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# Target size and optimal life history when individual growth and energy budget are stochastic

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ABSTRACT

I extend my previous work on life history optimization when body mass is divided into reserves and structure components. Two important innovations are: (1) effect of finite target size on optimal structural growth; (2) incorporating reproduction in the optimization objective. I derive optimal growth trajectories and life histories, given that the individual is subject to both starvation mortality and exogenous hazards (e.g., predation). Because of overhead costs in building structural mass, it is optimal to stop structural growth close to the target size, and to proceed only by accumulating reserves. Higher overhead costs cause earlier cessation of structural growth and smaller final structures. Semelparous reproduction also promotes early cessation of structural growth, compared to when only survival to target size is maximized. In contrast, iteroparous reproduction can prolong structural growth, resulting in larger final structures than in either the survival or the semelparous scenarios. Increasing the noise in individual growth lowers final structural mass at small target sizes, but the effect is reversed for large target sizes. My results provide predictions for comparative studies. I outline important consequences of my results to additional important evolutionary questions: evolution of sexual dimorphism, optimization of clutch size and evolution of progeny and adult sizes.



## 1. Introduction

Life history and developmental transitions often require the attainment of some target size. For example, "in a variety of animals and plant species adult size appears to be determined by a size threshold for maturation" (Roff, 2002, p. 201). Developmental transitions in insects and amphibians are dependent upon reaching some critical size (Day and Rowe, 2002; Nijhout, 2003; Mirth and Riddiford, 2007). More generally, such thresholds play a key role in recent approaches to developmental plasticity and phenotypic evolution (West-Eberhard, 2003).

Thus, target size is an important life history variable, and should affect optimal life history decisions during growth and development of an individual (e.g., Day and Rowe, 2002). In this paper, I study the effect of a given finite target size on optimal life history, when individual growth is stochastic. I employ a dynamic optimization approach (e.g., Perrin and Sibly, 1993; Iwasa, 2000; Irie and Iwasa, 2005), and extend my previous work (Filin, 2009), by considering not only survival, but also reproduction, as part of the optimization objective.

Following much recent work on size-structured populations and individual growth (e.g., Persson et al., 1998; Kooijman, 2000; Gurney and Nisbet, 2004; Filin, 2009), I divide the total body mass of an individual into a reversibly growing component (hereafter, reserves) and an irreversible component (hereafter, structure). The mass of reserves varies stochastically in time, for example, because of fluctuations in consumption, assimilation and metabolic maintenance. I explore how costs of structural growth, noise level in reserves dynamics, and mode of reproduction (e.g., semelparous or iteroparous), all affect optimal investment in structure versus reserves, when the individual is subject to both starvation risk and exogenous mortality. Finally, I discuss how my model and results can be easily applied to a wealth of additional evolutionary problems: evolution of sexual dimorphism, optimal clutch size, and optimal progeny and adult sizes.

## 2. Effect of target size on life history optimization

### 2.1. Basic formulations

I denote structural mass by $z(t)$ and reserves mass by $y(t)$. Total body mass is then $z(t)+y(t)$. I define the time-varying maximum of total body mass

$$\theta(t)=\max_{t_0\le\tau\le t}[y(\tau)+z(\tau)] \quad (1)$$

i.e., the maximal total mass reached up to time $t$. (Below, I interchangeably refer to $\theta$ as total body mass, total mass, or body

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mass.) Both $\theta(t)$ and $z(t)$ grow irreversibly, i.e., can only increase through time, while $y(t)$ is free to either increase or decrease. Because $y$ represents reserves mass, when $y(t)$ drops to a level $a$ (the *starvation boundary*), the individual dies of starvation. Throughout the rest of this paper I set the starvation boundary to zero (i.e., $a=0$), to avoid unnecessarily cumbersome notation, and because a nonzero starvation boundary results in only minor modifications (if any) to the expressions presented below (see Appendix B), and does not qualitatively change the general conclusions. Therefore, an individual dies of starvation only after exhausting all of its reserves (i.e., when $y[t]$ hits zero).

Following Filin (2009), structural growth occurs only each time a new body mass threshold is reached. In mathematical guise, the irreversibly growing structural mass $z$ is a non-decreasing function of $\theta$, i.e., $z(t)=z[\theta(t)]$. Whenever the organism reaches a new maximum of total mass (i.e., a new, higher value of $\theta$), there can be structural growth associated with this crossing of a new body mass threshold. In addition, structural growth always occurs on the expense of reserves $y$. A unit of structural mass is built by consuming $1+\alpha$ units of reserves mass. The dimensionless parameter $\alpha$ represents *overhead costs* of building structural mass.

In between events of structural growth (while $\theta[t]$ and thus $z[t]$ remain unchanged), the dynamics of reserves $y(t)$ is described by a diffusion process on the interval $[0, \theta-z]$. This diffusion process is characterized by $g(y, z)$ (the *mean growth rate*) and by $\sigma^2(y, z)$ (the *growth variance*) representing, respectively, the mean balance of input (e.g., assimilation) and output (e.g., metabolic maintenance) fluxes of energy and mass, and the random fluctuations around this mean balance. In general, both mean growth rate $g$ and growth variance $\sigma^2$ are dependent on both reserves mass $y$ and structural mass $z$ (but see following sections). (The units of $g$ are that of [mass/time], while the units of $\sigma^2$ are that of [mass$^2$/time].)

In addition to starvation mortality (if $y[t]$ hits the starvation boundary), the individual is subject to various exogenous hazards (e.g., predation and disease). These are captured by the mortality or hazard rate $\mu(y, z)$. Survival probability from initial reserves mass $y_1$ to final reserves mass $y_2$ ($\geq y_1$), while keeping $z$ fixed, is given by

$$S_y(y_1,y_2)=\exp\left(-\int_{y_1}^{y_2}\eta(y,z)\,dy\right) \tag{2}$$

where $\eta(y, z)$ is the *hazard density*, the hazard function for survivorship through transitions in the value of reserves mass (i.e., along the $y$-axis, from a lower value of reserves $y_1$ to a higher value $y_2$). The hazard density $\eta$ describes mortality per unit of gain in mass (analogously, mortality rate $\mu$, describes mortality per unit of time, i.e., along the time axis), and encapsulates within it both starvation mortality and exogenous mortality.

In the following, initial body mass of an individual will be denoted by $\theta_0$ and target body mass by $\theta_2$ ($\theta_1$ will denote the size at which structural growth ceases; see below). Survival probability from initial total mass $\theta_0$ to final total mass $\theta_2$, when structural mass grows according to $z=z(\theta)$, is given by

$$S_\theta(\theta_0,\theta_2)=\exp\left(-\int_{\theta_0}^{\theta_2}\eta(\theta-z,z)(1+\alpha\dot{z})\,d\theta\right) \tag{3}$$

(Filin, 2009), where $\dot{z}$ stands for $\partial z/\partial\theta$ (hereafter, the dot sign will always stand for derivative with respect to $\theta$). Note that the hazard function for transitions in total mass (i.e., along the $\theta$-axis; the integrand in Eq. (3)) is inflated by a factor $(1+\alpha\dot{z})$, dependent on rate and overhead costs of structural growth.

Finally, the optimization problem consists of finding the optimal form of the structural growth curve, $z^*(\theta)$, that maximizes the following objective function

$$\log F(\theta_0,\theta_2)=-\int_{\theta_0}^{\theta_2}\eta(\theta-z,z)(1+\alpha\dot{z})\,d\theta+\log R(\theta_2,z_2) \tag{4}$$

i.e., fitness is given by $F=S_\theta R$, namely, survival probability to target size multiplied by a terminal reward obtained at that size. The terminal reward $R$ (e.g., Houston and McNamara, 1999, ch. 3; also known as final function; Perrin and Sibly, 1993, appendix therein) depends on target size $\theta_2$ and on structural mass $z_2$ obtained at this target size.

The state variable in this optimization problem is $z$, with which a costate variable is associated, denoted by $\lambda$. The value of the costate variable at each body mass $\theta$ between $\theta_0$ and $\theta_2$ (i.e., $\lambda[\theta]$), quantifies the benefit of investing in structural growth, compared to just accumulating reserves. For analyzing the effect of target size on the optimal structural growth curve, $z^*(\theta)$, I require the boundary condition for the value of $\lambda$ at the target size $\theta_2$

$$\lambda(\theta_2)=\frac{\partial\log R}{\partial z_2} \tag{5}$$

(Appendix A).

In the following sections I derive the optimal structural growth curve for several forms of the objective (fitness) function (Eq. (4); more precisely, for several forms of the terminal reward $R$). First, I consider maximization of survival probability to target size (Eq. (3)). This is also the optimization objective in Filin (2009). Later, I extend the analysis to consider objective functions that include reproduction.

### 2.2. Maximizing survival probability to target size: optimal cessation of structural growth due to overhead costs

When maximizing survival probability to final size the terminal reward function in Eq. (4) is $R=1$. The boundary condition for $\lambda$ (Eq. (5)) then becomes $\lambda(\theta_2)=0$. I have previously studied optimal structural growth for this case (Filin, 2009; Eq. (12) therein). However, in that study I did not consider the possible effect of a finite target size on the optimal structural growth curve $z^*(\theta)$ (in effect, $\theta_2$ is taken to be infinite in Filin, 2009).

Filin (2009) showed that when nonzero structural growth occurs (i.e., $\dot{z}>0$), $z^*(\theta)$ follows a *singular arc* (a term borrowed from dynamic optimization theory; e.g., Perrin and Sibly, 1993). Along singular arcs the costate variable $\lambda$ and the structural mass $z$ satisfy the condition $\lambda(\theta)=\alpha\eta(\theta-z, z)$ (Appendix A). However, because $\lambda(\theta_2)=0$, this condition can never be satisfied at the target size, unless there are no overhead costs of building structural mass (i.e., if $\alpha=0$; $\eta$ is always positive, unless growth is completely deterministic, i.e., $\sigma^2=0$, and there are no exogenous hazards, i.e., $\mu=0$). Therefore, the pair $(\theta_2, z_2)$ (where $z_2=z^*[\theta_2]$) never lies on a singular arc, unless $\alpha=0$. I conclude that, for finite target size $\theta_2$ and nonzero overhead costs of structural growth ($\alpha>0$), the optimal structural growth curve $z^*(\theta)$ always ends with a plateau, along which structural mass remains constant ($z^*[\theta]=z_2$), and only reserves grow.

As we consider increasingly lower body masses below $\theta_2$, $\lambda(\theta)$ may gradually increase from its boundary value $\lambda(\theta_2)=0$ until the condition $\lambda(\theta)=\alpha\eta(\theta-z, z)$ is finally satisfied at some body mass, denoted by $\theta_1$ ($\leq\theta_2$; the equality may hold only if $\alpha=0$). At that point, the optimal growth curve connects with a singular arc, and as we proceed backwards towards even lower values of $\theta$ (i.e., $\theta<\theta_1$), the optimal growth curve exhibits positive structural growth ($\dot{z}^*>0$). The overall pattern of optimal structural growth is to grow along the singular arc until $\theta_1$, and then proceed to the target size $\theta_2$, by switching to only accumulating reserves. Therefore, $\theta_1$ is termed *switching size*.

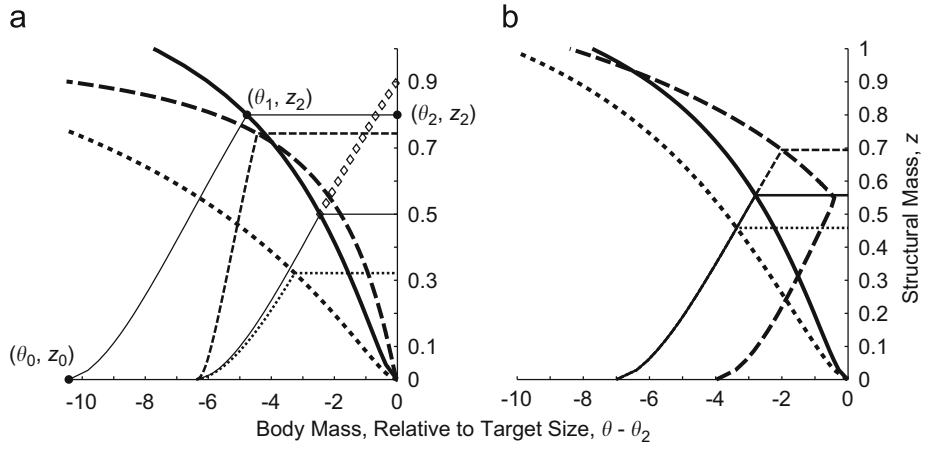


**Fig. 1.** **(a)** Switching curves (thick lines) and sample growth trajectories (thin lines) given: the standard specific model, described below ($\boldsymbol{\alpha}$=1; solid lines);$\boldsymbol{\alpha}$ is increased to 3 (dotted lines); growth variance $\boldsymbol{\sigma}^2$ is uniformly reduced by a factor of 10 (dashed lines). The optimization objective is survival to target size (i.e., no reproduction). In the uppermost sample growth trajectory I marked the points representing (from left to right) the initial, switching and target sizes. The other three sample trajectories all emanate from the same initial point. However, because of differences in $\boldsymbol{\alpha}$ or $\boldsymbol{\sigma}^2$ both the growth path and the switching size are different. When overhead costs of structural growth (i.e., $\boldsymbol{\alpha}$) are increased the switching curve becomes lower, but the rate of structural growth is only slightly reduced, causing smaller final structural mass and earlier cessation of structural growth (compare solid with dotted lines). When noise in individual growth is reduced (i.e., $\boldsymbol{\sigma}^2$ is reduced), the switching curve is higher for small target sizes, but lower for large target sizes (compare solid and dashed curves). Because a reduced growth variance strongly affects the rate of structural growth, cessation of structural growth occurs earlier, but at higher final structural mass. **(b)** Switching curves and sample growth trajectories for the standard specific model (below), given: no reproduction (solid lines), semelparous reproduction (dotted lines), iteroparous reproduction (dashed lines; $y_3$=4). Because in all three cases the values and functional forms of the model parameters are identical (see below), the growth paths are identical (given the same initial condition). The only variation is that structural growth ceases at different body masses (i.e., switching sizes) according to the mode of reproduction. The examples provided in both panels have been obtained using the following standard specific model: $g=3[z^{2/3}-(2/3)z]$, $\boldsymbol{\sigma}^2=4(0.1z^{2/3}+0.9z)$, $\boldsymbol{\mu}$=0.2, and $\boldsymbol{\alpha}$=1. The motivation for these specific expressions follows from the work of Kooijman (2000, ch.3), where $z$ here is comparable to Kooijman's *structural volume*. The mean growth rate $g$ is the difference between assimilation, proportional to surface area, and maintenance, proportional to volume. Time and size were rescaled such that maximum mean growth rate is $g$=1, and it occurs at $z$=1. The growth variance $\boldsymbol{\sigma}^2$ is the sum of noise in assimilation (again proportional to surface area) and noise in maintenance (proportional to structural mass; e.g., representing independent fluctuations in metabolism among cells). Mortality rate is taken to be constant, independent of structural mass (but see online Figs. 2 and 3 in the supplementary material for examples with size-dependent mortality).

The switching size, in turn, determines the optimal final structural mass achieved by the growing individual, because $z_2=z^*(\theta_2)=z^*(\theta_1)$ (and the latter is determined by the singular arc). Both $\theta_1$ and $z_2$ will change as target size $\theta_2$ varies. I can therefore obtain a *switching curve* $z_2(\theta_2)$ that determines the value of the optimal final structural mass as a function of target size. Fig. 1a provides specific examples of such switching curves and optimal structural growth curves for different target sizes. It is important to note that, in general, the singular arc is also defined for values of $\theta$ above the switching size $\theta_1$ (diamonds in Fig. 1a). However, given a finite target size, it is optimal to abandon the singular arc once structural mass has hit the switching curve (i.e., at the switching size).

When reserves growth and exogenous mortality depend only on structural mass (i.e., $g$, $\sigma^2$ and $\mu$ are functions of $z$ only), Filin (2009) provides the following expression for the hazard density

$$\eta(y,z)=\chi\coth(\chi y)-\varphi \tag{6}$$

where

$$\chi=\sqrt{\frac{g^2}{\sigma^4}+\frac{2\mu}{\sigma^2}},\quad \varphi=\frac{g}{\sigma^2} \tag{7}$$

are both functions of structural mass $z$ (i.e., $\chi=\chi[z]$ and $\varphi=\varphi[z]$). The quantity $\chi$ combines both starvation risk (decreasing with $g$ and increasing with $\sigma^2$) and exogenous hazards (increasing with $\mu$), to determine the range of values of reserves mass in which starvation is the predominant cause of mortality (as opposed to exogenous hazards). (This range can be described by the interval $[0, \chi^{-1}]$.) The quantity $\varphi$ measures how stochastic/deterministic the reserves dynamics is: when $\varphi\to\pm\infty$, the growth of reserves mass is practically deterministic, while when $\varphi\to 0$ reserves dynamics behaves like Brownian motion. The units of $\varphi$, $\chi$ and $\eta$ are that of $[\text{mass}^{-1}]$.

The approach to calculating the switching curve is to solve for the switching size and target size ($\theta_1$ and $\theta_2$, respectively), given a value of final structural mass $z_2$. For that purpose, I define the following dimensionless variables

$$Y=\chi y,\quad A=\frac{\chi^2}{\chi'},\quad B=\frac{\varphi'}{\chi'},\quad \Phi=\frac{\varphi}{\chi} \tag{8}$$

where $\varphi'=d\varphi/dz$ and $\chi'=d\chi/dz$ (note the change in the definitions of $Y$ and $A$, compared to Filin, 2009). I then derive (Appendix A) the following expression for the switching curve

$$f_{tar}(Y_2,A,B)=f_{tar}(Y_1,A,B)-\alpha A(\coth Y_1-\Phi) \tag{9}$$

where the function $f_{tar}$ is defined as $f_{tar}(Y_2, A, B)=(Y-A)\coth Y-BY$. In addition, $Y_2=\chi(\theta_2-z_2)$, $Y_1=\chi(\theta_1-z_2)$, and $\chi$, $A$, $B$ and $\Phi$ are all evaluated at structural mass $z=z_2$. Because $(\theta_1, z_2)$ lies on the singular arc, $Y_1$ obeys

$$f_{arc}(Y_1,B)=2(1+\alpha)A \tag{10}$$

where $f_{arc}(Y, B)=[2Y-\sinh(2Y)]+B[\coth(2Y)-1]$ (the singular arc equation; Eq. (14) in Filin, 2009; using the revised definitions of $Y$ and $A$ in Eq. (8)). I used these expressions (Eqs. (9) and (10)) to calculate the switching curves and growth trajectories presented in Fig. 1a.

### 2.3. Maximizing expected allocation to reproduction: semelparity and iteroparity

Both Filin (2009) and this study, up to this point, have only considered survival probability as the objective (fitness) function. Clearly, however, a full description and optimization of a lifecycle

must include reproduction as well. The simplest (yet still realistic) form of introducing reproduction into the optimization objective is choosing the terminal reward function in Eq. (4) to be $R=\theta_2-z_2$, i.e., the mass of reserves accumulated at target size, and available for production of progeny. Alternatively, $R$ can describe yield, for example, in agricultural crops. The role of reserves is now dual, serving as both insurance against starvation during growth (in addition to providing raw material and fuel for structural growth), and as the terminal reward gained once target size is achieved. Therefore, in addition to the overhead costs of structural growth, discussed above, investment in structure entails an additional cost in terms of reduced fecundity or yield. The boundary condition in Eq. (5) becomes $\lambda(\theta_2)=-1/(\theta_2-z_2)$.

The expression for the terminal reward from the previous paragraph describes semelparous mode of reproduction. All reserves are utilized in a single burst of reproduction, causing the individual to die of starvation immediately after. However, if the individual retains some reserves after reproduction (denoted by $y_3$) it may survive to reproduce additional times, i.e., iteroparous mode of reproduction. The individual sacrifices immediate reproduction (by not utilizing all of its reserves mass in a single reproduction event), for the sake of surviving to future reproduction events. Thus, the tradeoff between present and future reproduction (e.g., between fecundity and parental survival; Roff, 2002, pp. 126–150, pp. 188–198) is generated mechanistically in this model, mediated by the value of $y_3$ (i.e., the level of reserves retained after reproduction).

I adopt here the concept of a *reproduction buffer* (Kooijman, 2000; p. 115), such that immediately after each reproduction event, reserves mass is $y_3$ (reproduction buffer emptied), and subsequent reproduction events occur each time the individual regains reserves mass $\theta_2-z_2$ (reproduction buffer full). Survival between reproduction events is then given by $s=S_y(y_3,\theta_2-z_2)$ (recall that $S_y$ represents survival along reserves mass transitions, while holding structural mass fixed; Eq. (2)). The expected number of reproduction events is $1/(1-s)$, and the terminal reward is then $R=(\theta_2-z_2-y_3)/(1-s)$. The boundary condition in Eq. (5) becomes $\lambda(\theta_2)=-1/(\theta_2-z_2-y_3)+(\partial s/\partial z_2)/(1-s)$. Additional structural growth does not occur after the individual begins to reproduce (i.e., after reaching $\theta_2$ for the first time; determinate growth).

For semelparous reproduction, $\lambda(\theta_2)$ is now negative ($\lambda[\theta_2]=-1/[\theta_2-z_2]$), compared with $\lambda(\theta_2)=0$ when the objective function was survival probability to target size (previous section). Integrating backwards from target size $\theta_2$ towards lower body masses, $\lambda(\theta)$ would take longer to reach the singular arc $\lambda(\theta)=\alpha\eta(\theta-z,\ z)$ (the right-hand-side of this equation is non-negative). Therefore, for a given target size, $\theta_2$, I expect earlier cessation of structural growth (smaller switching size, $\theta_1$) and smaller final structural mass ($z_2$), when semelparous reproduction is taken into consideration in the objective function. The effect of iteroparous reproduction on the switching curve is less straightforward (see below).

The following analysis again concerns the case with reserves growth and exogenous mortality dependent only on structural mass (i.e., $g$, $\sigma^2$ and $\mu$ are functions only of $z$). For semelparous reproduction, the expression for the switching curve is

$$f_{tar}(Y_2,A,B)+\frac{A}{Y_2}=f_{tar}(Y_1,A,B)-\alpha A(\coth Y_1-\Phi) \tag{11}$$

where again $Y_1$ obeys the singular arc equation (Eq. (10)). Fig. 1b demonstrates that, indeed, structural growth ceases earlier for semelparous reproduction (dotted curves; compared with the case of maximizing only survival to target size; previous section; solid curves in Fig. 1b).

For iteroparous reproduction, Appendix A provides the expression for the switching curve. As Fig. 1b demonstrates the switching curve, in this case, has two branches: upper and lower. Only the upper branch is a solution of the switching curve equation (Appendix A). Thus, such a solution exists only if the value of the final structural mass $z_2$ is high enough. The lower branch of the switching curve represents the additional constraint $\theta_2\geq z_2+y_3$ that must be obeyed in the case of iteroparous reproduction (not surprisingly the lower branch intersects the abscissa axis at $\theta-\theta_2=-y_3=-4$ in the case of Fig. 1b). When the equality $\theta_2=z_2+y_3$ holds, we obtain the limit of continuous reproduction. The amount of reserves spent in each reproduction event goes to zero ($\theta_2-z_2-y_3=dy\to 0$), survival probability between reproduction events goes to one ($s\to[1-\eta(y_3,\ z_2)dy]$; Eq. (2)), and the expected number of reproduction events becomes infinite (the terminal reward is nonetheless finite: $R=[\theta_2-z_2-y_3]/[1-s]\to 1/\eta[y_3,\ z_2]$). From a biological perspective, however, such continuous reproduction, which consists of an infinite number of infinitesimally small progeny, can only be regarded as an approximation, at best. I further consider this issue below in the discussion.

## 3. Discussion

In this study, I extended my previous work (Filin, 2009) and considered optimal stopping conditions for structural growth, when individuals must grow to some given finite target size, and when reproduction is included in the optimization objective. I found that even when the optimization objective is maximizing survival to target size (as in Filin, 2009; i.e., no reproduction) it is optimal to abandon structural growth altogether close to the target size, and to proceed only by accumulating reserves.

Only when there are no overhead costs of building structural mass ($\alpha=0$), is it optimal to keep investing in structure all the way to the target size. The reason is that the individual pays for such overhead losses of reserves in increased mortality, due to the extra time required to regain those lost reserves. Close to the target size, it is optimal to avoid any such losses of reserves mass (and thus instantaneous total body mass $z[t]+y[t]$). In addition, Fig. 1a demonstrates that the higher the overhead costs (i.e., higher $\alpha$), the earlier structural growth ceases and the smaller final structural mass is (see also Figs. 2 and 3 in the supplementary material).

The effect of reducing the level of noise in the dynamics of reserves (i.e., reducing the growth variance $\sigma^2$) is less straightforward. At small target sizes, a lower noise level increases the final structural mass. However, the cessation of structural growth occurs earlier (i.e., at a smaller total body mass), because lower noise levels also promote faster structural growth. At large target sizes the effect is reversed, lower noise levels cause smaller final structural masses (Fig. 1a; see also Figs. 2 and 3 in the supplementary material).

Filin (2009) also found that varying the growth variance may either increase or decrease final structural mass, depending on the function of the structural trait (e.g., foraging-related or defensive). However, the effect of growth variance in this paper is fundamentally different from the one found by Filin (2009). Because target size is, in effect, infinite in Filin (2009), the results described in that work concern the effect of growth variance on the asymptotic structural mass (i.e., at very large total body masses: $\theta\to\infty$). Here, by contrast, I consider the effect of the growth variance on optimal cessation of structural growth due to a finite target size, and at final structural masses potentially far from the asymptotic value. As discussed above, the signs of these two different effects may in fact be opposite.

When I incorporate reproduction into the optimization objective, the results vary depending on whether reproduction is semelparous or iteroparous (Fig. 1b). Semelparous reproduction always causes earlier cessation of structural growth and smaller final structural masses, compared to the case with no reproduction (when survival is the optimization objective). Iteroparous reproduction introduces a new parameter into the model, $y_3$, the amount of reserves retained by the individual after a reproduction event. As Fig. 1b demonstrates, at very small target sizes (but nonetheless larger than $y_3$) the switching size is the initial size and there is no structural growth at all throughout the entire lifetime of the individual (the region right of the iteroparity switching curve in Fig. 1b). At larger target sizes, cessation of structural growth is delayed, compared to either semelparous reproduction or no reproduction, causing larger final structural mass for a given initial condition (see growth trajectory examples in Fig. 1b). Finally, at even larger target sizes the switching curve for iteroparous reproduction falls below that for no reproduction and asymptotically approaches that of semelparous reproduction.

The above qualitative conclusions remain unchanged also when mortality is size-dependent, decreasing with structural mass (see Figs. 2b and 3b in the supplementary material). The effect of size-dependent mortality in this case is only quantitative, prolonging structural growth and thus resulting in larger final structural mass. The effects of increased overhead costs or reduced noise level remain qualitatively the same.

The growth trajectory examples in Fig. 1b demonstrate that, given identical initial conditions, target size and parameter values (including identical functional forms of $g$, $\sigma^2$ and $\mu$), the reproduction mode (i.e., no reproduction, semelparity or iteroparity) does not affect the growth trajectory itself. The reproduction mode only affects the stopping condition, i.e., the body and structural mass at which structural growth ceases. (Mathematically, that is because the reproduction mode only affects the terminal reward function $R$ in Eq. (4).). This provides an interesting prediction for comparative studies, where closely related species or populations, or even different individuals within the same population, may exhibit different growth patterns, depending on the reproduction mode they adopt (semelparous or iteroparous).

A related question is that of sexual dimorphism (this issue was also briefly addressed in the context of deterministic dynamic optimization models in the discussion of Kozlowski and Wiegert, 1987). Because the benefits and costs of body size and structures vary between males and females within a species, the terminal reward function should also depend on sex. As discussed in the previous paragraph, this would affect the stopping condition, i.e., males and females will cease structural growth at different body mass and attain different final sizes of structures. However, early growth and development will be identical. This is a pattern of bimaturism, and in the context of heterochrony, may lead to males, for example, being hypermorphic compared to females (McNamara, 1995; i.e., sexual dimorphism due to differences in timing of developmental transitions between males and females). However, if there are additional sex-specific differences in consumption, assimilation, metabolism or mortality, the optimal growth trajectories of males and females may diverge earlier in life, and before final structural size is attained. When such differences are caused by differences in behavior between males and females (e.g., Rennie et al., 2008), the model may be extended to include reversible behavioral transitions (which can then be optimized separately for males and females), in addition to the irreversible structural growth, as presented and discussed in Filin (2009).

As discussed in the previous section, when $\theta_2=z_2+y_3$, iteroparous reproduction becomes continuous, consisting of an infinite number of infinitesimally small progeny. There are at least two ways to remedy this biologically questionable result. First, one can define an upper bound $s_{max}<1$ for the survival probability between reproduction events (i.e., $s$). As a result, the expected number of reproduction events can never exceed $1/(1-s_{max})$ (which is finite because $s_{max}<1$). The parameter $s_{max}$ may embody mortality factors during the reproduction event itself, e.g., due to increased susceptibility to predation. (See also Fig. 3 in the supplementary material.)

A second more mechanistic way to avoid continuous reproduction is to introduce a minimum nonzero amount of reserves that the individual must expend during each reproduction event. For example, this amount may represent the costs of producing a single egg. Denoting this amount by $a_{egg}$, the constraint on target size becomes $\theta_2 \geq z_2+y_3+a_{egg}$. When the equality holds, the individual reproduces in single eggs, i.e., clutch size is one, rather than continuously as before (if $a_{egg}=0$). Fig. 4 in the supplementary material further explores these modifications. It is important to note that, although not explicitly formulated as part of the model, using this last modification, optimal clutch size arises as a byproduct of the life history optimization within this model. Thus, the theoretical framework presented in this study also addresses this important ecological and evolutionary problem.

There exists an analogy between the model described in this paper and dynamic optimization models for optimal size at maturity when season length is finite (Cohen, 1971; Vincent and Pulliam, 1980; Kozlowski and Wiegert, 1986). A comparison with such models, demonstrates that body mass $\theta$ is analogous to the time-coordinate in those models. Target size is analogous to season length, and $\theta-\theta_2$ of Fig. 1 is analogous to the 'time-to-go' until season end, which determines the optimal switch between growth and reproduction in those models. However, growth in those models is deterministic. Therefore, exploring, for example, the effect of noise in growth is not within their scope.

This study explored the optimal way to invest in structural growth, starting from some initial condition and finishing at a given final target size. Target size was taken to be a fixed parameter, and growth and life history were optimized under that constraint. Elsewhere I will additionally explore the simultaneous optimization of the endpoints, i.e., of initial and target sizes (e.g., representing optimal progeny and adult sizes, respectively). Kozlowski (1996) previously obtained such optimal initial and adult sizes for a deterministic life history model. The stochastic model in this work enables me to investigate additional questions, such as the effect of noise in the growth of individuals on optimal progeny and adults sizes.

Concerning the above discussion of sexual dimorphism and optimal clutch size, optimizing target size itself will enable to explore sexual size dimorphism in the adult (total) body mass, in addition to sexual dimorphism in the allocation between structure and reserves. Optimization of initial and adult sizes will also result in simultaneous optimization of egg and clutch size, as $a_{egg}$ will no longer be a fixed parameter but will depend on the initial size, subject to optimization. Finally, I am also currently working on an extension of the model to indeterminate growers.

In conclusion, the theoretical framework, presented in this study, provides a powerful tool for addressing a wide variety of life-history and evolutionary questions, under biologically realistic conditions, including: subjection to both starvation mortality and exogenous hazards, noise in the dynamics of individual state, and distinction between reversible and irreversible components of individual size. These two last aspects of the growth and development of individuals have important implications to evolution of life history (as well as to population dynamics; Filin, 2009), that have not yet been fully explored and assessed.

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## Appendix A. Derivation of the optimal structural growth curve and the switching curve

Applying the Pontryagin maximum principle (Intriligator, 1971, pp. 344–348; Perrin and Sibly, 1993, appendix therein; Perrin et al., 1993), I obtain the Hamiltonian

$$H=-\eta(\theta-z,z)(1+\alpha u)+\lambda u$$

where $z$ is the state variable, $\lambda$ is the costate variable associated with $z$, and $u$ is the control variable, defined as $\dot{z}=u$ (recall that the dot sign stands for derivative with respect to $\theta$). The dynamics of the costate variable is given by

$$\dot{\lambda}=-\frac{\partial H}{\partial z}=(1+\alpha u)\left(\frac{\partial \eta}{\partial z}-\frac{\partial \eta}{\partial y}\right)\begin{array}{l} y=\theta-z \\ z=z^*(\theta)\end{array} \tag{A.1}$$

with boundary condition given by the derivative of the final function (terminal reward $R$ in Eq. (4)) with respect to the state variable $z$

$$\lambda(\theta_2)=\frac{\partial \log R}{\partial z_2}$$

(i.e., Eq. (5); Intriligator, 1971, p. 348).

The switching function for investing in structural mass is given by $\Sigma=\partial H/\partial u$, or

$$\Sigma=-\alpha\eta(\theta-z,z)+\lambda$$

when $\Sigma=0$, I obtain singular control, i.e., this is the equation satisfied by a singular arc, to which I refer throughout the main text. Singular control additionally requires $d\Sigma/d\theta=0$, which results in the general equation for the singular arc presented by Filin, (2009)

$$\left[\frac{\partial \eta}{\partial z}-(1+\alpha)\frac{\partial \eta}{\partial y}\right]_{y=\theta-z}=0$$

(see derivation therein).

I confine the analysis, hereafter, to the special case of reserves growth and exogenous mortality dependent only on structural mass $z$ (i.e., Eqs. (6)–(8)). Applying the conclusion that between $\theta_1$ and $\theta_2$ no structural growth occurs (i.e., the optimal control is $u^*=0$; thus, $z^*(\theta)=const=z_2$), Eq. (A.1) becomes

$$\dot{\lambda}=\chi'\coth Y-\chi' Y\ \mathrm{csch}^2 Y+\chi^2\ \mathrm{csch}^2 Y-\varphi'$$

where $Y=\chi y=\chi(\theta-z)$ (Eq. (8)), $\varphi'=d\varphi/dz$ and $\chi'=d\chi/dz$, all evaluated at $z=z_2$. Integrating from $\theta_1$ to $\theta_2$ gives

$$\lambda(\theta_2)=\lambda(\theta_1)+\left[\left(\frac{\chi'}{\chi}\right)Y\coth Y-\chi\coth Y-\left(\frac{\varphi'}{\chi}\right)\right]_{Y_1}^{Y_2}$$

where $Y_1=\chi(\theta_1-z_2)$ and $Y_2=\chi(\theta_2-z_2)$. Multiplying both hand sides by $\chi/\chi'$, using the definitions of the dimensionless variables in Eq. (8), and recalling that $(\theta_1,\ z_2)$ lies on the singular arc (therefore, $\lambda[\theta_1]=\alpha\eta[\theta_1-z_2,\ z_2]=\alpha\chi\coth Y_1-\alpha\varphi$), I finally obtain

$$[(Y_2-A)\coth Y_2-BY_2]-\Lambda=[(Y_1-A)\coth Y_1-BY_1]-\alpha A(\coth Y_1-\Phi) \tag{A.2}$$

where

$$\Lambda=\left(\frac{\chi}{\chi'}\right)\lambda(\theta_2) \tag{A.3}$$

And using the definition of $f_{tar}$ in the main text (under Eq. (9)), Eq. (A.2) can be rewritten as

$$f_{tar}(Y_2,A,B)-\Lambda=f_{tar}(Y_1,A,B)-\alpha A(\coth Y_1-\Phi) \tag{A.4}$$

When the objective function is survival probability to final size, $\Lambda=0$, and I obtain Eq. (9). When the objective function includes semelparous reproduction $\lambda(\theta_2)=-1/(\theta_2-z_2)=\chi/Y_2$. Eq. (A.4) then becomes Eq. (11). For iteroparous mode of reproduction, $\lambda(\theta_2)=-1/(\theta_2-z_2-y_3)+s'/(1-s)$ (where $s'$ stands for $\partial s/\partial z_2$). The survival probability $s$ between reproduction events is given by

$$s=\frac{\sinh Y_3}{\sinh Y_2}\exp[\Phi(Y_2-Y_3)] \tag{A.5}$$

(the expression for $S_y$ in Eq. (3) of Filin, 2009) where $Y_3=\chi y_3$ ($y_3$ is taken to be a fixed parameter of the model). Consequently, $s'/s=\partial\log s/\partial z_2=Y_3'(\coth Y_3-\Phi)-Y_2'(\coth Y_2-\Phi)+\Phi'(Y_2-Y_3)$. Given that $Y_2=\chi(\theta_2-z_2)$, and $\Phi=\varphi/\chi$, I obtain $(\chi/\chi')Y_2'=Y_2-A$ and $(\chi/\chi')\Phi'=B-\Phi$ (using definitions in Eq. (8)). The mass of reserves immediately after a reproduction event is $y_3$, and its dimensionless counterpart is $Y_3=\chi y_3$. Given that $y_3$ is a fixed parameter, $(\chi/\chi')Y_3'=Y_3$, and then Eq. (A.3) becomes

$$\Lambda=-\frac{A}{Y_2-Y_3}-\frac{s}{1-s}[(Y_2-A)\coth Y_2-BY_2+A\Phi+BY_3-Y_3\coth Y_3]$$

Finally, by additionally utilizing the definition of $f_{tar}$, I arrive at the expression for the switching curve in the case of iteroparous reproduction

$$\begin{aligned}&\frac{1}{1-s}f_{tar}(Y_2,A,B)+\frac{A}{Y_2-Y_3}+\frac{s}{1-s}[A\Phi+BY_3-Y_3\coth Y_3]\\&\quad=f_{tar}(Y_1,A,B)-\alpha A(\coth Y_1-\Phi)\end{aligned} \tag{A.6}$$

where $s$ is given by Eq. (A.5).

## Appendix B. Modifications for nonzero starvation boundary

For the survival-probability-to-target-size and iteroparous-reproduction scenarios the expressions are easily modified for nonzero starvation boundary $a$ by redefining $Y$ (Eq. [8]) as

$$Y=\chi(y-a) \tag{B.1}$$

Similarly the hazard density in Eq. (6), is given by

$$\eta(y,z)=\chi\coth[\chi(y-a)]-\varphi \tag{B.2}$$

Subsequently, the expressions for the switching curves in these cases remain the same (Eqs. (9) and (10), and Eq. (A.6) in Appendix A).

For semelparous reproduction, I define $\tilde{a}$ as the amount of reserves that cannot be utilized for reproduction, once target size is achieved. It may be possible that the amount of reserves that cannot be utilized for maintenance to avoid starvation, i.e., the starvation boundary $a$, can nonetheless be utilized for reproduction (in this case $\tilde{a}\le a$). In contrast, some reserves may be utilized for maintenance, but not mobilized for reproduction (in that case $\tilde{a}\ge a$). Regardless of which case occurs, the terminal reward is $R=\theta_2-z_2-\tilde{a}$, and the boundary condition in Eq. (5) takes the form $\lambda(\theta_2)=-1/(\theta_2-z_2-\tilde{a})$. The expression for the switching curve is then

$$f_{tar}(Y_2,A,B)+\frac{A}{Y_2-\tilde{Y}}=f_{tar}(Y_1,A,B)-\alpha A(\coth Y_1-\Phi) \tag{B.3}$$

where $Y_2=\chi(\theta_2-z_2-a)$ and $\tilde{Y}=\chi(\tilde{a}-a)$ ($\tilde{Y}$ can be either positive or negative depending on which of the two above-mentioned cases occur). Eq. (B.3) becomes Eq. (11) if $\tilde{a}=a$ (i.e., if $\tilde{Y}=0$).

## Appendix C. Supplementary material

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jtbi.2010.02.031.

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