Unified theory of interspecific allometric scaling

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Abstract
A general simple theory for the interspecific allometric scaling is developed
in the $d + 1$-dimensional space (d biological lengths and a physiological time)
of metabolic states of organisms. It is assumed that natural selection shaped
the metabolic states in such a way that the mass and energy $d + 1$-densities are
size-invariant quantities (independent of body mass). The different metabolic
states (basal and maximum) are described by considering that the biological
lengths and the physiological time are related by different transport processes of
energy and mass. In the basal metabolism, transportation occurs by ballistic and
diffusion processes. In $d = 3$, the 3/4 law occurs if the ballistic movement is
the dominant process, while the 2/3 law appears when both transport processes
are equivalent. Accelerated movement during the biological time is related
to the maximum aerobic sustained metabolism, which is characterized by the
scaling exponent $2d/(2d + 1)$ (6/7 in $d = 3$). The results are in good agreement
with empirical data and a verifiable empirical prediction about the aorta blood
velocity in maximum metabolic rate conditions is made.

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Metabolic rate B and body mass M are connected by the relation $B = aM^b$, where b is
the allometric exponent and a is a constant. For several decades it was accepted that the
basal metabolic rate (BMR) among almost all organisms [1–3] (interspecific scaling) was
characterized by $b = 3/4$ (Kleiber's law [4]). A few years ago, theoretical explanations
of the ubiquity of the 3/4-law based on the resource distribution network common to all
organisms [5] and on network geometry optimization [6, 7] were proposed. Kleiber's law
has however been questioned recently. On the observational side, it is not clear whether the
value of b is 3/4, 2/3 or even variable in both interspecific and intraspecific (same species)
scaling [8–10]. On the theoretical side, there are several debates about the validity of these

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models [8, 11] undermining the rational basis for the scaling law. A related open question is why maximum aerobic sustained metabolic rate (MMR) of endothermic animals scales with an exponent larger than that of BMR [9–14]. In this work we show how, on the basis of a few quite general hypotheses, all the aspects of metabolic scaling mentioned above may be accounted for. The central assumption of our approach is that, if one characterizes the metabolic state of an organism as a point in a space comprised of d biological lengths and a biological time τ, then the $(d + 1)$-dimensional mass and energy densities are size-invariant under natural selection. Using this idea we may, given the dominant mass and energy transport processes, obtain dynamical relations involving lengths and time. The latter are characterized by invariant quantities (diffusion coefficient, velocity etc.). Each transport mechanism is related to a different metabolic state, with its own values for allometric exponents, in agreement with data.

The key questions in interspecific allometry are: (1) Is there a universal interspecific BMR exponent b? (2) Is there a model that can describe both BMR and MMR scaling? (3) Which quantities have their scaling determined by the BMR, and which by the MMR?

We present a simple and general theory that answers, at least partially, these questions and explain some aspects of the allometric scaling of organisms. As all biological processes depend on characteristic times, it is natural to include a characteristic time, as well as various characteristic lengths, when specifying the metabolic state of an organism [15, 16]. We therefore associate the metabolic state of an organism with a point in a $d + 1$-dimensional space with d biological lengths $L_1, L_2 \ldots, L_d$ and a biological time τ. Here d is the number of spatial dimensions; although we usually have $d = 3$, we will work in general dimension d. Examples of biological lengths L and times τ are the total aorta length in mammals, the length of capillaries, the mean distance from cell surface to mitochondria in unicellular organisms, the duration of one heartbeat, the capillary blood transit time or the turnover time for glucose [1, 2, 7, 16].

We now look for simple, general relations constraining the distribution of points in the space of metabolic states. It is usual for an animal to make several transitions between states A_{basal} and A_{max}, through a series of complex biochemical processes. We do not consider these transitions. Rather we are interested in describing how natural selection has shaped the state A_{basal} and other ones belonging to the space of metabolic states of all organisms (or a group of them). An organism is characterized by the fundamental quantities of mass and available energy for the metabolic processes. We therefore identify the mass M and available energy E as the fundamental variables characterizing an organism. Since we have a $d + 1$-dimensional space we shall use the mass density $\rho_{d+1}(L_1, \ldots, L_d, \tau)$ (mass per unit volume and unit time) and the energy density $\sigma_{d+1}(L_1, \ldots, L_d, \tau)$ (available energy per unit volume and unit time). We assume that during evolution, natural selection enforces the constraints of *size-invariant* (independent of body mass) ρ_{d+1} and σ_{d+1} (our first and second hypotheses, respectively). Our third hypothesis is that the scaling of the metabolic states is determined by the *dominant dynamical transport processes of nutrients* (mass and energy), which are characterized by size-invariant quantities (diffusion coefficient, average velocity, etc.). Note that the first and second hypotheses furnish two relations valid for all metabolic regimes. Different metabolic scalings will appear because there are different ways to transport nutrients.

From the second hypothesis we obtain that $E = \sigma_{d+1}\tau V_d$, where τV_d is the $d + 1$-volume and $V_d = L_1 L_2 \ldots L_d$. Since power is defined as $P = dE/dt$, energy can be written in terms of B, the power averaged over the time scale τ, as $E = B\tau$. We identify B as the metabolic rate. Therefore from the first and second hypotheses we obtain that

$$M = \rho_{d+1}\tau V_d, \qquad \text{and} \qquad B = \sigma_{d+1}V_d. \qquad (1)$$

Let us briefly present a qualitative argument about an optimal delivery that supports our postulates. Consider two organisms with the same body mass M belonging to the same group. The organism with the larger biological volume V_d has the larger nutrient distribution network and more fuel and oxygen are arriving to the cells in a unit time. Therefore its cells must have a fast metabolism in order to consume the fuel. Of course, a fast metabolism is related to a small biological time. On the other hand, a small V_d implies in a larger biological time τ. These arguments suggest that the product τV_d is constant for these organisms.

An immediate consequence of our hypotheses is that the power per mass (specific metabolic rate), namely

$$\frac{B}{M} = \frac{\sigma_{d+1}}{\rho_{d+1}} \frac{1}{\tau},$$

is inversely proportional to the metabolic time. Animals with a small τ, such as small mammals, require larger power by unit mass than ones with a large τ (large mammals) because their cells have a large mitochondrial density [17].

Obviously, τ cannot be 0 neither ∞; there must be a minimal and a maximal metabolic times $\tau_{\min}$ and $\tau_{\max}$. For animals, a lower bound for $\tau_{\min}$ can be found from the observation that the biological volume of an organism V_d cannot be larger than its *spatial volume V*, since the biological lengths L_i characterize the organism's anatomy on the scale of the body, or some organ or cellular structure. For compact animal bodies we have that $M = \rho V$, where ρ is the usual d-dimensional mass density, which is approximately constant. Using $\rho V = M = \rho_{d+1} \tau V_d$ in $V > V_d$, we obtain $\tau > \tau_{\min} = \rho/\rho_{d+1}$. Since B/M cannot be zero it must exist an size-invariant $(B/M)_{\min}$ related to the minimum power per mass to keep the organisms just alive, the so-called tissue maintenance specific metabolic rate [10]. This implies that $\tau_{\max} = \sigma_{d+1}/(\rho_{d+1}(B/M)_{\min})$. Note if τ is size-invariant, such as $\tau_{\max}$, we have isometric scaling of the metabolic rate ($b = 1$).

The relation $M \propto \tau V_d$, derived from the first hypothesis, is a generalization of the result of Banavar *et al* [6], namely $V_{\text{net}} \propto L^{d+1}$, where V_{net} is the total volume of an efficient distributive network. Using that the blood volume V_{net} is proportional to mass, they obtained $M \propto L^{d+1}$, a basic relation to deduce the 3/4-law. The two relations are equal when $\tau \propto L$, a condition valid for the BMR. Moreover, the result $M \propto L^{d+1}$ is also crucial to obtain the BMR exponent in the model of West, Brown and Enquist (WBE) [5]. This relation is also a generalization of the equation $F \propto (L_p/u)B$ of Banavar *et al* [7], where L_p is the physical length of the system and u is the characteristic length scale. If we rewrite this equation as $M \propto \rho t V_d$, where ρ is the tissue density and t is the physiological time related with the rate of energy use per unit volume, it becomes similar to equation (1).

Although, from our third assumption, we need some dynamical size-invariant quantities, like the blood flow speed velocity v_0 in the aorta or in the capillaries, the length l_c and the radius r_c of capillaries *are not necessarily invariants*.

Let us first study the case of transport via diffusion. We have only one metabolic length scale ($L_1 \propto L_2 \propto \cdots \propto L_d \propto L$), so the biological volume is given by $V_d \propto L^d$. Since diffusion over short distances is fast, it is possible that the metabolic rate of very small organisms is governed by this process. It is well known that $L = D_0 \tau^{1/2}$, where D_0 is the size-invariant diffusion coefficient. Since $\tau = (L/D_0)^2$, we obtain from equation (1) that $M \propto L^{d+2}$. This relation furnishes how L depends on M and we can use again equation (1) to obtain that

$$L \propto M^{\frac{1}{2+d}}, \qquad \tau \propto M^{\frac{2}{2+d}}, \qquad B \propto M^{\frac{d}{2+d}}.$$

In $d = 3$, the metabolic exponent is $b = 3/5$.

For larger organisms diffusion is inadequate. Transport by convection is then utilized on large length scales. In mammals, for example, we find the cardiovascular system that transports blood to the capillaries, where the cells are fed by diffusion. Since blood circulates in an ballistic regime, we consider that the BMR is basically driven by *ballistic transport*, namely $L = v_0\tau$, where the velocity v_0 is size-invariant. Now we must specify how the different metabolic steps are related. We call BMR-1 the scenario of *a single metabolic relevant length* $L_1 \propto L = v_0\tau$ and *a single time* τ, both related to the ballistic transport. The other lengths, related to other metabolic steps, have evolved to meet it, namely $L_2 \propto \ldots \propto L_d \propto L$. Using that $V_d \propto L^d$ in equation (1), we write

$$L \propto \tau \propto M^{\frac{1}{d+1}}, \qquad B \propto M^{\frac{d}{d+1}}.$$

For $d = 3$ we find the 3/4-law, namely $\tau \propto L \propto M^{1/4}$ and $B \propto M^{3/4}$. These results are the same as those of WBE [5] and Banavar *et al* [6, 7].

In the BMR-2 scenario, we have *different relevant lengths and times* related to the metabolic processes. However, due to the concept of symmorphosis [18], which states that all metabolic steps have co-evolved in order that no step is more limiting than another, we will end up with a single time τ and $d - 1$ rescaled lengths. In a 'cylindrical' symmetry we have $L_1 \propto L = v_0 t_1$ (ballistic term) and $d - 1$ lengths proportional to $R = D_0 t_2^{1/2}$ (diffusion). Both v_0 and D_0 are size-invariant. From the symmorphosis principle ($t_1 = t_2 = \tau$), it follows that $R = (D_0/v_0^{1/2})L^{1/2}$. The biological volume is $V_d \propto R^{d-1}L$. From equation (1) we obtain that

$$L \propto \tau \propto M^{\frac{2}{3+d}}, \qquad R \propto M^{\frac{1}{3+d}}, \qquad B \propto M^{\frac{1+d}{3+d}}.$$

Then in $d = 3$ the BMR-2 scenario yields the 2/3 law, without mention of the area/volume ratio. We obtained both 3/4 and 2/3 laws from the same transport processes: convection and diffusion. If convection is the dominant limiting process we have the 3/4 law; if the two processes are equivalent we obtain the 2/3 law.

The circulatory networks of endothermic animals are dynamical ones which are adjusted according to the metabolic state. The transition from resting to maximum activity can be described very briefly as follows: (a) the heart increases its rate and output; (b) the mean arterial pressure and peripheral extramuscular resistance increase; (c) arterial blood volume increases due to constriction of the veins; (d) extramuscular flow remains essentially constant, somewhat reduced in some organs but increased in others; and (e) total flow and muscular flow increase, with all muscular capillaries activated. The items (a), (c) and (e) suggest that we have a 'forced movement' during the characteristic time τ. This means that the typical constant velocity can be written as $v = a_0\tau$, where a_0 is a size-invariant acceleration. Consequently the MMR is driven by an *inertial movement accelerated during time τ*, implying that $L = v\tau = a_0\tau^2$.

If inertial transport is the only relevant process (MMR-1 scenario), it follows that $L_1 \propto L_2 \ldots \propto L_d \propto L = a_0\tau^2$. Since $V_d \propto L^d$ and $\tau \propto L^{1/2}$, we obtain from equation (1) the metabolic relations:

$$L \propto M^{\frac{2}{2d+1}}, \qquad \tau \propto M^{\frac{1}{2d+1}}, \qquad B \propto M^{\frac{2d}{2d+1}}.$$

For $d = 3$ we have that $L \propto M^{2/7}$, $\tau \propto M^{1/7}$ and $B \propto M^{6/7}$. This results agree with the ones obtained trough a generalization of WBE ideas to MMR scenario [14].

In the MMR-2 scenario, diffusion and inertial movement are equally relevant. We again choose a 'cylindrical' symmetry so that have that $L_1 \propto L = a_0\tau^2$ while the remaining $d - 1$ lengths are of order $D_0\tau^{1/2}$. It follows from equation (1) that

$$L \propto M^{\frac{4}{d+5}}, \qquad \tau \propto M^{\frac{2}{d+5}},$$
$$B \propto M^{\frac{d+3}{d+5}}, \qquad R \propto M^{\frac{1}{d+5}}.$$

When $d = 3$, we obtain that $L \propto M^{1/2}$, $\tau \propto M^{1/4}$, $R \propto M^{1/8}$ and $B \propto M^{3/4}$.

Table 1. Allometric exponent y describing the dependence of a variable Y on body mass M ($Y \sim M^y$). Under parenthesis is the error in the last significative of the observed quantities.

Variable	Exponent		
	Predicted	Observed	Reference
MMR	0.86 (MMR-1)	0.83(7)	[9]
		0.88(2)	[12]
		0.87(3)	[13]
		0.85	[21]
		0.87(5)	[22]
Capillary density	−0.14 (MMR-1)	−0.14(7)	[3, 19]
Heart rate at MMR	−0.14 (MMR-1)	−0.17(2)	[23]
		−0.16(2)	[24]
		−0.15	[25]
BMR	0.75 (BMR-1)	0.74(2)	[9]
	0.66 (BMR-2)	0.67	[8]
		0.69(1)	[26]
Heart rate at BMR	−0.25 (BMR-1)	−0.25(2)	[1, 9]
	−0.33 (BMR-2)	−0.27	[3, 26]
Aorta radius	0.36 (MMR-1)	0.36	[3, 26]
	0.38 (BMR-1)		
	0.33 (BMR-2)		
Aorta length	0.29 (MMR-1)	0.32	[1, 3]
	0.25 (BMR-1)	0.31	[26]
	0.33 (BMR-2)		

The transportation network can be characterized by 'aorta' and 'capillaries'. Note that the aorta L_a and capillary l_c lengths are both proportional to L. Since the nutrient fluid is conserved, the volume rate of flow is given by

$$\dot{Q} = \pi R_a^2 v_a = N_c \pi r_c^2 v_c,$$

where R_a and v_a are the aorta radius and fluid velocity and N_c, r_c and v_c are capillary number, radius and fluid velocity, respectively. It is natural to write that $\dot{Q} \propto B$. In the basal regime, v_a and v_c are size-invariant. Then we obtain that $R_a \propto B^{1/2}$ and $N_c r_c^2 \propto B$. Making the *extra assumption* that r_c is invariant, it follows that the capillary density $\rho_c = N_c/M$ is $\rho_c \propto B/M \propto 1/\tau$. For $d = 3$ we have the following results: (a) BMR-1 - $R_a \propto M^{3/8}$ and $\rho_c \propto M^{-1/4}$; (b) BMR-2 - $R_a \propto M^{1/3}$ and $\rho_c \propto M^{-1/3}$. In the maximum regime, we have that $v_a = a_0\tau$, implying that now v_a depends on the mass. Note that this suggests new empirical studies. Then, we obtain that $R_a^2 \propto B/\tau$. Now v_c is not necessarily invariant, as in the basal case. Since $\rho_c r_c^2 v_c \propto 1/\tau$ we obtain that $\rho_c \propto 1/\tau$ if we make the *extra assumptions* that both v_c and r_c are independent of body mass. The results are: (c) MMR-1 - $R_a \propto M^{5/14}$ and $\rho_c \propto M^{-1/7}$; (d) MMR-2 - $R_a \propto M^{1/4}$ and $\rho_c \propto M^{-1/4}$.

Let us compare our predictions for $d = 3$ with experimental data (see table 1). The values predicted in the MMR-2 context are far from the experimental ones. On the other hand, the MMR-1 scenario describes very well the MMR data. The exponent $b = 6/7 \approx 0.86$, larger than the basal value, is in very good agreement with data. Muscular capillary density of mammals is linked to MMR, instead of BMR, because only during exercise are all the muscular

capillaries perfused. The capillary density scales as $\rho_c = N_c/M \propto M^{-1/7}$, in good agreement with the average experimental value for various regions of muscle [3, 19]. Since $\tau \propto M^{1/7}$, frequencies must scale as $F \propto \tau^{-1} \propto M^f$ with $f = -1/7 \approx -0.14$. This value, smaller than the basal one, is also in good agreement with data for heart and respiration rates in strenuous exercise. The results for the capillary invariant radius r_c and $l_c \propto M^{2/7}$, agree roughly with the theoretical-empirical estimation of Dawson [20]. ($l_c \propto M^{0.21}$ and $r_c \propto M^{0.08}$).

Since the length of aorta cannot change from basal to maximum metabolic regimes, it should scale as the prediction of MMR scaling. The predicted exponent 0.29 agrees well with data. The aorta radius could in principle follow the two scalings because of the elasticity of the aorta and the dynamical body adaptations of mammals in the transition BMR - MMR. The experimental value 0.36 has however a better agreement with the MMR-1 value 5/14.

Consider now the predictions of BMR scaling. Recently the empirical values of the BMR exponent of mammals were reanalyzed using diverse procedures [8, 9, 22] that furnished values in the interval between $b_2 = 2/3$ and $b_1 = 3/4$, which are the predicted values of BMR-2 and BMR-1, respectively. Heart and respiration rates are close to the BMR-1 value $-1/4$ and other biological variables [5] have values close to multiples of $1/4$. On the other hand, empirical data near multiples of $1/3$ are also reported [10]. Therefore, the two scenarios are possible. This last possibility explains why b is greater in large versus small mammals data: diffusion and ballistic transports can be equally important in small organisms (BMR-2) but not in large ones, where ballistic transport is crucial (BMR-1). Finally, let us emphasize that we make a verifiable empirical prediction: the aorta blood velocity (v_a), which is scaling-invariant in BMR conditions, grows with mass in the exercise-induced MMR condition ($v_a \propto \tau$). The related exponent, which is predict to have the value -0.14 in the MMR-1 scenario, was never measured. Its empirical determination can be an experimental test of the importance of the transportation processes for the allometric scaling of metabolism.

Acknowledgments

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