 0.45)$ occurs where the reproductive effort is a maximum. Future reproductive effort is $V = 0.5$, and the initial damage is $D_0 = 10$. All intakes are in units of maintenance.

2.3. Lifetime allocation strategy

Given an organism's damage state $D_0(x)$, where x is the age of the organism measured in breeding cycles, the allocation strategy throughout an organism's lifetime can be calculated by an iterated backward propagation of Eq. (4). This is achieved by equating the expected reproductive effort at age x with the future reproductive effort at age $x-1$ (Mangel and Clark, 1988; Houston and McNamara, 1999; Clark and Mangel, 2000). The algorithm assumes that an organism is reproductively active for all the breeding cycles being considered. The optimal allocation strategy at age x, $(i_R^\star(x), i_P^\star(x))$, is given by

$$V(x-1) = \max_{i_R, i_P}\{[\gamma i_R(x) + V(x)]S(D_0(x), i_T(x), i_P(x))\}. \tag{5}$$

Normally, Eq. (5) can be solved at the maximum age of an organism because at this age the future reproductive effort, V, is zero. This solution then provides the future reproductive effort of the proceeding breeding cycle. This process is then iterated back until $x = 1$. However, the optimal solution of Eq. (5) requires solving for $i_T(x)$, $i_P(x)$ and $D_0(x)$. Finding this solution is complicated by the fact that an organism's state, $D_0(x)$, is itself a function of i_T and i_P at earlier ages. This is because the initial damage at age x is related to the initial damage at age $x-1$

$$D_0(x) = D(D_0(x-1), i_T(x-1), i_P(x-1)). \tag{6}$$

Because the optimal strategy depends upon both future and past allocation strategies, a single backward propagation of Eq. (5) will not find the optimal strategy. Instead, the optimal lifetime strategy is found by iterating both Eqs. (5) and (6). An initial guess is made for $D_0(x)$, and then Eq. (5) is solved by backward propagation. Then this solution together with Eq. (6) is used to give a new set of values for $D_0(x)$. This process is iterated until successive iterations change the solution by less than 0.001%.

2.4. Default parameter values

An initial set of parameter values were chosen for the model based, wherever possible, upon observations. The default values are summarized in Table 1. The model's sensitivity to these parameter values was investigated and is presented in the results.

The conversion efficiency of protein and energy, γ, is generally thought to lie somewhere between 0.8 and 0.9 (Kyriazakis and Emmans, 1992). We chose a value of γ to be 0.8, although the model's behaviour is insensitive to this parameter. We further assumed that the conversion efficiency is constant, which is likely to be the case for nutrients (Kyriazakis and Emmans, 1992), but not for energy intake (Tolkamp and Ketelaars, 1992). Letting conversion efficiency be a decreasing function of resource intake (using the exponential function from Tolkamp and Ketelaars, 1992) did not change the qualitative results, although a general reduction in the predicted optimal intake rates was observed.

Direct estimates for the remaining five parameters, α, β, σ, t and $D_0(1)$ could not be found in the literature. We assume that an organism enters its first breeding cycle with no accumulation of damage, so that $D_0(1) = 0$. Data exists on the magnitude of allocation strategies, and this data can be used to constrain the parameter space for the remaining parameters. Field metabolic rates commonly lie within the range of 2.5–3 times maintenance requirements (Peters, 1986; Nagy et al., 1999), total energy intake rate is not normally expected to exceed five times maintenance (Kirkwood, 1983) and

Table 1
The default parameter values used in the model

Parameter		Default value
Intake conversion efficiency	γ	0.8
Severity of damage	σ	0.2
Extrinsic mortality rate	μ	ln(0.9)
Nonlinearity of damage to food intake	α	2
Nonlinearity of protection	β	0.5
Specific rate of damage repair for process 1	b	1
Rate of approach to equilibrium	τ	0.2
Initial damage for first breeding cycle	$D_0(1)$	0
Maintenance requirement		1

Justification of the values used is given in the text. The state-independent model sets $\tau = 100$ which effectively made the damage at the end of a breeding cycle independent of the damage at the start of that cycle.

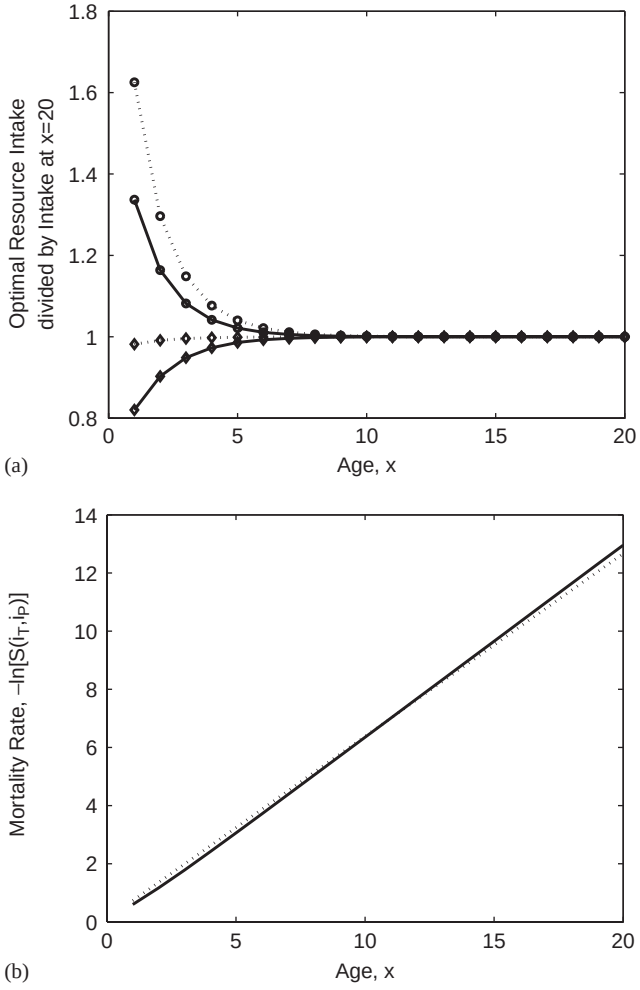

Fig. 2. (a) The optimal resource intake and allocation for targeted repair (Process 1) during an organism's lifetime as a fraction of the intake at age $x = 20$, and (b) the associated mortality rate for unconstrained (solid lines) and constrained (dotted lines) acquisition (acquisition constrained to be $i_T = 4$). The intakes at age $x = 20$ are $i_R^\star(20) = 3.1$, $i_P^\star(20) = 0.004$ for the unconstrained acquisition $i_R^\star(20) = 2.9$, $i_P^\star(20) = 0.1$ for the constrained acquisition. Lines marked with diamonds and circles correspond to reproductive and repair allocation, respectively. Default parameters are given in Table 1.

only in extreme cases it can exceed seven times maintenance (Mellish et al., 2000). Nutrient intake rate is likely to be slightly higher, resulting in an upper limit to resource intake being set as $i_T < 10$. Allocation to protection, such as immune functions against parasites, and cell maintenance (Jessop, 2000) are generally observed to be less than 50% of maintenance requirements, and normally nearer 10–20% (Houdijk et al., 2001), so we set the upper limit for allocation to protection as $i_P < 0.5$. Using these constraints on i_T, i_P, the remaining default parameters were set to be: $\sigma = 0.2$, $\mu = 0.1$, $\alpha = 2$, $\beta = 0.5$, $b = 1$ and $\tau = 0.2$. These values were arbitrarily chosen to ensure that the above constraints were obeyed by both the default model and moderate parameter variations about this default. The value of $b = 1$ implies that somatic damage can never decrease in the targeted repair model.

3. Results

3.1. Targeted repair (process 1)

For targeted repair there are no state-independent results because there is no equilibrium level of somatic damage. The optimal allocation strategy for the state-dependent, process 1 model is shown in Fig. 2a for both optimal and constrained resource intakes. These simulations are for the default parameter settings in Table 1, and assume that an organism has a maximum of 20 breeding cycles (although an organism is unlikely to survive until its 20th breeding cycle). For both optimal and constrained resource intake the allocation to reproduction is predicted to increase with age and allocation to repair is predicted to decrease with age. Optimal resource intake is predicted to reach a plateau at late ages as the organism's damage state dominates the cost–benefit mechanism of food intake regulation. The effect of reducing the maximum number of breeding cycles (bringing the final time horizon closer to the first breeding cycle) is to shrink the size of the plateau, and to increase the importance of the time horizon on the strategies of the final breeding cycles (i.e. to increase total resource intake).

The mortality rate under these optimal allocation strategies is shown Fig. 2b. Both optimal and constrained resource intake show a linearly increasing mortality rate with age, corresponding to the gradual accumulation of somatic damage. Under these parameter values an organism is unlikely to survive more than a few breeding cycles.

3.2. Non-targeted repair (process 2)

3.2.1. State-independent model

The simplest form of the non-targeted repair model occurs when an organism's level of mortality risk is always at its equilibrium value, D_∞ (Eq. (2b)), ensuring that the current mortality risk is independent of an organism's past history of damage. This is a state-independent model, and corresponds to the limit of $\tau \to \infty$. The optimal resource allocation to reproduction $i_R^\star$ and repair $i_P^\star$ for this state-independent model can be explicitly found by solving Eq. (5), giving

$$\frac{i_R^\star(x)}{i_T^\star(x)} = \frac{1}{\sigma\alpha}\left(\frac{i_P^\star(x)}{i_T^\star(x)}\right)^\beta (i_T^\star(x))^{2+\beta-\alpha} - \frac{V(x)}{\gamma\, i_T^\star(x)}, \tag{7a}$$

$$\frac{i_P^\star(x)}{i_T^\star(x)} = \frac{\beta}{\alpha}. \tag{7b}$$

Eqs. (7a) and (7b) can be used to solve for $i_T^\star$, since $i_T^\star(x) = 1 + i_R^\star(x) + i_P^\star(x)$. Changes in the severity of damage accumulation, σ, and the future reproductive effort, $V(x)$, have no effect upon the proportion of intake allocated to repair (Eq. (7b)). Therefore, the primary response to changes in damage severity and future reproductive effort, under the optimal strategy is to regulate acquisition rather than to control for the costs of acquisition through protection. The allocation strategy is independent of the extrinsic mortality, μ, and the conversion efficiency, γ.

Fig. 3 shows the lifetime allocation of resources to reproduction and repair in this state-independent model for two models of resource intake: constrained and optimized. Default parameters were used (Table 1) except τ which was set to be large enough that the model was state-independent ($\tau = 100$). Qualitative differences between constrained and optimized intake are only noticeable as the final time horizon approaches (set at an age of $x = 20$). Here the allocation to reproduction increases for both models of intake, and the difference lies in the allocation to repair. In the constrained intake model resource allocation to repair decreases because total resources are limited, in the optimized intake model allocation to repair increases because total resource intake increases.

In the example shown in Fig. 3a, very few organisms will survive long enough to experience the changes in allocation strategy as the time horizon is approached. If the time horizon is reduced then the behaviour of the allocation strategies in the final breeding cycles is preserved and it is the plateau regions in Fig. 3 which is reduced until eventually no plateau exists.

3.2.2. State-dependent model

The model becomes increasingly state-dependent as τ decreases because the amount of damage at the end of a breeding cycle is increasingly determined by the initial amount of damage (Eq. (2b)). In the state-dependent model, the optimal allocation strategy depends upon an organism's past allocation strategy, through $D_0(x)$, and its expected future allocation strategy, through $V(x)$. All results for the state-dependent model were found numerically.

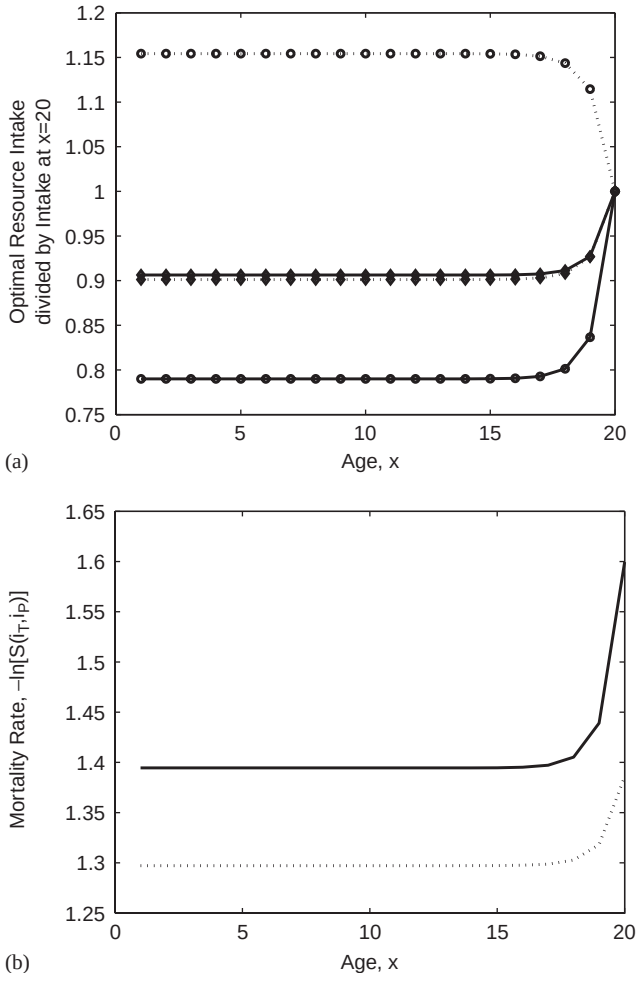

Fig. 3. (a) The optimal resource intake and allocation for state-independent non-targeted repair (process 2) during an organism's lifetime as a fraction of the intake at age $x = 20$, and (b) the associated mortality rate for unconstrained (solid lines) and constrained (dotted lines) acquisition (acquisition constrained to be $i_T = 2$). The model is state-independent, so that the somatic damage at age x is independent of the somatic damage at age $x-1$. The intakes at age $x = 20$ are $i_R^\star(20) = 0.8$, $i_P^\star(20) = 0.6$ for the unconstrained acquisition $i_R^\star(20) = 0.6$, $i_P^\star(20) = 0.4$ for the constrained acquisition. Lines marked with diamonds and circles correspond to reproductive and repair allocation, respectively. Default parameters are given in Table 1.

Fig. 4 shows the results of a state-dependent model with a time horizon of 20 breeding cycles. Unlike the state-independent model, the optimal allocation strategies are seen to vary throughout the lifespan of an organism because the state of the organism is changing (Fig. 4a). The behaviour of this state-dependent model also differs from the targeted repair model (Fig. 2), because in this model of non-targeted repair the changes

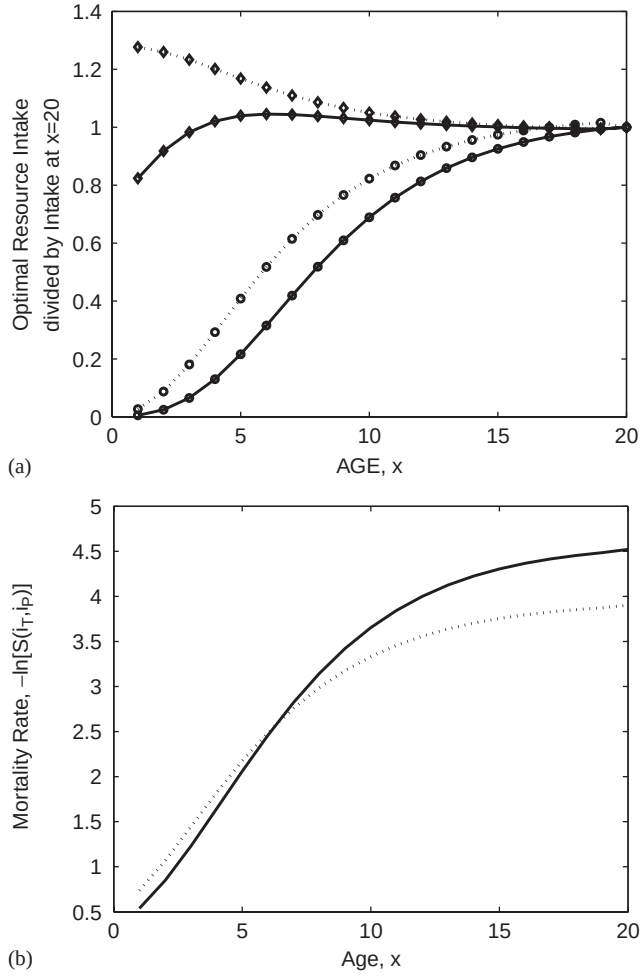

Fig. 4. (a) The optimal resource intake and allocation for state-dependent non-targeted repair (Process 2) during an organism's lifetime as a fraction of the intake at age $x = 20$, and (b) the associated mortality rate for unconstrained (solid lines) and constrained (dotted lines) acquisition (acquisition constrained to be $i_T = 4$). The intakes at age $x = 20$ are $i_R^\star(20) = 2.8$, $i_P^\star(20) = 1.2$ for the unconstrained acquisition $i_R^\star(20) = 2.3$, $i_P^\star(20) = 0.7$ for the constrained acquisition. Lines marked with diamonds and circles correspond to reproductive and repair allocation, respectively. Default parameters are given in Table 1.

in state feedback to an organism's rate of damage accumulation (Eq. (1b)). The changes in state lead to important differences in the optimal allocation patterns. Firstly, the magnitude of the changes in allocation strategy are considerably larger than those in the state-independent model. Secondly, the allocation to reproduction generally decreases with age, whilst the allocation to repair increases. Under the optimal strategy state-dependent changes in damage are primarily controlled by changing the allocation to protection, whereas for the state-independent model damage is modulated primarily through resource intake. Thirdly, the time horizon at $x = 20$ does not produce an important change in strategy because the behaviour is dominated by the organism's state (reducing the time horizon has little effect on the allocation strategies of early breeding cycles). Finally, the mortality rate under the state-dependent model is seen to decelerate and finally plateau in old age (Fig. 4b). This plateau occurs because the somatic damage is approaching an equilibrium level where the accumulation of damage is being balanced by the rate of damage repair.

Unlike the state-independent model, the regulation of resource intake in the state-dependent model (either constrained or optimal) has an important effect on the allocation strategy early in an organism's life history. The model with constrained resource intake requires that increases in reproduction are countered by decreases in repair allocation. In contrast, optimal intake is found to increase throughout the lifetime of an organism and this can allow both reproductive and repair allocation to increase. At early ages, when accumulated damage is low, the increase in optimal intake is used to increase allocation to reproduction (the allocation to repair at early ages is similar for both constrained and optimal intake models, Fig. 4a). Later in the lifespan of an organism, as damage further accumulates, the optimal intake is preferentially allocated to protection causing a decrease in allocation to reproduction.

3.3. Model robustness

The qualitative features in Figs. 2–4 were found to be robust to changes in the parameters. Table 2 shows the sensitivities of the state-dependent models for both optimal and constrained intake. In general the allocation strategies are most sensitive to parameters involved in the rate and severity of damage accumulation (i.e. σ, τ, and i_T). In addition, under optimal resource intake the nonlinearity in damage accumulation, α, is an important parameter, whereas for constrained resource intake the parameters affecting the rate of damage removal (b for process 1 and β for process 2) are more important. Sensitivities for reproductive allocation are higher than those for repair, indicating that changes in resource intake costs are primarily regulated through total resource intake. For optimal intake and non-targeted repair, the proportional increase in sensitivity between the ages $x = 1$ and 15 is greatest for allocation to repair, suggesting that as age increases there is a greater tendency to use repair as a means of regulating costs.

Since the qualitative features of targeted and non-targeted repair differ, the robustness of the results to the structural form of Eq. (1) was investigated by looking at a third model which changed Eq. (1) so that damage accumulated at a rate given by

$$\frac{\mathrm{d}D}{\mathrm{d}\tau} = a i_T^\alpha - b i_P^\beta D^\eta. \tag{8}$$

Apart from this change the model structure is unchanged. Eq. (8) simplifies to the structure of Eqs. (1a)

Table 2
The sensitivity of the optimal allocation strategy, i_R and i_P at ages $x = 1$ and 15 for constrained and optimal intake under the state-dependent, targeted (process 1) and non-targeted (process 2) repair model (all values to two decimal places)

	Parameter being varied	Sensitivity of optimal reproductive allocation		Sensitivity of optimal protection allocation	
		$i_R(x=1)$	$i_R(x=15)$	$i_P(x=1)$	$i_P(x=15)$
Optimal resource intake					
	σ	−6.24	−8.14	0.02	0.02
	α	−2.29	−2.78	−0.01	−0.01
Process 1:	β	0.04	0.01	−0.03	−0.03
targeted	τ	−6.24	−8.14	0.02	0.02
repair	b	−0.01	−0.00	0.01	0.01
	$D_0(1)$	0.15	0.67	0.02	0.04
	$\exp(-\mu)$	−0.86	−0.00	0.00	0.00
	σ	−5.61	−7.40	−0.02	−2.58
	α	−2.05	−2.59	−0.01	−1.54
Process 2:	β	−0.79	−0.84	−0.04	3.00
non−targeted	τ	−5.59	−7.42	0.04	−0.75
repair	$D_0(1)$	0.31	−0.01	0.01	0.03
	$\exp(-\mu)$	−1.25	−0.02	−0.01	−0.04
Constrained resource intake, $i_T = 4$					
	σ	−0.84	−0.83	0.84	0.83
	α	0.11	0.00	−0.11	0.00
Process 1:	β	0.02	0.10	−0.02	−0.10
targeted	τ	0.40	0.00	−0.40	−0.00
repair	b	−0.27	−0.17	0.27	0.17
	$D_0(1)$	0.13	0.00	−0.13	0.00
	$\exp(-\mu)$	0.08	0.00	−0.08	0.00
	i_T	0.96	0.94	0.05	0.06
	σ	−0.12	−2.51	−0.12	2.51
	α	−0.04	−0.77	0.04	0.77
Process 2:	β	0.07	−1.04	−0.07	1.04
non-targeted	τ	−0.31	−3.07	0.31	3.07
repair	$D_0(1)$	−0.02	−0.01	0.02	0.01
	$\exp(-\mu)$	−0.01	−0.02	0.01	0.02
	i_T	0.97	0.47	0.03	0.53

The model has zero sensitivity to γ. All other parameters are perturbed by 10% of their default values (Table 1), except $D_0(1)$ which is perturbed by 0.1.

and (1b) when $\eta = 0$ and 1, respectively. As η approaches zero the resources allocated to repair become increasingly targeted at the somatic damage. As η becomes more positive, repair becomes increasingly non-targeted. The robustness of our qualitative results to the structure of Eqs. (1a) and (1b) was investigated by varying η. The decrease in reproductive allocation seen in Fig. 4a was seen for values of η between 0.3 and 1.3, whilst the increase in allocation to repair with age was seen for all values of η greater than 0.05. For values of η below 0.02 allocation to repair is seen to always decrease with age similar to that in Fig. 2a (values of η between 0.02 and 0.05 showed an initial decrease in allocation to reproduction, and an increase at late ages). We conclude that the qualitative results in Fig. 4a seem to be robust to different forms of damage density-dependence. In contrast, the results of Fig. 2a for targeted damage repair only seem to hold for situations where the efficiency of the repair process is close to being independent of the level of somatic damage.

4. Discussion

Six versions of our model have been presented through combinations of targeted and non-targeted repair, state-dependent or state-independent, and constrained or optimal resource acquisition. All models show that an increase in the mortality risk of resource acquisition (quantified by parameter σ) leads to a reduction in reproductive allocation, and also to a decrease in total resource intake for the optimal intake

models (Table 2). Consistent with this, observations show that individuals under increased mortality risk reduce their resource intake, be it due to predation pressure (Korpimaki et al., 1994; McPeek et al., 2001; Kotler et al., 2002) or parasite infection (Kyriazakis et al., 1998). Other results differed between the models.

A large difference was seen in the mortality rate trajectories between the models (Figs. 2b, 3b and 4b). Data show that mortality rate generally increases with age (Vaupel et al., 1998), but mortality deceleration at late ages is also observed in a number of species: humans, the nematode *Caenorhabditis elegans*, the fruit fly *Drosophila melanogaster*, the medfly *Ceratitis capitata* and the beetle *Calosobruchus maculatus* (Vaupel et al., 1998; Partridge and Mangel, 1999). Several explanations have been proposed for mortality deceleration, but as yet there is no consensus of opinion (Vaupel et al., 1998). This pattern of decelerating mortality rate with age is consistent with the results of the non-targeted repair (process 2), state-dependent models (Fig. 4b), where the deceleration is due to an approaching equilibrium between damage creation and repair. This equilibrium exists because damage repair under process 2 depends upon the level of damage as well as the allocation to repair (Eqs. (1b)). The state-independent models show no change in mortality rate until the final time horizon approaches and the model of targeted damage repair shows a linear increase of mortality rate with age.

The non-targeted repair, state-dependent models further predict that resource allocation to repair should increase with age (Fig. 4a), which is a surprising result since the classical view is that allocation to repair should decrease with age (Kirkwood, 1981). However, increasing allocation to repair mechanisms with age is not without observational support. Experiments on the fruitfly (Zou et al., 2000) and the mouse (Lee et al., 1999) show that the transcription of stress response genes generally increase with age, although Lee et al. (1999) found that calorie restriction of mice prevented this increase in transcription. Bohr (2002) studied DNA repair in mammalian cells, and found that mitochondrial DNA repair activity increases with age, although recent results suggest that whilst repair activity in aged organisms is increased, it is also less efficient (Szczesny et al., 2003). Increased repair activity with age has also been found in mice (de Souza-Pinto et al., 2001) and rats (Hudson et al., 1998). Despite increased repair activity, DNA damage is still seen to accumulate in mitochondria since mitochondria are a major source of free-radical production. For nuclear DNA no consensus can be found for age-related changes in repair activity (Bohr, 2002). Finally, from an immunological perspective work on sheep shows a reduced immunological responsiveness in young organisms compared to mature organisms, which is not due to the reduced exposure of young organisms to pathogens (Colditz et al., 1996).

In the introduction three general predictions from previous somatic repair allocation models were introduced. For these models (Abrams and Ludwig, 1995; Cichoń, 1997; Teriokhin, 1998; Shanley and Kirkwood, 2000) resource intake was assumed to be constrained, so that resources were allocated from a fixed budget, and damage repair was assumed to be independent of the level of damage. How do these general predictions compare with our present results?

1. All models predict that some somatic damage should be tolerated. This is a robust prediction irrespective of the details of either the resource intake regulation or the mechanism of damage protection.
2. Contrary to the other published models, the models of non-targeted damage repair predict that allocation to damage repair should increase with age (assuming everything else to be constant), whilst the models of targeted damage repair are consistent with earlier models in predicting a decline in repair allocation with age. The predictions of increasing repair allocation with age are robust to changes in the model's details, such as the function for the amount of damage (Eq. (1b)) or variability in the model's parameters. The increasing allocation to repair is not sufficient to avoid an increase in the accumulation of damage. So although the absolute allocation to repair increases with age, the mortality rate also increases. This increase in somatic damage with age underlies the reason why repair is predicted to increase. For non-targeted repair, the efficiency of repair increases as damage increases (because the random distribution of the repair effort acts on proportionately more somatic damage as the amount of damage increases), and this provides a strong selective force to increase the resource allocation to repair.
3. Whilst existing models predict direct competition for resources between allocation strategies, which gives rise to a negative correlation between strategies. This competition is very much weakened in the optimal acquisition models considered here, because there is no fixed resource budget. Therefore, the general prediction of a direct trade-off between allocation strategies is no longer a necessary outcome if resource acquisition is optimally regulated.

Compared to the constrained acquisition model, the optimal acquisition model weakens the correlation between allocation strategies because the resource budget is not fixed. In turn, this produces a weak correlation between repair allocation and other life history traits, such as mortality risk or lifespan. Evidence for correlations between the allocation to repair and other life history traits is mixed. For example, somatic damage due to free-radicals and protection from anti-oxidants has received a lot of attention (e.g.

Stadtman, 1992; Sohal and Weindruch, 1996; Finkel and Holbrook, 2000; Melov et al., 2000; Zou et al., 2000; López-Torres et al., 2002), but it is still unclear whether increased allocation to anti-oxidant production is associated with increased survival. Some results seem to indicate that survival is positively correlated with protection (Kapahi et al., 1999; Kirkwood and Austad, 2000; Melov et al., 2000), whilst other results seem to show little or no evidence for any correlation (Viarengo et al., 1995; Selman et al., 2000; Mockett et al., 2001). Evidence is also mixed for a direct trade-off between reproduction and survival, with the strongest evidence coming from stressful situations where resources are clearly limiting (a situation which would favour intake maximization, Stearns, 1992). Previous explanations for this lack of apparent trade-off have been based upon inter-individual variation in resource intake (van Noordwijk and de Jong, 1986; de Jong and van Noordwijk, 1992), or successive resource allocations (de Jong, 1993). The lack of a clear picture may be an indication that additional factors, such as resource acquisition, are also playing a rôle in controlling life history costs.

Resource intake may not always be regulated to ensure an optimal balance between costs and benefits. As pointed out earlier, there may be circumstances when resource acquisition is effectively constrained. For our model this is most likely when the predicted optimal resource intake is high, exceeding an organism's capabilities, and may occur during periods when either damage severity is small, or resource quality is low, or future reproductive effort is small (i.e. towards the end of an organism's life). A further complication, not included in our model, is the possibility that repair may have negative as well as positive effects. For example, anti-oxidants are often acknowledged as being an important protective mechanism against the harmful effects of free-radicals, but they can also cause an increase in disease susceptibility in excessive doses (Erel et al., 1997; Salganik, 2001). Such effects could limit the degree to which the allocation to repair strategies is useful, and suggests another reason for using resource acquisition as one means of controlling somatic damage.

Finally, evidence for the relevance of state-independent damage is provided by one study on dietary restriction in fruitflys (*Drosophila melanogaster*) (Mair et al., 2003). Dietary restriction at any age was found to reduce mortality to the same level, suggesting that there was no difference in state between flies of different ages.

5. Conclusions

The model presented here develops an acquisition–allocation model with three novel aspects: it compares acquisition strategies which are constrained and optimal, it compares state-dependent and state-independent damage accumulation and it compares repair strategies which are targeted and non-targeted towards the somatic damage. The results emphasize that acquisition and allocation strategies are inter-dependent. In particular, the regulation of resource intake can be more important than allocation to repair strategies for controlling the costs inherent in gathering and consuming resources. The regulation of these costs over an organism's lifetime leads to an increasing mortality rate with age. Although senescence has several theoretical explanations (Rose, 1991), we believe our model is unique in suggesting that senescence can be as much to do with an organism's foraging behaviour as it is to do with its resource allocation.

The connection between resource acquisition and resource allocation has rarely been explored, despite the fact that the two are commonly interdependent (Boggs, 1992; Nilsson, 2002). The model presented here attempts to clarify the importance of intake regulation and show that, within the assumptions of the model, increasing damage and the associated increase in mortality can be due to an increase in the effort devoted to resource acquisition rather than a decrease in allocation to protection. However, although there is strong evidence for the presence of resource acquisition costs, there is a tantalizing lack of information on the importance of these costs in shaping an organism's resource acquisition behaviour. Our model suggests that the priorities for future experimental research programmes are: to clarify the link between resource intake regulation and the costs of resource intake, to identify the critical costs, and to identify the mechanisms used to protect against these costs (e.g. targeted vs. non-targeted repair mechanisms). If organism's regulate their resource intake in relation to the costs, then foraging theory, resource intake allocation theory and theories of aging may all be fundamentally linked.

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