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Biochemical universality of living matter and its metabolic implications

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Summary

1. Recent discussions of metabolic scaling laws focus on the model of West, Brown & Enquist (WBE). The core assumptions of the WBE model are the size-invariance of terminal units at which energy is consumed by living matter and the size-invariance of the rate of energy supply to these units. Both assumptions are direct consequences of the biochemical universality of living matter. However, the second assumption contradicts the central prediction of the WBE model that mass-specific metabolic rate q should decrease with body mass with a scaling exponent $\mu = -1/4$, thus making the model logically inconsistent.

2. Examination of evidence interpreted by WBE and colleagues in favour of a universal $\mu = -1/4$ across 15 and more orders of magnitude range in body mass reveals that this value resulted from methodological errors in data assortment and analysis.

3. Instead, the available evidence is shown to be consistent with the existence of a size-independent mean value of mass-specific metabolic rate common to most taxa. Plotted together, q-values of non-growing unicells, insects and mammals in the basal state yield $\mu \approx 0$. Estimated field metabolic rates of bacteria and vertebrates are also size-independent.

4. Standard mass-specific metabolic rates of most unicells, insects and mammals studied are confined between 1 and 10 W kg^{-1}. Plant leaves respire at similar rates. This suggests the existence of a metabolic optimum for living matter. With growing body size and diminishing surface-to-volume ratio organisms have to change their physiology and perfect their distribution networks to keep their q in the vicinity of the optimum.

Key-words: Allometry, body mass, endotherms, mass-specific metabolic rate, unicells

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Introduction

The distribution network model of West, Brown & Enquist (1997) (WBE) has recently become a focus of intense discussions, as illustrated by two recent forums in *Functional Ecology* vol. 18(2) and *Ecology* vol. 87(7). Supporters of the WBE model credit it for being derived from the first principles of physics and biology and for the universality of application both within and across taxa. Claims were even put forward that this model can constitute a conceptual basis for much of ecology (Brown *et al*. 2004). In the view of such a perspective, no effort should be spared to scrutinize the fundamental assumptions of the model, its internal logic and the quality of supporting evidence. Here such an analysis is undertaken with a focus on the proposed universality of the 3/4 scaling law across the entire domain of life (Gillooly *et al*. 2001; Brown *et al*. 2004).

Fundamentals of the WBE model

The distribution network of the WBE model represents a hierarchy of branching vessels, which become shorter, narrower and more numerous from the central vessel (e.g. aorta) to the terminal ones (e.g. capillaries). The size of terminal vessels (their length l_c and radius r_c) is assumed to be independent of the body size of the organism. The particular rules for shortening, narrowing and multiplication of vessels adopted by WBE lead to the following relationships between the number N_c of terminal vessels, total volume V_b of all vessels of the network and length l_0 of the vessel of zeroth hierarchical level (in cardiovascular terminology N_c corresponds to the total number of capillaries, V_b to blood volume and l_0 to aorta length) (West *et al*. 1997), see Appendix:

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$$N_c \propto V_b^{3/4}. \quad \text{eqn 1a}$$

$$V_b \propto l_0^4. \quad \text{eqn 1b}$$

Scaling relationships 1a and 1b represent a static, architectural property of the network. They bear no relation to metabolic processes. Whether blood flows through it, or this network represents an artificial metallic construction assembled in accordance with the mathematical branching rules described by WBE, equations 1a and 1b will hold.

In order to convert equation 1a into the $^3/_4$ metabolic power law, it must be additionally assumed that the rate of nutrient flow in terminal vessels is independent of body size, u_c = constant. Total flow $u_c r_c^2 N_c$ of nutrients in the terminal vessels with size-independent radius r_c is proportional to whole-body metabolic rate Q, as far as in the steady state living cells consume as much nutrients as are delivered to them through the distribution network. The assumption u_c = constant allows one to write $Q \propto u_c r_c^2 N_c \propto N_c$. This relationship will yield $Q \propto V_b^{3/4} \propto M^{3/4}$ from equation 1a, but only for those organisms where $V_b \propto M$ and $M \propto l_0^4$, as far equations 1a and 1b must be satisfied simultaneously within the WBE model. The central (longest) vessel of the distributive network is expected to stretch along the whole body of length l, so that in compact bodies, such as those of animals, the length of the central vessel l_0 is expected to scale as $l_0 \propto l \propto M^{1/3}$ and not as $M^{1/4}$. Later we discuss the mathematical contradictions resulting from applying the WBE model to compact bodies in greater detail (see below).

Despite numerous claims that the WBE model is derived from the first principles in physics and biology, the assumption u_c = constant has never been given any justification, neither by WBE in 1997 nor in subsequent works by their colleagues. This assumption makes the WBE model self-contradictory. Indeed, the assumed size-invariance of terminal units of distribution networks is, ultimately, a natural consequence of the universal biochemical organization of living matter – at the cellular level, energy consumption in organisms of drastically different sizes is governed by similarly sized enzyme molecules performing universal functions (West, Brown & Enquist 1999). Given such size-independence of function, it would be natural if the living matter required, on a large-scale average, a size-independent rate of energy supply per unit mass for its maintenance. This proposition is the only first principle which could justify the constancy of the rate of energy supply to the size-invariant terminal units of the network, u_c = constant. However, it contradicts the central result of the WBE model, where mass-specific metabolic rate $q \equiv Q/M$ must decrease with growing body size as $q \propto M^{-1/4}$.

Size-invariance of mass-specific metabolic rate across taxa

Size-invariance of mass-specific metabolic rate q (W kg^{-1}) for living matter can be directly tested. Obviously, one cannot question the allometric dependencies well established *within* many taxa. Our analysis will concentrate on large-scale patterns spreading *across* taxa over large body size intervals. To this end, it is particularly instructive to compare metabolic rates of the largest and the smallest organisms, bacteria and vertebrates. We start with the analysis of the available data on metabolic rates estimated for organisms under natural environmental conditions. Although much more scanty and imprecise than laboratory data, such evidence has the unique value of an insight into how life works when undisturbed.

THE LARGEST *VS* THE SMALLEST: WHO RESPIRES FASTER?

Nagy, Girard & Brown (1999) provide a comprehensive compilation of field metabolic rates in birds, mammals and reptiles. In all three groups, the number of studied species peaks between 10 and 100 g. A 'typical' 30-g reptile, mammal and bird respire in the field at rates of 1·6, 22 and 41 W kg^{-1}, respectively. Using the data of Stork & Blackburn (1993) on mean biomass of tropical arthropods and the available estimates of the rate of herbivory and mean productivity of tropical forests, Makarieva, Gorshkov & Li (2004a) found the mean field metabolic rate of tropical arthropods to be in the same range, at around 30 W kg^{-1}. (Note that this value represents a grand mean for all tropical arthropods ranging in body mass from approximately 10^{-4} g to 10 g. While informative for large-scale comparisons as the one we are undertaking, it does not in any way exclude a metabolic scaling among different-sized arthropods.)

Turning to the smallest living beings, Clarholm & Rosswall (1980) studied short-term changes in bacterial biomass in the peat of a sub-Arctic mire and in the humus and mineral soil layers of a pine forest. Daily changes in bacterial numbers, bacterial biomass and size distributions were monitored for several days. Mean bacterial productivity per unit biomass for peat, humus and mineral horizon were 0·40, 0·19 and 0·15 kg kg^{-1} day^{-1}, respectively. Assuming energy content of living matter to be 7×10^6 J kg^{-1} (Peters 1983), mass-specific bacterial productivity p in energetic units constitutes 32, 15 and 12 W kg^{-1} for peat, humus and mineral layers, respectively. Long-term study of bacterial biomass revealed that these estimates correspond to the most favourable growth period of the year (Clarholm & Rosswall 1980), thus representing upper estimates of bacterial productivity. Mass-specific productivity p is related to mass-specific metabolic rate q via growth efficiency η, $\eta = p/(p+q)$ and $q = p(1-\eta)/\eta$. Assuming $\eta = 0{\cdot}4$ (Del Giorgio & Cole 1998) we obtain that soil bacteria respired on average at rates of 48, 22 and 18 W kg^{-1} in peat, humus and mineral layers, respectively.

Smith & Prairie (2004) report cell volumes and cell-specific respiration rates for natural bacterial samples from 20 Canadian lakes. Mean cell volume in the samples studied ranges from 0·023 to 0·049 μm^3 and

Table 1. Estimated field metabolic rates of bacteria and higher organisms. See text for data sources

Organism	Body mass (g)	Metabolic rate (W kg^{-1})
Prokaryotes		
Aquatic bacteria	$0{\cdot}03 \times 10^{-12}$	<66
Soil bacteria	$(0{\cdot}3–10) \times 10^{-12}$	18–48
Thiovulum sp.	3×10^{-9}	33
Thioploca auracae	2×10^{-8}	1·5
Eukaryotes		
Forest arthropods (mean for the indicated body size range)	0·0001–10	30
Reptiles	30	1·6
Mammals	30	22
Birds	30	41

averages 0·031 μm^3. Cell-specific respiration rates measured in terms of emitted CO_2 carbon range from 0·41 to 7·16 fg C $cell^{-1}$ h^{-1} (1 fg = 10^{-15} g). Mass-specific metabolic rates of bacteria from different lakes calculated from cell-specific rates and known cell masses at 37×10^3 J $(g\ C)^{-1}$ range from 10 to 250 W kg^{-1} with a mean of 66 W kg^{-1}. These estimates are obtained during exponential growth of natural bacterial cultures diluted in the ambient water. Under natural conditions only a small portion of bacterial cells is in the state of fastest possible growth and the proportion of actively respiring bacteria is never significantly above 20% (Berman *et al*. 2001; Stenstrøm, Svensson & Johansson 2001). This means that the obtained figures are likely to overestimate field bacterial metabolism by several times.

Large filamentous sulphur bacteria belonging to the genus *Thioploca* form dense visible mats on the continental shelf off the coast of Chile and Peru. These gammaproteobacteria (belonging to the same class as *Escherichia coli*) oxidize sulphide by reducing their internally stored nitrate. Otte *et al*. (1999) estimated *in vivo* fluxes of sulphide oxidation in these microbial mats to be 20·4 nmol min^{-1} (mg of bacterial protein)$^{-1}$. If sulphide is fully oxidized to sulphate and nitrate reduced to ammonium ($H_2O + H_2S + NO_3^- = SO_4^{2-} + NH_4^+$), oxidation of 1 mol of sulphide releases about 360 kJ energy (Kelly 1991). Protein accounts for about 50% of dry mass, dry mass in *Thioploca* constitutes 24% of wet mass (Otte *et al*. 1999). This allows the estimation of *in vivo* mass-specific metabolic rate in *Thioploca* as 15 W (kg of wet mass of the cytoplasm)$^{-1}$. About 90% of cell volume in this species is occupied by a large vacuole. When metabolic rate is calculated per total cell mass, it becomes 1·5 W kg^{-1}.

Another large sulphur bacterium, *Thiovulum* sp., forms veils above sea sediments at the oxygen–sulphide interface. Jørgensen & Revsbech (1983) studied chemical microgradients in a stationary veil formation and estimated cell-specific rate of oxygen consumption in this species as $2{\cdot}6 \times 10^{-16}$ mol of O_2 $cell^{-1}$ s^{-1}. Complete oxidation of sulphide to sulphate yields 358 kJ $(mol\ O_2)^{-1}$ (Kelly 1991). Cell mass of *Thiovulum* sp. approximates 3×10^{-9} g (Schulz & Jørgensen 2001). Mass-specific metabolic rate of this species is therefore around 33 W kg^{-1}.

Summarized in Table 1, the above estimates are consistent with the proposition that on a large scale mass-specific metabolic rate supporting living matter is size-independent, with vertebrates exhibiting the same range of values as bacteria, from about 2 to 40 W kg^{-1}. The lowest value for prokaryotes, 1·5 W kg^{-1} for *Thioploca*, comes from cells containing large metabolically inactive volumes. A similar effect can be responsible for low metabolic rates observed in ectothermic vertebrates. For example, fish white muscle making up a significant portion of body mass is practically metabolically inactive in resting fish as compared with visceral organs and brain: $q = 0{\cdot}1$ W kg^{-1} for white muscle *vs* $q = 9$ W kg^{-1} for kidney and 4 W kg^{-1} for brain in *Pagrus major* at 20 °C (Oikawa & Itazawa 2003), see also changing proportions of metabolically active and inactive tissues.

MASS-SPECIFIC *VS* WHOLE-BODY METABOLIC RATE

The proposition of large-scale size invariance of mass-specific metabolic rate q has never been widely discussed by biologists. To our knowledge, besides the work of Gorshkov (1981) which remained largely unknown to western scientists, there has been only one attempt to compare mass-specific metabolic rates across the whole domain of life (Robinson, Peters & Zimmermann 1983).

Otherwise, the few existing large-scale comparisons of metabolism across different taxa were made on the basis of whole-body metabolic rates Q (Hemmingsen 1960; Gillooly *et al*. 2001). In the latest compilation, Brown *et al*. (2004; Fig. 1) argued that when the available data on whole-body metabolic rates of organisms ranging over 15 orders of magnitude in body mass (from unicells to vertebrates) are plotted on one log–log plot, the overall slope α in the $Q \propto M^{\alpha}$ dependence is $\alpha = 0{\cdot}71$, close to the predicted $^3/_4$. This result contradicts the proposed large-scale size-invariance of mass-specific metabolic rate q, implying that the latter should decrease with body mass as $q = Q/M \propto M^{\mu}$, $\mu = \alpha - 1 = -0{\cdot}29 \approx -^1/_4$. This would correspond to differences in q of thousands of times between the smallest and largest organisms.

There are good grounds to consider this result as an artefact of the applied methodology of analysis and insufficient data quality. This statement is first illustrated on a model example and then by analysis of empirical evidence.

In the model example the considered 15 orders of magnitude range of body mass is divided into three model taxa, unicells U (the smallest), invertebrates I (intermediate in size) and endotherms E (the largest). Each taxon is assumed to cover five orders of magnitude range in body mass. It is assumed that under natural conditions and in comparable physiological states

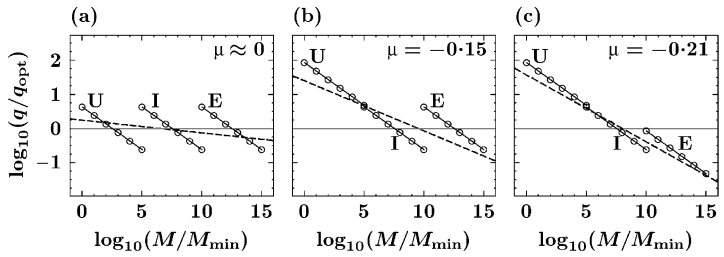

Fig. 1. Model examples for the origin of various scaling exponents μ for mass-specific metabolic rate q across 15 orders of magnitude range in body mass. Circles denote model points for which regressions are made. (a) The proposed size-invariance ($\mu \approx 0$) of mean $q = q_{opt}$ in unicells (U), invertebrates (I) and endotherms (E) when q is measured in comparable physiological states at natural temperatures. (b) $\mu = -0{\cdot}15$ results from comparison of standard metabolic rates of invertebrates and endotherms to growth metabolic rates of unicells assuming that the latter are about 20 times higher than 'standard' metabolic rates of unicells. (c) $\mu = -0{\cdot}21$ results from pattern (b) when basal metabolic rates of endotherms are corrected to 20 °C, a temperature incompatible with viability in most mammals.

mass-specific metabolic rates of organisms from each taxon vary around a uniform value q_{opt} as prescribed by scaling exponents μ_T established within each taxon. For the sake of definiteness, it is assumed that $\mu_T = -1/4$ and $\alpha_T = 3/4$ for all the three taxa. On the [log q] – [log M] plot such a situation corresponds to three straight parallel lines describing, for each taxon, how q varies with body mass within the interval from q_{min} to q_{max}, the latter also being size-invariant, Fig. 1(a). Within each taxon one model point per unitary logarithmic interval is plotted, to account for a similar binning procedure applied by Brown *et al*. (2004). As expected, linear regression of these model points reveals a size-invariant metabolic rate and yields an overall slope $\mu = -0{\cdot}037 \approx 0$. (The small negative value of μ results from the fact that the the smallest body mass corresponds to the highest q within the first taxon U, while the largest body mass corresponds to the lowest q within the third taxon E. Regression of the means $q = q_{opt}$ for each taxon gives $\mu = 0$ exactly.)

How can empirical evidence presumably consistent with $\mu \approx 0$ be modified to yield $\mu \approx -1/4$? In higher organisms one most commonly measures standard metabolic rate, which corresponds to adult, postabsorptive animals at rest. Accurate monitoring of physiological state in vertebrates is critically important for correct interpretation of the observed patterns (see e.g. Steffensen 2002). For microscopic unicellular organisms accurate definition of physiological state is much more problematic than in larger organisms. First, the notion of rest (immobility) remains largely undefined for unicells continuously moving in the aquatic media. Second, metabolic rates of unicells are most often measured in the state of growth in the presence of various substrates, as, for example, the data for about 50 bacterial species reported by Altman & Dittmer (1974). As is well known, metabolic rates of growing and actively moving animals can significantly exceed standard metabolic rate.

This means that comparing published values of metabolic rate for unicells with those of higher organisms introduces a systematic error and results in overestimation of metabolic rates in unicells. This is equivalent to elevating line U in Fig. 1(a) above the common mean. The magnitude of such elevation can be estimated comparing metabolic rates of growing and non-growing unicells. Analysis of published data on respiration of growing and non-growing bacteria (a total of 80 species) revealed that growth metabolic rates are on average about 20 times higher (A. Makarieva, V. Gorshkov & B.-L. Li, unpublished data). If line U is elevated by log 20 = 1·3 units above the common mean, Fig. 1(b), the resulting regression of binned values produces an overall slope of $\mu = -0{\cdot}15$, corresponding to $\alpha = 0{\cdot}85$.

Another factor influencing the overall slope is the temperature correction of metabolic rates. Most studied unicells, as well as invertebrate species, live in environments with temperatures never rising significantly above 20–30 °C and often being much lower. For these organisms the traditional correction of published values to 20 °C does not represent a significant departure from mean natural ambient temperatures. In contrast, body temperatures of most endotherms rarely deviate significantly from 37 to 40 °C. For endotherms, correction of metabolic rates to 20 °C performed by Brown *et al*. (2004) strongly contradicts organismal physiology. Namely this correction causes the overall slope μ to decrease even further. For the mean value of $E = 0{\cdot}67$ eV adopted by Brown *et al*. (2004) in their temperature term $\exp(-E/kT)$, correction from 39 to 20 °C corresponds to a fivefold decrease in metabolic rate. This moves line E in Fig. 1 by log 5 = 0·70 units downward, Fig. 1(c). The resulting overall slope becomes $\mu = -0{\cdot}21$, Fig. 1(c), approaching the −0·29 value obtained by Brown *et al*. (2004).

Importantly, the overall slope μ is largely insensitive to the particular values of slopes μ_T within each taxon, but is profoundly affected by the degree of vertical displacement of the three lines with respect to the common mean. For example, adopting $\mu_T = -0{\cdot}4$ for each taxon and performing the same displacements as in Fig. 1(c) (moving line U by 1·3 units up and line E by 0·7 units down) yielded an overall slope $\mu = -0{\cdot}23$, close to the –0·21 value obtained at $\mu_T = -0{\cdot}25$. The overall slope μ therefore contains no information about slopes within taxa and cannot be interpreted against or in favour of any particular value of μ_T. Heusner (1982) illustrated the same idea for whole-body metabolic rates. It can be similarly shown that the obtained result is not significantly affected by the particular value of body mass range within each taxon (five orders of magnitude in Fig. 1). For example, in a model example of three taxa ranging across seven orders of magnitude of body mass each (a situation resembling, e.g., insects and mammals) but partially overlapping to cover together a total range of 15 orders of magnitude in body mass, the adopted +1·3 and −0·7 displacements for the smallest and largest taxon yielded an overall slope $\mu = -0{\cdot}25$.

Finally, the third important factor (besides physiology and temperature corrections) influencing the overall slope obtained by Brown *et al*. (2004) is the data representativity. The overall slope of a regression line covering 15 orders of magnitude in body mass is particularly sensitive to the values corresponding to the smallest and largest body sizes. In the plot presented by Brown *et al*. (2004) the number of points for large organisms (with body mass exceeding 1 g) is well above 300, but there are only 30 points for unicells, with body masses ranging from approximately 10^{-11} to 10^{-4} g.

Analysis of these points reveals that they contain measurements of metabolic rates of *Bresslaua insidiatrix*, *Chaos chaos*, *Collozum inerme*, *Colpidium* sp. and *Paramecium* sp. (one measurement per each species); three species with multiple measurements of metabolic rate taken at different temperatures: *Amoeba chaos* (five measurements), *Anabaena variabilis* (seven), *Paramecium caudatum* (two); three measurements for 'unspecified microbes' and eight measurements for an organism laconically called 'yeast' (Gillooly *et al*. 2001).

It is instructive to compare this limited data set with the available extensive compilations of metabolic rates in unicells. Vladimirova & Zotin (1985) compiled an exhaustive data set on metabolic rates in Protozoa (554 observations for 108 species were collected from 320 published sources and corrected to 20 °C using empirically established temperature dependence $q/q_0 = 0{\cdot}166e^{0{\cdot}087t}$, where temperature t is in °C and q_0 is metabolic rate at 20 °C). Among these data, 205 observations for 50 species pertain to endogenous respiration, that is, respiration of non-growing cells in the absence of exogenous substrates. In Fig. 2(a) these 205 points are plotted together with the 626 points for mammalian basal mass-specific metabolic rates collected by Savage *et al*. (2004) and with the 402 points for standard mass-specific metabolic rates of insects at 25 °C from the updated data set of Addo-Bediako, Chown & Gaston (2002). The overall slope for the total of 1233 points is $\mu = -0{\cdot}026 \pm 0{\cdot}003$ (± 1 SE) ($R^2 = 0{\cdot}05$, $P < 10^{-5}$), remarkably close to the theoretical value obtained under the assumption that mass-specific metabolic rate of living matter is size-independent across taxa, cf. Figs 1(a) and 2(a).

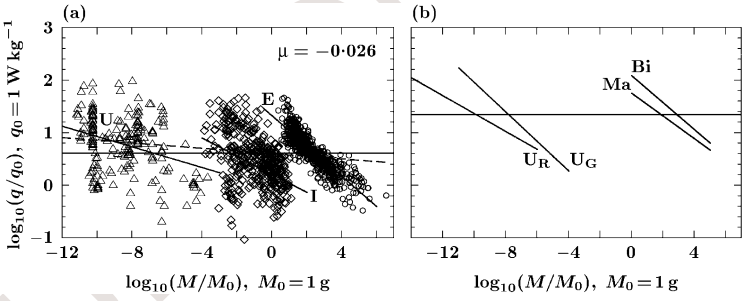

Fig. 2. Large-scale across-taxa comparison of mass-specific metabolic rates q. (a) Comparison of q-values in non-growing *Protozoa* at 20 °C (triangles, regression line U, $n = 205$) (Vladimirova & Zotin 1985), insects (standard metabolism at 25 °C) (diamonds, regression line I, $n = 402$) (Addo-Bediako, Chown & Gaston 2002), and endotherms (mammals) in the basal state (circles, regression line E, $n = 626$) (Savage *et al*. 2004). Dashed line: overall log–log regression $q \propto M^{-0{\cdot}026}$; horizontal line $q = 4{\cdot}1$ W kg^{-1} is the geometric mean for the entire sample ($n = 1233$). (b) Lines U_R, U_G, Bi, Ma describe the allometric relationships corresponding to the data of Robinson *et al*. (1983) and Gillooly *et al*. (2001) for unicells at 20 °C and to field metabolic rates of birds and mammals studied by Nagy *et al*. (1999), respectively. Horizontal line $q = 22$ W kg^{-1} is the geometric mean of three q-values corresponding to the midpoints of lines U_R, Bi and Ma.

Robinson *et al*. (1983), based on published data for 67 species of unicells with body masses ranging from 10^{-14} to 10^{-6} g and unspecified physiological state, established an equation relating mass-specific metabolic rates of unicells to body mass and temperature. When expressed in energetic units assuming 20 J per 1 ml O_2 this equation at 20 °C reads as $q_R = 23M^{-0{\cdot}17}$, where q is in W kg^{-1} and M is in 10^{-10} g. At the midpoint of the studied body size ranges, $M = 10^{-10}$ g, unicells respire at a rate of approximately 23 W kg^{-1}. Log–log regression of the temperature-corrected points of Gillooly *et al*. (2001) leads to equation $q_G = 91M^{-0{\cdot}28}$ ($R^2 = 0{\cdot}88$, $P < 10^{-5}$). For the data of Vladimirova & Zotin (1985) plotted in Fig. 2(a) the following equation was obtained: $q_{VZ} = 8{\cdot}4M^{-0{\cdot}10}$ ($R^2 = 0{\cdot}11$, $P < 10^{-5}$). For a characteristic body mass of 10^{-10} g one can see that q_{VZ} is 2·7 and 11 times lower than q_R and q_G, respectively. The predicted difference becomes even more pronounced for the smallest body sizes of $M \approx 10^{-14}$ g, where q_R and q_G exceed q_{VZ} by 5·2 and 57 times, respectively. This supports the statement that, on average, published metabolic rates overestimate 'standard' metabolic rates of the smallest organisms by several times and more, and cannot be meaningfully compared with standard metabolic rates of higher organisms, cf. Figs 1(b) and 2(b).

On the other hand, it is likely that published metabolic rates of unicells that are normally measured in solutions resembling natural media might approximate their field metabolic rates. If so, this would be an independent indication in favour of a size-invariant field mass-specific metabolic rate across taxa. The value of 23 W kg^{-1} obtained by Robinson *et al*. (1983) for 10^{-10} g unicells is similar to the observed mean q of free-ranging mammals (16 W kg^{-1}) and birds (28 W kg^{-1}) at the midpoint body mass $M = 100$ g of the body mass range from 1 to 10^4 g studied by Nagy *et al*. (1999), Fig. 2(b).

It should be also noted that both maximum and minimum q-values displayed by unicells and endotherms, Fig. 2, approximately coincide, supporting the existence of universal size-independent upper and lower limits to mass-specific metabolic rate (Gorshkov 1981; Robinson *et al*. 1983; Singer *et al*. 1993; Makarieva, Gorshkov & Li 2003). For example, 5% lowest q-values from Fig. 2(a) average 0·54 W kg^{-1} and 0·78 W kg^{-1} for unicells ($n = 10$) and mammals ($n = 31$), respectively. Also, within each body size interval there are likely to be taxa with mass-specific metabolic rates significantly lower or higher than mean q_{opt}. Among vertebrates, reptiles exhibit much lower field metabolic rates than birds and mammals (Nagy *et al*. 1999). Among invertebrates, such slowly metabolizing taxa can be exemplified by ticks, scorpions and centipedes (Lighton & Fielden 1995; Lighton *et al*. 2001; Klok, Mercer & Chown 2002), which, similarly to reptiles *vs* endotherms,

exhibit abnormally low metabolic rates as compared with other terrestrial arthropods of similar body size.

To conclude, the overall slope $\mu = -0{\cdot}29 \approx -1/4$ obtained by Brown *et al*. (2004) results from a systematic error, which was introduced by comparing different physiological states in the smallest and largest organisms and further aggravated by using a limited and non-representative data set for unicells. This means that the proposed interpretations of empirical evidence claimed to support the large-scale predictions of the WBE model stemming from $q \propto M^{-1/4}$ should similarly suffer from errors identifiable after detailed analysis. For example, ecosystem-level implications of the WBE model are based on the assumption of a size-independent rate of environmental resource supply, which is theoretically unjustified and empirically unsupported (Li, Gorshkov & Makarieva 2004). The ontogenetic growth model developed on the basis of the WBE model to describe growth of organisms as diverse in body size as zooplankton and mammals (West, Brown & Enquist 2001; Gillooly *et al*. 2002) violates the energy conservation law (Makarieva, Gorshkov & Li 2004b).

Metabolic scaling laws within taxa

CHANGING PROPORTIONS OF METABOLICALLY ACTIVE AND INACTIVE TISSUES

Large scale size-invariance of q is not equivalent to strict uniformity. Mass-specific metabolic rate of different tissues can be different reflecting different functions and biochemistry. Scaling relationships between Q and M observed within studied taxa can therefore be profoundly affected by the allometry of body composition, i.e. the changing proportion of tissues with lower and higher q in the living body of a given size (see e.g. Porter 2001; Oikawa & Itazawa 2003 and references therein). For example, brain and digestive organs account together for 41% of whole-body metabolic rate in 1 g fish *Pagrus major*. Mean mass-specific metabolic rate of these body parts is dozens of times higher than that of white muscles at rest. This rate changes only negligibly with body size, $q \propto M^{-0{\cdot}05}$ in brain and $q \propto M^{-0{\cdot}07}$ in digestive organs. The relative mass of digestive organs is approximately independent of body mass, while relative mass of brain decreases as rapidly as $M^{-0{\cdot}4}$ (Oikawa & Itazawa 2003). Given the possible variety of mass-specific metabolic rates, scaling exponents for mass-specific metabolic rate and relative mass of organs, it is not surprising that no universal scaling exponent for the resulting whole-body metabolic rate was established in the study of several hundred fish species (Bokma 2004).

These observations suggest that mass-specific metabolic rates are much more profoundly affected by the particular tissue biochemistry than by body size. For example, the observed 90-fold scope between mass-specific metabolic rates of fish kidney and white muscle that coexist within one and the same organism (Oikawa & Itazawa 2003) would correspond to a $M_2/M_1 = [q(M_1)/q(M_2)]^4 = 90^4 \approx 66 \times 10^6$-fold difference in body masses $M_2 > M_1$, if this scope pertained to whole-body-averaged mass-specific metabolic rates $q(M) \propto M^{\mu}$ of two organisms with body masses M_2 and M_1 at $\mu = -1/4$. Taxonomic differences in characteristic tissue biochemistry (mitochondrial efficiency, volume density, membrane permeability, etc.) may account for the existence of taxa with different metabolic rates within the same body mass interval, like ticks, scorpions and centipedes having significantly lower metabolic rates than the rest of arthropods or ectothermic vertebrates having significantly lower metabolic rates than similarly sized endotherms (Lighton *et al*. 2001; Klok *et al*. 2002; Darveau *et al*. 2002; Weibel *et al*. 2004).

THE WBE MODEL AS APPLIED TO PLANTS AND ANIMALS

Metabolic implications of changing proportions of tissues with high and low q can be illustrated with the following example. Suppose that the organism consists of two types of tissues, one of which is the main tissue driving all organismal energetics, while the second one is of subsidiary (mechanical, structural) nature and features a negligibly low metabolic rate. A straightforward example of such an organism is a tree, where metabolism is concentrated in leaves, while wood, which is largely metabolically inactive, makes up the distribution network and the bulk of total tree mass. While solving the task of supplying its metabolically active tissues at a size-independent rate, the tree must, with growing number of leaves, increase the number N_c of terminal vessels of its distribution network (petioles) bringing inorganic nutrients to each leaf. At size-independent mass-specific metabolic rate of leaves q_L = constant and constant leaf size, whole-body metabolic rate of the plant is proportional to total leaf mass M_L and to the number of leaf petioles:

$$Q \propto N_c \propto M_L. \qquad \text{eqn 2}$$

In woody plants total plant mass M can be approximated by mass of the wood, which also makes up the major part of the distributive network, $M \approx V_b\rho$, where ρ (kg m^{-3}) is wood density. If the distribution network of the tree conforms to the requirements of the WBE model, then the number N_c of leaf petioles should scale with total plant mass M approximated by mass of the wood in accordance with equation 1a, $N_c \propto M^{3/4}$. In such a case equation 2 yields $Q \propto M^{3/4}$. This pattern was discussed by WBE and colleagues when extending their model to plant energetics (see e.g. Enquist & Niklas 2002).

However, the described pattern could only be possible and not mathematically and biologically contradictory, if mass-specific metabolic rates of leaves q_L and wood q_W were, first, size-independent and, second, q_W were

much less than q_L and close to zero. Indeed, at $q_W \approx 0$ and q_L = constant >> q_W whole-body metabolic rate of the plant equals:

$$Q = q_L M_L + q_W M_W \approx q_L M_L \propto M_L. \quad \text{eqn 3}$$

If mass-specific metabolic rate q_L of leaves was not constant but changed with total plant body mass M as $M^{-1/4}$, i.e. following the prediction of the WBE model for mean mass-specific metabolic rate, then the whole-body metabolic rate $Q \approx q_L M_L$ would scale as $Q \approx q_L M_L \propto M^{-1/4} N_c \propto M^{-1/2}$, that is, contradicting the WBE model. In other words, the WBE model will only yield $Q \propto M^{3/4}$ for plants if q_L = constant, not if $q_L \propto M^{-1/4}$.

Stating that whole-body metabolic rate of plants scales as $M^{3/4}$, the WBE model is obscuring the fact that this scaling is only due to varying proportions of leaves and wood, tissues with different but size-independent mass-specific metabolic rates, $q_W \approx 0$, q_L = constant > 0. The resulting conclusion of the WBE model that mean mass-specific metabolic rate of plants decreases as $M^{-1/4}$ is therefore biologically invalid, because, according to the WBE model itself, there are no tissues in the plant where mass-specific metabolic rates would follow the predicted pattern.

Generally, there are strict limitations for the applicability of the WBE model for description of distribution networks. In the WBE model if the total volume V_b of all vessels is proportional to body mass M, $V_b \propto M$, then the length l_0 of the central vessel (aorta of mammals, trunk of trees) must scale as $^1/_4$ power of body mass (West *et al*. 1997), see equations 1a and 1b (remember that $Q \propto N_c$):

$$Q \propto M^{3/4}. \quad \text{eqn 4a}$$

$$l_0 \propto M^{1/4}. \quad \text{eqn 4b}$$

Simultaneous fulfilment of equations 4a and 4b is a mathematical requirement of the WBE model. If for some taxon equation 4b does not hold, while equation 4a does, this discrepancy is an unambiguous indication that the observed metabolic scaling cannot be explained by the WBE model but must have a different explanation. In mammals, for example, the aorta scales as $M^{0.32}$ (Peters 1983), $l_0 \propto M^{0.32}$. Putting this relationship into equation 1b and 1a of the WBE model predicts $Q \propto N_c \propto M^{0.96}$, instead of the intended $M^{3/4}$. Therefore all evidence in favour of the $^3/_4$ rule for mammals (Savage *et al*. 2004) invalidates the WBE model instead of supporting it.

The failure of the mammalian distribution network to conform to the WBE model is caused by the fact that mammals are compact bodies, whose characteristic linear body size l scales approximately as one-third power of body mass, $l \propto M^{1/3}$. The distribution network is designed to deliver nutrients to all parts of the body, which means that it must stretch along the body itself. In the result, length l_0 of the central vessel of the network, which characterizes the linear size of the network, must scale isometrically with body length l and, hence, proportionally to $M^{1/3}$ and not to $M^{1/4}$ as demanded by the WBE model. Therefore N_c cannot scale as $M^{3/4}$ and must be proportional to M, as was noted by Kozłowski & Konarzewski (2004) in their critique of the WBE model. Note that the same mathematical contradiction is inherent to the distributive network model of Banavar, Maritan & Rinaldo (1999) and Banavar *et al*. (2002), where it is stated that at the same time $l \propto M^{1/3}$, $V_b \propto l^4$ and $M \propto V_b^{3/4}$, three relationships that cannot be satisfied simultaneously.

In plants, which are not compact bodies, the relationship between linear size and mass is not so strictly defined. Height H of plant trunks, $l_0 \equiv H$, can, in principle, conform to equations 4a and 4b, i.e. $Q \propto H^3 \propto M^{3/4}$. However, such a relationship is not general. For example, it does not hold for small plants such as tree seedlings or herbs (Chen & Li 2003; Chang *et al*. 2004). Similarly, it should not hold for large mature trees, where growth of diameter D is accompanied by only negligible increase in height H (Niklas 2002). If mass of large trees can be roughly estimated as trunk mass, the latter being a function of the product D^2H, then growth of the tree in the horizontal direction at constant height corresponds to complete absence of dependence between M and H. Thus, rigorous testing of all involved parameters is needed to establish whether the WBE model is valid for description of a particular type of plant architecture. We note once again, however, that even for those plants where $H \propto M^{1/4}$, as demanded by the WBE model, the model will yield the $^3/_4$ scaling for whole-plant metabolic rate only if the mass-specific metabolic rate of leaves is size-independent.

Discussion

Clarke & Fraser (2004) suggested that life has overcome the 'tyranny' of Boltzman's law. They referred to the fact that the activity of key metabolic enzymes appears to be relatively temperature-independent when compared among organisms adapted to live at different ambient temperatures. Here we argued that life has similarly overcome the physical limitations imposed by body size. Despite dramatically changing surface-to-volume ratios from the smallest to the largest organisms of the biosphere, the mean absolute values of mass-specific metabolic rate they display are similar. In unicellular eukaryotes 54% of observations for endogenous metabolic rate (Fig. 2a) are confined between 1 and 10 W kg^{-1}. The same interval accounts for 80% of basal metabolic rates in mammals and for 67% of standard metabolic rates in insects, Fig. 2(a). [Note that in insects most data come from the studies of species with body mass confined between 10 mg and 10 g, Fig. 2a. In the meantime, in natural ecosystems the largest number of arthropod species corresponds to a smaller body size of around 0·1–1 mg (e.g. Gorshkov 1985; Morse, Stork & Lawton 1988; Ulrich 2004). For

example, species numbers of tropical beetles peak at a body length of about $l \approx 2$ mm (Morse *et al*. 1988), which corresponds to a body mass of about $M \approx 0{\cdot}2$ mg using the formula $M = 0{\cdot}0305L^{2{\cdot}62}$, where l is in mm and M in mg (Rogers, Hinds & Buschbom 1976). The existing data might therefore somewhat underestimate the mass-specific metabolic rate of the majority of insect species.] The reported values for dark respiration of plant leaves fall remarkably close to the q-values observed in animals. In an analysis of over 60 North American plants it was found that q is around 2 W (kg leaf mass)$^{-1}$ for dark respiration of leaves in conifers, 6 W (kg leaf mass)$^{-1}$ in broad-leaved trees and 12 W (kg leaf mass)$^{-1}$ in forbs (Reich *et al*. 1998; 25 °C). Seedlings of 12 Chilean trees respired at a mean rate of 4·2 W (kg leaf mass)$^{-1}$ (Lusk & del Poso 2002).

Narrowing intertaxa comparisons from mean mass-specific metabolic rates to mass-specific metabolic rates of particular tissues is likely to reveal even greater similarity. For example, while ectothermic vertebrates display several times lower metabolic rates than similarly sized endotherms at the same body temperature, ectothermic and endothermic brains, i.e. some of their metabolically most active and life-essential tissues, respire at very similar mass-specific rates (Nilsson 1996).

These observations suggest the existence of a metabolic optimum q_{opt} for the living matter, as far as the observed size-independent mean q-values are apparently favoured by natural selection across diverse kingdoms and body size intervals. Each organism consumes energy from the environment via some part of its body surface area S and spends this energy within its body mass M at a rate q per unit mass. The energy balance equation can be written as $fS = qM$ or

$$q = fS/M, \qquad \text{eqn 5}$$

where f is flux of energy per unit body surface area, W m^{-2}. With growing body size, from bacteria to largest vertebrates, the ratio S/M rapidly declines, while q, as we have shown, is on a large scale size-independent. From equation 5 this means that in order to become larger, the organisms must invent new ways of obtaining energy from the environment at an ever-increasing rate f growing proportionally to mean taxonomic ratio M/S, with an ultimate goal to keep their q in the vicinity of q_{opt}. In order to efficiently deliver the growing flux f of consumed energy and nutrients to all cells of the body, large organisms first had to invent and then continuously perfect their distribution networks.

Another factor profoundly impacting the closeness of organismal metabolic rate q to q_{opt} is the natural ambient temperature at which the organism lives and to which it is evolutionarily adapted. It is interesting to compare characteristic temperatures at which different-sized organisms flourish in natural ecosystems. Unicellular organisms such as bacteria, fungi and *Protozoa* are typical inhabitants of soils and water bodies. Owing to the high heat capacity of water and soil, the temperature of these media in closed ecosystems such as forests fluctuates much less than that of the air and is close to the mean daily temperature. For temperate Europe, for example, mean daily temperature is around 16 °C in July (data for the inland climate of England; Manley 1970) and even lower in the autumn, when most part of microbial decomposition in soil takes place. Larger organisms such as non-aquatic invertebrates enjoy a richer choice of temperatures, as far as air and ground surface temperatures change more significantly during the day than those of deeper soil and water. During the temperate summer, the magnitude of daily temperature changes is around 10 °C (Manley 1970). It is remarkable that in the temperate zone the majority of ectothermic animals, including arthropods and vertebrates, are active during the warmer parts of the day. The behavioural preference of arthropods for the higher environmental temperatures as compared with the smaller unicells can serve as one of possible mechanisms compensating the size-related drop of mass-specific metabolic rates in the larger ectothermic animals. The largest animals in the temperate zone, mammals, maintain the highest body temperatures due to endothermy. Not by chance, it has become common with researchers studying different groups of organisms to report metabolic rates at a reference temperature of 20 °C for unicells (the smallest body sizes) (Fenchel & Finlay 1983; Vladimirova & Zotin 1985); at a somewhat higher temperature of 25 °C for arthropods (the intermediate body sizes) (Lighton *et al*. 2001; Addo-Bediako *et al*. 2002) and at yet higher natural body temperatures $T \approx 37$–39 °C for endotherms (the largest body sizes) (Robinson *et al*. 1983). These temperatures, which were also employed by us in Fig. 2(a), were likely chosen by the researchers as most closely resembling the mean natural temperatures of the corresponding groups of organisms. Interestingly, there is no uniform temperature for reporting metabolic rates of vertebrate ectotherms. For example, Bennett & Dawson (1976) report three scaling relationships for Q in lizards, at 20, 30 and 37 °C. With respect to non-aquatic ectotherms it is remarkable that in the temperate zone their energy consumption at the population level (W m^{-2}) is negligible compared with that of mammals, but approaches or even exceeds the latter in the tropics, at temperatures around 35 °C (Bennett & Gorman 1979; Rodda *et al*. 2001), that is, at those temperatures where metabolic rates of endothermic and ectothermic brains approximately coincide (Nilsson 1996). Further studies are needed to test the prediction that closeness of mass-specific metabolic rates of particular tissues (or bodies on average) to q_{opt} is likely to be associated with energetic dominance of the corresponding species at the ecosystem level as compared with similarly sized species with their q significantly deviating from q_{opt}.

To sum up, we have argued that there are rational grounds for changing the current focus in metabolic research from scaling exponents, which, as we showed on the example of the WBE model, can be misleading

and obscuring the biological meaning of the observed patterns, to absolute values of mass-specific metabolic rates. Exposing the proposition of a size-independent q_{opt} to a careful scrutiny may lead to important insights into observed biological and ecological phenomena.

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Appendix: Derivation of equations 1a and 1b

The WBE distributive network is space-filling, which means that vessels of all hierarchical levels are evenly distributed within one and the same volume. The single ($N_0 = 1$) vessel of the zeroeth hierarchical level has length l_0 and occupies a spatial volume proportional to l_0^3. The same volume is occupied by all the N_k vessels of length l_k at any kth hierarchical level, including the terminal level with N_c vessels of a constant length l_c:

$$N_c l_c^3 = N_k l_k^3 = N_{k+1} l_{k+1}^3 = l_0^3. \qquad \text{eqn A1}$$

The second condition employed in the model is the condition of area preservation, that is, the sum of cross-sections r_k^2 of all the N_k vessels at the kth level is conserved across all levels:

$$N_c r_c^2 = N_k r_k^2 = N_{k+1} r_{k+1}^2 = r_0^2. \qquad \text{eqn A2}$$

From equations A1 and A2 we note that

$$\frac{N_k}{N_{k+1}} = \frac{r_k^2}{r_{k+1}^2} = \frac{l_k^3}{l_{k+1}^3} \qquad \text{eqn A3}$$

The second equality in equation A3 can be re-written as

$$\frac{r_k^2}{l_k^3} = \frac{r_{k+1}^2}{l_{k+1}^3} = \frac{r_0^2}{l_0^3} = \frac{r_c^2}{l_c^3} \qquad \text{eqn A4}$$

As far as the cross-section r_c^2 and length l_c of the terminal vessels are size-independent in the WBE model, the last equality in equation A4 means that cross-section r_0^2 of the central vessel scales as the cube of its length l_0:

$$r_0^2 = \frac{r_c^2}{l_c^3} l_0^3 \qquad \text{eqn A5}$$

From equation A2 we have $N_k r_k^2 = r_0^2$, so the total volume V_b of all vessels of the network is

$$V_b = \sum_{k=0}^{N} N_k r_k^2 l_k = r_0^2 \sum_{k=0}^{N} l_k = l_0^3 \frac{r_c^2}{l_c^3} \sum_{k=0}^{N} l_k \qquad \text{eqn A6}$$

Here summation is done over all the N hierarchical levels of the network. Note that the total volume V_b of all vessels of the network is not equal to the spatial volume occupied by the network. (For example, the cumulative volume of tree branches is not equal to the spatial volume embraced by the tree crown.)

In the WBE network the vessels shorten from one level to another, $l_k = \gamma l_{k-1} = \gamma^k l_0$, where $\gamma < 1$ is a constant. This allows equation A6 to be rewritten as

$$V_b = l_0^4 \frac{r_c^2}{l_c^3} \sum_{k=0}^{N} \gamma^k = l_0^4 \frac{r_c^2}{l_c^3} \frac{1-\gamma^{N+1}}{1-\gamma} \qquad \text{eqn A7}$$

As far as $\gamma^N = l_c/l_0$, the last ratio in eqn A7 is independent of l_0 at $l_0 >> l_c$, which is usually the case. This means that V_b is proportional to l_0^4, i.e. equation 1b follows. Equation 1a follows immediately from equation 1b by noting from equation A1 that $N_c = l_0^3/l_c^3$, where l_c is a constant. This means that $N_c \propto l_0^3 \propto V_b^{3/4}$.