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A diffusion-based approach to stochastic individual growth and energy budget, with consequences to life-history optimization and population dynamics

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Abstract

Using diffusion processes, I model stochastic individual growth, given exogenous hazards and starvation risk. By maximizing survival to final size, optimal life histories (e.g. switching size for habitat/dietary shift) are determined by two ratios: mean growth rate over growth variance (diffusion coefficient) and mortality rate over mean growth rate; all are size dependent. For example, switching size decreases with either ratio, if both are positive. I provide examples and compare with previous work on risk-sensitive foraging and the energy–predation trade-off. I then decompose individual size into reversibly and irreversibly growing components, e.g. reserves and structure. I provide a general expression for optimal structural growth, when reserves grow stochastically. I conclude that increased growth variance of reserves delays structural growth (raises threshold size for its commencement) but may eventually lead to larger structures. The effect depends on whether the structural trait is related to foraging or defence. Implications for population dynamics are discussed.

Introduction

In the past three decades, dynamic optimization has been widely and successfully applied to a myriad of questions and problems in evolutionary biology. Dynamic optimization models can roughly be divided into deterministic and stochastic. In deterministic models, individuals that are initially identical, remain identical in subsequent times, given that all follow the same strategy. Such models have used a variety of optimization methods: static optimization (e.g. Kozlowski & Wiegert, 1986), Pontryagin's maximum principle (e.g. Schaffer, 1983; Perrin & Sibly, 1993), dynamic programming (e.g. Ludwig & Rowe, 1990) and have derived many important results; for example, the dependence of optimal switches between growth and reproduction and other life-history transitions (e.g. metamorphosis), on the ratio of size-dependent production over size-dependent mortality (e.g. Werner & Gilliam, 1984; Perrin & Sibly, 1993; Hutchinson *et al.*, 1997; Kozlowski, 2006); conditions for determinate vs. indeterminate growth (e.g. King & Roughgarden, 1982; Sibly *et al.*, 1985; Perrin *et al.*, 1993; Kozlowski & Teriokhin, 1999) and optimal patterns of simultaneous allocation to several structures (i.e. optimal patterns of allometric growth; Iwasa & Roughgarden, 1984; Perrin, 1992; Irie & Iwasa, 2005).

Stochastic models additionally incorporate random components in the dynamics of an individual's state (e.g. due to random variation in prey capture success; Tenhumberg *et al.*, 2000). In these models, even initially identical individuals that follow the same optimal strategy diverge in their subsequent growth and development. Optimization under such stochastic state dynamics was mainly investigated using stochastic dynamic programming (e.g. Houston & McNamara, 1999; Clark & Mangel, 2000). Important results of these studies concern risk-sensitive foraging, the energy–predation trade-off and state-dependent life histories.

Diffusion models may serve as a framework for analysing situations where consumption and growth vary stochastically. Diffusion processes have played prominent roles in many subfields of ecology and

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evolutionary biology: dispersal and animal movement (e.g. Okubo & Levin, 2001), stochastic population dynamics (e.g. Lande *et al.*, 2003) and population and quantitative genetics (e.g. Kimura, 1965). In relation to individual growth and life history, McNamara (1983, 1984) (Houston & McNamara, 1985; see also McNamara *et al.*, 2001) used diffusion processes to model optimal risk-sensitive foraging. Houston *et al.* (1993) used a diffusion approximation in the context of the energy–predation trade-off, when energy gain is stochastic. In addition, Iwasa (1991) used a diffusion process to study optimal patterns of allocation to vegetative growth vs. reproduction in plants, under fluctuating resource levels. Finally, Hara (1984a,b) and Hara *et al.* (1991) applied diffusion equations to model 'noisy' growth of individuals in the context of dynamics of plant size distributions. Kirkpatrick (1984) similarly used a white-noise process in modelling stochastic growth in a size-based demographic model.

In this paper, I extend the use of such diffusion models, to investigate the effect of stochastic growth and energy budget on optimal life histories, under starvation risk and exogenous hazards (e.g. predation). I first provide some general results, regarding a reversibly growing size measure (e.g. total mass or mass of energy reserves), and relate these results to previous work (e.g. Houston *et al.*, 1993). Then I extend the analysis to consider simultaneous growth of both structural mass (growing irreversibly) and energy reserves (growing reversibly according to a diffusion process). I derive general expressions for the optimal growth of the structural trait, given that stochasticity in reserves entails starvation risk, and individuals are also exposed to exogenous hazards.

I then use the results of this general analysis to examine some special cases. For example, a purely defensive trait that reduces exogenous mortality but also impairs foraging ability (or conversely, for a foraging-improving trait). I study the effects of increasing the noise in the growth dynamics of reserves, on the optimal (irreversible) growth of the structural trait. Finally, I draw in the Discussion section some important consequences to population dynamics and stability.

Basic derivations

Survival-to-size when growth is formulated as a diffusion process

I define an individual's state by its size, a continuous variable, denoted by y. The size of an individual can both increase and decrease over time (but see the section concerning structural growth). A typical such size variable may be the mass of energy reserves. Moreover, the dynamics of $y(t)$ (where t denotes time) is described by a diffusion process with mean growth rate, given by $g(y)$, and variance in growth, given by $\sigma^2(y)$. (It is important to note that the units of σ^2 are [size^2/time], and not [size^2/time^2] as expected for variance in growth rates. That is because σ^2 is a diffusion coefficient, i.e. it describes the rate of increase in the variance of size, due to stochasticity in growth.) I shall refer to g as the *mean growth rate*, and to σ^2 as the *growth variance*.

The mean growth rate $g(y)$ captures the mean balance of input and output flows of energy and mass (Kooijman, 2000) that cause changes in the state of individuals. For example, assimilation of consumed food (input) and metabolic maintenance costs (output). The variance $\sigma^2(y)$ captures the random fluctuations around this mean growth rate. For example, due to random variation in food availability or capture success (Iwasa, 1991; Tenhumberg *et al.*, 2000; Fujiwara *et al.*, 2004). Because of this random variation in the growth of individuals, some may experience size decrease over an extended period, even if the mean (population-wise) growth rate is positive (as well as vice versa). If size $y(t)$ falls below a threshold level, denoted by a, the individual dies of starvation. I refer to a as the *starvation boundary*.

In addition to starvation risk, the individual is subject to various exogenous hazards, for example: predation and disease. These are captured by the mortality or hazard rate $\mu(y)$ (also known as killing rate; Karlin & Taylor, 1981, p. 161). Survival probability to some final size b ($b > a$) from an initial size y ($a \le y \le b$), given both risk of starvation and exogenous hazards, will be denoted by $S(y; a, b)$.

Derivations and sections below will use the following two quantities: $\varphi(y) = g(y)/\sigma^2(y)$, and $\rho(y) = \mu(y)/g(y)$. Using these expressions, I find that $S(y; a, b)$ obeys the following differential equation

$$\frac{\mathrm{d}^2 S}{\mathrm{d}y^2} + 2\varphi(y)\frac{\mathrm{d}S}{\mathrm{d}y} - 2\varphi(y)\rho(y)S = 0 \qquad (1)$$

with boundary conditions

$$S(a) = 0, \quad S(b) = 1 \qquad (2)$$

(see Appendix 1). (Note that the signs of φ and ρ are always the same, and depend on the sign of g. So, the product $\varphi\rho = \mu/\sigma^2$ is always non-negative).

Given functions $\varphi(y)$ and $\rho(y)$, I can solve eqn 1 and obtain $S(y; a, b)$. Figure 1 presents some examples. Additionally, Fig. 1a demonstrates that the quantity φ serves to measure how stochastic the growth dynamics is. When $\varphi \to \pm\infty$, the growth process behaves deterministically (e.g. as $\varphi \to +\infty$, $S \to 1$ for all $y > a$; Fig. 1a), whereas, when $\varphi \to 0$, the random fluctuations in growth are the dominant factor, and growth behaves like Brownian motion (i.e. the growth process is an unbiased random walk; in that case, $S(y)$ is linear; Fig. 1a). I note that φ is not dimensionless, as it has units of [size^{-1}].

The quantity $\rho(y)$ captures the exogenous hazard per unit of (mean expected) growth. If the organism may choose among several options (e.g. habitats), each with

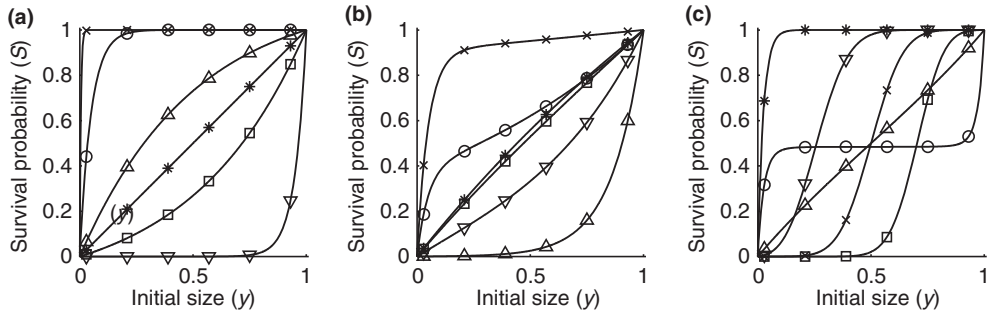

Fig. 1 Examples of survival to size as a function of initial size. In these graphs the size axis has been rendered dimensionless by translation and rescaling, such that the starvation boundary is $a = 0$ and the final target size is $b = 1$. (a) The effect of the value of φ on the form of the survival probability curve ($\rho = 0$ and φ is independent of size for all curves in this panel): $\varphi = 100$ (×); $\varphi = 10$ (○); $\varphi = 1$ (△); $\varphi = 0$ (*); $\varphi = -1$ (□); $\varphi = -10$ (▽). Note that as φ increases (tends towards $+\infty$), the survival curve tends towards $S(y) = 1$ for all $y > a$, i.e. the case of deterministic growth with positive growth rate ($g > 0$). As φ decreases (tends towards $-\infty$), the survival curve tends towards $S(y) = 0$ for all $y < b$, i.e. the case of deterministic growth with negative growth rate. Finally, as φ tends to 0, the survival curve approaches a straight line, i.e. the case of Brownian motion-like growth ($g = 0$, $\sigma^2 \neq 0$). (b) Exogenous hazard has been added (i.e. $\rho \neq 0$): $\rho = 0.1$, $\varphi = 10$ (×); $\rho = 1$, $\varphi = 10$ (○); $\rho = 10$, $\varphi = 10$ (△); $\rho = 0.1$, $\varphi = 0.25$ (*); $\rho = 1$, $\varphi = 0.25$ (□); $\rho = 10$, $\varphi = 0.25$ (▽). Note that differences among survival curves, due to different ρ-values, decrease as the value of φ approaches 0. Thus, as growth dynamics becomes increasingly less deterministic and approaches the limit of Brownian motion (i.e. φ tends to 0), it is starvation mortality (rather than exogenous hazards) that most affects survival to final size. Moreover, note that, for high values of ρ (△ and ▽ in panel b), making the growth dynamics more stochastic (i.e. decreasing the absolute value of φ) increases survival to final size. (c) φ is size dependent ($\rho = 0$ for this panel): $\varphi = 20(2y - 1)$ (×), i.e. increasing linearly from −20 at $y = 0$ to +20 at $y = 1$; $\varphi = 20(1 - 2y)$ (○); $\varphi = 0.5(1 - 2y)$ (△); $\varphi = 20$ (*); $\varphi = 20(2y^2 - 1)$ (□); $\varphi = 20(2y^{0.5} - 1)$ (▽). Compare the form of $S(y)$ when φ is increasing from −20 to +20, with that of φ decreasing from +20 to −20 (× and ○ respectively). Additionally, when the range of φ-values induced by the size dependence is such that φ is never far from zero (△), $S(y)$ is still very much close to the Brownian motion limit (i.e. S linear in y). When $\varphi(y)$ may receive values far from zero, size dependence may cause different $S(y)$ curves, than those attainable by size-independent φ (for example, compare * with ×). Finally, for the three cases of ×, □ and ▽, φ increases from −20 at $y = 0$ to +20 at $y = 1$. However, the specific form of this size dependence of φ clearly has an important effect on the form of $S(y)$, because φ receives values far from zero in all three cases, but goes through $\varphi = 0$ at different values of y.

different expected growth rate $g(y)$ and hazard rate $\mu(y)$, then $\rho = \mu/g$ weighs the costs (in terms of exogenous hazards) vs. the benefits (in term of expected growth rate) for each such option. I note that ρ is exactly the quantity that is minimized in the well-known 'minimize μ/g' rule of Werner & Gilliam (1984), concerning optimal decisions such as niche shifts and timing of metamorphosis (see also Werner, 1988; Ludwig & Rowe, 1990; Houston *et al.*, 1993). This rule applies only when growth is deterministic. For stochastic growth, Houston *et al.*(1993) provide a different expression, that involves the variance in growth as well (see below). ρ also has units of [size^{-1}].

The special case of φ and ρ constants, independent of y, can be solved analytically:

$$S(y; a, b) = \frac{\sinh[\chi(y-a)]}{\sinh[\chi(b-a)]}\exp[\varphi(b-y)] \qquad (3)$$

where

$$\chi = \sqrt{\varphi^2 + 2\varphi\rho} = \sqrt{\frac{g^2}{\sigma^4} + \frac{2\mu}{\sigma^2}} \qquad (4)$$

The quantity χ (eqn 4) combines both starvation risk (decreasing with φ) and exogenous hazards (increasing with ρ), to determine how fast survival probability to b increases with initial size y.

In the following sections, I repeatedly use eqns 3 and 4 (and related expressions, e.g. eqn 9). Therefore, some justification for considering φ and ρ independent of y is required. First, mathematically, this is the simplest case, and therefore useful in illustrating several of the (more general) conclusions I arrive at in following sections. Second, in some biologically relevant cases, growth (g and σ^2) and mortality (μ) may be independent of y (or only weakly dependent on it). For example, if y represents energy reserves (and these have no metabolic maintenance costs as Kooijman, 2000 suggests, p. 90 therein), then the growth and exogenous mortality rates are probably much more sensitive to other variables (e.g. control variables, representing behavioural decisions or additional individual state variables, such as size of structural traits; see below). Third, eqns 3 and 4 are useful also when φ and ρ are piecewise independent of y, for example, as when there is step-like habitat or diet shift that is dependent on the level of energy reserves (represented by y) (see below). Finally, I also consider cases where φ and ρ do depend explicitly on y (e.g. Fig. 2d), so to demonstrate how my general methods and conclusions are applied in more complicated situations.

The maximum process and the hazard density

Survival probability from initial time t_0 to some subsequent time t, i.e. over the time interval $[t_0, t]$, is given by the probability that an individual, alive at time t_0, has a

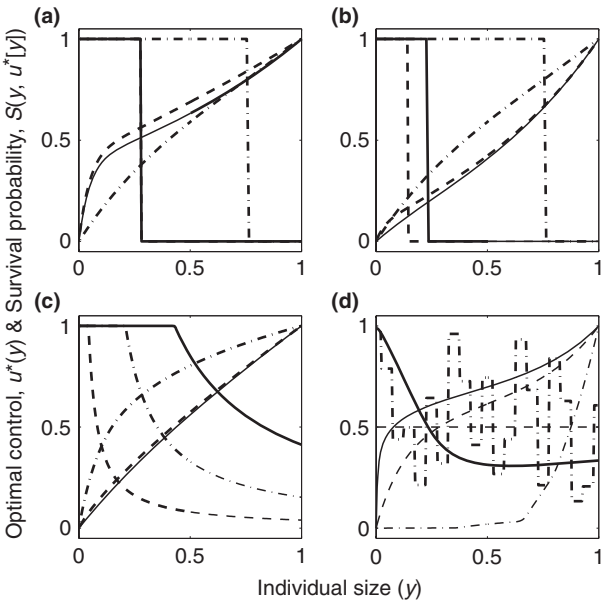

Fig. 2 Optimal control curves, $u^*(y)$ (thick lines), and their respective survival curves $S(y, u^*[y])$ (thin lines). The control variable u is confined to vary between 0 and 1, e.g. representing a fraction of the day spent foraging. (a) $\rho = 1$, $\varphi = 1 + u$ (dash-dotted); $\rho = 1$, $\varphi = 10(1 + u)$ (solid); $\rho = 1$, $\varphi = 1 + 19u$ (dashed). Note that the value of the switching size y^* depends only on the maximal value of φ (e.g. y^* is the same for the two latter cases). (b) Effect of changing the value of ρ on the switching size y^*: $\rho = 1$, $\varphi = 1 + u$ (dash-dotted); $\rho = 4$, $\varphi = 1 + u$ (solid); $\rho = 4$, $\varphi = 1 + 19u$ (dashed). (c) Both φ and ρ are proportional to the control variable u: $\rho = u$, $\varphi = 10u$ (dash-dotted); $\rho = u$, $\varphi = u$ (solid); $\rho = 10u$, $\varphi = 10u$ (dashed). Note that despite the great difference in optimal control, the survival probability curves of the two latter cases are similar. In all three cases, bang–bang control is no longer optimal, and $u^*(y)$ takes intermediate values between 0 and 1. As the hazard density η decreases with increasing size, ρ must be decreased (by decreasing u) so to keep the difference $(\eta - \rho)$ in eqn 10 positive. (d) A model with ρ and φ depending on both the control variable u and size y. Food assimilation rate depends on both searching effort u and handling effort $1 - u$ such that mean growth rate is $g(u, y) = 4u(1 - u) - y$, where the last term represents metabolic maintenance costs proportional to size y. The growth variance is the sum of variance in food assimilation and variance in metabolic maintenance costs: $\sigma^2(u,y) = u(1 - u)^2 + 0.01(0.01 + y^2)$. Mortality rate is proportional to searching effort, and increases at an accelerating manner with size: $\mu(u, y) = u(0.5 + y^2)$. Curves presented in the panel: the optimal control (solid); nonoptimal randomly generated control (dash-dotted); suboptimal control based on maximization of mean expected assimilation: $u(y) = 0.5$ (dashed).

failure time that exceeds t. This can be written as $S(t_0, t) = \Pr\{T > t\}$, where T represents failure time. In a similar fashion, I may define survival to size (i.e. $S[y; a, b]$ of the previous section) as the probability of survival through a size interval $[y_0, b]$ $(b > y_0)$. In Appendix 2, I demonstrate that

$$S(y_0, b) = \Pr\{\theta(T) \geq b | y(t_0) = y_0\} \tag{5}$$

where T is again the failure time and $\theta(t)$ is the maximum process of size (i.e. the maximum process of $y[t]$)

$$\theta(t) = \max_{t_0 \leq \tau \leq t} y(\tau) \tag{6}$$

(The use of the term 'process' in this context is in relation to stochastic processes. That is, $y[t]$ is the stochastic process of size vs. time. Similarly, $\theta(t)$ is also a stochastic process, obtained from $y[t]$ by use of eqn 6.) $\theta(t)$ is a 'memory' that retains the maximal size reached up to time t, and is updated each time the individual reaches a new maximum size. Clearly, $\theta(T)$ (in eqn 5) is the maximum size reached by an individual during its entire lifetime (more accurately, between initial time t_0 and its time of death). I shall return to the maximum process below, in the section concerning structural growth.

Equation 5 defines a survivor function (Kalbfleisch & Prentice, 2002, ch. 1), where the value of the maximum process at failure (i.e. $\theta[T]$) plays the role of failure time. I can now appropriately define a hazard function for survival to size (eqn 5) (analogous to the relationship between survival over time and the hazard rate; Kalbfleisch & Prentice, 2002; Appendix 2). Thus,

$$S(y_0, b) = \exp\left(-\int_{y_0}^{b} \eta(y)\mathrm{d}y\right) \tag{7}$$

where $\eta(y)$ is the hazard function of survival to size. For brevity, I call η *hazard density* (like φ, ρ and χ, it has units of $[\text{size}^{-1}]$). The hazard density describes mortality per unit of increase in size (in a similar manner to mortality/hazard rate, describing mortality per unit of time). As such, the hazard density, $\eta(y)$, summarizes the different ways by which an individual may die, starting from size y, and before reaching size $y + \mathrm{d}y$ (where $\mathrm{d}y$ is small). These include dropping in size all the way to the starvation boundary, or being exposed to external hazards for variable durations, because growth is stochastic (and thus, there are many different growth trajectories the individual may follow from y to $y + \mathrm{d}y$).

When growth is described by a diffusion process (previous section), $\eta(y)$ satisfies

$$\frac{\partial \eta}{\partial y} = -\eta^2 - 2\varphi(\eta - \rho) \tag{8}$$

(see Appendix 2). Equation 8 describes the size dependence of the hazard density. I can gain further insight into this size dependence of $\eta(y)$ by considering the special case of φ and ρ independent of y. Using eqns 3 and 7, I obtain

$$\eta(y) = \chi \coth[\chi(y - a)] - \varphi \tag{9}$$

$(y \geq a)$. For small values of $(y - a)$ eqn 9 becomes $\eta(y) \approx [1/(y - a) - \varphi]$. Thus, for sizes close enough to

the starvation boundary a (as a rule of thumb: $[\chi(y-a)] < 0.5$) the hazard density decreases steeply with increasing size, and goes to infinity as y approaches a. The expression $1/\chi$ determines the range of sizes, above a, in which starvation is the predominant cause of mortality (as opposed to exogenous hazards).

As $(y-a)$ increases, η approaches the limit $\eta_\infty = \chi - \varphi$. Given eqn 4 for χ, this last expression for η_∞ is identical to an expression by Houston *et al.* (1993, their eqn 7). Interestingly, when φ, $\rho > 0$, η_∞ is always lower than ρ, which is the value of η when growth is deterministic (Werner & Gilliam, 1984). Therefore, once the individual is large enough such that starvation no longer poses a threat (as a rule of thumb: $[\chi(y-a)] > 2$), stochasticity in growth decreases the hazard density, i.e. the mortality per unit of size gained, by allowing the individual to achieve higher than average growth rates, and thus reach the final size faster (see also Houston *et al.*, 1993; Houston & McNamara, 1999, p. 123).

Optimal life histories when growth is stochastic

State-dependent life histories

In addition to size (y), φ and ρ may also depend on some control variables, summarized by a vector $\mathbf{u}$, i.e. $\varphi = \varphi(y,\mathbf{u})$ and $\rho = \rho(y,\mathbf{u})$. For example, $\mathbf{u}$ may represent foraging effort or habitat choice. In such cases, the organism may choose $\mathbf{u}$ (e.g. habitat or level of foraging effort) in order to satisfy some optimality criterion. I would like to find the optimal control at each size, i.e. a function $\mathbf{u}(y)$ that maximizes fitness. As a fitness measure, I use $F = S(y_0,b)R(b,y_0)$, where $S(y_0,b)$ is given by eqn 7, and $R(b,y_0)$ represents a terminal reward (Houston & McNamara, 1999, p. 26), gained once the organism survived to the final size b. For example, $R(b,y_0)$ may represent fecundity, which depends on both the adult size (final size b) and the offspring/propagule size (initial size y_0). Maximizing

$$\log F = \log R + \log S = \log[R(y_0,b)] - \int_{y_0}^{b} \eta(y)\mathrm{d}y,$$

and given eqn 8 for the size dependence of η, I obtain the following condition for the optimal control

$$\max_{\mathbf{u}}\{\varphi(y,\mathbf{u})[\eta(y) - \rho(y,\mathbf{u})]\} \qquad (10)$$

As an illustration, consider the case of φ independent on size and ρ constant, i.e. $\varphi = \varphi(\mathbf{u})$, $\rho = \text{const}$. A constant ρ (independent of both y and $\mathbf{u}$) may, for example, describe a situation where both mean growth rate and exogenous mortality rate are proportional to the level of foraging effort (e.g. fraction of a day spent foraging), represented by $\mathbf{u}$. The conclusions below, concerning the optimal control (see next paragraph), extend to situations where ρ depends on y but is independent of $\mathbf{u}$ (or only weakly dependent on the control variables, i.e. on behavioural decisions made by the individual).

Let both φ and ρ be positive for all possible values of $\mathbf{u}$. In that case, at small sizes, close to the starvation boundary a (where $\eta > \rho$), $\varphi(\mathbf{u})$ should be maximized, whereas at larger sizes (where $\eta < \rho$) the optimal control minimizes $\varphi(\mathbf{u})$ (see Fig. 2a). The size, at which the optimal control changes from maximizing φ to minimizing it, is the *switching size*, denoted by y^*.

The interpretation of this 'first maximize then minimize φ' rule may vary among cases, depending on whether it is the mean growth rate g or the growth variance σ^2 that vary with $\mathbf{u}$ (recall that $\varphi = g/\sigma^2$). If $g = \text{const}$ and $\sigma^2 = \sigma^2(\mathbf{u})$, then when size is small ($y < y^*$) one should minimize the growth variance, whereas when size is large enough ($y > y^*$) the growth variance should be maximized. This result has been previously derived by Merad & McNamara (1994) (see also Houston *et al.*, 1993). Once individuals are clear of the range of sizes where starvation risk is high, it is optimal to increase stochasticity of growth. This is a consequence of Jensen's inequality, where the benefit (in terms of survival probability to final size b) of growing above mean rate (due to high stochasticity), outweighs the costs due to the possibility of also growing below that mean rate.

However, if it is the mean growth rate that varies with the control variable, i.e. $g = g(\mathbf{u})$ and $\sigma^2 = \text{const}$, the interpretation of the above 'first maximize then minimize φ' rule is different. The initial maximization of φ is now interpreted as maximizing growth rate close to the starvation boundary, in order to most rapidly 'escape' the size range where starvation probability is high. For larger sizes ($y > y^*$) the minimization of φ takes the interpretation of minimizing mortality rate: $\mu = \rho g(\mathbf{u})$. Therefore, even though these two extreme cases are biologically distinct, the same expression (i.e. eqn 10 with $\varphi = \varphi[\mathbf{u}]$ and $\rho = \text{const}$) defines the optimal control for both, as well as for all intermediate cases where both g and σ^2 vary with $\mathbf{u}$.

Finally, the value of the switching size, y^*, can be found using eqn 9: y^* satisfies $\chi_{\max}\coth[\chi_{\max}(y^* - a)] = \varphi_{\max} + \rho$, where $\varphi_{\max} = \max[\varphi(\mathbf{u})]$ and $\chi_{\max}$ is given by eqn 4 with $\varphi = \varphi_{\max}$. Figure 2a,b demonstrates that y^* decreases as either $\varphi_{\max}$ or ρ increases. Additional examples of optimal control, for cases other than $\varphi = \varphi(\mathbf{u})$ and $\rho = \text{const}$, are also presented in Fig. 2.

Irreversible (structural) growth when reserve dynamics is stochastic

So far, I have considered the state of the individual to be one dimensional, namely a reversible size measure, such as mass of energy reserves. However, much recent work considered the overall size or mass of an individual, to be divided into reversible (e.g. reserves) and irreversible (e.g. structural) components (e.g. Persson *et al.*, 1998; Kooijman *et al.*, 1999; Muller & Nisbet, 2000; Lika &

Kooijman, 2003; Gurney & Nisbet, 2004; van der Meer, 2006). For example, in Kooijman's (2000) dynamic energy budget (DEB) framework, an individual's state is described by its energy reserves (reversible component of mass) and by its structural volume (which grows irreversibly). Next, I consider optimal irreversible structural growth when the energy reserves budget is stochastic.

I denote structural mass by $z(t)$ and reserves mass by $y(t)$. So total mass is given by $z(t) + y(t)$. At this point, I do not commit to any specific functional forms of mean growth rate $g(y,z)$, growth variance $\sigma^2(y,z)$, and mortality $\mu(y, z)$. The maximum process is now defined with respect to total mass, i.e.

$$\theta(t) = \max_{t_0 \le \tau \le t} [y(\tau) + z(\tau)] \tag{11}$$

So $\theta(t)$ is the maximal total mass that an individual have reached up to time t. I note that both $\theta(t)$ and $z(t)$ can only increase over time; never decrease, whereas $y(t)$ is free to either increase or decrease. Because $y(t)$ represents reserves mass, when it drops to a level a, the individual dies of starvation. In the following, I set $a = 0$, i.e. the organism dies only after exhausting all of its energy reserves. A model with $a > 0$ can just as easily be formulated (see previous sections), for example, if some physiological constraint prevents full utilization of reserves.

My model will be an 'assimilation model' (see Kooijman, 2000, p. 365), i.e. assimilated energy and mass is added to reserves, and reserves are then used to fuel metabolic maintenance and structural growth, as well as providing the building materials for the latter. The growth coefficients of the reserves, $g(y, z)$ and $\sigma^2(y, z)$, already summarize both assimilation and metabolic maintenance costs, paid directly by the reserves. In addition, any growth of the structural mass z (e.g. of feeding or defensive structures) is on the expense of reserves y, and not directly from assimilates. An increment dz of structural mass is built by consuming $(1 + \alpha)dz$ mass units of reserves, where α is a non-negative constant parameter representing 'overhead costs' of building structural mass.

Structural mass $z(t)$ grows irreversibly, and thus, like $\theta(t)$, can only increase over time. Moreover, starvation experiments have demonstrated that once feeding is resumed, the original energetic state of individuals is first regained, before any additional structural growth takes place (e.g. Perrin *et al.*, 1990; Kooijman, 2000; Johnsson & Bohlin, 2006). Especially in insects, developmental transitions (moulting and metamorphosis) are dependent upon reaching some critical size thresholds (Nijhout, 2003; Mirth & Riddiford, 2007). Similar developmental thresholds also occur in amphibians. Such thresholds have been advocated as an important (but often missing) component for theories of life history (Day & Rowe, 2002). These observations suggest that the structural growth is associated with attainment of new total mass maxima, i.e. with an increase in the value of $\theta(t)$. For example, after a starvation period, the individual must exceed the previously attained maximum (prior to starvation; most likely, the mass at which starvation began, if individuals were initially fed *ad libitum*), before structural growth can resume.

I shall, thus, connect structural growth with the maximum process of total mass, and assert that structural mass is a nondecreasing function of θ, i.e., $z = z(\theta)$, or $z(t) = z[\theta(t)]$. Whenever the organism reaches a new maximum of total mass, there can be structural growth associated with this attainment of a new maximum. Moreover, between consecutive increases of the maximum mass, i.e. during periods when $\theta(t)$ remains constant, only reserves mass $y(t)$ can change over time. The reserves dynamics is then described by a diffusion process with $g(y, z)$ and $\sigma^2(y, z)$ obtained by keeping z constant at $z = z(\theta)$.

During such periods of no increase in θ, and for a given value of θ, reserves mass $y(t)$ is constrained to vary between 0 and $y_{\max}(\theta, z) = \theta - z$. Therefore, as structural mass increases (for a given value of θ), reserves mass tends to have lower values; thus, starvation risk increases as well. On the other hand, a larger structural mass (for a given value of θ) may translate to higher mean growth rates of reserves or lower mortality rates, thus decreasing starvation risk and/or exogenous hazards. Additionally, reserves mass itself (y) may affect growth rates (g and/or σ^2) or mortality risk (μ). For example, a 'fatter' individual may be less successful in escaping predators, and reserves mass may incur its own metabolic maintenance costs. The problem then boils down to finding the optimal functional form of $z(\theta)$, given all the above considerations.

I derive in (Appendix 3) the following expression for the optimal growth curve of structural mass, $z^*(\theta)$

$$\left[\frac{\partial \eta}{\partial z} - (1+\alpha)\frac{\partial \eta}{\partial y}\right]_{\substack{y=y^*_{\max}(\theta) \\ z=z^*(\theta)}} = 0 \tag{12}$$

where $y^*_{\max}(\theta) = y_{\max}(\theta, z^*[\theta]) = \theta - z^*(\theta)$. Note that η still represents, as before, the hazard density for transitions along the axis of reserves mass, i.e. y-axis (eqns 7–10). However, because growth and mortality also depend on structural mass, η is now a function of both y and z, i.e. $\eta = \eta(y, z)$. Equation 12 states that the optimal structural growth $z^*(\theta)$ is such that the decrease in the hazard density $\eta(y,z)$ because of gain in structural mass, i.e. $(\partial\eta/\partial z)dz$, exactly compensates the increase $-(\partial\eta/\partial y)(1 + \alpha)dz$ due to the associated loss of $(1 + \alpha)dz$ of reserves mass. If the function $\eta(y, z)$ is known, one can find the optimal structural growth graphically, by considering the points where contours of equal η have a slope equal to $-(1 + \alpha)$ (where z is the abscissa and y is the ordinate). Figure 3 provides some examples.

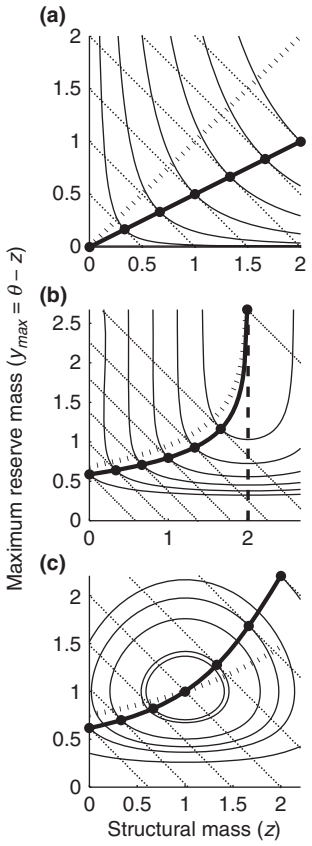

Fig. 3 Optimal growth curve of structural mass for different forms of the hazard density function $\eta(y, z)$. In all three panels, the thick solid line represents the optimal growth curve $y_{max}(z)$ (where $\theta(z) = y_{max}[z] + z$) for $\alpha = 0$, i.e. no overhead cost for building structural mass. The thick dotted line represents optimal growth for $\alpha = 1$, i.e. it takes two mass units of reserves to build one mass unit of structure. The thin solid lines represent contours of the function $\eta(y, z)$. The thin dotted lines represent straight lines of slope $-(1 + \alpha) = -1$ (for $\alpha = 0$) that are tangent to the $\eta(y, z)$ contours at points along the optimal growth curve. (a) η monotonically decreases with both y and z. (b) η monotonically decreases with y but (for a given value of y) has a minimum at $z = 2.01$. This minimum point serves as an asymptotic value of structural mass, approached as $\theta \to \infty$, and never exceeded. (c) η has a minimum point at $y = 1$, $z = 1$. However, if the individual is forced to grow in size beyond this minimum point, then the optimal growth curve represents simultaneous allocation to reserves and structure, along which η increases most slowly (as θ continues to grow beyond its value at $z = 1$, $y_{max} = 1$).

I next consider the special class of cases where φ ($= g/\sigma^2$) and ρ ($= \mu/g$) are functions of structural mass only (i.e. $\varphi = \varphi[z]$ and $\rho = \rho[z]$). For these cases, it is possible to do direct calculations using eqns 3, 4 and 9. For the sake of graphical representations and the following derivations, I define dimensionless variables that naturally arise in the analysis of these cases (see Appendix 3)

$$A = 2(1+\alpha)\frac{\chi^2}{\chi'}, \quad B = \frac{\varphi'}{\chi'}, \quad Y = 2\chi y_{max} \qquad (13)$$

where $\varphi' = d\varphi/dz$, $\chi' = d\chi/dz$ (χ is given by eqn 4). I can rewrite the dimensionless variable A as $A = 2(1 + \alpha)/(-d\chi^{-1}/dz)$, where $\chi^{-1} = 1/\chi$, as discussed previously, determines the range of reserve mass values (above the starvation boundary; here $a = 0$) in which starvation is the predominant cause of mortality. Thus, A summarizes considerations at low reserve mass: on the one hand, the cost of constructing a unit of structural mass, i.e. $1 + \alpha$; on the other hand, the benefit of structural growth in decreasing χ^{-1}, i.e. decreasing susceptibility to starvation mortality.

By contrast, the dimensionless variable B summarizes considerations at high reserve mass, as it represents the ratio of φ' and χ', where the asymptotic hazard density (i.e. η at high values of y) changes with structural growth according to $d\eta_\infty/dz = \chi' - \varphi'$. (For example, if χ' and φ' are both positive, i.e. both increase with structural mass z, then the asymptotic hazard density decreases with z for $B > 1$ and increases for $B < 1$.) Note that the overhead cost of structural mass α appears in A but not in B (eqn 13). That is because this 'extra' loss of reserves due to structural growth is significant only at small reserves masses, where starvation poses a significant risk. Finally, the third dimensionless variable, Y, measures the susceptibility of the individual to starvation mortality, when reserve mass is at its maximum (i.e. $y = y_{max}$). (Based on the rule of thumb discussed previously for $[\chi(y - a)]$, if $Y > 4$ the individual is far enough from the starvation boundary such that $\eta(y_{max}) \approx \eta_\infty$, whereas if $Y < 1$ starvation mortality is very high and $\eta(y_{max}) \approx [1/y_{max} - \varphi]$.)

I then derive (Appendix) from eqn 12 the following expression for the optimal structural growth curve, $z^*(\theta)$

$$f(Y^*) = [Y^* - \sinh(Y^*)] + B[\cosh(Y^*) - 1] = A \qquad (14)$$

(Y^* stands for the dimensionless value of y^*_{max}, i.e. obtained for $z^*[\theta]$.) Based on eqn 14, I find the combinations of A and B values, for which it is optimal to invest in structural growth, rather than just accumulate mass in the form of reserves. I present these in Fig. 4a as the shaded regions in A–B parameter space (see Appendix 3 for explicit expressions). Every choice of the functions $\varphi(z)$ and $\rho(z)$ (and, hence, also $\chi[z]$) projects into an orbit in the A–B parameter space. Figure 4a provides three examples.

Figure 4 also presents some examples of optimal growth curves $z^*(\theta)$, obtained for several forms of $\varphi(z)$ and $\rho(z)$. In Fig. 4b there is no exogenous hazard ($\mu = \rho = 0$; i.e. $\chi = |\varphi|$, and thus, $B = \pm 1$), and φ increases linearly with z from an initially negative value. Increasing the overhead costs of structural growth (i.e. increasing α) causes structural growth to begin later (in terms of θ), and to proceed more slowly. Additionally, increasing the rate at which φ increases with z (i.e. increasing φ')

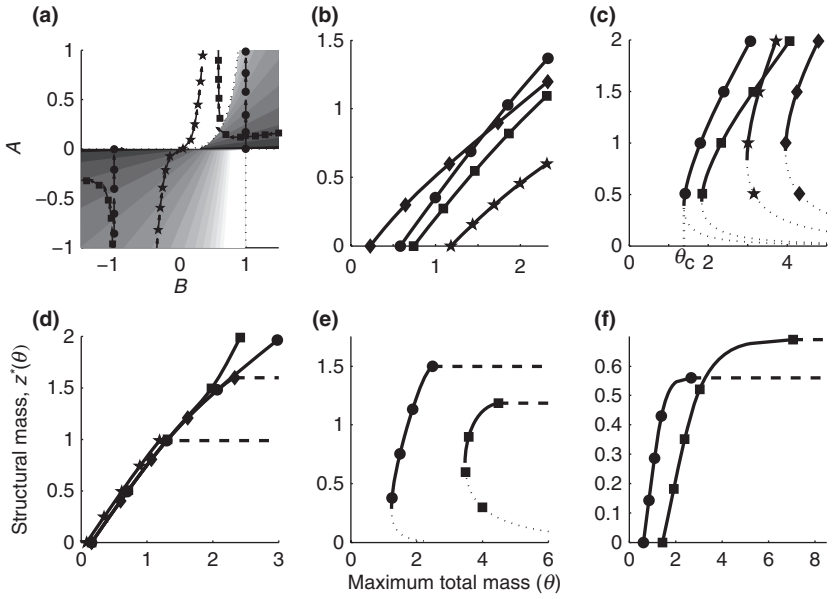

Fig. 4 (a) Shaded areas (bordered by thin dotted lines) represent regions in the *A*–*B* parameter space that admit a (non-negative) solution of eqn 14, i.e. for which it is optimal to invest in structural growth. Level of shading represents the value of Y^* for a given combination of *A* and *B*. Shading varies between $Y^* = 0$ (black) and $Y^* > 3$ (white). Thus, in most cases presented, there is moderate risk of starvation mortality (as discussed in the main text regarding rule of thumb for the value of *Y*). In addition, the *A*–*B* orbits for three choices of the functions $\varphi(z)$ and $\rho(z)$ are drawn (● for all cases of in panels b and c, as well as case ● in panel d; ★ for case ★ in panel d; ■ for case ■ in panel e). One moves along such orbits, as structural mass, *z*, increases. When such an orbit leaves the shaded regions in the *A*–*B* parameter space, structural growth stops and the individual continues only to accumulate reserves mass. Because *A* and *B* depend only on the value of *z* (as φ and χ are functions only of *z*; see eqn 14), the individual gets 'stuck' on the last obtained values of *A* and *B* (and, thus, *z*), and structural growth can never resume. (b) Optimal structural growth curves when $\rho = 0$, i.e. no exogenous hazards, and φ increases linearly with size: $\varphi' = 1.5$, $\alpha = 0$ (●); $\varphi' = 1.5$, $\alpha = 1$ (■); $\varphi' = 1.5$, $\alpha = 9$ (★); $\varphi' = 15$, $\alpha = 0$ (◆). (c) φ increases with *z* in an accelerating manner (proportional to z^2): $\sigma^2 = 1$ (arbitrary units), $\alpha = 0$ (•); $\sigma^2 = 1$, $\alpha = 1$ (■); $\sigma^2 = 10$, $\alpha = 0$ (★); $\sigma^2 = 10$, $\alpha = 1$ (◆). The thin dotted lines represent branches of the optimal growth curves that are unattainable, as *z* decreases with θ along them. The point of growth curve discontinuity is marked by θ_c (marked only for case ●). (d) φ increases linearly with *z*, $\alpha = 0$, and: no exogenous mortality: $\rho = \mu = 0$ (●); constant mortality rate: $\mu = 0.5$ (■); $\rho = \pm 10$ (★); $\rho = \pm 0.1$ (◆). In the latter two cases, ρ is constant, up to a change of sign (because the sign of φ and ρ must always be the same). The thick dashed lines represent the fixed value of structural mass, maintained after structural growth ceases. (e) A foraging-enhancing trait (φ increases with *z* at an accelerating manner) that also incurs a cost in terms of increased susceptibility to exogenous hazards (μ also increases with *z*): low growth variance (●); high growth variance (■); (f) A defensive trait (μ decreases with *z*) that also impairs foraging ability (φ also decreases with *z*): low growth variance (●); high growth variance (■).

causes structural growth to begin earlier but again to proceed at a slower rate. That is because as φ increases (e.g. as mean growth rate increases), starvation mortality becomes increasingly unlikely; so, the marginal value of spending valuable reserves on additional structural growth decreases.

Figure 4c presents an example where φ increases with *z* at an accelerating rate, but at low values of *z* it does so very slowly (i.e. φ' is close to zero at low values of *z*). In such cases, the optimal growth curve $z^*(\theta)$ has two branches. However, because *z* grows irreversibly, only the upper branch (where *z* increases with θ) is attainable. Consequently, as in Fig. 4b, structural growth commences only after some threshold reserves mass is attained (a critical value of θ is reached; denoted by θ_c in Fig. 4c). However, unlike in Fig. 4b, the optimal structural growth curve, in this case, has a discontinuity at $\theta = \theta_c$. That is, once enough reserves have accumulated, there is rapid construction of a large structural mass, whereas the maximum total mass is kept constant (at $\theta = \theta_c$). Only after this initial structural mass has been fully built, does structural mass proceed to grow gradually with θ.

Figure 4c also demonstrates that increasing the overhead cost of constructing structural mass causes structural growth to commence later (in terms of θ; i.e. θ_c increases), but the initial structural mass (i.e. $z[\theta_c^+]$) changes only slightly. By contrast, increasing the growth variance of reserves (i.e. increasing $\sigma^2[z]$ uniformly across all values of *z*), both postpones structural growth and increases the initial structural mass. Uniformly inflating the growth variance is, in this case, equivalent to reducing the rate at which φ increases with *z* (compare with the above discussion of Fig. 4b).

I note that all cases presented in Fig. 4b,c (for which $\mu = \rho = 0$, and thus, $\chi = |\varphi|$) correspond to a single orbit in *A*–*B* parameter space (i.e. $B = -1$ for $A < 0$ and $B = 1$ for $A > 0$; the orbit marked with circles in Fig. 4a). Thus, although describing markedly different biological scenarios, the optimal structural growth curves discussed above all collapse to the same orbit in *A*–*B* parameter space. Moreover, the positive branch of this orbit ($B = 1$ for $A > 0$) corresponds to $\varphi' = \chi'$, i.e. $d\eta_\infty/dz = \chi' - \varphi' = 0$. So, if minimization of the asymptotic hazard density, η_∞, serves as an optimization criterion (as suggested by Houston *et al.*, 1993), structural growth is no longer optimal once φ becomes positive (i.e. B becomes 1). As this study demonstrates, using this criterion would lead to greatly truncated nonoptimal structural growth curves.

Figure 4d–f presents cases with exogenous hazards, i.e. $\rho = \rho(z) \neq 0$. Compare the orbits in *A*–*B* parameter space of two of these cases (squares and stars in Fig. 4a) with the simple orbit for $\mu = \rho = 0$ (previous paragraph; circles in Fig. 4a). These two orbits eventually leave the region in *A*–*B* parameter space that admits non-negative solutions of eqn 14 (i.e. leave the shaded area in Fig. 4a). Therefore, there is in these cases, an optimal final structural mass, beyond which further structural growth is not optimal. Figure 4d demonstrates that an optimal final size exists, for example, when ρ is constant (i.e. when mortality rate μ increases proportionally to mean growth rate g).

In Fig. 4e, φ increases with structural mass in an accelerating manner (as in Fig. 4c); however, mortality rate increases as well. This is a scenario where a foraging-enhancing structure also incurs some costs in terms of increased mortality. In this case, there is a nonzero initial structural mass, which is built once θ exceeds θ_c, as in Fig. 4c. However, unlike in Fig. 4c, there is also a final structural mass, i.e. structural growth eventually ceases, to avoid high mortality rates. Figure 4f describes the optimal growth of a defensive structure that not only reduces mortality rate but also impairs foraging ability (decreases φ). Note that, the effect of increasing the growth variance (of reserves) is opposite in Fig. 4e,f, i.e. final size decreases in Fig. 4e but increases in Fig. 4f.

Discussion

As McNamara *et al.* (2001) pointed out, 'when the animal makes repeated decisions, its level of reserves can be modelled as a diffusion process, with the decisions controlling the mean and variance of this process'. In this study, I developed a diffusion-based model that serves as a framework for investigating optimal growth and life history, with stochastically varying individual state. The strength of the approach is in providing general analytical expressions, thus complementing other, more computer/simulation-based, approaches to stochastic individual-based modelling in evolutionary biology and population dynamics. Different functional forms can then be substituted in the general expressions, in order to study specific cases (as demonstrated in Figs 1–4). Next, I summarize the findings of this paper, and compare them with those of previous studies. I then outline some additional interesting topics in life-history evolution and population dynamics that can be explored using my model.

I determined survival to a final size, as a function of initial size (eqn 1), using two ratios: φ = mean growth rate over growth variance, and ρ = hazard rate over mean growth rate. Size-dependent survival does not depend on these three quantities directly, but only through the ratios φ and ρ. I identified φ as a measure of how deterministic the growth process is (e.g. when $\varphi = 0$, growth resembles Brownian motion). I identified ρ with Gilliam's μ/g criterion for optimal size-dependent life-history decisions (e.g. optimal size at metamorphosis; Werner & Gilliam, 1984). However, minimization of ρ is not the proper optimization criterion when growth is stochastic (Houston *et al.*, 1993; Houston & McNamara, 1999, p. 122), because it does not account for the risk of starvation.

For the purpose of finding an optimization criterion, when growth is stochastic, I defined the hazard density, which is mortality per unit of increase in size, and determined its size dependence (eqns 7–9). As size approaches the starvation boundary, the hazard density tends to infinity, because of starvation mortality. As size grows away from the starvation boundary, the hazard density tends to fall below ρ (the value of the hazard density when growth is deterministic). Thus, stochasticity in growth tends, at large sizes, to decrease the mortality per unit of size, a conclusion also reached by Houston *et al.* (1993).

I found that, in some situations, optimal size-dependent life history maximizes survival to some final size by maximizing φ at small sizes, where starvation risk dominates, and minimizing it at larger sizes, where exogenous hazards are the more important mortality factor. As a special case of this rule, I derived Merad & McNamara's (1994) result, regarding minimization of growth variance at small sizes (or low energy reserves) and maximization of variance at large sizes. In the most general case, I use eqns 9 and 10 to derive the optimal control (see examples in Fig. 2).

Finally, I derive the optimal growth curve for an irreversibly growing trait (e.g. structural volume or mass; *sensu* Kooijman, 2000), when energy reserves grow stochastically (and reversibly). I assert, based on observational and experimental evidence, that structural growth is associated with attainment of new total size maxima. I find an expression for the optimal investment of accumulated reserves into structural growth, given both exogenous hazards and risk of starvation (eqn 12). Conclusions that arise from examining specific cases (Fig. 4) are: (1) there is usually an initial delay in the

growth of a structural trait, because at small sizes, reserves are more important, in order to avoid starvation. (2) Depending on its function, there may also be an initial discontinuity in the growth of a structural trait. That is, once the individual has accumulated enough reserves to begin optimally investing in structural growth, it should initially build a relatively large structure (see Fig. 4c,e). (3) Increasing the growth variance of reserves increases the initial delay in structural growth and raises the initial discontinuity (e.g. Fig 4b,c). (4) Increasing the growth variance also has a large effect on optimal final sizes of a structure (see Fig. 4c,e,f). Interestingly, it may either increase or decrease the optimal final size of a structural trait, depending on the function of that trait (i.e. foraging-related or defensive trait; compare Fig. 4e with Fig. 4f).

I note that Clark & Mangel (2000, pp. 61–67) presented a dynamic state variable model that also includes dynamics of both reserves and structural mass (the latter growing irreversibly). They used specific functional forms for probabilities of finding food and survival between consecutive time steps (both increasing with structural mass). Clark and Mangel concluded that, as food supply becomes increasingly variable, instances of structural growth occur more rarely. Their conclusion is partly similar to my conclusion (3) of the previous paragraph, concerning the increased delay in structural growth (e.g. compare with their fig. 2.3). However, Fig. 4c,f indicates that once structural growth commences, it may proceed as fast, or even faster, when the growth variance of reserves is increased. In other cases, increasing the growth variance indeed slows down structural growth (e.g. compare with Fig. 4e). Therefore, I demonstrate that the effect of increasing the variance in growth on the rate of structural growth depends on the specific functional forms of the structural trait dependence of growth and mortality.

The above conclusions can readily be translated into predictions for experimental and comparative studies. For example, the effect of increased variance in growth on optimal structural growth is particularly interesting. Closely related species, or geographically distinct populations of the same species, may experience different levels of noise in resource intake, growth efficiency (e.g. due to short-term variations in food quality) or metabolic maintenance costs. This study provides predictions on how patterns of structural growth should vary between such populations or species. Moreover, it may be possible to examine in experimental settings the effect of different levels of growth variance on the growth of reserves and structural traits, e.g. in experiments of artificial selection or phenotypic plasticity. The level of noise in growth modulates the trade-off between energy gain and avoiding exogenous hazards (e.g. see eqn 4), and thus, depending on whether a structural trait is related to foraging or defence, my model predicts different effects of increased growth variance (e.g. Figs 4e,f).

Irie & Iwasa (2005) studied optimal shell growth in molluscs, i.e. a purely defensive structure. Their model is deterministic, as it does not include noise in the growth process. An initial delay in structural growth also occurs in their model, as the individual initially invests only in the growth of the soft body, in order to increase production rate. In my model, the initial delay in the growth of a defensive structure (Fig. 4f) occurs because initially the individual should accumulate reserves, as insurance against starvation mortality. (In Fig. 4f I use the same functional form for structural mass-dependent mortality rate, as in Irie & Iwasa, 2005.)

Irie & Iwasa (2005) also discussed other modes of defence, such as chemical defence (e.g. production of toxins; e.g. Longson & Joss, 2006) or defensive behaviour (e.g. increased vigilance; Brown, 1999). They noted that their model is not applicable to such defences, because it assumes that the effect of investment in defence is cumulative. Unlike shells, which are irreversibly growing defensive structures, chemical or behavioural defences are reversible, in the sense that they can be switched on and off without a lasting effect on the individual. In my model, it is straightforward to incorporate such reversible defences, together with the irreversibly growing structural/physical defence, by considering additional control variables (affecting mean growth rate, growth variance and mortality rate). The reversible modes of defence can be switched on and off as reserves mass fluctuates, resulting in optimal control that is a function of instantaneous reserves mass, as I studied above (e.g. Fig. 2). In general, this optimal control will also depend on structural mass (i.e. $u^* = u^*[y, z]$). Similarly, the reversibly changing and irreversibly growing traits can also be related to foraging, e.g. feeding structures, growing irreversibly and habitat preference, diet choice or foraging effort, serving as reversible behavioural traits.

Energy allocation to reproduction is another behaviour that the individual can reversibly switch on and off depending on its nutritional state. In this work, however, I only considered survival while growing from an initial size (i.e. offspring size) to some final size (i.e. adult size). In that respect, the current model aims mainly at immature or nonreproducing individuals. Reproduction was summarized by a terminal reward, a function of both initial size and final size. This terminal reward function can be used to derive optimal initial and final sizes, as Kozlowski (1996) obtained for a deterministic life-history model. Using my stochastic model, I can additionally investigate how optimal offspring and adult sizes depend, for example, on the growth variance.

In addition to an explicit treatment of reproduction, another important component of dynamic optimization, missing in the current model, is an explicit treatment of time, for example, the effect of a final time horizon. This can be incorporated, for example, by using a time penalty (Houston *et al.*, 1993). Another possibility is to use the backward Kolmogorov equation (Karlin & Taylor, 1981,

p. 216), as demonstrated by Iwasa (1991) in the context of an environment that fluctuates stochastically over time.

Fluctuations in the environment over time (e.g. environmental stochasticity, in contrast to demographic stochasticity; see below) has also been the focus of most optimization models, concerned with simultaneous investment in both structure and reserves (also called storage, or storage organs; e.g. Perrin & Sibly, 1993; Iwasa & Kubo, 1997). In such models, however, the growth of individuals is deterministic in the sense that there is only variability in growth rate over time, not individual variability. That is, identical individual, having the same state (e.g. size) at a given time, have exactly the same growth rate.

The stochasticity in growth considered in this study, however, is demographic. That is, 'because of differences in luck in obtaining food, avoiding predators, etc.' (Houston & McNamara, 1999, p. 220). Demographic stochasticity can further be divided into demographic stochasticity proper, i.e. sampling variance in individual fate (associated with a given value of a demographic trait) and demographic heterogeneity, i.e. individual variability in the values of demographic traits (Kendall & Fox, 2002, 2003; Melbourne & Hastings, 2008). In this study, randomly generated variation in growth rate among identical individuals (i.e. growth variance) has important effects on how survival probability varies with size, as demonstrated by Fig. 1. This has important consequences to population stability, as I next demonstrate.

Consider a simple scenario of no exogenous mortality ($\mu = \rho = 0$) and an environment that fluctuates stochastically between a positive value of mean growth rate (e.g. a year with abundant resource) and a negative such value (e.g. a year with scarce resource). When growth variance is small survival probability in 'good years' is close to 1, whereas in 'bad years' it is very low (see Fig. 1a; compare $\varphi = 10$ and -10; recall that $\varphi = g/\sigma^2$). Thus, there are large fluctuations in survival probability among years. On the other hand, when growth variance is high, between-year fluctuations in survival probability are greatly reduced (compare $\varphi = 1$ and -1 in Fig. 1a).

Interestingly and counter intuitively, growth variance, although considered a component of demographic stochasticity (e.g. generated because of differences in luck in obtaining food), has a stabilizing effect on environmentally driven fluctuations in population dynamics. This stabilizing effect is different from the one attributed to individual variation in demographic traits [e.g. (size-related) variation in survival and fecundity; Bjørnstad & Hansen, 1994; Grimm & Uchmanski, 2002; Filin & Ovadia, 2007], as it occurs even in the absence of such variation. However, a similarity still exists in the sense that both an initial structure in the population (which can become exaggerated with time) and randomly generated variability in an initially homogeneous population, increase the within-generation variability in individual fate, but decrease the between-generation variability in the average fate (see also Uchmanski, 2000).

In conclusion, the present paper along with many recently published empirical and theoretical studies emphasizes the need to document individual variability in growth, and not only mean responses, in experimental and field work (e.g. Pfister & Stevens, 2002; Gurney & Veitch, 2007). In addition, it is important to distinguish between different components of the size or mass of individuals (e.g. reserves and structure; Kooijman, 2000). As this work clearly demonstrates, these two aspects of the growth of individuals have important implications to life history and population dynamics, and need to be better addressed both by experimentalists and theoreticians.

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Appendix 1: Derivation of eqns 1 and 2

Survival probability to b, given starvation boundary a ($< b$) and initial size y ($a \le y \le b$) can be written as $S(y) = u(y)P(y)$. The first factor in the product is $u(y)$ = Pr{the growth process reaches b before a}, i.e. the probability that a sample path of the growth process crosses level b before crossing level a, starting from y. The second factor is $P(y)$ = Pr{no exogenous mortality event before reaching b, given that b is reached before a}, i.e. the probability that a sample path is not killed before reaching level b, prescribed to the process confined to sample paths that reach b before a. Thus, the expression $S(y) = u(y)P(y)$ is a simple consequence of the definition of conditional probability. (Note that any exogenous mortality event, along sample paths of the growth process that reach a before b, is irrelevant as far as survivorship is concerned, because any individual following this sample path is destined to die before reaching level b, either by exogenous hazards or by starvation.)

The growth process is a diffusion process with infinitesimal mean and variance given by $g(y)$ and $\sigma^2(y)$ respectively. The probability $u(y)$ of reaching level b before crossing a different level a is given for time homogenous diffusion by

$$\frac{1}{2}\sigma^2(y)\frac{d^2u}{dy^2} + g(y)\frac{du}{dy} = 0 \qquad \text{(A1)}$$

$$u(a) = 0, \quad u(b) = 1 \qquad \text{(A2)}$$

(Karlin & Taylor, 1981, pp. 192–193). $u(y)$ is in fact a *scale function* of the (growth) diffusion process (Karlin & Taylor, 1981, pp. 194–196).

The second probability $P(y)$ is given by the Kac functional (eqns 3.41 and 3.42 in Karlin & Taylor, 1981, p. 204). However, because I confine myself in this calculation only to those sample paths (of the growth process) that reach b before a, I must use the respective conditioned diffusion process in obtaining an expression for $P(y)$. All sample paths of the growth process that reach b before a describe a new (conditioned) diffusion process with the following infinitesimal mean and variance

$$g^*(y) = g(y) + \frac{u'(y)}{u(y)}\sigma^2(y) \qquad \text{(A3)}$$

$$\sigma^{2*}(y) = \sigma^2(y) \qquad \text{(A4)}$$

(Karlin & Taylor, 1981, pp. 261–264). With the addition of the exogenous hazard (killing) rate, $\mu(y)$, I obtain the following expression for the probability $P(y)$

$$\frac{1}{2}\sigma^{2*}(y)\frac{d^2P}{dy^2} + g^*(y)\frac{dP}{dy} - \mu(y)P = 0 \qquad \text{(A5)}$$

$$P(a) = 1, \quad P(b) = 1 \qquad \text{(A6)}$$

(Karlin & Taylor, 1981, p. 204). Note that although $P(a) = 1$ is a boundary condition of eqn A5, the state $y = a$ becomes an entrance boundary, and thus unattainable from within the interval $(a, b]$, for the conditioned diffusion. So, the conditioned process can only display absorption at b; see Karlin & Taylor, 1981, pp. 226–250 and 263–264).

The total survival probability to b, given the original diffusion process is $S(y) = u(y)P(y)$ = Pr{reaching b before a}Pr{the conditioned process is not killed before reaching b}. Combining eqns A1–A6, I obtain z

$$\frac{1}{2}\sigma^2(y)u\frac{d^2P}{dy^2} + [g(y)u + \sigma^2(y)u']\frac{dP}{dy} - \mu(y)uP + P\left[\frac{1}{2}\sigma^2(y)\frac{d^2u}{dy^2} + g(y)\frac{du}{dy}\right] = 0$$

$$S(a) = u(a)P(a) = 0, \quad S(b) = u(b)P(b) = 1 \qquad \text{(A7)}$$

Rewriting eqn A7 as

$$\frac{1}{2}\sigma^2(y)(uP'' + 2u'P' + u''P) + g(y)(u' + uP') - \mu(y)uP = 0$$

and dividing by $\sigma^2/2$, I finally obtain eqns 1 and 2.

Appendix 2: Survival to size, the maximum process of growth, and the hazard density

As a useful illustration, consider a cohort of identical individuals all starting at an initial size y_0 at time t_0, but diverging in subsequent growth trajectories. Individual i has failure time (i.e. time of death) given by T_i and, thus, size at death $Y_i = y(T_i)$. By definition, $S(t_0, t)$ = Pr{$T_i > t$} (Kalbfleisch & Prentice, 2002, ch. 1). However, can I analogously define $S(y_0, b)$ as Pr{$Y_i > b$}? Next, I demonstrate that such a definition is problematic and suggest an alternative definition.

First, although an individual never exceeds T_i during its lifetime, it may exceed Y_i if it grows to a larger size before eventually decreasing to Y_i. Second, consider two individuals, both starting at y_0 and dying at size y_1 (i.e. $Y_i = y_1$ for both). Although one individual grows monotonically from y_0 to y_1, where it eventually dies, the second individual passes through $y_2 > y_1$. Naturally, I would claim that the second individual contributes to survival $S(y_0, y_2)$ from initial size y_0 to subsequent size y_2, even though later its size decreases below y_2, but not so in the first individual that never reaches y_2 during its lifetime. However, if I define $S(y_0, y_2)$ = Pr{$Y_i > y_2$}, both individuals do not contribute to survival to size y_2.

I can fix the definition of $S(y_0, b)$, by defining the maximum process

$$\theta(t) = \max_{t_0 \le \tau \le t} y(\tau)$$

(see eqn 6 in the main text). If I now define

$$S(y_0, b) = \Pr\{\theta(T) \ge b | y(t_0) = y_0\}$$

(seeeqn 5 in the main text) I observe that the two problems presented in the previous paragraph no longer plague this definition. First, $\theta(t)$ cannot decrease over time (unlike $y[t]$). Second, our two hypothetical individuals are now distinguishable based on their final values of the maximum process: $\theta(T_1) = y_1 < y_2 \le \theta(T_2)$.

Because a hazard function is an alternative representation of a survivorship function (Kalbfleisch & Prentice, 2002, p. 7), I can define a hazard function for survival to size $S(y_0, b)$ in a manner analogous to the hazard rate, which is the hazard function of survival to time. Thus,

$$S(y_0, b) = \Pr\{\theta(T_i) \ge b\} = \exp\left(-\int_{y_0}^{b} \eta(y)\mathrm{d}y\right)$$

(see eqn 7 in the main text) where $\eta(y)$ is the *hazard density*, i.e. the hazard function of $S(y_0, b)$. Similarly to the hazard rate, $\eta(b)\mathrm{d}b$ is the increment in mortality as I slightly raise the survival threshold from b to $b + \mathrm{d}b$, taking into account all the possible different growth trajectories that reach b but do not reach $b + \mathrm{d}b$, i.e. $S(y_0, b + \mathrm{d}b) \approx S(y_0, b)[1 - \eta(b)\mathrm{d}b]$. Similarly, $S(y_0, b) \approx S(y_0 + \mathrm{d}y_0, b)[1 - \eta(y_0)\mathrm{d}y_0]$. When growth is deterministic $\eta(y) = \mu(y)/g(y) = \rho(y)$.

When growth is described by a diffusion process, I obtain from eqn 1

$$S\left[\frac{\mathrm{d}^2 \ln S}{\mathrm{d}y^2} + \left(\frac{\mathrm{d}\ln S}{\mathrm{d}y}\right)^2 + 2\varphi(y)\frac{\mathrm{d}\ln S}{\mathrm{d}y} - 2\varphi(y)\rho(y)\right] = 0$$

and given that $\eta(y) = \partial \ln S(y,b)/\partial y$ (eqn 7), and b is a constant parameter representing final size, I obtain eqn 8.

Appendix 3: Optimal irreversible structural growth

My criterion for optimality would be maximizing survival from initial total mass $\theta_\mathrm{I} = z_\mathrm{I} + y_\mathrm{I}$ to final total mass θ_F, i.e. $S(\theta_0, \theta_\mathrm{F})$. As before, $S = \exp(-\Lambda)$, where

$$\Lambda(\theta_\mathrm{I}, \theta_\mathrm{F}) = \int_{\theta_\mathrm{I}}^{\theta_\mathrm{F}} h(\theta)\mathrm{d}\theta$$

is the *cumulative hazard*. However, note that now the hazard density, denoted here by h, is a function of the maximum of total mass process, θ (eqn 11). In the following, $h(\theta)$ refers to the hazard density for transitions from one value of θ to a higher value of θ, whereas $\eta(y)$, as before, refers to transitions along the axis of reserves mass, i.e. y-axis (see eqns 7–10). In general, both may also depend on the value of the structural mass z. So I can also denote these hazard densities by $h(\theta, z)$ and $\eta(y, z)$ respectively.

Thus, survival probability from total mass θ to total mass $\theta + \mathrm{d}\theta$ (where $\mathrm{d}\theta$ is sufficiently small) is given by $S(\theta, \theta + \mathrm{d}\theta) = 1 - h(\theta)\mathrm{d}\theta + o([\mathrm{d}\theta]^2)$. I wish to find an expression for $h(\theta)$, given the specification of mortality and growth (of both y and z) in the main text.

Given a value of θ and its corresponding value of $z = z[\theta]$, reversible mass y changes according to a diffusion process with $g = g(z, y)$ and $\sigma^2 = \sigma^2(z, y)$. As long as $y(t)$ remains within the interval $(0, y_{\max}(\theta, z)]$ (where $y_{\max}[\theta, z] = \theta - z$), the individual does not starve to death, and there is also no additional structural growth. However, once $y(t)$ exceeds $y_{\max}$, a new maximum total mass θ is obtained and structural mass z may also increase. Let us assert that structural growth occurs only at evenly spaced points along the θ-axis, with intervals of size $\mathrm{d}\theta$. That is, if the last structural growth increment occurred at $\theta(t_1) = \theta_1$, then z will remain unchanged at least until $\theta(t_2) = \theta_2 = \theta_1 + \mathrm{d}\theta$, where the next structural growth increment may occur. The next structural growth after t_2 occurs at $\theta(t_3) = \theta_3 = \theta_2 + \mathrm{d}\theta = \theta_1 + 2\,\mathrm{d}\theta$, and so forth.

Immediately before t_2, I have:

$$y(t_2^-) = \theta_2 - z(\theta_1) = y_{\max}(\theta_1, z[\theta_1]) + \mathrm{d}\theta \quad \text{and} \quad z(t_2^-) = z(\theta_1).$$

Immediately after t_2, I have:

$$y(t_2^+) = y_0(\theta_2) \quad \text{and} \quad z(t_2^+) = z(\theta_2) = z(\theta_2) + \mathrm{d}z,$$

where $y_0(\theta)$ represents the initial amount of reserves immediately after a structural growth increment occurred, given the value of θ. If growth increments occur at small enough intervals (i.e. $\mathrm{d}\theta$ is small), then $\mathrm{d}z = (\mathrm{d}z/\mathrm{d}\theta)|_\theta\,\mathrm{d}\theta + o([\mathrm{d}\theta]^2)$, where the derivative $(\mathrm{d}z/\mathrm{d}\theta)|_\theta$ is evaluated at $\theta = \theta_1$.

Given this formulation, I can derive the following expression for survival from θ to $\theta + \mathrm{d}\theta$

$$S(\theta, \theta + \mathrm{d}\theta) = S(y = y_0[\theta], y = y_{\max}[\theta, z(\theta)] + \mathrm{d}\theta)$$

or given $S = \exp(-\Lambda)$

$$\Lambda(\theta, \theta + \mathrm{d}\theta) = -\ln S(y = y_0[\theta], y = y_{\max}[\theta, z(\theta)] + \mathrm{d}\theta) \quad \text{(A8)}$$

The initial amount of reserves immediately after a structural growth increment, $y_0(\theta)$, can be found if I consider that in order to produce mass $\mathrm{d}z$ of structure one requires $(1 + \alpha)\mathrm{d}z$ of reserves. Therefore $y_0(\theta) = y_{\max}(\theta, z[\theta]) - \alpha\,\mathrm{d}z$ (where the 1 in $(1 + \alpha)$ is already absorbed in $y_{\max}$). Thus, eqn A8 can now take the form

$$\Lambda(\theta, \theta + \mathrm{d}\theta) = -\ln S(y = y_{\max}[\theta, z(\theta)] - \alpha\,\mathrm{d}z, y = y_{\max}[\theta, z(\theta)] + \mathrm{d}\theta)$$

and finally I obtain (when taking the limit $\mathrm{d}\theta \to 0$)

$$h(\theta) = \eta(y = y_{\max}[\theta, z(\theta)])\left(1 + \alpha \left.\frac{dz}{d\theta}\right|_{\theta}\right) \qquad (A9)$$

Notice that the hazard density for transitions along the θ-axis (eqn A9) is inflated by the rate of structural growth ($dz/d\theta$), if there are overhead costs for building structural mass (i.e. if $\alpha > 0$). That is because after each increment of structural growth, the individual needs to spend extra time in regaining reserves mass, before any additional structural growth may occur.

Once I obtained an expression for $h(\theta)$ I wish to find the optimal functional form of $z(\theta)$. For that purpose, I use the Pontryagin's maximum principle. My state variable is z, and let u denote the control variable: $u = dz/d\theta$. Then, the Hamiltonian is given by $H = -h + u\lambda_z$ (the minus sign in front of h is because I wish to maximize survival or, equivalently, minimize the cumulative hazard Λ), i.e.

$$H = -\eta(y_{\max}[\theta, z])(1 + \alpha u) + \lambda_z u$$

where λ_z is the co-state variable of z. The switching function for investing in structural mass is given by $\Sigma = \partial H/\partial u$, or

$$\Sigma = -\alpha\eta(y_{\max}[\theta, z]) + \lambda_z.$$

When $\Sigma = 0$, I obtain singular control, which should provide us with an expression for the optimal growth curve $z^*(\theta)$. A singular control additionally requires $d\Sigma/d\theta = 0$, which results in the following condition (given $d\lambda_z/d\theta = -\partial H/\partial z$)

$$\frac{d\Sigma}{d\theta} = \left[\frac{\partial}{\partial z} - \alpha\frac{\partial}{\partial \theta}\right]\eta(y_{\max}[\theta, z]) = 0 \qquad (A10)$$

which is identical to eqn 12. (Recall that $\eta[y_{\max}] = \eta[y, z]$ for $y = \theta - z$. So, given the values of θ and z,

$$\begin{aligned}\partial\eta[y_{\max}]/\partial\theta &= \partial\eta[y, z]/\partial y \quad \text{and} \quad \partial\eta[y_{\max}]/\partial z \\ &= -\partial\eta[y, z]/\partial y + \partial\eta[y, z]/\partial z,\end{aligned}$$

all evaluated at z and $y = y_{\max} = \theta - z$.)

Hereafter, I shall only refer to the special case of φ and ρ independent of y, i.e. η is given by eqn 9, where φ and χ are functions of z. Equation 12 then becomes

$$\begin{aligned}\chi' \coth(\chi y_{\max}) - \chi\chi' y_{\max}\ \mathrm{cosech}^2(\chi y_{\max}) - \varphi' \\ + (1 + \alpha)\chi^2\ \mathrm{cosech}^2(\chi y_{\max}) = 0\end{aligned}$$

where $\varphi' = d\varphi/dz$, $\chi' = d\chi/dz$. Multiplying by $-2\sinh^2(\chi y_{\max})$ I obtain

$$\begin{aligned}\chi'[(2\chi y_{\max}) - \sinh(2\chi y_{\max})] + \varphi'[\cosh(2\chi y_{\max}) - 1] \\ = 2(1 + \alpha)\chi^2\end{aligned}$$

and from this eqn 14 is obtained by using expressions 13 for A, B and Y. Note that $f[Y]$ in eqn 14 is the sum of an odd function and an even function.

Using eqn 14, and the definitions for the dimensionless variables (eqn 13), I find that a singular control solution $Y^* > 0$ exists if:

(a) $A > 0$ (i.e. $\chi > 0$ and $\chi' > 0$) and $B \geq 1$;
(b) $A < 0$ (i.e. $\chi > 0$ and $\chi' < 0$) and $B < 1$;
(c) $0 < B < 1$ and $0 < A < [\ln(1 + B) - \ln(1 - B) - 2B]$.

(Note that in this case, eqn 14 admits two positive solutions of Y^*. However, only the smaller one is optimal. I conclude that using the second-order condition for the local maximality of singular control; see below).

Additionally:

(d) if $A = 0$ (i.e. $\chi = 0$), then $y^*_{\max} = \theta - z^*(\theta) = [(1 + \alpha)/\varphi']^{1/2}$;
(e) if $\chi' = 0$, then $Y^* = a\cosh(1 + A/B) = a\cosh\{1 + [2(1 + \alpha)\chi^2/\varphi']\}$.

In both (d) and (e) a singular control solution exists only if $\varphi' > 0$. These can be rewritten in terms of the forms of the functions $\chi(z)$ and $\varphi(z)$:

(I) $\chi > 0$ and $\varphi' > \chi'$ [from (a), (b) and (e)]; or
(II) $\chi > 0$ and $\varphi' = \chi' > 0$ [from (a)]; or
(III) $\chi = 0$ and $\varphi' > 0$ [from (d)]; or
(IV) $\chi' > \varphi' > 0$ (i.e. $0 < B < 1$) and $0 < A < [\ln(1 + B) - \ln(1 - B) - 2B]$ [from (c)].

The second-order condition for the local maximality of singular control is given by

$$\frac{\partial}{\partial u}\left(\frac{d^2}{d\theta^2}\right)\frac{\partial H}{\partial u} > 0$$

(e.g. Iwasa & Roughgarden, 1984; eqn 18 therein). By substituting $\Sigma = \partial H/\partial u$, and using eqn A10 I can obtain from this condition the following one:

$$\frac{\partial}{\partial z}\left(\frac{d\Sigma}{d\theta}\right) > 0$$

By noticing that for singular control $d^2\Sigma/d\theta^2 = [\partial/\partial\theta + u\,\partial/\partial z](d\Sigma/d\theta) = 0$ and $u > 0$, I now obtain the following inequality

$$\frac{\partial}{\partial \theta}\left(\frac{d\Sigma}{d\theta}\right) < 0$$

And finally, for the case of φ and ρ dependent only on z, I obtain the condition

$$\chi'\chi\ \mathrm{cosech}^2\left(\frac{1}{2}Y^*\right)f'(Y^*) > 0 \qquad (A11)$$

for local maximality of the singular control described by eqn 14.

Condition (A11) can be summarized as follows:

(i) $f'(Y^*) > 0$ for $A > 0$ (i.e. for $\chi > 0$ and $\chi' > 0$),
(ii) $f'(Y^*) < 0$ for $A < 0$ (i.e. for $\chi > 0$ and $\chi' < 0$),
(iii) $\varphi' > 0$ for $\chi = 0$ and/or $\chi' = 0$).

In conjunction with conditions (a)–(e) for the existence of a singular control, I finally conclude that whenever a singular control exists it is optimal.

Condition A11 also helps to determine that in case (c), which admits two solutions of eqn 14, it is only the smaller solution that is optimal. I note that case (c) also admits a singular control solution $Y^* > 0$ when $A = [\ln(1 + B) - \ln(1 - B) - 2B]$. However, condition (i) is not satisfied in this case. Finally, if the initial structural mass z_I and reserves mass y_I lie outside the singular control curve (eqn 12), then the switching function Σ is not zero, and the individual should grow in such a manner to eventually hit the singular control curve, and proceed along it.

If $\Sigma < 0$ the optimal strategy is $u^*(\theta) = 0$, i.e. no investment in structural mass as total mass θ increases. This case corresponds to initial reserves below the level prescribed by the singular control for a given value of structural mass z_I. In this case, the individual only accumulates reserves. Conversely, when $\Sigma > 0$ the Hamiltonian is maximized by having $u^*(\theta) = dz/d\theta = +\infty$, that is an initial step in $z(\theta)$. This case corresponds to an initial structural mass below that prescribed by the singular control for the value of θ_I ($=z_I + y_I$). Thus, there is a transient phase where structural mass grows without increasing total mass (i.e. $\theta(t) = \theta_I$), until the singular control curve is hit at the point (θ_I, $z^*[\theta_I]$). (in θ–z space; as in Fig. 4b–f).

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