Metabolic Scaling in Animals: Methods, Empirical Results, and Theoretical Explanations

Craig R. White[*1] *and Michael R. Kearney*[2]

▶ ABSTRACT

Life on earth spans a size range of around 21 orders of magnitude across species and can span a range of more than 6 orders of magnitude within species of animal. The effect of size on physiology is, therefore, enormous and is typically expressed by how physiological phenomena scale with mass[b]. When $b \neq 1$ a trait does not vary in direct proportion to mass and is said to scale allometrically. The study of allometric scaling goes back to at least the time of Galileo Galilei, and published scaling relationships are now available for hundreds of traits. Here, the methods of scaling analysis are reviewed, using examples for a range of traits with an emphasis on those related to metabolism in animals. Where necessary, new relationships have been generated from published data using modern phylogenetically informed techniques. During recent decades one of the most controversial scaling relationships has been that between metabolic rate and body mass and a number of explanations have been proposed for the scaling of this trait. Examples of these mechanistic explanations for metabolic scaling are reviewed, and suggestions made for comparing between them. Finally, the conceptual links between metabolic scaling and ecological patterns are examined, emphasizing the distinction between (1) the hypothesis that size- and temperature-dependent variation among species and individuals in metabolic rate influences ecological processes at levels of organization from individuals to the biosphere and (2) mechanistic explanations for metabolic rate that may explain the size- and temperature-dependence of this trait. © 2014 American Physiological Society. *Compr Physiol* 4:231-256, 2014.

Introduction

Living species span a size range of around 21 orders of magnitude, from the smallest single-celled micro-organisms (∼0.1 pg) to giant sequoias *Sequoiadendron giganteum* and redwoods *Sequoia sempervirens* weighing several thousand tonnes. To put this size range in perspective, the earth weighs 6×10^{27} g, which is about 10^{21}-times heavier than an elephant. The difference in size between the largest and smallest organisms is therefore equivalent to the difference in size between the largest extant terrestrial vertebrate and the earth itself. Even relatively similar organisms span a large size range: the smallest adult vertebrate is the fish *Paedocypris progenetica*, which measures 7.9 mm long at maturity (216) and probably weighs less than 1 mg; the largest is the blue whale *Balaenoptera musculus* that weighs up to 190 t (294). During ontogenetic development, some animals can increase in size by at least six orders of magnitude (205, 280). Biological processes are tightly related to the physical dimensions of the system (cell and organism) they occur in, especially through the implications of surface area, volume and their interaction. The effect of size on the biology of organisms is, therefore, enormous. The study of how aspects of biology vary with size is called scaling; when a trait varies in proportion to body mass it is said to scale isometrically (from Greek, meaning "equal measure"). Surprisingly, however, many characteristics of animals do not vary in direct proportion to their size; such scaling is referred to as allometric (meaning "by another measure").

That many aspects of biology vary allometrically with animal size was probably first formally recognized by Galileo Galilei (1637, cited by 356) and the study of allometric scaling has attracted considerable attention ever since (5, 44, 52, 97, 145, 178, 188, 221, 314, 357, 381, 386, 387, 404). The effect of mass (M) on an aspect of biology (Y) is commonly described using a power function of the form:

$$Y = aM^b$$

where a represents the value of Y for an animal of unit mass and b represents the scaling exponent. When $b = 1$ the relationship is isometric and Y is proportional to M; when $b = 0$, Y is independent of M; when b takes other values the relationship is allometric. For example, the volume (V) of an object with characteristic length l is proportional to l^3 and its surface area (SA) is proportional to l^2. Thus, since $l \propto SA^{1/2} \propto V^{1/3}$ and $V \propto M$, it follows that surface area is proportional to mass$^{2/3}$. This is true for simple objects such as spheres and squares, but

---

*Correspondence to craig.white@uq.edu.au; mrke@unimelb.edu.au

[1]School of Biological Sciences, The University of Queensland, St Lucia, Queensland, Australia

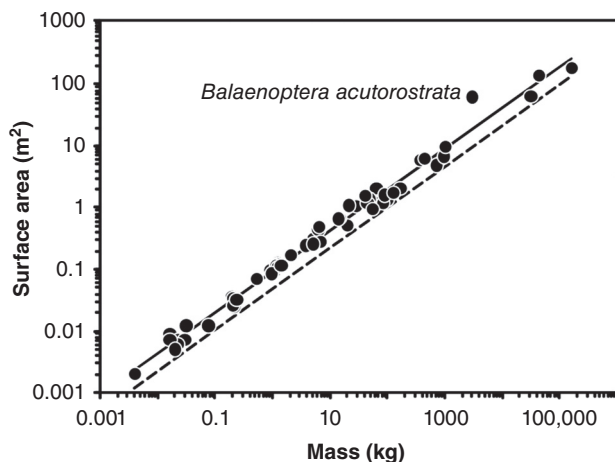[2]Department of Zoology, The University of Melbourne, Victoria, Australia

Figure 1 Scaling of surface area with body mass (M, kg) in mammals (data from 333). The solid line is the phylogenetically informed scaling relationship (surface area = $0.092 M^{0.67}$) (333). The lower dashed line is the relationship between the surface area and volume of a sphere with a density of 1.08 g cm^{-3}, a rough estimate of a mixture of muscle, fat, and bone: muscle has a density of 1.06 g cm^{-3}; bone has a density of 2.00 g cm^{-3}; and fat has a density of 0.93 g cm^{-3} (7).

also for organisms with relatively complex shapes (e.g., mammals: 333). An advantage of the power function for describing scaling relationships is that the relationship between Y and M becomes linear when expressed on log-log axes:

$$\log(Y) = \log(a) + b \, \log(M)$$

The relationship between surface area and body mass, therefore, appears as a straight line with a slope of 2/3 (Fig. 1). Allometric scaling with an exponent between 0 and 1, as is the case for body surface area, indicates that relative to M, Y is smaller in large animals (e.g., the $SA{:}V$ ratio decreases with size). When b is greater than 1, on the other hand, large animals have relatively large values of Y. For example, the scaling exponent for the size of weapons and ornaments is typically in the range 1.5 to 2.5 (209), indicating that large animals have relatively larger weapons for their body size than small animals.

Although the use of power functions to describe scaling patterns is widespread (44, 52, 138, 314, 357), it must be borne in mind that models that violate rules for dealing with dimensions are useful only as empirical descriptions, because the meaning of a model is lost if it contains transcendental functions of variables that are not dimensionless (212). The dimensionality problem inherent in the standard two-parameter power equation can be solved by measuring mass with reference to a characteristic mass M_0:

$$Y = Y_0(M/M_0)^b$$

However, the relationship now has three rather than two parameters, and there is no natural reference value M_0 for weights (17, 212). The use of the standard power function

$Y = aM^b$ also introduces the problem that a takes units of YM^{-b}, and is, therefore, a function of b (455). These shortcomings do not devalue the standard two-parameter power equations as a useful empirical description of scaling patterns, providing that the units of mass are defined and are used in a consistent and clear fashion. For example, if the units of M in Figure 1 were not defined then the scaling relationship between surface area (SA, in m^2) and M (surface area = $0.092 M^{0.67}$) would be nonsensical because the value of SA estimated from M would differ if M was measured in mg, g, or kg. Providing that any scaling relationship is treated as conditional upon measurement of M in consistent units, no such problems arise.

Describing scaling relationships

Statistical descriptions of the patterns of the relationship between Y and M are key to the development of an understanding of the scaling of biological traits. A distinction that is not often made in the physiological literature is that between the various forms and levels of allometric scaling (451). In the morphological literature, three types are recognized: ontogenetic, static, and evolutionary (see, e.g., 62, 313, 371). Ontogenetic scaling considers growth trajectories of the relationship between Y and M for a single individual, although cross-sectional comparisons of multiple individuals from the same species at different stages of ontogenetic development are more common than longitudinal comparisons in the physiological literature (e.g., 205, 280, 363, 382, 389). Static scaling considers the relationship between Y and M among individuals of the same developmental stage within a species, such as within an instar for insects (e.g., 342, 408). Evolutionary scaling considers the relationship between Y and M among individuals of different species, again at the same developmental stage. The distinction between these forms of scaling is particularly important when comparing among models that have been proposed to explain scaling relationships (see below), because some models have different explanations for different forms of scaling, whereas others do not (180, 202, 213, 249, 462).

The most common statistical description of the relationship between Y and M, irrespective of the form or level of scaling being considered, is a linear relationship between $\log(Y)$ and $\log(M)$, usually calculated using ordinary least squares (OLS) regression. This approach makes three key assumptions about the data: (1) that the X-variable, M, is measured without error, (2) that linear regression of log-log data is an appropriate statistical model for describing the relationship between Y and M, and (3) that the data are independent. Depending on the data being analyzed, one or more of these assumptions are likely to be violated.

Regression models—measurement error

Measurement error refers to any variation that causes an observed value to be randomly different from the "true" value

of interest (192, 252). It is important to recognize that in this context measurement error includes not only the sources of variation that biologists usually regard as error (e.g., instrument and transcription error), but also encompasses genuine biological variation such as interindividual differences as well as diel and seasonal variation (160, 307). Such interindividual differences present a problem for scaling studies, because although they are important (i.e., they are repeatable, heritable, and have consequences for performance: 29, 50, 56, 291, 437, 442), they nevertheless contribute variance to the species mean which is typically used for such analyses. In scaling studies, instrument error is likely to be small (307, 314), but other sources of variation are likely to be non-negligible (e.g., 22, 75, 276, 318). When the X-variable in a scaling study is measured with error (e.g., when each data point represents the mean mass of multiple individuals, multiple measurements of the same individual, or measurements of mass taken from the literature for individuals other than those for which the Y-variable was measured), a commonly advocated approach is to used reduced major axis (RMA) regression rather than OLS regression (226, 383). The use of RMA regression is increasing, both because of its utility in estimating functional relationships (226, 360, 383), and because of an increased appreciation of the assumptions of OLS regression. However, RMA regression also makes assumptions about the error distribution of the data, and this assumption may also be violated. Specifically, RMA regression assumes that that error variance in Y is equal to that in X. McArdle (253) suggests that OLS is the better technique to use as long as the error variance in the independent variable is less than one third of that in the dependent variable, but few scaling studies assess these error variances. In the case of the interspecific relationship between metabolic rate (MR) and M, the coefficient of variation (CV = s.d. divided by mean) of MR and M are reasonably similar (374), but interspecific differences in MR between species of similar size that are associated with ecology and life-history are rather large and amount to sixfold to sevenfold for endotherms and ~25-fold for ectotherms (e.g., 431, 441, 444). For at least these data, Monte Carlo simulations suggest that OLS regression is appropriate (429). OLS regression is also considered appropriate when the purpose of a scaling relationship is to enable prediction of Y from measurements of X (383), and when X is thought to be affecting Y, rather than the reverse (380).

Regression models—data transformation

Related to the problem of measurement error is the problem of choosing an appropriate regression form. Linear OLS regression of log-log transformed data is the most commonly applied approach, but a number of recent studies have discussed potential problems associated with this approach (163, 183, 184, 298-305, 304). The use of log-log transformation potentially introduces bias when estimating data on the original untransformed scale, and also potentially introduces bias into estimates of the elevation and exponent of scaling

relationships. A possible solution to these problems is to fit the power equation $Y = aM^b$ directly, using nonlinear estimation without log-log transformation (e.g., 300, 304). Nonlinear regression methods can be implemented in many standard statistical packages, and generally result in models that fit values for large species better than power functions fitted by linear regression of log-log transformed data (184). A nonlinear fit to untransformed data applies a model with additive error on the original (untransformed) scale

$$Y = aM^b + \varepsilon$$

whereas linear regression of log-log transformed data applies a model with additive error to the transformed data

$$\log(Y) = \log(a) + b \log(M) + \varepsilon$$

which, when back-transformed to the original scale, results in a model with multiplicative error

$$Y = aM^b 10^\varepsilon$$

The extent to which this is a problem depends upon the data in question. Since many biological phenomena are inherently multiplicative (60, 135, 203), linear regression of log-log transformed ontogenetic or interspecific data may be appropriate (112, 159, 292, 329, 429, 454, 456). In such cases, nonlinear models may poorly fit data for small values of M (429).

Of course, the foregoing discussion regarding the best way to fit a power equation $Y = aM^b$ to empirical data hinges upon the appropriateness of this relationship for describing the relationship between Y and M. Early proponents of the use of such power equations were Snell (381), who used power functions to describe the relationship between brain size and body size, and Krogh (221), who suggested that the metabolic rate of endotherms was proportional to M^b rather than M; power functions have remained in widespread use since then (5, 44, 52, 145, 178, 188, 314, 357). While many relationships are well described by a power function, it may not be appropriate for all types of data. Strict adherence to a power function may, therefore, hinder examination of the relationship between Y and M (379). Not all species and traits conform to linear relationships on log-log axes, and relationships may be multilinear or curved on log-log axes. Examples include, but are not limited to, aspects of sexual size dimorphism (105), morphology (e.g. 26, 177, 182, 197, 345, 375, 388), maximum speed during running or swimming (e.g., 61, 72, 127, 447), metabolic rate (e.g., 95, 138, 165, 210, 300, 309, 429), and population density (e.g., 376). For example, a critical assumption of a two-parameter power function estimated by linear regression of log-log transformed data is that the original data conform with a power function having an intercept of 0 in a plot with arithmetic coordinates (304). This assumption is rarely checked and is not always met (300, 305). An alternative is a three-parameter power function, which

accommodates a nonzero intercept through the addition of a mass-independent constant (c):

$$Y = aM^b + c$$

This statistical model can be estimated by nonlinear regression of untransformed data, and assumes a component of variation in Y that is independent of body mass (c), and a component that scales allometrically in proportion to M^b.

Phylogenetic signal in interspecific scaling

Most methods for line-fitting (including both OLS and RMA) assume that each data point in the analysis is independent of the others. This assumption is likely to be violated for comparative studies including data for multiple species, because closely related species are likely to be more similar that distantly related ones. The problem also potentially applies to intraspecific studies of related individuals because closely related individuals are more likely to be similar that distantly related ones, though this is rarely acknowledged except in quantitative genetic studies that partition phenotypic variation into genetic and other components (106, 243; see also 153 for a demonstration of the link between quantitative genetic and comparative analyses). Violation of the assumption of independence in interspecific analyses leads to inflated degrees of freedom, increased Type I error rates, overestimation of the strength of regression relationships, and a significant increase in the variance of the scaling exponent estimate (119, 125, 160, 295, 343). Blomberg et al. (34) have shown that for studies with 20 or more species, most traits show significant phylogenetic signal, defined as the tendency for related species to resemble each other. The traits considered included morphological, physiological, ecological, and behavioral ones, and significant phylogenetic signal was evident even for traits that are thought to be evolutionarily malleable or subject to relatively strong environmental effects, as well as for those traits subject to high levels of measurement error (34). This demonstrates unambiguously that many types of comparative data show phylogenetic non-independence, and that comparative studies (including those aimed at understanding the effect of body size) should be analyzed in a phylogenetic context. Indeed, incorporating phylogenetic information in allometric analyses is essential because, when data show phylogenetic nonindependence, the inclusion of phylogenetic information can significantly alter estimates of the scaling exponent (e.g., 69, 432, 443), although this is not always the case (e.g., 116, 431). A common measure of phylogenetic signal is λ (119, 306), which normally varies between 0 and 1 and quantifies the degree to which trait evolution deviates from Brownian motion ($\lambda = 1$). λ is mathematically equivalent to phylogenetic heritability, defined as the proportion of variance in a character that is explained by the relationship among taxa as given by the phylogeny (153, 181, 242), thereby further consolidating the conceptual link between the estimation of phylogenetic signal in comparative analyses and heritability in quantitative genetic analyses, since heritability is the ratio of additive genetic to total phenotypic variance.

A range of methods are available for undertaking phylogenetically informed analyses. These include phylogenetic generalized least squares (128, 146, 225, 251), independent contrasts (110), phylogenetic eigenvector regression (92), phylogenetic autocorrelation (63, 64), and phylogenetic mixed models (153, 181, 242), though not all methods explicitly incorporate evolutionary models (118). Many of these can be implemented using freely available software packages. For example, independent contrasts (110) can be implemented in Mesquite (244) using the Phenotypic Diversity Analysis Package module (277) and phylogenetic generalized least squares can be implemented in R (330) using the Analysis of Phylogenetics and Evolution package (310), or in Matlab using the Regressionv2 program (225). The "caper" (Comparative Analyses of Phylogenetics and Evolution in R) package in R implements both PGLS and independent contrasts (297). Recent work has provided methods for incorporating measurement error into comparative analysis (192), for disentangling phylogenetic and spatial effects (120, 223), for conducting phylogenetically informed logistic regression (191), and has applied Markov chain Monte Carlo algorithms to phylogenetic generalized linear mixed models, thereby extending the generality of phylogenetically informed analysis to a range of non-Gaussian data distributions (153).

Although some discussion remains regarding the need to account for phylogeny in comparative analyses (230, 269, 266, 265, 424-426), the value of this approach is now well appreciated (119, 125, 160, 225, 336, 343). The phylogenetically informed approach requires a phylogeny, and although large trees including thousands of species are becoming more common (e.g., 27, 28, 121, 195, 327, 328), appropriate phylogenies are nevertheless unavailable for some groups. It is clear, however, that it is better to include an incomplete tree than no tree at all (90, 91, 326), especially if the poorly known aspects of the tree (e.g., soft polytomies and unknown branch lengths) are appropriately accounted for (reviewed in 125). Errors in topology, for example, are most problematic when near the tips of the tree, but such errors tend to be conservative in that they generally act to conceal real relationships (394). The best approach for conducting an allometric analysis with an incomplete tree is, therefore, to include phylogenetic information, but reduce the impact of topological errors by representing uncertain relationships as polytomies and conducting statistical tests with appropriately reduced degrees of freedom (124, 126, 325). The extent of phylogenetic nonindependence in the data can then be tested for using metrics such as λ (119, 306) and the K-statistic (34). Such tests have revealed that phylogenetic nonindependence is common in most biological data (34), but is not ubiquitous (e.g., 237, 255, 449). The degree of phylogenetic nonindependence for a given data set can be explicitly incorporated in the analyses by modifying the covariance matrix in phylogenetic generalized least squares to accommodate the degree of nonindependence

in the data (e.g., 53, 98, 431-433). Such an approach ensures reliable estimation of the elevation (*a*) and scaling exponent (*b*) of allometric relationships.

Size-dependent covariates

Many traits in addition to metabolic rate are correlated with body mass, and the incorporation of these into scaling relationships can influence the values of the estimated parameters. For example, body temperature is weakly associated with body size in some mammalian lineages, and inclusion of body temperature in the relationship between metabolic rate and body mass decreases the estimated scaling exponent of metabolic rate (443). Similarly, in accordance with Bergmann's rule (24), body size generally increases with latitude in endotherms (e.g., 14, 15, 32, 275), and some, but not all, groups of ectotherm (3, 25, 66, 85). This potentially introduces a problem when describing the relationship between metabolic rate and body mass, because species from high-latitude and cold environments often have higher metabolic rates than those from warmer environments (e.g., 4, 11, 186, 194, 238, 385, 391, 392, 430, 431, 449). Among species of mammals, body mass may also be associated with diet and life-history traits, including litter size and maximum longevity (199, 267), and these may also have confounding associations with metabolic rate (e.g., 267, 286, 393, 429, 444). Though most studies of metabolic scaling fail to do so, it is, therefore, important to consider other biotic and abiotic variables that may be associated with both mass and metabolic rate when describing the scaling of metabolic rate. In doing so, however, it is important not to conflate correlation and causation; for example, an animal's body temperature is dictated by its metabolic rate and thermal conductance, so body temperature is not a cause of variation in metabolic rate. Nonetheless, variation in body temperature can be used to explain variation in metabolic rate statistically, and can, therefore, be used to improve predictions of metabolic rate beyond those that can be made on the basis of body mass alone. As always, manipulative experiments should be used to establish causation; the inclusion of covariates in scaling relationships can establish only correlations.

Data requirements

When compiling data for scaling relationships, a commonly raised question concerns the quantity of data required to generate a robust relationship, both in terms of the number of data points included in the data set and the mass range of species being considered. Previous studies of intraspecific scaling of metabolic rate have included mass ranges that vary from less than 1.5-fold (e.g., 79) to over 3-million-fold (280). Data included in studies of the interspecific scaling of metabolic rate with broad taxonomic representation now include data spanning 16 or 20 orders of magnitude range in body mass (87, 250, 436), a range that comes close to encompassing all living organisms. For mammals, interspecific metabolic scaling relationships including 150 species spanning three to four
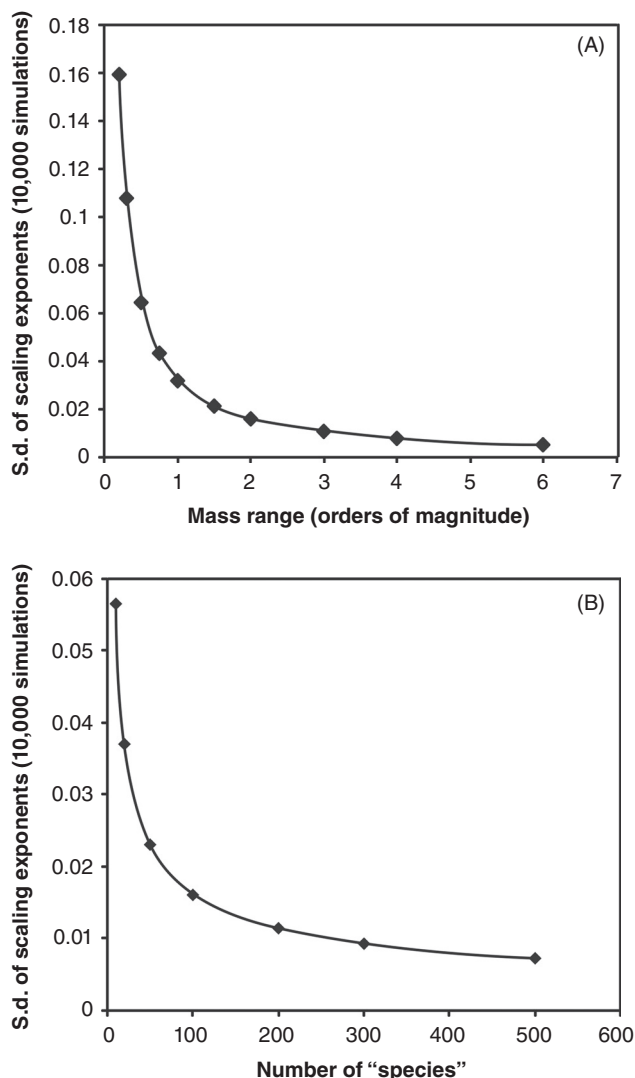

Figure 2 (A) Standard deviation of 10,000 scaling exponents (*b*, where $Y = aM^b$) calculated for mass ranges of 0.2 to 6 orders of magnitude. (B) Standard deviation of 10,000 scaling exponents (*b*, where $Y = aM^b$) calculated for sample sizes of 10 to 500 "species." For each of the 10,000 simulations in (A) 100 values of log(*M*) spanning the appropriate range were randomly generated and values of *Y* were calculated as $M^{0.75}$ plus a normal deviate with a mean of 0 and a standard deviation equal to 20% of $M^{0.75}$. For each of the 10,000 simulations in (B), values of log(*M*) spanning two orders of magnitude were randomly generated and values of *Y* were calculated as $M^{0.75}$ plus a normal deviate with a mean of 0 and a standard deviation equal to 20% of $M^{0.75}$. Values of *b* were then calculated as the slope of the relationship between log(*Y*) and log(*M*).

orders of magnitude variation in body mass are sufficient to distinguish between scaling exponents of 2/3 and 3/4 (446), but less data may be sufficient for many purposes. Monte Carlo simulations suggest that variation in the calculated scaling exponent decreases most dramatically as the mass range approaches one to two orders of magnitude, suggesting that a data set spanning this mass range is sufficient to reliably estimate the value of *b* (Fig. 2A; see also Ref. 76 who presented similar relationships for the population density of mammals). Similarly, the increase in precision associated with increases

of sample size above approximately 100 suggests that this number is sufficient (Fig. 2B). It must be borne in mind, however, that while 100 species spanning approximately one to two orders of magnitude range in body mass may be sufficient to reliably estimate the scaling exponent for a given data set, extrapolation beyond the mass range of the data is inappropriate, and considerably more data may be required to detect subtleties in the data, such as curvature, and small data sets may not provide sufficient power to detect differences among subsets within the data.

A related problem for comparative studies concerns the number of individuals that are required to be measured for a species mean to be included in a data set. In their analysis of metabolic scaling in birds McKechnie and Wolf (256) considered only data that unambiguously met the criteria for basal metabolic rate (BMR, the metabolic rate of a postabsorptive inactive nonreproductive adult endotherm thermoregulating in a thermoneutral environment during its inactive phase: 262, 314) and had sample sizes with n ≥ 3, and showed convincingly that for such analyses including data measured under poorly defined conditions biases the outcome of scaling analyses. However, McNab (264) demonstrated that the estimated BMR of a single individual is usually within 2% to 8% of the species' mean when the individual is measured eight or more times. Since the difference between individual mean BMR and the mean of individual means is minimized when an individual is measured eight times (264), future work should attempt to obtain eight measurements per individual if a reasonable estimate of a species mean is required from measurements of a single individual (such data could be complimented by calculation of a phylogenetically informed prediction for the species of interest: see "*Predicting traits from body mass*," below). Similarly, the inclusion of data for rare, elusive, or endangered species with small sample sizes in comparative studies is warranted to maximize phylogenetic or spatial representation. Again, single individuals should be measured multiple times (eight, ideally), though the inclusion of data for individuals measured a smaller number of times is unlikely to be problematic, because such data are unlikely to introduce bias. Such data are likely to increase the variance of the data set, however. If this is suspected to be a problem, funnel plots of mass-independent residuals against sample size or log-transformed sample size can be used to determine if data should be weighted by sample size; such approaches are common in meta-analyses (see, e.g., 100, 434, 435).

Interpreting scaling relationships

Interpreting the value of the scaling exponent

Once a data set has been gathered and the scaling relationship determined, the interpretation of the relationship depends on the question at hand. For some studies, the value of the scaling exponent itself may be of interest, and may be compared to a null expectation to test a given hypothesis. For example, the role of gravity in the cardiovascular systems of terrestrial animals has been of interest for decades (16, 41, 129, 130,
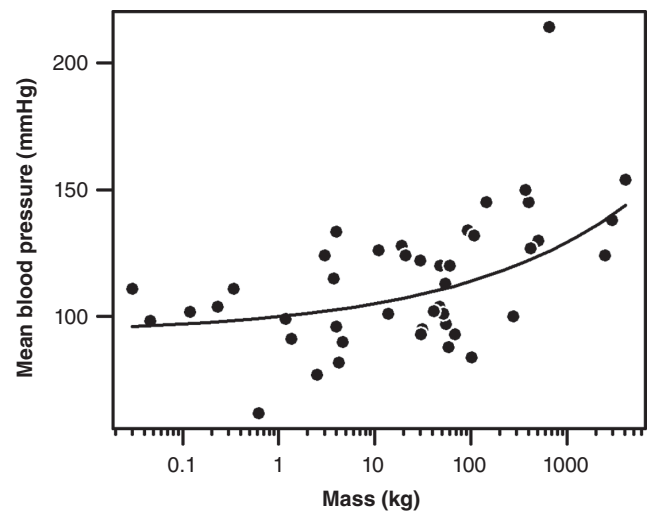

Figure 3 The scaling of mean arterial blood pressure (*P*, mmHg) in mammals (data and analysis from 448). Solid line is the significant three parameter power equation: $P = 6.9\,M^{0.24} + 93$. The scaling exponent is significantly greater than 0, and not significantly different from the scaling exponent of 0.33 predicted by geometric scaling of the vertical distance between the heart and head (448).

174, 175, 231, 232, 278, 311, 362, 364-366, 448). Since the total height of the blood column above the heart increases with animal size, and the hydrostatic pressure at the bottom of a column of fluid is calculated as the product of fluid density (ρ), gravitational acceleration (g) and the height of the column (h), then blood pressure should be positively related to body size if the heart must work against gravity to pump blood to the head. If the heart does not work against gravity, then blood pressure should be size-invariant. The hypothesis that the heart works against gravity can therefore be tested by examining the scaling of systolic and diastolic blood pressure in terrestrial mammals, and testing for a scaling exponent that is significantly greater than zero (448). Such an analysis reveals that blood pressure increases with size in accordance with the predicted scaling of head-heart distance, in favor of the hypothesis (Fig. 3).

A similar approach is taken to test hypotheses concerning any hypothesized value of the scaling exponent. For example, because the weight of a bone varies in proportion to its volume ($b = 1$), but its strength varies in proportion to its cross-sectional area ($b = 0.67$), it can be hypothesized that the skeletal elements of large terrestrial species should be relatively more robust than those of small species to support their mass against gravity. Thus, it can be predicted that the skeletal mass of animals that must support their weight against gravity should increase allometrically ($b > 1$), because the cross sectional area, not the mass, of bones must increase in proportion to animal mass. This hypothesis can be tested by examining the scaling of skeletal mass with body mass for terrestrial animals. When all available data for skeletal mass are considered; however, the scaling of skeletal mass for terrestrial animals ranging in size from 7 g arctic shrews *Sorex arcticus* to 9 t African elephants *Loxodonta africana* does
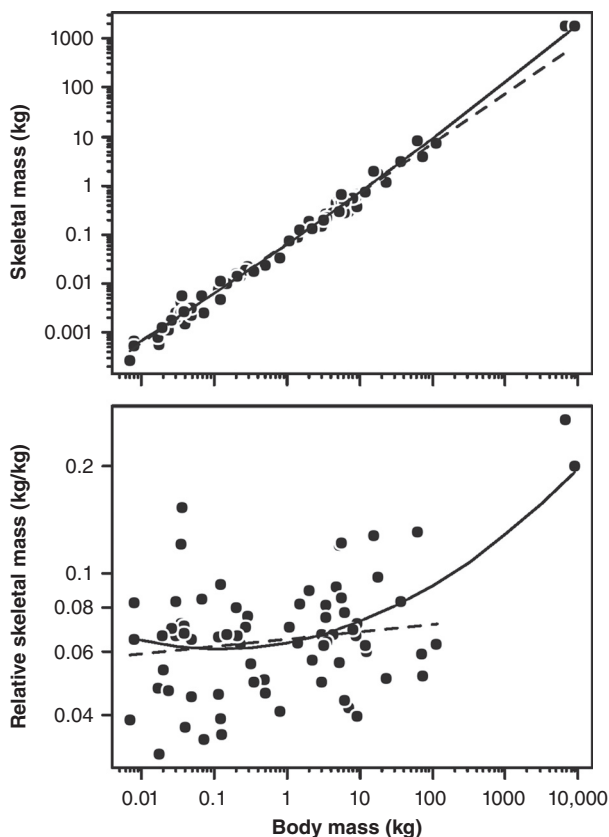
Figure 4 Scaling of skeletal mass (M_s) and relative skeletal mass with body mass (M) in mammals. Relative skeletal mass is calculated by dividing skeletal mass by body mass. Data are for 73 species and were compiled from published sources (21, 241, 319, 322, 335), and matched to a supertree of mammals (28). Data were analyzed using phylogenetic generalized least squares (PGLS) (128, 146, 251) in the APE (Analysis of Phylogenetics and Evolution) package (310) within R (189) according to established procedures (98, 156, 432). In addition to the dated branch lengths associated with the supertree, a range of branch length transformations were compared: star, $\log_e$, punctuated, Grafen's (146), Nee's (324), and Pagel's (308). For each of these models, a measure of phylogenetic correlation, λ (119, 306), was estimated by fitting PGLS models with different values of λ and finding the value that maximizes the log likelihood. The degree to which trait evolution deviates from Brownian motion ($\lambda = 1$) was accommodated by modifying the covariance matrix using the maximum likelihood value of λ, which is a multiplier of the off-diagonal elements of the covariance matrix (i.e., those quantifying the degree of relatedness between species). All models were compared on the basis of Akaike's Information Criterion (AIC) as a measure of model fit (49). The solid line is a significant second-order polynomial regression relating $\log(M_s)$ to $\log(M)$ and $[\log(M) + 4]^2$: the best model included a phylogeny with all branch lengths equal to 1 ($w_i = 0.18$, $\lambda = 0.32$); $\log(M_s) = -1.53 + 0.87 \log(M) + 0.02 [\log(M) + 4]^2$. Dashed line is the best model for data excluding *Loxodonta africana* and *Elephas maximas* and including a phylogeny with all branch lengths equal to 1 ($w_i = 0.68$, $\lambda = 0.37$), which is a linear model: $\log(M_s) = -1.19 + 1.02 \log(M)$. The 95% confidence interval of the scaling exponent for the linear model includes 1 (95%CI: 0.98-1.06).

not conform to a simple two-parameter power function, but instead to a curved relationship on log-log axes (Fig. 4). This finding highlights the importance of considering nonpower-function allometric scaling, but also highlights the potential influence of high leverage values on the interpretation of scaling patterns. In this case, excluding data for the two species

of elephant leads to a different interpretation: if only species up to and including 113 kg *Gorilla* are considered, then the scaling exponent of skeletal mass is not significantly different from one (Fig. 4). These analyses suggest that the scaling of skeletal mass is isometric for most terrestrial mammals, as is also the case for cetaceans (236, 334) and fish (447), but the largest terrestrial mammals have relatively heavy skeletons. It is noteworthy, however, that the relationship in Figure 4 includes no data for terrestrial mammals between 113 kg and 6 t, so this conclusion should be reevaluated as more data become available.

Comparing scaling relationships: Similar exponents for a single trait

One of the most common and valuable uses of scaling relationships lies in their ability to compare traits between groups of animals while accounting for the allometric relationship between the trait of interest and body mass. When the scaling exponents for two or more groups of animals are not significantly different, analysis of covariance (ANCOVA) can be used to test for differences between the groups to determine if there are mass-independent differences in the mean value of the trait of interest (302, 303, 383, 456). The major assumptions of ANCOVA are that (i) data are randomly selected from the population and randomly assigned to groups, (ii) within group regressions are homogenous, (iii) the covariate and treatment are statistically independent, (iv) covariate values are fixed and error free, (v) within-group regression are linear, (vi) conditional Y scores are normally distributed, (vii) conditional Y scores show homogeneity of variance, (viii) treatment levels are fixed (185). It is important to recognize that the ANCOVA approach is not equivalent to comparing mean values of mass-specific data ($Y_{m-s} = Y/M$), because Y_{m-s} is independent of M only when Y scales isometrically (i.e., when Y is proportional to M^1). When Y scales allometrically with M, the scaling exponent of Y_{m-s} (b_{m-s}) is the negative complement of the whole-body one ($b_{m-s} = b - 1$). When $b \neq 1$, the error introduced by using mass-specific data to account for variation in mass is substantial, even when the mass range is small. For a twofold range in M, for example, the error is 16 or 20% if $b = 0.75$ or 0.67, respectively.

The ANCOVA approach has been used in a phylogenetic context to compare the metabolic rates of arid and nonarid species on multiple occasions (e.g., 255, 289, 405, 429, 428). Species living in arid environments may be predicted to have low metabolic rates because resources are sparse and widely distributed (239, 259, 281), or, in the case of endotherms, because of constraints imposed by high temperatures on heat dissipation (272, 385, 431). Since the scaling exponent of field metabolic rate (FMR) is not significantly different between arid and non-arid birds (405), it is possible to test for differences in FMR between them while simultaneously accounting for the effect of mass using ANCOVA. Such an analysis reveals that species from arid environments have, on average for a given body mass, lower metabolic rates than those from
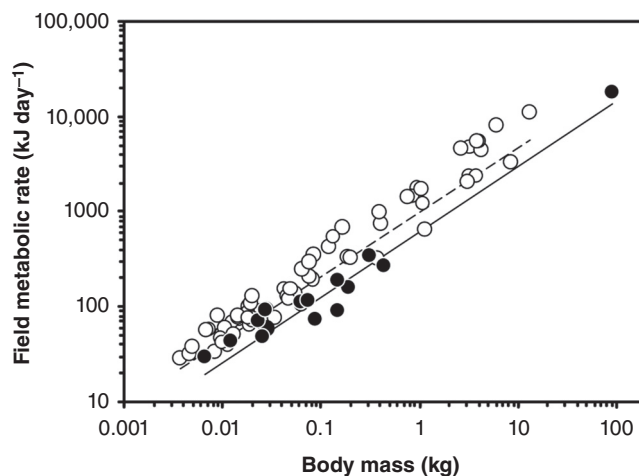
Figure 5 Scaling of field metabolic rate (FMR) with body mass (M) in arid (filled symbols, solid line) and nonarid (unfilled symbols, dashed line) birds (data from 405). Phylogenetically informed relationships: arid birds, FMR = 5.24 (2.86-9.59) $M^{0.691\ (0.610\text{-}0.772)}$; non-arid birds, FMR = 9.31 (7.79-11.12) $M^{0.676\ (0.383\text{-}0.969)}$; values in parentheses are 95% confidence limits. Analyzed using independent contrasts (110), the scaling exponent of FMR does not differ among arid and non-arid birds ($t = -1.57$, $P = 0.12$), whereas environment (desert or non-desert) does have a significant effect ($t = 2.11$, $P < 0.04$) (405).

Figure 6 Scaling of heart mass with body mass for birds (unfilled symbols, dashed line) and mammals (filled symbols, dashed line) (data from 362). The scaling exponents of heart mass are different for birds ($b = 0.91$) and mammals ($b = 1.06$). Application of the Johnson-Neyman technique (196) demonstrates that at $P = 0.05$, regression elevations are not significantly different at masses above 4.26 kg (427), thus confirming the conclusion that the hearts of flightless birds are not significantly larger than those of similarly sized mammals (362). The vertical dashed line represents the lower limit of the region of non-significance. Within the region of nonsignificance there is no significant difference in elevation between the scaling relationships, so in this example the groups differ significantly in elevation to the left of the vertical line. Birds: heart mass (g) = 8.08 $M^{0.91}$, mammals: heart mass = 4.04 $M^{1.06}$.

nonarid ones (Fig. 5). Of course, the term "arid" conceals within it a multitude of characteristics including low primary productivity, low and temporally unpredictable precipitation, extreme ambient temperatures and high potential evapotranspiration, and any or all of these might explain the relationship between metabolic rate and aridity. When the dichotomous distinction between arid and non-arid environments is abandoned in favor of analyses including continuous predictor variables such as net primary productivity (NPP), mean annual habitat temperature, and precipitation, it becomes clear that NPP explains little of the variation in metabolic rate (431). Instead, after body mass, metabolic rate is generally most strongly associated with mean habitat temperature (194, 238, 385, 431). This negative relationship between metabolic rate and temperature explains why low metabolic rates are observed in bird species from both arid (e.g., 255, 289, 405, 429, 428) and tropical environments (154, 450, 452).

Comparing scaling relationships: Dissimilar exponents for a single trait

In the previous examples, mean values of a trait for two groups were compared while accounting for the relationship between the trait and body mass by including body mass as a covariate in ANCOVA. A requirement of ANCOVA is that the relationship with the covariate is uniform across groups; that is, the regression slopes are homogenous (assumption ii of ANCOVA, above). In practice, a prerequisite for ANCOVA is, therefore, demonstration that the scaling exponent of the trait is not significantly different between groups. When the scaling exponents differ between groups, it is not possible
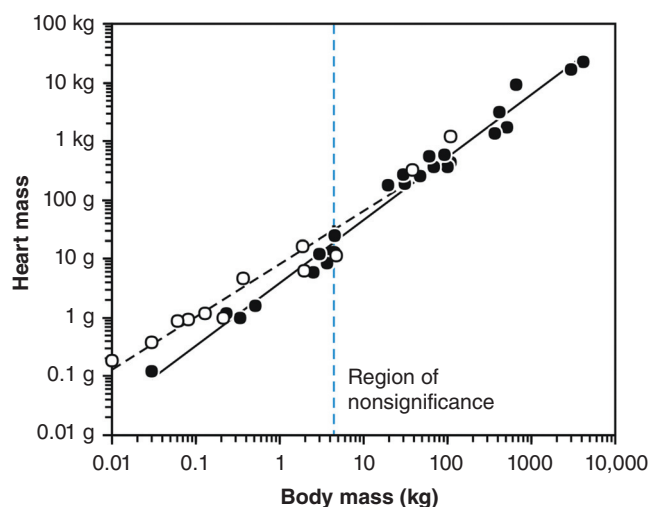
to compare them using ANCOVA (456). Such a problem arises when comparing the heart masses of mammals and birds (Fig. 6), the BMRs of wild and captive birds (255) and the FMRs of mammals and reptiles (289, 385). Demonstration of scaling exponents that differ significantly between groups can be regarded as evidence of a significant treatment effect (73, 301), leading to the conclusion that the groups are significantly different, but in some situations, it may be valuable to analyze the data further. In the case of heart mass, for example, Seymour and Blaylock (362) demonstrated significantly different scaling exponents between mammals and birds, and concluded that "the bird hearts were obviously heavier within the range of similar body mass" and that "the scaling factor was twice as high at a body mass of 1 kg, but the data converge in larger species" (p. 395). In such a situation it would be valuable to determine the range of body masses over which the heart masses of birds and mammals are different, and the range of masses over which they are not. Such an analysis can be undertaken by applying the Johnson-Neyman technique (78, 96, 104, 185, 196, 227, 320, 329, 427, 457).

The Johnson-Neyman technique is used to identify the range of X values for which two groups are not significantly different (henceforth referred to as "the region of nonsignificance") by determining the confidence interval associated with the point at which nonparallel regression lines intersect (245, 329). The technique has previously been applied to

the fields of medical and behavioral science, sociology, and ecology (e.g., 96, 132, 185, 227, 457), and more recently to the field of comparative physiology, including: analyses of growth and development in birds (347, 367) and marsupials (113, 395); metabolic physiology in birds (12, 157), mammals (428), and fish (176, 296); and digestive physiology in birds and mammals (59, 225). Application of the Johnson-Neyman technique to the heart mass data provided by Seymour and Blaylock (362) yields a region of nonsignificance that ranges from a lower mass of 4.26 kg to an upper mass well beyond the largest animal in the data set (Fig. 6). Thus, the analysis confirms the conclusion that the hearts of flightless birds are not significantly larger than those of similarly sized mammals. The hearts of small (<4.26 kg) birds, on the other hand, are significantly larger than those of similarly sized mammals.

The major assumptions of the Johnson-Neyman technique are similar to those of ANCOVA (185): (i) the residuals of the within-group regressions of Y on X are independent, and individuals have been randomly selected from a specified population and randomly assigned to groups; (ii) the residuals are normally distributed; (iii) the residuals have homogeneous variance for each value of X; (iv) the residuals have homogeneous variance across treatment groups; (v) the regression of Y on X is linear; (vi) the levels of the covariate are fixed; and (vii) the covariate is measured without error. Assumption (i) is likely to be violated for comparative data because closely related species tend to be more similar than distantly related ones, but unfortunately there is not yet a phylogenetically informed implementation of the Johnson-Neyman technique. Nevertheless, the technique has been applied in studies that incorporate phylogenetic information wherever possible (225, 428). It is also unfortunate that the technique is not implemented in commercially available statistical packages. A Microsoft Excel spreadsheet that performs both ANCOVA and the Johnson-Neyman technique accompanies an earlier presentation of the technique (427) and is available for distribution via email from CRW. The limits of the region of nonsignificance are calculated according to

$$X_{\text{lower}} = \frac{-B - \sqrt{B^2 - AC}}{A}$$

$$X_{\text{upper}} = \frac{-B + \sqrt{B^2 - AC}}{A}$$

Where

$$A = \frac{-F_{(\alpha,1,N-4)}}{N-4}(SSres_i)\left(\frac{1}{\sum x_1^2} + \frac{1}{\sum x_2^2}\right) + (b_1 - b_2)^2$$

$$B = \frac{F_{(\alpha,1,N-4)}}{N-4}(SSres_i)\left(\frac{\overline{X}_1}{\sum x_1^2} + \frac{\overline{X}_2}{\sum x_2^2}\right) + (a_1 - a_2)(b_1 - b_2)$$

$$C = \frac{-F_{(\alpha,1,N-4)}}{N-4}(SSres_i)\left(\frac{N}{n_1 n_2} + \frac{\overline{X}_1^2}{\sum x_1^2} + \frac{\overline{X}_2^2}{\sum x_2^2}\right) + (a_1 - a_2)^2$$

$$SSres_i = \left(\sum y_1^2 - \frac{(\sum xy_1)^2}{\sum x_1^2}\right) + \left(\sum y_2^2 - \frac{(\sum xy_2)^2}{\sum x_2^2}\right)$$

$F_{(\alpha,1,N-4)}$ = critical value of F statistic at α for 1 and $N-4$ degrees of freedom; N = total number of observations = $n_1 + n_2$; n_1, n_2 = number of observations in groups 1 and 2, respectively; $\overline{X}_1, \overline{X}_2$ = covariate means for groups 1 and 2, respectively; a_1, a_2 = regression intercepts for groups 1 and 2, respectively; b_1, b_2 = regression slopes for groups 1 and 2, respectively. The quantities $\sum x_1^2, \sum x_2^2, \sum y_1^2, \sum x_2^2, \sum xy_1$, and $\sum xy_2$ are calculated according to:

$$\sum x_1^2 = \sum X_1^2 - \frac{(\sum X_1)^2}{n_1}$$

$$\sum y_1^2 = \sum Y_1^2 - \frac{(\sum Y_1)^2}{n_1}$$

$$\sum xy_1 = \sum XY_1 - \frac{(\sum X_1)(\sum Y_1)}{n_1}$$

$$\sum x_2^2 = \sum X_2^2 - \frac{(\sum X_2)^2}{n_2}$$

$$\sum y_2^2 = \sum Y_2^2 - \frac{(\sum Y_2)^2}{n_2}$$

$$\sum xy_2 = \sum XY_2 - \frac{(\sum X_2)(\sum Y_2)}{n_2}$$

Comparing scaling relationships: Multiple traits and the analysis of residuals

In the previous examples, scaling exponents for a single trait (e.g., metabolic rate) were compared between groups of species (e.g., birds and mammals, or mammals from arid and nonarid environments) as a prelude to a comparison of these groups using ANCOVA or the Johnson-Neyman Technique. A further application of allometry lies in the comparison of scaling exponents for separate traits for a single group of animals to test hypotheses that the traits are related. Such an approach is often applied to test hypotheses of animal design, such as the hypothesis that heat loss through the body surface influences the scaling of metabolic heat production, as first proposed by Sarrus and Rameaux in the 1830s (39, 339, 346). Their logic was as follows: since the heat produced as a by-product of metabolism must ultimately be lost through the body surface, the rate at which heat is produced by animals (i.e., their metabolic rate) should be matched to the area over which it is dissipated. Thus, since body surface area scales as $M^{0.67}$ (Fig. 1), the scaling exponent of metabolic rate should be similar. Indeed, a number of studies have reported scaling exponents close to 0.67 for endotherms (168, 170, 221, 339, 346, 443), though other studies have reported different values (23, 39, 40, 53, 206, 207, 210, 240, 350, 374, 432). It is important to recognize, however, that demonstration of statistically similar scaling exponents for multiple traits demonstrates only that they share a similar relationship with body mass; it does not demonstrate that the traits are functionally related (see, e.g., 271). Demonstration of a functional association requires demonstration that traits are related independent

Table 1 Parameter estimates for the relationship between basal metabolic rate (BMR, mL h^{-1}), body mass (M, g), and body surface area (SA, m^2) for mammals (logBMR = 1.04 + 0.87 logM − 0.30 logSA)

Parameter	Estimate	SE	P
Intercept	1.04	0.27	0.001
logM	0.87	0.21	0.0005
logSA	−0.30	0.30	0.33

Data from Dawson and Hulbert (83), Reynolds (333), and White and Seymour (443).

Table 2 Parameter estimates for the relationship between exercise-induced maximum aerobic metabolic rate ($\dot{V}O_2$ max, mL min^{-1}) and body mass (M, g), muscle mitochondrial volume (V_{mt}, mL), and muscle capillary volume (V_c, mL)

Term	Estimate	SE	P
Mitochondrial volume			
Intercept	0.56	0.26	0.06
logM	−0.09	0.18	0.62
logV$_{mt}$	1.10	0.18	0.0003
Capillary volume			
Intercept	0.92	0.34	0.03
logM	−0.44	0.40	0.30
logV$_c$	1.42	0.40	0.007

Data from Weibel et al. (412) and references therein.

of their shared relationships with body mass. In the case of the putative relationship between metabolic rate and body surface area, it must be demonstrated not that body surface area and metabolic rate scale with similar exponents, but that species with a relatively high body surface area also have a relatively high metabolic rate. Such associations are often examined using mass-independent residual values that are calculated by subtracting the trait value predicted by a scaling relationship from the measured trait value, and testing for an association between the traits (e.g., 444). Such an approach has been criticized as a statistically flawed *ad hoc* procedure (117, 123, 164). The most strident criticisms are that parameter estimates are biased and the error degrees of freedom in the analysis of residuals are overestimated because the estimation of the regression coefficients for the traits is not considered. An approach preferable to the analysis of residuals is to use standard or phylogenetically informed multiple regression to control for the potentially confounding effect of body mass (117). Application of this approach to test for an association between BMR and the body surface area of mammals demonstrates that these traits are not associated (444), despite scaling similarly with body mass (Table 1). It should be borne in mind, however, that just as size-dependent covariates can influence the relationship between traits and body mass (see "*Size-dependent covariates*," above), mass-independent associations among traits may also be influenced by biotic and abiotic covariates (i.e., a mass-independent relationship between surface area and metabolic rate could be obscured by differences in ambient temperature among species and studies, for example).

The multiple regression approach has also been applied to tests of the symmorphosis hypothesis that animals are designed optimally (411, 414, 415). In this case, one might hypothesize that the maximum aerobic metabolic rate of an animal during exercise ($\dot{V}O_2$max), which represents the maximum rate of oxygen transport through the oxygen cascade from the lungs to the mitochondria, should be matched to capillary volume of the locomotory musculature (V_{cap}, the location of the final convective step of the oxygen cascade) and to total mitochondrial volume (V_{mt}, where the oxygen is consumed). When the scaling of these traits is examined, they are found to scale similarly with body mass ($b = 0.962, 0.984,$ and

0.956 for $\dot{V}O_2$max, V_{cap}, and V_{mt}, respectively) (412). Again, however, this is not sufficient to demonstrate functional associations. Multiple regression indicates that $\dot{V}O_2$max is significantly correlated with both V_{cap} and V_{mt} independent of their shared relationship with body mass (Table 2), supporting the hypothesis that $\dot{V}O_2$max is associated with the aerobic capacity of the locomotory musculature (412).

The preceding examples concern cases where putative associations are examined between traits that scale similarly with body mass, but associations can also exist between traits that scale with significantly different scaling exponents. If the BMR of mammals is assumed to scale as a simple power function of mass, the scaling exponent is ~0.71 (53, 374, 432; see 71, 210, 283, 429 for discussion of curvature in the scaling of metabolic rate). Heart rate, on the other hand, scales with an exponent of −0.26 (Fig. 7). Heart rate and metabolic rate are related according to the Fick equation (111), which describes the relationship between rate of oxygen consumption ($\dot{V}O_2$), heart rate (f_H), stroke volume (V_s), and the oxygen contents of arterial (C_aO_2) and mixed venous blood (C_vO_2):

$$\dot{V}O_2 = f_H V_s (C_aO_2 - C_vO_2)$$

According to the Fick equation, a high metabolic rate (high $\dot{V}O_2$) is matched to an increase in either or both of heart rate and oxygen pulse (= V_s multiplied by oxygen extraction: $C_aO_2-C_vO_2$). The positive relationship between heart rate and metabolic rate underpins the "heart rate" method for estimating the energy expenditure of free-living animals (51, 148). Similarly, it can be hypothesized that species with high rates of metabolism and oxygen consumption will have high heart rates, but scaling relationships alone fail to support this hypothesis: metabolic rate increases with size ($b = 0.71$) whereas heart rate decreases with size ($b = -0.26$). Thus, the
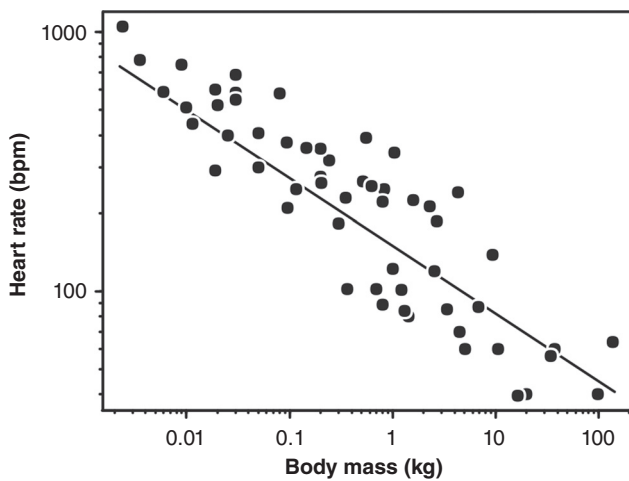
Figure 7 Scaling of heart rate (f_H, bpm) with body mass (M, kg) for mammals. Data are for 58 species compiled by White and Seymour (444), matched to a supertree of mammals (28), and analyzed according to the PGLS methods described in the legend to Figure 3. Solid line is the scaling relationship fitted by PGLS with all branch lengths equal and equal to 1, with a maximum likelihood λ of 0.71 ($f_H = 150\ M^{-0.26}$ [95% CI: −0.30 to −0.22]).

Figure 8 Interspecific relationship between heart rate (f_H, bpm) and basal metabolic rate (BMR, mL h^{-1}) assessed using phylogenetic generalized least squares. Data are for 58 species (444) matched to a supertree of mammals (28). The relationship between BMR and f_H was then assessed by comparing the fit of statistical models that either included BMR (logf_H ~ logM + logBMR) or did not include BMR (logf_H ~ logM). The best model was chosen on the basis of its Akaike weight (w_i, the probability that a model is the best of a candidate set, given the data). The best model included logBMR and logM ($w_i = 0.19$) with a maximum likelihood λ of 0.64 and all branch lengths set equal to one (a punctuated model of evolution). Summed over all evolutionary models (dated, punctuated, Grafen's, Nee's and Pagel's), the probability that the best model for logf_H includes logBMR in addition to logM is 0.66 (see legend to Figure 3 for full details of analysis procedure). Residuals are shown to account for the relationships between logM and both logBMR and logf_H, but the analysis of the relationship between f_H and BMR was not assessed using residuals; the solid line is the parameter estimate for logBMR from the best model (logf_H ~ logM + logBMR) plotted through the bivariate mean of residuals.

largest animals have the highest absolute metabolic rates and the lowest heart rates, which seems counterintuitive. A test for an interspecific association between metabolic rate and heart rate remains valid; however, it is possible to test for an association between heart rate and metabolic rate, while accounting for body mass, using the multiple regression approach. Such a test reveals that heart rate and metabolic rate are indeed positively associated so, for a given size, species with high metabolic rates have high heart rates (Fig. 8). The discrepancy between the scaling exponents of heart rate ($b = -0.26$, Fig. 7) and metabolic rate ($b = 0.71$) can then be explained by the scaling of cardiac stroke volume ($b = 1.03$: 362), since the product of stroke volume and heart rate increases with size as $M^{0.77}$, which is similar to the scaling of metabolic rate.

While the multiple regression approach overcomes the problems associated with the analysis of residuals and has provided significant insight regarding the associations among metabolic rate and a range of other traits, it is nonetheless a potentially imperfect solution. This is because, as discussed above, many traits in addition to metabolic rate covary with body mass (52, 314, 357). This collinearity among traits can result in spurious conclusions about the relationship between dependent and independent variables in multiple regressions, because the partial regression coefficients associated with the independent variables may not be representative of the relationship that exists in the population (456). Such problems can be overcome by moving from a univariate approach where associations between a single dependent and multiple independent variables are examined to a multivariate approach in which covariances among traits are estimated in a mixed model framework that incorporates phylogenetic information (153).
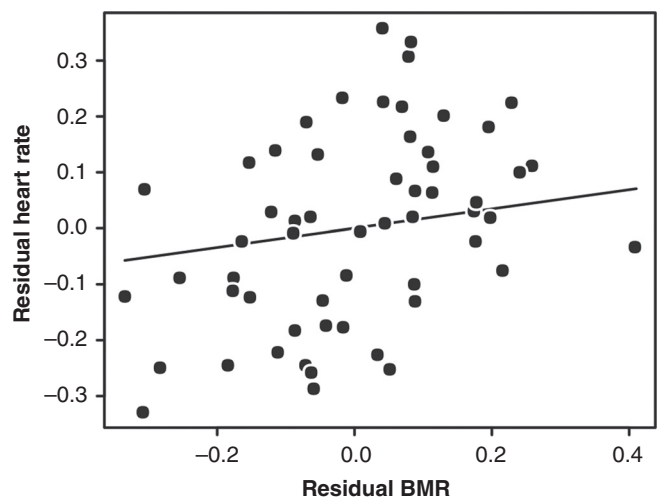
Using scaling relationships
Predicting traits from body mass

When a trait of interest is significantly related to body mass, the regression describing the relationship can be used to predict the value of the trait, based on measurements of only body mass. This is an often quoted use of allometry (e.g., 149, 200, 235, 246, 247, 256, 267, 268, 287, 289) and is particularly prevalent in the human literature where scaling principles have been used to predict rates of metabolism (e.g., 172, 173, 284) and drug clearance (e.g., 396, 397, 398). Such analyses of intraspecific scaling for humans now routinely include data for metabolic rate and organ sizes of hundreds of individuals, and have been particularly useful for establishing the influence of body composition and stature on the scaling of metabolic rate with size (171, 172, 173, 284), and for estimating *in vivo* metabolic rates of organ-tissue compartments (409, 410).

Scaling relationships can also be applied to the prediction of physiological characteristics of extinct species (114, 270, 348, 359, 361, 368), and, because of the allometric relationship between body mass and other morphological variables, can be used to predict morphological characteristics that are
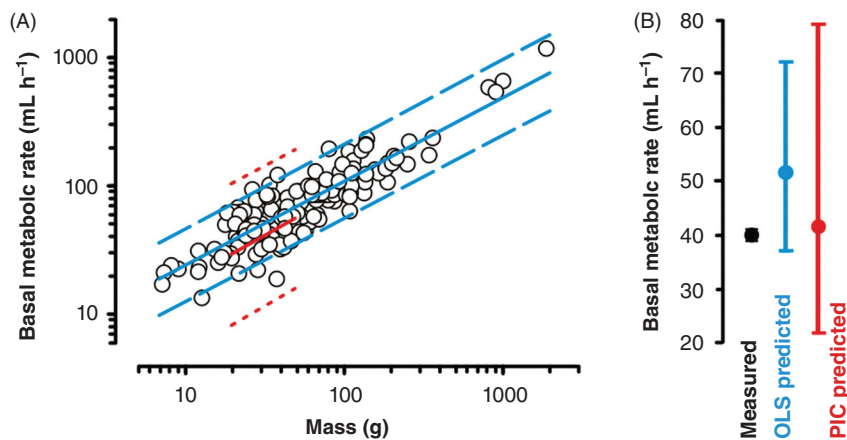
Figure 9 (A) scaling of basal metabolic for 148 species of murid rodent. Data adapted, with permission, from White and Seymour (443) synonymized to match the supertree of Bininda-Emonds et al. (28); see White et al. (432) for details. Solid blue line is the ordinary least squares relationship; dashed blue lines are the 95% prediction interval of the ordinary least squares (OLS) relationship. Solid red line is the relationship for *Notomys alexis* estimated by independent contrasts (PIC) (110) following Garland and Ives (128); dotted red lines are the 95% prediction interval for the phylogenetically informed regression. (B) BMR measured for 11 individual *Notomys alexis* (mean mass 33 g) using indirect calorimetry (440) shown ± SEM and compared with predicted BMR for a 33 g murid rodent (shown ± SEE) for the OLS and PIC regressions presented in (A). Error bounds of BMR value predicted by OLS encompass the measured value, but absolute BMR is overestimated by 29%, and the OLS relationship estimates BMR with considerable uncertainty. The PIC estimate of BMR for *Notomys alexis* is more accurate and overestimates BMR by only 4%, an error similar to the measurement error associated with experimental determination of metabolic rate by indirect calorimetry (e.g., 439), but the error bounds associated with the PIC estimate of BMR are wider than those of OLS. Note that the error bars for predicted BMR are asymmetric because of back-transformation from log-transformed data.

difficult or impossible to measure, such as the body mass of sharks (279) and extinct species (10, 136, 358). Making such predictions based on scaling relationships seems reasonable, because body mass typically accounts for most of the interspecific variation in a range of traits (e.g., metabolic rate: 429, 441, 445). It is important to recognize, however, that the coefficient of determination r^2 can be a poor descriptor of the goodness of fit of scaling relationships (76, 261, 314, 378); for a given magnitude of residual variation, r^2 increases with both the slope of a relationship and the X-range of the data, and high values of r^2 can conceal surprisingly large amounts of residual variation. For example, for the murid rodents in Figure 9A, BMR varies 84-fold from 13.5 mL h^{-1} in least gerbils *Gerbillus pusillus* weighing 12.6 g (47) to 1141 mL h^{-1} in Gambian pouched rats *Cricetomys gambianus* that weigh 1.9 kg (208). Among the 148 species of Muridae for which data are available, the coefficient of determination $r^2 = 0.79$, so mass alone explains 79% of the variation in BMR. Thus, just over one-fifth of the variation in BMR remains unexplained. This seems a relatively small amount, but is actually considerable. For example, fat mice *Steatomys pratensis* and silver mountain voles *Alticola argentatus* both have a mean body mass of 38 g, yet their BMRs differ by over sixfold (103, 416). When larger mass ranges are considered, it is not uncommon for mass to explain over 95% of the variation in metabolic rate, yet large residual differences between species are observed at all body masses and amount to several fold

in endotherms (56, 444), and over an order of magnitude in ectotherms (250, 441).

Smith (379) and McNab (261) suggest that the standard error of estimate ($s_{Y·X}$) provides a better measure of residual variation than the coefficient of nondetermination ($1 - r^2$), which expresses the proportion of variance of a variable that is not explained by another variable (383). $s_{Y·X}$ is calculated from the residual mean square $s^2_{Y·X}$:

$$s^2_{Y·X} = \frac{\sum (Y_i - \hat{Y}_i)^2}{n - 2}$$

The standard error of estimate provides an overall indication of the accuracy with which the fitted regression function predicts the dependence of Y on X (456), and has desirable properties not shared by the coefficient of determination: for a given magnitude of residual variation, r^2 increases with the mass range of the sample, whereas $s_{Y·X}$ does not (Fig. 10). Quantification of residual variation in terms of standard error of estimate ($s_{Y·X}$) and residual mean square ($s^2_{Y·X}$) also permits calculation of the error associated with values of Y predicted by X. The standard error of a predicted value of Y for a given value of X is (383):

$$s_{\hat{Y}} = \sqrt{s^2_{Y·X}\left[1 + \frac{1}{n} + \frac{(X_i - \overline{X})^2}{\sum x^2}\right]}$$
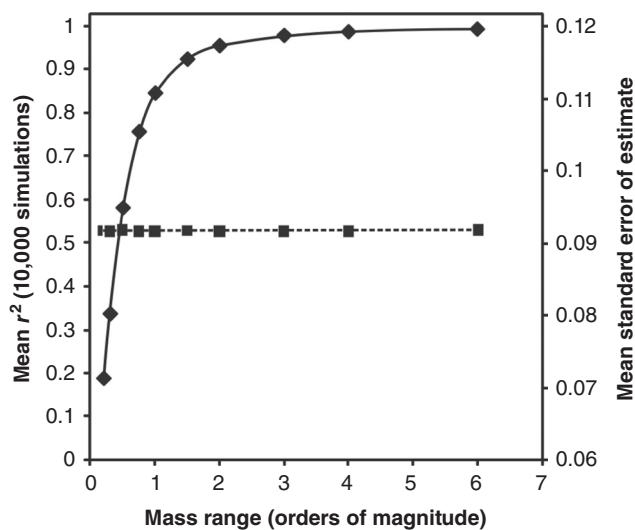
Figure 10 Mean coefficient of determination (r^2, filled diamonds and solid line) and standard error of estimate (filled squares and dashed line) for 10,000 scaling exponents (b, where $Y = aM^b$) calculated for mass ranges of 0.2 to 6 orders of magnitude. For each of the 10,000 simulations, 100 values of log(M) spanning the appropriate range were randomly generated and values of Y were calculated as $M^{0.75}$ plus a normal deviate with a mean of 0 and a standard deviation equal to 20% of $M^{0.75}$. The value of b was then calculated as the slope of the relationship between log(Y) and log(M). For a given quantity of residual variation, the coefficient of determination increases with mass range but the standard error of estimate does not.

where

$$\sum x^2 = \sum (X_i - \overline{X})^2 = \sum X_i^2 - \frac{\left(\sum X_i\right)^2}{n}$$

The error associated with estimates of Y from a regression is rarely calculated, but this error can be very large and the estimates themselves can be rather inaccurate when compared to measured data (Fig. 9B). In many cases, the precision and accuracy of allometric estimates can be improved by incorporating a phylogenetic perspective (e.g., Fig. 9B), because closely related species are often more similar than distantly related ones (see 128 for further details and examples).

A further risk associated with estimates based on allometric relationships is that linear regressions of log-log transformed data estimate the arithmetic mean of log(Y), which is geometric mean of Y. This introduces bias into estimates of Y, which must be accounted for if the objective is to predict Y rather than log(Y) (163). As discussed above, a possible solution to this problems is to fit the power equation $Y = aM^b$ directly, using nonlinear estimation without log-log transformation (e.g., 300, 304). Such an approach may not be suitable for all data sets, however, particularly when the range of body masses considered extends over several orders of magnitude (429). In such cases, linear regression can be used to estimate log(Y) and a range of methods are available to adjust for the bias introduced by this procedure (163).

Comparing newly measured to existing data

When comparing newly obtained data for a species to those available for other species, a common approach is to compare the measured value to that predicted from a scaling relationship. The percentage of the species' value relative to the "expected" value from the allometric equation for an animal of equivalent mass is then often interpreted to be relatively high (somewhat greater than 100%), relatively low (somewhat less than 100%), or approximately as expected (around 100%) (74). Such qualitative comparisons are potentially valuable, but are necessarily subjective because it is not clear how high or low new data need to be before the difference is important. For example, it has previously been hypothesized that mammals of >200 g body mass that feed extensively on termites have metabolic rates lower than predicted from body mass (260, 263), presumably because a termite diet has a low net energy yield (1, 260, 332). Indeed, comparison of the BMRs of termitivorous numbats *Myrmecobius fasciatus* and aardwolves *Proteles cristatus* to the BMR predicted on the basis of their body mass reveals that numbats and aardwolves have BMRs that are 83.6% and 74.2% of that predicted by their respective body masses on the basis of scaling relationships for dasyurid marsupials and Carnivora, respectively (74). Similarly, the FMR of short-beaked echidnas *Tachyglossus aculeatus* varies from ~25% to 50% of that expected on the basis of body mass (38, 147, 288, 354). To determine if such differences are statistically significant; however, it is necessary to establish the 95% prediction confidence limits (74, 383, 456).

The 95% prediction confidence limits of a predicted value of Y ($\hat{Y}$) are calculated from the standard error of a predicted value of Y ($S_{\hat{Y}}$) as:

$$\hat{Y} \pm t_{\alpha(2),(n-2)} S_{\hat{Y}}$$

where t is the critical t value and α is the probability value (e.g., 0.05) for a two-tail t test for $n-2$ degrees of freedom (see also 124, 128 for phylogenetically informed implementations of this approach). If the newly measured value falls outside of the 95% prediction interval, then it can be considered to be significantly different from the remaining data at the specified α level. Prediction intervals tend to be wide (e.g., Fig. 9A), and are much wider than the 95% confidence interval of the regression mean. Thus, the BMRs of numbats and aardwolves are not significantly lower than those of other mammals (74), and a murid rodent weighing 33 g would need to have a BMR lower than 52% of that predicted by mass to be significant different from the remaining murids (Fig. 9A). The power of such comparisons can be increased when data for multiple species are available (e.g., a comparison of multiple ant and termite eating species with other mammals), and groups of species are compared (see "*Comparing scaling relationships*," above).

Metabolic Scaling

Perhaps the most widely examined physiological scaling relationship, and certainly the most controversial, is that between metabolic rate and body mass. Measures of metabolic rate integrate a wide variety of functions performed by animals (42, 155, 213, 390). Animals expend energy for many processes including the maintenance of homeostasis, foraging for and digesting their food, to cover overhead costs of processes such as growth, and to search for mates and reproduce. All of these aforementioned processes involve energy being used to do metabolic work (rather than being stored in the body as new tissue including growth or reproduction) and, therefore, contribute to the metabolic rate. Variation in metabolic rate is therefore linked to Darwinian fitness (see, e.g., reviews: 29, 50, 55, 211, 437). In larval radiated shanny *Ulvaria subbifurcata* and juvenile garden snails *Helix aspersa*, for example, standard metabolic rate (SMR, the metabolic rate of an inactive nonreproductive postabsorptive ectotherm measured at a known temperature during its inactive phase: 115, 314) is under a combination of directional and stabilizing selection such that individuals with low and intermediate metabolic rates are favored over those with high metabolic rates (13, 35). Low BMR improves starvation resistance in rats (338), but individual cockroaches *Nauphoeta cinerea* with low SMR do not live longer than individuals with high BMR under conditions of food and water restriction (351). Individual male Leach's storm-petrel *Oceanodroma leucorrhea* with low BMR breed earlier and produce offspring that grow faster than individuals with high BMR (33), and individual female cockroaches with low SMR have shorter gestation durations than those with high BMR (352). BMR is also associated with over-winter survival in some, but not all, species (36, 37, 193, 224), and interindividual differences in metabolic rate are associated with differences in behavior (29). Metabolic rate has also been hypothesized to underlie a range of ecological patterns (9, 42, 46, 215, 273, 274, 285).

Metabolic rate is usually measured in a laboratory setting by direct calorimetry as heat production (201) or by indirect calorimetry as oxygen consumption or carbon dioxide production (228, 229, 417, 453). For free-living animals, metabolic rate is typically measured using either doubly labeled water (51, 370, 384) or heart rate (51, 148). Measurements of metabolic rate are now available for several thousand species (250, 385, 412), and studies of the scaling of metabolic rate have a history going back almost 200 years. The earliest work (Sarrus and Rameaux, 1838, cited in 39) suggested that because the heat produced as a by-product of metabolism must ultimately be lost through the body surface, the scaling exponent of metabolic rate should be similar to that of body surface area ($b = 0.67$, Fig. 1). This hypothesis was later supported by Rubner's (346) measurements of heat production and body surface area in dogs, and a number of subsequent studies reported a scaling exponent close to 0.67 for the BMR of endotherms (168, 170, 221, 256, 315, 443).

Beginning with Kleiber (206, 207) and Brody and Proctor (40), however, other studies reported that metabolic rate scaled with an exponent greater than 0.67 in a wide array of animals (e.g., 23, 39, 167, 350), and by the 1980s, there was a broad consensus that metabolic rate scaled, on average, with an exponent of 0.75 (52, 314, 357). The perceived ubiquity of quarter-power scaling in turn led to the search for explanations for the origin of this non-Euclidean scaling (e.g., 214, 249, 421, 419), with the potential that explanations for metabolic scaling might also underlie scaling patterns observed in biochemical, physiological, and ecological systems (e.g., 42, 418, 422). The most recent phylogenetically informed studies of endotherms, however, have rejected both 0.67 and 0.75 as universal scaling exponents for these animals, and have stressed that no single scaling exponent adequately describes the mass dependence of metabolic rate (53, 255, 374, 432). Other studies have demonstrated that when plotted on log-log axes, the relationship between mass and metabolic rate for mammals is actually curved, such that the scaling exponent of metabolic rate increases with size (71, 95, 138, 165, 210, 309, 429).

Variation in the scaling exponent of metabolic rate is not limited to a single group of organisms, or to the resting state. The scaling exponent of metabolic rate differs between endothermic and ectothermic animals (69, 107, 315, 434, 441), and the scaling exponent for prokaryotes is higher than for other organisms (87). The scaling exponent of metabolic rate is also typically higher during exercise than during rest (140, 139, 141, 143, 205, 341, 412, 434, 449), and varies with habitat, phylogenetic affinity, and developmental mode in mammals (240, 283, 432, 444), with captivity in birds (255), with laboratory acclimation in scorpions (402), with lifestyle and temperature in fish (204), and with a range of other factors (138, 143, 437). A consequence of the variation in the scaling exponents for $\dot{V}O_2max$, FMR, and BMR (Fig. 11) is that aerobic scope ($= \dot{V}O_2max$ divided by BMR) tends to increase with size (30, 48), whereas activity scope ($=$ FMR divided by BMR) tends to decrease (423). Thus, the largest animals have the greatest capacity to increase metabolic rate above basal levels, but free-living large animals routinely operate at metabolic levels only a few fold higher than basal (Fig. 12). Humphries and Careau (187) used scaling relationships to predict the influences of allometric scaling, air temperature, and mode of activity on the extent to which heat generated as a by-product of activity could be substituted for heat generated for thermoregulation. They predicted that the opportunity for heat substitution should increase with body size, leading to more similar metabolic rates between active and resting rate in large than small animals and thereby perhaps explaining the triangular pattern observed in Figure 12.

Mechanistic theories for metabolic scaling

The methods for scaling analyses presented thus far have focused largely on describing patterns of variation between physiological traits and body mass. Such exploratory analyses are valuable, because they document the association between
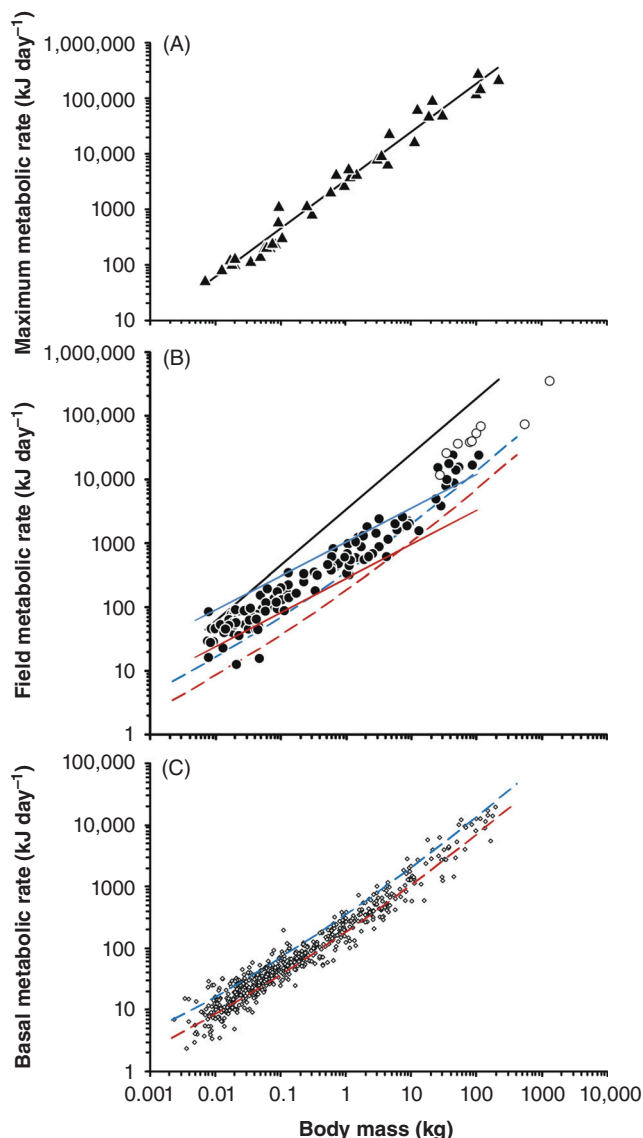
Figure 11   Scaling of (A) maximum metabolic rate ($\dot{V}O_2$max, filled diamonds and solid black line), (B) field metabolic rate (FMR, filled and unfilled circles, blue and red solid lines), and (C) basal metabolic rate (BMR, unfilled diamonds, blue and red dashed lines) with body mass (M) for mammals. $\dot{V}O_2$max = 3300 $M^{0.87}$ (see also 93 for a recent analysis including additional species that reported a scaling exponent of 0.85, data adapted, with permission, from 445). Data for FMR are shown for terrestrial (filled circles) and aquatic (unfilled circles) animals (2, 289, 385); blue and red solid lines are maximum sustained metabolic rates predicted by the Heat Dissipation Limit theory for endotherms at 10 and 30°C, respectively (385). Blue and red dashed lines in (B) and (C) are predicted BMR for tropical/xeric/desert and widespread species, respectively, and are curvilinear on log-log axes (429).

mass and the traits of interest and provide predictive equations that can be used to estimate the value of traits based on measurements of only body mass. Such descriptions do not, however, tell us *why* body mass is associated with traits in the manner that it is. For the case of metabolic rate, a wide range of mechanistic hypotheses have been proposed, beginning with the heat-loss hypothesis proposed by Sarrus and
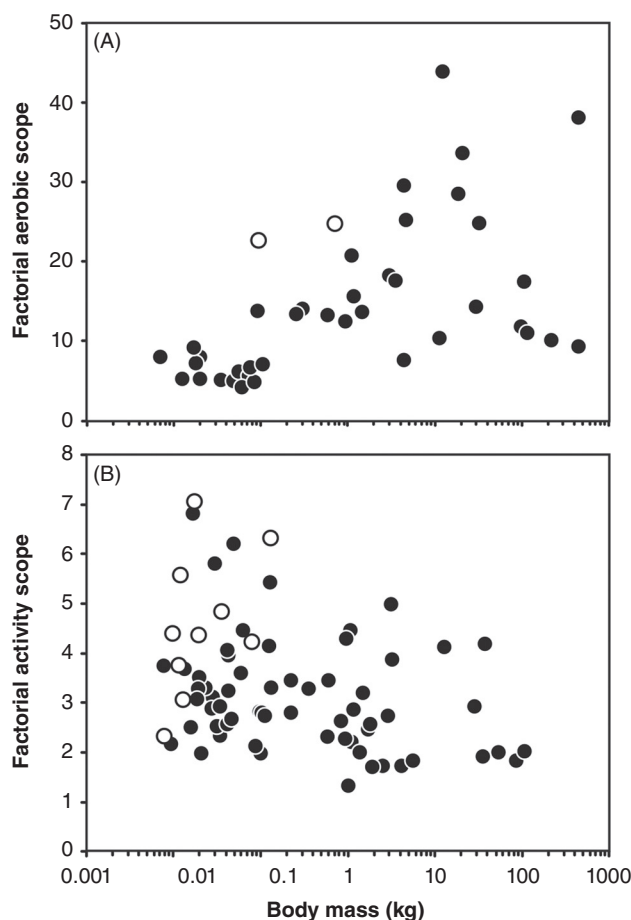

Figure 12   Size dependence of (A) factorial aerobic scope (= maximum aerobic metabolic rate, $\dot{V}O_2$max, divided by basal metabolic rate, BMR) and (B) factorial activity scope (= field metabolic rate, FMR, divided by BMR) for bats (unfilled symbols) and other mammals (filled symbols). Both aerobic and activity scope show a triangular pattern, with a wider range of aerobic scopes observed for large mammals than small ones, and a wider range of activity scopes observed for small animals than large ones. Aerobic and activity scopes were calculated using published data for $\dot{V}O_2$max (198, 234, 412, 445), FMR (385), and BMR (101, 267).

Rameaux that predicts metabolic rate should be proportional to body mass$^{2/3}$ (39, 339, 346). Many of these hypotheses are reviewed and critiqued in detail elsewhere (5, 6, 95, 138, 143, 314), and the debate concerning the factors that cause metabolic rate to scale allometrically with body mass remains one of the most enduring in biology (5, 6, 18-20, 45, 69, 82, 88, 89, 95, 99, 108, 109, 133, 137, 138, 143, 150-152, 168-170, 207, 214, 217-221, 257, 258, 312, 337, 340, 339, 369, 412, 413, 421, 419, 434, 449). Rather than repeat detailed critiques of these theories here, we instead highlight a small number of mechanistic theories that make quantitative predictions about the scaling of metabolic rate and use these to illustrate the difficulties involved in selecting between competing theories. We then discuss methods for evaluating the theories, and demonstrate that these theories can be used to provide a mechanistic basis for ecological and other patterns that are linked to metabolic rate.

Distribution network geometry

Probably the most well-known hypothesis proposed to explain the allometric scaling of metabolic rate is the theory of West and co-workers (418, 419, 422) that predicts the allometric scaling of metabolic rate based on the minimization of transport costs through fractally branching networks that distribute oxygen and nutrients. Although the classic prediction for metabolic scaling based on these models is a scaling exponent of $3/4$ (e.g., 419), the authors of the theories acknowledge that variation exists (e.g., 87) and the theory can predict a range of other values of the scaling exponent depending on the geometry of the networks (20, 210, 323, 349). Nonetheless, the canonical prediction of $3/4$-power scaling of metabolic rate continues to be used (e.g., 179, 462). An alternative model of nutrient transport predicts an exponent of 2/3 for the scaling of whole animal metabolic rate (94).

Dynamic energy budget theory

The Dynamic Energy Budget (DEB) theory (213, 214) considers metabolic rate to comprise a weighted sum of three processes: assimilation, growth, and dissipation. The latter category encompasses somatic maintenance (including foraging and locomotion), maturation, maturity maintenance and any overhead costs of reproduction. The theory is based on generalized surface area (source) and volume (sink) relationships, with body mass decomposed into two indirectly measurable state variables, the "reserve" and the "structure." The composition of reserve and structure are assumed to remain constant (the "strong homeostasis" assumption) but may differ from each other. This compositional assumption enables a complete elemental analysis of the mass budget in terms of "macro-chemical equations" and provides a mechanistic underpinning to indirect calorimetry. Under constant food, the ratio of reserve to structure remains constant and hence so does the entire body composition (the "weak homeostasis" assumption). Energy and matter are assimilated in proportion to structural surface area (e.g., of cells or the gastrointestinal tract), and directed first to the reserve pool of the organism before being mobilized and allocated in fixed proportions to (i) the growth and maintenance of structure and (ii) maturation and maintenance of maturity, and to reproduction once puberty is reached.

A important distinction between DEB and other theories, and one that can be used to design empirical tests among DEB and other theories (202), is that the mechanisms invoked to explain intraspecific scaling relationships are different to those that explain interspecific scaling. Interspecifically, allometric scaling arises because the contribution of nonrespiring reserves to body mass increases with body size. For example, body fat (which is not strictly equivalent to reserve) scales as $M^{1.19}$ in mammals (52, 316) and has a very low mass-specific metabolic rate (102). Intraspecific metabolic rate, on the other hand, varies with size as a consequence of variation in the relative contributions of assimilation, maintenance,

growth, and maturation during development. DEB theory predicts that metabolic rate scales with exponents between 0.5 and 1 for two-dimensional organisms, and between 2/3 and 1 for three-dimensional organisms, and predicts many other intra- and interspecific scaling relationships in addition to metabolic rate (e.g., 54, 213, 214, 293, 407). Notably, in the limit of infinite mass, DEB theory predicts $3/4$ power scaling, as does the more recent distribution network theory of West and co-workers, but for completely different reasons (214, 249).

Metabolic level boundaries

The metabolic level boundaries (MLB) hypothesis predicts variation in the scaling exponent of metabolic rate based on variation in the relative importance of two boundary constraints between groups and activity levels (138, 143). The boundary constraints are surface-area related effects on fluxes of metabolic resources, wastes, and (or) heat; and volume-related effects on energy use and power production. Volume-related effects scale isometrically with mass and apply at low and high levels of metabolic intensity, whereas surface-area-related constraints scale as mass$^{2/3}$ and are more prominent at intermediate levels of metabolism. Thus the scaling exponent of metabolic rate is predicted to vary in a U- or V-shaped pattern with metabolic intensity, bounded by 1 at low and high levels of metabolism and 2/3 at intermediate levels, in good agreement with data for a wide range of organisms (140, 139-143).

Heat dissipation limits

The heat dissipation limits (HDL) hypothesis predicts that that the daily energy expenditure of endotherms is constrained not by their ability to acquire and process energy but by their capacity to dissipate the heat produced as a by-product of metabolic processes (385). The model builds on an elegant experiment demonstrating that lactating mice increase food intake and milk production when shaved (222), although the importance of heat stress for milk production had been demonstrated previously for large (>50 kg) domestic animals with low surface:area volume ratios (e.g., 31, 122, 385). Expansion of these experimental tests of the HDL theory beyond lactation to include a greater emphasis on exercise would be valuable (e.g., 459, 460); many studies have examined the influence of exercise on heat dissipation (e.g., 198, 233, 248, 321, 353, 399-401), and quantitative examination of this wealth of information may provide a useful basis opportunity for further tests of the HLD hypothesis. The HDL hypothesis predicts the scaling of metabolic rate for free-living endotherms on the basis of size-dependent variation in their maximal capacity to dissipate heat, yielding metabolic scaling exponents ranging from 0.47 to 0.50 (385), which is similar the scaling exponent of 0.46 predicted for the metabolic rate of thermoregulating furred endotherms at an ambient temperature 20°C (317).

Muscle aerobic capacity

During exercise-induced maximal aerobic metabolism most (>90%) metabolic activity is associated with work done by the locomotor muscles and delivery of substrates and oxygen to these (412). Given that most of the oxygen consumed during intense activity is used by the mitochondria of the locomotory musculature, it is reasonable to expect that $\dot{V}O_2$max should scale in proportion to the total volume of mitochondria in the working muscles, and therefore, that the scaling exponent of $\dot{V}O_2$max should approximate that of the scaling exponent of the total volume of mitochondria in the locomotory muscles. Weibel and colleagues (412) compiled estimates of total muscle mitochondrial volume for 11 species varying in size from 20 g to 475 kg, and report a scaling exponent of 0.956, which is very close to the scaling exponent of $\dot{V}O_2$max for the same species (0.962).

Comparing theories for metabolic scaling

How should one select among the set of theories for the allometric scaling of metabolic rate described above? Each clearly provides a good fit to at least some of the metabolic data that they seek to describe, and so by the criteria of fit to data alone each must be considered acceptable, at least for some situations. However, comparing models on the basis of how well they predict available data will fail to distinguish between competing theories that predict the same value of the scaling exponent. For example, the original heat loss hypothesis for the scaling of metabolic rate predicts 2/3-power scaling of the BMR of endotherms; the same prediction is made by an alternative theory based on heat loss (339), a theory based on dimensional analysis and biological similarly (150, 151), two theories based on nutrient supply networks (20, 94), and the MLB hypothesis (138, 143). Similarly, the canonical 3/4-power scaling of metabolic rate can be predicted by the same theory of dimensional analysis and biological similarity, if a small empirical adjustment is made (150-152), as well by other theories based on biological similarity (99), elastic similarity (109, 257), nutrient supply networks (18, 19, 421, 419), and reserve/structure geometry (214, 249), among others. Given that the predictions of theories for metabolic scaling often overlap, it is therefore essential that the fit to data is not the only basis on which the theories are compared. Theories must also be evaluated on the legitimacy of the assumptions that underpin them (202).

A potential additional criterion by which competing explanations for metabolic scaling can be compared is their relative complexity, so that the best of two models that describe a given dataset equally well is the one that describes the data with the smallest number of parameters that must be estimated from the data. Indeed, simple explanations that incorporate a minimum of detail are sometimes regarded as more parsimonious than more complicated ones (461). Such ideas form the basis of several methods of formal model comparison (see 49), and applications of such approaches to the scaling of metabolic rate generally reveal that more complicated models describe the available data better than simple ones (e.g., 190, 436). Again, however, theories that predict the same scaling relationship with the same number of free parameters will be indistinguishable.

Given the problem of distinguishing between competing theories that make overlapping predictions, it is essential that the presentation of theories include clear descriptions of the unique predictions made by the theory, to facilitate tests that distinguish between alternatives (77, 162, 202, 372, 438). Such predictions should emerge from the theory, but will ideally complement the support provided to the theory by observed patterns of metabolic scaling, and will also ideally incorporate some form of experimental manipulation (138). For example, the HDL hypothesis has been tested by manipulating heat loss through fur removal or cold exposure (e.g., 222, 377, 458), the hypothesis that muscle aerobic capacity dictates the scaling of $\dot{V}O_2$max could be tested by measuring the size dependence of training-induced changes in muscle mitochondria and quantitatively examining the consequences of this for variation in the scaling of metabolic rate. At present, the MLB hypothesis (138, 143), has been tested predominantly using comparative data gleaned from the literature, though intraspecific studies have begun (57), and further experimental tests are underway (D.S. Glazier, personal communication). Such tests are sorely needed, as are manipulative tests of the distribution network geometry and DEB theories (202).

A common and usually insurmountable problem associated with tests of explanations for metabolic scaling has been a reliance on correlational approaches to understand the scaling of physiological traits with body mass. This approach precludes examination of the causal effect of mass on the trait of interest. A potential solution to this problem is the examination of scaling relationships for colonial organisms. The size of colonies can be manipulated experimentally and the consequences of the manipulation for scaling relationships can be examined in light of the explicit predictions of competing theories (e.g., 158, 290, 438).

On the metabolic basis of ecology

A valuable research agenda that arose following the publication of the original distribution network geometry models for metabolic scaling over a decade ago (420, 421) was the development of a metabolic theory of ecology (MTE), which attempts to link the size and temperature dependence of individual metabolic rates to size- and temperature-dependent ecological processes at levels of organization from individuals to the biosphere (42, 373). Such links have been recognized for decades (e.g., 43, 314), and an early example of the possible association between metabolism and ecology arises from the observation that of population energy use (W ha^{-1}) is independent of species mass when estimated as the product of metabolic rate (W individual^{-1}, approximately proportional to $M^{3/4}$) and population density (individuals ha^{-1}, approximately proportional to $M^{-3/4}$) (80, 81).

The most prominent MTE is grounded in the first principles explanation for metabolic scaling of the fractal distribution network geometry model of West, Brown and Enquist (421, 419) (WBE hereafter). The WBE-MTE combines the predictions of their model for metabolic scaling with a prediction of the temperature dependence of metabolic rate based on the kinetics of biochemical reactions (134) to arrive and the fundamental equation of WBE-MTE, which describes the size and temperature dependence of metabolic rate with only one free parameter (42). The fundamental equation of WBE-MTE is deliberately simple, yet has been remarkably successful in explaining some ecological patterns (9, 42, 46, 166, 273, 274, 285). Its failure in other cases (8, 84, 161, 282, 331, 344, 403) may stem, at least in part, from violation of assumptions of the basic model (58), or an imprecise description of the effects of temperature and mass on MR (436). Recent work by the proponents of WBE-MTE acknowledges the existence of such variation (e.g., 86, 87), and an interesting avenue for future work is determination of the extent to which incorporation of this variation into the fundamental equation of WBE-MTE improves its predictive power.

While the WBE-based theory is the most well-known MTE, an alternative theory of individual metabolism (DEB) was proposed over 10 years prior to WBE theory, but based on different principles (406). DEB has also been lauded as having the potential to unite hierarchical levels of biological organization (293), and therefore, also represents a possible mechanistic basis for the putative link between individual metabolism and ecological patterns. That a robust MTE can be built upon two very different foundations highlights the important distinction between the general idea behind a MTE (i.e., the hypothesis that metabolic variation underlies ecological patterns and processes) and the particular mechanistic theory that underlies any given MTE (e.g., DEB or WBE). Indeed, any theory that explains the size and temperature dependence of metabolic rate has the potential to form the basis of a robust MTE. Given this, it is essential that the putative mechanistic basis of any MTE is subjected to rigorous experimental testing, again preferably using tests that can distinguish between alternatives (202).

More generally, descriptions of metabolic scaling relationships can be used as a basis for understanding ecological patterns without invoking mechanistic explanations for the scaling relationships themselves. For example, Humphries and Careau (187) used published scaling relationships for the metabolic cost of transport (355) and the HDL model for heat dissipation capacity (385) to predict the influences of body size, ambient temperature, and mode of activity on the extent to which heat generated as a by-product of activity can substitute for thermogenesis. They predicted that, regardless of activity mode, activity-thermoregulatory heat substitution increases with body size and ambient temperature and propose that this offers a simple, null-model alternative to niche-based interpretations of the macroecology of endotherm metabolism (187). Because activity-thermoregulatory heat substitution is predicted to be more common in cold environments than warm ones, the range of energy expenditures observed for animals in warm environments is predicted to be wider than for animals in cold environments, because the energy expenditure of inactive and active animals is similar in the cold but widely divergent in warmer conditions (187). Such a pattern is observed in data for endotherm FMRs (11, 187, 385), and has been suggested to demonstrate that warm low-latitude environments provide a greater variety of feasible metabolic niches than do cool high-latitude environments, and has therefore, been proposed to be a mechanism contributing to latitudinal diversity gradients (11, 70). In contrast to this niche-based interpretation, the analysis of Humphries and Careau (187), based on simple scaling relationships, demonstrates that the latitudinal gradient of variation in endotherm energy expenditure can arise as a simple consequence of temperature dependent activity-thermoregulatory substitution, and does not require an explanation based on hypotheses concerning the variety of available metabolic niches. Such approaches, based on the scaling of physiological traits, have great potential to interface with the continuing documentation of variation in physiological traits over large geographical and temporal scales (Macrophysiology: 68, 131), and can serve to provide a mechanistic underpinning to the understanding of macroecological patterns and to understand and predict the consequences of global change (65, 67).

A complimentary way of viewing the association between metabolism and ecology is not to view ecological patterns as being dictated by variation in metabolic rate, but to view metabolic rate as being shaped by ecological factors. Such an ecological theory of metabolism, as recently proposed by Glazier and colleagues (138, 143, 144, 204), emphasizes that both intrinsic and extrinsic influences on metabolic rate, and the interaction among them, must be considered in any synthetic theory of the scaling of metabolic rate. Certainly, there are many examples where biotic and abiotic variables influence metabolic rate and the scaling thereof (see 138 for a comprehensive review); recent examples include (i) the hypothesis that lifestyle influences the scaling of metabolic rate in fish because differences in lifestyle are associated with differential investment in energetically expensive tissues as required for swimming during predator-prey interactions (204); (ii) the observation that predation influences the growth rate and the intraspecific scaling of metabolic rate in amphipods (144); (iii) the suggestion that an association between catchment land cover and the scaling of FMR in freshwater crayfish is mediated by among site differences in the availability and quality of food (254); and (iv) the hypothesis that the allometric scaling of metabolic rate in marine bryozoans may mean that populations experiencing high rates of habitat fragmentation have greater energy requirements than populations experiencing lower rates of fragmentation (438). Such efforts to understand how ecology shapes metabolism will benefit from consideration of metabolic theories, because these aim to capture physical constraints on metabolic possibilities, and provide null expectations for scaling relationships.

Conclusion

The history of the quantitative study of allometric scaling spans well over a century of investigation, and has encompassed many traits. Throughout much of this period, scaling exponents relating physiological variables to body mass have been determined using ordinary least-squares linear regression of log-log transformed data. During recent years it has become clear that physiological data often violate the assumptions of such an approach, which is appropriate only when data are independent and conform to a two-parameter power relationship with multiplicative error on the arithmetic scale. However, many traits show significant phylogenetic signal, indicating that a phylogenetic perspective is necessary when analyzing such data. Some traits do not conform to a two-parameter power function with multiplicative error, and alternative models including nonlinear regression of untransformed data, three-parameter power functions, and polynomial relationships should be considered for such data. Such approaches have been valuable in describing the scaling of metabolic rate, which remains one of the most widely investigated scaling relationships. The scaling exponent of metabolic rate varies between approximately 0.5 and 1, and varies with activity level as well as between endotherms and ectotherms. A number of explanations for the allometric scaling of metabolic rate have been proposed, but none has gained widespread acceptance. Choosing between these explanations is difficult, because they generally predict similar values of the scaling exponent, and manipulative experiments that test among the unique predictions of the explanations are sorely required.

Acknowledgements

References

1. Abensperg-Traun M, Dickman CR, Boer ESD. Patch use and prey defence in a mammalian myrmecophage, the echidna (*Tachyglossus aculeatus*) (Monotremata: Tachyglossidae): A test of foraging efficiency in captive and free-ranging animals. *J Zool* 225: 481-493, 1991.
2. Acquarone M, Born EW, Speakman JR. Field metabolic rates of walrus (*Odobenus rosmarus*) measured by the doubly labeled water method. *Aquat Mamm* 32: 363-369, 2006.
3. Adams DC, Church JO. Amphibians do not follow Bergmann's rule. *Evolution* 62: 413-420, 2008.
4. Addo-Bediako A, Chown SL, Gaston KJ. Metabolic cold adaptation in insects: A large-scale perspective. *Funct Ecol* 16: 332-338, 2002.
5. Agutter PS, Tuszynski JA. Analytic theories of allometric scaling. *J Exp Biol* 214: 1055-1062, 2011.
6. Agutter PS, Wheatley DN. Metabolic scaling: Consensus or controversy? *Theor Biol Med Model* 1: 13 (http://www.tbiomed.com/content/1/1/13), 2004.
7. Alexander RM. *Locomotion of Animals*. Glasgow: Blackie, 1982.
8. Algar AC, Kerr JT, Currie DJ. A test of Metabolic theory as the mechanism underlying broad-scale species-richness gradients. *Glob Ecol Biogeogr* 16: 170-178, 2007.
9. Allen AP, Brown JH, Gillooly JF. Global biodiversity, biochemical kinetics, and the energetic-equivalence rule. *Science* 297: 1545-1548, 2002.
10. Anderson JF, Hall-Martin A, Russell DA. Long-bone circumference and weight in mammals, birds and dinosaurs. *J Zool* 207: 53-61, 1985.
11. Anderson KJ, Jetz W. The broad-scale ecology of energy expenditure of endotherms. *Ecol Lett* 8: 310-318, 2005.
12. Arens JR, Sheldon JC. Seasonal and diurnal variation in metabolism and ventilation in house sparrows. *The Condor* 107: 433-444, 2005.
13. Artacho P, Nespolo RF. Natural selection reduces energy metabolism in the garden snail, *Helix aspersa* (*Cornu aspersum*). *Evolution* 63: 1044-1050, 2009.
14. Ashton KG. Patterns of within-species body size variation of birds: Strong evidence for Bergmann's rule. *Glob Ecol Biogeogr* 11: 505-523, 2002.
15. Ashton KG, Tracy MC, de Queiroz A. Is Bergmann's rule valid for mammals? *Am Nat* 156: 390-415, 2000.
16. Badeer HS. Is the flow in the giraffe's jugular vein a "free" fall? *Comp Biochem Physiol A* 118: 573-576, 1997.
17. Banavar JR, Damuth J, Maritan A, Rinaldo A. Allometric cascades. *Nature* 421: 713-714, 2003.
18. Banavar JR, Damuth J, Maritan A, Rinaldo A. Supply-demand balance and metabolic scaling. *Proc Natl Acad Sci U S A* 99: 10506-10509, 2002.
19. Banavar JR, Maritan A, Rinaldo A. Size and form in efficient transportation networks. *Nature* 399: 130-131, 1999.
20. Banavar JR, Moses ME, Brown JH, Damuth J, Rinaldo A, Sibly RM, Maritan A. A general basis for quarter-power scaling in animals. *Proc Natl Acad Sci U S A* 107: 15816-15820, 2010.
21. Barclay RMR. Constraints on reproduction by flying vertebrates: Energy and calcium. *Am Nat* 144: 1021-1031, 1994.
22. Bednekoff PA, Biebach H, Krebs J. Great tit fat reserves under unpredictable temperatures. *J Avian Biol* 25: 156-160, 1994.
23. Benedict FG. *Vital Energetics: A Study in Comparative Basal Metabolism*. Washington, D.C.: Carnegie Institution of Washington, 1938.
24. Bergmann C. Ueber die Verhältnisse der Wärmeökonomie der Thiere zu ihrer Grösse. *Göttinger Studien* 3: 595-708, 1847.
25. Berke SK, Jablonski D, Krug AZ, Roy K, Tomasovych A. Beyond Bergmann's rule: Size–latitude relationships in marine Bivalvia worldwide. *Glob Ecol Biogeogr* 22: 173-183, 2012.
26. Bertram JEA, Biewener AA. Differential scaling of the long bones in the terrestrial carnivora and other mammals. *J Morphol* 204: 157-169, 1990.
27. Bininda-Emonds ORP. The evolution of supertrees. *Trends Ecol Evol* 19: 315-322, 2004.
28. Bininda-Emonds ORP, Cardillo M, Jones KE, MacPhee RDE, Beck RMD, Grenyer R, Price SA, Vos RA, Gittleman JL, Purvis A. The delayed rise of present-day mammals. *Nature* 446: 507-512, 2007.
29. Biro PA, Stamps JA. Do consistent individual differences in metabolic rate promote consistent individual differences in behavior? *Trends Ecol Evol* 25: 653-659, 2010.
30. Bishop CM. The maximum oxygen consumption and aerobic scope of birds and mammals: Getting to the heart of the matter. *Proc Roy Soc B-Biol Sci* 266: 2275-2281, 1999.
31. Black JL, Mullan BP, Lorschy ML, Giles LR. Lactation in the sow during heat stress. *Livest Prod Sci* 35: 153-170, 1993.
32. Blackburn TM, Hawkins BA. Bergmann's rule and the mammal fauna of northern North America. *Ecography* 27: 715-724, 2004.
33. Blackmer AL, Mauck RA, Ackerman JT, Huntington CE, Nevitt GA, Williams JB. Exploring individual quality: Basal metabolic rate and reproductive performance in storm-petrels. *Behav Ecol* 16: 906-913, 2005.
34. Blomberg SP, Garland T, Jr, Ives AR. Testing for phylogenetic signal in comparative data: Behavioral traits are more labile. *Evolution* 57: 717-745, 2003.
35. Bochdansky AB, Grønkjær P, Herra TP, Leggett WC. Experimental evidence for selection against fish larvae with high metabolic rates in a food limited environment. *Mar Biol* 147: 1413-1417, 2005.
36. Boratyński Z, Koskela E, Mappes T, Oksanen TA. Sex-specific selection on energy metabolism – selection coefficients for winter survival. *J Evol Biol* 23: 1969-1978, 2010.
37. Boratyński Z, Koteja P. The association between body mass, metabolic rates and survival of bank voles. *Funct Ecol* 23: 330-339, 2009.
38. Brice PH. Thermoregulation in monotremes: Riddles in a mosaic. *Aust J Zool* 57: 255-263, 2009.
39. Brody S. *Bioenergetics and growth*. New York: Reinhold Publishing Corporation, 1945, p. 1023.

40. Brody S, Proctor RC. Relation between basal metabolism and mature body weight in different species of mammals and birds. *Univ. Missouri Agric. Exp. Stn. Res. Bull.* 166: 89-101, 1932.

41. Brøndum E, Hasenkam JM, Secher NH, Bertelsen MF, Grøndahl C, Petersen KK, Buhl R, Aalkjær C, Baandrup U, Nygaard H, Smerup M, Stegmann F, Sloth E, Østergaard KH, Nissen P, Runge M, Pitsillides K, Wang T. Jugular venous pooling during lowering of the head affects blood pressure of the anesthetized giraffe. *Am J Physiol* 297: R1058-R1065, 2009.

42. Brown JH, Gillooly JF, Allen AP, Savage VM, West GB. Toward a metabolic theory of ecology. *Ecology* 85: 1771-1789, 2004.

43. Brown JH, Marquet PA, Taper ML. Evolution of body size: Consequences of an energetic definition of fitness. *Am Nat* 142: 573-584, 1993.

44. Brown JH, West GB editors. *Scaling in Biology*. New York: Oxford University Press, 2000, p. 352.

45. Brown JH, West GB, Enquist BJ. Yes, West, Brown and Enquist's model of allometric scaling is both mathematically correct and biologically relevant. *Funct Ecol* 19: 735-738, 2005.

46. Buckley LB, Rodda GH, Jetz W. Thermal and energetic constraints on ectotherm abundance: A global test using lizards. *Ecology* 89: 48-55, 2008.

47. Buffenstein R, Jarvis JUM. Thermoregulation and metabolism in the smallest African gerbil, *Gerbillus pusillus*. *J Zool* 205: 107-121, 1985.

48. Bundle MW, Hoppeler H, Vock R, Tester JM, Weyand PG. High metabolic rates in running birds. *Nature* 397: 31-32, 1999.

49. Burnham KP, Anderson DR. *Model Selection and Multi-Model Inference: A Practical Information-Theoretic Approach*. New York: Springer, 2010, p. 488.

50. Burton T, Killen SS, Armstrong JD, Metcalfe NB. What causes intraspecific variation in resting metabolic rate and what are its ecological consequences? *Proc R Soc Lond B Biol Sci* 278: 3465-3473, 2011.

51. Butler PJ, Green JA, Boyd IL, Speakman JR. Measuring metabolic rate in the field: The pros and cons of the doubly labelled water and heart rate methods. *Funct Ecol* 18: 168-183, 2004.

52. Calder WA, III. *Size, Function, and Life History*. Cambridge: Harvard University Press, 1984, p. 431.

53. Capellini I, Venditti C, Barton RA. Phylogeny and metabolic scaling in mammals. *Ecology* 91: 2783-2793, 2010.

54. Cardoso JFMF, van der Veer HW, Kooijman SALM. Body-size scaling relationships in bivalve species: A comparison of field data with predictions by the Dynamic Energy Budget (DEB) theory. *J Sea Res* 56: 125-139, 2006.

55. Careau V, Garland T, Jr. Performance, personality, and energetics: Correlation, causation, and mechanism. *Physiol Biochem Zool* 85: 543-571, 2012.

56. Careau V, Thomas D, Humphries MM, Réale D. Energy metabolism and animal personality. *Oikos* 117: 641-653, 2008.

57. Carey N, Sigwart JD, Richards JG. Economies of scaling: More evidence that allometry of metabolism is linked to activity, metabolic rate and habitat. *J Exp Mar Biol Ecol* 439: 7-14, 2013.

58. Cassemiro FAS, Diniz-Filho JAF. Deviations from predictions of the metabolic theory of ecology can be explained by violations of assumptions. *Ecology* 91: 3729-3738, 2010.

59. Caviedes-Vidal E, McWhorter TJ, Lavin SR, Chediack JG, Tracy CR, Karasov WH. The digestive adaptation of flying vertebrates: High intestinal paracellular absorption compensates for smaller guts. *Proc Natl Acad Sci U S A* 104: 19132-19137, 2007.

60. Cawley GC, Janacek GJ. On allometric equations for predicting body mass of dinosaurs. *J Zool* 280: 355-361, 2010.

61. Chappell R. Fitting bent lines to data, with applications to allometry. *J Theor Biol* 138: 235-256, 1989.

62. Cheverud JM. Relationships among ontogenetic, static, and evolutionary allometry. *Am J Phys Anthropol* 59: 139-149, 1982.

63. Cheverud JM, Dow MM. An autocorrelation analysis of genetic variation due to lineal fission in social groups of rhesus macaques. *Am J Phys Anthropol* 67: 113-121, 1985.

64. Cheverud JM, Dow MM, Leutengger W. The quantitative assessment of phylogenetic constraints in comparative analyses: Sexual dimorphism in body weight among primates. *Evolution* 39: 1335-1351, 1985.

65. Chown SL, Gaston KJ. Macrophysiology for a changing world. *Proc Roy Soc B-Biol Sci* 275: 1469-1478, 2008.

66. Chown SL, Gaston KJ. Body size variation in insects: A macroecological perspective. *Biol Rev* 85: 139-169, 2010.

67. Chown SL, Gaston KJ, Kleunen M, Clusella-Trullas S. Population responses within a landscape matrix: A macrophysiological approach to understanding climate change impacts. *Evol Ecol* 24: 601-616, 2010.

68. Chown SL, Gaston KJ, Robinson D. Macrophysiology: Large-scale patterns in physiological traits and their ecological implications. *Funct Ecol* 18: 159-167, 2004.

69. Chown SL, Marais E, Terblanche JS, Klok CJ, Lighton JRB, Blackburn TM. Scaling of insect metabolic rate is inconsistent with the nutrient supply network model. *Funct Ecol* 21: 282-290, 2007.

70. Clarke A, Gaston KJ. Climate, energy and diversity. *Proc Roy Soc B-Biol Sci* 273: 2257-2266, 2006.

71. Clarke A, Rothery P, Isaac NJB. Scaling of basal metabolic rate with body mass and temperature in mammals. *J Anim Ecol* 79: 610-619, 2010.

72. Clemente CJ, Thompson GG, Withers PC. Evolutionary relationships of sprint speed in Australian varanid lizards. *J Zool* 278: 270-280, 2009.

73. Cochran WG. Analysis of covariance: Its nature and uses. *Biometrics* 13: 261-281, 1957.

74. Cooper CE, Withers PC. Numbats and aardwolves—how low is low? A re-affirmation of the need for statistical rigour in evaluating regression predictions. *J Comp Physiol A* 176: 623-629, 2006.

75. Cresswell W. Diurnal and seasonal mass variation in blackbirds *Turdus merula*: Consequences for mass-dependent predation risk. *J Anim Ecol* 67: 78-90, 1998.

76. Currie DJ. What shape is the relationship between body size and population density? *Oikos* 66: 353-358, 1993.

77. Currie DJ, Mittelbach GG, Cornell HV, Field R, Guégan J-F, Hawkins BA, Kaufman DM, Kerr JT, Oberdorff T, O'Brien E, Turner JRG. Predictions and tests of climate-based hypotheses of broad-scale variation in taxonomic richness. *Ecol Lett* 7: 1121-1134, 2004.

78. D'Alonzo KT. The Johnson-Neyman procedure as an alternative to ANCOVA. *Western J Nurs Res* 26: 804-812, 2004.

79. Daan S, Masman D, Strijkstra A, Verhulst S. Intraspecific allometry of basal metabolic rate: Relations with body size, temperature, composition, and circadian phase in the kestrel, *Falco tinnunculus*. *J Biol Rhythms* 4: 267-283, 1989.

80. Damuth J. Interspecific allometry of population density in mammals and other animals: The independence of body mass and population energy use. *Biol J Linn Soc* 31: 193-246, 1987.

81. Damuth J. Population density and body size in mammals. *Nature* 290: 699-700, 1981.

82. Darveau CA, Suarez RK, Andrews RD, Hochachka PW. Allometric cascade as a unifying principle of body mass effects on metabolism. *Nature* 417: 166-170, 2002.

83. Dawson TJ, Hulbert AJ. Standard metabolism, body temperature, and surface areas of Australian marsupials. *Am J Physiol* 218: 1233-1238, 1970.

84. de Castro F, Gaedke U. The metabolism of lake plankton does not support the metabolic theory of ecology. *Oikos* 117: 1218-1226, 2008.

85. de Queiroz A, Ashton KG. The phylogeny of a species-level tendency: Species heritability and possible deep origins of Bergmann's rule in tetrapods. *Evolution* 58: 1674-1684, 2004.

86. Dell AI, Pawar S, Savage VM. Systematic variation in the temperature dependence of physiological and ecological traits. *Proc Natl Acad Sci U S A* 108: 10591-10596, 2011.

87. DeLong JP, Okie JG, Moses ME, Sibly RM, Brown JH. Shifts in metabolic scaling, production, and efficiency across major evolutionary transitions of life. *Proc Natl Acad Sci U S A* 107: 12941-12945, 2010.

88. Demetrius L. The origin of allometric scaling laws in biology. *J Theor Biol* 243: 455-467, 2006.

89. Demetrius L, Tuszynski JA. Quantum metabolism explains the allometric scaling of metabolic rates. *J Royal Soc Interface* 7: 507-514, 2010.

90. Díaz-Uriarte R, Garland T, Jr. Effects of branch length errors on the performance of phylogenetically independent contrasts. *Syst Biol* 47: 654-672, 1998.

91. Díaz-Uriarte R, Garland T, Jr. Testing hypotheses of correlated evolution using phylogenetically independent contrasts: Sensitivity to deviations from Brownian motion. *Syst Biol* 45: 24-47, 1996.

92. Diniz JAF, De Sant'ana CER, Bini LM. An eigenvector method for estimating phylogenetic inertia. *Evolution* 52: 1247-1262, 1998.

93. Dlugosz EM, Chappell MA, Meek TH, Szafrańska PA, Zub K, Konarzewski M, Jones JH, Bicudo JEPW, Nespolo RF, Careau V, Garland T, Jr. Phylogenetic analysis of mammalian maximal oxygen consumption during exercise. *J Exp Biol* 2013. [Epub ahead of print].

94. Dodds PS. Optimal form of branching supply and collection networks. *Phys Rev Lett* 104: 048702, 2010.

95. Dodds PS, Rothman DH, Weitz JS. Re-examination of the "3/4-law" of metabolism. *J Theor Biol* 209: 9-27, 2001.

96. Dorsey SG, Soeken KL. Use of the Johnson-Neyman technique as an alternative to analysis of covariance. *Nurs Res* 45: 363-366, 1996.

97. Dubois E. Sur le rapport du poids de l'encéphale avec la grandeur du corps chez les mammifères. *Bull Mem Soc Anthrop Paris* 8: 337-376, 1897.

98. Duncan RP, Forsythe DM, Hone J. Testing the metabolic theory of ecology: Allometric scaling exponents in mammals. *Ecology* 88: 324-333, 2007.

99. Economos AC. On the origin of biological similarity. *J Theor Biol* 94: 25-60, 1982.

100. Egger M, Smith GD, Schneider M, Minder C. Bias in meta-analysis detected by a simple, graphical test. *BMJ* 315: 629-634, 1997.

101. Eisenberg JF. *The Mammalian Radiations*. Chicago: The University of Chicago Press, 1981.

102. Elia M. Organ and tissue contributions to metabolic rate. In: Kinney JM, Tucker HN, editors. *Energy Metabolism: Tissue Determinants and Cellular Corollaries*. New York: Raven, 1992.

103. Ellison GTH. Thermoregulatory responses on cold acclimated fat mice (*Steatomys pratensis*). *J Mammal* 76: 240-247, 1995.

104. Engqvist L. The mistreatment of covariate interaction terms in linear model analyses of behavioural and evolutionary ecology studies. *Anim Behav* 70: 967-971, 2005.

105. Fairbairn DJ, Preziosi RF. Sexual selection and the evolution of allometry for sexual size dimorphism in the water strider, *Aquarius remigis*. *Am Nat* 144: 101-118, 1994.

106. Falconer DS, Mackay TFC. *Introduction to Quantitative Genetics*. Edinburgh: Longman, 1996.

107. Farrell-Gray CC, Gotelli NJ. Allometric exponents support a 3/4-power scaling law. *Ecology* 86: 2083-2087, 2005.

108. Feldman HA. On the allometric mass exponent, when it exists. *J Theor Biol* 172: 187-197, 1995.

109. Feldman HA, McMahon TA. The 3/4 mass exponent for energy metabolism is not a statistical artifact. *Respir Physiol* 52: 149-164, 1983.

110. Felsenstein J. Phylogenies and the comparative method. *Am Nat* 125: 1-15, 1985.

111. Fick A. Über die Messung des Blutquantums in den Herzventrikeln. *Verh Phys Med Ges Würzburg* 2: 16, 1870.

112. Finney DJ. Was this in your statistics textbook? V. Transformations of data. *Exp Agric* 25: 165-175, 1989.

113. Foster WK, Taggart DA. Gender and parental influences on the growth of a sexually dimorphic carnivorous marsupial. *J Zool* 275: 221-228, 2008.

114. Franz R, Hummel J, Kienzle E, Kölle P, Gunga H-C, Clauss M. Allometry of visceral organs in living amniotes and its implications for sauropod dinosaurs. *Proc Roy Soc B-Biol Sci* 276: 1731-1736, 2009.

115. Frappell PB, Butler PJ. Minimal metabolic rate, what it is, its usefulness, and its relationship to the evolution of endothermy: A brief synopsis. *Physiol Biochem Zool* 77: 865-868, 2004.

116. Frappell PB, Hinds DS, Boggs DF. Scaling of respiratory variables and the breathing pattern in birds: An allometric and phylogenetic approach. *Physiol Biochem Zool* 74: 75-89, 2001.

117. Freckleton RP. On the misuse of residuals in ecology: Regression of residuals vs. multiple regression. *J Anim Ecol* 71: 542-545, 2002.

118. Freckleton RP, Cooper N, Jetz W. Comparative methods as a statistical fix: The dangers of ignoring an evolutionary model. *Am Nat* 178: E10-E17, 2011.

119. Freckleton RP, Harvey PH, Pagel M. Phylogenetic analysis and comparative data: A test and review of evidence. *Am Nat* 160: 712-726, 2002.

120. Freckleton RP, Jetz W. Space versus phylogeny: Disentangling phylogenetic and spatial signals in comparative data. *Proc Roy Soc B-Biol Sci* 276: 21-30, 2009.

121. Fritz SA, Rahbek C. Global patterns of amphibian phylogenetic diversity. *J Biogeogr* 39: 1373-1382, 2012.

122. Fuquay JW. Heat stress as it affects animal production. *J Anim Sci* 52: 164-174, 1981.

123. García-Berthou E. On the misuse of residuals in ecology: Testing regression residuals vs. the analysis of covariance. *J Anim Ecol* 70: 708-711, 2001.

124. Garland T, Jr, Adolph SC. Why not to do two species comparative studies: Limitations on inferring adaptation. *Physiol Biochem Zool* 67: 797-828, 1994.

125. Garland T, Jr, Bennett AF, Rezende EL. Phylogenetic approaches in comparative physiology. *J Exp Biol* 208: 3015-3035, 2005.

126. Garland T, Jr, Diaz-Uriarte R. Polytomies and phylogenetically independent contrasts: Examination of the bounded degrees of freedom approach. *Syst Biol* 48: 547-558, 1999.

127. Garland T, Jr. The relation between maximal running speed and body mass in terrestrial mammals. *J Zool* 199: 157-170, 1983.

128. Garland T, Jr., Ives AR. Using the past to predict the present: Confidence intervals for regression equations in phylogenetic comparative methods. *Am Nat* 155: 346-364, 2000.

129. Gartner GEA, Hicks JW, Andrade DV, Secor SM, Garland T, Jr. Reply to "Heart position in snakes". *Physiol Biochem Zool* 84: 102-106, 2011.

130. Gartner GEA, Hicks JW, Manzani PR, Andrade DV, Abe AS, Wang T, Secor SM, Garland T, Jr. Phylogeny, ecology, and heart position in snakes. *Physiol Biochem Zool* 83: 43-54, 2010.

131. Gaston KJ, Chown SL, Calosi P, Bernado J, Bilton DT, Clarke A, Clusella-Trullas S, Ghalambor CK, Konarzewski M, Peck LS, Porter WP, Pörtner HO, Rezende EL, Schulte PM, Spicer JI, Stillman JH, Terblanche JS, van Kleunen M. Macrophysiology: A conceptual reunification. *Am Nat* 174: 595-612, 2009.

132. Gillanders BM. Comparison of growth rates between estuarine and coastal reef populations of *Achoerodus viridis* (Pisces: Labridae). *Mar Ecol Prog Ser* 146: 283-287, 1997.

133. Gillooly JF, Allen AP. Changes in body temperature influence the scaling of Vo_2max and aerobic scope in mammals. *Biol Letters* 3: 99-102, 2007.

134. Gillooly JF, Brown JH, West GB, Savage VM, Charnov EL. Effects of size and temperature on metabolic rate. *Science* 293: 2248-2251, 2001.

135. Gingerich PD. Arithmetic or geometric normality of biological variation: An empirical test of theory. *J Theor Biol* 204: 201-221, 2000.

136. Gingerich PD, Smith BH, Rosenberg K. Allometric scaling in the dentition of primates and prediction of body weight from tooth size in fossils. *Am J Phys Anthropol* 58: 81-100, 1982.

137. Ginzburg L, Damuth J. The space-lifetime hypothesis: Viewing organisms in four dimensions, literally. *Am Nat* 171: 125-131, 2008.

138. Glazier DS. Beyond the '3/4-power law': Variation in the intra- and interspecific scaling of metabolic rate in animals. *Biol Rev* 80: 1-52, 2005.

139. Glazier DS. Effects of metabolic level on the body size scaling of metabolic rate in birds and mammals. *P Roy Soc B-Biol Sci* 22: 1405-1410, 2008.

140. Glazier DS. Activity affects intraspecific body-size scaling of metabolic rate in ectothermic animals. *J Comp Physiol B* 179: 821-828, 2009.

141. Glazier DS. Metabolic level and size scaling of rates of respiration and growth in unicellular organisms. *Funct Ecol* 23: 963-968, 2009.

142. Glazier DS. Ontogenetic body-mass scaling of resting metabolic rate covaries with species-specific metabolic level and body size in spiders and snakes. *Comp Biochem Physiol A* 153: 403-407, 2009.

143. Glazier DS. A unifying explanation for diverse metabolic scaling in animals and plants. *Biol Rev* 85: 111-138, 2010.

144. Glazier DS, Butler EM, Lombardi SA, Deptola TJ, Reese AJ, Satterthwaite EV. Ecological effects on metabolic scaling: Amphipod responses to fish predators in freshwater springs. *Ecol Monogr* 81: 599-618, 2011.

145. Gould SJ. Allometry and size in ontogeny and phylogeny. *Biol Rev* 44: 587-640, 1966.

146. Grafen A. The phylogenetic regression. *Philos T R Soc Lon B* 326: 119-157, 1989.

147. Green B, Griffiths M, Newgrain K. Seasonal patterns in water, sodium and energy turnover in free-living echidnas, *Tachyglossus aculeatus* (Mammalia: Monotremata). *J Zool* 227: 351-365, 1992.

148. Green JA. The heart rate method for estimating metabolic rate: Review and recommendations. *Comp Biochem Physiol A* 158: 287-304, 2011.

149. Green JA, White CR, Butler PJ. Allometric estimation of metabolic rate from heart rate in penguins. *Comp Biochem Physiol A* 142: 478-484, 2005.

150. Günther B. Dimensional analysis and theory of biological similarity. *Physiol Rev* 55: 659-699, 1975.

151. Günther B, León de la Barra B. A unified theory of biological similarities. *J Theor Biol* 13: 48-59, 1966.

152. Günther B, Morgado E. Theory of biological similarity revisited. *J Theor Biol* 96: 543-560, 1982.

153. Hadfield JD, Nakagawa S. General quantitative genetic methods for comparative biology: Phylogenies, taxonomies and multi-trait models for continuous and categorical characters. *J Evol Biol* 23: 494-508, 2010.

154. Hails CJ. The metabolic rate of tropical birds. *Condor* 85: 61-65, 1983.

155. Halsey LG. The challenge of measuring energy expenditure: Current field and laboratory methods. *Comp Biochem Physiol A* 158: 247-251, 2011.

156. Halsey LG, Butler PJ, Blackburn TM. A phylogenetic analysis of the allometry of diving. *Am Nat* 167: 276-287, 2006.

157. Halsey LG, White CR, Enstipp MR, Jones DR, Martin GR, Butler PJ. When cormorants go fishing: The differing costs of hunting for sessile and motile prey. *Biol Letters* 3: 574-576, 2007.

158. Hartikainen H, Humphries S, Okamura B. Form and metabolic scaling in colonial animals. *J Exp Biol* doi: 10.1242/jeb.093484, 2013.

159. Harvey PH. On rethinking allometry. *J Theor Biol* 95: 37-41, 1982.

160. Harvey PH, Pagel MD. *The Comparative Method in Evolutionary Biology*. New York: Oxford University Press, 1991, p. 239.

161. Hawkins BA, Albuquerque FS, Araújo MB, Beck J, Bini LM, Cabrero-Sañudo FJ, Castro-Parga I, Diniz-Filho JAF, Ferrer-Castán D, Field R, Gómez JF, Hortal J, Kerr JT, Kitching IJ, León-Cortés JL, Lobo JM, Montoya D, Moreno JC, Ollalla-Tárraga MÁ, Pausas JG, Qian H, Rahbek C, Rodríguez MÁ, Sanders NJ, Williams P. A global evaluation of metabolic theory as an explanation for terrestrial species richness gradients. *Ecology* 88: 1877-1888, 2007.

162. Hawkins BA, Diniz-Filho JAF, Bini LM, Araújo MB, Field R, Hortal J, Kerr JT, Rahbek C, Rodríguez MÁ, Sanders NJ. Metabolic theory and diversity gradients: Where do we go from here? *Ecology* 88: 1898-1902, 2007.

163. Hayes JP, Shonkwiler JS. Allometry, antilog transformations, and the perils of prediction on the original scale. *Physiol Biochem Zool* 79: 665-674, 2006.

164. Hayes JP, Shonkwiler JS. Analyzing mass-independent data. *Physiol Zool* 69: 974-980, 1996.

165. Hayssen V, Lacy RC. Basal metabolic rates in mammals: Taxonomic differences in the allometry of BMR and body mass. *Comp Biochem Physiol A* 81: 741-754, 1985.

166. Hechinger RF, Lafferty KD, Dobson AP, Brown JH, Kuris AM. A common scaling rule for abundance, energetics, and production of parasitic and free-living species. *Science* 333: 445-448, 2011.

167. Hemmingsen AM. Energy metabolism as related to body size and respiratory surfaces, and its evolution. *Rep. Steno Meml Hosp. Nordisk Insulinlab.* 9: 1-110, 1960.

168. Heusner AA. Energy metabolism and body size: 1. Is the 0.75 mass exponent of Kleibers equation a statistical artifact? *Respir Physiol* 48: 1-12, 1982.

169. Heusner AA. Energy metabolism and body size: 2. Dimensional analysis and energetic nonsimilarity. *Respir Physiol* 48: 13-26, 1982.

170. Heusner AA. Size and power in mammals. *J Exp Biol* 160: 25-54, 1991.

171. Heymsfield SB, Childers D, Beetsch J, Allison DB, Pietrobelli A. Body size and human energy requirements: Reduced mass-specific resting energy expenditure in tall adults. *J Appl Physiol* 103: 1543-1550, 2007.

172. Heymsfield SB, Gallagher D, Kotler ZW, Allison DB, Heshka S. Body-size dependence of resting energy expenditure can be attributed to nonenergetic homogeneity of fat-free mass. *Am J Physiol* 282: E132-E138, 2005.

173. Heymsfield SB, Thomas D, Bosy-Westphal A, Shen W, Peterson CM, Müller MJ. Evolving concepts on adjusting human resting energy expenditure measurements for body size. *Obes Rev* 13: 1001-1014, 2012.

174. Hicks JW, Badeer HS. Gravity and the circulation: "open" vs. "closed" systems. *Am J Physiol* 262: R725-R732, 1992.

175. Hicks JW, Badeer HS. Siphon mechanism in collapsible tubes: Application to circulation of the giraffe head. *Am J Physiol* 256: R567-R571, 1989.

176. Hölker F. Effects of body size and temperature on metabolism of bream compared to sympatric roach. *Anim Biol* 56: 23-37, 2006.

177. Hongo Y. Evolution of male dimorphic allometry in a population of the Japanese horned beetle *Trypoxylus dichotomus septentrionalis*. *Behav Ecol Sociobiol* 62: 245-253, 2007.

178. Hoppeler H, Weibel ER. Scaling functions to body size: Theories and facts. *J Exp Biol* 208: 1573-1574, 2005.

179. Hou C, Bolt KM, Bergman A. A general model for ontogenetic growth under food restriction. *Proc Roy Soc B-Biol Sci* 278: 2881-2890, 2011.

180. Hou C, Zuo W, Moses ME, Woodruff WH, Brown JH, West GB. Energy uptake and allocation during ontogeny. *Science* 322: 736-739, 2008.

181. Housworth EA, Martins EP, Lynch M. The phylogenetic mixed model. *Am Nat* 163: 84-96, 2004.

182. Howland HC, Merola S, Basarab JR. The allometry and scaling of the size of vertebrate eyes. *Vision Res* 44: 2043-2065, 2004.

183. Hui C, Terblanche JS, Chown SL, McGeoch MA. Parameter landscapes unveil the bias in allometric prediction. *Meth Ecol Evol* 1: 69-74, 2010.

184. Hui D, Jackson RB. Uncertainty in allometric exponent estimation: A case study in scaling metabolic rate with body mass. *J Theor Biol* 249: 168-177, 2007.

185. Huitema BE. *The Analysis of Covariance and Alternatives*. New York: John Wiley and Sons, 1980, p. 445.

186. Humphries MM, Boutin S, Thomas DW, Ryan JD, Selman C, McAdam AG, Berteaux D, Speakman JR. Expenditure freeze: The metabolic response of small mammals to cold environments. *Ecol Lett* 8: 1326-1333, 2005.

187. Humphries MM, Careau V. Heat for nothing or activity for free? Evidence and implications of activity-thermoregulatory heat substitution. *Integr Comp Biol* 51: 419-431, 2011.

188. Huxley JS. *Problems of Relative Growth*. London: Methuen & Co., 1932.

189. Ihaka R, Gentleman R. R: A language for data analysis and graphics. *J Comput Graph Stat* 5: 299-314, 1996.

190. Isaac NJB, Carbone C. Why are metabolic scaling exponents so controversial? Quantifying variance and testing hypotheses. *Ecol Lett* 33: 728-735, doi: 10.1111/j.1461-0248.2010.01461.x, 2010.

191. Ives AR, Garland T, Jr. Phylogenetic logistic regression for binary dependent variables. *Syst Biol* 59: 9-26, 2010.

192. Ives AR, Midford PE, Garland T, Jr. Within-species variation and measurement error in phylogenetic comparative methods. *Syst Biol* 56: 252-270, 2007.

193. Jackson DM, Trayhurn P, Speakman JR. Associations between energetics and over-winter survival in the short-tailed field vole *Microtus agrestis*. *J Anim Ecol* 70: 633-640, 2001.

194. Jetz W, Freckleton RP, McKechnie AE. Environment, migratory tendency, phylogeny and basal metabolic rate in birds. *PLoS ONE* 3: e3261, 2007.

195. Jetz W, Thomas GH, Joy JB, Hartmann K, Mooers AO. The global diversity of birds in space and time. *Nature* 491: 444-448, 2012.

196. Johnson PO, Neyman J. Tests of certain linear hypotheses and their application to some educational problems. *Stat. Res. Mem.* 1: 57-93, 1936.

197. Jolicoeur P. A simplified model for bivariate complex allometry. *J Theor Biol* 140: 41-49, 1989.

198. Jones JH, Taylor CR, Lindholm A, Straub R, Longworth KE, Karas RH. Blood gas measurements during exercise: Errors due to temperature correction. *J Appl Physiol* 67: 879-884, 1989.

199. Jones KE, Bielby J, Cardillo M, Fritz SA, O'Dell J, Orme CDL, Safi K, Sechrest W, Boakes EH, Carbone C, Connolly C, Cutts MJ, Foster JK, Grenyer R, Habib M, Plaster CA, Price SA, Rigby EA, Rist J, Teacher A, Bininda-Emonds ORP, Gittleman JL, Mace GM, Purvis A. PanTHERIA: A species-level database of life history, ecology, and geography of extant and recently extinct mammals. *Ecology* 90: 2648, 2009.

200. Kabat AP, Blackburn TM, McKechnie AE, Butler PJ. Phylogenetic analysis of the allometric scaling of therapeutic regimes for birds. *J Zool* 275: 359-367, 2008.

201. Kaiyala KJ, Ramsay DS. Direct animal calorimetry, the underused gold standard for quantifying the fire of life. *Comp Biochem Physiol A* 158: 252-264, 2011.

202. Kearney MR, White CR. Testing metabolic theories. *Am Nat* 180: 546-565, 2012.

203. Kerkhoff AJ, Enquist BJ. Multiplicative by nature: Why logarithmic transformation is necessary in allometry. *J Theor Biol* 257: 519-521, 2009.

204. Killen SS, Atkinson D, Glazier DS. The intraspecific scaling of metabolic rate with body mass in fishes depends on lifestyle and temperature. *Ecol Lett* 13: 184-193, 2010.

205. Killen SS, Costa I, Brown JA, Gamperl AK. Little left in the tank: Metabolic scaling in marine teleosts and its implications for aerobic scope. *Proc Roy Soc B-Biol Sci* 274: 431-438, 2007.

206. Kleiber M. Body size and metabolism. *Hilgardia* 6: 315-353, 1932.

207. Kleiber M. *The Fire of Life*. New York, London: John Wiley & Sons, Inc., 1961, p. 454.

208. Knight MH. Thermoregulation in the largest African cricetid, the giant rat *Cricetomys gambianus*. *Comp Biochem Physiol A* 89: 705-708, 1988.

209. Kodric-Brown A, Sibly RM, Brown JH. The allometry of ornaments and weapons. *Proc Natl Acad Sci U S A* 103: 8733-8738, 2006.

210. Kolokotrones T, Savage VM, Deeds EJ, Fontana W. Curvature in metabolic scaling. *Nature* 464: 753-756, 2010.

211. Konarzewski M, Książek A. Determinants of intra-specific variation in basal metabolic rate. *J Comp Physiol B* 183: 27-41, 2013.

212. Kooijman SALM. *Dynamic Energy and Mass Budgets in Biological Systems*. Cambridge: Cambridge University Press, 2000.

213. Kooijman SALM. *Dynamic Energy Budget Theory for Metabolic Organisation*. Cambridge: Cambridge University Press, 2010.

214. Kooijman SALM. Energy budgets can explain body size relations. *J Theor Biol* 121: 269-282, 1986.

215. Kooijman SALM. Waste to hurry: Dynamic energy budgets explain the need of wasting to fully exploit blooming resources. *Oikos* 122: 348-357, 2013.

216. Kottelat M, Britz R, Hui TH, Witte K-E. *Paedocypris*, a new genus of Southeast Asian cyprinid fish with a remarkable sexual dimorphism, comprises the world's smallest vertebrate. *Proc Roy Soc B-Biol Sci* 273: 895-899, 2006.

217. Kozłowski J, Konarzewski M. Is West, Brown and Enquist's model of allometric scaling mathematically correct and biologically relevant? *Funct Ecol* 18: 283-289, 2004.

218. Kozłowski J, Konarzewski M. West, Brown and Enquist's model of allometric scaling again: The same questions remain. *Funct Ecol* 19: 739-743, 2005.

219. Kozłowski J, Konarzewski M, Gawelczyk AT. Cell size as a link between noncoding DNA and metabolic rate scaling. *Proc Natl Acad Sci U S A* 100: 14080-14085, 2003.

220. Kozłowski J, Konarzewski M, Gawelczyk AT. Intraspecific body size optimization produces intraspecific allometries. In: Blackburn TM, Gaston KJ, editors. *Macroecology: Concepts and Consequences*. Malden: Blackwell Science Ltd, 2003, pp. 299-320.

221. Krogh A. *Respiratory Exchange of Animals and Man*. London: Longmans, Green and Co., 1916.

222. Król E, Murphy RW, Speakman JR. Limits to sustained energy intake. X. Effects of fur removal on reproductive performance in laboratory mice. *J Exp Biol* 240: 4233-4243, 2007.

223. Kühn I, Nobis MP, Durka W. Combining spatial and phylogenetic eigenvector filtering in trait analysis. *Glob Ecol Biogeogr* 18: 745-758, 2009.

224. Larivée ML, Boutin S, Speakman JR, McAdam AG, Humphries MM. Associations between over-winter survival and resting metabolic rate in juvenile North American red squirrels. *Funct Ecol* 24: 597-607, 2010.

225. Lavin Shana R, Karasov William H, Ives Anthony R, Middleton Kevin M, Theodore G, Jr. Morphometrics of the avian small intestine compared with that of nonflying mammals: A phylogenetic approach. *Physiol Biochem Zool* 81: 526-550, 2008.

226. LeBarbera M. Analyzing body size as a factor in ecology and evolution. *Annu Rev Ecol Syst* 20: 97-117, 1989.

227. Leon AC, Portera L, Lowell K, Rheinheimer D. A strategy to evaluate a covariate by group interaction in an analysis of covariance. *Psychopharmacol Bull* 34: 805-809, 1998.

228. Lighton JRB. *Measuring Metabolic Rates: A Manual for Scientists.* Oxford: Oxford University Press, 2008.

229. Lighton JRB, Halsey LG. Flow-through respirometry applied to chamber systems: Pros and cons, hints and tips. *Comp Biochem Physiol A* 158: 265-275, 2011.

230. Lillywhite H, Albert J. Evolutionary physiology, comparative data, and phylogenetic methods. In: Morris S, Vosloo A, editors. *Proceedings of the 4th CPB Meeting in Africa: MARA 2008 Molecules to Migration: The Pressures of Life.* Pianoro: Medimond, 2009, pp. 613-620.

231. Lillywhite HB, Albert JS, Sheehy CM, III, Seymour RS. Gravity and the evolution of cardiopulmonary morphology in snakes. *Comp Biochem Physiol A* 161: 230-242, 2012.

232. Lillywhite HB, Seymour RS. Heart position in snakes: Response to "Phylogeny, ecology, and heart position in snakes". *Physiol Biochem Zool* 84: 99-101, 2011.

233. Lindinger MI. Exercise in the heat: Thermoregulatory limitations to performance in humans and horses. *Can J Appl Physiol* 24: 152-163, 1999.

234. Lindstedt SL, Hokanson JF, Wells DJ, Swain SD, Hoppeler H, Navarro V. Running energetics in the pronghorn antelope. *Nature* 353: 748-750, 1991.

235. Lindstedt SL, Schaeffer PJ. Use of allometry in predicting anatomical and physiological parameters of mammals. *Lab Anim* 36: 1-19, 2002.

236. Lockyear C. Body weights of some species of large whales. *J. Cons. Int. Explor. Mer* 36: 259-273, 1976.

237. Losos JB. Seeing the forest for the trees: The limitations of phylogenies in comparative biology. *Am Nat* 177: 709-727, 2011.

238. Lovegrove BG. The influence of climate on the basal metabolic rate of small mammals: A slow-fast metabolic continuum. *J Comp Physiol B* 173: 87-112, 2003.

239. Lovegrove BG. The metabolism of social subterranean rodents: Adaptation to aridity. *Oecologia* 69: 551-555, 1986.

240. Lovegrove BG. The zoogeography of mammalian basal metabolic rate. *Am Nat* 156: 201-219, 2000.

241. Lydersen C, Ryg MS, Hammill MO, O'Brien PJ. Oxygen stores and aerobic dive limit of ringed seals (*Phoca hispida*). *Can J Zool* 70: 458-461, 1992.

242. Lynch M. Methods for the analysis of comparative data in evolutionary biology. *Evolution* 45: 1065-1080, 1991.

243. Lynch M, Walsh B. *Genetics and Analysis of Quantitative Traits.* Sunderland: Sinauer Associates, 1998.

244. Maddison WP, Maddison DR. Mesquite: A modular system for evolutionary analysis. Version 2.75. http://mesquiteproject.org. 2011.

245. Magnusson William E. Significance versus magnitude: Use of the Johnson-Neyman technique in comparative biology. *Physiol Biochem Zool* 78: 105, 2005.

246. Mahmood I. Prediction of clearance, volume of distribution and half-life by allometric scaling and by use of plasma concentrations predicted from pharmacokinetic constants: A comparative study. *J Pharm Pharmacol* 51: 905-910, 1999.

247. Mahmood I, Martinez M, Hunter RP. Interspecies allometric scaling. Part I: Prediction of clearance in large animals. *J Vet Pharmacol Ther* 29: 415-423, 2006.

248. Mahoney SA. Cost of locomotion and heat balance during rest and running from 0 to 55 degrees C in a patas monkey. *J Appl Physiol* 49: 789-800, 1980.

249. Maino JL, Kearney MR, Nisbet RM, Kooijman SALM. Reconciling theories for metabolic scaling. *J Anim Ecol* DOI: 10.1111/1365-2656.12085, 2013. [Epub ahead of print].

250. Makarieva AM, Gorshkov VD, Li B-L, Chown SL, Reich PB, Gavrilov VM. Mean mass-specific metabolic rates are strikingly similar across life's major domains: Evidence for life's metabolic optimum. *Proc Natl Acad Sci USA* 105: 16994-16999, 2008.

251. Martins EP, Hansen TF. Phylogenies and the comparative method: A general approach to incorporating phylogenetic information into the analysis of interspecific data. *Am Nat* 149: 646-667, 1997.

252. McArdle BH. Lines, models, and errors: Regression in the field. *Limnol Oceanogr* 48: 1363-1366, 2003.

253. McArdle BH. The structural relationship: Regression in biology. *Can J Zool* 66: 2329-2339, 1988.

254. McFeeters BJ, Xenopoulos MA, Spooner DE, Wagner ND, Frost PC. Intraspecific mass-scaling of field metabolic rates of a freshwater crayfish varies with stream land cover. *Ecosphere* 2: art13, 2011.

255. McKechnie AE, Freckleton RP, Jetz W. Phenotypic plasticity in the scaling of avian basal metabolic rate. *Proc Roy Soc B-Biol Sci* 273: 931-937, 2006.

256. McKechnie AE, Wolf BO. The allometry of avian basal metabolic rate: Good predictions need good data. *Physiol Biochem Zool* 77: 502-521, 2004.

257. McMahon T. Size and shape in biology. *Science* 179: 1201-1204, 1973.

258. McMahon TA, Bonner JT. *On Size and Life.* New York: Scientific American, 1983.

259. McNab BK. Climatic adaptation in the energetics of heteromyid rodents. *Comp Biochem Physiol A* 62: 813-820, 1979.

260. McNab BK. Physiological convergence amongst ant-eating and termite-eating mammals. *J Zool* 203: 485-510, 1984.

261. McNab BK. Complications inherent in scaling the basal rate of metabolism in mammals. *Q Rev Biol* 63: 25-54, 1988.

262. McNab BK. On the utility of uniformity in the definition of basal rate of metabolism. *Physiol Zool* 70: 718-720, 1997.

263. McNab BK. The standard energetics of mammalian carnivores: Felidae and Hyaenidae. *Can J Zool* 78: 2227-2239, 2000.

264. McNab BK. Sample size and the estimation of physiological parameters in the field. *Funct Ecol* 17: 82-86, 2003.

265. McNab BK. Standard energetics of phyllostomid bats: The inadequacies of phylogenetic-contrast analyses. *Comp Biochem Physiol A* 135: 357-368, 2003.

266. McNab BK. The evolution of energetics in eutherian "insectivorans": An alternate approach. *Acta Theriol* 51: 113-128, 2006.

267. McNab BK. An analysis of the factors that influence the level and scaling of mammalian BMR. *Comp Biochem Physiol A* 151: 5-28, 2008.

268. McNab BK. Ecological factors affect the level and scaling of avian BMR. *Comp Biochem Physiol A* 152: 22-45, 2009.

269. McNab BK. Energy expenditure cannot be effectively analyzed with phylogenetically based techniques. In: Morris S, Vosloo A, editors. *Proceedings of the 4th CPB Meeting in Africa: MARA 2008 Molecules to Migration: The Pressures of Life.* Pianoro: Medimond, 2009, pp. 621-626.

270. McNab BK. Resources and energetics determined dinosaur maximal size. *Proc Natl Acad Sci USA* 106: 12184-12188, 2009.

271. McNab BK, Eisenberg JF. Brain size and its relation to the rate of metabolism in mammals. *Am Nat* 133: 157-167, 1989.

272. McNab BK, Morrison P. Body temperature and metabolism in subspecies of *Peromyscus* from arid and mesic environments. *Ecol Monogr* 33: 63-82, 1963.

273. Meehan TD. Energy use and animal abundance in litter and soil communities. *Ecology* 87: 1650-1658, 2006.

274. Meehan TD, Jetz W, Brown JH. Energetic determinants of abundance in winter landbird communities. *Ecol Lett* 7: 532-537, 2004.

275. Meiri S, Dayan T. On the validity of Bergmann's rule. *J Biogeogr* 30: 331-351, 2003.

276. Michard-Picamelot D, Zorn T, Gendner JP, Mata AJ, Le Maho Y. Body protein does not vary despite seasonal changes in fat in the white stork *Ciconia ciconia. Ibis* 144: E1-E10, 2002.

277. Midford PE, Garland T, Jr, Maddison W. PDAP Package for Mesquite. Version 1.16. 2011.

278. Mitchell G, Maloney SK, Mitchell D, Keegan DJ. The origin of mean arterial and jugular venous blood pressures in giraffes. *J Exp Biol* 209: 2515-2524, 2006.

279. Mollet HF, Cailliet GM. Using allometry to predict body mass from linear measuremetns of the white shark. In: Klimley AP, Ainley DG, editors. *Great White Sharks: The Biology of Carcharodon carcharias.* San Diego: Academic Press, 1996, pp. 81-89.

280. Moran D, Wells RMG. Ontogenetic scaling of fish metabolism in the mouse-to-elephant mass magnitude range. *Comp Biochem Physiol A* 148: 611-620, 2007.

281. Mueller P, Diamond J. Metabolic rate and environmental productivity: Well-provisioned animals evolved to run and idle fast. *Proc Natl Acad Sci U S A* 98: 12551-12554, 2001.

282. Muller-Landau HC, Condit RS, Chave J, Thomas SC, Bohlman SA, Bunyavejchewin S, Davies S, Foster R, Gunatilleke S, Gunatilleke N, Harms KE, Hart T, Hubbell SP, Itoh A, Kassim AR, LaFrankie JV, Lee HS, Losos E, Makana J-R, Ohkubo T, Sukamar R, Sun I-F, Supardi N, Tan S, Thompson J, Valencia R, Muñoz GV, Wills C, Yamakura T, Chuyong G, Dattaraja HS, Esufali S, Hall P, Hernandez C, Kenfack D, Kiratiprayoon S, Suresh HS, Thomas D, Vallejo MI, Ashton P. Testing metabolic ecology theory for allometric scaling of tree size, growth and mortality in tropical forests. *Ecol Lett* 9: 575-588, 2006.

283. Müller DWH, Codron D, Werner J, Fritz J, Hummell J, Griebeler EM, Clauss M. Dichotomy of eutherian reproduction and metabolism. *Oikos* 121: 102-115, 2012.

284. Müller MJ, Langemann D, Gehrke I, Later W, Heller M, Glüer CC, Heymsfield SB, Bosy-Westphal A. Effect of constitution on mass of individual organs and their association with metabolic rate in humans—a detailed view on allometric scaling. *PLoS ONE* 6: e22732, 2011.

285. Munch SB, Salinas S. Latitudinal variation in lifespan within species is explained by the metabolic theory of ecology. *Proc Natl Acad Sci U S A* 106: 13860-13864, 2009.

286. Muñoz-Garcia A, Williams JB. Basal metabolic rate in carnivores is associated with diet after controlling for phylogeny. *Physiol Biochem Zool* 78: 1039-1056, 2005.

287. Nagilla R, Ward KW. A comprehensive analysis of the role of correction factors in the allometric predictivity of clearance from rat, dog, and monkey to humans. *J Pharm Sci* 93: 2522-2534, 2004.

288. Nagy KA. Field metabolic rate and body size. *J Exp Biol* 208: 1621-1625, 2005.

289. Nagy KA, Girard IA, Brown TK. Energetics of free-ranging mammals, reptiles and birds. *Annu Rev Nutr* 19: 247-277, 1999.

290. Nakaya F, Saito Y, Motokawa T. Experimental allometry: Effect of size manipulation on metabolic rate of colonial ascidians. *Proc Roy Soc B-Biol Sci* 272: 1963-1969, 2005.

291. Nespolo RF, Franco M. Whole-animal metabolic rate is a repeatable trait: A meta-analysis. *J Exp Biol* 210: 2000-2005, 2007.

292. Nevill AM, Holder RL. Scaling, normalizing, and per ratio standards: An allometric modelling approach. *J Appl Physiol* 79: 1027-1031, 1995.

293. Nisbet RM, Muller EB, Lika K, Kooijman SALM. From molecules to ecosystems through dynamic energy budget models. *J Anim Ecol* 69: 913-926, 2000.

294. Nowak RM. *Walker's Mammals of the World*. Baltimore: Johns Hopkins University Press, 1999, p. 1936.

295. O'Conner MP, Agosta SJ, Hansen F, Kemp SJ, Sieg AE, McNair JN, Dunham AE. Phylogeny, regression, and the allometry of physiological traits. *Am Nat* 170: 431-442, 2007.

296. Ohlberger J, Mehner T, Staaks G, Hölker F. Temperature-related physiological adaptations promote ecological divergence in a sympatric species pair of temperate freshwater fish, *Coregonus* spp. *Funct Ecol* 22: 501-508, 2008.

297. Orme D, Freckleton RP, Thomas GH, Petzoldt Y, Fritz S, Isaac N, Pearse W. Caper: Comparative Analyses of Phylogenetics and Evolution in R package version 0.5. 2012.

298. Packard GC. On the use of logarithmic transformations in allometric analyses. *J Theor Biol* 257: 515-518, 2009.

299. Packard GC, Birchard GF. Traditional allometric analysis fails to provide a valid predictive model for mammalian metabolic rates. *J Exp Biol* 211: 3581-3587, 2008.

300. Packard GC, Birchard GF, Boardman TJ. Fitting statistical models in bivariate allometry. *Biol Rev* 83: 254-563, 2011.

301. Packard GC, Boardman TJ. The misuse of ratios to scale physiological data that vary allometrically with body size. In: Feder ME, Bennett AF, Burggren WW, Huey RB, editors. *New Directions in Ecological Physiology*. Cambridge: Cambridge University Press, 1987, pp. 216-239.

302. Packard GC, Boardman TJ. The misuse of ratios, indices and percentages in ecophysiological research. *Physiol Zool* 61: 1-9, 1988.

303. Packard GC, Boardman TJ. The use of percentages and size-specific indices to normalize physiological data for variation in body size: Wasted time, wasted effort? *Comp Biochem Physiol A* 122: 37-44, 1999.

304. Packard GC, Boardman TJ. Model selection and logarithmic transformation in allometric analysis. *Physiol Biochem Zool* 81: 496-507, 2008.

305. Packard GC, Boardman TJ. A comparison of methods for fitting allometric equations to field metabolic rates of animals. *J Comp Physiol A* 179: 175-182, 2009.

306. Pagel M. Inferring the historical patterns of biological evolution. *Nature* 401: 877-884, 1999.

307. Pagel M, Harvey PH. The taxon-level problem in the evolution of mammalian brain size: Facts and artifacts. *Am Nat* 132: 344-359, 1988.

308. Pagel MD. A method for the analysis of comparative data. *J Theor Biol* 156: 431-442, 1992.

309. Painter PR. Data from necropsy studies and *in vitro* tissue studies lead to a model for allometric scaling of basal metabolic rate. *Theor Biol Med Model* 2: 39, 2005.

310. Paradis E, Claude J, Strimmer K. APE: Analyses of Phylogenetics and Evolution in R language. *Bioinformatics* 20: 289-290, 2004.

311. Patterson JL, Goetz RH, Doyle JT, Warren JV, Gauer OH, Detweiler DK, Said SI, Hoernicke H, McGregor M, Keen EN, Smith MH, Hardie EL, Reynolds M, Flatt WP, Waldo DR. Cardiorespiratory dynamics in the ox and giraffe, with comparative observations on man and other mammals. *Ann N Y Acad Sci* 127: 393-413, 1965.

312. Patterson MR. A mass transfer explanation of metabolic scaling relations in some aquatic invertebrates and algae. *Science* 255: 1421-1423, 1992.

313. Pélabon C, Bolstad GH, Egset CK, Cheverud JM, Pavlicev M, Rosenqvist G. On the relationship between ontogenetic and static allometry. *Am Nat* 181: 195-212, 2013.

314. Peters RH. *The Ecological Implications of Body Size*. Cambridge: Cambridge University Press, 1983, p. 329.

315. Phillipson J. Bioenergetic options and phylogeny. In: Townsend CR, Calow P, editors. *Physiological Ecology: An Evolutionary Approach to Resource Use*. Sunderland: Sinauer Associates, 1981, pp. 20-45.

316. Pitts GC, Bullard TR. Some interspecific aspects of body composition in mammals. In: Reid JT, Bensadoun A, Bull LS, editors. *Body composition in animals and man*. Washington, DC: National Academy of Science, 1968.

317. Porter WP, Kearney M. Size, shape, and the thermal niche of endotherms. *Proc Natl Acad Sci U S A* 106: 19666-19672, 2009.

318. Portugal SJ, Green JA, Butler PJ. Annual changes in body mass and resting metabolism in captive barnacle geese (*Branta leucopsis*): The importance of wing moult. *J Exp Biol* 210: 1391-1397, 2007.

319. Potter B. The allometry of primate skeletal weight. *Int J Primatol* 7: 457-466, 1986.

320. Potthoff R. On the Johnson-Neyman technique and some extensions thereof. *Psychometrika* 29: 241–256, 1964.

321. Powers DR, Getsinger PW, Tobalske BW, Wethington SM, Powers SD, Warrick DR. Respiratory evaporative water loss during hovering and forward flight in hummingbirds. *Comp Biochem Phys A* 161: 279-285, 2012.

322. Prange HD, Anderson JF, Rahn H. Scaling of skeletal mass to body mass in birds and mammals. *Am Nat* 113: 103-122, 1979.

323. Price CA, Enquist BJ, Savage VM. A general model for allometric covariation in botanical form and function. *Proc Natl Acad Sci USA* 104: 13204-13209, 2007.

324. Purvis A. A composite estimate of primate phylogeny. *Philos T R Soc Lon B* 348: 405-421, 1995.

325. Purvis A, Garland T, Jr. Polytomies in comparative analyses of continuous characters. *Syst Biol* 42: 569-575, 1993.

326. Purvis A, Gittleman JL, Luh H-K. Truth or consequences: Effects of phylogenetic accuracy on two comparative methods. *J Theor Biol* 167: 293-300, 1994.

327. Pyron RA, Burbrink FT, Wiens JJ. A phylogeny and revised classification of Squamata, including 4161 species of lizards and snakes. *BMC Evol Biol* 13: 93, 2013.

328. Pyron RA, Wiens JJ. A large-scale phylogeny of Amphibia including over 2800 species, and a revised classification of extant frogs, salamanders, and caecilians. *Mol Phylogenet Evol* 61: 543-583, 2011.

329. Quinn GP, Keough MJ. *Experimental Design and Data Analysis for Biologists*. Cambridge: Cambridge University Press, 2002.

330. R Development Core Team. R: A Language and Environment for Statistical Computing. Vienna, Austria: R Foundation for Statistical Computing, 2013.

331. Rahbek C, Gotelli NJ, Colwell RK, Entsminger GL, Rangel TFLVB, Graves GR. Predicting continental-scale patterns of bird species richness with spatially explicit models. *Proc Roy Soc B-Biol Sci* 274: 165-174, 2007.

332. Redford KH, Dorea JG. The nutritional value of invertebrates with emphasis on ants and termites as food for mammals. *J Zool* 203: 385-395, 1984.

333. Reynolds PS. Phylogenetic analysis of surface areas of mammals. *J Mammal* 78: 859-868, 1997.

334. Reynolds WW. Skeleton weight allometry in aquatic and terrestrial vertebrates. *Hydrobiologia* 56: 35-37, 1977.

335. Reynolds WW, Karlotski WJ. The allometric relationship of skeleton weight to body weight in teleost fishes: A preliminary comparison with birds and mammals. *Copeia* 1977: 160-163, 1977.

336. Rezende EL, Diniz-Filho JAF. Phylogenetic analyses: Comparing species to infer adaptations and physiological mechanisms. *Compr Physiol* 2: 639-674, 2012.

337. Riveros AJ, Enquist BJ. Metabolic scaling in insects supports the predictions of the WBE model. *J Insect Physiol* DOI: 10.1016/j.jinsphys.2011.1001.1011, 2011.

338. Rixon RH, Stevenson JAF. Factors influencing survival of rats in fasting. Metabolic rate and body weight loss. *Am J Physiol* 188: 332-336, 1957.

339. Roberts MF, Lightfoot EN, Porter WP. A new model for the body size–metabolism relationship. *Physiol Biochem Zool* 83: 395-405, 2010.

340. Roberts MF, Lightfoot EN, Porter WP. Basal metabolic rate of endotherms can be modeled using heat-transfer principles and physiological concepts: Reply to "Can the basal metabolic rate of endotherms be explained by biophysical modeling?". *Physiol Biochem Zool* 84: 111-114, 2011.

341. Robinson WR, Peters RH, Zimmermann J. The effects of body size and temperature on metabolic rate of organisms. *Can J Zool* 61: 281-288, 1983.

342. Rogowitz GL, Chappell MA. Energy metabolism of eucalyptus-boring beetles at rest and during locomotion: Gender makes a difference. *J Exp Biol* 203: 1131-1139, 2000.

343. Rohlf FJ. Comparative methods for the analysis of continuous variables: Geometric interpretations. *Evolution* 55: 2143-2160, 2001.

344. Rombouts I, Beaugrand G, Ibaňez F, Chiba S, Legendre L. Marine copepod diversity patterns and the metabolic theory of ecology. *Oecologia* 166: 349-355, 2011.

345. Rowland JM, Emlen DJ. Two thresholds, three male forms result in facultative male trimorphism in beetles. *Science* 323: 773-776, 2009.

346. Rubner M. Über den Einfluss der Körpergrösse auf Stoff- und Kraftwechsel. *Zeischrift für Biologie* 19: 536-562, 1883.

347. Runciman S, Seymour RS, Baudinette RV, Pearson JT. An allometric study of lung morphology during development in the Australian pelican, *Pelicanus conspicillatus*, from embryo to adult. *J Anat* 207: 365-380, 2005.

348. Ruxton GD, Houston DC. Could *Tyrannosaurus rex* have been a scavenger rather than a predator? An energetics approach. *Proc Roy Soc B-Biol Sci* 270: 731-733, 2003.

349. Savage VM, Deeds EJ, Fontana W. Sizing up allometric scaling theory. *PLoS Computational Biology* 4: e1000171, 2008.

350. Savage VM, Gillooly JF, Woodruff WH, West GB, Allen AP, Enquist BJ, Brown JH. The predominance of quarter-power scaling in biology. *Funct Ecol* 18: 257-282, 2004.

351. Schimpf NG, Matthews PGD, White CR. Cockroaches that exchange respiratory gases discontinuously survive food and water restriction. *Evolution* 66: 597-604, 2012.

352. Schimpf NG, Matthews PGD, White CR. Standard metabolic rate is associated with gestation duration, but not clutch size, in speckled cockroaches *Nauphoeta cinerea*. *Biol Open* 1: 1185-1191, 2012.

353. Schlader ZJ, Stannard SR, Mündel T. Human thermoregulatory behavior during rest and exercise—A prospective review. *Physiol Behav* 99: 269-275, 2010.

354. Schmid J, Andersen NA, Speakman JR, Nicol SC. Field energetics of free-living, lactating and non-lactating echidnas (*Tachyglossus aculeatus*). *Comp Biochem Physiol A* 136: 903-909, 2003.

355. Schmidt-Nielsen K. Locomotion: Energy cost of swimming, flying and running. *Science* 172: 222-228, 1972.

356. Schmidt-Nielsen K. Scaling in biology: The consequences of size. *J Exp Zool* 194: 287-307, 1975.

357. Schmidt-Nielsen K. *Scaling: Why is Animal Size so Important?* Cambridge: Cambridge University Press, 1984, p. 241.

358. Seebacher F. A new method to calculate allometric length-mass relationships of dinosaurs. *J Vertebr Paleontol* 21: 51-60, 2001.

359. Seebacher F, Grigg GC, Beard LA. Crocodiles as dinosaurs: Behavioural thermoregulation in very large ectotherms leads to high and stable body temperatures. *J Exp Biol* 202: 77-86, 1999.

360. Seim E, Sæther B-E. On rethinking allometry: Which regression model to use? *J Theor Biol* 104: 161-168, 1983.

361. Seymour RS. Raising the sauropod neck: It costs more to get less. *Biol Letters* 5: 317-319, 2009.

362. Seymour RS, Blaylock AJ. The principle of Laplace and scaling of ventricular wall stress and blood pressure in mammals and birds. *Physiol Biochem Zool* 73: 389-405, 2000.

363. Seymour RS, Gienger CM, Brien ML, Tracy CR, Manolis SC, Webb GJW, Christian KA. Scaling of standard metabolic rate in estuarine crocodiles *Crocodylus porosus*. *J Comp Physiol B* 183: 491-500, 2013.

364. Seymour RS, Hargens AR, Pedley TJ. The heart works against gravity. *Am J Physiol* 265: R715-R720, 1993.

365. Seymour RS, Lillywhite HB. Blood pressure in snakes from different habitats. *Nature* 264: 664-666, 1976.

366. Seymour RS, Lillywhite HB. Hearts, neck posture and metabolic intensity of sauropod dinosaurs. *Proc R Soc Lond B* 267: 1883-1887, 2000.

367. Seymour RS, Runciman S, Baudinette RV, Pearson JT. Developmental allometry of pulmonary structure and function in the altricial Australian pelican *Pelecanus conspicillatus*. *J Exp Biol* 207: 2663-2669, 2004.

368. Seymour RS, Smith SL, White CR, Henderson DM, Schwarz-Wings D. Blood flow to long bones indicates activity metabolism in mammals, reptiles and dinosaurs. *Proc Roy Soc B-Biol Sci* 279: 451-456, 2012.

369. Seymour RS, White CR. Can the basal metabolic rate of endotherms be explained by biophysical modeling? Response to "A new model for the body size–metabolism relationship". *Physiol Biochem Zool* 84: 107-110, 2011.

370. Shaffer SA. A review of seabird energetics using the doubly labeled water method. *Comp Biochem Physiol A* 158: 315-322, 2011.

371. Shingleton AW, Frankino WA, Flatt T, Nijhout HF, Emlen DJ. Size and shape: The developmental regulation of static allometry in insects. *BioEssays* 29: 536-548, 2007.

372. Shipley B. *Cause and Correlation in Biology*. Cambridge: Cambridge University Press, 2000.

373. Sibly RM, Brown JH, Kodric-Brown A editors. *Metabolic Ecology: A Scaling Approach*. Wiley Blackwell, 2012, p. 392.

374. Sieg AE, O'Conner MP, McNair JN, Grant BW, Agosta SJ, Dunham AE. Mammalian metabolic allometry: Do intraspecific variation, phylogeny, and regression models matter? *Am Nat* 174: 720-733, 2009.

375. Silva M. Allometric scaling of body length: Elastic or geometric similarity in mammalian design. *J Mammal* 79: 20-32, 1998.

376. Silva M, Downing JA. The allometric scaling of density and body mass: A nonlinear relationship for terrestrial mammals. *Am Nat* 145: 704-727, 1995.

377. Simons MJP, Reimert I, van der Vinne V, Hambly C, Vaanholt LM, Speakman JR, Gerkema MP. Ambient temperature shapes reproductive output during pregnancy and lactation in the common vole (*Microtus arvalis*): A test of the heat dissipation limit theory. *J Exp Biol* 214: 38-49, 2011.

378. Smith RJ. Allometric scaling in comparative biology: Problems of concept and method. *Am J Physiol* 246: R152-R160, 1984.

379. Smith RJ. Rethinking allometry. *J Theor Biol* 87: 97-111, 1980.

380. Smith RJ. Use and misuse of the reduced major axis for line-fitting. *Am J Phys Anthropol* 140: 476-486, 2009.

381. Snell O. Die Abhängigkeit des Hirngewichtes von dem Körpergewicht und den geistigen Fähigkeiten. *Arch Psychiat Nervenkr* 23: 436-446, 1891.

382. Snelling EP, Seymour RS, Matthews PGD, Runciman S, White CR. Scaling of resting and maximum hopping metabolic rate throughout the life cycle of the locust *Locusta migratoria*. *J Exp Biol* 214: 3218-3224, 2011.

383. Sokal RR, Rohlf FJ. *Biometry*. W H Freeman and Co., 1995.

384. Speakman JR. *Doubly Labelled Water: Theory and Practice*. London: Chapman and Hall, 1997.

385. Speakman JR, Król E. Maximal heat dissipation capacity and hyperthermia risk: Neglected key factors in the ecology of endotherms. *J Anim Ecol* 79: 726-746, 2010.

386. Stahl WR. Organ weights in primates and other mammals. *Science* 150: 1039-1042, 1965.

387. Stahl WR. Scaling of respiratory variables in mammals. *J Appl Physiol* 22: 453-460, 1967.

388. Stern DL, Emlen DJ. The developmental basis for allometry in insects. *Development* 126: 1091-1101, 1999.

389. Streicher JW, Cox CL, Birchard GF. Non-linear scaling of oxygen consumption and heart rate in a very large cockroach species (*Gromphadorhina portentosa*): Correlated changes with body size and temperature. *J Exp Biol* 215: 1137-1143, 2012.

390. Suarez R. The biology of energy expenditure. *J Exp Biol* 214: 163-163, 2011.

391. Swanson DL, Garland T, Jr. The evolution of high summit metabolism and cold tolerance in birds and its impact on present-day distributions. *Evolution* 63: 184-194, 2009.

392. Swanson DL, Liknes ET. A comparative analysis of thermogenic capacity and cold tolerance in small birds. *J Exp Biol* 209: 466-474, 2006.

393. Symonds MRE. Life history of the Insectivora: The role of phylogeny, metabolism and sex differences. *J Zool* 249: 315-337, 1999.

394. Symonds MRE. The effects of topological inaccuracy in evolutionary trees on the phylogenetic comparative method of independent contrasts. *Syst Biol* 51: 541-553, 2002.

395. Taggart DA, Schultz D, White C, Whitehead P, Underwood G, Phillips K. Cross-fostering, growth and reproductive studies in the brush-tailed rock-wallaby, *Petrogale penicillata* (Marsupialia : Macropodidae): Efforts to accelerate breeding in a threatened marsupial species. *Aust J Zool* 53: 313-323, 2005.

396. Tang H, Hussain A, Leal M, Fluhler E, Mayersohn M. Controversy in the allometric application of fixed- versus varying-exponent models: A statistical and mathematical perspective. *J Pharm Sci* 100: 402-410, 2011.

397. Tang H, Hussain A, Leal M, Mayersohn M, Fluhler E. Interspecies prediction of human drug clearance based on scaling data from one or two animal species. *Drug Metab Dispos* 35: 1886-1893, 2007.

398. Tang H, Mayersohn M. Controversies in allometric scaling for predicting human drug clearance: An historical problem and reflections on what works and what does not. *Curr Top Med Chem* 11: 340-350, 2011.

399. Taylor CR, Dmi'el R, Shkolnik A, Baharav D, Borut A. Heat balance of running gazelles: Strategies for conserving water in the desert. *Am J Physiol* 226: 439-442, 1974.

400. Taylor CR, Rowntree VJ. Temperature regulation and heat balance in running cheetahs: A strategy for sprinters? *Am J Physiol* 224: 848-851, 1973.

401. Taylor CR, Schmidt-Nielsen K, Dmi'el R, Fedak MA. Effect of hyperthermia on heat balance during running in the African hunting dog. *Am J Physiol* 220: 823-827, 1971.

402. Terblanche JS, Janion C, Chown SL. Variation in scorpion metabolic rate and rate-temperature relationships: Implications for the fundamental equation of the metabolic theory of ecology. *J Evol Biol* 20: 1602-1612, 2007.

403. Terribile LC, Diniz-Filho JAF. Spatial patterns of species richness in New World coral snakes and the metabolic theory of ecology. *Acta Oecol* 35: 163-173, 2009.

404. Thompson DW. *On Growth and Form*. Cambridge: Cambridge University Press, 1917.

405. Tieleman BI, Williams JB. The adjustment of avian metabolic rates and water fluxes to desert environments. *Physiol Biochem Zool* 73: 461-479, 2000.

406. van der Meer J. Metabolic theories in ecology. *Trends Ecol Evol* 21: 136-140, 2006.

407. van der Veer HW, Kooijman SALM, van der Meer J. Body size scaling relationships in flatfish as predicted by Dynamic Energy Budgets (DEB theory): Implications for recruitment. *J Sea Res* 50: 257-272, 2003.

408. Vogt JT, Appel AG. Standard metabolic rate of the fire ant, *Solenopsis invicta* Buren: Effects of temperature, mass, and caste. *J Insect Physiol* 45: 655-666, 1999.

409. Wang Z, Ying Z, Bosy-Westphal A, Zhang J, Heller M, Later W, Heymsfield SB, Müller MJ. Evaluation of specific metabolic rates of major organs and tissues: Comparison between men and women. *Am J Hum Biol* 23: 333-338, 2011.

410. Wang Z, Ying Z, Bosy-Westphal A, Zhang J, Heller M, Later W, Heymsfield SB, Müller MJ. Evaluation of specific metabolic rates of major organs and tissues: Comparison between nonobese and obese women. *Obesity* 20: 95-100, 2012.

411. Weibel ER. *Symmorphosis: On Form and Function in Shaping Life.* Cambridge: Harvard University Press, 2000.

412. Weibel ER, Bacigalupe LD, Schmidt B, Hoppeler H. Allometric scaling of maximal metabolic rate in mammals: Muscle aerobic capacity as a determinant factor. *Resp Physiol Neurobi* 140: 115-132, 2004.

413. Weibel ER, Hoppeler H. Exercise-induced maximal metabolic rate scales with muscle aerobic capacity. *J Exp Biol* 208: 1635-1644, 2005.

414. Weibel ER, Taylor CR, Bolis L editors. *Principles of Design: The Optimization and Symmorphosis Debate.* Cambridge: Cambridge University Press, 1998.

415. Weibel ER, Taylor CR, Hoppeler H. The concept of symmorphosis: A testable hypothesis of structure-function relationship. *Proc Natl Acad Sci U S A* 88: 10357-10361, 1991.

416. Weiner J, Górecki A. Standard metabolic rate and thermoregulation in 5 species of Mongolian small mammals. *J Comp Physiol B* 145: 127-132, 1981.

417. Welch KC, Jr. The power of feeder-mask respirometry as a method for examining hummingbird energetics. *Comp Biochem Physiol A* 158: 276-286, 2011.

418. West GB, Brown JH. The origin of allometric scaling laws in biology from genomes to ecosystems: Towards a quantitative unifying theory of biological structure and organization. *J Exp Biol* 208: 1575-1592, 2005.

419. West GB, Brown JH, Enquist BJ. A general model for the origin of allometric scaling laws in biology. *Science* 276: 122-126, 1997.

420. West GB, Brown JH, Enquist BJ. A general model for the structure and allometry of plant vascular systems. *Nature* 400: 664-667, 1999.

421. West GB, Brown JH, Enquist BJ. The fourth dimension of life: Fractal geometry and allometric scaling of organisms. *Science* 284: 1677-1679, 1999.

422. West GB, Woodruff WH, Brown JH. Allometric scaling of metabolic rate from molecules and mitochondria to cells and mammals. *Proc Natl Acad Sci U S A* 99: 2473-2478, 2002.

423. Westerterp KR, Speakman JR. Physical activity energy expenditure has not declined since the 1980s and matches energy expenditures of wild mammals. *Int J Obes* 32: 1256-1263, 2008.

424. Westoby M, Leishman M, Lord J. Further remarks on phylogenetic correction. *J Ecol* 83: 727-729, 1995.

425. Westoby M, Leishman M, Lord J. Issues of interpretation after relating comparative datasets to phylogeny. *J Ecol* 83: 892-893, 1995.

426. Westoby M, Leishman MR, Lord JM. On misinterpreting the phylogenetic correction. *J Ecol* 83: 531-534, 1995.

427. White CR. Allometric analysis beyond heterogeneous regression slopes: Use of the Johnson-Neyman technique in comparative biology. *Physiol Biochem Zool* 76: 135-140, 2003.

428. White CR. The influence of foraging mode and arid adaptation on the basal metabolic rate of burrowing mammals. *Physiol Biochem Zool* 76: 122-134, 2003.

429. White CR. Allometric estimation of metabolic rates in animals. *Comp Biochem Physiol A* 158: 346-357, 2011.

430. White CR, Alton LA, Frappell PB. Metabolic cold adaptation in fish occurs at the level of whole animal, mitochondria, and enzyme. *Proc R Soc Lond B Biol Sci* 279: 1740-1747, 2012.

431. White CR, Blackburn TM, Martin GR, Butler PJ. Basal metabolic rate of birds is associated with habitat temperature and precipitation, not primary productivity. *Proc Roy Soc B-Biol Sci* 274: 287-293, 2007.

432. White CR, Blackburn TM, Seymour RS. Phylogenetically informed analysis of the allometry of mammalian basal metabolic rate supports neither geometric nor quarter-power scaling. *Evolution* 63: 2658-2667, 2009.

433. White CR, Blackburn TM, Terblanche JS, Marais E, Gibernau M, Chown SL. Evolutionary responses of discontinuous gas exchange in insects. *Proc Natl Acad Sci USA* 104: 8357-8361, 2007.

434. White CR, Cassey P, Blackburn TM. Allometric exponents do not support a universal metabolic allometry. *Ecology* 88: 315-323, 2007.

435. White CR, Cassey P, Schimpf NG, Halsey LG, Green JA, Portugal SJ. Implantation reduces the negative effects of bio-logging devices on birds. *J Exp Biol* 216: 537-542, 2013.

436. White CR, Frappell PB, Chown SL. An information-theoretic approach to evaluating the size and temperature dependence of metabolic rate. *Proc Roy Soc B-Biol Sci* 279: 3616-3621, 2012.

437. White CR, Kearney MR. Determinants of inter-specific variation in basal metabolic rate. *J Comp Physiol B* 183: 1-26, 2013.

438. White CR, Kearney MR, Matthews PGD, Kooijman SALM, Marshall DJ. A manipulative test of competing theories for metabolic scaling. *Am Nat* 178: 746-754, 2011.

439. White CR, Martin GR, Butler PJ. Pedestrian locomotion energetics and gait characteristics of a diving bird, the great cormorant, *Phalacrocorax carbo. J Comp Physiol B* 178: 745-754, 2008.

440. White CR, Matthews PGD, Seymour RS. Balancing the competing requirements of saltatorial and fossorial specialisation: Burrowing costs in the spinifex hopping mouse, *Notomys alexis. J Exp Biol* 209: 2103-2113, 2006.

441. White CR, Phillips NF, Seymour RS. The scaling and temperature dependence of vertebrate metabolism. *Biol Letters* 2: 125-127, 2006.

442. White CR, Schimpf NG, Cassey P. The repeatability of metabolic rate declines with time. *J Exp Biol* 216: 1763-1765, 2013.

443. White CR, Seymour RS. Mammalian basal metabolic rate is proportional to body mass$^{2/3}$. *Proc Natl Acad Sci U S A* 100: 4046-4049, 2003.

444. White CR, Seymour RS. Does BMR contain a useful signal? Mammalian BMR allometry and correlations with a selection of physiological, ecological and life-history variables. *Physiol Biochem Zool* 77: 929-941, 2004.

445. White CR, Seymour RS. Allometric scaling of mammalian metabolism. *J Exp Biol* 208: 1611-1619, 2005.

446. White CR, Seymour RS. Sample size and mass range effects on the allometric exponent of basal metabolic rate. *Comp Biochem Physiol A* 142: 74-78, 2005.

447. White CR, Seymour RS. Physiological functions that scale to body mass in fish. In: Farrell AP, editor. *Encyclopedia of Fish Physiology: From Genome to Environment*. San Diego: Academic Press, 2011, pp. 1573-1582.

448. White CR, Seymour RS. The role of gravity in the evolution of mammalian blood pressure. *In Review*, 2013. [Epub ahead of print].

449. White CR, Terblanche JS, Kabat AP, Blackburn TM, Chown SL, Butler PJ. Allometric scaling of maximum metabolic rate: The influence of temperature. *Funct Ecol* 22: 616-623, 2008.

450. Wiersma P, Muñoz-Garcia A, Walker A, Williams JB. Tropical birds have a slow pace of life. *Proc Natl Acad Sci U S A* 104: 9340-9345, 2007.

451. Wieser W. A distinction must be made between the ontogeny the phylogeny of metabolism in order to understand the mass exponent of energy metabolism. *Respir Physiol* 55: 1-9, 1984.

452. Wikelski M, Spinnery L, Schelsky W, Scheuerlein A, Gwinner E. Slow pace of life in tropical sedentary birds: A common-garden experiment on four stonechat populations from different latitudes. *Proc Roy Soc B-Biol Sci* 270: 2383-2388, 2003.

453. Withers PC. Design, calibration and calculation for flow-through respirometry systems. *Aust J Zool* 49: 445-461, 2001.

454. Xiao X, White EP, Hooten MB, Durham SL. On the use of log-transformation vs. nonlinear regression for analyzing biological power-laws. *Ecology* 92: 1887-1894, 2011.

455. Xiao Y. What are the units of the parameters in the power function for the length–weight relationship? *Fish Res* 35: 247-249, 1998.

456. Zar JH. *Biostatistical Analysis.* Upper Saddle River: Pearson, 2010.

457. Zerbe GO, Archer PG, Banchero N, Lechner AJ. On comparing regression lines with unequal slopes. *Am J Physiol* 242: R178-R180, 1982.

458. Zhao Z-J. Energy budget during lactation in striped hamsters at different ambient temperatures. *J Exp Biol* 214: 988-995, 2011.

459. Zhao Z-J, Król E, Moille S, Gamo Y, Speakman JR. Limits to sustained energy intake. XV. Effects of wheel running on the energy budget during lactation. *J Exp Biol* 216: 2316-2327, 2013.

460. Zub K, Fletcher QE, Szafrańska PA, Konarzewski M. Male weasels decrease activity and energy expenditure in response to high ambient temperatures. *PLoS ONE* 8: e72646, 2013.

461. Zuo W, Moses ME, Hou C, Woodruff WH, West GB, Brown JH. Response to comments on "Energy uptake and allocation during ontogeny". *Science* 325: 1206-c, 2009.

462. Zuo W, Moses ME, West GB, Hou C, Brown JH. A general model for effects of temperature on ectotherm ontogenetic growth and development. *Proc Roy Soc B-Biol Sci* 279: 1840-1846, 2012.