A Comparison of Dynamic-State-Dependent Models of the Trade-Off Between Growth, Damage, and Reproduction

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abstract: Fast growth can be costly, so trade-offs between growth and fitness are to be predicted when organisms adjust their growth to compensate for earlier environmental conditions. We developed four generic models of increasing complexity with different processes to predict the indeterminate growth of vertebrate ectotherms, which is sensitive to ambient temperature even when food is not limiting. We contrast the predictions of the models with observed experimental data on growth trajectories, feeding activity, and reproductive investment of three-spined sticklebacks and inferred patterns of accumulation of biomolecular damage arising from activity and growth. All models predicted observed patterns of compensatory growth (both accelerating and decelerating) in response to earlier temperature perturbations, but the more complex models provided the best fit to experimental data. Growth trajectories influenced future reproductive investment regardless of final body size at breeding. Our findings suggest that while models with fewer parameters can predict basic patterns of growth in stable conditions, they cannot capture the costly long-term effects of deviations from steady growth trajectories. In contrast, models in which foraging activity is assumed to carry costs are capable of predicting the complex patterns of feeding, growth, and reproductive investment seen in animals, with the cost of a heightened mortality risk (e.g., through predation) being more important than the cost of increased physiological damage.

Keywords: compensatory growth, damage, life-history strategy, reproductive investment, activity, allocation.

Introduction

The growth rate of an organism influences its future survival and reproduction and is affected by energy supply and environmental conditions, in particular those pertaining to when growth rates are normally fastest (usually in early life). Growth rate has known effects on life-history traits: for instance, attaining larger size in early life typically leads to higher survival and fecundity in later life (Quentin and Richard 2001). However, there may be costs induced by the rapid growth needed to reach large size, such as an increased risk of physiological damage to molecules, cells, and tissues (Metcalfe and Monaghan 2001, 2003; Mangel and Munch 2005) and increased metabolic rate in adulthood (Criscuolo et al. 2008). Alternatively, or perhaps in addition, a faster overall rate of early growth might cause a mismatch in relative growth and development of component tissues or organs, producing a suboptimal adult phenotype (Martell et al. 2006) that would then fail sooner.

Compensatory growth is a well-known strategic adjustment that occurs when growth rate is accelerated when conditions improve after a period of poor conditions (e.g., starvation or low temperature); if it is completed, compensation results in normal adult size still being attained despite the earlier setback (Arendt 1997). While compensatory growth may enhance survival, feeding, and mating success (Metcalfe and Monaghan 2001), recent work has shown that early accelerated growth after a period of food deprivation can have negative effects on whole-organism performance in later life. These include reductions in locomotor performance (Álvarez and Metcalfe 2005; Lee et al. 2010), reproductive output (Auer et al. 2010), and life span (Inness and Metcalfe 2008). When maximizing Darwinian fitness, the growth strategy adopted by an individual faced with a finite energy resource is predicted to depend on allocation trade-offs that in turn often depend on the state of the individual (McNamara and Houston 1996).

In a theoretical examination of the consequences of such long-term costs of growth rate, Mangel and Munch (2005) showed that if rapid growth increased the rate of accumulation of damage in body tissues, this could lead to different optimal long- or short-term growth strategies. However, their model did not consider temperature, which in ectotherms has diverse effects on organismal perfor-

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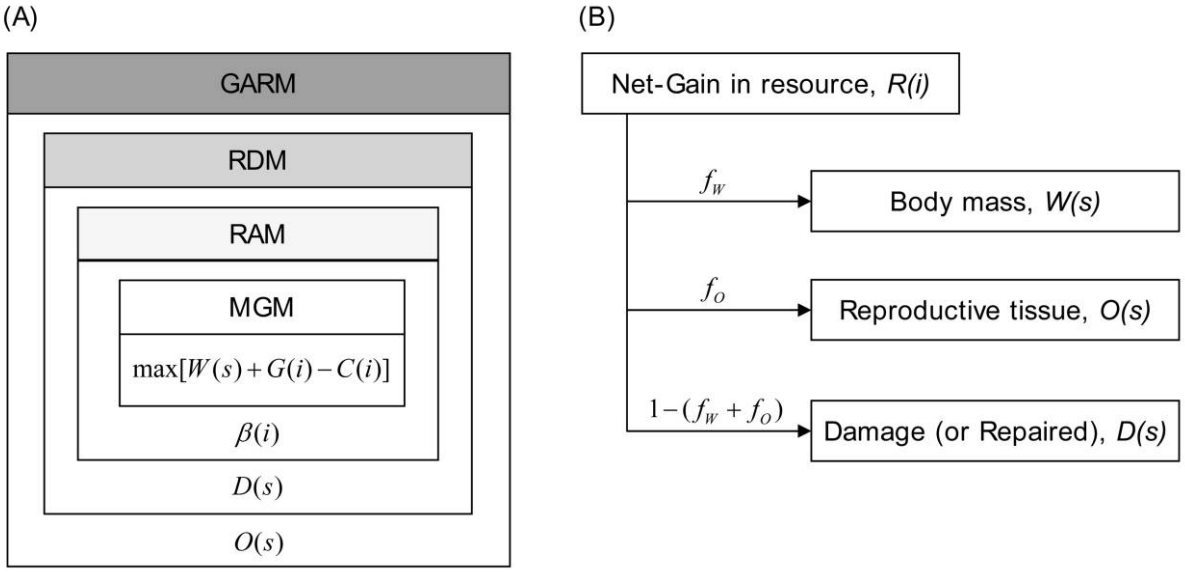

Figure 1: Illustration of *A*, the structure of models (MGM, maximize growth model; RAM, response to activity model; RDM, response to damage model; GARM, gonadal accumulation and repair model; see "Material and Methods" for more detail), and *B*, the resource allocation process in the GARM model. $W(s)$, body mass; $G(i)$, daily food consumption; $C(i)$, catabolic costs; $\beta(i)$, mortality; $D(s)$, damage (or repair); $O(s)$, reproductive tissue.

mance (Wootton 1998). For example, moderate increases in ambient temperature are associated with faster growth, but they also cause a higher metabolic rate and hence a higher food requirement, leading to more active foraging behavior and hence a greater risk of being detected by predators. Higher metabolism may also lead to a greater rate of damage accumulation due to an increased production of damaging reactive oxygen species (Metcalfe and Alonso-Alvarez 2010). Therefore, environmental conditions that affect growth may have both benefits and costs, making it difficult a priori to predict the optimal rate of growth for a given set of environmental conditions.

In this article, we develop a range of life-history models to understand the trade-offs faced by vertebrate ectotherms between early growth and damage in relation to both temperature and food supply, taking into account the level of activity required to obtain a given amount of food and the resulting pattern of energy allocation. We develop four models of increasing complexity with different growth-damage scenarios that range from assuming that the animal maximizes growth regardless of any costs (the maximize growth model [MGM]), through assuming a relationship between activity and mortality risk (the response to activity model [RAM]), to assuming that growth is mediated by the response to the accumulation of macromolecular and cellular damage (the response to damage model [RDM]), and then to a model that allows the animal to apportion resources between somatic growth, gonadal growth, and investment in repair of damage (gonadal accumulation and repair model [GARM]). We then compare the growth trajectories predicted by each model with each other and with experimental data from three-spined sticklebacks *Gasterosteus aculeatus* that had been induced during their juvenile growth phase to follow three different growth trajectories (accelerating, decelerating, and steady) by temperature manipulations. Our results suggest how early growth trajectory is likely to cause long-term effects through the accumulation of physiological damage and how this trade-off between growth tempo and damage level can influence optimal life-history strategies.

Material and Methods

Dynamic State Models

To find the growth and activity levels that maximize expected fitness, we consider four possible dynamic models of increasing complexity (fig. 1*A*). Initially we consider a life history to be governed by one state variable, mass W, but the later models include two additional state variables, the accumulation of oxidative or cellular damage D and of reproductive tissue O. To model growth rates, we combine a model of fish growth (Mangel and Munch 2005), a food-consumption model for three-spined sticklebacks

Table 1: Summary of variable and parameter definitions and the range of values used in simulations

Variable or parameter	Description	Range or values			
		MGM	RAM	RDM	GARM
Time:					
s	Time	1–40	1–40	1–40	1–40
S	Final time, at which reproduction is assessed				
State variables:					
$W(s)$	Current body mass at time s				
$O(s)$	Current mass of reproductive tissue at time s				
$D(s)$	Current level of damaged tissue at time s				
$T(s)$	Temperature (°C) at time s				
$G(i)$	Daily food consumption at activity i				
$C(i)$	Basic catabolic costs at activity i				
$\alpha_s(i)$	Specific metabolic cost at activity level i				
Model outputs:					
w	Body mass	1–2,500	1–2,500	1–2,500	1–2,500
d	Damage	1–1,000	1–1,000	1–1,000	1–1,000
o	Reproductive tissue				
Parameters for growth:					
i	Activity	0–1	0–1	0–1	0–1
c_0	Constant for food consumption	−12	−12	−12	−12
c_1	Weight coefficient for food consumption	.388	.388	.388	.388
c_2	Temperature coefficient for food consumption	19.312	19.312	19.312	19.312
c_3	Constant for catabolic cost	.021	.021	.021	.021
α	Weight-specific catabolic rate	.125	.125	.125	.125
m_r	Reduction in metabolic cost due to resting	.002	.002	.002	.002
φ	Multiple for fitness value	1	1	1	1
Parameters for damage:					
k	Parameter for damage accumulation	.51	.51	.51	.51
i_d	Activity level at zero damage	.005	.005	.005	.005
ρ	Efficiency of repair				.001
Parameters for mortality:					
μ	Mortality rate when active		.015	.015	.015
μ_r	Mortality rate when resting		.014	.014	.014
μ_d	Mortality rate due to damage			.005	.005
μ_b	Mortality rate during breeding season			.002	.002

Note: See text for details of simulations. GARM, gonadal accumulation and repair model; MGM, maximize growth model; RAM, response to activity model; RDM, response to damage model.

(Wootton et al. 1980), and a basal catabolic model for fish (Brett and Groves 1979). As with dynamic energy budget models but without resorting to hidden state variables, we incorporate an interaction between activity levels and consumption, using insights from optimal foraging theory (Clark and Mangel 2000; Satterthwaite et al. 2010).

To begin, we assume that the net gain of resource based on anabolism G and catabolism C at activity level i is allocated to an increase in body mass, an increase in reproductive tissue (e.g., oocyte), and/or repair of damaged tissue (fig. 1*B*). If f_W is the fraction of resources allocated to mass gain, then body mass W at time $s + 1$ is described by

$$W(s + 1) = W(s) + f_W(G(i) - C(i)). \quad (1)$$

The amount of food G a fish consumes during time s when its activity level is i is (Wootton et al. 1980)

$$G(i) = i[c_0 + (c_1W(s))^{0.75} + c_2T(s)], \quad (2)$$

where $T(s)$ is temperature at time s and c_0, c_1, and c_2 are constants associated with the activity, activity interacting with mass, and activity interacting with temperature dependence of growth, respectively (table 1).

Consumption thus depends on fish size and temperature T at that time s, but the extent to which it reaches the maximal intake possible at that time depends on the fish's activity i. The basic catabolic costs of a fish (Brett and Groves 1979),

$$C(i) = \alpha_s(i)e^{0.021T(s)}W(s), \quad (3)$$

also depend on its mass and temperature and the specific metabolic cost $\alpha_s(i)$ at activity level i. Here, $e^{0.021\,T(s)}$ characterizes the temperature dependence of growth costs (Brett and Groves 1979). The specific metabolic cost for a given level of activity,

$$\alpha_s(i) = \alpha i + (1 - i)\alpha m_r, \tag{4}$$

depends on the weight-specific catabolic rate α and the multiplier for time spent resting m_r. Note that increases in activity level i cause increases in both consumption and total catabolic costs, but at different rates (Mangel and Munch 2005), so there is an intermediate, optimal level of activity. The basal catabolic term depends on the measure of weight-specific catabolic costs α and the effect of temperature T. Thus, the net gain of resources $R(i)$ at activity level i is

$$R(i) = G(i) - C(i). \tag{5}$$

We assume that the probability of survival for the RDM and for the GARM depends on both activity and accumulated damage (see Mangel and Munch 2005):

$$\beta_1(i) = e^{-\mu i - \mu_r(1-i) - \mu_d d}, \tag{6}$$

where the mortality rate while active (e.g., as a result of predation) is μ, the mortality rate while resting is μ_r, the mortality rate due to damage is μ_d, and the level of damage is d (table 1). In the RAM, we assume the activity-dependent probability of survival to be

$$\beta_2(i) = e^{-\mu i - \mu_r(1-i)}. \tag{7}$$

While we use equations (1)–(4) as the general structure of growth based on anabolism and catabolism, in the GARM the individual at each time step allocates resources to the three options of growth, repair of damage, and investment in reproduction. If f_o is the fraction of resources allocated to reproductive tissue, the allocation to repair would be $(1 - f_W - f_O)$. So, the level of damaged tissue $D_1(s)$ at time $s + 1$ is described by

$$D_1(s + 1) = D_1(s) - (1 - f_W - f_O)R(i)\rho + k(i - i_d), \tag{8}$$

where ρ is the efficiency of repair (and is thus the parameter that links investment in repair to actual reduction in damage), i_d is the activity level at zero net production of damage (i.e., repair = production), and k connects activity to damage.

Over time, all organisms accumulate damage to molecules, cells, and tissues that will reduce their capacity in a range of ways. The rate of damage accumulation is not fixed but will vary with metabolic processes. In the MGM, this is captured as simply as possible, with damage accumulating passively. We model the accumulated damage $D_2(S)$ in the individual at the end time S at which reproduction is assessed as

$$D_2(S) = k\sum_{s=1}^{S-1}(i^*(s) - i_d), \tag{9}$$

where the optimal activity level to maximize the net growth rate is indicated by $i^*(s)$. Accumulated damage in the RAM is also predicted passively (i.e., is not taken into account when determining the optimal behavior). We model the accumulated damage as

$$D_3(S) = k\sum_{s=1}^{S-1}(i^*(W^*(s), s) - i_d), \tag{10}$$

where the optimal mass at time s is indicated by $W^*(s)$ and the other parameters are as before. Thus, the level of activity determines the level of damage, but in this model the accumulation of damage does not influence the optimal activity level, nor does it affect mortality rate and reproduction.

The RDM allows a more dynamic approach to damage: the level of physiological damage influences the optimal level of activity, and the animal can repair damage in order to reduce its impact on fitness (hence the animal "responds" to the damage, in contrast to the MGM and the RAM, where damage does not influence behavior). Moreover, since damaged cells and tissues have diverse impacts, we let the level of damage influence mortality risk as well as reproductive output. Thus in the RDM, we calculate accumulated damage levels D_4 by time s and activity level $i^*(s)$ as

$$D_4(s + 1) = D_4(s) + k(i^*(s) - i_d). \tag{11}$$

Terminal Fitness and Dynamic Programming Equation

The MGM allows us to predict the maximum possible net growth rate, but it also takes no account of other factors such as mortality risk through predation, so that

$$W(s + 1) = W(s) + \max_i [G(i) - C(i)]. \tag{12}$$

In contrast with the MGM, in the RAM we assume that the aim is to optimize (rather than necessarily maximize) growth rate, taking account of its effect on survival. There is an intrinsic trade-off faced by organisms while foraging: a curtailment of foraging activity (i.e., increased time spent at rest) may reduce predation risk as well as food intake (Houston et al. 1993). That is, because of the impact of foraging on predation (and hence mortality) risk, growth should not necessarily always be maximized.

We next determine the optimal time- and state-dependent pattern of activity for the parameters (which then determines the pattern of growth and survival). We assume that expected reproductive success at the end of the fixed growth interval S, when mass is $W(S)$ is $(W(S) - w_c)^\varepsilon$,

where w_c is a critical mass required for reproduction and φ is an allometric parameter relating mass and reproductive output (table 1).

For previous times, we define fitness as

$$F(w, s) = \max \mathrm{E}[(W(S) - w_c)^{\varphi} \mid W(s) = w], \quad (13)$$

where the maximum is taken over the level of activity and the expectation refers to the probability of surviving from the current time until the end of the growth interval. Then $F(w, S) = (w - w_c)^{\varphi}$, and for previous times,

$$F(w, s) = \max_i [\beta_1(i)F(w + G(i) - C(i), s + 1)]. \quad (14)$$

In the RDM, we now define $F(w, d, s)$ in analogy to equation (14) as representing maximum expected terminal reproduction given the current mass and level of damage. The final condition becomes

$$F(w, d, S) = (w - w_c)^{\varphi} e^{-\mu_b d}, \quad (15)$$

where μ_b is the parameter for mortality rate during the breeding season (note that this is multiplied by the level of damage d, so that damage reduces breeding life span and hence fitness), and for previous times it is

$$F(w, d, s) = \max_i [\beta_2(i)F(w + G(i) - C(i), d + k(i - i_d), s + 1)]. \quad (16)$$

The fitness function in the GARM is now $F(w, o, d, s)$ and depends on current mass, accumulated oocytes, and accumulated damage. The end condition is

$$F(w, o, d, S) = oe^{-\mu_b d}, \quad (17)$$

and for previous times it is

$$F(w, o, d, s) = \max_i \max_{f_w, f_o} [\beta_2(i)F(w + f_w R(i), o + f_o R(i), d - (1 - f_w - f_o)R(i)_\rho + ki, s + 1)]. \quad (18)$$

We solve equations (14), (16), or (18) using backward iteration (Mangel and Clark 1988; Clark and Mangel 2000). At each time and state, we generate the optimal level of activity $i^*(w, s)$ that maximizes the fitness function. Given an initial mass, the trajectory of growth can then be calculated by forward Monte Carlo simulation, feeding the calculated values for optimal activity (given the animal's current size) at each time step into equation (1).

Experimental Design

In order to parameterize the growth models, we used data on the growth of three-spined sticklebacks that were captured with a dip net and minnow traps in the River Endrick, in Scotland (56°04′N, 4°23′W), on November 1, 2007. Fish were kept under an ambient photoperiod throughout the experiment and were initially held at 9.7° ± 0.1°C before the start of experiments. On November 21, 2007, juvenile sticklebacks were sorted into groups of five of differing size (to aid in individual identification), with each group of five fry kept in a separate small tank (335 × 170 × 185 mm). There were two different experimental periods before the breeding season: first, a 4-week temperature-manipulation period, in which four replicate tanks of five fry were randomly assigned to each of three treatment groups (giving a total of 12 tanks), defined in relation to temperature (high [14°C], low [6°C], and intermediate [10°C]). This was followed by a period in which all fish were kept at 10°C until the breeding season. Temperatures were achieved by using temperature-controlled aquarium rooms; at the end of the 4-week temperature-manipulation period, the locations of tanks were randomized with respect to earlier treatment to rule out any effects of the room microenvironment on subsequent growth. We predicted that those individuals that experienced slowed growth by being held initially at 6°C would then show (compensatory) growth acceleration during the second period, while the high-temperature-manipulation group were predicted to show the opposite growth pattern (faster growth during the 4 weeks at 14°C, followed by a deceleration). The group kept at 10°C throughout were predicted to exhibit steady growth. Food was provided ad lib. throughout in the form of a single meal per day of previously frozen *Chironomid* larvae; by recording whether food was present at hourly intervals after feeding, we produced an index of feeding activity in each tank, using a five-point scale from 1 (all food consumed in <1 h) to 0 (still some food left after 4 h).

Fish were remeasured for length and mass every 2 weeks during the temperature manipulations and every 3 weeks thereafter; all fish were starved for 24 h before measuring to reduce variation in the weight of stomach contents. We used only empirical data on the growth of female sticklebacks ($n = 36$), as our models predict reproductive output (which is difficult to quantify in males); fish were sexed and the sexes were separated when males gained nuptial coloration. Female sticklebacks are capable of producing a series of clutches throughout the breeding season (Wootton 1976), and so whenever they became fully gravid with eggs they were manually stripped and the clutch mass was recorded (see detailed experiments description in Lee et al. 2010). All statistical analyses of growth rate used tank

as a random factor to control for the nonindependence of data points from the same tank (full details of analyses of the empirical data are given in Lee et al. 2010).

Compensatory Growth Rate

For both models and experiments, we computed the compensatory growth rate (CGR; % per time s) after the temperature manipulation as

$$\mathrm{CGR} = 100\frac{\ln\left[W(s_c)W(s_i)^{-1}\right]}{s_c - s_i}, \tag{19}$$

where $W(s_i)$ is the initial wet mass at the end of the manipulation period and $W(s_c)$ is the wet mass when fish in the different manipulation groups had finished the phase of compensatory growth and had appeared to converge on the same mean mass before breeding (on the basis of inspection of growth trajectories).

Simulation and Model Comparison

The models are generally applicable to any vertebrate ectotherm, but in order to compare the performance of the various growth models in relation to the observed data we randomly generated a population of 20 juvenile fish with the same mean and standard deviation for initial body mass as the observed data. Using growth data of fish held at a constant 10°C, we fitted models to the empirical data, finding the best fit by changing parameter values as appropriate. Since the empirical data come from fish that were already juveniles at the time of first capture, we were unable to fit the models to observed growth trajectories over the first few months of life. In addition, our data fitted better to a nonsigmoid curve ($r^2 = 0.868$) than a sigmoid one ($r^2 = 0.690$), each with two parameters, and so we developed the models as a nonsigmoid curve. However, our focus in this study is to compare model predictions of the responses to growth perturbations in the time leading up to reproduction rather than to accurately model growth throughout development.

We now must consider a subtlety of model comparison. The four models each predict the pattern of growth, activity, and damage accumulation, but only one of them (GARM) predicts reproductive output at the end of the season. We have empirical data on growth, activity, and reproductive output to compare against model predictions but no measurements of the accumulation of damage, so that measurements of damage remain as a testable prediction from the models. Furthermore, only one of the models predicts reproductive output. Comparing models in a situation like this one is a topic for which even the most recent statistical literature on model selection is silent (Krnjajić and Draper 2011).

One solution to this problem would be to expand the MGM, RAM, and RDM models to include reproductive output. To do so, we used an indirect approach to predict final gonad size in all models. The maximum mass of eggs that can be produced by female fish is known to be tightly related to body mass (Wootton 1998). We therefore assume this maximum reproductive tissue to be

$$O_{\max}(w) = 0.25w, \tag{20}$$

where 0.25 is the mean observed ratio of total clutch mass (over the entire breeding season) to body mass (measured at the start of the breeding season) from the experimental data. However, if an individual has incurred significant damage during early life, then the total resources available to produce eggs may be reduced. Thus, we assume that a fish's total reproductive mass will be reduced in proportion to the damage it has accumulated:

$$O(W(S)) = 0.25W(S) - k_c D(S), \tag{21}$$

where the parameter for clutch damage k_c is set to 10.5. In the GARM, the reproductive tissue (here envisaged as oocytes) $O(s)$ at time $s + 1$ is described by

$$O(s + 1) = O(s) + f_O R(i). \tag{22}$$

This solution, while seemingly scientifically defensible, may not be statistically defensible (Krnjajić and Draper 2011; D. Draper, personal communication; S. Fienberg, personal communication) since the addition is an ad hoc extension of the model, with no method of computing an associated likelihood. However, it allows us to compare all four of the models with respect to reproductive output.

Consequently, we compared experimental data with all four models' predictions on the basis of the values that they all predict (growth trajectories at a constant 10°C, compensatory growth trajectories after a growth perturbation [low- and high-temperature manipulation], and feeding activity) and using the ad hoc extension for reproductive activity. We did this using Akaike Information Criteria (AIC; Akaike 1973), assuming that observational errors are normally distributed with variance proportional to mass.

We ran forward versions of the MGM, RAM, and RDM in R (R Development Core Team 2007) and the GARM in Microsoft Visual Basic 2008 Express (Microsoft 2008).

Results

Optimum Growth and Activity Patterns

In order to find appropriate growth rate parameter values for the stickleback system, we ran the MGM, the RAM,

the RDM, and the GARM with a wide range of parameter values until model outputs best matched the observed growth trajectories of fish kept at 10°C throughout. Using the parameter values in table 1, the predicted growth trajectories from the models were similar to those observed in the experiment (fig. 2). All models were able to broadly replicate the observed growth trajectories for fish in the intermediate-temperature group (i.e., a constant 10°C temperature), although they all predicted a greater reduction in the within-population variation in mass over time than was observed in the experimental data (fig. 2). The GARM provided the best fit to the experimental results obtained at 10°C (table 2).

Results from each model led to the prediction of an acceleration in growth during the period after the temperature reduction (i.e., when the fish were transferred from 6°C to 10°C) relative to those from the model kept at 10°C throughout. In addition, fish in the high-temperature group (which had experienced a period at 14°C) were predicted to show decelerated growth when transferred to 10°C. The predicted strength of this compensation differed between the models, with the least compensation (whether in terms of accelerated or decelerated growth) predicted by the MGM and the strongest predicted by the GARM (which was marginally the best at matching the experimental data; table 2). However, none of the models predicted as extreme an acceleration of growth as was observed in the experimental fish in the low-temperature group (fig. 3; table 2).

The MGM predicted that activity levels of the three

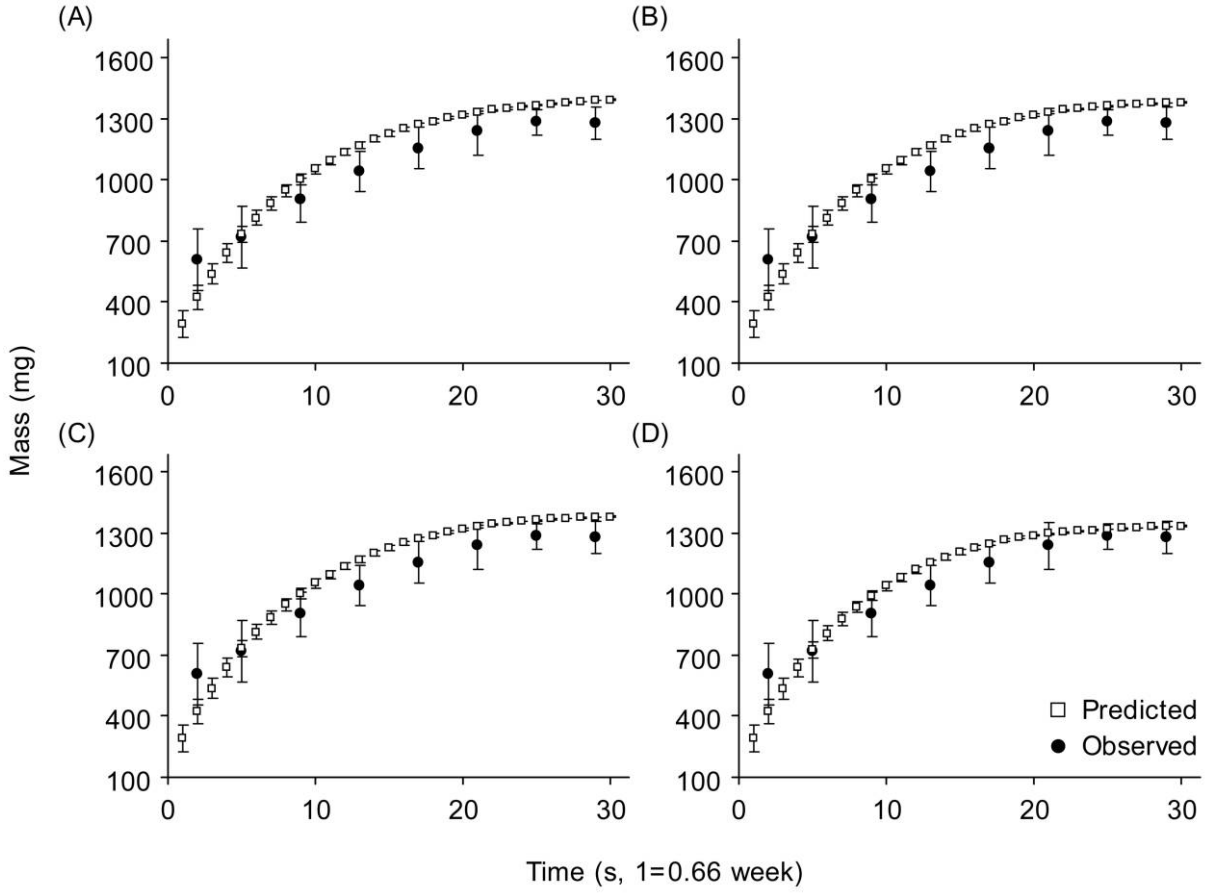

Figure 2: Predicted and observed growth trajectories at time $s = 1$ to time $s = 30$ for fish under conditions of ad lib. food and constant 10°C. The four plots show the predicted optimized growth trajectories (open squares; mean mass ± SD) for a simulated population of 20 fish with the same initial mean size and SD as the experimental population (see "Material and Methods") according to the four growth models: *A*, the maximize growth model; *B*, the response to activity model; *C*, the response to damage model; and *D*, the gonadal accumulation and repair model. Note that the error bars are indistinct at later time periods because of a predicted reduction in the variation in size among individuals over time. The closed circles and error bars show the observed mean size ± SD of three-spined sticklebacks in the intermediate-temperature (i.e., constant 10°C) group in the lab experiment.

Table 2: Comparison of experimental data with the predictions of the four models

Model	AIC	ΔAIC	AIC weight
Growth trajectory at 10°C:			
MGM	255.2	4.0	.089
RAM	254.5	3.2	.131
RDM	254.5	3.2	.131
GARM	251.2	0	.651
Compensatory growth:			
MGM	121.7	5.2	.057
RAM	121.3	4.7	.072
RDM	120.7	4.1	.098
GARM	116.5	0	.773
Feeding activity:			
MGM	286.7	4.5	.073
RAM	285.8	3.6	.115
RDM	285.8	3.6	.115
GARM	282.1	0	.697

Note: Data are Akaike Information Criteria (AIC), AIC differences (ΔAIC), and AIC weights for the four models. Comparisons were made on the basis of the values that the models all predict, without the additional assumption concerning reproductive output. Growth trajectories were at a constant 10°C; compensatory growth trajectories were after a growth perturbation (low- and high-temperature manipulation) and feeding activity. MGM, maximize growth model; RAM, response to activity model; RDM, response to damage model; GARM, gonadal accumulation and repair model.

temperature-treatment groups should remain at maximal levels (since in this model there is no cost to activity). However, the three other models (RAM, RDM, and GARM) lead to predictions that activity levels would change over time and would differ between the treatment groups. The three models gave broadly similar patterns of maximal activity in all fish during the temperature-manipulation period but then a decline when all fish were at 10°C, with the activity of the high-temperature group declining first, followed by the intermediate-temperature group and finally the low-temperature group (fig. 4). The GARM predicted a smaller difference in activity between treatment groups than the RAM and RDM, which predicted that the activity of fish in the high-temperature group would have dropped to very low levels by the onset of the breeding season (fig. 4). The pattern predicted by the GARM was the closest match to the observed data on feeding activity (i.e., time taken to consume each meal; fig. 4; table 2).

Accumulated Damage

While the MGM predicted the same value of accumulated damage for the three temperature-treatment groups (since activity levels did not vary between them and there was no repair of damage in this model), other models (RAM, RDM, and GARM) predicted that the accumulated damage (measured at the end time *S*, at which reproduction is assessed) would differ among the experimental temperature groups (low-, intermediate-, or high-temperature treatments; fig. A2, available online): in each of these three models, the damage levels were predicted to be highest in animals initially subjected to the low temperature and least in those initially exposed to warmer temperatures; the predicted relative levels of accumulated damage were remarkably similar across these three models despite their different assumptions (see details in the appendix, available online), suggesting that the optimal activity pattern is little affected by taking the consequences for damage into account (i.e., comparison of RAM and RDM predictions).

Investment in Reproductive Tissue

The GARM incorporates resources that are allocated over time to gonadal growth. The prediction from this model was that initially there should be no gonadal growth but

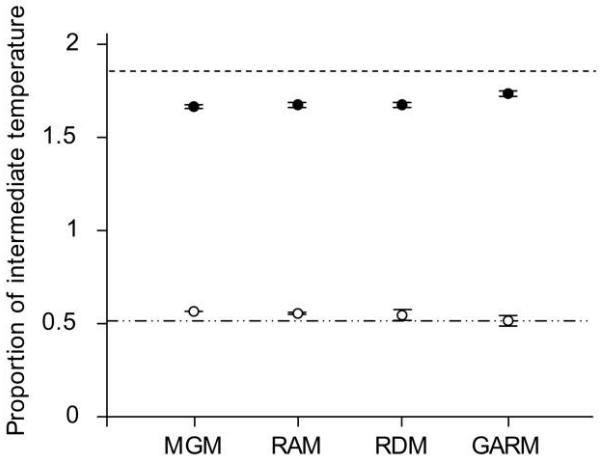

Figure 3: Predicted growth rates over the period from $s = 7$ to $s = 25$. This time corresponds to the period when the temperature was 10°C after a 4-week period (from $s = 1$ to $s = 6$) when it was either 6°C (low-temperature group; filled circles) or 14°C (high-temperature group; open circles). Values are expressed as a proportion of the predicted growth rate of the intermediate-temperature group, which was held at a constant 10°C. Predicted growth rates are shown for the four separate models: the maximize growth model (MGM), the response to activity model (RAM), the response to damage model (RDM), and the gonadal accumulation and repair model (GARM). The dashed line and the dotted-dashed line indicate the means of observed values for the low-temperature and high-temperature groups, respectively, similarly expressed as a proportion of the observed growth rate of the intermediate-temperature group. Data are shown as mean values ± SD for the simulated populations (see text for explanation).

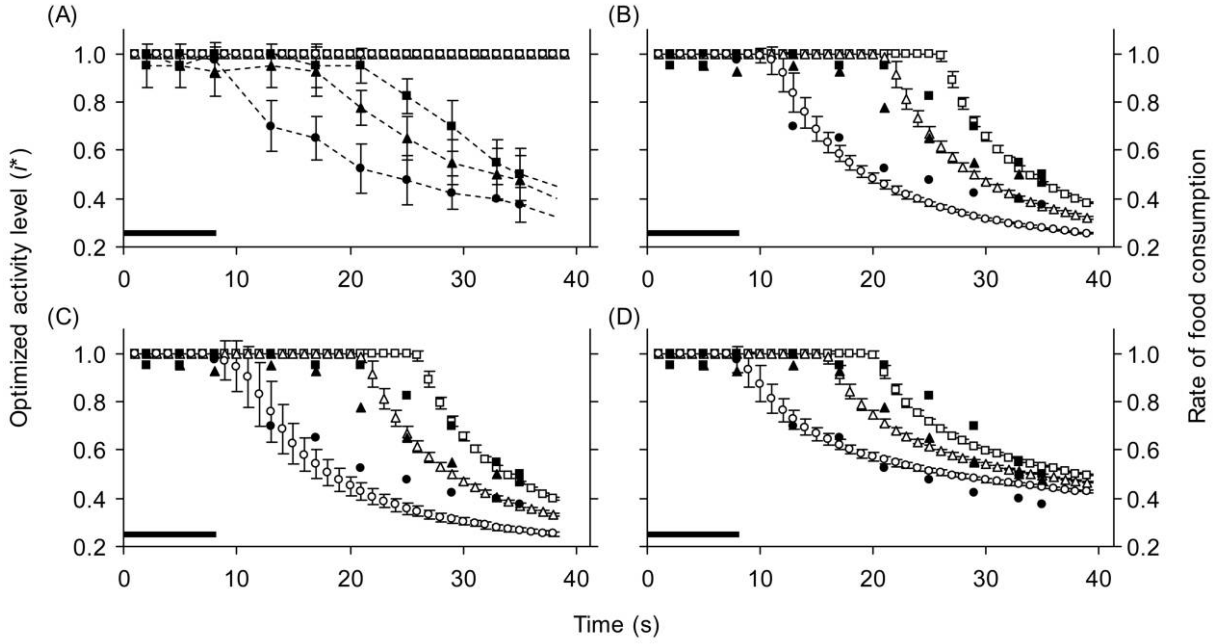

Figure 4: The optimum activity levels (i^*) of individuals in the three temperature-treatment groups (high [14°C], open circles; intermediate [10°C], open triangles; low [6°C], open squares) as predicted by the four different models (*A*, maximize growth model; *B*, response to activity model; *C*, response to damage model; and *D*, gonadal accumulation and repair model). Data show the mean ± SD predicted activity for the simulated populations of 20 fish per treatment. Also shown with filled symbols are the observed times taken by experimental fish to consume food after presentation of food (expressed by the rate of food consumption). Data are shown separately for the three temperature-treatment groups (high, circles; intermediate, triangles; low, squares; values are plotted as means ± SD in *A*, but in *B–D* only the mean values are plotted for clarity). The thick bar indicates the period during which temperatures differed between the groups, after which time all fish were at 10°C.

that it should accelerate later as the breeding season approached (fig. 5). There were effects of the mortality rate when active (μ) on the predicted growth of reproductive tissue, with reduced/suppressed investment in the gonads when the mortality risk during foraging was higher (fig. 5). Furthermore, the temporal pattern of reproductive investment was predicted to differ between the temperature treatments, with an earlier onset of gonadal growth in the higher-temperature treatments but equal rates of growth thereafter (leading to larger final gonad size in the higher-temperature treatments).

Although fish in all three groups reached a similar mean size by the time of spawning (fig. A1, available online), their observed egg production was different, with high-temperature females producing more eggs and low-temperature females producing fewer eggs than the intermediate-temperature females. A similar pattern was predicted by all but the MGM, with the GARM predicting values that are marginally closer to the observed data than the other two (fig. 6). Thus, the observed reproduction allocation of the low-temperature treatment was 82% of the control, while our prediction from the GARM was 85% ± 1%; conversely, the reproductive allocation of females from the high-temperature treatment was observed to be 138% of that of the intermediate-temperature females, and our prediction from the GARM was 123% ± 1% (fig. 6).

We thus conclude that the GARM can better predict both steady and compensatory growth trajectories as measured by the AIC weight when comparing predicted and observed results (table 2), and it is capable of predicting reproductive output without the need for prior assumptions of the link between reproductive output and damage, something that none of the other models can do.

Discussion

The dynamic-state-dependent models presented here are applicable to any vertebrate ectotherm (and have relevance for other animal groups) but were validated using data from our model system of the three-spined stickleback. Our theoretical and empirical results suggest that growth trajectories in early life affect later fitness. We have assumed that the cause of the reduction in fitness is an

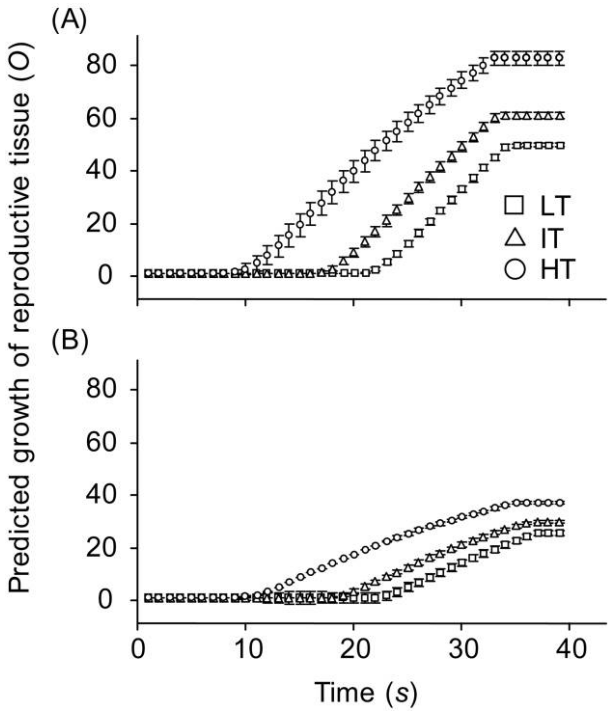

Figure 5: Effects of mortality rate when active (μ) on the growth of reproductive tissue ($O(s)$) in the gonadal accumulation and repair (GARM) model. The panels illustrate different values for the mortality parameter ($\mu = 0$ [*A*] and 0.015 [*B*]); in each case, the predictions are plotted separately for the three temperature-treatment groups, low (squares), intermediate (triangles), and high (circles).

increased activity level (necessary to achieve a higher food intake), which causes both a higher mortality risk (e.g., through predation) and an accumulation of damage to molecules, cells, and tissues. All but the simplest model were able to approximately replicate the different growth trajectories seen in the experimental fish resulting from variations in activity levels: accelerated growth (induced by a period of lower temperatures) was associated with longer periods over which the activity level was maximized, whereas decelerated growth (seen in the high-temperature group) was linked to reduced activity levels. These variations in growth trajectory were predicted to lead to differences in damage levels, with the accelerated growth trajectory having the highest expected damage and the decelerated trajectory having the least. Therefore, variations in growth rate induced by environmental temperatures in early life were predicted to cause differences in the accumulation of physiological damage as a consequence of changes in the optimal level of activity. In the more complex models, this variation in damage levels influenced reproductive investment (i.e., the timing and extent of egg production), with a greater accumulated damage level causing a lower expected reproductive rate and delayed breeding since animals would require more time and investment to repair tissues before breeding. However, the close similarity of the RAM and RDM predictions suggests that taking into account the future effect of damage on breeding performance has little impact on the calculated optimum level of activity.

The theoretically predicted effects of damage have a well-established empirical basis. It is well known that compensatory growth, particularly growth acceleration, has both costs and benefits in many species from diverse taxa (Arendt 1997; Metcalfe and Monaghan 2001). In our particular model system (sticklebacks) it may increase the chance of reproduction (Wootton 1976, 1998), but it also reduces locomotor performance (Álvarez and Metcalfe 2005; Lee et al. 2010) and life span (Inness and Metcalfe 2008). The costs of growth may be general to all animals: recent reviews (Monaghan et al. 2009) have highlighted the likelihood of elevated levels of reactive oxygen species during juvenile development due to the high metabolic activities required for growth. It is possible that animals mount defenses against such free radical attacks (see De Block and Stoks 2008), but this increased investment will itself be a cost even if it prevents the faster accumulation

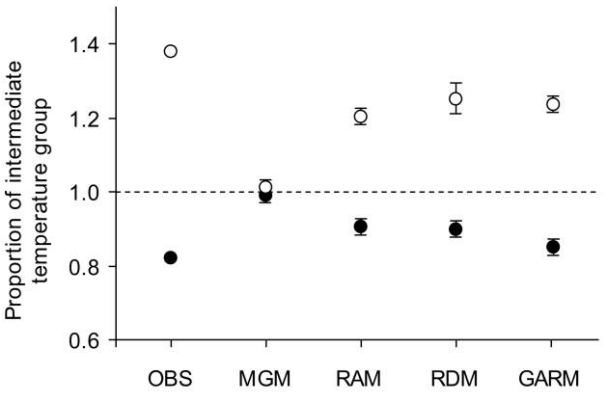

Figure 6: Investment in reproduction, quantified as the total mass of eggs produced during the breeding season for the observed (OBS) experimental data and the total reproductive mass for the four models. Values are shown for the low-temperature (filled circles) and the high-temperature (open circles) treatment groups, and are expressed as a proportion of the mean value for fish in the intermediate-temperature group; data are plotted as means ± SD for the observed and simulated populations. MGM, the maximize growth model; RAM, the response to activity model; RDM, the response to damage model; GARM, the gonadal accumulation and repair model. The output for the GARM is shown for the calculation based on modeled ovary growth, while the other three models calculate the output only on the basis of final somatic mass and accumulated damage (see text for explanation).

of damage. Our models and the experimental data also showed "negative compensatory growth" after an earlier period of higher-temperature treatment, with growth rates suppressed compared with animals that had been at a constant temperature. The theoretical models predicted that this growth deceleration would be associated with a lower accumulated damage level; although damage levels were not measured as a part of this study, there is nonetheless indirect support for this from the finding that the growth deceleration after temperature manipulation had a positive effect on locomotor performance (Lee et al. 2010) and reproductive investment (Lee et al., forthcoming). Conversely, as a consequence of rapid growth rate incurring a higher level of oxidative stress as a result of an elevated metabolic rate, the models predicted an elevated level of accumulated damage in animals that had undergone accelerated growth (Criscuolo et al. 2008; Metcalfe and Alonso-Alvarez 2010).

All but the MGM predicted differences in reproductive investment between animals with contrasting growth trajectories, even if by the breeding season their sizes did not differ. Thus fish undergoing accelerated growth were predicted to have the lowest investment while the decelerated growth fish were expected to have the highest. The biggest effects of growth trajectory on reproduction were predicted by the GARM, which also predicted that the onset of oocyte investment would be faster in the decelerated growth than in the accelerated growth. The predictions of the GARM best matched the observed differences in egg production between growth treatment groups. Furthermore, this was the only model to predict reproductive investment without an ad hoc assumption about the link between damage and reproductive output, making it a better model scientifically as well as statistically. The empirical results for breeding output, described in more detail by Lee et al. (forthcoming), have parallels in the recent study of Auer et al. (2010), who showed that accelerated compensatory growth (induced by prior food deprivation) reduced the rate of offspring production in female guppies *Poecilia reticulata*. While we did not explicitly include the effect of growth trajectory on male reproduction, we have experimental data showing that the duration of nuptial coloration in male sticklebacks varies with growth pattern, being shortest in accelerated growth groups (Lee et al., forthcoming). This may again be associated with damage levels, since Pike et al. (2007) found that high levels of oxidative damage (in this case, caused by reduced availability of dietary antioxidants) led to male sticklebacks being less able to invest in their nuptial signal. However, an unusual aspect of our study was the positive effect of decelerated growth, with both models and data showing that fish undergoing rapid growth early in life but suppressed growth in the lead-up to the breeding season perform better than those fish growing steadily throughout their juvenile lives. Our models suggest that the positive as well as the negative long-term effects of early growth rate arise through changes to the rate of accumulation of physiological damage, but this remains to be tested by empirical measurements.

Environmental conditions affect both early development and their ecological consequences in later life. There are clear costs of impaired development in early life, but natural selection is expected to lead to a life-history strategy that reduces these negative effects so as to maximize expected reproductive success (e.g., through compensatory growth). This is illustrated by the predictions of our models, particularly those related to the optimal level of activity, which take into account the trade-off between early growth rate and accumulated damage level. The optimal activity level was predicted to be the maximum that is possible when the organisms are still relatively small (Swallow et al. 1999; Tou and Wade 2002). While the animals undergoing accelerated growth were predicted to continue at maximal activity for longer in order to catch up in body size and hence improve their probability of survival and successful reproduction (e.g., Kraak et al. 1999; Garvey et al. 2004), all but the simplest model predicted that the optimal activity level would then drop, especially in the decelerated growth group during the compensatory period. A decreased activity level may be selected for in animals that have previously experienced good growing conditions, since it reduces both the risk of mortality and the level of damage accumulation by the onset of the breeding season. If such females were instead to maintain a maximized activity level, they would be larger still by the time of the breeding season if they survived that long, but the high level of damage by accumulation would (according to the GARM) reduce and delay their capacity to produce eggs, thus having a net negative effect on reproductive success. Therefore, animals undergoing decelerated growth are predicted to reduce their activity (and hence mortality risk and damage and the need to divert resources into repair) and save the surplus resources for egg production rather than growth. Benefits are thus maximized over the long rather than the short term. Thus, our results suggest that the trade-offs between early growth rate, mortality, and accumulated damage level would result in the optimal activity level being determined by a maximization of future reproductive investment.

It is well documented that early growth rates have long-term consequences, but the pattern of growth observed (especially the degree of compensation for an earlier period of altered growth) still varies between different species and contexts. Some of this variation may be due to the current environmental conditions: for example, our models predicted that the differences in compensatory growth rate

and accumulated damage level between temperature-manipulation groups would disappear when the mortality rate (predation pressure) was higher (tables A1, A2, available online). Predation pressure affects the balance of time spent feeding versus being inactive (Beukema 1968), since more active organisms are more likely to be caught by a predator. While there is no direct evidence of the effects of predation risk on compensatory growth rate (Dmitriew and Rowe 2005; Stoks et al. 2005), our findings suggest that predation pressure may nonetheless influence (both directly and indirectly) the trade-off between compensatory growth rate and its long-term consequences.

The four models predicted similar patterns of growth, especially for animals in constant conditions, but they differed in the accuracy with which they matched the empirical data, as a result of each model being based on different assumptions (see "Material and Methods"). The predictions of the GARM, which is the most complex model, were the best match for the experimental data, while the simplest model (MGM) was least able to match the observed patterns since it led to the prediction of the same levels of optimal activity, accumulated damage, and reproductive investment irrespective of temperature treatment. Many studies (Ricklefs 1969; Case 1978; Sibly et al. 1985; Arendt and Wilson 1997; Mangel and Munch 2005) found that growth models that excluded a consideration of the costs of growth (i.e., the damage level) could not adequately predict compensatory growth patterns, and the poor predictive power of the MGM is likewise a consequence of the assumption that growth can be maximized without any effect on mortality rate. While the RAM and the RDM predicted similar growth trajectories under constant conditions and similar activity levels, the pattern of compensatory growth and reproductive output was marginally better predicted by the RDM since a consideration of damage was included in the optimal decision process, whereas growth in the RAM was determined independently of the damage level. The GARM incorporated the idea of resource allocation between growth, damage, and repair, and produced the most accurate predictions. This supports the approach of Mangel and Munch (2005) and also suggests that the greater the extent to which life-history theory is incorporated into growth models, the better the predictions.

In conclusion, we have shown by the use of four dynamic-state-dependent models that a consideration of the costs of rapid growth (in terms of its effect on immediate mortality risk, long-term damage accumulation, and future reproductive investment) allows prediction of complex growth trajectories that match empirical data. Moreover, the predictions are better when the models include more aspects of this trade-off between the benefits and costs of rapid growth. Our results also emphasize that animal growth trajectories may take account of life-history consequences—ultimately, fitness—as well as current ecological conditions.

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