a subduction-related andesite that was not used to promote plate tectonics on Mars.

The classification of "andesite" rocks at the Mars Pathfinder landing site is tenuous: this name (actually icelandite) was originally assigned on the basis of its major-element chemistry, noting that a sedimentary origin or weathering rind could not be ruled out without textural or mineralogical data[9]. A re-analysis of α-proton X-ray spectrometer chemistry[10] indicates that Pathfinder rocks may have a high water content, which supports a non-igneous classification.

Formation mechanisms on a global scale are required to explain the extensive distribution of type-2 materials in the northern lowlands. The weathered-basalt interpretation would predict a large body of water or sedimentary depocentre in the northern lowlands, but does not account for local occurrences of type-2 materials in the southern highlands. These might be either andesites formed by igneous fractionation or the result of local weathering processes.

The production of fractionated magmas of intermediate composition is an inefficient process unless it is promoted by dissolved water, so the occurrence of vast amounts of andesite would probably require a wet source region and efficient transport of less dense magmas. The intermediate-volcanism interpretation requires large-scale melting of the thin northern crust and flooding with high-silica volcanics, as well as isolated melting pockets in the thick and ancient southern highlands.

However, the mechanism for rejuvenated mantle melting, the degree of magma fractionation and crustal assimilation, and the creation of local hotspots are all unresolved issues. Furthermore, there is a poor correlation between the transition of surface type-1 and type-2 materials with the transition from thick to thin crust, which suggests that the distribution of materials is a result of surficial processes rather than crust–mantle ones.

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COMMUNICATIONS ARISING

Physiology

Why does metabolic rate scale with body size?

A long-standing problem has been the origin of quarter-power allometric scaling laws that relate many characteristics of organisms to their body mass[1,2] — specifically, whole-organism metabolic rate, $B = aM^b$, where M is body mass, a is a taxon-dependent normalization, and $b \approx 3/4$ for animals and plants. Darveau *et al.*[3] propose a multiple-cause model for mammalian metabolic rate as the "sum of multiple contributors", B_i, which they assume to scale as $B_i = a_i M^{b_i}$, and obtain $b \approx 0.78$ for the basal and 0.86 for the maximally active rate, $\dot{V}_{O_2}^{max}$. We argue, however, that this scaling equation is based on technical, theoretical and conceptual errors, including misrepresentations of our published results[4,5].

All of the results of Darveau *et al.*[3] follow from their equation (2), $B = a\Sigma c_i M^{b_i}$, which they neither derive nor prove. As control coefficients[6], c_i, and exponents, b_i, are dimensionless, this must be incorrect because it violates dimensional homogeneity, leading to different results for b that depend on the units of mass: for the basal rate, $b \approx 0.76$ when M is in kilograms, and $b \approx 1.08$ when M is in picograms.

Now, by definition, $c_i \equiv \partial \ln B / \partial \ln B_i$, which leads to the standard sum rules[6] $\Sigma c_i = 1$ and $\Sigma c_i \varepsilon_i = 0$, where $\varepsilon_i = b - b_i$ with $b(M) \equiv d\ln B / d\ln M$, the slope of $\ln B$ versus $\ln M$, and $b_i(M) \equiv d\ln B_i / d\ln M$. This gives $b = \Sigma c_i b_i$, the equations that Darveau *et al.* should have used to determine b from the empirical c_i and b_i. These formulae are very general, requiring no assumptions about how B and B_i scale, or whether the B_i are connected in parallel or in series. Darveau *et al.*, however, use $B = \Sigma B_i$, implying that the B_i are added in parallel and, as such, their model is simply a consistency check on the conservation of energy, which requires all "ATP-utilizing processes"[3] (in parallel) to sum to B and so must be trivially correct. This gives $c_i = (a_i/a)M^{-\varepsilon_i}$ and $B = a\Sigma(c_i M^{\varepsilon_i})M^{b_i}$, which is the (dimensionally) correct version of equation (2).

As Darveau *et al.* take a and a_i as constant, their c_i must scale as $M^{-\varepsilon_i}$. However, they assume that c_i (and b_i) $\propto M^0$, which requires b (which equals $\Sigma c_i b_i$) $\propto M^0$, thereby contradicting their equation (2), in which b depends on M. This inconsistency in the M-dependence of b is concealed in their plots, which cover only three orders of magnitude in M, over which b is almost constant (about 0.78 for basal). However, when their analysis is extended to the realistic eight orders of magnitude spanned by mammals, their b increases with M to an average value of about 0.85 and, for $\dot{V}_{O_2}^{max}$, to about 0.98, which are both inconsistent with other data[1,2].

Darveau *et al.* have taken their value for b_i from empirical data, without explaining why B_i or B_i scales nonlinearly with M, or why most $b_i \approx 3/4$. Understanding these features is the real challenge — the formulation of Darveau *et al.* is therefore hardly fundamental. By contrast, our theory[4,5] is grounded in basic principles of geometry, physics and biology, and offers a general unifying explanation for these and the other quarter-power scalings that are so pervasive in biology.

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Physiology

Allometric cascades

An allometric power-law relationship between metabolic rate and the mass of living organisms has been observed over many orders of magnitude in mass, indicating that (among other things) a characteristic mass scale is not applicable. Darveau *et al.*[1] present a multiple-cause cascade model of metabolic allometry, which has been hailed as a new perspective on comparative integrative physiology[2] and scaling relationships[3]. Here we show that this cascade model is flawed and is therefore meaningless both for control of metabolic rate in an organism of a given size and for scaling of the metabolic rate.

The basic equation of the cascade model[1] is

$$\mathrm{MR} = a\sum_i c_i M^{b_i} \qquad (1)$$

where MR is the metabolic rate in any given state, M is body mass, a is a coefficient, b_i is the scaling exponent of the process i, and c_i is the control coefficient of the process i. The control coefficients are chosen so that

$$\sum_i c_i = 1 \qquad (2)$$

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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