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Review

Control of metabolic rate is a hidden variable in the allometric scaling of homeotherms

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Summary

The allometric scaling exponent of the relationship between standard metabolic rate (SMR) and body mass for homeotherms has a long history and has been subject to much debate. Provided the external and internal conditions required to measure SMR are met, it is tacitly assumed that the metabolic rate (B) converges to SMR. If SMR does indeed represent a local minimum, then short-term regulatory control mechanisms should not operate to sustain it. This is a hidden assumption in many published articles aiming to explain the scaling exponent in terms of physical and morphological constraints. This paper discusses the findings of a minimalist body temperature (T_b) control model in which short-term controlling operations, related to the difference between T_b and the set-point temperatures by specific gains and time delays in the control loops, are described by a system of differential equations of T_b, B and thermal conductance. We found that because the gains in the control loops tend to increase as body size decreases (i.e. changes in B and thermal conductance are speeded-up in small homeotherms), the equilibrium point of the system potentially changes from asymptotically stable to a centre, transforming B and T_b in oscillating variables. Under these specific circumstances the very concept of SMR no longer makes sense. A series of empirical reports of metabolic rate in very small homeotherms supports this theoretical prediction, because in these animals B seems not to converge to a SMR value. We conclude that the unrestricted use of allometric equations to relate metabolic rate to body size might be misleading because metabolic control itself experiences size effects that are overlooked in ordinary allometric analysis.

Key words: control, body temperature, metabolic rate, allometry, dynamic system.

Introduction

The observation that the metabolic rate of individual organisms changes with body mass either ontogenetically or phylogenetically has its roots in the early literature. In 1883, Max Rubner reported that the relationship between the change in body mass (M_b) and 'basal' metabolic rate of mammals scaled with 2/3 (see, for example, White and Seymour, 2003). The first tentative explanation for this relationship had its basis on the proportion between body volume and surface area, thus giving a geometric perspective to the problem, and some support for the 2/3 scaling parameter. A few decades later (ca. 1924), Huxley (see Prothero, 1986; Klingenberg, 1998) proposed a more general perspective to scaling problems and set the foundations of what became known as 'allometry'. The formulation of Huxley is of the type $f(M_b)=f_1M_b^a$, where f is the trait associated with the changing size, has been the subject of vivid disagreement in the scientific literature. Whereas for some "*The use of power laws in biology is so well established that these are called allometric equations …*" (Brown et al., 2000), others state that "*It is well known that the allometric equation of Huxley does not have a solid theoretical basis …*" (Slack, 1999). Additionally, the debate was heated by new insights into the geometric view of Rubner, including the addition of a physical perspective to the issue. D'Arcy Thompson, for example, formulated problems relating 'Growth and Form' based on an energy minimisation principle (Thompson, 1942; Goodwin, 1999).

Within the specific field of metabolic physiology, the basis of current thought originated from the data compilations and analyses of Kleiber (1932, 1961), Brody (1945; see, for

example, Calder, 1996) and Hemmingsen (1960; see, for example, Calder, 1996), who reported what they interpreted as a scaling rule of the basal metabolic rate that was characterized by an exponent of 3/4 instead of the proposed 2/3. Additional questions were then raised concerning the appropriate interpretation of the 3/4 exponent (e.g. Bertalanffy, 1968; Heusner, 1982; Dodds et al., 2001; White and Seymour, 2003; Hochachka et al., 2003; Kaitaniemi, 2004) and there has been much discussion since on the possible 'causes' of such an exponent, including the elastic energy scale of McMahon (1973), the similarity principles of Gunther (1975), the heterogeneous catalytic bioreactor of Sernetz et al. (1985), the constructal law of Bejan (1996, 2000); the fractal similitude of West et al. (1997), the similitude in cardiovascular systems of Dawson (2001), the central source and distribution of sinks of Dreyer (2001), and the fluid dynamics approach of Rau (2002) and Santillan (2003). A common trait of most of these postulates is that they rely on the same principles as Rubner and Thompson, i.e., a geometric/physical law leading to an energy minimisation or constraint.

From the above discussion it must be clear that the validity of the allometric approach, and the values, causes and consequences of its associated exponents in energy metabolism, merits examination. It is not surprising, then, that certain characteristics of the metabolic rate of animals, which appear to be specific to given size ranges, have been rather overlooked in the literature. In contrast to the common tendency to pool animals along a size continuum Bertalanffy (1968), for example, divided rats in two size groups (animals weighing more or less than 110 g) because a 110 g body mass "*corresponds to many physiological modifications*". Along the same lines, McNab (2002) pointed out some important metabolic differences occurring in medium-sized (100 g<M_b<2 kg) homeotherms due to highly different patterns in thermal conductance, and claimed that these differences are related not only to body mass but to food availability and climate as well.

These problems are particularly evident when the subjects of study are very small endotherms. Besides food and climate effects on the scaling of metabolism, analysis of data sets revealed that the pattern of this scaling is different for small and large masses (for a detailed study of this subject, see McNab, 1983). Additionally, Schuchmann and Schmidt-Marloh (1979) reported an unusual pattern of body temperature (T_b) control and oxygen consumption in two species of Jamaican hummingbirds, for which they described a fairly large thermoneutral zone (20–29°C). Within this temperature range, T_b was constant, but metabolic rate was twice the minimum recorded. Our own studies of hummingbirds in an open-respirometry system designed for the fast acquisition of metabolic rate data also revealed unusual features (Chaui-Berlinck et al., 2002a): the birds did not show a steady-state condition of T_b and metabolic rate. Time-series analysis revealed that the observed oscillatory pattern of metabolic rate exhibited long-range correlation, an observation that is compatible with the existence of control mechanisms operating to maintain such a pattern. Regarding very small mammals, it has been long recognised that some shrews maintain a body temperature and metabolic rate much higher than would be expected with the 0.75 allometric scaling rule as the null hypothesis (e.g. Gehr et al., 1980; Sparti, 1990; Brown et al., 1997). Moreover, the body temperature of these animals is highly variable (e.g. Nagel, 1991; Brown et al., 1997). Some bats also seem to have both low and variable body temperatures, instead of the 'homeothermic' condition they supposedly share with other mammals (e.g. Chappell and Roverud, 1990; Hosken and Withers, 1997; Bartels et al., 1998). The metabolic pattern observed by Bartels et al. (1998) in 14 g bats suggests an absence of steady-state conditions, a result compatible with that of Corp et al. (1997) who detected remarkable variation in both T_b and metabolic rate in supposedly euthermic wood mice. Variations around 4°C in 'euthermic' body temperature also occur in elephant shrews (Macroscelidea), small mammals of body mass 40–60 g (Lovegrove et al., 2001). Finally, it is noteworthy that even size inheritance in mammals weighing less than 18 g might operate differently, as suggested by Smith et al. (2004). The authors argue that this is probably due to biomechanical and thermoregulatory problems in such small animals.

The purpose of this paper is to investigate whether very small homeotherms constitute a special case regarding the control of body temperature, and the extent to which this situation would lead to unexpected problems in the search for a causative explanation to the ¾ allometric exponent based on geometric (morphological) and physical factors. We first analyse what is meant by the concept of standard metabolic rate and discuss the assumptions that this concept carries with it. Then, we outline a minimalist control system of body

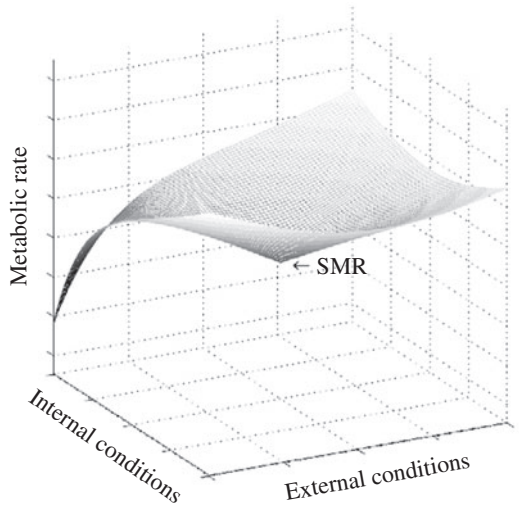


Fig. 1. Standard metabolic rate concept. There are internal and external conditions that once met supposedly lead the metabolic rate B of a homeotherm to the SMR. This is, thus, a local minimum of B (δB=0). See text for further discussion.

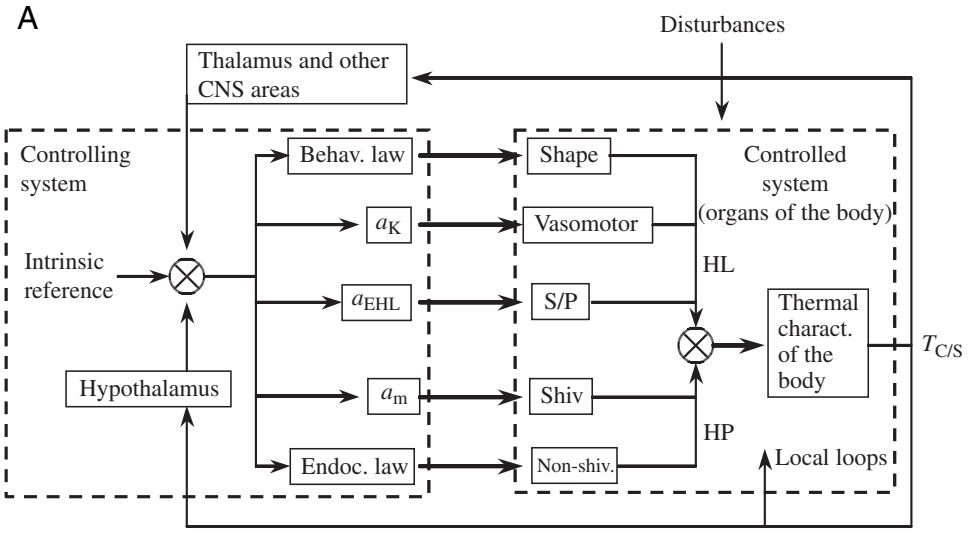
temperature, assuming that the parameters of the model follow scaling rules. From the effects of these scaling rules in the behaviour of the system we verify whether the assumptions implied by the standard metabolic rate concept can be fulfilled over the entire range of homeotherm body mass.

The definition of the metabolic rate and what this implies

To measure standard metabolic rate (SMR) as defined by the IUPS Thermal Commission (2003), '*The specified standard conditions are usually that the organism is rested (or as near to rested as is possible), fasting (if possible), awake, and in a thermoneutral environment*'. The definition excludes torpor and other metabolic depressed states, as well as the reproductive phase (McNab and Brown, 2002). Thus SMR is a function of external (i.e. surrounding ambient) and internal (i.e. organismic) environmental conditions. Considering that once these conditions are met SMR is the minimal energetic situation, it is tacitly assumed that the function B (the metabolic rate of a homeotherm) converges to SMR. Standard metabolic rate is, then, a local minimum of B and thus, at the SMR, $\delta B=0$ (δ indicates infinitesimal variation; see Fig. 1). A corollary of this first assumption is that when an animal is in the SMR condition, the short-term control mechanisms that maintain body temperature (T_b) do not operate, since δB is zero. This hidden assumption must be satisfied if a scaling rule is settled in terms of physical and morphological causes. Otherwise, the SMR scaling rule would be subjected to the 'volition' of metabolic controllers and physical/morphological arguments would no longer make sense. In engineering terms, this is equivalent to operating in an open-loop system.

A minimalist T_b control system and its analysis

Body temperature control in homeotherms is hierarchical, the major control centre being the hypothalamus (Boulant, 2000). This organ receives and integrates information from peripheral and core temperature sensors and modulates a number of responses, including motor outputs modifying metabolic rate (shivering and non-shivering processes) and changes in the thermal conductance through skin and peripheral blood flow, fur/feather positioning, etc (Graener et al., 1984; Gordon, 1986; Boulant, 2000; Cooper, 2002). Empirical evidence to date indicates that the control law is proportional to the error between T_b and a reference temperature (Graener et al., 1984; Gordon, 1986; Webb, 1995; Hexamer and Werner, 1996; Boulant, 2000; Cooper, 2002). Fig. 2A illustrates the biological basis of the process described above. Thus a very simple control system of T_b in homeotherms should comprise three dimensions: (1) body temperature, (2) metabolic rate and (3) thermal conductance.



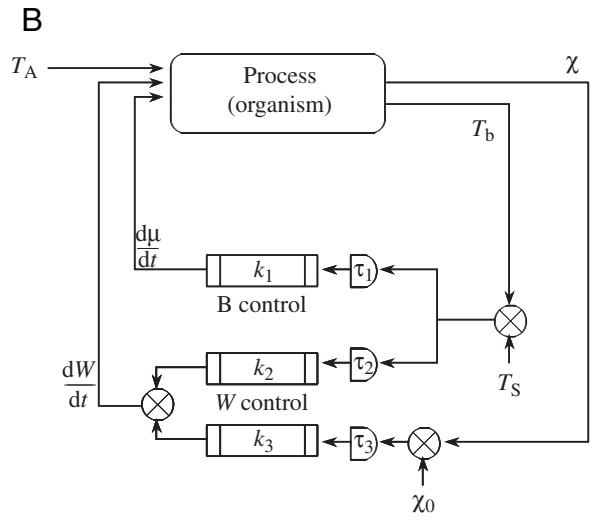


Fig. 2. (A) Biological basis of T_b control. a_m, metabolic proportionality factor; endoc. law, endocrine controllers law; shiv, shivering thermogenesis; non-shiv, non-shivering thermogenesis. These four blocks constitute the metabolic rate controller/process in our model (see B). Behav. law, posture controllers law; a_K, proportionality factor for non-evaporative heat transfer; a_{EHL}, proportionality factor for evaporative heat transfer; shape, body positioning; vasomotor: peripheral blood perfusion; S/P, sweating and panting. These six blocks constitute the thermal conductance controller/process in our model (see B). HP, heat production; HL, heat loss; $T_{C/S}$, core and skin temperatures. The thermal characteristics of the body correspond to the thermal inertia and 'disturbances' to T_A in our model. Notice that our model does not take into account local loops and other central nervous system areas interfering in the control. (Scheme based on fig. 2 of Cooper, 2002). (B) Schematic representation of the T_b control system modelled in Eq. 1–3. See text for details. Compare this control system to the biological one presented in A.

The time variation of T_b can be macroscopically described by the relationship between heat input (metabolic rate) to and heat loss (due to temperature difference) from the system (the organism; e.g. Chaui-Berlinck et al., 2002a,b):

$$dT_b / dt = b[B - W(T_b - T_a)] , \quad (1)$$

where B is the metabolic rate, W is the wet thermal conductance, T_A is the ambient temperature and b is the inverse of the product body mass × thermal capacitance of the body. The time variation of metabolic rate can be expressed, in our minimalist model, as:

$$dB / dt = k_1[T_s - T_b(t-\tau_1)] , \quad (2)$$

where k_1 is the gain in the closed control loop and T_S is the set-point (or reference) temperature of the organism. In a simple statement, metabolic rate B increases as the difference between the set-point and the sensed body temperature increases. Notice that the loop response undergoes a time delay $t-\tau_1$, as expected in biological systems. The time variation in thermal conductance is expressed as:

$$dW / dt = -k_2[T_s - T_b(t-\tau_2)] - k_3[W(t-\tau_3) - W_0] , \quad (3)$$

where k_2 is the gain in the closed control loop of thermal conductance in relation to T_b, k_3 is the gain in a control loop that tends to match W to a given thermal conductance W_0. This last term is a minimal value of thermal conductance for a given organism. In contrast to what happens with metabolic rate, the thermal conductance of the organism is expected to decrease as the difference between the set-point and the sensed body temperatures increases (hence the minus signal in k_2). Each loop has its own response time delay. Fig. 2B illustrates the scheme of this control system.

Steady-state conditions: equilibrium point of the system

Considering that the definition of SMR presumes steady-state conditions of the organism, we searched for sets (T_b*, B*, W*) of values that render all derivatives simultaneously equal to zero. There is only one such set:

$$T_b^* = T_S , \quad (4a)$$

$$W^* = W_0 , \quad (4b)$$

$$B^* = W_0 \Delta T , \quad (4c)$$

where ΔT is the difference $T_S - T_A$. Notice that the decaying rate of thermal conductance, $-k_3(W-W_0)$ in Eq. 3, becomes a 'forcing term' for minimisation of energetic demand: the animal tends to adjust its heat loss to as minimal a value as possible, thus decreasing the metabolic rate B* associated with a given ambient temperature. Loosely speaking, the model satisfies a condition of energy minimisation.

Stability

The next step is to check the stability of the equilibrium point (EP) in order to determine whether the system tends to the EP once perturbed. In the analysis (e.g. Murray, 1993; Monteiro, 2002), we obtain the characteristic polynomial of the linearised system as:

$$\lambda^3 + bW_0\lambda^2 + bk_5e^{\lambda\tau_5} + \lambda b(k_1e^{\lambda\tau_1} + \Delta T k_2e^{\lambda\tau_2} + W_0k_3e^{\lambda\tau_3}) + \lambda^2k_3e^{\lambda\tau_3} = 0 , \quad (5)$$

where λ represents an eigenvalue of the system, k_5 is the product k_1k_3 and τ_5 is the sum $\tau_1+\tau_3$. In order to have an asymptotically stable EP, all the three eigenvalues of the system must have negative real part. Eq. 5 is a transcendental equation and thus has no analytical solution, so insights into the behaviour of the system can be obtained from a graphical analysis. Before proceeding in this direction, however, we must return to the original problem we are investigating.

The scaling problem

There are several 'scaling' exponents that we should take into account in the analysis. In the following relationships, the subscript '1' refers to a reference value of a 'standard' homeotherm of 1 kg. The first obvious exponent is the one from the SMR scaling:

$$SMR = SMR_1M_b^{\beta} . \quad (6a)$$

Another exponent is the one from the scaling of wet thermal conductance. As shown in many reports (e.g. Schleucher and Withers, 2001), W increases with body mass as:

$$W = W_1M_b^{\alpha} . \quad (6b)$$

Taking together Eq. 4c, 6a and 6b, a scaling in the temperature difference ΔT emerges (see, for example, Schleucher and Withers, 2001). However, with this approach, there is no need to assume that body temperature is under control because only the difference between the set-point and ambient temperatures is taken into account. We do require that T_S is an 'un-scaled' constant, i.e. that set-point temperature is a constant reference value across the species under consideration. With this important constraint, we obtain a scaling rule for the minimal critical ambient temperature:

$$T_{A,\,min} = T_S(1-M_b^{\gamma}) + T_{A1}M_b^{\gamma} , \quad (6c)$$

where $\gamma=\beta-\alpha$, and T_{A1} is the minimal critical ambient temperature for a standard homeotherm of 1 kg. Notice that Eq. 6c is the one that preserves body temperature control in the phylogenetic or ontogenetic scaling analysis.

The factor b, which represents the inverse of thermal inertia, scales with the inverse of body mass. Considering thermal capacitance due to tissue composition of the organisms as roughly constant across species, we have:

$$B = b_1M_b^{-1} . \quad (6d)$$

Finally we consider the gains and time delays in the control loops. This is a relatively unexplored issue so we must trust a sensible speculation. Some empirical findings suggest that the gains in the loops in which we are interested increase as body mass decreases. These come from data on rates of rewarming after metabolic depression (Geiser and Baudinette, 1990; Stone

and Purvis, 1992) and on the hypoxic respiratory 'drive' (Boggs and Tenney, 1984). Here, we generalise that the scaling of a given gain is:

$$k = k_{ref} M_b^{\xi} ,$$

where

$$\xi < 0 . \quad (6e)$$

We also assume that time delays decrease as body mass decreases, simply because size is reduced and thus the distances between controllers and processes. We generalise the scaling of a given time delay as:

$$\tau = \tau_{ref} M_b^{\zeta} . \quad (6f)$$

Let us summarize the scaling laws that will drive our analysis, i.e. Eq. 6a–f. On the one hand, standard metabolic rate, wet thermal conductance and time delays increase as body mass increases. On the other hand, minimal critical ambient temperature (the lower limit of the thermoneutral zone), the inverse of thermal inertia, and the gains in the control loops, decrease as body mass increases. Therefore, a large homeotherm is subjected to a condition of low gains associated with high thermal inertia, while a small homeotherm faces a condition of high gains and low thermal inertia. Also, on a mass-specific basis, wet thermal conductance is lower in large homeotherms because α (Eq. 6b) is <1. We now return to the problem of evaluating the stability of the system with time delays, expressed in its full form in Eq. 5.

Scaling and stability

To explore the relationship between the scaling rules from Eq. 6 and the behaviour of the equilibrium point (i.e. its stability), it is convenient to first separate Eq. 5 into two main components, a pure polynomial and one containing the exponential terms of λ:

$$\lambda^3 + bW_0\lambda^2 = -[bk_5 e^{\lambda\tau_5} + \lambda b(k_1 e^{\lambda\tau_1} + \Delta T k_2 e^{\lambda\tau_2} + W_0 k_3 e^{\lambda\tau_3}) + \lambda^2 k_3 e^{\lambda\tau_3}] .$$

The crossings of these two functions correspond to the solutions of Eq. 5. Table 1 shows the numerical values we employed in most of the graphical solutions and simulations.

Fig. 3A shows the general solution obtained for hypothetical large homeotherms. We can identify three crossings between the polynomial and exponential functions of λ, meaning that, under the situation considered, the equilibrium point is an asymptotically stable node. This situation extends to small animals in the range of 100 g, but we can see that the exponential part of the function exhibits an upwards shift in relation to the polynomial part (Fig. 3B). The 50 g animal represents a borderline situation. There is one crossing near the region $\lambda \leq\cong 0$ and then the two functions touch each other at a lower value of λ that represents a double root (Fig. 3C). The system is still an asymptotically stable node at this situation, but oscillations are about to emerge: a further decrease in body mass leads to a single crossing between the functions belonging to $\mathrm{Im}(\lambda)=0$. The other two solutions have $\mathrm{Im}(\lambda)\neq 0$ and thus the system has an asymptotically stable focus. Extending the decrease in body mass to even lower values, the real part of the conjugate root may become positive, rendering the system unstable (Fig. 3D). At this point, non-linearities in both the control system and the process itself become important and the oscillatory pattern would be sustained within some boundaries. In other words, an unstable focus in the minimalist system of body temperature control becomes a centre in a more complete system due to non-linearities that the minimalist system does not take into account. For instance, in the simulations of the model, these other non-linearities were represented by limited ranges of B and *W*, proportional to values estimated by Eq. 6a,b. These simulations are shown in Fig. 4.

Table 1. *Values of the 'standard 1 kg' and scaling exponents used in most of simulations*

Variable	Standard 1 kg value	Scaling coefficient	
B	6.4	β	0.75
W	0.8	α	0.60
$T_{A,min}$	29	γ	0.15
b	1		–1
k_1	0.0070	ξ	–0.2
k_2	0.0035	ξ	–0.2
k_3	0.0035	ξ	–0.2
τ	4	ζ	0.33
T_S	37		0

The γ exponent is the difference between β and α (see text for explanation).

In the conditions depicted above, asymptotically stable systems converge spontaneously on the metabolic rate anticipated by the allometric scaling rule (Eq. 6a, with β=0.75). Therefore, these organisms would fulfil the measuring requirements of SMR, or, in other words, the metabolic rate corresponds to the one where δB=0. Nevertheless, if the gains of the control system change it to the unstable situation, the saturation of the responses turns the system into a centre. Under these circumstances, metabolic rate, body temperature and thermal conductance oscillate continuously (Fig. 4C), and the very definition of SMR no longer makes sense. Note that the oscillatory behaviour of the system shifts upwards the mean value of the metabolic rate in the function of its metabolic scope under the experimental situation. This means that measurements of metabolic rate of an animal of this type would result in values higher than the one anticipated by the allometric scaling rule.

Scaling T_S

The previous analysis was based on the maintenance of the set-point temperature across the size variation (see Eq. 6c). This is a crucial assumption, as explained earlier, because it would be pointless to group homeotherms of different body temperatures to analyse 'size effects'. However, body temperature tends to decrease as size decreases, i.e. many small

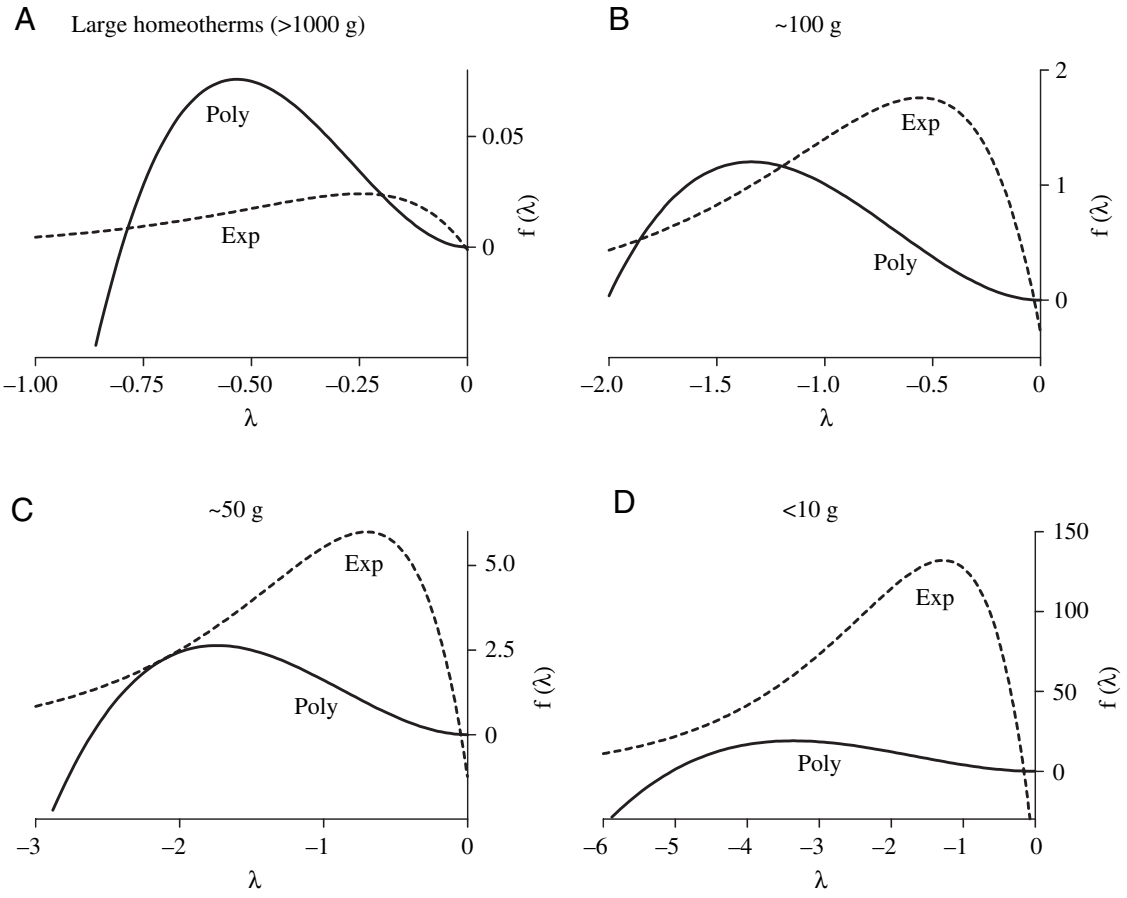


Fig. 3. Exponential (exp; broken lines) and pure polynomial (poly; solid lines) parts of Eq. 5 plotted separately as functions of λ. The crossing points between the functions are the solutions to the characteristic polynomial of the system. λ values are shown in the negative real axis portion, thus, crossing points correspond to pure real negative λ and, therefore, asymptotically stable nodes in the corresponding eigenvector. Notice that at λ=0, the polynomial part is zero and the exponential part is negative ($-bk_5$). (A) A putative large homeotherm (>1000 g) is represented. Notice the existence of three crossing points. (B) Small (≅100 g) homeotherm. (C) 50 g homeotherm, represents a putative limiting condition. The functions touch each other just twice. Any further decrease in body mass would make the functions fall apart and two eigenvalues would have imaginary parts, rendering the system a focus. The focus is asymptotically stable while the real part of each complex conjugate λ belongs to the negative real axis. The focus would become unstable when the complex conjugate λ has positive real part. This potentially occurs when the functions are 'far away' from each other, as depicted in (D), representing a 10 g homeotherm.

homeotherms tend to have low body temperatures compared with their larger-sized counterparts (e.g. White and Seymour, 2003).

When the term ΔT in Eq. 5 is diminished, a downwards shift occurs in the descending portion of the exponential function, approaching the polynomial and the exponential parts of the characteristic polynomial (Eq. 5). This indicates a potential change from the oscillatory behaviour to an asymptotically stable focus, and so the metabolic rate measured would fall on the expected by the allometric scaling rule. However, this would occur due to changes operating at the controller level and should not be considered as a pure law of 'geometrical/physical optimisation or constraint' alone.

Why maintain a system with high gains?

It is interesting to speculate on the putative reasons why a small homeotherm should maintain high gains in the T_b control system. We may first claim influences of the phylogenetic history of the lineage, for example, a size reduction over evolutionary time in which cladogenesis involves the retention of key features related to temperature control. This explanation, however, is not causal, and the question of why a high gain trait would be retained through evolution remains. An alternative or complementary, yet tentative, explanation, arises from looking at the T_b control system itself: the biological control system certainly has more than one dimension (see above), and higher than first-order systems exhibit a rich variety of behaviours, potentially becoming oscillating when gains increase (Nise, 2000). These fast-responding systems would cope well with a rapidly changing external environment (inputs). For instance, changes in ambient temperature would lead to changes in the amplitude of the oscillations, but the mean T_b value would be kept constant all the time. Conversely, a slow-responding

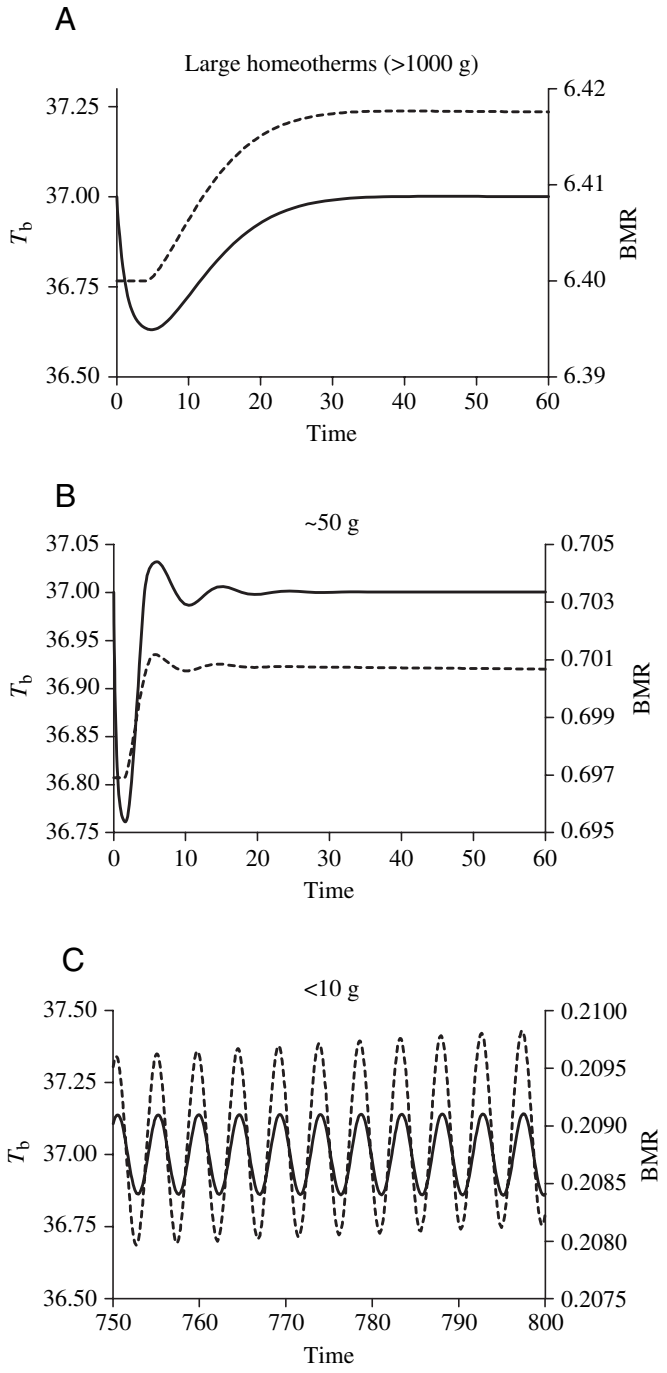


Fig. 4. Temporal profiles of T_b and B in conditions representing a large homeotherm (A), a homeotherm weighing a little less than 50 g (B) and a 10 g homeotherm (C). Time in arbitrary units. Solid lines, T_b; broken lines, B. Notice the asymptotically stable node in the large homeotherm condition, the asymptotically stable focus (damped oscillations) in the ≅50 g condition, and the centre (sustained oscillations) in the 10 g condition. Simulations were done in MatLab 6.1 and Simulink.

system would experience a shift in T_b that would persist for a period, depending on the change of the external temperature.

Conclusions

The present study is intended to offer a new perspective to the relationship between body mass and standard metabolic rate of homeotherms, a perspective in which body temperature control acts on the metabolic rate of an endotherm. Our model offers important support to the idea that size effects can modify the intrinsic characteristics of the steady-state condition (i.e. the equilibrium point of the system), potentially changing the behaviour from asymptotically stable to a centre, generating a situation where no steady-state conditions would apply. The control system presented here is extremely simple but the model is solid, to convey the main points discussed here. We recognize that many possible non-linearities of the process and of the controller have not been considered in the analysis. Although we offered saturation of responses as an example, we also acknowledge that a multiplicity of controllers, spatial heterogeneities in temperature distribution, non-linearities in the controllers themselves, and others (e.g. Gordon and Heath, 1983; Werner et al., 1989) would render the temporal patterns of the system more realistic, yet much more complex. The important point is that such temporal patterns would be more distant from the well-behaved δB=0 of the classical SMR. Therefore, terms of size–temperature covariance are potentially more complicated than might be evident by treating them as isolated sources of variance.

Finally, we argue that control loops operating to maintain a stable elevated body temperature (homeothermy) must be taken into account in the conundrum of metabolic rate scaling. The operating status of the control system imposes another facet to the issue, lying outside the realm of geometrical and physical constraints. Ignoring the scaling of these control mechanisms would be to mask the allometric phenomenon itself, particularly at very small body sizes. Physical and geometrical principles are classical and relevant causes in scaling. However, control systems add a new dimension to the problem that must be included in the guideline principles of ‘allometric scaling causes’.

List of abbreviations and symbols

B	metabolic rate
b	inverse of the product body mass × thermal capacitance of the body
ΔT	the difference T_S–T_A
EP	equilibrium point
k	gain in the control loop
M_b	body mass
SMR	standard metabolic rate
T_A	ambient temperature
$T_{A,min}$	minimum critical ambient temperature
T_b	body temperature
T_S	set-point temperature
W	wet thermal conductance
δ	infinitesimal change
λ	an eigenvalue of the system
τ	time delay

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