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FORUM

West, Brown and Enquist's model of allometric scaling again: the same questions remain

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Introduction

Brown, West & Enquist (2005, BWE hereafter) rebut our critique (Kozłowski & Konarzewski 2004; K & K hereafter). In our paper we analysed the model published by West, Brown & Enquist (1997; WBE hereafter), showing the steps where either implicit assumptions were made or, in our opinion, the model was logically inconsistent. To our disappointment, BWE did not present a thorough mathematical analysis showing why we are wrong. However, in rebuttal of our critique they formulated some assumptions of their model more explicitly. This encouraged us to pinpoint once again the fundamental logical inconsistency of WBE's model.

Fundamental inconsistency in WBE's model

As stated by BWE, one of the crucial assumptions of the model is the size-invariance of the final branch (such as a capillary in the circulatory system). Size-invariance means the same length l_c, radius r_c and flow velocity irrespective of animal body size and, consequently, of its metabolic rate. They write additionally: 'WBE clearly state that only the characteristics of the capillaries themselves are assumed to be invariant. Nevertheless, K & K incorrectly interpreted this size-invariance to mean that each capillary must supply a constant volume of tissue.' Indeed, this is a critical point in the discussion. We shall show therefore that allowing the volume of tissue supplied by a capillary (the service volume) to increase with body size, although declared in the original WBE's paper (p. 125), is neither proved nor even allowed in the framework of their model.

At the beginning of the model presentation, WBE assume that because the fluid transports oxygen and nutrients for metabolism, blood flow through all capillaries should be proportional to the metabolic rate (B), and because metabolic rate is proportional to M^a (where M is body mass), the total number of capillaries must scale with exponent a. This assumption seems reasonable, but it is only an arbitrary assumption, because we can imagine that the concentration of nutrients and oxygen may decrease with size (see *Biological relevance of the model*). The exponent a is not specified at this point; it may equal 1 as well as 3/4 or any other value.

To prove that the ratio of vessel lengths between two consecutive branching levels, γ, is independent of the level and that $\gamma \approx n^{-1/3}$ (where n is the number of smaller branches at each node), WBE write in their original paper, 8 lines below equation 5: 'The network must branch so that a group of cells, referred to here as a "service volume", is supplied by each capillary. Because $r_k << l_k$ and the total number of branchings N is large, the volume supplied by the total network can be approximated by the sum of spheres whose diameters are that of a typical kth-level vessel, namely $4/3\pi(l_k/2)^3 N_k$' (r_k is vessel radius and l_k is the vessel length). Because this equation must also hold for the final branches, the body volume must equal $4/3\pi(l_c/2)^3 N_c$. This means that body volume is proportional to the number of capillaries N_c. Thus, service volume is not free to vary with size, at least in this part of the model. Here we arrive at the fundamental inconsistency of WBE's model: unless the metabolic rate exponent equals one, the number of capillaries must scale allometrically to satisfy the assumption that blood flow through all capillaries should be proportional to the metabolic rate, and at the same time the number of capillaries must scale isometrically to make it possible to compose a body with spheres having size-invariant radii, which is required for a space-filling fractal. Thus, WBE's claim that $\gamma_k \approx n^{-1/3} \approx \gamma$ is valid only for isometric scaling of metabolic rate. Because WBE write, 'This result for γ_k is a general property of all space-filling fractal systems that we consider', 3/4 scaling is proved neither in a rigid-pipe model nor in a pulsatile system model.

There are three solutions to the contradiction specified above: (i) the unlikely assumption that tissue density increases with body mass with the exponent $1 - a$; (ii) relaxing the assumption of capillary size-invariance; or (iii) assuming that $a = 1$. Only solution (ii) is supported on empirical grounds: according to Gehr *et al.* (1981) and Dawson (2001, 2003) both the radius and length of capillaries increase with body mass. Although in our critique we cited Dawson's (2001) paper invoking this experimental result challenging WBE's assumptions, BWE did not respond to this important point. Furthermore,

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Table 1. Slopes for BMR scaling against body mass in mammalian orders. Data were drawn from Savage *et al.* (2004). Only orders with 10 or more species are shown

Order	*N*	Slope (± SE)	Intercept (± SE)
Artiodactyla	20	0·753 (0·031)[ns]	–1·672 (0·146)[b]
Carnivora	52	0·784 (0·034)[ns]	–1·891 (0·127)[b]
Chiroptera	79	0·780 (0·028)[ns]	–1·824 (0·042)[b]
Dasyuromorpha	33	0·752 (0·015)[ns]	–1·882 (0·034)[b]
Primates	25	0·772 (0·046)[ns]	–1·918 (0·118)[b]
Diprotodontia	24	0·711 (0·015)[a]	–1·755 (0·057)
Insectivora	50	0·457 (0·040)[a]	–1·220 (0·067)
Lagomorpha	11	0·629 (0·044)[a]	–1·278 (0·137)
Rodentia	281	0·669 (0·013)[a]	–1·579 (0·027)
Xenarthra	15	0·658 (0·040)[a]	–1·751 (0·145)

[ns]Not significantly different from 0·75 at $P = 0·05$.
[a]Different from 0·75 at $P = 0·05$.
[b]Significant heterogeneity of intercepts (ANOVA, $P < 0·0001$).

several classic studies (e.g. Hoppeler *et al.* 1981) demonstrated that the proportion of capillaries opened to blood flow varies depending on the intensity of metabolism, which clearly violates the assumption of a constant number of smaller branches originating at each node of the fractal network. This is one of the major mechanisms underlying significantly steeper scaling of maximal metabolic rates, as compared to resting rates (Weibel *et al.* 2004) – yet another principal feature of metabolic scaling unexplained by WBE's model.

K & K's Table 1 shows the case with size-invariance of service volume following size-invariance of capillary length. This table was built to indicate that size-invariance of capillary length is impossible over a broad size range. The main point of our critique was that the assumption of capillary size-invariance cannot be defended if service volume is defined as a sphere with diameter equal to capillary length. In their Table 1, BWE propose an obvious solution with service volume scaling allometrically as $M^{1/4}$, but they ignore the fact that such scaling is impossible under size-invariance of capillary length. The only difference between BWE's Table 1 and K & K's Table 1 is body mass. In our table, body volume (assumed proportional to body mass) is calculated according to WBE's prescription, that is, as the sum of spheres with diameters equal to capillary length. Instead, BWE write in their table heading: 'The WBE model by contrast requires that blood volume, V_b, scales linearly with body mass, and this forces the number of capillaries, N_c, and hence metabolic rate to scale allometrically as $M^{3/4}$'. In fact, BWE calculate body mass from this relationship, and ignore WBE's assumption that the service volume for capillaries must sum up to the same volume (body volume) as for other levels of the system. This inconsistency is not apparent in their Table 1, because they do not check the necessary invariance of body volume at all vessel levels as K & K did.

BIOLOGICAL RELEVANCE OF THE MODEL

In their rebuttal, BWE did not address the major problems raised in our critique of the biological relevance of the model. Instead, they focused on minor points, and restated earlier claims without proving them. Below, we briefly comment on the major points of their rebuttal.

Oxygen or nutrients. Our question was not 'oxygen or nutrients', but whether the rate of their transport is proportional to blood volume. BWE acknowledge WBE's implicit assumption of proportionality. Since the size and number of erythrocytes (per blood unit volume) differ considerably between small and large animals (e.g. Kostelecka-Myrcha & Cholostiakow-Gromek 2001; Gregory 2002), we argue that WBE's assumption is an obvious oversimplification which ignores a plethora of physiological mechanisms.

Plant vascular system. To defend their statements, BWE refer to their 1999 paper (West, Brown & Enquist 1999) in which they present a model of the structure and allometry of plant vascular systems. However, the reference paper, like the original WBE model, is based on identical reasoning with regard to self-similar fractal characteristics of the modelled structures. Foundations of their 1999 model have the same flaws as those in WBE model. In particular, their 1999 model is based on the unproven assumption, borrowed from the WBE model, that the ratio of daughter to parent branch length (γ) scales as $n^{-1/3}$, where n is the number of daughter branches derived from a parent branch. Furthermore, as pointed out in our critique, the topography of the plant vascular system (both stem and leaf systems) is far from an ideal fractal structure – a point also recently made by McCulloh, Sperry & Adler (2003). BWE elected not to respond directly to this statement. Instead, they stated that they considered the leaf petiole as the invariant, terminal unit. Indeed, BWE rightly point out that by doing so they avoid 'complications of within-leaf network structure', but then where else should one look for the self-similar fractals envisaged by their model for plant vascular systems?

Insect tracheal system and vertebrate lungs. About the insect tracheal system BWE write: 'WBE do not present a model for insect tracheal system'. We do not understand therefore why WBE stressed in their abstract, 'More generally, the model predicts structural and functional properties of vertebrate cardiovascular and respiratory systems, plant vascular systems, insect tracheal tubes, and other distribution networks'. Later they refer to the tracheal system three times. Moreover, BWE write, 'if whole-organism metabolic rates of insects scale as $M^{3/4}$ … , then similar principles of fractal-like design should apply to the structure and function of the tracheal system'. This statement must be rejected on purely methodological grounds: different causes may lead to similar results. We understand that BWE admit, albeit not explicitly, that their model in its current form cannot explain metabolic rate scaling in insects. It also cannot be applied to amphibian or reptilian lungs,

because BWE write, 'neither WBE nor K & K have attempted to model explicitly the design of such "spongy lungs" in relation to the allometric scaling of reptilian and amphibian metabolic rates'. Thus the claim of the model's universality, so strongly stressed in the abstract of WBE's basic paper, and intensively exploited in their other papers, is still unjustified.

Scaling of metabolic rates. BWE repeatedly referred to the recent paper of their research group (Savage *et al.* 2004) in which they re-analysed scaling of mammalian BMR. Savage *et al.*'s analysis of the largest-ever data set yielded a regression slope of 0·712, which is significantly different from 3/4. Savage *et al.* pointed out that their failure to demonstrate 3/4 scaling was due to over-representation of the smallest species, with body masses < 1 kg. To remedy the problem of unbalanced representation, Savage *et al.* divided the data set into equally spaced logarithmic mass bins, each ranging 0·1 log mass units. They then averaged the data, which gave a single data point for each bin. Analysis of the thus-averaged data set yielded a slope not significantly different from 3/4, which according to Savage *et al.* supports WBE's model.

To check the validity of WBE's and Savage *et al.*'s claims, we analysed the 'binned' data-set given in Appendix 2 of Savage *et al.* (2004). We fitted a second order polynomial to their data and found that the square term is highly significant ($P < 0{\cdot}001$), clearly indicating non-linearity. Thus, the statistical validity of Savage *et al.*'s estimate of the slope of scaling must be rejected right from the start. It is important to note, however, that WBE claim that their model actually predicts slight curvilinearity of the scaling of mammalian metabolic rates located at the lower end of the body mass range (M < 1 kg). To test this prediction, we pinpointed the breakpoint of body mass in Savage *et al.*'s 'binned' data set by means of a computer program based on the modified method of Welch (1978). Briefly, the algorithm implemented in the program looks for the best fit of the two-segmented linear model, where the best fit is quantified as the minimum of least square means summed over the two segments. To avoid the discontinuity between fitted segments in each run the algorithm assigns two or more data points common to both segments. The fit is evaluated as successful when the breakpoint lies between the smallest and the largest of the common points. Otherwise the number of common points is increased and the fitting is repeated.

The best-fit model included just two common points, with the breakpoint abscissa equal to 3·63 log units, which is equivalent to body mass of 4·25 kg. The breakpoint is located almost in the middle of the log range of mammalian body masses (Fig. 1a). This is clearly at variance with BWE's claim that the curvilinearity of scaling of mammalian BMR is due to the undue effect of the smallest species. The slope of the left segment equals 0·67 (SE = 0·01) and, not surprisingly, differs very significantly from 3/4 ($F_{1,30} = 25{\cdot}7$, $P < 0{\cdot}001$). The slope of the right segment equals 0·85, and due

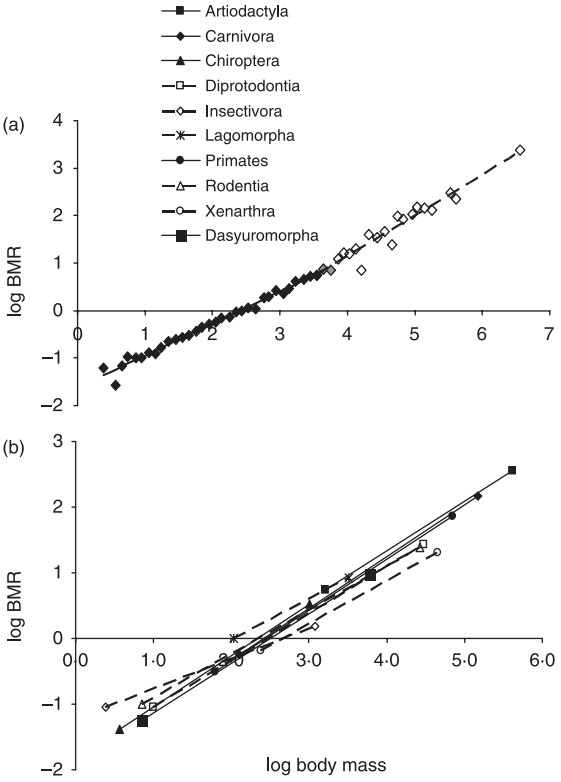

Fig. 1. (a) Two-segmented straight line model fitted to 'binned' data on mammalian log body mass *vs* log BMR reported in Appendix 2 of Savage *et al.* (2004). The breakpoint with abscissa equal to 3·63 log units is located between the two closed symbols. (b) Allometries of BMR in 10 mammalian orders, computed from the species averages assembled by Savage *et al.* (2004). Solid lines represent orders whose slopes of scaling do not differ from 3/4, whereas broken lines depict orders with scaling significantly different from 3/4. Slopes are given in Table 1.

to a high SE = 0·05 does not significantly differ from 3/4 ($F_{1,18} = 3{\cdot}85$, $P = 0{\cdot}065$).

It is also important to note that according to WBE's model the undue effect of the smallest species is not due to their over-representation, but rather to their higher than average metabolic rates. Notably, WBE's model 'predicts small deviations from quarter-power scaling ($a = 3/4$), observed in the smallest mammals'. To test the consistency of this prediction with real data, we computed the slopes of scaling of mammalian basal metabolic rates for species averages analysed by Savage *et al.* For mammals with $M < 0{\cdot}25$ kg, $M < 0{\cdot}5$ kg and $M < 1{\cdot}0$ kg the slopes were 0·66 (SE = 0·01), 0·64 (SE = 0·01) and 0·66 (SE = 0·02), respectively, and differed very significantly from 3/4 ($P < 0{\cdot}001$ in all cases). Clearly, higher than average metabolic rates of the smallest mammals (e.g. insectivores) are not achieved by steeper scaling, as predicted by WBE's model, but by higher elevation of the allometry (Fig. 1b). Thus, mammalian data falsify rather than support WBE's model.

The impressive data set compiled by Savage *et al.* (2004) allowed us to demonstrate the taxonomic heterogeneity of slopes of scaling of mammalian BMR. Systematic differences in the metabolic rates of different taxa are totally ignored in WBE's model, whereas an ample

body of evidence shows that it is highly informative and biologically meaningful (e.g. Lovegrove 2000; McNab 2002; Kozłowski, Konarzewski & Gawelczyk 2003b; Lovegrove & Haines 2004). In Table 1 we report the slopes calculated from Savage *et al.*'s data for mammalian orders represented by at least 10 species. The slopes do differ significantly (ANCOVA, $F_{9,570} = 11{\cdot}61$, $P < 0{\cdot}0001$). In five out of 10 orders the slopes significantly differ from 0·75. Interestingly, the grand slope calculated for the species belonging to the remaining five orders, whose slopes do not differ from each other and 3/4, equalled 0·775 and differed significantly from 3/4 ($P < 0{\cdot}001$). Obviously such deviation from 3/4 was due to a significant effect of differences in the intercepts of parallel allometric lines (ANCOVA, $F_{4,209} = 6{\cdot}69$, $P < 0{\cdot}0001$) – a point already made by Heusner (1982). This exposes another weakness of WBE's reasoning, which disregarded not only the taxonomic differences in slopes but also in the intercepts of allometries of taxonomic groups.

What's next? The future of metabolic rate scaling

In our view, it is high time to thoroughly and critically re-examine both the mathematical and biological foundations of WBE's model. We urge the authors to present the model once more, carefully and at greater length. The model should be presented step by step, without invoking future results (e.g. 'where a will later be determined to be 3/4' in WBE below equation 2, or 'As shown below, one can also prove from the energy minimization principle that $V_b \propto M$' below equation 4). Such excursions to future results make it difficult to distinguish results from assumptions, or to detect circular reasoning. Most importantly, in their model the authors should address the seeming inconsistency in treating the service volume of a capillary as increasing with body size and at the same time constant as a volume of a sphere with size-invariant diameter equal to capillary length. They should also address Dodds, Rothman & Weitz's (2001) criticism of their pulsatile system model. We anticipate that such a critical re-examination of WBE's model will force the authors to abandon the assumption of body-size invariant capillary size, which is clearly at variance with experimental data.

Our criticism by no means undermines the very significance of the problem of biological scaling. Contrary to BWE's claim, we did offer an alternative analytical model of scaling. We argue that the scaling exponents of interspecific allometries of metabolic rates are by-products of evolutionary diversification of genome size within narrow taxonomic groups, which underlines the participation of cell size and cell number in body size optimization (Kozłowski & Weiner 1997; Kozłowski, Konarzewski & Gawelczyk 2003a; Kozłowski *et al.* 2003b). Unlike WBE, we believe that only a pluralistic approach to scaling, founded on life-history theory, can explain the patterns of metabolic rate scaling. We do not expect a single, universal exponent, but a distribution with a mode somewhere between 2/3 and 3/4. The diversity of physiological solutions to the same problems is a striking feature of nature. Should the scaling of metabolic rates be an exception to this rule?

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References

Brown, J.H., West, G.B. & Enquist, B.J. (2005) Yes, West, Brown and Enquist's model of allometric scaling is both mathematically correct and biologically relevant. *Functional Ecology* **19**, 735–738.

Dawson, T.H. (2001) Similitude in the cardiovascular system of mammals. *Journal of Experimental Biology* **204**, 395–407.

Dawson, T.H. (2003) Scaling laws for capillary vessels of mammals at rest and in exercise. *Proceedings of the Royal Society of London B* **270**, 755–763.

Dodds, P.S., Rothman, D.H. & Weitz, J.S. (2001) Re-examination of the '3/4-law' of metabolism. *Journal of Theoretical Biology* **209**, 9–27.

Gehr, P., Mwangi, D.K., Ammann, A., Maloig, G.M.D., Taylor, C.R. & Weibel, E.R. (1981) Design of the mammalian respiratory system. V. Scaling morphometric pulmonary diffusing capacity to body mass: wild and domestic mammals. *Respiration Physiology* **44**, 61–86.

Gregory, T.R. (2002) A bird's-eye view on C-value enigma: genome size, cell size and metabolic rate in the class *Aves*. *Evolution* **56**, 121–130.

Heusner, A.A. (1982) Energy metabolism and body size. I. Is the 0·75 mass exponent of Kleiber's equation a statistical artifact? *Respiration Physiology* **48**, 1–12.

Hoppeler, H., Mathieu, O., Weibel, E.R., Krauer, R., Lindstedt, S.L. & Taylor, C.R. (1981) Design of the mammalian respiratory system. VIII. Capillaries in skeletal muscles. *Respiratory Physiology* **44**, 129–150.

Kostelecka-Myrcha, A. & Cholostiakow-Gromek, J. (2001) Body mass dependence of the haemoglobin content to surface area ratio of avian erythrocytes. *Acta Ornithologica* **36**, 123–128.

Kozłowski, J. & Konarzewski, M. (2004) Is West, Brown and Enquist's model of allometric scaling mathematically correct and biologically relevant? *Functional Ecology* **18**, 283–289.

Kozłowski, J., Konarzewski, M. & Gawelczyk, A.T. (2003a) Cell size as a link between noncoding DNA and metabolic rate scaling. *Proceedings of the National Academy of Sciences of the USA* **100**, 14080–14085.

Kozłowski, J., Konarzewski, M. & Gawelczyk, A.T. (2003b) Intraspecific body size optimisation produces interspecific allometries. *Macroecology: Concepts and Consequences* (eds T.M. Blackburn & K.J. Gaston), pp. 299–320. Blackwell Publishing, Oxford.

Kozłowski, J. & Weiner, J. (1997) Interspecific allometries are byproducts of body size optimization. *American Naturalist* **149**, 352–380.

Lovegrove, B.G. (2000) The zoogeography of mammalian basal metabolic rate. *American Naturalist* **156**, 201–219.

Lovegrove, B.G. & Haines, L. (2004) The evolution of placental mammal body sizes: evolutionary history, form, and function. *Oecologia* **138**, 13–27.

McCulloh, K.A., Sperry, J.S. & Adler, F.R. (2003) Water transport in plants obeys Murray's law. *Nature* **421**, 939–942.

McNab, B.K. (2002) *The Physiological Ecology of Vertebrates*. Cornell University Press, Ithaca.

Savage, V.M., Gillooly, J.F., Woodruff, W.H., West, G.B., Allen, A.P., Enquist, B.J. & Brown, J.H. (2004) The predominance of quarter-power scaling in biology. *Functional Ecology* **18**, 257–282.

Weibel, E.R., Bacigalupe, L.D., Schmitt, B. & Hoppeler, H. (2004) Allometric scaling of maximal metabolic rate in mammals: muscle aerobic capacity as determinant factor. *Respiratory Physiology and Neurobiology* **140**, 115–132.

Welch, W.R. (1978) Fitting segmented straight lines. *A Mini-Manual for Use with the Minitab Computing System*. Technical Report No. 2, The University of Wisconsin, Madison.

West, G.B., Brown, J.H. & Enquist, B.J. (1997) A general model for the origin of allometric scaling laws in biology. *Science* **276**, 122–126.

West, G.B., Brown, J.H. & Enquist, B.J. (1999) A general model for the structure and allometry of plant vascular systems. *Nature* **400**, 664–667.

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