The justification for the model is that the allometric cascade arises from the layering of function at various levels of organization[1], with the numerous steps involved in pathways of demand and supply each characterized by their own b_i and c_i values. Differences in the scaling of the basal metabolic rate and the maximum metabolic rate are then accounted for by noting that the c_i coefficients are different in the two cases and thus the calculation involves mixing different 'cocktails' of the components, as was assumed in ref. 4.

Dimensional analysis shows that the units of c_i depend crucially on the exponent b_i. This follows on noting that, because the units of MR and a are fixed, the units of $c_i M^{b_i}$ are independent of i. Thus the requirement in equation (2) that the c_i values add up to 1 is erroneous because it is meaningless to add quantities with different units and require that the sum be unity.

Furthermore, the model (equation (1)) critically depends on the units of mass that are chosen. Consider a toy example of a two-process system with c_1 and c_2 equal to 1/2 (in different units, as above) and the exponents b_1 and b_2 being 1 and 2, respectively, with a body mass of 1 kg. (Note that this point does not depend on the choice of these numbers.) When body mass is measured in kilograms, the contributions of the two processes to MR/a are equal. However, if the mass were measured in grams, the second process would contribute 1,000 times as much as the first.

This absurd result of an arbitrary relative contribution of the processes depending on the mass scale used is a consequence of the flaw discussed earlier. For a linear combination of different power laws, it does not follow that an effective exponent is simply a weighted average of the individual exponents[4].

The cascade model cannot be salvaged by recasting equation (1) in the form $\mathrm{MR} = \mathrm{MR}_0 \Sigma_i c_i (M/M_0)^{b_i}$, where the body mass is measured in units of a 'characteristic' body mass, M_0, and MR_0 is the metabolic rate of an organism of this mass. First, as the power law in question spans 20 orders of magnitude in mass, there is no characteristic mass scale and the choice of value for M_0 is completely arbitrary.

Second, the value of MR calculated for a species of any given body mass will differ with the choice of M_0, unless the c_i values are allowed to vary in response (in which case they are neither constants nor are they constrained to sum to 1, as equation (2) requires of control coefficients).

Third, both the relative contributions to MR of each of the i terms in the summation, and the relative values of MR calculated for species of different masses, depend on the choice of M_0. For any given set of body masses, the effective slopes of the log–log mass–metabolic-rate plots, such as those in ref. 1 and based on this new equation, will exhibit any value between the lowest and highest b_i value, depending on the value that is chosen for M_0.

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Darveau et al. *reply* — West *et al.* and Banavar *et al.* criticize our results on mathematical grounds, but they overlook the consistency of our multiple-cause model (concept) of metabolic scaling[1] with what is known from biochemical[2] and physiological[3] analysis of metabolic control. Their single-cause explanations[4,5] are based on the assumption that whole-body metabolism in animals is exclusively supply-limited, whereas there are many factors that together explain the observed patterns of metabolic scaling[6,7]. Our concept can accommodate these multiple causes, the range of metabolic scaling exponents observed in various taxa[8], and variation in exponents due to physiological state[7].

Allometric equations are mathematical descriptions of empirical relationships, rather than derived physical laws[6]. Our equation[1] is a first approximation that attempts to express our concept in mathematical terms. It does not distinguish between energy-demand processes that occur in parallel and supply processes that operate in series. It suffers from a semantic flaw that imposes units on the control coefficient, c_i.

In a modified equation (J. Endelman) to determine the basal metabolic rate, $\mathrm{BMR} = \mathrm{MR}_0 \Sigma c_i (M/M_o)^{b_i}$, where MR_0 is the characteristic metabolic rate of an animal with a characteristic body mass M_0, c_i is rendered dimensionless while the exact meaning of the original equation[1] is retained. With M_0 of 1 unit mass, MR_0 now takes the place of the value a, as found in the standard scaling equation[6] and in our original. For mammalian maximum metabolic rate, MMR, the same equation applies with a roughly tenfold higher MR_0. We were able to find the relevant b_i values and estimate c_i for various processes in mammals to demonstrate the utility of our model.

Although using mammalian data precludes extrapolation to non-mammalian species, our concept can be used to understand metabolic scaling in other taxa. Our equation is not a power-law function, but yields meaningful results when a biologically realistic range of b_i values is used in simulations. The examples we used yield results that are indistinguishable from power functions, reflected in r^2 values that are greater than 0.999. Lower r^2 values result when b_i values outside the biological range are used.

The inherent limitations of the data, and estimates based on them, offer new directions for experiments, and the shortcomings of our equation highlight the need for better ways to express our multiple-cause model. Branching distributive structures and supply limitations[4,5] may contribute to metabolic scaling, although supply limitations contribute minimally to BMR, which scales with an exponent that is close to 0.75. Supply limitations have a greater influence on MMR[3,9], but the allometric exponent for this is paradoxically higher[7]. These and the many factors that contribute to the allometric scaling of metabolic rates[6,7,10], as well as the observation that cellular metabolic rates *in vitro* decline with increasing body mass[10,11], should give pause to advocates of single-cause explanations for metabolic scaling.

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