Sizing the effects of temperature on ectotherms - from physiology to ecology

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Abstract

Increasing temperatures under climate change are thought to affect individual physiology of ectotherms through increases in metabolic demands, leading to decreases in body-size with concomitant effects on species ecology. Although intuitively appealing, the magnitude, as well as when and where such changes are expected in an ecosystem context remains unclear. Here, we develop a general trait-based model of carbon and oxygen budgets to determine the relative influence of thermal sensitivity, oxygen limitation, and environmental conditions on ectotherm body size. [RESULTS SUMMARY HERE] Our model provides a physiological basis for changes in species size and observed ecological consequences of temperature, and ties together various strands of thought about the ecological consequences of increased temperatures.

1   Introduction

Temperature, through its effects on individual physiology, is a dominant driver of species ecology and bio-geography ([6, 14, 3]). As a consequence, current and predicted temperature increases under climate change will act as a strong agent of change in many ecosystems ([6, 18]). However, the nature of these changes can be difficult to predict as temperature effects scale from individuals to species and ecosystems. Through this cascade of scales, incorrect or approxiamte model assumptions at the individual scale can have disproportiante effects on ecosystem level outcomes ([2, 12]). In marine fish, recent predictions of decreasing organism size, and resulting decreases in the size of fisheries catches ([4]), have been criticized as overly simplistic and not in line with physiological constraints ([2, 12]). While it is not disputed that organism size can change as a result of changing temperatures, the question is when, where and why this is so.

Empirically, most ectotherms are found to grow larger at colder temperatures - a phenomenon called the temperature-size rule. Models of ectotherm growth can reproduce this pattern (under assumptions of optimal energy allocation between growth and reproduction) if increases in metabolic costs (metabolism) with temperature outpace increases in energy acquisition ([10]). While increasing metabolic costs are real, energy acquisition is an abstract, phenomenological description that lumps ecological aspects of foraging activity and prey availability. At this more mechanistic level of energy acquisition, consistent temperature patterns have remained elusive ([7, 17]), with highly variable responses across habitats and taxa.

Physiologically, a long held view has been that temperature is a controlling factor while oxygen supply sets the physiological limits ([8, 5, 11]). Other factors, such as salinity and pH are masking factors that influence physiology via their effects on oxygen availability and temperature sensitivity. How exactly temperature influences physiological rates and limits, however, has been a matter of debate, not least because of the variable responses observed among different species.

In most species, the standard metabolism (i.e., the metabolic cost of maintenance and routine activity such as ventilation) increases steadily with temperature. A prevalent view is that the maximum metabolic rate (i.e., the metabolic rate at maximum sustained exercise) has a dome-shaped response to temperature, whereby it can be increased (passively and actively) up to a point, but plateaus or decreases thereafter ([8, 5, 11, 15]). This leads to the view of a modal curve for metabolic scope, the available oxygen/energy for activity, and suggests that towards the upper end of the curve. This view was formalized in the Theory of Oxygen and Capacity Limitation of Temperature ([16]), which suggests that the decrease in metabolic scope towards extreme temperatures limits species limits their ability to sustain core functions such as foraging and growth. In some species, however, maximum metabolic rate may be steadily increasing ([11]), suggesting that oxygen may not be the limiting factor. Consequently, the difference in observed physiological responses will at least partially determine temperature effects on body size for individual species. In addition to physiological differences, the assumed coefficients for temperature sensitivity of metabolism and size scaling exponents are different between species, and can disproportionately affect inferences about future organism size.

A general framework for understanding temperature effects on size should reflect physiology, and also allow exploring effects of physiological traits on temperature dependence of body size. Here, we develop a trait based framework to investigate temperature on ectotherm physiology, growth and body size. We discuss traits that have large effects on body size, and discuss expectations under climate change.

2 Setting up the model

2.1 Key assumptions

Our model assumes that physiology is described by two key budgets: the energy and oxygen budgets. In setting up the model we make a number of key assumptions, which are similar to those made in Holt & Jorgensen. At a high level, we assume that animals will adapt activity levels to optimise available energy for growth and reproduction. Growth is limited either by food capture, food processing capacity, or by available oxygen. We further assume that temperature acts directly on rates that are determined by enzymatic activity: digestive activity (via maximum consumption) and metabolic costs. The scaling with temperature (determined by the activation energy E_a) is assumed the same for these processes. Consequently, temperature only acts on ecological rates (food acquisition) via optimisation given temperature driven changes in enzymatic rates.

2.2 Metabolically constrained activity model

In the constrained activity model, ectotherms adjust the relative amounts of time (τ) spent foraging and resting/hiding to optimise the net energy/mass gain P_C relative to natural mortality. Since both energy gain and loss are sensitive to temperature and oxygen limitations, both the activity level and the net energy gain will be subject to these environmental constraints. Their interplay thus determines available energy for growth, reproduction, and, ultimately, organisms final size.

The model is written in terms of carbon (or energy) and oxygen balance equations:

$$f_C = \frac{\tau}{\tau + \frac{h c_T w^{q-p}}{\gamma \Theta}} \tag{1}$$

$$P_C = S_C - D_C \tag{2}$$

$$= (1 - \beta - \phi) f_C h c_T w^q - (1 + \tau \delta) c_T k w^n \tag{3}$$

$$P_{0_2} = S_{O_2} - D_{O_2} \tag{4}$$

$$= f_{O_2} w^n - \omega \left(\beta f_C h c_T w^q + (1 + \tau \delta) c_T k w^n \right) \tag{5}$$

where f is the feeding level ([0,1]) at weight w, determined by the fraction of time spent foraging (or proportion of maximum attack rate) τ, consumption rate $\gamma w^p \Theta$ (search rate γw^p times prey availability Θ) and maximum consumption $h w^q$. In the following, we will refer to τ as the activity fraction for sake of generality. Maximum consumption, determined by digestive (enzymatic) processes (**[jeschke2002predator**, **sentis2013parsing**]), is assumed to scale with temperature as $c_T = e^{E_a(T - T_0)/kTT_0}$. Available carbon P_C is determined by supply (S_C) from prey consumption ($f_C h c_T w^q$), with β a loss due to specific dynamic action (SDA, or heat increment; the energy spent absorbing food), and ϕ is

the fraction of food excreted and egested. Metabolic costs (D_C) are those of standard metabolism (kw^n), as well as active metabolism (scaled in units of standard metabolism as δkw^n), with the activity fraction τ determining the fraction of time that the active metabolism cost applies.

The oxygen budget determines the metabolic scope P_{0_2}. Metabolic scope is the difference between oxygen supply $S_{O_2}/w^n = f_{O_2}$, the amount of oxygen supplied per unit weight, and oxygen demand. Demand (D_{O_2}) is the sum of oxygen used for SDA (i.e., the conversion of food into energy and growth - $\beta f_C h c_T w^q$) and catabolism ($[1 + \tau\delta]c_T kw^n$), with ω determining amount of oxygen required per unit of metabolised carbon (note: oxygen is measured in the same units as the carbon. To find the actual amount of oxygen used multiply by the amount of oxygen used to respire one unit of carbon).

The maximum oxygen consumption—usually assumed to occur at the maximum metabolic rate (MMR), or active metbolic rate—is the oxygen consumption during maximal activity level that can be sustained over some time. This level is determined by oxygen delivery to organs and muscles. At constant temperature T, oxygen supply is a function of ambient oxygen and is assumed to follow a saturating function [e.g., 13]. We specify P_{50} as the point where the MMR (or oxygen supply) has dropped by 50% relative to the saturation level l [1]. Oxygen supply, and hence the MMR, are usually found to be temperature dependent [**lefelefevre˙are˙2016**]. We follow [9, 13] and use a dome shaped function that peaks at the optimal temperature (optimal in the sense of maximising the MMR). Thus:

$$f_{O_2} = \lambda(1 - e^{O_2 \log(0.5)/P50}), \tag{6}$$

$$\lambda = \zeta(\frac{T_{\max} - T}{T_{\max} - T_{\mathrm{opt}}})^\eta \times \exp(-\eta\frac{T_{\max} - T}{T_{\max} - T_{\mathrm{opt}}}), \tag{7}$$

with f_{O_2} the oxygen supply (MMR), $T_{\max}$ the lethal temperature for the species, T_{opt} the temperature at which the MMR is optimised; η determines the width of the dome-shape, and ζ its height (Figure 1). Note that we can emulate a MMR that increases up to the lethal temperature by setting the optimal temperature for MMR close to the maximum temperature.

We now assume that organisms will adjust their activity level to maximise available energy relative to mortality M: $\tau_{\mathrm{opt}} = \max_\tau(F) = \max_\tau(P_C/M)$, with F a measure of fitness. We further assume here that mortality scales with activity level and weight as w^{1-n} ([1]), such that $M_{\tau,w} = \tau^\eta \rho w^{1-n}$, where ρ is the natural mortality at mass $w = 1$ and $\tau = 1$, that is, at constant maximum activity and M is the effective mortality.

The optimal activity level (τ_{opt}) is found at $\frac{dF}{d\tau} = 0$. We assume that the metabolic scope dictates the upper limit of this activity, such that at $\tau_{\max}$, oxygen demand D_{0_2} equals total supply S_{0_2}. Both temperature and oxygen will influence τ, such that at a given temperature and oxygen concentration,

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[1]Could also use the same functional form as the consumption of food for simplicity: $f_{O_2} = \Gamma\Theta_{O_2}/\Gamma\Theta_{O_2} + h_{O_2} = \Theta_{O_2}/(\Theta_{O_2} + P_{50})$.
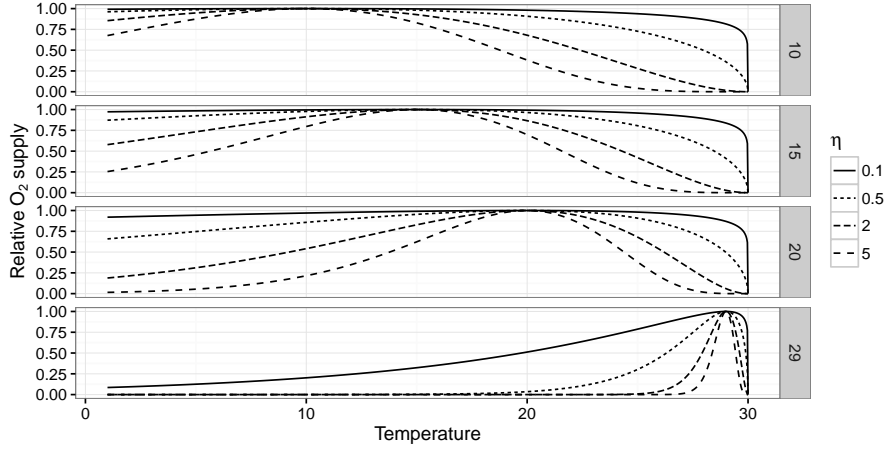
Figure 1: Maximum metabolic rate, determined by oxygen supply, as a function of temperature, for four different optimal temperatures (panels—see panel label for optimal temperature in degrees celsius), and increasing values of η.
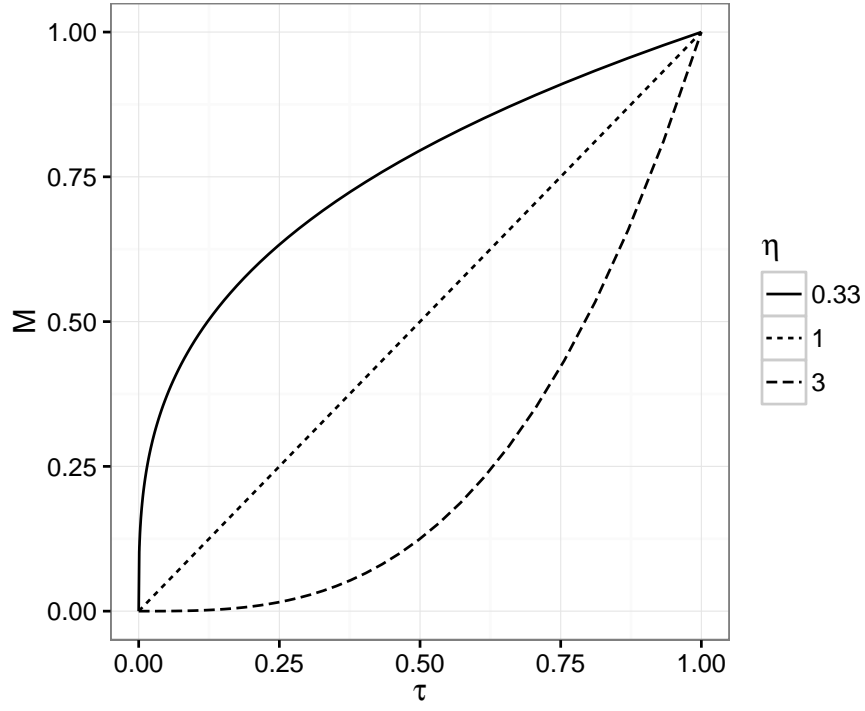

Figure 2: Mortality as a function of activity τ for three levels of the mortality scaler η

Table 1: Parameters

Description	Value
Specific dynamic action	$\beta = 0.15$
Egestion and excretion	$\phi = 0.25$
Consumption rate[1]	$\Theta\gamma \approx hw^{p-q}$ (g_C/time)
Coef. for maximum consumption rate	$h \approx 5g_{WW}^{1-n}$/yr
Critical feeding level	$f_c \approx 0.1$
Activity coefficient	$\delta \approx 1$
Exponent for max. consumption	$q = 0.75$
Exponent for clearance rate	$p = 0.8$
Exponent for std. metabolism	$n = 0.75$

[1] This is under the assumption that the amount of encountered food Θ is roughly independent of body size. This gives and order-of-magnitude estimate of the product $\Theta\gamma$.

$\tau_{T,O_2} = \min(\tau_{\text{opt}}, \tau_{\text{max}})$, meaning we assume that animals will adapt their effort to optimise fitness F given temperature and oxygen constraints.

Life history theory suggests that the maximumasymptotic weight W_∞ is found at the size at which all energy is allocated to reproduction, which optimally occurs at $\frac{\partial P_c}{\partial w} = M$ (CITE Quince, Kozlovski). Note that W_∞ depends on temperature only through τ and changes in P_C as temperature changes metabolic and enzymatic rates. We thus impose no direct constraint on W_∞. We followed tests outlined in Giacomini et al (CITE) to verify the optimally of this allocation assumption.

3   Model results

$$\frac{(\delta\tau+1)T_c km^n + \frac{(\beta+\phi-1)\tau T_c hm^q}{T_c hm^{-p+q}/\gamma+\tau}r\tau^{r-1}}{\tau^{2r}Mm^{n-1}} - \frac{\frac{\delta T_c km^n + (\beta+\phi-1)T_c hm^q}{T_c hm^{-p+q}/\gamma+\tau} - \frac{(\beta+\phi-1)\tau T_c hm^q}{(T_c hm^{-p+q}/\gamma+\tau)^2}}{Mm^{n-1}\tau^r} \tag{8}$$

$$\frac{(\delta\tau+1)T_c km^n + \frac{(\beta+\phi-1)\tau T_c hm^q}{T_c hm^{-p+q}/\gamma+\tau}m^{n-2}(n-1)}{\tau^r Mm^{2n-2}}$$
$$-\frac{\frac{(\beta+\phi-1)h^2 m^{-p+2q-1}(p-q)}{(T_c hm^{-p+q}/\gamma+\tau)^2\gamma} + (\delta\tau+1)km^{n-1}nT_c + \frac{(\beta+\phi-1)\tau T_c hm^{(q-1)q}}{hm^{-p+q}T_c/\gamma+\tau}}{Mm^{n-1}\tau^r} \tag{9}$$

4   Discussion

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