Notes and Comments

The Space-Lifetime Hypothesis: Viewing Organisms in Four Dimensions, Literally

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ABSTRACT: Much of the debate about alternative scaling exponents may result from unawareness of the dimensionality appropriate for different data and questions; in some cases, analysis has to include a fourth temporal dimension, and in others, it does not. Proportional scaling simultaneously applied to an organism and its generation time, treating the latter as a natural fourth dimension, produces a simple explanation for the 3/4 power in large-scale interspecies comparisons. Analysis of data sets of reduced dimensionality (e.g., data sets constructed such that one or more of the four dimensions are fixed), results in predictably lower metabolic exponents of 2/3 and 1/2 under one and two constraints, respectively. Our space-lifetime view offers a predictive framework that may be useful in developing a more complete mechanistic theory of metabolic scaling.

Keywords: metabolism, life span, scaling, dimensionality, metabolic ecology.

The 3/4-power scaling of metabolism with animal body mass (Kleiber's law; Kleiber 1932), generalized to all forms of life (Hemmingsen 1960; Brown et al. 2004; Savage et al. 2004), has been not unlike Fermat's theorem in the theory of integers: it is an observation that has been relatively easy to see but hard to explain. From the beginning, dimensional arguments have played an important role in attempts to account for metabolic scaling. Before Kleiber, metabolism was thought to scale as the 2/3 power of mass, since organisms metabolize through two-dimensional (2-D) surfaces but supply a three-dimensional (3-D) body (Rubner 1883). Recent work has produced an explanation of the observed tendency for metabolic rates to scale interspecifically according to Kleiber's law (instead of the 2/3 power) by focusing on the geometry of organisms' internal distribution networks for metabolites or nutrients (West et al. 1997, 1999; Banavar et al. 1999, 2002). In this theoretical approach, the exponent 3/4 results because the network scales as if it has a metaphorical "extra" spatial dimension related to the extra distances that a functional network requires as it increases in size (but for different reasons, depending on the models of different research groups). This characteristic of networks has been dubbed the "fourth dimension of life" (West et al. 1999). However, here we discuss something different: we argue that there is a distinct and literal sense in which the conventional fourth dimension—time—may be profitably incorporated into biological scaling theory. Our goal here is to adopt this literal (rather than metaphorical) four-dimensional (4-D) view of organismic scaling and explore novel predictions arising from it.

Part of our motivation is that even if network geometry explains the prevalence of Kleiber's law, there is considerable variation in the degree to which different subsets of organisms and taxa conform to it (Glazier 2005; White et al. 2007). The field of metabolic ecology, recently "baptized" by Brown and colleagues (Brown et al. 2004), has developed quickly over the last decade and incorporates many previously discovered $\pm 1/4$-power allometries, including those for generation time (Bonner 1965), rate of population increase (Fenchel 1974), population density (Damuth 1987, 2007), and many others discovered and summarized by earlier workers (Peters 1983; Calder 1984; Savage et al. 2004). All exhibit variation and most are interrelated, so that articulating an adequate theoretical account of the empirical complexity of metabolic ecology appears to be a daunting task (Glazier 2005). A 4-D approach reveals order and simplicity not readily apparent in the traditional 3-D view.

Our point of departure is a well-known observation: with respect to body mass (M) in a wide range of taxa,

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most life-history traits scale either as approximately $M^{-1/4}$ (rates of physiological processes and reproduction) or as $M^{1/4}$ (various times, including generation time and life span; Calder 1984; Brown et al. 2004). It is striking that such life-history scaling, when combined with the 3/4 interspecific scaling of metabolism, gives rise to a host of invariants or isometries with respect to body mass (Calder 1984; Charnov 1993). For example, lifetime metabolism scales as $M^{3/4} \times M^{1/4} = M^1$ and thus is proportional (isometric, not allometric) to body size. As a consequence, since mass-specific metabolism scales as $M^{-1/4}$, the lifetime metabolism of each gram of an organism is independent of body size. Though frequently remarked on, this characteristic of the life span is usually considered an outcome of other scaling relationships (Lindstedt and Calder 1981; Brown et al. 2004) and has not been treated as a primary principle of scaling theory—although it has formed the basis of a theory of aging (Pearl 1928). To us, these observations suggest instead that the scaling of lifetimes may reflect a fundamental manner in which organisms of all body masses are ecologically and evolutionarily functionally similar. Thus, we would expect that adding ecological time to scaling theory would simplify the theory with no loss of explanatory power.

Here we build forcefully on this suggestion by defending a simple proposition: it is productive to view organisms as 4-D objects with three spatial dimensions and one temporal dimension that is equal to the generation time. This space-lifetime hypothesis has immediate implications. Scaling now has to be thought of as simultaneous proportional change in all spatial dimensions and in generation time. In this view, 3/4 scaling of metabolism is not at all surprising since the exchange of energy with the environment takes place through a 3-D surface (two spatial and one temporal) and expenditures are correspondingly 4-D (three spatial and one temporal). All the 1/4-power allometries for linear dimensions and life history follow simultaneously from this simple view.

Blum (1977) reasoned similarly that if organisms were literally four-dimensional, then the exponent 3/4 follows easily, but he did not suggest what that fourth dimension should be. Time associated with physiological processes has been treated as an explicit dimension in some physiological models of metabolism (Heusner 1982; da Silva et al. 2006), and it of course plays a key role in many others (e.g., Banavar et al. 2002). However, in this note we are concerned with ecological time—specifically, generation times. Ecological time–related characters have been mentioned in the literature as candidates for a fourth dimension, but this topic has not been explored further (Hainsworth 1981; Calder 1984).

It is a straightforward observation that, to a first approximation, the power of unity in the lifetime metabolic-expenditure isometry ($M^{3/4} \times M^{1/4} = M^1$) is subdivided into approximately equal quarters among the four total temporal and spatial dimensions: life span scales as $M^{1/4}$ and metabolic rate per chronological unit of time as $M^{3/4}$. Purely equal subdivision among the dimensions does not have to occur, and in fact there may be many exceptions. For example, using the database of Froese and Pauly (2000), we determined (L. Ginzburg, unpublished manuscript) that the slope of metabolic rate of fishes, after adjusting for temperature, is 0.84, higher than 3/4. We found that, at the same time, fish generation time scales with the exponent of 0.16, so the lifetime metabolism scales again as power 1. In contrast, mammals show a more even distribution between temporal and spatial dimensions (Calder 1984). Reviews by Atanasov (2005, 2007) show that lifetime metabolism scales approximately as 1 widely within and among taxa, in spite of variations in metabolic exponents.

Generation Time as a Dimension

Why should generation time be so significant that it forms a fourth dimension for organisms? Time units driven by astronomical events do not form a natural timescale for biology. Although organisms may respond to various astronomical cycles, the periodicity of such cycles depends on accidental properties of the solar system and not the functional requirements of biological systems. When we adopt a timescale more suitable for organisms, we would expect it to exhibit a clear relationship to processes important for organismic function and fitness.

Since populations of established species tend to be roughly stable over the long run, the per capita rate of survival to the next generation has to be approximately unity. That is, one surviving daughter of a size equal to its mother has to replace each mother per generation. This is a requirement for ecological and evolutionary success. Constructing one viable and reproductively capable daughter requires a certain duration (a "generation time") that is conveniently viewed as an organism's fourth dimension. So, on average, it takes a generation time of metabolism for a mother to guarantee the existence of her replacement. On this basis, we deduce that the generation time (and correlated lifetime) metabolism should be isometric to body size, as described above. Thus, generation time is a plausible constraint inseparably linked to the size dimensions of an organism through metabolism. The generation time acting as such a constraint may even ultimately determine the values of other physiological or life-history scaling exponents.

It is the average metabolic rate under natural conditions—the field metabolic rate (FMR)—that is most relevant to this 4-D view, since organisms do not typically

live their entire lives at basal, or standard, metabolic rates. However, our analyses are necessarily restricted to using basal rates, since there are too few species for which both published FMR and life-history data are available (Nagy et al. 1999; Anderson and Jetz 2005). In any case, FMR scales roughly parallel to basal rates in vertebrate taxa and is close to 3/4 in placental mammals (Nagy 2005). We expect that the results of using basal rates will thus be comparable to use of FMR directly.

Within species, data on life prolongation due to caloric restriction give an idea of an "exchange rate" of metabolism versus longevity. Reducing caloric input by up to 30% extends life by the equivalent percentage (Weindruch and Sohol 1997). This topic has been investigated among diverse organisms, including mice, protozoans, water flies, spiders, and guppies. We further found that the residuals of the interspecific scaling of basal metabolism and the scaling of maximum life span co-vary negatively (226 species shared by data sets of Ernest 2003 and Savage et al. 2004; correlation coefficient -0.25, $P < .0002$), although the scatter is large. That is, a species that is overmetabolic with respect to the metabolism line has a tendency to be below the line for generation time allometry, and vice versa, as if the 4-D volume matters more than each of the four dimensions separately.

We venture below to make some specific predictions based on our 4-D view. We have been able to test some of them with satisfactory results; others remain conjectures for future testing.

Predicted and Actual Allometries for Subsets of Reduced Dimensionality

First, consider a set of organisms of different sizes that all share the same generation time. This means that one dimension out of four is fixed, and the organisms differ only in three dimensions rather than four. Metabolism in a 3-D system would be expected to scale not as 3/4 but as 2/3, consistent with the reasoning of Rubner (1883) and other pre-Kleiber workers. However, from our 4-D view, the reason that the slope will be different is simply that one dimension has been removed.

An important special case of such 3-D sets is that members of a single species have essentially the same generation time. Thus, we would predict that intraspecific metabolism would scale with a lower exponent, ideally 2/3. This prediction is in complete agreement with the well-known observation that intraspecific scaling exponents for metabolism are often different from interspecific exponents and tend to be closer to 2/3 than to 3/4 (Feldman and McMahon 1983; Glazier 2005; Chown et al. 2007).

Second, note that if, in a 3-D set of organisms, we standardize an additional dimension (e.g., one of the three spatial dimensions, say, body length), we effectively remove two of the four dimensions. By the foregoing reasoning, we would then expect the slope to be 1/2 (i.e., the remaining variability is 2-D).

Substantial data are available to test these predictions for *Homo sapiens*. As a single species, it is 3-D and thus should exhibit a metabolic scaling exponent of 2/3; in fact,

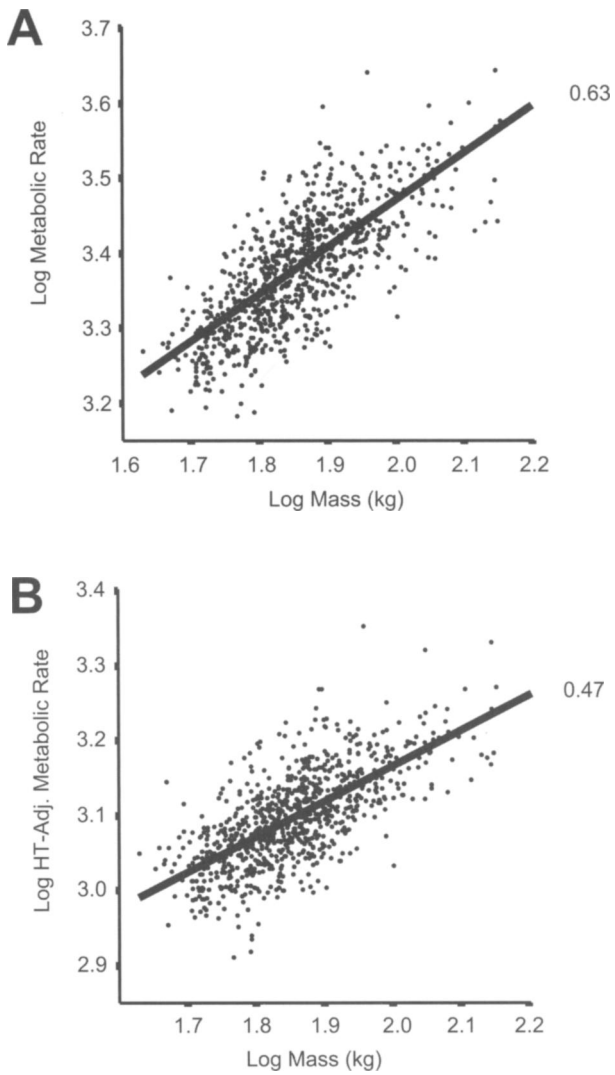

Figure 1: Intraspecific relationship (in humans) between mass and metabolic rate in systems of reduced dimensionality. Data are resting metabolic rate, mass, and height of 890 adult individuals, with metabolism adjusted for age effects; from S. Heymsfield (personal communication). *A*, Metabolism regressed on mass alone agrees with our predicted intraspecific slope of 0.67 (0.63 ± 0.04, 95% confidence interval). *B*, Height-adjusted metabolism plotted against body mass from a multiple regression of metabolism on both mass and height. This multiple regression effectively decreases one spatial dimension by standardizing height (see text), and the partial regression coefficient associated with mass (i.e., the slope in the plot) agrees with our predicted slope of 0.50 (0.47 ± 0.04).

the data we analyzed show the exponent equal to 0.63, with a 95% confidence interval of 0.59–0.67 (fig. 1*A*). We can further reduce the dimensionality by performing a multiple regression of metabolic rate on both mass and height, in which case we would expect a value of 1/2 for the partial regression coefficient associated with mass. In agreement with the prediction, the observed value is 0.47 (0.43–0.51; fig. 1*B*). If, equivalently, we bin the individuals into groups of equal heights (0.01 log height [cm]), the mean slope for the scaling of metabolism within groups gives the same result: 0.47 (0.42–0.52). Standard textbook formulas used in human physiology that regress surface area for humans on their height and weight have the exponents of weight varying between 0.43 and 0.54, in agreement with our own estimate (Dubois and Dubois 1916; Dubin and Zietz 1996; Verbraecken et al. 2006).

We can perform the same test on an interspecific scale across placental mammal species, with some caveats. The mammal data certainly incorporate a wider range of variation in ecological and physiological constraints than do intraspecific data. In particular, it is known that metabolism in small mammals (<50 g) scales with a much shallower slope than it does in large mammals (McNab 1988; Glazier 2005). Accordingly, we will restrict our analysis to species >100 g in body mass, among which the allometric relationships are relatively uniform. By necessity, we also used maximum recorded life span to represent generation time; though an imperfect proxy, life span does scale similarly to the other life-history characters that jointly determine actual generation times (Lindstedt and Calder 1981). Finally, we have not investigated whether phylogenetic nonindependence affects our estimates of slopes. Our interest here is in a direct comparison with the human data for which no comparable genealogical information is available. Moreover, published phylogenetically based and nonphylogenetic studies tend to yield similar exponents for the relevant allometries in mammals, though some life-history traits may be exceptions (Martin et al. 2005; Nagy 2005; Duncan et al. 2007). We expect that the results of a phylogenetically based analysis would be qualitatively the same as ours, but an exploration of this additional complexity is beyond the scope of this work.

Table 1 shows that the results for mammals are similar to those for humans. In the 4-D (unconstrained) case, the metabolic exponent is not different from 3/4, and the 95% confidence interval does not include 2/3. In the 3-D case (controlling for life span), the exponent is lower, but variation is such that it is consistent with either 2/3 or 3/4. In the 2-D case (controlling for both life span and length), the exponent is 0.46, not significantly different from 1/2 and almost exactly the value that we obtained in the intraspecific case (fig. 2).

The focal values of 3/4, 2/3, and 1/2 correspond to integer reductions in dimensionality, and they seem to represent the modal values seen widely in metabolic scaling (Glazier 2005). However, we can easily imagine fractional dimension reduction, which would produce metabolic scaling exponents of various intermediate values. For example, mammals are not perfect cubes, and the slope of the regression of body mass to length tends to be slightly larger (up to 3.6) than the expected 3.0 in most orders (Damuth 1990; Silva 1998). The same exponent is closer to 2.8 for fishes (this note) and for mammalian carnivores (Van Valkenburgh 1990). Thus, constraining by body length would be expected to have different effects in different groups, because slightly more or slightly less than a full spatial dimension contributing to body mass is being standardized.

Actual morphological, developmental, or temporal constraints (as opposed to those imposed statistically by the investigator) may also cause observed metabolic allometries with powers outside of this simple set of $(n - 1)/n$ fractions or with powers unexpected from the apparent dimensionality of the system. For example, the low exponents for metabolic scaling observed in small (<50 g) mammals (1/2 or even 1/3; Glazier 2005) immediately

Table 1: Effect of reduction of dimensionality for metabolic scaling among placental, nonvolant mammals with >100 g body mass

Regression	Exponent	95% CI	Expected value	Dimensionality	*n*
A: BMR vs. mass	.73	.70–.76	.75	4	149
B: BMR vs. mass, life span	.71	.67–.76	.67	3	149
C: BMR vs. mass, life span, and head-body length	.46	.29–.63	.50	2	148

Note: Data include those for the 149 overlapping species from Savage et al. (2004), for basal metabolic rate (BMR); Ernest (2003), for maximum life span; and Silva (1998, personal communication), with some additions from the literature, for head-body length. In all regressions, BMR is the dependent variable. The exponent is the regression coefficient, or partial regression coefficient in the case of multiple regressions, associated with mass. Expected values are those based on consideration of dimensionality (see text). Regressions A–C are illustrated in figure 2; CI = confidence interval.

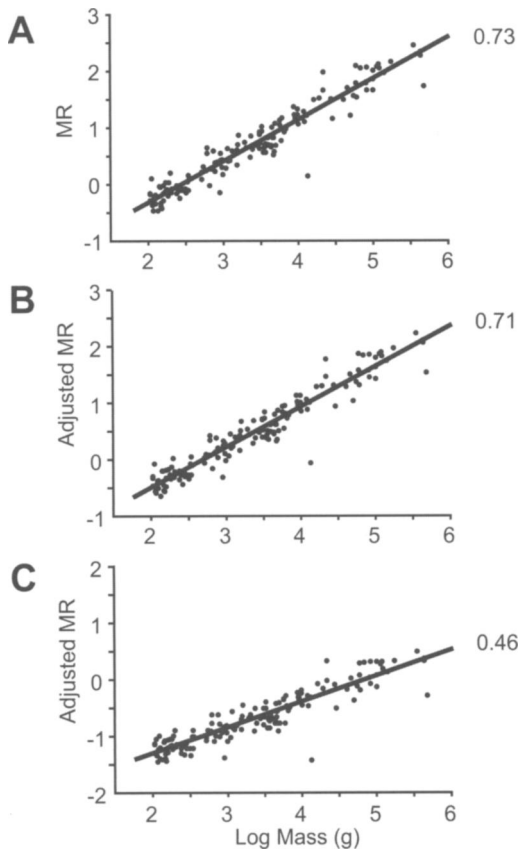

Figure 2: The interspecific relationship (in mammals) between mass and metabolic rate in systems of progressively reduced dimensionality. Data are basal metabolic rate (BMR, in W), mass (g), maximum life span (LS, years), and head-body length (HBL, cm) of 149 species of mammals (over 100 g in body mass). Table 1 gives references and shows data for each regression. *A*, Simple regression of BMR vs. mass; *B*, life span–adjusted BMR vs. mass (adjustment from multiple regression of BMR vs. mass, LS); *C*, HBL- and LS-adjusted BMR vs. mass (adjustment from multiple regression of BMR vs. mass, LS, HBL).

suggest to us that small mammal species effectively form at most a 2-D set. We conjecture that small mammals experience constraints in both spatial and temporal dimensions. We have no suggestions for the source of the apparent reduction by an additional dimension. Nevertheless, the 4-D view allows us to frame a novel question about the system that may lead to further understanding.

Discussion

The space-lifetime view predicts the 3/4 exponent for metabolic scaling across species. Significantly, it also successfully predicts the exponents of metabolic scaling in sets of organisms of progressively lower dimensionality, and it further correctly predicts that intraspecific metabolic slopes will tend to be lower than interspecific slopes—and ordinarily closer to 2/3. Considering these observations and other conjectures discussed above, we suggest that our proposed 4-D view of metabolic scaling is in many ways simpler than the conventional 3-D view but with a similar and, in some cases, superior predictive power.

We are aware that there are multiple explanations within the 3-D framework for many of the same patterns that we address (Glazier 2005). Perhaps surprisingly, we would argue that our theory is not likely to be a competing causal theory, nor does it necessarily contradict existing 3-D theories. We rely, informally, on the concept of duality to suggest how this can be so.

Duality is a widely used concept in modern physics. The two dual theories describe the same facts in different ways, typically by differing by one dimension. In a sense, they are the same theory but distinct formulations that emphasize different aspects or package the ingredients differently (Randall 2005). Neither 3-D nor 4-D metabolic theory has been developed sufficiently to determine whether the theories are formally dual. But it is in the spirit of such a possible duality that we offer our 4-D view. The fact that we do not have a mechanistic 4-D model yet see predictable relationships from that perspective strongly suggests duality with 3-D mechanistic theory rather than an alternative or replacement.

We thus present our view without a mechanistic underpinning. Knowledge of regular patterns in nature without a concurrent understanding of their underlying mechanisms is more common (and useful) in science than people often think (Greene 2001). Darwin's lack of knowledge of the mechanisms of heredity (which we now understand) or physics' lack of a mechanism for gravity (which we still do not understand) are just two examples. Our presentation of a nonmechanistic framework means only that this represents less of an intellectual advance than one would strive for.

When we add generation time to scaling theory as an organism's fourth dimension, we see order involving metabolic exponents that was previously obscured. The exponents depend in a simple way on the dimensionality of the set of organisms being considered: 1/2 for two dimensions, 2/3 for three, 3/4 for four. We believe that our view can serve as an organizing framework, within which various theories and mechanisms may coexist peacefully, occupying their own (sub)space of correctly identified dimensionality. Instead of expecting universal applicability of one of the exponents (e.g., 3/4, 2/3, or 1/2), we expect to see various exponents based on variation in dimensionality. The 4-D view thus embraces network theory, aimed at explaining the central tendencies of interspecific scaling, and simultaneously other approaches, including those involving multiple constraints (e.g., Kooijman 2000;

Glazier 2005; Demetrius 2006) that seek to explain much of the variation in metabolic scaling at various scales and in particular groups. At the same time, the scaling patterns predicted and successfully explained by the 4-D view offer a challenge to traditional theories, which must account for them.

Including the temporal dimension as an integral part of the organism's phenotype may have broader applications in ecology than just those involved with metabolism and scaling. If organisms are considered to occupy a 4-D space, then time, like the dimensions of 3-D space, can be considered a resource. Where time for growth and reproduction is in short supply, it can be viewed as a resource that can be divided, with implications for diversity, resource partitioning, and biogeography. Other ecological processes ultimately depending on reproductive rates (such as population fluctuations and local extinction probability) must depend partly on generation time. We speculate that an extended 4-D view, if confirmed by additional studies, may provide clarification in other areas of theoretical ecology that are currently based in three dimensions.

Generation time has always been the fundamental unit of time for understanding evolution. Our suggested view of metabolic ecology is that a generational timescale is equally fundamental for ecology (Ginzburg and Colyvan 2004). A well-known metaphor by Hutchinson (1965) sets ecology as a theater and evolution as a play. We believe that the theater's clock ticks at the same rate at which the play is being performed. The coincidence of the basic timescale of ecology to that of evolution is another confirmation of the unity of the two fields of biology.

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