

# Revising the distributive networks models of West, Brown and Enquist (1997) and Banavar, Maritan and Rinaldo (1999): Metabolic inequity of living tissues provides clues for the observed allometric scaling rules

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## Abstract

Basic assumptions of two distributive network models designed to explain the  $3/4$  power scaling between metabolic rate and body mass are re-analysed. It is shown that these models could have consistently accounted for the observed scaling patterns if and only if body mass  $M$  had scaled as  $L^4$ , where  $L$  is body length, in the model of Banavar et al. (1999, *Nature* 399, 130–132), or if spatial volume  $V_F$  occupied by the distributive network had scaled as  $M^{3/4}$  in the model of West et al. (1997, *Science* 276, 122–126). Lack of agreement between these predictions and observational evidence invalidates both models rendering them mathematically controversial. It is further shown that consideration of distributive networks can nevertheless yield realistic values of scaling exponents under the major assumption that living organisms are designed so as to keep the mass-specific metabolic rate of important functional tissues in the vicinity of a size-independent optimum value. Mass-specific metabolic rate of subsidiary mechanical tissues can be small and vary with body mass. Different patterns of spatial distribution of metabolically active biomass within the organism result in different patterns of allometric scaling. From the available evidence the presumable optimum value of mass-specific metabolic rate of living matter is estimated to be in the vicinity of  $1\text{--}10 \text{ W kg}^{-1}$ .

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## 1. Introduction

Recent years witnessed a conspicuous rise in the interest to theoretically account for the observed scaling exponents  $\alpha$  between whole-body metabolic rate  $B$  and body mass  $M$ ,  $B \propto M^\alpha$ . In particular, two models have been developed, hereafter WBE97 (West et al., 1997) and BMR99 (Banavar et al., 1999), both intending to explain  $\alpha = 3/4$  by considering properties of the distributive networks delivering nutrients to all parts of the organism.

In this paper it is shown that the WBE97 and BMR99 models make use of essentially the same, although differently formulated, basic assumptions. Those very assumptions yielding  $B \propto M^{3/4}$  unambiguously constrain the dependence between body mass  $M$  and  $L_d$  as  $M \propto L_d^4$ , where  $L_d$  is a linear size of the distributive network. It is demonstrated that this prediction contradicts the empirical evidence. This fact indicates that the observed scaling patterns must have a different explanation.

It is proposed that living matter can be characterized by an optimum mass-specific metabolic rate  $b_{\text{opt}}$ , which is independent of body size and varies little from taxon to taxon (Makarieva et al., 2005). Living bodies are

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organized so as to keep the mass-specific metabolic rate of the most important tissues and controlling organs near the optimum. Mass-specific metabolic rates of subsidiary mechanical tissues can be much lower than  $b_{opt}$  and vary with body size. The various scaling patterns for whole-body metabolism follow from different patterns of spatial distribution of the metabolically active mass with  $b = b_{opt}$  within the organism. The developed approach also explains why the relative mass of visceral organs in mammals should decrease with body mass.

**2. Distributive network of Banavar, Maritan and Rinaldo (1999)**

*2.1. Model's structure*

Everywhere below the following notations are used:  $M$ , mass of the organism;  $L$ , linear size of the spatial volume occupied by the organism;  $V_d$ , volume of the distributive network (the cumulative volume of all vessels). Note that in compact bodies of animals  $M \propto L^3$ , but this is not true in the general case.

In the BMR99 model, living body of linear size  $L$  is divided into the so-called transfer sites, i.e. sites where the distributive network terminates. If the mean distance between neighboring sites is  $l_s$ , the total number of sites in the  $D$ -dimensional body is

$$N_s = (L/l_s)^D. \tag{1}$$

BMR99 state that the number of transfer sites scales as  $L^D$ ,  $N_s \propto L^D$ . By doing so, BMR99 implicitly assume that  $l_s$  is size invariant,  $l_s \propto L^0$ . Size invariance of  $l_s \ll L$  agrees with the postulate of BMR99 that “the fundamental processes of nutrient transfer at the microscopic level are independent of organism size”.

BMR99 define whole-body metabolic rate  $B$  as “the total amount of nutrients being delivered to the sites per unit time” and state that  $B$  “scales as the number of sites or as  $L^D$ ”,  $B \propto L^D$ . In a subsequent paper developing this model (Banavar et al., 2002) it is explicitly stated that “ $D = 3$  in most cases of interest” and that body mass and volume scale as  $L^D$ ,  $M \propto L^D$ . From this one unambiguously concludes that  $B$  scales proportionally to  $M$ ,  $B \propto M$  and  $\alpha = 1$ . A similar conclusion has been reached by Kozłowski and Konarzewski (2004) with respect to the WBE97 model.

Hence, the intended derivation of  $\alpha = 3/4$  by BMR99 contains a mathematical error. BMR99 state that as far as  $B \propto L^D$  and the total volume of an efficient distributive network  $V_d$  scales as  $L^{D+1}$ , one has  $B \propto V_d^{D/(D+1)}$ , which gives  $B \propto V_d^{3/4}$  for  $D = 3$ . Demanding additionally that  $V_d$  is proportional to body mass,  $V_d \propto M$ , BMR99 conclude that  $B \propto M^{3/4}$ .

Here lies the mathematical contradiction, because the three relationships needed to obtain  $B \propto N_s \propto M$ ,

$$\left. \begin{matrix} N_s \propto L^3 \\ V_d \propto L^4 \\ V_d \propto M \end{matrix} \right\} \Rightarrow M \propto L^4, \tag{2}$$

constrain body mass as  $M \propto L^4$ . This cannot be reconciled with the observed  $M \propto L^3$  for animals. That is, either  $V_d \propto L^4$  and  $M \propto L^3$ , but then  $V_d$  and  $M$  cannot scale isometrically. Or  $V_d$  and  $M$  scale isometrically, but then, as far as  $M \propto L^3$ ,  $V_d$  should also scale as  $L^3$ , and the main result of the BMR99 model, the  $V_d \propto L^4$  scaling, is invalidated.

Notably, this major problem with the BMR99 model was first noted by Dodds et al. (2001), but, despite citing the work of Dodds et al. (2001), Banavar et al. (2002) did not respond to this fundamental criticism. Dodds et al. (2001) suggested that the mathematical controversy in the BMR99 model could be possibly avoided by abandoning size invariance of the distance between transfer sites  $l_s$ . However, as shown above, the size-invariance of  $l_s$  is a basic assumption of the BMR99 model, from which all scaling relationships, including  $B \propto M^{3/4}$ , originate. If  $l_s$  were assumed to scale as  $M^x$ , where  $x \neq 0$ , then, at  $D = 3$ ,  $N_s$  would scale as  $N_s \propto M^{1-3x}$ , see Eq. (1). As long as  $V_d$  scales as  $N_s L$  (the distributive network brings nutrients to  $N_s$  sites distributed along a linear scale  $L$ ), this would give  $V_d \propto M^{4/3-3x}$ . Demanding additionally that  $V_d \propto M$ , see Eq. (2), one obtains an equation on  $x$ ,  $4/3 - 3x = 1$ , which solves at  $x = 1/9$ . From this, applying the assumption of BMR99 that  $B \propto N_s$  and taking into account that  $N_s \propto M^{1-3x} = M^{2/3}$ , one obtains  $B \propto M^{2/3}$ . Hence, if the BMR99 model were saved from the mathematical controversy captured in Eq. (2), it would yield the conventional  $2/3$  scaling instead of the  $3/4$  scaling. Note that this result,  $B \propto M^{2/3} \propto L^2$ , is directly obtained from  $V_d \propto L N_s$ ,  $V_d \propto L^3$  and  $B \propto N_s$ . In the framework of the BMR99 model it corresponds to the case when transfer sites are spread not within the body volume ( $N_s \propto L^3$ ), but over the body surface ( $N_s \propto L^2$ ).

*2.2. Implicit assumptions of the BMR99 model*

The  $V_d \propto L^{D+1}$  scaling is proved by BMR99 to be valid for all efficient distributive networks irrespective of their geometry. This scaling can be therefore illustrated on the simplest example of tubes with cross-section  $r_c^2$  going from a single point (central source of nutrients) along a body of length  $L$  to  $N_s$  transfer sites distributed evenly within the  $D$ -dimensional volume  $L^D$ . The mean length of such tubes is close to  $L$  (with a geometric coefficient of the order of unity).

One has therefore:

$$V_d = Lr_c^2 N_s = Lr_c^2 (L/l_s)^D. \quad (3)$$

BMR99 state that  $V_d \propto L^{D+1}$ . This means that  $r_c^2$ , the cross-section of terminate vessels, is implicitly assumed to be size invariant,  $r_c \propto L^0$ , otherwise the scaling exponent in the  $V_d - L$  scaling would be different from  $D + 1$ .

Flow of nutrients to each transfer site is

$$F_s = \rho r_c^2 u_c. \quad (4)$$

Here  $\rho$  is the volume density of nutrients and  $u_c$  is the flow rate in the terminate vessel. Whole-body metabolic rate thus becomes  $B = F_s N_s = \rho r_c^2 u_c (L/l_s)^3$ . BMR99 state that  $B \propto N_s \propto L^D$ . This means that BMR99 implicitly assume that  $F_s \propto L^0$ . Coupling this assumption with the above-discussed size invariance of  $r_c$  and the explicit assumption made by Banavar et al. (2002) that  $\rho \propto L^0$ , size invariance of  $F_s$  is equivalent to size invariance of  $u_c$ , the rate of nutrient flow in the terminal vessels.

Summing up, BMR99 implicitly assume that  $\rho$ ,  $u_c$ ,  $l_s$ , and  $r_c$  are independent of  $L$ . As shown above, within these assumptions one cannot obtain  $B \propto M^{3/4}$ . On the other hand, if these parameters were free to depend on  $L$ , the  $B-M$  scaling would be fully determined by the character of these dependencies. However, the BMR99 model would then be unable to predict the resulting relationship between  $B$  and  $M$ , as far as this model does not constrain these parameters.

It should also be noted that the reasoning of Banavar et al. (2002) that the BMR99 model allows one to derive  $\alpha = 3/4$  irrespective of how the distance  $l_s$  between neighboring sites scales with  $L$  is incorrect (see also the previous section). It is based on the statement (equivalent to Eq. (3) of Banavar et al. (2002)) that  $V_d \propto (L/l_s)B$ . Banavar et al. (2002) state that BMR99 proved  $V_d \propto (L/l_s)B$  as a mathematical theorem. However, it is clear from Eq. (3), which describes a simple network conforming to the requirements of the BMR99 model, that the relationship  $V_d \propto (L/l_s)B$  is only true for a size-independent  $l_s$ , i.e.  $V_d \propto L N_s \propto LB$ , which is the main result obtained by BMR99. Inserting  $l_s$  into the latter proportionality is misleading. On the other hand, for a size-independent  $l_s$  the relationship  $V_d \propto LB$  is only true if  $B \propto L^3 \propto M$ , see Eq. (2). Hence, the derivation of  $B \propto M^{3/4}$  from  $V_d \propto (L/l_s)B$  by Banavar et al. (2002) contains the same mathematical controversy as described by Eq. (2).

### 3. Distributive network of West, Brown and Enquist (1997)

#### 3.1. Model structure

The distributive network of The WBE97 model is a space-filling fractal. At each  $k$ th hierarchical level from

aorta to capillaries (a total of  $N$  levels) each vessel branches into  $n$  smaller vessels, each smaller vessel being  $\gamma$  times shorter,  $l_{k+1}/l_k \equiv \gamma$ , and  $\beta$  times narrower,  $r_{k+1}/r_k \equiv \beta$ , than the parent vessel. At each  $k$ th level there are  $N_k$  vessels. WBE97 explicitly assume that  $u_c$  and  $r_c$ , see Eq. (4), are size invariant and implicitly assume the size invariance of  $\rho$ . Instead of fixing the linear size between transfer sites  $l_s$ , WBE97 demand that the length  $l_c$  of the terminal vessels (capillaries in the cardiovascular terminology) is independent of body size.

In space-filling fractals, vessels of all hierarchical levels are evenly distributed within one and the same volume  $V_F$ . Mathematically, this can be expressed as the condition of volume preservation (West et al., 1997):

$$l_k^3 N_k = l_{k+1}^3 N_{k+1} = l_0^3 \propto V_F. \quad (5)$$

Here  $l_0$  is the length of the zeroth level (aorta,  $N_0 = 1$ ) in the distributive network. The value of  $l_0$  characterizes the linear size of the total volume  $V_F$  occupied by the fractal network. Note that the volume  $V_d$  of the distributive network (i.e. blood volume in animals, volume of wood in plants) is not equal to the spatial volume  $V_F$  occupied by the network.

Writing Eq. (5) for the terminal vessels (capillaries),  $l_c^3 N_c = l_0^3$ , one obtains

$$N_c = (l_0/l_c)^3. \quad (6)$$

The number  $N_c$  of capillaries, terminal vessels, is equivalent to the number of transfer sites in the BMR99 model,  $N_c \equiv N_s$ . The length of terminal vessels,  $l_c$ , is size invariant as well as the distance  $l_s$  between transfer sites in the BMR99 model. Thus, Eq. (6) in WBE97 is mathematically equivalent to Eq. (1) in BMR99 if one uses aorta length  $l_0$  instead of linear size of the organism  $L$  and length of terminal vessels  $l_c$  instead of distance between transfer sites  $l_s$ .

WBE97 assume that whole-body metabolism is proportional to the number of capillaries,  $B \propto N_c$ . BMR99 make an equivalent assumption that  $B$  is proportional to the total number of transfer sites. (As pointed out by the anonymous referee, this assumption is not self-evident for basal metabolism, because in the resting state some capillaries can be quiescent; this assumption should be therefore more plausible for maximum metabolism. Here it is worthy noting that in mammals maximum whole-body metabolic rate does not scale as  $M^{3/4}$  (Weibel et al., 2004).)

WBE97 calculate the total blood volume  $V_d$  in their distributive network as

$$V_d \approx \frac{\pi r_c^2 l_c (\gamma \beta^2)^{-N}}{1 - n \gamma \beta^2} \quad (7)$$

(Eq. (4) of West et al. (1997), see also Makarieva et al. (2005) for a short derivation of the  $V_d$  scaling from the assumptions of the WBE97 model). As far as  $\gamma \equiv l_{k+1}/l_k$ , one has  $l_0 = \gamma^{-N} l_c$ . For the simplest case of

area-preserving fractals ( $\beta = \gamma^{3/2}$ ) Eq. (7) yields  $V_d \propto l_0^4$ . Additionally demanding that volume  $V_d$  be proportional to body mass  $M$ , the WBE97 model must satisfy a system similar to Eq. (2):

$$\left. \begin{aligned} N_s &\propto l_0^3 \\ V_d &\propto l_0^4 \\ V_d &\propto M \end{aligned} \right\} \Rightarrow M \propto l_0^4. \tag{8}$$

In other words, the 3/4 scaling in the WBE97 model is retrieved from the fact that the number of capillaries  $N_c \propto l_0^3$  scales as 3/4 power of the network volume  $V_d \propto l_0^4$ . If  $V_d$  is put proportional to  $M$ , this yields  $B \propto M^{3/4}$ . If  $V_d$  scaled differently with  $M$ , a different scaling of metabolism with body mass would result.

The three relations in the left-hand side of Eq. (8) yield  $B \propto N_s \propto M^{3/4}$  if only  $M \propto l_0^4$ . The BMR99 model starts from  $l_0 = L$  by stating that the linear size of the distributive network coincides with that of the body size. This immediately results in the contradiction with the observed  $M \propto L^3$  in animals. The WBE97 model avoids this mathematical pitfall by introducing  $l_0$  as an independent scale, which does not have to be proportional to body length  $L$ . However, this creates another, logical contradiction; because the distributive network designed to “supply all parts of the organism” (West et al., 1997) must stretch along the whole body of length  $L$  rather than to be confined to its own spatial scale  $l_0$ . As shown below, the available evidence for animals disproves the possibility of two linear scales  $l_0$  and  $L$  existing in the organism.

### 3.2. WBE97 as applied to animals

First, direct observations show that aorta length in mammals scales approximately as one-third power of body mass, that is,  $M \propto l_0^3$  instead of  $M \propto l_0^4$  demanded by the WBE97 model. Peters (1983) cites the work of

Günter and Léon de la Barra (1966) who found that total aorta length scales as  $M^{0.32}$ . Calder (1984) lists several more scaling exponents for lengths of various aorta parts based on the study of Holt et al. (1981) of mammals ranging 38,000-fold in body mass (from mice to cows). In agreement with the previous work, Holt et al. (1981) found that total aorta length (from the valves to bifurcation) scales as  $M^{0.32}$  and equals about 16 cm for a 1-kg mammal. Length of aorta from the valves to the left and right renal arteries scales as  $M^{0.33}$  and  $M^{0.34}$ , respectively, and is about 11 cm for a 1-kg mammal. Length of aorta from the valves to brachiocephalic artery scales as  $M^{0.28}$ , while length of aorta between intercostal arteries scales as  $M^{0.36}$ . Note that the latter two lengths pertain to very small segments of aorta (1 cm and 0.6 cm, respectively, for a 1-kg mammal) not relevant to the characteristic linear space scale of aorta. Summarized in Table 1, the available estimates are in disagreement with the 1/4 scaling for aorta length expected from the WBE97 model. West et al. (1997) make an explicit prediction for aorta length as scaling as  $M^{1/4}$ ; the data reported by Peters (1983) and Calder (1984) are used by West et al. (1997) in their Table 1 aimed to test the agreement between observations and the various scaling predictions of the WBE97 model. However, in Table 1 of West et al. (1997) there is no mentioning of aorta length; the evidence given in Peters (1983) and Calder (1984) on the scaling of aorta length as  $M^{1/3}$ , unsupportive of the WBE97 model, was ignored by West et al. (1997). The observed scaling of aorta length as  $M^{1/3}$  is consistent with the physically and biologically sound expectation that the distributive network should stretch along the whole body of length  $L$ ,  $l_0 \propto L \propto M^{1/3}$ . However, putting  $l_0 \propto L$  turns Eq. (8) into Eq. (2) of the BMR99 model and leads to the contradiction between the demanded  $M \propto L^4$  and the observed  $M \propto L^3$  in animals.

Table 1  
Scaling of aorta length and capillary density in mammals;  $\delta$  is the observed scaling exponent in the relationship  $y \propto M^\delta$ , where  $y$  is a variable from the first column of the table,  $M$  is body mass

Variable	$\delta$	Reference
Total aorta length	0.32	Günter and Léon de la Barra (1966)
Total aorta length	0.32	Holt et al. (1981)
Aorta length, from valves to left renal artery	0.33	Holt et al. (1981)
Aorta length, from valves to right renal artery	0.34	Holt et al. (1981)
Aorta length, from valves to brachiocephalic artery	0.28	Holt et al. (1981)
Aorta length, between intercostal arteries	0.38	Holt et al. (1981)
Mean	0.33 versus WBE97 predicted 1/4 = 0.25	
Capillary density (CD, mm <sup>-2</sup> ) per unit cross-sectional area of tissue		
CD, semitendinosus	-0.138	Hoppeler et al. (1981)
CD, longissimus dorsi	-0.10	Hoppeler et al. (1981)
CD, vastus medialis	-0.097	Hoppeler et al. (1981)
CD, diaphragm	-0.045	Hoppeler et al. (1981)
Mean	-0.095 versus WBE97 predicted -1/6 = -0.17	

Second, if  $M$  scaled as  $l_0^4$ , then the spatial volume  $V_F \propto l_0^3$  occupied by the distributive network, see Eq. (5), should scale as  $V_F \propto M^{3/4}$  and increase in relative size with decreasing body size. This yields an unrealistic pattern that in small animals the volume occupied by the distributive network will be larger than body volume itself. To demonstrate this, it is necessary to express  $V_F$  in terms of measurable physiological variables. Assuming spherical geometry as WBE97 did,  $V_F$  can be written as  $V_F = 3/4\pi(l_0/2)^3 \approx 0.5l_0^3$ , see Eq. (5) and West et al. (1997).

Then one can write the ratio  $V_F/V$  (where  $V \propto M$  is body volume) as

$$\frac{V_F}{V} = 0.5 \frac{l_0^3}{V} = 0.5 \frac{N_c l_c^3}{V} \equiv 0.5 \frac{l_c^3}{V_s} \quad (9)$$

Here  $V_s \equiv l_s^3 \equiv V/N_c$  is the service volume, that is, the body volume served by one capillary. Capillary density  $\rho_S$  per unit cross-sectional area of tissue is then equal to  $\rho_S = 1/l_s^2$ . Another variable commonly measured in physiological studies is the capillary length density  $\rho_{ld}$ , which is the total length of capillaries in a given volume,  $\rho_{ld} = l_c/l_s^3$ . The dimensionless ratio of these variables is equal to  $\rho_{ld}/\rho_S = l_c/l_s$ . These relationships allow to rewrite Eq. (9) as

$$\frac{V_F}{V} \equiv 0.5 \frac{l_c^3}{l_s^3} = 0.5 \left( \frac{\rho_{ld}}{\rho_S} \right)^3 \quad (10)$$

Kanatous et al. (2001) report  $\rho_{ld}$  and  $\rho_S$  for a variety of muscle tissues in seal and dog with body masses of about 20 kg. The mean ratios  $\rho_{ld}/\rho_S$  are very similar in both species and constitute 1.31 and 1.28 in seal and dog, respectively. From Eq. (10) this gives  $V_F/V \approx 1$ . (Note that numerically this result is true to the accuracy of a geometric coefficient of the order of unity, which describes the particular geometric patterns of vessels orientation in tissues.) This remarkable result means that the distributive networks in these mammals are indeed filling the entire body volume.

Hence, if the volume  $V_F$  filled by the distributive network is already equal to body volume  $V$  in animals with  $M_1 = 20$  kg, but it scales as  $V^{3/4}$ , this leads to an unrealistic result that in a shrew with  $M_2 = 2$  g the volume occupied by the distributive network (that is, by the cardiovascular system) should be  $(M_1/M_2)^{1/4} = 10$  times larger than body volume itself! Note that this controversy inherent to the WBE97 model is different from the situation discussed by Banavar et al. (2002), who wrote that blood volume  $V_d$  cannot grow as  $M^{4/3}$ , because in such a case large animals should have largely consisted of blood only. In the case of the WBE97 model the controversy pertains not to the scaling of the blood volume  $V_d$ , but to the scaling of the spatial volume  $V_F$  embraced by the distributive network.

On the other hand, if one does not put  $V_F = V$  but introduces  $V_F$  as a formal variable changing freely with body size, there are no longer physical or biological grounds for Eq. (5), which therefore becomes just a mathematical axiom. If so, one is free to introduce any other similar condition at one's discretion: for example, to make the network fill surfaces ( $N_k l_k^2 = N_{k+1} l_{k+1}^2$ ) or lengths ( $N_k l_k = N_{k+1} l_{k+1}$ ) or, referring to the fashionable fourth dimension, equate the fourth powers of the corresponding variables ( $N_k l_k^4 = N_{k+1} l_{k+1}^4$ ). In other words, if no physical or biological interpretation in terms of measurable variables is assigned to  $V_F$ , then the only reason for choosing the particular form of Eq. (5) is the goal to obtain the 3/4 scaling, which makes the approach of WBE97 circular. At the same time, putting  $V = V_F$  in the WBE97 model leads to the biological controversy discussed above. It can be therefore concluded that the WBE97 model cannot account for the scaling exponents in animals remaining simultaneously within the domains of biological and physical plausibility and mathematical coherence.

Finally, the WBE97 model predicts that the service volume  $V_s \equiv V/N_c$ , i.e. volume served by one capillary, should scale as  $M^{1/4}$ . This means that capillary density per unit volume,  $\rho_V$ , should scale as  $\rho_V \propto M^{-1/4}$ ; capillary density per unit cross-sectional area of tissue,  $\rho_S$ , should scale as  $\rho_S \propto \rho_V^{2/3} \propto M^{-1/6}$ ; capillary density per unit body length,  $\rho_L$ , should scale as  $\rho_L \propto \rho_V^{1/3} \propto M^{-1/12}$ . (Note that  $\rho_L$ , the number of capillaries per unit body length, dimension  $[\text{mm}^{-1}]$ , is not equal to  $\rho_{ld}$ , dimension  $[\text{mm}^{-2}]$ , which is the total length of capillaries in a unit volume.) In their Table 1, West et al. (1997) list a variable called “density of capillaries”. It has a predicted exponent of  $-1/12 = -0.083$  and, hence, corresponds to  $\rho_L$ , capillary density per unit body length, dimension  $[\text{mm}^{-1}]$ . In the column “observed exponent” West et al. (1997) list a value of  $-0.095$ , which is relatively close to the predicted value  $-0.083$ .

Where does the value of  $-0.095$  originate from? Peters (1983), one of the three sources of the observed scaling exponents in Table 1 of West et al. (1997), lists four values of the scaling exponent for mammalian capillary density in different parts of the body based on the study of Hoppeler et al. (1981). These four values,  $-0.138$ ,  $-0.10$ ,  $-0.097$  and  $-0.045$ , Table 1, have a mean value of  $-0.095$ , suggesting that Peters (1983) is the source of data for the observed exponent for the variable called “capillary density” in Table 1 of West et al. (1997). However, the scaling exponents reported by Peters (1983) refer to capillary density per square millimetre (!), that is, to the capillary density per unit cross-sectional area of tissue,  $\rho_S$ , for which the WBE97 model prediction is  $-1/6 = -0.17$  and not  $-1/12 = -0.083$ , as for  $\rho_L$ , capillary density per unit body length, dimension  $[\text{mm}^{-1}]$ .

The difference between the observed exponents for  $\rho_S$  and the one predicted by the WBE97 model is in fact very substantial,  $-0.095$  versus  $-0.17$  on average and  $-0.05$  versus  $-0.17$  in the diaphragm, Table 1. Hence, the relative agreement between the columns “predicted exponent”,  $-0.083$ , and “observed exponent”,  $-0.095$ , in Table 1 of West et al. (1997) with respect to variable “capillary density” was obtained by West et al. (1997) by erroneously matching the predicted and observed exponents for different variables of different dimensions.

### 3.3. WBE97 model as applied to plants

Plants are not compact bodies and can therefore disobey the trouble-making  $M \propto L^3$  law. The distributive network in vascular plants constitutes most part of plant mass, so the condition  $V_d \propto M$  in plants is automatically guaranteed. Length  $l_0$  of the distributive system naturally corresponds to plant height  $H$ ,  $l_0 \approx H$ . The distributive network in plants terminates in  $N_c$  leaf petioles servicing  $N_c$  leaves. If leaf size is independent of plant height, then the total foliage mass  $M_L$  is proportional to  $N_c$ . Hence, if the WBE97 model correctly captures the plant architecture, it would predict  $M_L \propto M^{3/4}$  from Eq. (8). But this result would be only supportive of WBE97 approach if the second prediction is also empirically confirmed, namely that  $H \propto M^{1/4}$  (West et al., 1999), see Eq. (8). In the general case this is not so.

There are very few studies reporting an allometric relationship between plant height and total plant mass (or stem mass, which is an estimate of total plant mass in

woody plants). For example, among over 150 allometric relationships reported for Australian plants (Eamus et al., 2000; Keith et al., 2000) only 9 have the form  $M \propto H^\delta$  and all these pertain to shrubs with  $H < 5$  m. The majority of studies relate  $M$  to stem diameter  $D$ .

Such a situation is conditioned by the fact that there is no general dependence between plant mass and plant height. As is well known, growth of the plant in the vertical direction greatly decelerates as the plant approaches maturity, which is mathematically described by various sigmoid-like curves (Yuancai and Parresol, 2001). At sufficiently large size the dependence between plant mass and plant height vanishes altogether,  $H \propto M^0$  instead of  $H \propto M^{1/4}$  demanded by the WBE97 model.

For those species where height is a good predictor of plant mass, the available evidence rules out the  $M \propto H^4$  scaling demanded by the WBE97 model, Table 2. The observed mean scaling exponent for seventeen species  $\delta = 2.54 \pm 0.19$  (S.E.).

There is indirect evidence that  $M \propto H^4$  can be observed in some tree species. For the case of surface-preserving fractals the WBE97 model constrains the dependence between vessel radius  $r_k$  and vessel length  $l_k$  as  $r_k^2 \propto l_k^3$ . This follows from Eq. (5),  $l_{k+1}/l_k = N_{k+1}/N_k \equiv n$  and the condition of area preservation,  $r_k^2 = nr_{k+1}^2$ . When applied to the plant as a whole, this means that plant height  $H$  scales as two thirds of stem diameter  $D$ ,  $H \propto D^{2/3}$ . If the total plant mass is approximated by stem mass, a cylinder of height  $H$  and diameter  $D$ , one has  $M \propto D^2 H \propto H^4$ . The  $H \propto D^{2/3}$  scaling is indeed observed in a diversity of species (Prothero, 1999). (McMahon (1973) theoretically

Table 2

Parameters of the log-log regression of plant mass  $M$  on plant height  $H$  for several species of vascular plants,  $\log M = a + \delta \log H$ . T total plant mass, S stem mass, n number of plants studied. We retrieved the data of Walker et al. (1996) from the LTER web page, <http://luq.lternet.edu/data/lterdb52/metadata/lterdb52.htm>, and performed the OLS log-log regression

Species	Height range, m		$\delta$	$R^2$	$n$	$P$	Source
<i>Acacia harpophylla</i>	0.6–3.2	T	2.150	0.86	29		Eamus et al. (2000)
<i>Cassia nemophila</i>	0.6–2.0	S	3.297	0.88	19		Eamus et al. (2000)
<i>Dodonaea viscosa</i>	0.2–2.0	S	3.380	0.88	40		Eamus et al. (2000)
<i>Eremophila bowmanii</i>	0.2–1.8	S	3.522	0.94	18		Eamus et al. (2000)
<i>Eremophila mitchelli</i>	0.6–5.0	S	3.002	0.92	18		Eamus et al. (2000)
<i>Geijera parviflora</i>	0.6–4.5	S	3.442	0.96	9		Eamus et al. (2000)
<i>Myoporum deserti</i>	0.2–2.0	S	3.030	0.92	17		Eamus et al. (2000)
<i>Acacia aneura</i>	< 4.5	S	2.404	0.94	19		Keith et al. (2000)
<i>Eremophila sturtii</i>	< 4.5	S	3.246	0.94	22		Keith et al. (2000)
<i>Anthriscus sylvestris</i>	0.1–1.8	S	3.46	0.82		<0.05	Chang et al. (2004)
<i>Changium smyrnioides</i>	0.1–1.7	S	2.26	0.95		<0.01	Chang et al. (2004)
<i>Cecropia peltata</i>	0.05–5	T	1.39	0.85	11	<0.001	Walker et al. (1996)
<i>Heterotrichum cymosum</i>	0.03–2.2	T	1.54	0.89	12	<0.00001	Walker et al. (1996)
<i>Phytolacca rivinoides</i>	0.025–1.8	T	1.71	0.96	10	<0.00001	Walker et al. (1996)
<i>Piper hispidum</i>	0.3–1.5	T	2.23	0.93	7	<0.001	Walker et al. (1996)
<i>Piper treleaseanum</i>	0.035–1.1	T	1.50	0.95	10	<0.0001	Walker et al. (1996)
<i>Solanum torvum</i>	0.08–2.7	T	1.67	0.90	9	<0.0001	Walker et al. (1996)
Mean	$\delta = 2.54 \pm 0.19$ (S.E.) versus WBE97 predicted $\delta = 4.00$						

accounted for the  $H \propto D^{2/3}$  scaling based on the principle of elastic similarity.) For such species the WBE97 model could possibly account for the dependence between foliage mass and stem mass of the species.

However, this prediction of WBE97 model is not equivalent to  $B \propto M^{3/4}$ , which WBE97 claim to have explained. In order to obtain  $B \propto M^{3/4}$  from  $M_L \propto M^{3/4}$  it is necessary to postulate that  $B \propto M_L$  (Enquist and Niklas, 2002). This statement means, first, that plant metabolism  $B$  is equal to metabolism of leaves  $B_L$  and, second, that mass-specific metabolic rate of leaves  $b_L$  is independent of plant size. Then one has  $B \approx B_L = b_L M_L \propto M_L$  and  $B \propto M^{3/4}$  is obtained from  $M_L \propto M^{3/4}$ . However, if one postulated  $b_L \propto M^x$ ,  $x \neq 0$ , instead of  $b_L = \text{const} \propto M^0$ , one would obtain from the WBE97 predicted  $M_L \propto M^{3/4}$  that  $B \propto M^{3/4-x}$ . The postulate  $b_L = \text{const}$  corresponds to one of the basic WBE97 assumptions that  $u_c$ , the rate of flow in terminal vessels, is independent of body size. Namely, this assumption, for which WBE97 do not give any justification, fully determines the character of the dependence between metabolism and body size.

The predicative power of the WBE97 model is therefore confined to evaluating the interdependence between the various parts of the distributive network WBE97 constructed, e.g. how the number of capillaries scales with the total network volume, etc. The extension of these predictions to the dependence between metabolic rate and body size is made axiomatically by postulating  $u_c = \text{const}$ . Neither  $\alpha = 3/4$  nor any other metabolic scaling is an inherent property of WBE97 distributive network.

#### 4. Optimal value of mass-specific metabolic rate in the living matter

BMR99 postulated that “the fundamental processes of nutrient transfer at the microscopic level are independent of organism size”. WBE97 implicitly used  $b_L = \text{const}$  to derive  $\alpha = 3/4$  for plants. As demonstrated below, these statements reflect a fundamental property of the living matter (Makarieva et al., 2005), which reconciles the controversies discussed in the previous sections and allows one to obtain realistic values of  $\alpha$  from consideration of distributive networks.

We propose that living organisms are designed so as to keep the energy supply of their most important tissues at a constant, size-independent rate  $b_{\text{opt}}$ , which constitutes the optimal value of mass-specific metabolic rate  $b$  of the living matter (Gorshkov et al., 2000). Metabolic requirements  $b_i$  of subsidiary tissues (e.g. mechanical ones) can be much lower than  $b_{\text{opt}}$  and vary freely with body size. The living body can be therefore approximately divided into metabolically active  $M_a$  and metabolically inactive  $M_i$  parts,  $M = M_a + M_i$ . We

also assume that  $b_a = b_{\text{opt}} \gg b_i$  to satisfy

$$B = b_a M_a + b_i M_i \approx b_{\text{opt}} M_a = B_a. \quad (11)$$

The proposed universality of  $b_{\text{opt}}$  in the metabolically active tissues imparts a profound biological meaning to the central assumption of the BMR99 and WBE97 models, namely to the size invariance of terminal microscopic processes delivering nutrients to living tissues. Indeed, size invariance of the flow rate  $F_s$ , Eq. (4), to each unit mass of the metabolically active tissues is dictated by the need to maintain mass-specific metabolic rate  $b$  near the optimum at all sizes,  $b = b_{\text{opt}} \propto F_s \propto L^0$ . If  $b$  were free to vary with body size, the size invariance of parameters determining  $F_s$ , in particular, the size invariance of flow rate  $u_c$  in the terminal vessels explicitly demanded by WBE97, would have no biological justification and remain just a formal mathematical assumption.

Our distributive network terminates in  $N_s$  vessels supplying  $N_s$  metabolically active service volumes  $V_s \equiv l_s^3$  at a size-independent rate  $F_s$ . Service volumes are independent of body size, so that at  $b_{\text{opt}} = \text{const}$  metabolic rate  $B$  is simply proportional to their number,  $B = b_{\text{opt}} M_a = b_{\text{opt}} V_s N_s$ . Note that the size-independent service volumes are assumed to be located in the metabolically active tissue only and their cumulative mass  $M_a$  is not equal to total body mass  $M$ .

We also make use of the fact that the volume of efficient distribution network  $V_d$  is proportional to the number  $N_s$  of service volumes multiplied by the mean distance  $L_d$  between the central source of nutrients and service volumes:

$$V_d \propto L_d N_s \quad (12)$$

##### 4.1. Metabolic scaling in mammals

As is well known, most part of basal metabolism in mammals is due to visceral organs, which constitute a small portion of total body mass, but feature very high mass-specific metabolic rates (Couture and Hulbert, 1995; Wang et al., 2001; Porter, 2001). For example, in humans the mass-specific metabolic rate of the brain is 23 and 33 times higher than that of skeletal muscles and skin, respectively (Aiello and Wheeler, 1995). The same situation is observed in other vertebrates, particularly fish (Nilsson, 1996; Oikawa and Itazawa, 2003).

To introduce a general framework for deriving scaling exponents in organisms containing metabolically active and metabolically inactive tissues, we put for simplicity  $M_a = M_{\text{brain}}$  and  $B \approx B_{\text{brain}} = b_{\text{opt}} M_{\text{brain}}$  ignoring the other visceral organs. The number of service volumes  $N_s$  in the brain is proportional to  $M_{\text{brain}}$ ,  $N_s \propto M_{\text{brain}}$ . The brain is concentrated in a particular part of the animal body, while the distributive network spreads over the entire body of length  $L$ . Hence, the mean distance  $L_d$  to

the brain from the central source of nutrients is proportional to  $L$ , so  $V_d \propto LM_{\text{brain}}$ . Demanding that  $V_d$  scales proportionally to body mass  $M$  ( $V_d$  has the meaning of blood volume in mammals), one obtains the following analogue of Eq. (2):

$$\left. \begin{array}{l} N_s \propto M_{\text{brain}} \\ V_d \propto LM_{\text{brain}} \\ V_d \propto M \end{array} \right\} \Rightarrow M_{\text{brain}} \propto M/L \quad (13)$$

Assuming  $M \propto L^3$ , one obtains from Eq. (13) that brain mass, as well as the whole-body metabolic rate, should scale as

$$B \propto M_{\text{brain}} \propto L^2 \propto M^{2/3}. \quad (14)$$

In a sample of 174 mammals for which *both* the basal metabolic rate  $B$  and brain size were known,  $M_{\text{brain}}$  scaled as  $M^{0.68}$ , while  $B$  scaled as  $M^{0.69}$  (McNab and Eisenberg, 1989).

This consideration adds support to the statement that the 2/3 scaling is the basic one in the organisms where, like in mammals and birds, basal metabolism is largely due to the functioning of compact visceral organs with slowly changing mass-specific metabolic rate (Dodds et al., 2001; Makarieva et al., 2003; White and Seymour, 2003). Recently, Savage et al. (2004) proposed that an  $\alpha$  value close to 3/4 in mammals can be obtained if one formally diminishes the relative portion of the smallest mammals in the considered database. Savage et al. did so by dividing the mammalian body mass range into equal logarithmic intervals and plotting the log-transformed mean  $B$  values against mean body mass values in each logarithmic intervals. This procedure moved the resulting scaling exponent towards the intended 3/4 value. However, such derived  $\alpha$  value is not comparable to  $\alpha$  values obtained in other taxa by the conventional procedure (i.e. log–log plotting all the species). According to Makarieva et al. (2003), reducing the share of small species and, consequently, elevating the share of large species in the log–log plot should lead to an increase in the scaling exponent from  $\alpha = 2/3$  towards unity. Small-sized species constitute the majority in virtually all taxa, see, for example May (1978). The procedure suggested by Savage et al. may change the scaling exponents in other taxa as well, including those which currently provide support for the 3/4 rule.

It should be emphasized that Eq. (13) provides a general framework for understanding the fact why the relative size of visceral organs should decrease with growing body size. The particular scaling exponents in Eqs. (13) and (14) come from the proposition that the animals try to keep the mass-specific metabolic rate of most important tissues close to the metabolic optimum  $b_{\text{opt}}$  irrespective of body size. Direct measurements indicate that this goal appears not to be 100% achievable. Mass-specific metabolic rate of visceral

organs does decrease with growing body mass, but this decrease is significantly slower than for the mean mass-specific metabolic rate of the whole body. While mean mass-specific metabolic rate in mammals decreases as  $b \propto M^{-\mu}$ , where  $\mu \approx 0.3$ , the characteristic  $\mu$  values for tissue slices from various organs are all much lower, 0.17 for liver, 0.07–0.11 for kidney cortex, 0.10 for lung and 0.07 for brain (Couture and Hulbert, 1995). Similar exponents are derived from in vivo estimates, all, with but one exception lower than 0.25 ( $\mu = 0.27$  for liver, 0.14 for brain, 0.12 for heart and 0.08 for kidney) (Wang et al., 2001). At the same time, mass of visceral organs grows more rapidly than  $M^{2/3}$  (Wang et al., 2001). It is interesting that these deviations from the proposed  $\mu = 0$  and the 2/3 scaling of organs' body mass appear to compensate each other. Metabolic rate  $B_a \approx B$  of all the visceral organs combined (liver, brain, kidneys and heart) scales as  $M^{0.69}$  (Wang et al., 2001), close to the  $M^{2/3}$  scaling expected for metabolically active tissues from Eq. (14).

#### 4.2. Scaling of plant architecture

Metabolically active mass of plants can be approximated by the mass of all leaves,  $M_a \approx M_L$ . Wood which constitutes most part of plant mass in trees is largely metabolically inactive (Makarieva et al., 2003). Plant stems incorporate vessels bringing nutrients to each leaf at a size-independent rate  $F_s$  dictated by the value of  $b_{\text{opt}}$ . Total flux via the stem is therefore proportional to the stem cross-section, which means that  $M_L$  should scale as squared stem diameter  $D^2$ ,  $M_L \propto D^{\alpha_L}$ ,  $\alpha_L = 2$ . This prediction is on average supported by observations, see below. However,  $\alpha_L$  can differ from species to species, as far as in many species only some part of the stem (sapwood) conducts nutrients. The particular value of  $\alpha_L$  will be determined by how the cross-sectional area of this part of the stem depends on stem diameter.

Solar energy, the source of energy for plants, is delivered per unit area. To make use of all incoming energy, leaves must form a continuous cover. That is, the neighbouring projections of leaf blades on the ground plane must be adjacent to each other. Maximum thickness  $d$  of this continuous cover (determined by leaf thickness and the degree of overlap between leaves) is dictated by the incoming flux  $F$  of solar energy,  $F \propto b_{\text{opt}}d$ , and is independent of plant size (Makarieva et al., 2003). This means that the maximum density of metabolically active mass per unit surface area is generally size independent.

If foliage of mass  $M_L \propto D^2$  is spread over the surface of a spherical crown at a size-independent density, then the mean distance  $L_d$  from the stem to the leaves is proportional to the sphere radius and, hence, to stem diameter  $D$ ,  $L_d \propto D$ . One thus obtains  $V_d \propto D^3$  from Eq. (12). The volume  $V_d$  of the distributive network

within the crown can be approximated by mass of branches,  $V_d \propto M_B$ . One has therefore  $M_B \propto D^3$  and  $M_L \propto M_B^{2/3}$ . Generally, if  $M_L \propto D^{\alpha_L}$  and  $M_B \propto D^{\alpha_B}$ , the above logic for a surface-spread foliage predicts  $\alpha_B = 3/2 \alpha_L$ .

If foliage of mass  $M_L \propto D^{\alpha_L}$  is spread within the volume of a spherical crown, then the radius of such a crown describing the mean length of the branches is proportional to  $D^{\alpha_L/3}$ ,  $L_d \propto D^{\alpha_L/3}$  and  $M_B \propto D^{4\alpha_L/3}$ , which gives  $M_B \propto D^{8/3}$  for  $\alpha_L = 2$ . Hence, for a volume-spread foliage  $\alpha_B = (4/3)\alpha_L$ . Note that particular form of the crown is not essential for deriving these relationships.

Finally, for tall trees with long branches and a narrow crown, the distance from the stem to the leaves is largely determined by crown height and can be independent of diameter  $D$ . In such cases the mass of branches and the mass of leaves will scale isometrically, proportionally to crown height, that is,  $\alpha_L = \alpha_B$ .

Using several sources of data (Eamus et al., 2000; Keith et al., 2000; Martin et al., 1998; Vann et al., 1998; Ross et al., 2001) 79 pairs of values  $\alpha_B, \alpha_L$  corresponding to 79 studies of over 50 tree species were collected, Fig. 1. Mean values were  $\alpha_L = 2.07 \pm 0.07$  (S.E.) and  $\alpha_B = 2.63 \pm 0.07$ . Linear regression of  $\alpha_B$  on  $\alpha_L$  revealed a great deal of correlation between the two parameters,  $R^2 = 0.38$ ,  $P < 0.00001$ . Three predicted lines,  $\alpha_B = 3/2 \alpha_L$ ,  $\alpha_B = 4/3 \alpha_L$  and  $\alpha_B = \alpha_L$ , dictated by different spatial distributions of leaves, embrace the majority of empirical points indicating that all the three patterns of

crown geometry are likely to be realized in the trees studied.

For comparison, regression of  $\alpha_L$  on  $\alpha_S$  and  $\alpha_T$ , where  $\alpha_S$  and  $\alpha_T$  describe how total mass  $M_T$  and stem mass  $M_S$  scale with stem diameter,  $M_T \propto D^{2T}$ ,  $M_S \propto D^{2S}$  based on the same data revealed much lower correlation ( $R^2 = 0.17$ ,  $P = 0.003$ ,  $n = 49$  for  $\alpha_T$  and  $R^2 = 0.08$ ,  $P = 0.02$ ,  $n = 66$  for  $\alpha_S$ ). This indicates that there is no direct functional dependence between the properties of the metabolically active mass of leaves and metabolically inactive wood, which principally serve different functions.

While the role of leaves is to drive the plant's energetics, the role of woody tissues mainly consists of overcoming the gravity and distributing leaves in the three-dimensional space above the ground, in order to ensure maximum light capture. Aquatic photosynthesizing organisms do not have this problem, as long as their specific density approximates that of water and the gravitational forcing is almost absent. Therefore, most part of primary productivity in the oceans is due to unicellular organisms lacking mechanical structures like wood.

These different roles played by mechanical metabolically inactive and functional metabolically active tissues in plants result in the observed lack of any general relationship between apparent plant size (largely determined by mechanical tissues) and its metabolism. Indeed, at a given latitude, primary productivity of forests and bogs or pastures that may differ by two or more orders of magnitude in characteristic plant height can be similar (Whittaker, 1975). This is the natural consequence of the major feature of plant energetics—solar energy, the source of energy for plants, is delivered per unit area, so whatever the plant height, its surface-specific metabolic rate is dictated by the size-independent value of the incoming flux of solar energy. It was argued (Makarieva et al., 2003) that there is no biological sense in relating the total metabolic rate of plants to total plant mass. However, by recognizing the metabolic inequity of leaves and wood it is possible to describe some simple properties of plant architecture from consideration of the distributive networks.

## 5. Discussion

The performed analysis of the internal logic of the WBE97 and BMR99 models revealed that these models could have explained the observed  $B \propto M^{3/4}$  if and only if body mass  $M$  had scaled as  $L^4$ , where  $L$  is body length, in the BMR99 model and if volume  $V_F$  occupied by the distributive network had scaled as  $M^{3/4}$  in the WBE97 model. These scaling relationships strongly

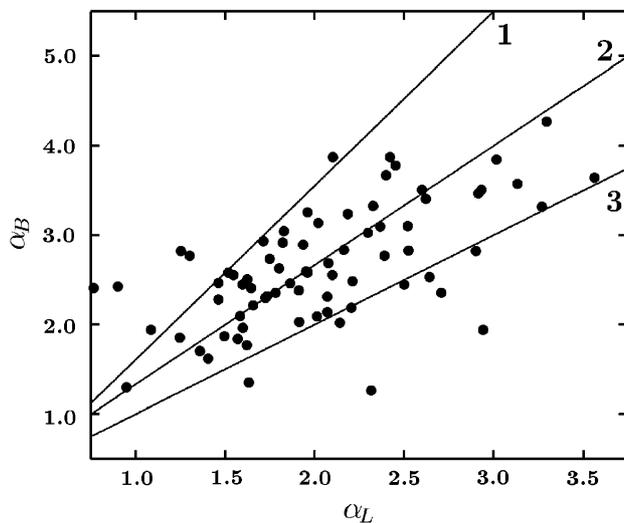


Fig. 1. Dependence of the scaling exponent  $\alpha_L$ ,  $M_L \propto D^{\alpha_L}$ , where  $M_L$  is foliage mass,  $D$  is stem diameter, on the scaling exponent  $\alpha_B$ ,  $M_B \propto D^{\alpha_B}$ , where  $M_B$  is mass of branches based on 79 studies of over 50 tree species (Eamus et al., 2000; Keith et al., 2000; Martin et al., 1998; Vann et al., 1998; Ross et al., 2001). Straight lines correspond to theoretically predicted relationships for 1:  $\alpha_B = 3/2 \alpha_L$  surface-spread foliage, 2:  $\alpha_B = 4/3 \alpha_L$  volume-spread foliage, 3:  $\alpha_L = \alpha_B$  foliage of tall narrow crowns.

contradict the empirical evidence in animals and are generally not true in plants.

We have argued that consideration of distributive networks can yield realistic values of  $\alpha$  if one accepts that living matter has a preferred optimum size-independent value of mass-specific metabolic rate  $b_{\text{opt}}$ . Living organisms strive to keep their mass-specific metabolic rate near the optimum, at least in the most important functional tissues, like, for example, plant leaves or animal brains. Mass-specific metabolic rate of subsidiary tissues can be either negligibly small (plant wood) or decrease rapidly with growing body size in animals.

What is the absolute value of the optimum mass-specific metabolic rate, and is it uniform for the entire domain of life? The above consideration suggests that within each animal taxon there is likely an optimum body size  $L_{\text{opt}}$ , for which all tissues of the organism feature one and the same optimum mass-specific metabolic rate. As is well known, the distribution of species numbers over body size within different taxa peaks in the region of smaller body sizes (May, 1978). It is natural to expect that these peaks correspond to the optimum body size  $M_{\text{opt}}$ , corresponding to the optimum mass-specific metabolic rate  $b_{\text{opt}}$ . Thus, comparison of mass-specific metabolic rates at those body sizes where species number peaks across different taxa would help reveal the degree of universality of  $b_{\text{opt}}$  in the living world.

Here we present an outline of such an analysis. For terrestrial arthropods  $B = 0.97 M^{0.856}$ , where  $B$  is in mW and  $M$  in grams (Lighton et al., 2001). The number of species in tropical beetles, the largest clade in terrestrial arthropods, peaks in the vicinity of  $L_{\text{opt}} \approx 2$  mm body length (Morse et al., 1988), which corresponds to a body mass of around  $M_{\text{opt}} \approx 2 \times 10^{-4}$  g (Stork and Blackburn, 1993) and  $b_{\text{opt}} \approx 3.3 \text{ W kg}^{-1}$ . Mammalian species numbers (excluding bats), on the other hand, peak at around  $M_{\text{opt}} \approx 300$  g (May, 1978). This corresponds to a metabolic rate of around  $b_{\text{opt}} \approx 4.0 \text{ W kg}^{-1}$  (Savage et al., 2004) and is close to the corresponding value for terrestrial arthropods.

It is interesting to compare these figures with mass-specific metabolic rates of mammalian brains and plant leaves. The mean value for five mammals with available in vivo estimates (Mink et al., 1981) is  $b_{\text{brain}} = b_{\text{opt}} \approx 10.5 \text{ W kg}^{-1}$  (range from 7.7 to 17.7  $\text{W kg}^{-1}$ ). For plants, Reich et al. (1998) collected data on mass-specific rates  $b$  of dark respiration of leaves in vascular plants from different geographic regions in the USA and Venezuela. Using species descriptions we matched the reported  $b$  values with characteristic maximum height  $H$  for 60 plant species studied by Reich et al. (1998). It appeared that at 25 °C leaves respire at a rate of 2.2  $\text{W kg}^{-1}$  in conifers (mean  $H \sim 35$  m), 5.8  $\text{W kg}^{-1}$  in broad-leaved shrubs and trees ( $H \sim 10$  m) and 12  $\text{W kg}^{-1}$  in forbs

Table 3

Characteristic values of preferred mass-specific metabolic rate  $b_{\text{opt}}$  in various groups of organisms,  $H$  is linear size of the organism (height in plants). See text for sources of the data

Taxon	$b_{\text{opt}}$ , $\text{W kg}^{-1}$	$T$ , °C	$H$ , m
Mammals, basal rate, 300 g	4.0	37	$\sim 0.1$
Mammalian brain, in vivo mean for 5 species	10.5	37	
Terrestrial arthropods, standard rate, 0.0002 g	3.3	25	0.002
Conifers, leaf dark respiration	2.2	25	35
Broad-leaved trees and shrubs, leaf dark respiration	5.8	25	11
Broad-leaved trees, leaf dark respiration	4.2	19–26	$\sim 0.1$
Forbs, leaf dark respiration	12	25	0.6

( $H \sim 0.6$  m). Seedlings of 12 Chilean rainforest trees (mean actual dry mass 11 g, mean maximum species height 27 m) respired at a rate of 4.2  $\text{W kg}^{-1}$  (Lusk and del Poso, 2002).

These estimates of  $b_{\text{opt}}$  are summarized in Table 3. Despite dramatic differences in apparent body size (over five orders of magnitude in linear size) and biology (arthropods, mammals, plants), the values of mass-specific metabolic rates displayed by most species are remarkably similar to each other. Additionally, comparison of standard mass-specific metabolic rates of eukaryotic unicells (205 observations for 50 species), insects (402 species) and mammals (626 species) has shown that in all the three taxa the majority of measured mass-specific metabolic rates are confined between 1 and 10  $\text{W kg}^{-1}$  (Makarieva et al., 2005).

Referring to the fact that in inter-specific comparisons the catalytic activity of proteins appears largely independent of preferred temperature, Clarke and Fraser (2004) wrote that life has overcome the tyranny of Boltzmann's law. Our results suggest that life has similarly worked to overcome the size-related metabolic constraints imposed by the laws of physics and chemistry. Living matter appears to be able to function at its own preferred rhythm, whose quantitative characteristics thus acquire fundamental biological importance and demand further exploration.

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