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REPORT

Growth autocorrelation and animal size variation

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Abstract

It has long been recognized that variability in animal size is affected by how individual growth rate is autocorrelated in time. Earlier studies have attributed the mechanism generating the autocorrelation primarily to size-dependent growth rate and autocorrelation in resource abundance. All of these studies have shown that positive autocorrelation in individual growth rate always translates into increased variability in size. We show that energy reserves in individuals induce growth autocorrelation by acting like a low pass filter between the resource and the internal energy that is available for metabolism, growth and reproduction. However, the reserve also reduces the variance in growth rate. Consequently, reserve-induced growth autocorrelation has relatively little effect on size variability in the population, contradicting existing ideas about the relationship between the growth autocorrelation and size variability.

Keywords

Bioenergetics, dynamic energy budget, energy reserves, fluctuating food, growth autocorrelation, low-pass filter, pink noise, size variability, stochastic growth model.

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INTRODUCTION

Animal size varies naturally among individuals born at the same time and place, despite growing under what appears to be the same environmental conditions. For example, a cohort of fish often exhibits substantial size variability (Ricker 1958; DeAngelis *et al.* 1993; Quinn & Deriso 1999). The origin of such size variability is not fully understood. Nevertheless, size is a good predictor of individual performance in survival and reproduction (section 3.2.1 of Caswell 2001), and individual size is also one of the easiest variables to measure in the field (Quinn & Deriso 1999). Consequently, the basis of size variability is of great scientific interest.

Ricker (1958) (section 9G) recognized that the variability in individual size increased within the same cohort through time when the individual growth rate was positively correlated with itself in time (autocorrelated). Ricker called this phenomenon 'growth depensation.' Originally, growth depensation was attributed to a positive correlation between size and individual growth rate. This correlation is a common feature among young, growing individuals (Ricker 1958) because larger individuals are more capable of capturing food, and thus have more potential to grow quickly. In this scenario, when individuals grow large during one time period, they tend to grow even larger again during the following time period while smaller individuals do not perform as well. This produces positive autocorrelation in the growth over time. Later, using individual-based models, DeAngelis *et al.* (1993) demonstrated that autocorrelation in general, regardless of its origin, could potentially broaden the variability in animal size at any given age.

Recently, Pfister & Stevens (2002) demonstrated that even after accounting for the positive correlation between size and growth, a variety of species exhibited additional autocorrelation in growth rate, and showed that that was important in determining the size variability within a cohort. We called this additional type of autocorrelation 'residual autocorrelation' to emphasize that it was the autocorrelation in residuals of individual growth rate after removing the size effect. Subsequently, Pfister & Peacor (2003) showed that when organisms were inefficient in detecting the location of abundant food in a spatially structured resource environment, they could induce heterogeneity in resource availability. The authors further demonstrated that the induced heterogeneity might generate the residual autocorrelation in growth rate.

Here, we demonstrated that residual autocorrelation could also be generated by physiological delays *within* an organism that encountered a fluctuating resource. We considered simple models of individual growth in which energy obtained from food was transferred to an energy reserve (or buffer) and ultimately to the structural biomass of an individual. We showed that the reserve translates randomly fluctuating but

serially uncorrelated signals in food into an individual growth rate that was positively autocorrelated. We called this 'physiologically induced residual autocorrelation' to distinguish it from the previously described resource-induced 'residual autocorrelation' (Pfister & Stevens 2002; Pfister & Peacor 2003). Some form of reserve is a ubiquitous feature of living organisms and allows individuals to store energy or nutrients for future use; thus, physiologically induced residual autocorrelation is likely to occur widely. However, unlike resource-induced residual autocorrelation, we showed that physiologically induced residual autocorrelation is associated with *reduced* variability in size.

We studied residual autocorrelation using two models. Our first model was a fully specified dynamic energy budget model (Kooijman 2000). We used it to investigate (numerically) ontogenetic change in size variability for organisms subsisting on a randomly fluctuating resource. Next, we investigated the residual autocorrelation (analytically) using a simple caricature of the energy flow within an organism. This second model took the form of a set of linear difference equations with stochastic forcing, and made explicit the mechanisms whereby reserves influence growth rate autocorrelation.

MODELS OF GROWTH IN A FLUCTUATING ENVIRONMENT

Organisms encounter and assimilate resources (henceforth termed 'food') from the environment. The energy content of the food is transformed into a metabolically inactive storage and distribution form (such as protein or fat) that we term 'reserves.' Energy is subsequently extracted from reserves and used for metabolic maintenance, growth and reproduction. The generic pathways are displayed in Fig. 1a, and have been extensively investigated using dynamic energy budget (DEB) models (Kooijman 2000).

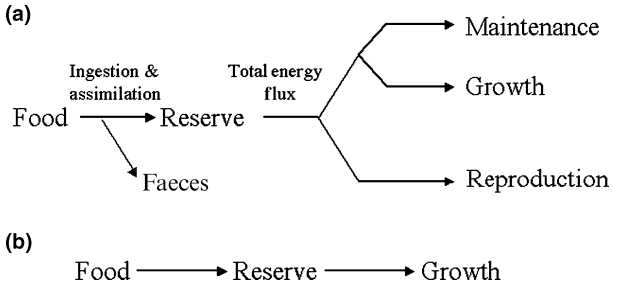

Figure 1 Energy flow diagram. (a) Basic energy allocation scheme. Food is ingested, assimilated and stored in reserve. The stored energy is used for individual functions such as maintenance, growth and reproduction. See Kooijman (2000) for detail. (b) Simplified energy utilization scheme. Food is stored in a reserve as energy, and it is used for growth.

A reserve fulfils multiple roles, most notably energy storage for a period of food shortage. Our focus here is a previously unexamined role played by the reserve in translating food variability into growth and size variability. In the models that follow, we assume that an individual's food assimilation rate through time is a stochastic process, which might be temporally autocorrelated. When we examine properties of cohorts, we assume that each individual's food environment is independent, but with common statistical properties.

Dynamic energy budget model

We first investigate the implications of a stochastically fluctuating food environment using a stochastic version of Kooijman's dynamic energy budget model (Kooijman 2000). The model variables and equations are detailed in Table 1. Below, we summarize the model assumptions, but for details and for broader discussion of the underlying rationale, readers are referred to Nisbet *et al.* (2000) and Kooijman (2000). For further discussion of issues relating to variable environments, see Muller & Nisbet (2000).

The model represents a sequence of energy transformations illustrated in Fig. 1a. The organism is initially characterized by two state variables: structural biovolume (V) – hereafter called 'size' – and density of stored energy (E). Ingestion rate is assumed to depend multiplicatively on the organism's surface area ($V^{2/3}$) and on food density through a Holling type 2 functional response. Assimilation efficiency is constant. Energy is released from the reserves for growth, reproduction, metabolic maintenance and reproductive maintenance at a rate determined by an equation representing *reserve homeostasis* (see Section 3.4 of Kooijman 2000). By default, a fixed fraction of the flux from reserves is assigned to reproduction (or in juveniles for development); the remainder is used for maintenance and growth with priority to maintenance. Maintenance rate is proportional to size.

The model has special rules that apply when food is scarce. If the default allocation of energy to growth plus maintenance is insufficient for maintenance alone (a situation we call 'moderate starvation'), growth stops, and maintenance needs are met by reducing reproduction.

At very low food levels (a situation we call 'severe starvation'), the individuals must increase the flux from the reserve to meet the minimum requirement for survival. This severe starvation has previously been modeled in various ways (e.g. chapter 7 of Kooijman 2000; Muller & Nisbet 2000). Here, we assume that starving individuals must meet all maintenance needs (including a component called 'maturity maintenance' by Kooijman). If individuals deplete the reserve completely, they starve to death.

The model variables and parameters are detailed in Table 1. For the current investigation, a particularly important

Table 1 Dynamic energy budget model. This table was adapted from Table 2 in Nisbet et al (2000). Parameter values are from Muller and Nisbet (2000); these are set for marine mussel (*Mytilus edulis*) growth

Parameters/variables	Description	Value/unit*
State variables		
$V(t)$	Structural biovolume at time t	$V(0) = 0.0001$
$E(t)$	Energy density in reserve at time t	$E(0) = 0.75$
Other variables		
$z(t)$	Noise in the logarithm of food (see eqn 6)	$z(0) = 0$
$\eta(t)$	Standard Gaussian white noise	
$f(t)$	Scaled functional response (see eqn 7)	$f(0) = 0.75$
Parameters		
A_m	Maximum assimilation rate per surface area	$0.075a$ J m^{-2} y^{-1}
E_m	Maximum reserve per unit size	$0.1a$–$2.0a$ J m^{-3}
M	Maintenance energy per unit size per unit time	$0.375a$ J m^{-3} y^{-1}
G	Energy costs for a unit increase in size	$0.643a$ J m^{-3}
V_p	Volume at maturity	3.3×10^{-4} m^3
τ	Memory retention time of food	0.01–1.0
S	Intensity of food fluctuation	0.09
κ	Fraction of utilized energy spent on maintenance and growth	0.5
m	Maintenance rate coefficient $m = \frac{M}{G}$	y^{-1}
v	Energy conductance rate $v = \frac{A_m}{E_m}$	m y^{-1}
g	Energy investment ratio $g = \frac{G}{\kappa E_m}$	
Dynamics	Under abundant food and moderately starved conditions $\frac{dV(t)}{dt} = \frac{\left(vV^{2/3}E(t)/E_m - mgV(t)\right)_+}{g+\frac{E(t)}{E_m}}$ $\frac{dE(t)}{dt} = \frac{A_m}{V^{1/3}(t)}\left(f(t) - \frac{E(t)}{E_m}\right)$ Under severely starved condition $\frac{dV(t)}{dt} = 0$ $\frac{dE(t)}{dt} = A_m f(t) V^{2/3}(t) - \frac{1}{E_m}\left[\kappa mg + (1-\kappa)mg\min\left(V(t), V_p\right)\right]$	

*a: a factor that converts dry weight to energy content in Joules. In the papers used by Muller and Nisbet (2000) to estimate parameters, dry weight was used as a proxy for energy content of organisms. After reparameterization, this factor disappears.

parameter is the maximum reserve density E_m. Reducing E_m reduces the capacity of the reserve, and when $E_m \approx 0$, the model reduces to a simple growth model with no reserve.

We are interested in the response of the organisms to stochastically fluctuating food. We assume that the logarithm of food has a constant long-term mean with an added pink noise, $z(t)$, with memory retention time τ. Pink noise can be simulated as filtered white noise using a stochastic differential equation (see chapter 7 of Nisbet & Gurney 1982). Thus

$$\frac{dz(t)}{dt} = \frac{-z(t)}{\tau} + \left(\frac{S}{\tau}\right)^{1/2}\eta(t) \qquad (1)$$

where $\eta(t)$ is white noise and S is a constant that determines the intensity of the fluctuations. The amount of food available at time t is proportional to $\exp(\alpha + z(t))$. eqn 1 is formulated so that the variance of $z(t)$ (thus also of $f(t)$) remains constant with different values of the parameter τ. The scaled functional response is related hyperbolically to the food density; thus we write

$$f(t) = \frac{\exp(\alpha + z(t))}{1 + \exp(\alpha + z(t))} \qquad (2)$$

where α is related to the mean level of functional response, and we arbitrarily chose its value so that $\exp(\alpha)/(1 + \exp(\alpha)) = 0.75$.

When the scaled functional response is given by eqns 1 and 2, the model in Table 1 takes the form of a system of stochastic differential equations. They were simulated using the Ito interpretation of stochastic differential equations (Higham 2001). The equations in Table 1 and eqns 1 & 2 were

simulated simultaneously from $t = 0$ to 50 with time step $\Delta t \approx 1.9 \times 10^{-4}$. The simulation was individual-based, and we repeated it 1000 times to obtain 1000 trajectories of $f(t)$ and $V(t)$ (i.e. 1000 individuals that are experiencing independent noise). Then the result was saved at every 0.0488 time step from t = 0 to 50. Initial values and parameter values used are listed in Table 1. The simulation was repeated for each set of $\tau(\tau = 1,\ 0.1\ \&\ 0.01)$ and E_m ($E_m = 2.0,\ 1.0\ \&\ 0.1$), representing different scenarios in the amount of the food autocorrelation and the size of the reserve. This resulted in 1000 trajectories of scaled functional response $f(t)$, reserve density $E(t)$ and $V(t)$ under each scenario. We then calculated the residual autocorrelation in growth as following. Let $V_i(t)$ be the size of individual i at time t ($t = 0, 0.0488, 0.0976, \ldots$). We first calculated the growth rate as $\Delta V_i(V_i(t)) = V_i(t + 0.0488) - V_i(t)$. Based on the initial size $V_i(t)$ at time t, the growth rate was categorized into 1000 size classes ranging from the minimum to the maximum sizes. This was repeated for all individuals. Then the growth rates within the same size classes were pooled to obtain the mean growth rate for given size classes. The mean size-specific growth rate was subtracted from $\Delta V_i(V_i(t))$ to obtain the residual of the growth rate. Finally, we calculated the autocorrelation in the residual.

Sample trajectories of the functional response, reserve density, and individual size are plotted in Fig. 2a–c. The first two panels in Fig. 2 show that as the variability in food is transmitted to the reserve, it is smoothed, with the response in size even smoother. Note that our model does not allow individual size to shrink. Therefore, the individual size trajectories are a strictly increasing function of time.

Figure 2d–f shows the variances in scaled reserve density, growth rate, and individual size when $\tau = 1.00$ and $E_m = 1.00$. The variance of the reserve reaches an approximately constant value quickly (Fig. 2d). This is true for all combinations of τ and E_m that we investigated. However, the variances of the growth rate (dV/dt) and size always have an increasing period (growth depensation) followed by a decreasing period (growth compensation).

We investigated how variability in size is affected by autocorrelation in food and the size of the reserve by comparing the maximum variances of the reserve, the growth rate, and size, which correspond to the highest points in Fig. 2d–f. Table 2 shows their result for the nine

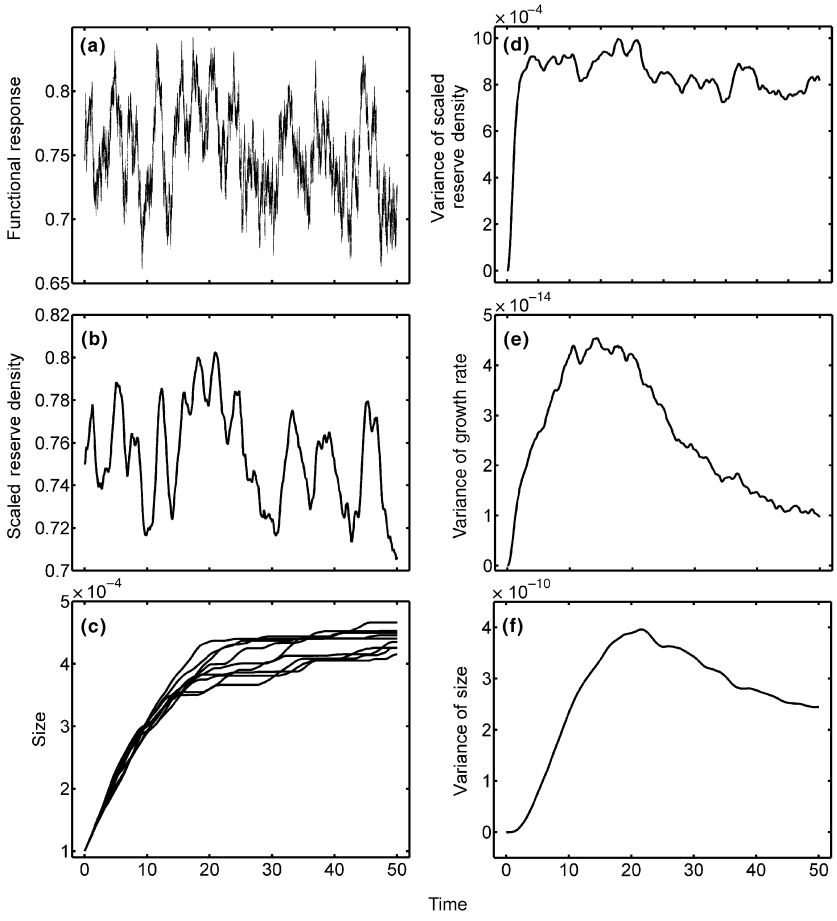

Figure 2 Individual based simulation result ($\tau = 1.00\ \&\ E_m = 1.00$). (a) Single simulation path of functional response $f(t)$ (Eq. 2). (b) Single simulation path of scaled reserve density $E(t)/E_m$. (c) 10 simulation paths (10 individuals) of volume $V(t)$. Variance over 1000 individuals of (d) scaled reserve density, (e) growth rate ($\Delta V(t)$) and (f) individual size ($V(t)$).

Table 2 Maximum variances of scaled reserve density, growth rate and size. Coefficients of variation were also calculated, but qualitative results remained the same

	τ		
E_m	0.01	0.10	1.00
Scaled reserve density ($E(t)/E_m$)			
2.00	1.2×10^{-5}	1.1×10^{-4}	6.8×10^{-4}
1.00	2.4×10^{-5}	2.2×10^{-4}	1.0×10^{-3}
0.10	2.0×10^{-4}	1.1×10^{-3}	8.9×10^{-3}
Growth rate ($\Delta V(t)$)			
2.00	3.8×10^{-16}	3.2×10^{-15}	1.8×10^{-14}
1.00	1.4×10^{-15}	1.2×10^{-14}	4.5×10^{-14}
0.10	2.8×10^{-14}	1.3×10^{-13}	1.8×10^{-13}
Size ($V(t)$)			
2.00	3.2×10^{-12}	2.9×10^{-11}	2.8×10^{-10}
1.00	4.5×10^{-12}	3.9×10^{-11}	4.0×10^{-10}
0.10	7.5×10^{-12}	6.6×10^{-11}	5.9×10^{-10}

different combinations of τ and E_m. Comparing across the rows of the table, an increase in τ results in increases in the variance of the scaled reserve density, the variance of the growth rate and the variance of the size. However, comparing across the columns of the tables, we note that an increase in E_m results in decreases in the variances of the scaled reserve density, the growth rate and size. The decrease in the variance of the size is only slight.

Finally, we calculated autocorrelation in food and the residual growth rate under the nine scenarios, and the result is plotted in Fig. 3. Comparing the top three panels, we note that as τ is decreased, the length of the autocorrelation in food is reduced, but the length of the autocorrelation in the residual growth remains almost the same. However, as E_m is reduced, the lengths of the autocorrelation of food and the residual growth rate become almost the same even at a low value of τ. This indicates that the residual autocorrelation is induced by inclusion of the reserve.

Simple energetic model

We now simplify the dynamics and characterize all processes that utilize energy under the broad heading 'growth' (Fig. 1b). We develop a discrete-time model of the assimilation and utilization of food. Our goal is to build a model that includes the key processes from the DEB model, but is simple enough to investigate analytically.

Let f_t be the food available to the individual, e_t be the quantity of reserves within the individual and g_t be its growth rate at time t. We denote variances of f_t, e_t, and g_t as σ_f^2, σ_e^2 and σ_g^2, respectively. If we omit additive and proportionality constants, essential dynamics can be summarized by the three equations

$$f_{t+1} = \gamma f_t + \sqrt{1-\gamma^2}\, z_t \tag{3}$$

$$e_{t+1} = \lambda e_t + f_t \tag{4}$$

$$g_{t+1} = (1-\lambda) e_{t+1}, \tag{5}$$

where γ is an autoregressive coefficient for food ($0 < \gamma < 1$), λ is an energy retention coefficient ($0 < \lambda < 1$), and z_t is a serially independent and identically-distributed random variable with mean 0 and variance σ_z^2. In

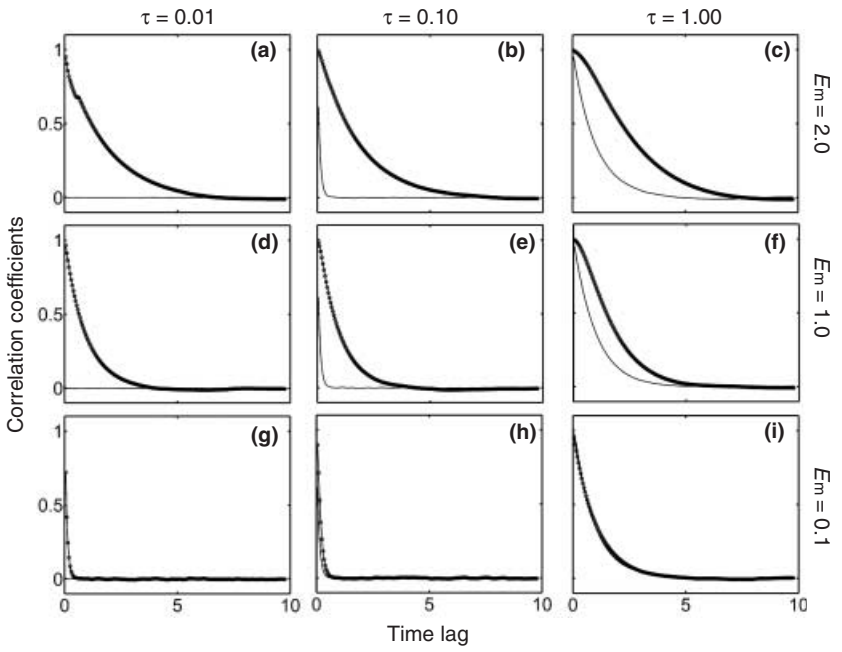

Figure 3 Autocorrelation in food (solid) and the residual growth (solid with circles).

each time-step, a fraction of the reserve is retained in the reserve; the remainder (1-λ) is released for growth. We assume that there is no size dependency in the individual growth because we are only interested in the residual autocorrelation, which does not include the size effect.

In this model, the autoregressive coefficient (γ) determines the length in time of food autocorrelation. When it is increased, the length of the autocorrelation becomes longer. In this sense, γ plays the same role as the parameter τ in eqn 1. The term multiplying z_t in eqn 3 ensures that the variance in food density σ_f^2 is independent of γ. The energy retention coefficient (λ) in eqn 4 is related to the mean retention time of the energy in a reserve and analogous to the parameter E_m in the DEB model (Table 1). Our goal is to determine how γ and λ affect the autocorrelation of growth and the size of σ_g^2.

Equations (3)–(5) can be reorganized to show that growth is described by the second order autoregressive model

$$g_{t+1} = (\lambda + \gamma)g_t - \lambda\gamma g_{t-1} + \frac{\sqrt{1-\gamma^2}}{1-\lambda} z_{t-1} \qquad (6)$$

Thus both food autocorrelation and reserve retention increase the first order autocorrelation of growth.

From eqn 6, we can show that

$$\sigma_g^2 = \frac{(1+\lambda\gamma)(1-\lambda)}{(1-\lambda\gamma)(1+\lambda)}\sigma_f^2 \qquad (7)$$

The variance of the growth rate is a monotonically increasing function of γ (Fig. 4), except that when $\lambda = 0$, it is constant. The variance of the growth rate is always a monotonically decreasing function of λ. Therefore, increased autocorrelation in food increases the variance of the growth rate as long as there is the reserve, and increased mean reserve retention time (i.e. increased λ) decreases the variance in the growth rate.

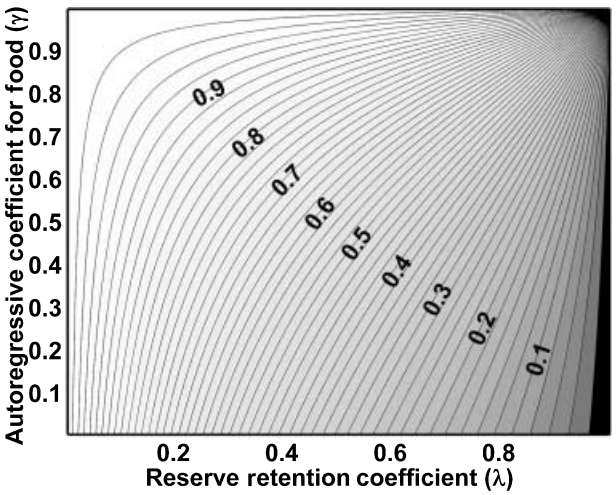

Figure 4 Variance of growth rate (σ_g^2) relative to the variance in food (σ_f^2). The relative variance is plotted as a contour against the reserve retention coefficient (horizontal axis) and the autoregressive coefficient for food (vertical axis).

As the signal (variability) in food is transmitted from food to a reserve, and finally to individual growth, it is smoothed because the energy reserve functions as a low-pass filter. As the maximum reserve density is increased, the ability to smooth the signal also increases. Therefore, in the same food environment (the same amount of autocorrelation and variance), increased maximum reserve density reduces the variance in the growth rate (Fig. 4). The reduced variance of the growth rate would translate into a reduction in the variance of size. However, the inclusion of the reserve in the model also induces autocorrelation in the reserve density. This, in turn, induces autocorrelation in the growth rate (compare the panels in each column of Fig. 3). This would have the effect of increasing the variance of size. It appears that, in the DEB model, the two effects of the reserve almost cancel each other.

DISCUSSION AND CONCLUSIONS

Our results using the two models consistently show that the residual growth is always autocorrelated as long as there is either a reserve, autocorrelation in food, or both (Fig. 4). However, in contrast to earlier studies (DeAngelis *et al.* 1993; Pfister & Stevens 2002), we demonstrated that the increased autocorrelation is *not* always associated with an increased variance of size. This is because, unlike resource autocorrelation, reserves affect both the autocorrelation and variance of growth, with counteracting effects on size variance.

Our conclusions on the effects of reduced variance of the growth rate and increased autocorrelation by a reserve is general in all individual growth in which the reserve acts as a low-pass filter. Consequently, qualitative results will hold for different types of individual growth strategy as long as there is a reserve buffer. However, quantitative results may differ among different models and different parameter values.

On the contrary to reserve induced autocorrelation, autocorrelation in food abundance directly increases the autocorrelation in reserve and the residual autocorrelation in growth rate (compare the panels in each row of Fig. 3) while having little effect on the variance of the reserve and the growth rate. Consequently, increasing the duration of the autocorrelation in food (while maintaining the same variance of food) increases the variance of size (compare across the rows of Table 2). Therefore, resource induced residual autocorrelation is associated with an increased variability in size. We note that the amounts of the increases in the

variance of the reserve and the growth rate are also proportional to the size of the maximum reserve density, and that when there is no reserve ($E_m = 0$ or $\lambda = 0$), these increases disappear (Fig. 4). Consequently, there is only the autocorrelation effect under no reserve situation.

When the maximum reserve density is small (see the bottom rows of Fig. 3 and Table 2), our model is essentially a simple growth model with no reserve. In this limiting case, our model becomes equivalent to the models of DeAngelis *et al.* (1993) and Pfister & Stevens (2002). Our result is consistent with their conclusion that the increased autocorrelation in the growth rate in a no reserve situation is associated with increased variability in size.

We analysed two types of mechanisms that generate the autocorrelation in the residual growth rate. Another important mechanism that could generate the autocorrelation is individual differences in traits. For example, the maximum reserve density or assimilation rate may be different among individuals. Such differences can be included in growth models as differences in parameter values among individuals. If an individual is born to be a fast grower, or acquired an ability to be a consistent fast grower, the individual always has higher than average growth rate for a given size. This creates strong autocorrelation in the residual growth (in fact, under this mechanism the autocorrelation does not disappear throughout the individual's lifetime) and also leads to increased size variability within a cohort. Influence of trait and resource (feeding) variations on individual growth rate is an active research topic in aquaculture (e.g. Imsland *et al.* 1998) and other animal farming (e.g. Meyer *et al.* 1993; Meyer 1999). Our study suggests the physiologically induced autocorrelation as another important factor to be considered when studying individual growth.

Disentangling these processes also offers practical benefits in view of the increasing use of measurements of the growth rate of organisms as an ecological indicator (e.g. Bennett *et al.* 1995). Growth rate measurements, combined with other measurements of physiological condition, have the potential to yield information on the history of environmental stress (e.g. from contaminants) experienced by an organism. However, our analysis shows that interpretation of these measurements will require careful evaluation of the mechanisms that generate autocorrelation in growth rate and size.

Variation among individuals in demographic traits of survival and fecundity has been shown to be important for the stochastic dynamics of the population (e.g. Conner & White 1999; Fox & Kendall 2002; Kendall & Fox 2002, 2003; Robert *et al.* 2003). Preliminary research likewise suggests that among-individual variation in growth rate can strongly affect the population growth rate, through its effect on mean time to maturity and mean size at maturity (B. E. Kendall and P. Vitt, unpublished manuscript). Thus our understanding of the population dynamics will be limited if we do not consider growth rate variability and its autocorrelation.

Our goal was to compare the effects of autocorrelation in food and functions of the energy reserve on the variability in individual size. Our analyses are not intended to include all possible mechanisms that influence the individual size variability. For example, size-dependent mortality could reduce the variability or difference in birth-size could be translated into magnified variability of adult size. Furthermore, combined effects of variability in traits over time and among individuals could lead to complicated individual growth dynamics. Such mechanisms are important but outside the scope of our current study. We also intentionally held the variance of the food constant (eqns 1 and 3) in order to control the effect of a change in the food variance. However, this does not imply that we do not think there is some relationship between the variance and autocorrelation of food. We believe that how they are related is still an open question that will need to be answered in a future study.

Our study emphasized *mechanisms* that generate autocorrelation in ecological data and their effects. Ecological signals are ubiquitously pink (Halley 1996), and this has been a focus of previous theoretical studies (Caswell & Cohen 1995; Cohen 1995; Halley 1996). Our result reemphasizes the importance of studying the causes of serial correlation in ecological data and their effects on ecological systems.

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