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How and Why Does Metabolism Scale with Body Mass?

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Most explanations for the relationship between body size and metabolism invoke physical constraints; such explanations are evolutionarily inert, limiting their predictive capacity. Contemporary approaches to metabolic rate and life history lack the pluralism of foundational work. Here, we call for reforging of the lost links between optimization approaches and physiology.

constraint; life history; metabolic rate; optimization; scaling

Introduction

Organisms vary in size to an almost impossible degree. The heaviest animal is the blue whale *Balaenoptera musculus*, with a maximum recorded weight of 190 tonnes (1). The smallest vertebrates include a New Guinea frog, *Paedophryne amauensis* (adult length ~7 mm; Ref. 2), and a fish, *Paedocypris progenetica* (adult length ~8 mm; Ref. 3), The smallest microbes weigh 0.1 pg. Living organisms therefore span at least a 10^{21}-fold size range. Size ranges for any one species are narrower, but still impressive. Giant clams *Tridacna gigas*, for example, vary by >11 orders of magnitude, from 100-µm eggs that weigh micrograms to 200-kg adults. Given the vast scales involved, it is hardly surprising that the influence of size has been well studied (4–9). Perhaps the most studied size relationship is the allometric (nonproportional) scaling relationship between an animal's size and its rate of energy use (metabolic rate).

How Does Metabolic Rate Scale with Body Mass?

The earliest attempts to link metabolic rate and body mass were rooted in assumptions about the physical principles that govern heat exchange rather than actual measurements. In a series of presentations to the Royal Academy of Medicine in Paris in the 1830s, Sarrus and Rameaux suggested that, because the heat produced by an animal as a by-product of metabolism must be lost through the body surface, the rate at which it produces heat (metabolic rate) should be proportional to the surface area over which the heat is lost, rather than body mass (10). Thus, metabolic rate was thought to scale as a power function of mass, with a scaling exponent of ~2/3 (i.e., metabolic rate was predicted to be proportional to mass$^{2/3}$).

In 1883, Rubner (11) reported that the rate of heat production of dogs scaled in proportion to their body surface area, and this two-thirds power scaling of metabolic rate came to be known as Rubner's surface law. In 1932, Kleiber (12) and Brody and Proctor (13) independently reported that the scaling exponent of metabolic rate of endotherms (birds and mammals) was >2/3, and a value of ~3/4 was subsequently adopted (in part because it simplified calculations undertaken with a slide rule; Ref. 8). In 1960, Hemmingsen (14) expanded the observation of three-quarter power scaling to a wide range of species. But in the decades that followed, it became clear that 3/4 scaling is not universal. Some studies continued to report a scaling exponent close to 2/3 for the basal metabolic rate of endotherms into the early 2000s (15–20), for example, whereas other studies reported a range of other values (10, 12, 13, 21–33). It has also been shown that scaling relationships may not follow a strict power function both within (e.g., Refs. 34, 35) and among (e.g., Refs. 26, 36–38) species.

The most reliable finding from more than a century of study is that metabolic rate almost always scales hypoallometrically with body mass (i.e., it exhibits a scaling exponent < 1), but there is no single "universal" scaling exponent that defines this relationship (e.g., Refs. 39, 40). Some consistent patterns have also emerged. For interspecific (among species) comparisons, the basal metabolic rate of endotherms (birds and mammals) scales with a shallower exponent than the standard metabolic rate of ectotherms (e.g., Refs. 33, 41–43). The maximum metabolic rate of endotherms scales with a steeper exponent than basal metabolic rate (29, 44–48), but the exponents of maximum and standard metabolic rate are similar for ectotherms (49). When metabolism is measured in the field, it tends to scale more steeply than (laboratory measured) standard metabolic rate in reptiles (50, 51), but no such differences occur in endotherms (47, 51–54). A striking deviation from hypoallometric scaling is observed among prokaryotes and protists, for which metabolic rate scales hyperallometrically (exhibits a scaling exponent > 1) or isometrically with mass, respectively (43, 55).

For the intraspecific (within species) scaling of metabolic rate with body mass, the average scaling exponent

of metabolic rate is strikingly close to 3/4 (56, 57), but there is considerable variation about this value, which is driven by diet, light intensity, oxygen availability, pH, salinity, starvation, temperature, water availability, and inbreeding, among others (reviewed in Refs. 58, 59). Most recently, the scaling exponent of metabolic rate has been shown to be negatively associated with growth performance and positively related to maximum reproduction rate (57). Some of the variation in the scaling exponent is certainly attributable to measurement error, such that exponents estimated with larger sample sizes and wider mass ranges are more precise (42, 57, 60). Examination of a recent dataset (57) also provides evidence of substantial publication bias: there is a conspicuous absence of hyperallometric scaling exponents for datasets with small mass ranges and also an absence of very shallow scaling exponents (FIGURE 1). These absences question the representativeness of published exponents collected for mass ranges smaller than ~1 order of magnitude, because these published values are likely to represent a nonrandom subset of the real values.

The consensus from the above is that, at least for metazoans, metabolic rate usually scales hypoallometrically across a wide range of physiological states and environmental contexts. But why such allometric scaling arises so frequently and consistently in biology has been hotly debated for decades with no clear resolution (39, 40, 58, 72–74).

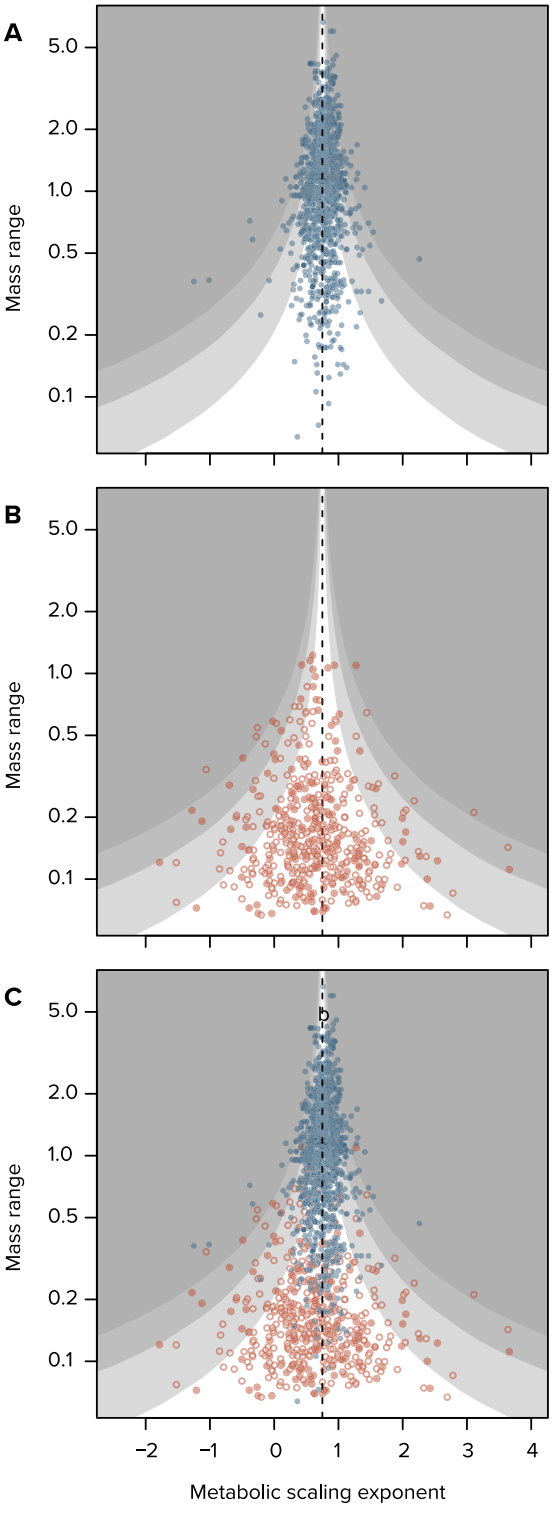

FIGURE 1. **Funnel plot showing the relationship between the intraspecific scaling exponent of metabolism and the mass range over which it was determined**

Funnel plot showing the relationship between the intraspecific scaling exponent of metabolism and the mass range over which it was determined for published (*A*, blue) and unpublished (*B*, red) data. (*C* shows both the published and unpublished data.) Published scaling exponents were taken from a recent compilation (57). Unpublished scaling exponents were calculated from raw data for species of insect, fish, amphibian, reptile, bird, and mammal provided in publications associated with our own research (open red symbols) (Refs. 33, 61, 62, 64–68, 145) and the wider literature (filled red symbols) (52, 69–71). The vertical dashed line represents a scaling exponent of 0.75, and the shaded areas delimit the 2.5th and 97.5th percentiles of the distributions of scaling exponents estimated for 50 mass ranges from 0.01 to 10 orders of magnitude. Relationships were simulated by generating a dataset with 17 data points (the median sample size in the published dataset) spanning the appropriate mass range and a mean metabolic scaling exponent of 0.75 and then adding normal deviates with a mean of 0 and a SD of either 0.3 (darker gray), 0.2 (intermediate gray), or 0.1 (lighter gray) units on a $\log_{10}$ scale. Each combination of mass range and residual standard deviation was simulated 100,000 times.

Why Does Metabolic Rate Scale with Body Mass?

Physical Geometric Constraints

Size is a physical property as well as a biological one. In trying to understand how size affects the function of organisms, classical approaches often emphasize the role of physical properties. In an early exploration of the relationship between size and function, Galileo argued in 1638 (75, 76) that the mass of an animal's skeleton must increase disproportionately with body mass, because the cross-sectional area of a bone must increase in proportion with the weight that it

supports. D'Arcy Thompson (77) similarly placed a heavy emphasis on physical explanations for size-function relationships. Such an emphasis is understandable, because the vast scales over which life exists result in very substantial changes in the dimensions of organisms, and these may impose strong constraints on function. Accordingly, multiple theories have assumed that physical constraints dictate the size dependence of metabolic rate. Below we deal with each very briefly (for a fuller account, see the papers that we reference) but emphasize the physical constraints that each theory base emphasizes.

Sarrus and Rameaux's prediction that metabolic rate should be proportional to body surface area (10), and therefore to mass$^{2/3}$, was the first attempt to understand how metabolic rate should be related to body mass. Similarly, heat dissipation limit theory (54) suggests that the daily energy expenditure of free-living endotherms is constrained by the capacity of animals to dissipate metabolically produced heat.

Dynamic energy budget (DEB) theory (78–81) separates the mass of an organism into "structure" and "reserve," where the latter is "…conceptualised as stored polymers (fats, carbohydrates and lipids) in 'blobs' within individual cells that must be accessed by the structure from the surface of the blobs at the subcellular level" such that "…the surface area interface of the reserve with the structure is a fundamental geometrical driver of the dynamics in DEB theory via the process of reserve mobilisation" (Ref. 82, p. 563). Food is assimilated into the reserve in proportion to surface area (volume$^{2/3}$), and the rate of mobilization of reserve depends on the surface area/volume interface of the reserve (83). Thus, two key assumptions of DEB are that assimilation and mobilization are proportional to the area of a surface.

Fractal network theory (84–88) proposes that the distribution of resources (most likely oxygen; Ref. 83) through a space-filling outward-directed fractally branching distribution network constrains the metabolic rate to that which minimizes the energy required to distribute resources. Thus, in fractal network theory the geometry of the circulatory system dictates the scaling of whole organism metabolic rate.

Gill-oxygen limitation theory (89–93) argues that the allometric scaling of gill surface area sets the allometric scaling of oxygen uptake rate (and therefore aerobic metabolic rate) for water-breathing ectotherms, because "…gill surface area cannot grow in three dimensions and thus cannot keep up with the 3D body that it supplies with oxygen" (93). Thus, in gill-oxygen limitation theory the scaling of whole organism metabolic rate is dictated by the geometry of the respiratory exchange surface.

These physical geometric theories may provide a proximate explanation for size-dependent patterns in biology, though this is hotly debated (38–40, 58, 72–74, 93–99). If they do provide reasonable descriptions of how organisms work, then they might also provide reasonable predictions of the proximate responses of animals to changes in their environment. The validity of the physical constraints invoked by each of these theories can and should be debated, but such debates overlook what is, at least for us, a critical shortcoming of all these theories: their outputs are all evolutionarily inert. That is, one could ask each of these theories to predict the consequence of fourfold increase in extrinsic mortality, and unless the physical environment also changed their model functions and outputs would remain unchanged. In other words, these models ignore how fitness (co)varies with evolutionary drivers that are unrelated to physiology. As biologists, we know life does not work that way: shifts in the selective milieu induce evolutionary change, organisms adapt such that their physiologies and life histories are altered. We would argue that there is a need to predict the evolutionary responses of organisms to environmental change, and that constraint-focused theories are ill suited to address this urgent problem we confront.

Life History Optimization

Rather than making assumptions about physical constraints, an alternative approach is to ask: what metabolic relationship might evolution favor? Life history theory (100–107) provides an analytical framework for understanding how different components of an organism's life affect the overall performance of that organism. Life history theory considers how evolution has shaped the way in which resources are allocated to the various functions of life and often focuses on the fitness consequences of different allocations, for example, to growth versus reproduction, producing many small versus few large offspring. One can use this approach to construct models based on life history optimization that can be used to predict the distributions of traits, be they metabolism, size, or their covariance with each other (108–111).

In the context of life history evolution, describing a combination of traits as "optimal" is shorthand for saying that those are the trait combinations that yield the highest fitness (112). The life history optimization approach has been applied to the study of size-dependent patterns in biology to predict, for example, optimal age and size at maturity (108), the among-species scaling of metabolic rate (and a range of other traits) with body size (109, 113), the within-species scaling of metabolic rate with body size (57, 112), and latitudinal gradients in age at maturation and the scaling of reproduction rate with body mass (114).

Before exploring the predictions of life history optimization with regard to metabolism, we must clarify a few important issues. First, the massive size range that exists among animals has exerted a strong gravitational pull on considerations of metabolic scaling. Many of the arguments for drivers of metabolic scaling emerge from among-species comparisons, which may explain why

many metabolic theories are evolutionarily inert: they focus on the products of evolution (i.e., among-species patterns) rather than the processes that yielded those patterns. But, as Kozłowski and others have pointed out (e.g., Refs. 109, 111, 115), among-species relationships provide very little information about within-species relationships. Accordingly, life history optimization incorporates within-species data and only makes predictions about within-species metabolic scaling.

Second, there is a tendency to consider metabolic scaling as a factor that is somehow separate from metabolic rate; indeed, physiologists often use different terms to describe the relationship between metabolism and size (i.e., metabolic scaling) and size-independent metabolism (often termed "metabolic level"). Distinguishing between metabolic level and scaling can sometimes be useful, but we think this distinction is somewhat artificial from both statistical and biological perspectives. Statistically, a negative covariance between metabolic level and scaling emerges as a statistical artifact (116). Biologically, within-species metabolic scaling functions are essentially descriptions of the ontogenetic trajectory of metabolism; breaking this continuous trajectory into two separate components seems odd to us. Thus, in our discussions of metabolic scaling and life history optimization, we discuss both metabolic level and scaling but really consider them to be two parts of a whole.

How then does life history optimization predict that metabolism should evolve under environmental change? The fulcrum upon which this model pivots is the risk of mortality. Changes to mortality risk are predicted to alter the fitness returns of life history events such as reproductive maturity and the fitness returns of rates such as growth and offspring production (57). To illustrate for a single hypothetical species, if mortality increases, life history optimization would predict that faster growth rates and earlier reproduction are favored. But to achieve faster growth and earlier reproduction, the model predicts that metabolic scaling becomes lower, as would the scaling of the relationship between body size and reproductive output (reproductive scaling). Finally, the model would predict that, all else being equal, metabolic level would increase and life span would decrease. This example illustrates the strong connection between metabolic traits and fitness components: changing one changes all of them. Thus, our model (57) would predict that anthropogenic stressors that increase mortality rates will impact not only the timing of life history events but also the energy fluxes that drive these events. We should therefore observe metabolic evolution when mortality rates increase. For example, the model would predict that fish species that have adapted to heavy fishing regimes should show evolutionary changes in their metabolic rates, from lower levels and higher scaling to higher levels and lower scaling relative to unfished species. This simple prediction awaits formal testing, but it illustrates how changes in mortality regimes, independent of change factors that might classically be expected to alter physiology (e.g., temperature), should generate evolutionary changes in rates of metabolism. In other words, the model emphasizes that metabolism, like any other trait, is evolutionarily labile. We think that life history optimization therefore holds tremendous promise for predicting how metabolism will coevolve with body size and life history with further anthropogenic change.

The Fragmented Field of Metabolic Scaling Research

Whereas we favor the use of life history to understand variation in metabolic scaling, many of the goals of life history theory are shared by other theories. Each seeks to understand growth and/or reproduction (FIGURE 2, A–D), for example, but each approaches the problem from a different perspective. The metabolic theory of ecology (MTE) builds on fractal network theory and posits that the metabolic rate of organisms is the fundamental biological rate, which governs most patterns in ecology (84, 122, 123). MTE predicts how metabolic rate controls ecological processes at all levels by determining resource uptake and allocation for survival, growth, and reproduction. Pace of life (POL) theory views behavior, physiology, and life history as interconnected components of a single integrated phenotype (120, 121). A slow POL is characterized by a long life span, slow development, and delayed reproduction, whereas a fast POL is characterized by a short life span, fast development, and immediate investment in reproduction. POL is strongly related to and sometimes simply reiterates classic life history theory, but physiologists seem to find POL more appealing because of its emphasis on physiological traits, based on the premise that "...the interaction between environment and fitness is mediated by behavioral and physiological responses, including basal metabolic rate (BMR), field metabolic rate (FMR), and testosterone..." (Ref. 120, p. 466). We would argue that integrating POL into classic life history theory would benefit both fields, as the former leverages new methodological techniques while the latter has an august history of deep thought and theoretical sophistication.

Fundamentally, life history theory and POL approach the problem from the perspective of optimization, whereas theories like DEB and MTE approach the problem from the perspective of constraint. To better understand how these intellectual traditions interact and exchange knowledge, we used a research weaving approach (117) to synthesize the literature associated with each of these theories (FIGURE 2). The analysis reveals that citations are strongly clustered by approach, although the optimization and constraint-based theories are not much more distinct than

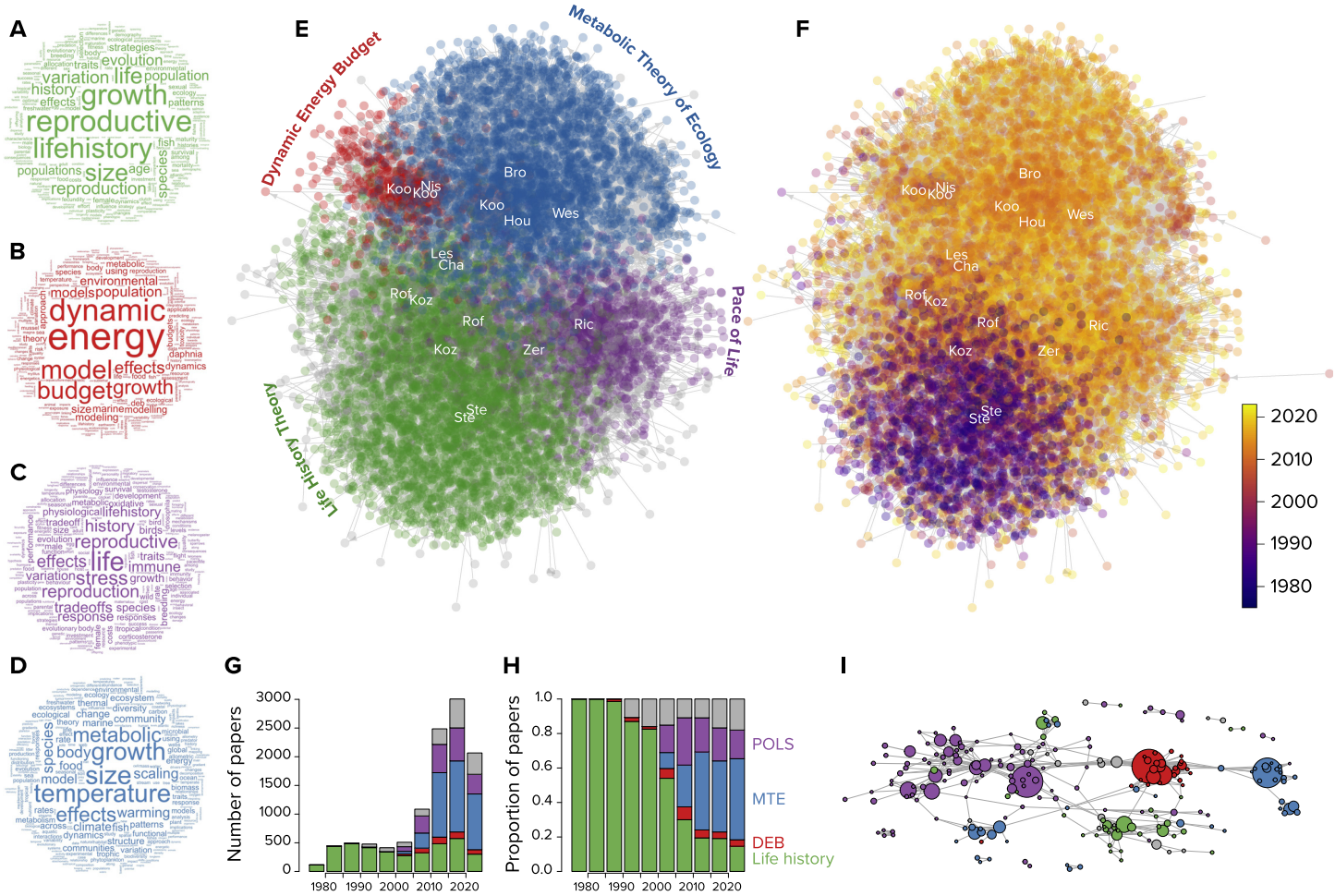

FIGURE 2. **Research weaving approach**

A research weaving (117) approach to understand the connections among life history theory, dynamic energy budget theory (DEB), pace of life theory (POLS), and metabolic theory of ecology (MTE). The candidate set of publications were identified with the prominent seed publications for life history theory (Ste, Les, Cha, Rof) (100–107), DEB (Koo, Nis) (78, 81, 118, 119), POLS (Zer, Ric) (120, 121), and MTE (Bro, Hou, Wes) (84, 122, 123), and each publication was coded to one of these theories based on the papers it cites: a paper was coded to a theory if it cites the seed papers for that theory and no others. *A–D* show word clouds generated from the titles of papers associated with each theory. *E* and *F* show the citation network for the papers, colored by theory base (*E*) or publication date (*F*). *G* and *H* show the frequency distributions of publications, colored by theory base. *I* shows a collaboration network for the most published authors in the candidate set of publications, with nodes colored by the predominant theory base cited by that author.

each of the theories within these categories (FIGURE 2E). Thus, although classic life history theory has seniority (FIGURE 2F), the more recent proliferation of new theories (FIGURE 2, G AND H) with related goals has given rise to a set of rather insular citation (FIGURE 2E) and collaboration (FIGURE 2I) networks.

The apparent divide between optimization and mechanistic frameworks for understanding life histories did not always exist. For example, R. A. Fisher, one of the founders of modern evolutionary biology, recognized the importance of physiology for the evolution of the life history in 1930: "It would be instructive to know not only by what physiological mechanism a just apportionment is made between the nutriment devoted to the gonads and that devoted to the rest of the parental organism, but also what circumstances in the life history and environment would render profitable the diversion of a greater or lesser share of the available resources towards reproduction" (Ref. 124, p. 43–44). And though D'Arcy Thompson hoped for physical explanations "[f] or the main features which appear to be common to all curves of growth. . ." (Ref. 77, p. 152), he also mused on the consequences of resource allocation: "After its last moult the stick-insect puts on more weight for a while; but growth soon draws to an end, and the bodily energies turn towards reproduction" (Ref. 125, p. 164), a view that is a decidedly life history-based perspective about allocation.

We view the emergence of distinctions between subdisciplines as unhelpful. The separation of pace of life syndromes and life history theory (FIGURE 2E) seems particularly unnecessary and serves only to draw physiologists away from classic life history theory, and vice versa. An example serves to illustrate the problems that such distinctions might introduce: past life history work has considered the optimization of complete energy budgets including rates of energy allocation to, e.g., ingestion, metabolism, excretion,

growth, and reproduction (e.g., Ref. 126), but the more physiological elements of the budget (e.g., standard metabolism) were incorrectly assumed to be inflexible and therefore not subject to optimization (127). In contrast to this assumption, decades of physiological work has demonstrated that metabolic rates are phenotypically plastic (e.g., Refs. 61, 62, 128), heritable (e.g., Ref. 129), and evolutionarily labile (e.g., Refs. 130, 131, 145). In any scientific endeavor, there is a perennial risk that one subfield will reinvent the wheel of another's: the lack of communication among subfields studying metabolism and life history is to everyone's detriment.

We think we need a little pluralism back: constraint-driven models would benefit from including optimization. And we think that examining how optimization changes in the face of absolute constraints (e.g., organisms cannot be infinitely large or small or quick, and metabolic rates cannot be zero or infinitely high) would add some necessary limits. We conclude our review by drawing attention to a suite of tools that we think can be fruitfully applied to understand the origin of allometric scaling. Importantly, these tools can be usefully applied to test hypotheses from the perspective of both optimization and constraint.

Future Directions

For the sake of discussion, let us allow that metabolism and its scaling with body mass is, at least partly, the product of life history optimization. What would that mean for the trait and our path forward for understanding it? We would argue that metabolic rate being an outcome of optimization both simplifies and complicates its evolution and simplifies and complicates our understanding of the problem.

First, if metabolic scaling is a product of life history optimization, then metabolic rate, like any other trait, will evolve in response to selection (e.g., Refs. 130, 131, 145). We can therefore use standard but powerful evolutionary approaches such as quantitative genetics and selection analyses to understand heritability and fitness consequences of variation in metabolic rate (e.g., Refs. 129, 132, 133). Using these approaches avoids the need to invoke strong, largely untested assumptions about why metabolism might scale with size in the way it does; instead, we can simply explore the fitness consequences and evolutionary constraints of a particular scaling. Furthermore, life history theory has successfully predicted how and why traits might change with shifts in evolutionary pressures; using this approach might provide insights into how metabolic rate might evolve in response to global change or anthropogenic pressures. In contrast, most constraint-based explanations for metabolic scaling tend to be more retrospective than prospective. Notably, constraint-based theories offer few predictions of how changes in the selective milieu will alter metabolic rate specifically. For these reasons, using life history optimization, to us at least, offers tremendous promise for gaining a better understanding of metabolic scaling now and how it will change in the future.

Quantitative genetics approaches can describe the heritability of a trait very well (134–137) and explicitly accommodate the multidimensionality of heritability. Likewise, selection analyses formally link trait variation to fitness outcomes (e.g., Ref. 138). Together, understanding the heritability of and selection on a trait allows us to predict changes in the trait across generations, using either the multivariate breeders equation or the Price–Robertson identity. But even these powerful approaches wilt under the demand of predicting evolutionary change across more than a few generations: instead, we must turn to population genetics theory, an intimidating theory base that requires parameters that are often unknowable for most species other than model microorganisms (e.g., the distribution of fitness effects). Despite these limitations, we still believe that using life history optimization as a framework for understanding metabolic scaling has the greatest promise.

Many discussions about metabolic rate, the pace of life, and even constraints on metabolism can be made soluble by approaching them with microevolutionary tools. For example, the idea that metabolic rate is strongly constrained to have a particular scaling implies that there is strong stabilizing selection about a particular scaling value (33); this is an invocation of selection that can be tested empirically (139, 140). Similarly, the idea that an evolutionarily favored outcome is inaccessible because of constraint is testable by examining whether genetic variation in that trait exists and is aligned with selection (141, 142). Finally, arguments about whether a slower or faster metabolic rate is favored in different habitats or conditions and whether different metabolic rates covary with different life history strategies can be resolved with estimates of selection on metabolic rate and estimates of genetic covariance between metabolism and life history traits, respectively. We believe that by bringing to bear the venerable traditions of quantitative genetics we can greatly simplify and formalize conceptual arguments about how metabolic rate (co)varies with other traits and fitness to understand and predict its evolution.

Ironically, if metabolic scaling is the product of life history optimization, then metabolic scaling is less constrained from a mechanistic perspective but might be more constrained from an evolutionary perspective. If we set aside the idea that metabolic scaling shows the patterns it does because physics constrains it to show those patterns, then metabolism is free to vary much more than traditionally appreciated (57). But, as Walsh and Blows (142) have emphasized, traits are subject to a complex web of selection and genetic covariances such that the capacity for traits to evolve quickly is very limited. To explain, even if a trait is subject to strong selection, it may not be able to evolve

because it genetically covaries (via pleiotropy, linkage disequilibrium, and other equally ghastly genetic processes) with other traits that also have strong fitness consequences. The more traits that covary with our trait of interest, the more complex that multidimensional trait is, and the more evolutionarily constrained that trait is likely to be. Worse, population genetics, specifically Fisher's geometric model (124, 143, 144), tells us that the closer a trait is to its fitness optimum, the less likely it is that any subsequent mutation will be beneficial. As such, traits are far less free to vary than we might intuit: changing a trait might have manifold fitness consequences such that it is evolutionarily static. As overwhelming as this concept can seem, from it a simple conclusion emerges: changing metabolism is unlikely to be selectively neutral. Instead, we are better off viewing metabolism as a trait that is likely to be highly integrated with a host of others such that changing metabolisms changes fitness. Einstein famously said that God does not play dice with the universe; neither would they be likely to play dice with metabolism. This integrated view of metabolic rate expands the scope but increases the complexity of factors that might shape metabolism and its scaling. Our model (57, 112) identifies the components that we believe are key—reproduction, growth, and life span—but it is important to recognize that metabolic rate likely covaries with myriad factors and univariate approaches will inevitably underestimate the complexity of the evolutionary processes shaping metabolic rate. ■

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