

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 data⁹. A re-analysis of α -proton X-ray spectrometer chemistry¹⁰ 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), $B = a \sum c_i M^{b_i}$, which they neither derive nor prove. As control coefficients⁶, 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 rules⁶ $\sum c_i = 1$ and $\sum c_i \epsilon_i = 0$, where $\epsilon_i = b - b_i$ 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 $b = \sum c_i b_i$, 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 $B = \sum B_i$, 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 $c_i = (a/a_i) M^{-\epsilon_i}$ and $B = a \sum (c_i M^{\epsilon_i}) M^{b_i}$, 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 $M^{-\epsilon_i}$. However, they assume that c_i (and b_i) $\propto M^0$, which requires b (which equals $\sum c_i b_i$) $\propto M^0$, 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 , 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 physiology² and scaling relationships³. 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 model¹ is

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

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

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