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Integrating developmental clocking and growth in a life-history model for the planktonic chordate *Oikopleura dioica*

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ABSTRACT: We present a novel modelling approach applied to the appendicularian *Oikopleura dioica*. Growth and development are represented as separate processes, and a developmental clock is assumed to regulate development. Temperature influences the organism globally through aging and not separately through individual physiological processes as commonly applied in ecological models, and physiological rates are outputs rather than model inputs. The model accounts for development, growth, metabolic rates and life-history characteristics, with relatively few equations and parameters. Parameter values needed for simulations were derived from published data on the development of *Caenorhabditis elegans* and *O. dioica*. Comparisons of simulated generation times, respiration rates, assimilation rates and growth rates with independent data demonstrated predictive capability, but also raised inconsistencies that deserve future experimentation. The present model frames several testable predictions concerning development, growth and metabolic rates in multicellular organisms. Specifically, it suggests that the allometric scaling of metabolism to temperature and body mass should change when food limitation slows growth but not development.

KEY WORDS: *Oikopleura* · Life history · Growth · Development · Aging · Allometry

INTRODUCTION

Growth is commonly defined as an increase in body mass, while development is the change from an undifferentiated to an increasingly more organised state. Due to the strong correlation between them, models of growth (West et al. 2001) and life history (Stearns 1992) usually do not explicitly differentiate between these processes. However, individuals of the same weight can, and often do, differ in developmental stage, as seen in copepods (Campbell et al. 2001). Development may proceed without growth in mass and vice versa. We therefore propose a model in which development is separated from growth, but in which they are linked such that development acts as a driver of growth. This explicit representation of development emerged from previous studies on the short-lived semelparous urochordate *Oikopleura dioica* (Ganot & Thompson 2002, Troedsson et al. 2002). The developmental time of this animal seems to be programmed as a function of temperature, but is unresponsive to food concentrations exceeding a minimum level necessary for survival (Troedsson et al. 2002). Food resources above this level are allocated to the reproductive organ, yielding clear differences in fecundity as a function of food regime. We model growth, i.e. increase in body mass, as a function of both external food availability and internally programmed development. Thus, body mass at a certain developmental stage depends on the amount of food provided in the simulation, while the developmental progression depends on temperature.

Our approach differs in several ways from the state-of-the-art individual growth modelling presented by Touratier et al. (2003). They simulated the growth of *Oikopleura dioica* by specifying a large number (18)

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of differential equations and process equations (>30) reflecting the state of the organism and the flow of both nitrogen and carbon through several defined periods of the life history. A very different growth modelling approach was presented by West et al. (2001). Rather than predicting individual growth trajectories at different food regimes for a particular species, they derived a single parameterless universal curve in order to describe general growth patterns of many diverse species. Concerning generality and complexity our approach is intermediate to those of West et al. (2001) and Touratier et al. (2003). Like Touratier et al. (2003) we do simulate the individual growth and life-history characteristics of a particular species as a function of environmental variables, but with a limited number of equations involving parameters primarily reflecting the general characteristics of cell proliferation dynamics. One important feature that separates our approach from both of the above is the explicit differentiation between development and growth, which also gives rise to several other unique features we will discuss. From published data on *Caenorhabditis elegans* (Sulston & Horvitz 1977, Sulston et al. 1983) and *O. dioica* (Staver & Strathmann 2002, Troedsson et al. 2002), we estimate the parameters required to simulate development, growth and life-history traits in *O. dioica*. We also simulate the scaling of metabolic rates with body mass and temperature. These simulated scaling relations are compared with independent observations on *O. dioica* (Lopéz-Urrutia 2003, Touratier et al. 2003), and lead us to a hypothesis on how food limitation should influence scaling relations in organisms more generally.

MATERIALS AND METHODS

The model consists of 2 submodels, one describing developmental progression and the age of a given developmental stage and another describing the growth increments from one developmental stage to the next (Table 1). The model parameters with units and values are given in Table 2, while equations representing model outputs are summarised in Table 3.

Modelling developmental progression and age. In a separate study (Aksnes et al. 2006, this volume) we have connected the age (t_i) of an organism to its developmental state (i), such that:

$$t_i = be^{ix - aT} - be^{-aT} \approx be^{ix - aT} \quad (1)$$

Here, b is proportional to a reference cell cycle duration at a reference temperature (for mathematical convenience taken as 0°C), a describes the temperature sensitivity, T is the temperature, and x is the product of one parameter describing cell proliferation and another reflecting the scaling of the time required for development versus the cell number (see Aksnes et al. 2006 for details. The approximation given by the last term of Eq. (1) is valid as i grows higher. Note that i is derived as a cell-cycle counter, which we interpret as a dimensionless developmental clock. One increment of this clock implies a more developed organism, and i can therefore also be seen as an index of developmental stages that reflect the cell number as explained in Aksnes et al. (2006). The developmental clock takes the values $[0,1,\ldots, i_{max}]$, whereby the egg is fertilised at $i = 0$ and spawning commences at $i = i_{max}$ with subsequent death for the semelparous *Oikopleura dioica*. We assume that the developmental regulation affecting the life-history traits and ontogeny can be programmed as on/off switches (Bornholdt 2005) according to the i-clock. Thus, growth of individual organs can be turned on or off as a function of i, according to the developmental programme of the organism. Here, we simply have to consider 2 body compartments, the soma and the gonad, and 3 switches so that when $i = i_{feed}$ organogenesis is fulfilled and feeding can start, when $i = i_{somoff}$ somatic growth is turned off and finally when $i = i_{max}$ spawning occurs. The actual time needed

Table 1. *Oikopleura dioica*. Representation of developmental progression, age and growth in the simulation model. A non-dimensional developmental clock i, also serving as an index for developmental stages, is assumed to control progression of the developmental events. The age (t_i) of Developmental Stage i is calculated from the ambient temperature according to Eq. (1) in the text. For each i-cycle the body mass, $M_i = S_i + G_i$, is updated according to the developmental forcing and the food concentration C (algal cells ml^{-1}). S_i and G_i are the mass of soma and gonad at Developmental Stage i respectively. See 'Materials and methods' for definition of parameters

Developmental progression and age

Stage: i =	0	1	2	...	i_{feed}	...	i_{somoff}	...	i_{max}
Event:	Egg is fertilised				Feeding starts		Soma growth stops		Spawning
Age (Eq. 1):	0	t_1	t_2	...	$t_{i_{feed}}$	...	$t_{i_{somoff}}$	...	$t_{i_{max}}$

State equations for body mass

Mass of soma: $S_{i+1} = S_i(1+F_{lim}A_s-R)$
$A_s = A$ when somatic growth is on ($i < i_{somoff}$), else $F_{lim}A_s = R$ for soma

Mass of gonad: $G_{i+1} = G_i(1+F_{lim}A-R)$

Food limitation on assimilation

$$F_{lim} = \frac{kC}{kC+K} \quad k = \frac{M_i}{G_i}$$ when somatic growth is off ($i \geq i_{somoff}$), else $k = 1$

Table 2. *Oikopleura dioica*. Parameter values used to simulate post-embryonic age, growth, physiological rates and life-history. *A* and *R* are given as fractions and are dimensionless (d.l.). Sources for the parameter values were 1: Aksnes et al. (2006, this volume); 2: Staver & Strathmann (2002); 3: estimates obtained by fitting to experiments by Troedsson et al. (2002); 4: Touratier et al. (2003)

Symbol	definition	Value	Unit	Source
x	Age expansion coefficient	0.11	per i-cycle	1
b	Reference age	33	h	1, 2
a	Temperature dependency	0.09	$^{\circ}C^{-1}$	2
A	Maximal assimilation during an i-cycle	1	d.l.	Assumed
R	Metabolic loss during an i-cycle	0.40	d.l.	3
M_e	Mass of an egg	0.013	µg C	4
G_{12}	Initial gonad mass (at i = 12)	0.004	µg C	3
i_{feed}	Start feeding	12	i-cycle	3
i_{somoff}	Stop soma growth	24	i-cycle	3
i_{max}	Spawn	27	i-cycle	3

Table 3. *Oikopleura dioica*. Model output equations predicting physiological rates and life-history characteristics. Values of A, R, M_e, x, b, a and i_{max} are given in Table 2, while the state equations for body mass (M_i), soma mass (S_i) and gonad mass (G_i) are given in Table 1 (including legend). The value of A_S depends on the value of i as explained in Table 1. t_i is given by Eq. (1). T: temperature

Generation time (h)	$D = t_{i_{max}} = be^{i_{max}x-aT}$
Respiration rate (µg C h^{-1})	$r_i = RM_i/(t_i - t_{i-1})$
Assimilation rate (µg C h^{-1})	$p_i = (A_sS_i + AG_i)/(t_i - t_{i-1})$
Growth rate over life time (h^{-1})	$g = \ln(M_{i_{max}}/M_e)/D$
Fecundity (number of eggs)	$E = G_{i_{max}}/M_e$
Maximal rate of population increase (h^{-1})	$r_{max} = \ln E/D$

to develop to Stage i is given by t_i, which corresponds to the age of the organism at that stage, and Eq. (1) therefore provides the age of the organism in our model.

Modelling growth. In addition to development, food also acts as a driver for growth in our model. The state variables for the soma and the gonad (S_i and G_i in Table 1) are updated for each i-cycle according to available food. Within each i-cycle a maximal assimilation (A), adjusted according to the prevailing food limitation (F_{lim}), and a metabolic loss (R) are assumed. In the present simulations, A and R are represented as constant fractions throughout the life cycle. No time dimension is attached to these parameters, and, provided that the growth of an organ is turned 'on', A = 1 simply means that cells within that compartment are, in the case of no food limitation, able to assimilate twice their mass during 1 i-cycle. Similarly, R specifies the fraction of the mass that is lost through metabolism during 1 i-cycle. We did not model the feeding process explicitly, but have introduced a dimensionless food-limitation term on growth, F_{lim}, that influences assimilation directly (Table 1). This limitation can be specified as a number between 0 and 1, where F_{lim} = 1 means no limitation. Alternatively, it can be derived from a normalised food concentration, $F_{lim} = F/(F + K)$, where F is food concentration and K is an experimentally determined half-saturation coefficient for assimilation (which will be different from the half-saturation constant commonly parameterised for food intake).

The growth of the 2 compartments is controlled in 2 ways. First, as already explained, through the start/stop regulation programmed according to the i-clock. Second, growing organs, or compartments in our case, compete for nutrients. When growth for both compartments is turned on, they obtain available nutrients in proportion to their size and according to environmental food limitation (Table 1). When somatic growth is turned off ($i \geq i_{somoff}$), only the metabolic requirement is applicable to the assimilatory need for soma and any surplus assimilated food is therefore entering the gonad only. This means reduced competition with the soma and was parameterised by an increase in available nutrients for the gonad, which is assumed proportional to the size ratio of the total body versus gonad mass ($k = M_i/G_i$, see Table 1).

Parameter values applied in the simulations. The value of the temperature sensitivity parameter a at $0.09°C^{-1}$ (Table 2) corresponds to that measured by Staver & Strathmann (2002) for cell cycle duration in *Oikopleura dioica*. This amounts to Q_{10} = 2.5. The value of x at 0.11, corresponds to that estimated for the post-embryonic period of *Caenorhabditis elegans* (Aksnes et al. 2006). The value of b at 33 h was also derived from the cell dynamics of *C. elegans*, but corrected according to observed lengths of cell cycling in *O. dioica* (Staver & Strathmann 2002) relative to that of *C. elegans* (Sulston et al. 1983). Using values for the 3 parameters of Eq. (1), the age (t_i [h]) of a post-embryonic *O. dioica* in Developmental Stage i at temperature T can then be calculated according to: $t_i = 33.0e^{0.11i - 0.09T}$. This equation, however, can also be used the other way around to calculate values of i that correspond to known timings of developmental events. Insertion of t_i = 170 h and T = 15°C, corresponding to the generation time measured by Troedsson et al. (2002), provided an estimate of i_{max} = 27 that was used in the simulations. Similarly, insertion of t_i = 120 h at 15°C, corresponding to the time when somatic growth ceased, yields i_{somoff} = 24. As shown in Aksnes et al. (2006), the embryonic and post-embryonic periods

require different parameters of the age equation (Eq. 1). By following the same procedure for the embryonic phase as for the post-embryonic phase, we obtained $t_i = 0.87e^{0.36i - 0.09T}$. Insertion of t_i = 15 h at T = 15°C, corresponding to completion of organogenesis and the start of active feeding in *O. dioica* (Troedsson et al. 2002), yielded an estimate of i_{feed} = 12, which was used as the start point for the simulation (i.e. only the feeding period was simulated). The initial body mass was set equal to 0.013 µg C, which corresponds to that of the egg (Touratier et al. 2003). The value of the maximal assimilation during 1 *i*-cycle was assumed as A = 1. Values of the 2 last parameters (metabolic loss per *i*-cycle, R = 0.4, and the initial weight of the gonad at i = 12, G_{12} = 0.004 µg C) were obtained by fitting simulated and experimental growth data obtained at 15°C under experimental eutrophic food conditions (Fig. 1, Table 4).

Model outputs. In order to describe model outputs, temperature influence requires some clarification. Temperature does not enter the submodel of growth. Both assimilation and metabolic loss are parameterised as fractions (A and R) that are independent of temperature. The simulated assimilation and metabolic *rates*, however, do depend on temperature (r_i and p_i in Table 3). This is because temperature affects the time it takes to develop (Eq. 1). A higher temperature means that the organisms develop more rapidly, because the durations of the *i*-cycles become shorter. The metabolic rates will increase accordingly, and this can be seen from the equations in Table 3. Of the 6 specific outputs listed here, there are 4 rates: respiration, assimilation and growth rate, together with maximal rate of population increase. These rates are all influenced by the durations of the *i*-cycles (i.e. by $t_i - t_{i-1}$ appearing in the rate equations), which, in turn, are affected by temperature according to Eq. (1). All rates in Table 3, however, are truly outputs and have no effects on simulated growth and life-history parameters. It is the parameter values of Table 2, together with the values for the 2 environmental variables temperature and food, that decide the outcome of a simulation.

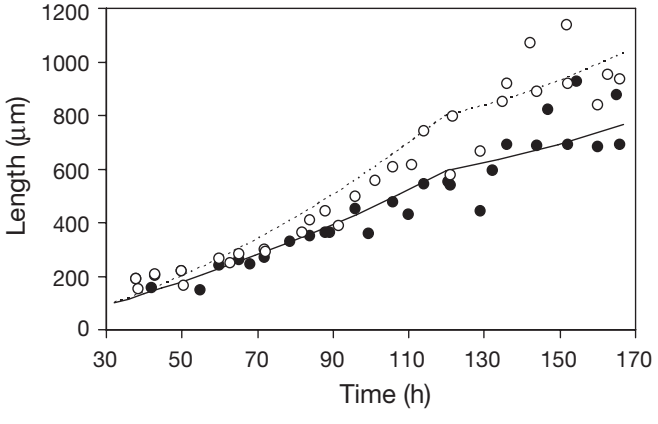

Fig. 1. *Oikopleura dioica*. Size (length) versus time (Troedsson et al. 2002). Experiments were conducted under eutrophic (○: 31 µg C l^{-1} prior to 96 h and 62 µg C l^{-1} after) and oligotrophic conditions (●: ⅙ of the eutrophic concentrations). Experiments conducted at 20 and 15°C were combined by standardising to 15°C. Simulations are indicated by lines (dashed: eutrophic; continuous: oligotrophic). Conversion from simulated body mass (M, µg C) to length (l, µm) was obtained by $l = 520M^{0.3806}$ (King et al. 1980)

Table 4. *Oikopleura dioica*. Simulated (Sim) fecundity (E) and maximal rate of population increase (r_{max}) as a function of temperature and food regime. See Table 3 for Sim E and Sim r_{max} equations. Observations (Obs) are from Troedsson et al. (2002). Oligotrophic and eutrophic conditions are defined in Fig. 1

Temp. (°C)	Food regime	E		r_{max} (h^{-1})	
		Obs	Sim	Obs	Sim
15	Oligotrophic	142 ± 64	139	0.029	0.028
	Eutrophic	303 ± 115	300	0.036	0.032
20	Oligotrophic	137 ± 65	123	0.037	0.044
	Eutrophic	363 ± 172	295	0.045	0.051

The 3 life-history parameters, generation time (D in Table 3), fecundity (E) and maximal rate of population increase (r_{max}) are given by (1) the insertion of i_{max} into Eq. (1); (2) the mass of the mature gonad divided by the mass of an egg; and (3) by the logarithm of fecundity divided by the generation time.

Independent observations used for comparisons. Two extensive reviews (Lopéz-Urrutia et al. 2003, Touratier et al. 2003) of *Oikopleura dioica* enabled comparisons of model predictions with independent observations. Except for the mass of an egg (M_e), these observations are independent in the sense that none of the measurements compiled by Lopéz-Urrutia et al. (2003) and Touratier et al. (2003) underlie the parameter values applied in the simulations. Lopéz-Urrutia et al. (2003) fitted empirical functions to data sets for ingestion, respiration, generation time and growth rates versus temperature and body mass. Respiration and growth rates are commonly described by exponential functions when related to temperature, while power functions are applied when these are related to body mass (Peters 1984, West et al. 2001, Gillooly et al. 2002). These 2 functional relationships were also assumed by Lopéz-Urrutia et al. (2003) and Touratier et al. (2003). In order to compare our simulation results with their functions based on measurements, we fitted the exponential and power functions to our simulated respiration, assimilation and growth rates.

RESULTS

Comparing simulated results with independent observations

As commonly observed in ecological measurements, the experimental data compiled by Lopéz-Urrutia et al. (2003) and Touratier et al. (2003) contain considerable variability. This observed variability is represented by the dashed lines in Fig. 2, while the solid lines represent our model simulations. Unlike generation time, growth rate is not explicitly expressed as a function of temperature in our model. However, we fitted the exponential function (solid line in Fig. 2B) to the simulated growth (given by the markers in Fig. 2B) at different temperatures. Similarly, power functions were fitted to simulated assimilation (Fig. 2C) and respiration rates (Fig. 2D) as a function of body weight. It should be noted that our model does not assume the validity of such allometric scaling of respiration and assimilation, although the simulation results were well described by the fitted functions (Fig. 2B–D). The accordance between the observations and simulations was robust (Fig. 2, Table 5) taking into account their independence and the absence of model tuning to these observations. Some deviations, however, deserve attention. The simulated assimilation rates for the 3 largest body masses were considerably below the trend

Table 5. *Oikopleura dioica*. Comparison of simulated processes with independent observations. Observed functional relationships for generation time (D), growth rate (g) and respiration rate (r) are from López-Urrutia et al. (2003); the relationship for assimilation (p) is from Touratier et al. (2003). These functions represent the estimated mean trends of the observations. The simulated relationships for body growth rate were obtained by fitting the exponential function to simulated growth rates for simulations carried out at different temperatures (Fig. 2B). Simulated relationships for respiration and assimilation rates (at 15°C) were obtained by fitting the power function to simulated values of respiration and assimilation rates at different body masses (Fig. 2C,D)

Process	Observed	Simulated
Generation time (h)	$D = 644e^{-0.085T}$	$D = 643e^{-0.089T}$
Body growth rate (h^{-1})	$g = 0.0096e^{0.081T}$	$g = 0.0097e^{0.089T}$
Respiration (µg C $ind.^{-1}$ h^{-1})	$r = 0.030M^{0.60}$	$r = 0.041M^{0.74}$
Assimilation (µg C $ind.^{-1}$ h^{-1})	$p = 0.17M^{0.75}$	$p = 0.10M^{0.76}$

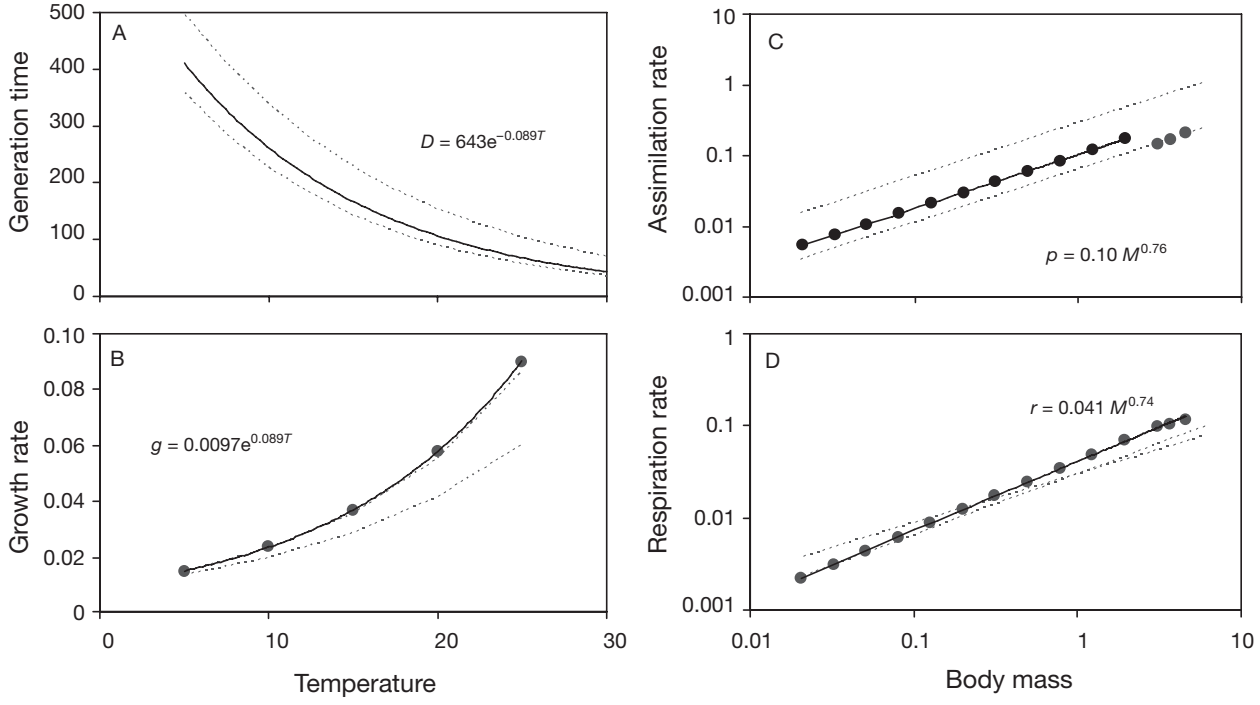

Fig. 2. *Oikopleura dioica*. Model predictions (solid lines) compared with independent observations (dashed lines) for (A) generation time (hours) (Lopéz-Urrutia et al. 2003, $D = 644e^{-(0.085 \pm 0.007)T}$); (B) body growth rate (h^{-1}) (Lopéz-Urrutia et al. 2003, $g = 0.0096e^{-(0.081 \pm 0.0072)T}$); (C) assimilation rate (µg C $ind.^{-1}$ h^{-1}) ($p = P \times 0.32\ M^{0.75}$; this equation is derived from the equation for maximal feeding rates given by Touratier et al. (2003), where P represents reported range of assimilation efficiency corresponding to 0.17–0.88); (D) respiration rate (µg C $ind.^{-1}$ h^{-1}) (Lopéz-Urrutia et al. 2003, $r = 0.030\ M^{0.5984 \pm 0.064}$ at 15°C). All simulations had eutrophic regimes (Fig. 1), and were started at $i = 12$ and run with the parameter values in Table 2; simulations for (C) and (D) were run at 15°C

line fitted for the smaller body masses. This is because simulated somatic growth was assumed to be turned off for $i \geq i_{somoff} = 24$, and at this point assimilation is restricted to the respiratory needs of the soma in addition to the growth demands of the gonad. Another discrepancy is seen in the simulated growth rate, which is consistently higher than that observed (Fig. 2B). This may suggest bias in the modelled maximal growth that is largely determined by the *A*-parameter (Table 2), but could also have been caused by different food conditions underlying simulations versus observations. The growth rates compiled by Lopéz-Urrutia et al. (2003) were obtained under variable food conditions, while a high food level was assumed in the simulation ($F_{lim} >$ 0.98). Such different food conditions may have also caused the most conspicuous deviation that is seen in the simulation of respiration rate versus body mass (Fig. 2D, Table 5). The simulated exponent of the allometric relationships between respiration rate and body mass was 0.74, while the observed was 0.60 ± 0.06. Closer inspection of the model behaviour revealed that the simulated respiration rates scale differently (i.e. the exponents are not constants) to both body size and temperature when the feeding histories are different. Thus, an unexpected result is that the simulated exponents of the allometric relations decrease as growth becomes food limited. The underlying mechanism for this simulation result is explained below.

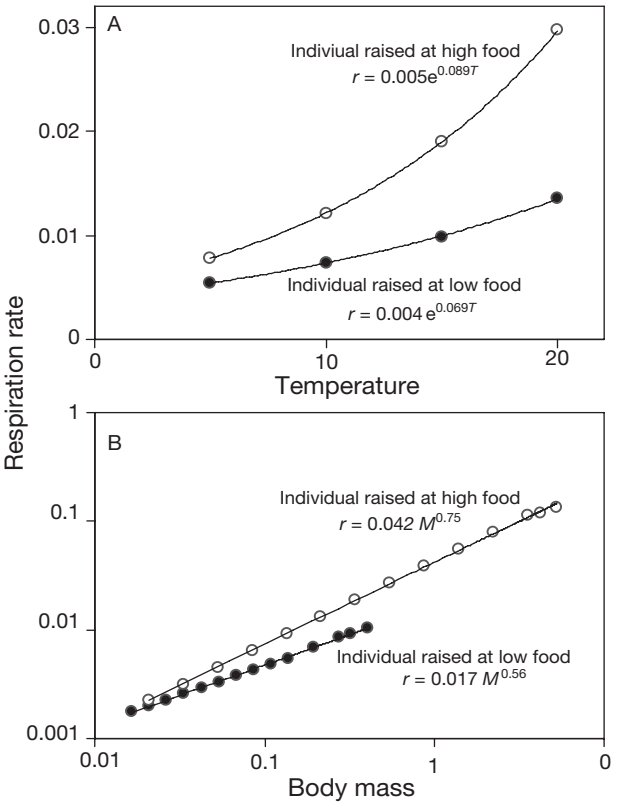

Fig. 3. Simulations of respiration rate (µg C ind.$^{-1}$ h^{-1}) (A) as a function of temperature (°C) for an individual of given body mass (0.34 µg C) raised at low (F_{lim} = 0.67) and high (F_{lim} = 0.95) food levels; (B) as a function of body mass (µg C) for individuals raised at low and high food levels. Equations were fitted to the simulated respiration rates (● and ○)

Hypothesis: metabolic scaling with body mass and temperature may depend on food limitation

In our model, temperature influences the organism globally through its influence on age (Eq. 1). Since only 1 parameter of the model defines the temperature sensitivity (a = 0.09; Table 2), one might expect the temperature sensitivities of the simulated rates to be invariant and equal to a. However, this was not the case. For individuals of a given body mass, simulations with a high food level gave a respiratory temperature sensitivity close to 0.09 (0.089; Fig. 3A), while simulations with a lower food level gave a much lower value (0.069; Fig. 3A). A similar prediction applies to the allometric relationship of respiration versus mass. Simulation with high food yielded the exponent 0.75, while simulation with low food gave 0.56 (Fig. 3B). Thus, the exponent (0.60 ± 0.06) estimated from observations by Lopéz-Urrutia et al. (2003) lies between these simulated exponents representing high and low food. The reason for the food-initiated variability in the simulated exponents can be explained as follows. Individuals exposed to food limitation in our model achieve a certain body mass at a later developmental stage (i.e. *i*-cycle) than an animal exposed to food surplus. Although respiration is calculated as a fixed fraction (R) of mass, there is a non-linearity in the way age (t_i) scales with developmental stage. Thus, the length of the age interval ($t_i - t_{i-1}$ appearing in the denominator of the respiration rate; Table 3) for which the respiration rate is calculated depends on which developmental stage, i, is considered. In other words, the simulated metabolic rates depend on the developmental stage of the organism as well as its body mass and temperature. This has no consequences as long as growth and development are in full synchrony, which, in our model, is the case when growth is not limited by food. However, when developmental progression and growth become unsynchronised due to food limitation, both the body mass and the developmental stage are needed as predictors for the metabolic rates. Thus, a testable hypothesis emerging from the simulations is that allometric coefficients of metabolic processes versus body mass and temperature should change for organisms when food limitation slows growth but not development.

DISCUSSION

We believe that the main advantage of our approach is that body mass and development, although related, are decoupled and that a simple on/off switch is assumed rather than a system of differential equations for developmental regulation (Bornholdt 2005). There are several consequences of these features. One is the change in the frame of reference for developmental events from time to the non-dimensional developmental clock (Table 1). In our approach the time dimension is uniquely represented by the age of the organism, which is predicted from a mechanistic model linking age to cell proliferation dynamics (Aksnes et al. 2006). Thus, age (and time) appears as a dependent variable and, consequently, physiological rates emerge as output from the model. These rates are affected by the aging of the organism, where aging means how fast the organism proceeds from one developmental stage, $i-1$, to the next, i, as given by $t_i - t_{i-1}$. This representation implies that temperature acts globally, through the speed of aging, rather than through separate physiological processes. In contrast, state-of-the-art models commonly require that each physiological rate is known from measurements or that they can be assumed from allometric relationships involving body mass and temperature. In our approach, measured physiological rates (Lopéz-Urrutia et al. 2003) serve as validation data rather than input data, and simulations revealed inconsistencies that raised the hypothesis about food-dependent metabolic scaling, which deserves future experimental attention.

Growth and life-history modelling frequently uses body size (mass or length) as the main state descriptor of the organism (Stearns 1992, Fiksen 1997, West et al. 2001, Touratier et al. 2003, Nisbet et al. 2004). In such models, developmental signposts are often parameterised so that maturation, entrance to the next developmental stage, etc., occur at a given body size. This assumption seems adequate for organisms in which developmental progression correlates strongly with body mass regardless of food intake. In such organisms we expect, contrary to our assumption for *Oikopleura dioica*, that the developmental clock is somehow regulated by food intake. Fundamental knowledge about the differences in the regulation of development and growth is obviously crucial to progress in modelling these phenomena. Understanding the temporal events that guide development is the focus of intensive research (Purnell 2003), and a number of internal clocks such as a cell division- (Matsuo et al. 2003), circadian- (Matsuo et al. 2003, Schultz & Kay 2003), segmentation- (Pourquié 2003) and *Hox*-clocks (Kmita & Duboule 2003) are implicated. In *Oikopleura*, tightly controlled clocking of cell cycles is intimately linked to pattern formation during development, and this occurs in a bilaterally symmetric fashion at a resolution of individual cells in the epithelium responsible for repetitive secretion of the filter-feeding house structure within which the animal lives (Ganot & Thompson 2002). Cell-division clocks are influenced by temperature (Staver & Strathmann 2002, Schibler 2003), and experiments on *O. dioica* (Troedsson et al. 2002) revealed that temperature was a major factor in translating the developmental programme into the timing of the growth trajectory. This observation was crucial for the separation of development and growth, and motivated the link of developmental clocking to cell cycling (Aksnes et al. 2006).

The predictive capacity of our model is most refined when the *i*-clock is most closely based on accurate determinations of fundamental developmental clocks, such as cell-division cycles, that reflect an organism's genetic programme. The detailed studies of cell lineages in *Caenorhabditis elegans* (Sulston & Horvitz 1977, Sulston et al. 1983) enabled a direct link of the *i*-clock to cell cycling for this species (Aksnes et al. 2006). As yet, such detailed data are not available for *Oikopleura dioica*. Instead we constructed the *i*-clock with the 27 developmental steps by applying data from *C. elegans*, corrected with measurements of cell cycle duration in *O. dioica* (Staver & Strathmann 2002) and scaled with the observed generation time in *O. dioica* (Troedsson et al. 2002). Hence, further research is required to connect the developmental clock more tightly to actual cell cycling in *O. dioica*. Since *O. dioica* develops polyploid cells (DNA replication without cytokinesis) during the post-embryonic phase, the *i*-clock for this species should be interpreted as an internal clock for rounds of DNA replication rather than cell divisions.

Temperature is the only environmental factor influencing developmental time in the present application. In reality, developmental timing, involving circadian rhythms and other environmental triggers, can be far more complex than suggested in our model, although our simple scheme seems appropriate for *Oikopleura dioica* being characterised by a relatively simple life history, where temperature determines generation time and food availability determines fecundity. In cases where food intake is insufficient to sustain the energetic demand of development, starvation mortality is a likely outcome that deserves experimental attention. In our application development affects growth, but growth does not affect development. In other words, control of development is linked to the organism's genetic programme rather than to its mass. Future applications will reveal if this is adequate or if developmental timing in one or another way also needs to be connected to growth and food intake.

Although much research remains to connect our life-history and growth-modelling approach more tightly to developmental regulation, several promising results appear in the present application. Development, growth, metabolic rates and life-history characteristics are accounted for with a relatively simple model containing few equations and parameters. Our modelling concept allows representation of internal (genomic and cellular control of development) as well as external (here represented as food and temperature) forcing. The model did fit the growth and life-history patterns obtained in the experiments of Troedsson et al. (2002) fairly well (Fig. 1, Table 4), but this is not surprising since some of the parameter estimates were derived from these experiments. Comparison with independent observations (Fig. 2, Table 5), however, indicates that our approach has explanatory and predictive merits. Simulations suggest that the parameter set $x = 0.11$, $A = 1$ and $R = 0.4$ per i-cycle is consistent with observed growth, life-history traits and metabolic rates in *Oikopleura dioica* (Figs. 1 & 2, Tables 4 & 5). However, these parameters, as well as $i_{max} = 28$, were estimated indirectly. A priority for future applications would be to measure these parameters directly at the cellular level and to analyse inter- as well as intraspecific variability. Such measurements would reveal under what circumstances the parameters can be considered invariant (or as reliable averages) as we have assumed in the present *O. dioica* simulation. Another priority for future experimentation is to test whether different feeding histories induce variations in the allometric relationship of metabolic rates versus body mass and temperature as we have suggested. Part of the variance observed in allometric relationships may potentially be accounted for by this hypothesis.

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