Evidence against universal metabolic allometry.

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A long-standing theoretical problem in biology is the relation between organism size M and metabolic rate Q. This long history of research into such a basic pattern is due to the particular shape of the relationship: it is generally observed that the relation is well described as $Q \propto M^b$ where b is most often in between 0.5 and 1. Numerous analyses have focused on determining b using interspecific data, in particular to test whether $b = ⅔$ or $b = ¾$. Here a large amount of intraspecific data is analysed, which shows that $b \neq ⅔$, $b \neq ¾$, and especially that there is no single, universal value of b.

Keywords: body size, metabolic rate, scaling

Introduction

Despite numerous established relations between body size and metabolic rate within as well as between species from various taxa (e.g. Peters 1986), there is still no consensus on what shapes the relation. An early theory suggested it is shaped by the geometry of organisms maintaining stable body temperature. In Euclidian geometry, the surface area of an object increases as the ⅔ power of its volume. If the rate of heat loss is assumed to be proportional to the surface area of the body, the power required to keep such an object at constant temperature increases with the ⅔ power of its volume. Initially this was found to be quite close to the observed pattern, but as empirical deviations from $b = ⅔$ accumulated it became replaced by $b \approx 0.72$-0.73, which was eventually replaced by $b = ¾$ (see Dodds et al. 2001 and references therein).

Although many accepted $b = ¾$ as a ubiquitous rule, the value remained purely empirical: there was no theoretical explanation except that it was observed that if organism geometry had a fourth dimension, metabolic rate would scale as body size to the power ¾ (Blum 1977). Only recently a serious potential fourth dimension was proposed. With the discovery of fractal geometry in biology, it was noted that the internal transport networks most organisms use (e.g. lungs, trachea, the blood vascular network) appear to have a fractal dimension. It was suggested that if organisms use such networks to minimize the cost of transporting substances inside the body, then metabolic rate scales as the ¾ power of body size (West, Brown & Enquist 1997, 1999). This theory, which elegantly relies on the intuitive supposition that organisms have evolved so as to maximize interal energetic efficiency, has proved powerful to explain various relationships associated with scaling in plants as well as animals (Enquist, Brown & West 1998, Gillooly et al. 2001, West, Brown & Enquist 2001).

There is little evidence against ¾-power scaling though some have questioned its generality (Whitfield 2001). It has been shown that the individual processes that make up the whole-organism metabolic rate (e.g. ventilation, cardiac work, circulation) have

different scaling characteristics (Darveau et al. 2002, Weibel 2002, see also Reich 2001). Also it was shown that in birds the observed relations are not statistically significantly different from the ⅔ scaling relationship (Dodds et al. 2001), and that mammal species below and above the modal body size of a taxon appear to have different scaling relations (Dodds et al. 2001, Lovegrove 2000).

However, much of the evidence for (and against) universal ¾-power scaling of metabolic rate comes from analyses of interspecific data affected by neglection of species' phylogenetic non-independence (Symonds and Elgar 2002). Thus, the variability of estimates of b in literature may be due to (*i*) measurement error, (*ii*) neglection of the historical relationships of species and (*iii*) true variation between species (Lovegrove 2000). The phylogenetic uncertainty in interspecific analyses can be considerable, especially if one takes into account that a character like metabolic rate may evolve in a punctuated manner rather than gradually over time (Lovegrove 2000, Bokma 2002). It is therefore more informative to investigate intraspecific allometry, avoiding assumptions on tempo and mode of metabolic rate evolution. If the relationship between body size and metabolic rate is shaped by geometrical factors -whether this geometry is Euclidian or fractal does not matter- metabolic rate should increase with body size within species in the same way as between species (West, Brown & Enquist 2001).

Methods

Measurements of fish metabolic rate were downloaded from the online database at www.fishbase.org. Of all available data on metabolic rate (n = 6555 measurements from 313 species) those obtained from measurements under stress were excluded. Also 1158 measurements obtained from active animals were excluded. Of the remaining 3575 another 3 were excluded as they did not specify the mass of the fish measured. The 3572 measurements that were finally used for analysis came from 113 species in 217 series, and ranged in temperature from -1.5 °C to 38°C and in mass from 0.01 g to 10.4 kg.

The relation between metabolic rate and body size within species was determined by least squares linear regression of the natural logarithm of oxygen consumption on the natural logarithm of body mass. Regression coefficients and intercepts (that is body size exponents and coefficients, respectively) were calculated for all combinations of temperature and salinity for which there were at least three measurements (after averaging measurements at identical body size). This was done because temperature affects metabolic rate (Gillooly et al. 2001) and also salinity might do so. A total of 198 regressions were analysed. For some species more than one regression was available. For example, the carp bream *Abramis brama* is represented twice, with a series of three measurements at 18 °C and a series of 32 measurements at 20 °C. Variance in the 198 estimates of b may be due to chance alone, or to true variation between species. Equality of regression coefficients was assessed by analysis of covariance (ANCOVA, Zar 1996 p. 362).

The data contains a number of measurements at constant body mass and constant conditions, which allows estimation of measurement error. Measurement error was

defined as the standard deviation of repeated measurements sd_e. 114 such series of repeats (containing 421 measurements) were used to determine the best linear relation between average (Q) and standard deviation of repeated measurements as: $sd_e = \exp(0.94 \ln Q - 1.72)$. This relation between sd_e and Q was used to obtain Monte Carlo estimates of statistical power for the 198 regressions. For all mass data supporting a certain observed regression coefficient, Q was calculated as $M^b + e$ where e is measurement error, sampled from a normal distribution with zero mean and standard deviation sd_e. From those simulated data regression coefficients were estimated (50 repeats for every regression). These estimates are on average b, but deviate more or less from this expectation depending on the number and spread of the mass data. Their variance indicates the statistical power of the observed regression. This procedure may be somewhat imprecise, but gives a relatively good picture of which are the most informative estimates of b.

Results

Intraspecific allometry

The regressions yielded a variety of body size exponents, as expected in a large set of estimates, especially as some were based on small sample sizes. Indeed, the variance of estimates decreases with increasing sample size (Fig. 1a) and with increasing body size range (Fig. 1b). The most informative estimates of body size exponents (with least sampling variance in the simulations) are from large samples spanning a large body size range. Even these estimates deviate considerably from $b = ⅔$ and $b = ¾$ (Fig. 1c). The most powerfull sample is one containing 37 measurements of sea trout *Salmo trutta trutta*, over a body size range from 0.1 to 600 grams. The corresponding estimate of the body size exponent of metabolic rate is 0.86 ($r^2 = 0.997$) which is highly significantly different from both ⅔ and ¾ ($P < 0.001$).

The weighted average of estimated body size exponents was calculated, with the weighting factor the inverse of the standard deviation of simulated estimates. The thus obtained average is 0.715, which in between the ”traditional” hypothesised values.

Estimates of the body size exponent were tested for significant departure from the hypotesised values ⅔ and ¾ using t-tests. When compared against a theoretical value of ¾, 21 out of 198 estimates are significant at the 5% level after sequential Bonferroni correction. When compared against the theoretical value ⅔, 16 out of 198 estimates remain significant after sequential Bonferroni correction. The data thus clearly reject both hypothesised body size exponents.

As the data do not agree with the hypothesised exponents, it is possible that there is another exponent, perhaps in between ⅔ and ¾, or that there is no universal scaling relation. In the latter case the individual estimates come from populations with different body size exponents b. ANCOVA revealed that the 198 estimates are very unlikely to come from populations with identical b ($F_{197,1342} = 7.33$, $P << 0.001$). If the analysis of covariance is restricted to the 25 most powerful estimates (thus excluding the estimates from small samples and small body size ranges) this conclusion remains ($F_{24,466} = 15.96$, $P << 0.001$).

Discussion

The data reject not only both hypothesised body size exponents $b = ⅔$ and $b = ¾$ but – more importantly- also the hypothesised existence of universal scaling of metabolic rate. That result does not rely on the estimates of b from small samples, which may be affected by the use of least squares regression where the use of reduced major axis regression or other statistics (Dodds et al. 2001) might have been more appropriate. The differences between these alternatives are neglegible in the statistically powerful samples if the correlation coefficients are high. In fact, the correlation coefficients in the 25 most powerful samples are high, and these samples also strongly reject the existence of universal scaling.

The finding that $b \neq ⅔$ contradicts the conclusion of Dodds et al. (2001) who carefully re-analysed available data on bird metabolic rate and found that the data do not reject $b = ⅔$. The traditional theoretical explanation for $b = ⅔$ is that heat leaves the body through the surface of the body, which in Euclidian geometry scales with the ⅔ power of body volume. It is tempting to argue that changes in shape during individual development cause deviations from intraspecific ⅔-power scaling, or that not all heat is lost through the body surface. However, it can be shown largely on theoretical grounds that the scaling of basal metabolic rate is not caused by scaling of the requirements to maintain stable body temperature. If basal metabolic rate is set by the requirement to maintain stable body temperature, organisms would decrease metabolic rate as far as permitted by the environmental circumstances. The contrary is observed in homeotherms, that do not decrease metabolic rate below a certain level as environmental temperature raises. This level is defined as basal metabolic rate, and its existence proves that there is a limit below which metabolic rate cannot go, and that this limit is not set by requirements to maintain stable body temperature. It is this limit that scales. Thus, basal metabolic rate does not scale due to the geometry of heat loss.

It is therefore more interesting that the data also reject $b = ¾$, the prediction of much recent physically inclined theoretical work (West, Brown & Enquist 1997, 1999). If the scaling of metabolic rate is determined by the fractal dimension of branching networks as suggested, deviations from $b = ¾$ may be due to non-fractal branching. Biological networks are not true fractals that break an organism into smaller but self-similar structures. Biological networks are instead what Bejan appropriately called ”constructals”: structures that are build up of small units that together form larger, more or less self-similar structures. As compared to adult individuals from different species, especially in individuals of increasing size of the same species that building-process may be dominantly present.

So far however the data support theories that explicitly allow for differences in scaling relations (Weibel 2002, Darveau et al 2002). Darveau et al.’s (2002) theory however ”predicts” the scaling of maximum and basal metabolic rate from observed scaling relationships of the individual processes that make up those overall rates. It thus fails to explain why metabolic rate would scale in the first place and why the individual processes would scale as they do. That is a serious shortcoming, as it is not unknown that

also non-living systems such as car engines and electrical amplifyers cannot increase their maximum power without increasing power at stationary performance, which suggest a universal, perhaps thermodynamic, reason for some sort of scaling.

We are just starting to understand the basic rules that appear to govern much of biology's seemingly dazzling complexity. It cannot be overemphasized how important those unifying developments are in the light of the biological tradition of emphasizing differences. It appears however that with respect to the scaling of metabolic rate there just are more differences than present unifying theories allow for.

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Figure 1. Estimates of the body size exponent of metabolic rate plotted against (a) the number of measurements, (b) the difference between the largest and smallest body sizes in the sample, and (c) a Monte Carlo estimate of statistical power which unites sample size and body size range. The best estimates place the exponent in between 0.5 and 1.

Figure 2. The relation between metabolic rate and body size in sea trout *Salmo trutta trutta*. The line through the dots is the least squares regression line $Q \propto M^{0.86}$, the line above the dots is the theoretical prediction $Q \propto M^{3/4}$, shifted up for clarity.