Linking scaling laws across eukaryotes

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Scaling laws relating body mass to species characteristics are among the most universal quantitative patterns in biology. Within major taxonomic groups, the 4 key ecological variables of metabolism, abundance, growth, and mortality are often well described by power laws with exponents near 3/4 or related to that value, a commonality often attributed to biophysical constraints on metabolism. However, metabolic scaling theories remain widely debated, and the links among the 4 variables have never been formally tested across the full domain of eukaryote life, to which prevailing theory applies. Here we present datasets of unprecedented scope to examine these 4 scaling laws across all eukaryotes and link them to test whether their combinations support theoretical expectations. We find that metabolism and abundance scale with body size in a remarkably reciprocal fashion, with exponents near ±3/4 within groups, as expected from metabolic theory, but with exponents near ±1 across all groups. This reciprocal scaling supports "energetic equivalence" across eukaryotes, which hypothesizes that the partitioning of energy in space across species does not vary significantly with body size. In contrast, growth and mortality rates scale similarly both within and across groups, with exponents of ±1/4. These findings are inconsistent with a metabolic basis for growth and mortality scaling across eukaryotes. We propose that rather than limiting growth, metabolism adjusts to the needs of growth within major groups, and that growth dynamics may offer a viable theoretical basis to biological scaling.

macroecology | biological scaling | metabolic theory

Scaling laws relating body mass to a variety of species characteristics are among the most general quantitative patterns in biology (1–3). These scaling laws encompass such core ecological characteristics as metabolism (1, 2, 4–9), abundance (10–13), growth (14–18), and mortality (1, 2, 19, 20). The relationship between a species characteristic (y) and its body mass (m) is often expressed as a power law, $y = cm^k$, where c is a constant for a given variable and k is a dimensionless scaling exponent, given by the slope of a straight line on a plot of $\log y$ vs. $\log m$. Many of these relationships are increasingly used to better understand and make large-scale predictions of the effects of the most critical global environmental problems, since they represent simple and efficient predictors of fundamental variables that hold across broad taxonomic groups (1, 2). Moreover, these relationships raise basic and persistently enigmatic problems: how are they linked, and where do they originate?

Body mass scaling research largely began with the study of basal metabolism across mammals, which found an exponent near $k = 3/4$, known as Kleiber's law (1, 2, 9, 21) and termed "allometry" ($k \neq 1$). This value did not match expectations of a constant energy flux per unit tissue mass ("isometry"; $k = 1$) nor of surface-volume constraints on heat dissipation over the surface of geometrically similar body plans ("surface law"; $k = 2/3$). These mismatches provoked questions as to the origin of near 3/4 metabolic scaling but also the generality of the exponent, which is now known to depend on many factors, including metabolic activity level, taxonomic group, taxonomic level, body mass range, temperature, other environmental conditions, life stage, and regression methods (1, 6, 8–10, 22, 23). Despite these many factors, an exponent value of 3/4 has become a canonical expectation for body mass scaling, especially as additional taxonomic groups and additional species characteristics have been found to scale with similar or related values (1–3, 7, 17, 18).

Most prevailing theories of metabolic scaling (with $k < 1$) are based on physical constraints on the structure of a body (3, 9, 24, 25), which in turn are thought to constrain the scaling of other variables with body mass (1, 2, 7, 11, 15, 18, 20, 26). For example, limits on energy supply can proximally limit abundance (2, 10–12) and the energy allocated to growth and reproduction (1, 2, 15, 18, 26–28). Metabolism is also known to produce harmful byproducts that hasten senescence and shorten life span (2, 9, 29). However, the many dependencies of the metabolic exponent, listed above, suggest that metabolism is also flexible and can adjust to different factors, some of which have been found to be growth factors. This has prompted some authors to suggest that basal metabolic scaling adjusts to growth rather than exerting fundamental control on growth and other characteristics (9, 14, 23, 27, 30–32).

One way to better understand the origin of near 3/4 scaling is to consider the generality of circumstances over which such scaling holds. For example, similar scaling both within and across taxonomic groups is consistent with a single underlying process, whereas different scaling regimes within or across groups are

Significance

Metabolic scaling theory has had a profound influence on ecology, but the core links between species characteristics have not been formally tested across the full domain to which the theory claims to apply. We compiled datasets spanning all eukaryotes for the foremost body mass scaling laws: metabolism, abundance, growth, and mortality. We show that metabolism and abundance scaling only follow the canonical ±3/4 slopes within some taxonomic groups, but across eukaryotes reveal reciprocal near ±1 slopes, broadly supporting the "energetic equivalence rule." In contrast to metabolism, growth follows consistent ∼3/4 scaling within many groups and across all eukaryotes. Our findings are incompatible with a metabolic basis for growth scaling and instead point to growth dynamics as foundational to biological scaling.

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consistent with multiple factors dominating under different circumstances but inconsistent with a single universal process.

In this work, we tested several of the principal assumptions and/or predictions of metabolic theories in ecology by linking scaling relationships for different characteristics, with a view toward the ultimate origin of ~3/4 scaling. These theories assume that metabolism is the "master rate," thus limited solely by physical factors related to body mass and so should apply to all eukaryotes. The most comprehensive test of current theory thus calls for data over the full eukaryote domain. Here we first consider the data for each characteristic separately and then examine the links between them.

Scaling Across Eukaryotes

We compiled data across eukaryotes for 4 core ecological variables, comprising 22,761 estimates drawn from nearly 2,800 published sources and meta-analyses (Fig. 1 and Table 1). These data are of varying quality, with unequal representation across the size spectrum. While data for mammals and birds are generally extensive, data are particularly limited for unicellular protists and less complete for some groups of invertebrates. Data often exhibit considerable residual variability, limiting the accuracy with which exponent values can be determined within particular taxonomic groups. At the largest scales, however, estimated exponent values are typically robust to different assumptions, measurement techniques, and regression methods, as well as more specific considerations (*Methods* and *SI Appendix*). These relationships offer a comprehensive view to distinguish the scaling across and within major groups, which helps delimit the generality of any underlying process.

Metabolism (W). Consistent with prior work (1, 2, 7, 21), we find that basal metabolism scales near 3/4 within some major groups (Kleiber's law), but when viewed across 20 orders of magnitude, it is clear that distinct shifts in elevation occur between major and minor groups (e.g., ectotherms and mammals). These shifts are such that across all taxa, metabolism scales near isometrically, with an exponent near 1 ($k = 0.96$; Fig. 1*A*) (4, 5). These shifts between groups are partially reduced by correcting metabolism to a standard temperature (2, 5, 6), but even correcting endotherm metabolism still results in slopes $k > 0.92$ across the full eukaryote domain (*SI Appendix*, Fig. S4). This implies that mass-specific basal metabolism is strongly bounded across eukaryotes (Fig. 2*A*), consistent with previous studies on smaller but equally broad datasets (4, 5).

Abundance (Individuals/m^2). Abundance (i.e., population density of a species) data are drawn from different ecosystems globally, with each estimate representing a snapshot for a species or an average value of several such points (*SI Appendix*). Consistent with prior work (1, 10, 11, 13), we find that abundance scales with body mass near −3/4 within some major groups (Damuth's law) (2, 10, 11, 13), but that across eukaryotes, the exponent is closer to −1 ($k = -0.95$; Fig. 1*B*). Surprisingly, we find that the slopes within groups and the shifts in elevation between groups are largely reciprocal to basal metabolism (Fig. 1*A*).

Species abundance is known to vary greatly through time or along an environmental gradient, and thus it is not surprising that the abundance-mass relationship reveals large residual variation. Contributing to this residual variation is the trophic level of a species, with, for example, carnivorous mammals approximately 10-fold less abundant than herbivorous mammals (Box 1), which

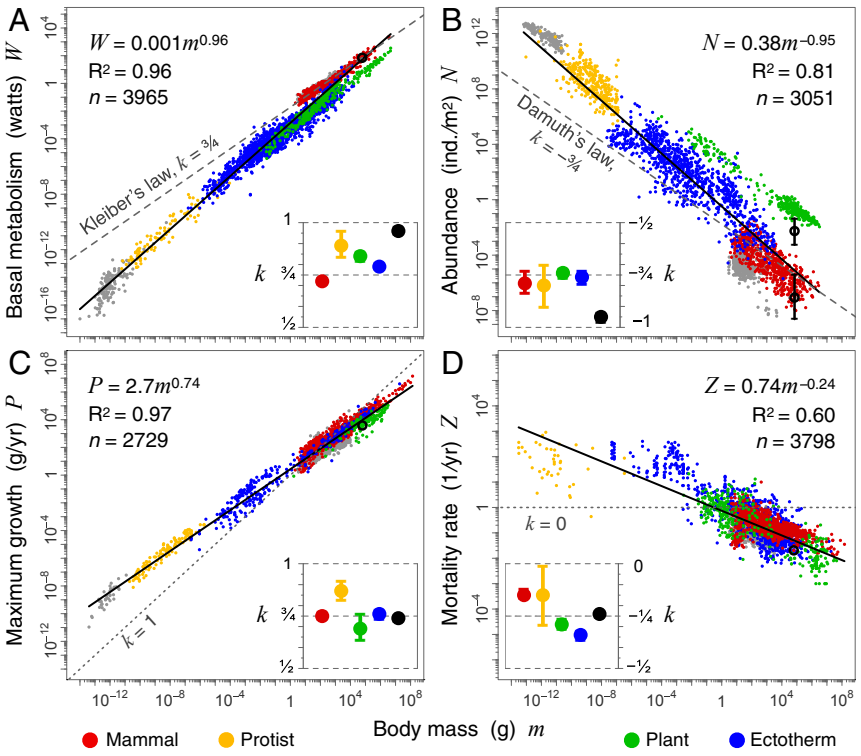


Fig. 1. Scaling of basic variables with body mass. (*A*) Basal metabolism, (*B*) population abundance, (*C*) maximum growth, and (*D*) mortality rate. Points (*n*) in all plots (*A–D*) are separate species values, except for plants represented by multiple points for the same species. Ectotherms and protists were also not aggregated into species values in *D*, due to limited mortality data among smaller sized species. For illustration, we have split eukaryotes into mammals, protists, plants, and ectotherm vertebrates and invertebrates. More resolved groups down to taxonomic orders are detailed in *SI Appendix*, Figs. S1–S3 and Tables S1–S4. Birds (gray points) often have similar rates to mammals and thus are difficult to see in the plots (*SI Appendix*). We show bacteria (also gray points) for reference, where available, but limit our discussion to eukaryotes. Scaling exponents *k* and 95% CIs are shown in the insets for major groups (these exclude birds and bacteria). Black empty circles are humans, with ranges shown in *B* for cities and hunter-gatherer communities (not included in the analysis).

Table 1. Scaling of basic, transformed and combined variables.

			Units	Expected Relation	Expected Scaling	Observed Scaling	C.I.
Basic variables							
i.	Metabolism	W	watts	$W \sim m^{\alpha}$	0.75	0.96	± 0.006
ii.	Abundance	N	ind./m²	$N \sim m^{-\alpha}$	–0.75	–0.95	± 0.016
iii.	Growth	P	g/yr	$P \sim m^{\alpha}$	0.75	0.74	± 0.005
iv.	Mortality rate	Z	1/yr	$Z \sim m^{\alpha-1}$	–0.25	–0.24	± 0.006
Transformed variables							
i.	Specific metabolism		watts/g	$W/m \sim m^{\alpha-1}$	–0.25	–0.04	± 0.006
ii.	Population biomass		g/m²	$Nm \sim m^{1-\alpha}$	0.25	0.05	± 0.016
iii.	Growth rate		1/yr	$P/m \sim m^{\alpha-1}$	–0.25	–0.26	± 0.005
iv.	Lifespan		yrs	$1/Z \sim m^{1-\alpha}$	0.25	0.24	± 0.006
Combined variables							
H1.	Population metabolism		watts/m²	$WN \sim m^{0}$	0	–0.01	± 0.013
H2.	Lifetime growth		unitless	$P/m/Z \sim m^{0}$	0	0.01	± 0.007
H3.	Growth efficiency		g/yr/watt	$P/W \sim m^{0}$	0	–0.19	± 0.009
H4.	Lifetime metabolism		watts/g/yr	$W/m/Z \sim m^{0}$	0	0.22	± 0.009

Each basic variable scales with body mass m raised to a power, often expected to be $\alpha = 3/4$. Observed scaling exponents are for all eukaryote data shown in Figs. 1 and 2 (with 95% CIs). Exponents that differ significantly from expectations ($\alpha \neq 3/4$) are boxed in red, while matches (~3/4) are boxed in green. Basic variables can be transformed and combined to yield 4 hypothesized equivalence relationships with expected mass exponents near 0 (H1 to H4). H1 is supported even though W and N do not themselves match expectations, because both basic variables remain inverse to one another. H3 and H4 are rejected because $\alpha \neq 3/4$ for metabolism (W) across eukaryotes.

themselves are at least 10-fold less abundant than plants (*SI Appendix*, Fig. S8). Further accounting for their very high densities, the plant data in Fig. 1*B* are for monoculture stands of trees rather than natural, more diverse assemblages (*SI Appendix*). Birds exhibit a very weak abundance-mass relationship with high residual variation, which is not fully understood (12). Much of the remaining residual variation reflects species population fluctuations rather than systematic variation between species, suggesting that residual variation across species is only marginally greater than that within species (*SI Appendix*, Fig. S5).

Both the scaling and residual variation in the abundance-mass relationship can also be linked to several other well-known abundance patterns. In Box 1, we transform the abundance data in Fig. 1*B* ($N \sim m^{k}$) by applying simple functions to N and m, which allows predictions for other types of relationships and distributions widely studied in ecology. Overall, the abundance data reveal broad consistency with other patterns, but also highlight several critical mismatches calling for further attention (*SI Appendix*, section 8 and Fig. S8).

Growth (g/yr). To ensure that data are comparable across very different groups, measures of growth were integrated over the entire life cycle and are equivalent to the maximum population growth rate (intrinsic growth rate, or r_{max}) multiplied by individual adult body mass (2, 15, 16, 33). Alternative measures of growth that apply only to particular groups or life stages tend to converge on similar measures, so that our confidence in the growth relationship across eukaryotes and within most major groups is relatively high (*SI Appendix*). Consistent with prior work (2, 15–17), maximum growth exhibits ~3/4 scaling within major groups, similar to within-group metabolic scaling. However, in contrast to metabolism, robust ~3/4 growth scaling is also preserved across groups (15–17) (Fig. 1*C*).

Mortality (1/yr). In general, mortality rate is the inverse of life span (years), but life span is commonly defined in different ways. Maximum life span is often measured in captivity under ideal conditions and represents an intrinsic physiological potential, often quite distinct from the extrinsic ecological reality. In contrast, average field life span is measured over a population in the wild and represents a more realistic but more variable measure. Estimating mortality in the wild is more difficult for species such as trees and fish that produce large numbers of offspring, most of which die in early ontogeny. Our data include both measures of mortality and, following previous studies, are normalized to field mortality rates when possible (20). As might be expected from the challenges in measuring mortality, there is significant dispersion in the relationship. However, consistent with prior work (1, 2, 19, 20), we find that mortality scales near −1/4, both across and within many major groups. Data are limited among smaller-sized species, so that we are less confident of the scaling within some groups, such as protists and invertebrates (Fig. 1*D*).

Testing Equivalence Hypotheses

By variously combining any 2 of these 4 scaling relationships through multiplication or division, we can obtain combined variables that have previously been proposed to be invariant with body mass, termed "equivalence relationships" (Table 1). By "equivalence," we do not mean to imply that everything is equal and residual variation is small, but simply that there is no significant trend with body mass. These equivalence relationships form core assumptions and/or predictions of the metabolic theory of ecology (2), but so far have only been validated within major groups. We treat these as hypotheses that can be formally tested across eukaryotes, where the null expectation is a mass exponent of 0 ($k = 0$). We have combined variables using taxonomic information down to the species level when available, to preserve as much of the residual variation as possible (*Methods* and *SI Appendix*). This allows us to

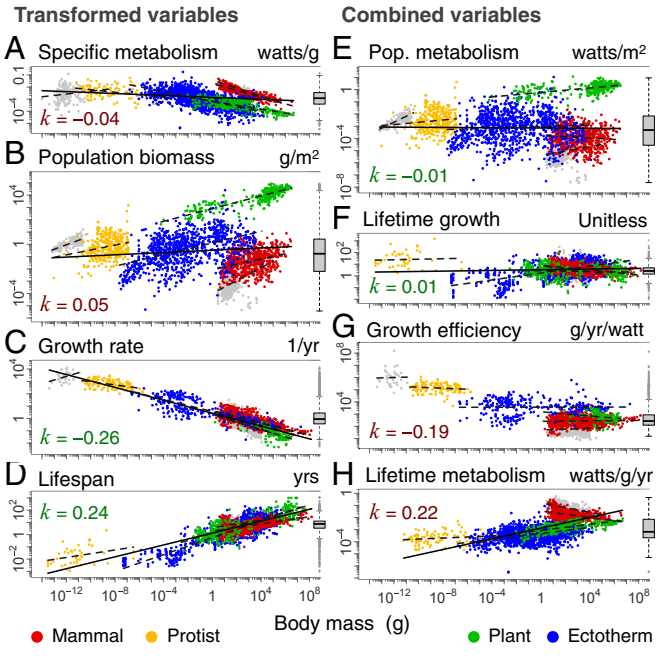
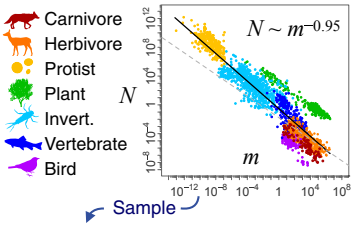


Fig. 2. Scaling of transformed and combined variables with body mass. Data are the same as in Fig. 1 but transformed (*A–D*) and combined (*E–H*) as per Table 1, using taxonomic information down to the species level, where available. The dashed lines show within-group relationships, while the solid line shows the cross-group relationship. Colors of the exponent k correspond to colors of boxes in Table 1. The x- and y-axes have equal order of magnitude spacing. Boxplots are shown for each variable.

Box 1. Linking abundance patterns.

Abundance can be viewed in many ways in ecology. The data from Fig. 1 B can be transformed by applying simple functions to N and/or m to make predictions for four other well-known ecological abundance patterns. These predictions are compared to the known patterns (**A** to **D**) to highlight consistency and identify mismatches.



Abundance-mass scaling

Different major groups are known to exhibit other known abundance relations and distributions. These groups are sub-sampled from the original data in Fig. 1 B.

Function	Prediction	Known pattern
Inverse of N	$A \sim m^{0.73}$	**A. Home range scaling** Mammal home range area, A is known to scale with mass, m, but as $A \sim m^1$ (1, 43, 44). → Suggests encounter rates and grouping scale with size.
Sum of N in bins of m	$f(m) \sim m^{-0.88}$	**B. Size spectra scaling** Size-frequency distributions in aquatic communities are power law, but $k \approx -1$ (1, 46). → Suggests diversity scales negatively with size class.
Mean & var. of Nm in bins of m	$\sigma^2_{Nm} \sim \mu_{Nm}^{1.92}$	**C. Mean-variance scaling** Population variation in space and time scales with the mean from $k = 1.5$ to 2 (47). → Consistent with variations in population biomass (Nm).
Number of species in bins of N	$f(N) \sim \mathcal{N}(\ln N; \mu, \sigma)$	**D. Species abundance** Community-level species abundance distributions are often lognormal (48). → Consistent with mammal species abundances globally.

Abundance patterns in **A** and **B** relate to the scaling of the $N \sim m$ relation, whereas **C** and **D** relate to the structure of residual variation in $N \sim m$. While the functional forms of all four known patterns are recovered, there are mismatches of predicted and known scaling in A and B, which suggest other factors (see SI Appendix and Fig. S8).

characterize the scaling and coefficients both within and across major groups for each of 4 equivalence hypotheses, H1 to H4 (Table 1 and Fig. 2 *E*–*H*).

H1: Population Metabolism. Population metabolism (W/m^2) estimates the total amount of basal energy used by a population per unit area. This hypothesis, also known as the "energetic equivalence rule," suggests an energetic basis of abundance, whereby energy partitioning among species in space exhibits no trend with body mass (2, 10, 12, 13). Although prior work has shown that within major groups, the product of the reciprocal ±3/4 scaling of basal metabolism and abundance gives an equivalence in population metabolism (10–13), this has not been previously tested across all eukaryotes. We find that the exponents and the shifts in elevation between groups of each variable are reciprocal. Specifically, the residual variation in each of the metabolism-mass and abundance-mass relationships (Fig. 1 *A* and *B*) is partly compensatory, so that when a species (or group) is above the line in 1 variable, it tends to be below the line in the other variable and vice versa. As a result, the product of these 2 variables shows an equivalence in energy use at the population level that appears to hold across 20 orders of magnitude in body mass (combining Fig. 2 *A* and *B* gives *E*).

H2: Lifetime Growth. Lifetime growth (dimensionless) estimates the maximum number of offspring produced over the average lifetime of an individual. This hypothesis, also known as "lifetime reproductive effort" (33) or the "equal fitness paradigm" (17), suggests that populations are broadly near a steady state in abundance, such that reproductive rates multiplied over an average life span in the wild should be nearly constant across species (2, 17, 33). Combining reproduction and survival in this way has been proposed as a key component of fitness (17, 33). Data are limited for field estimates of reproductive rates, and our use of maximum reproductive growth will tend to overestimate actual lifetime growth in the wild (*SI Appendix*). Moreover, data remain limited among smaller-sized classes for fully evaluating this hypothesis among protists and invertebrates. Despite these limitations, and consistent with prior work (2, 17, 33), we find broad support for this equivalence relationship across eukaryotes (combining Fig. 2 *C* and *D* gives *F*).

H3: Growth Efficiency. Growth efficiency (g/yr/W) estimates the maximum amount of mass produced per unit energy of basal metabolism. This hypothesis, also known as the "cost of growth," suggests that an approximately constant fraction of metabolism is allocated to growth (27, 28, 32), which forms a basic assumption in many growth models, going back to that of Bertalanffy (18, 25, 26, 34). Our use of basal metabolism for estimating growth efficiency may underestimate the energy needed to fuel maximum growth, so that this measure represents a possible upper limit of growth efficiency (27, 28). Consistent with prior work (27, 28), we find that within major groups, growth efficiency is largely equivalent but that across groups, this hypothesis is not supported, with clear shifts between groups, such that mammals and birds are several orders of magnitude less efficient than unicells in converting energy into new biomass (combining Fig. 2 *A* and *C* gives *G*).

H4: Lifetime Metabolism. Lifetime metabolism (W/g/yr) estimates the amount of basal energy used per gram of tissue over the maximum lifetime of an individual. This hypothesis, also known as the "rate of living" hypothesis (and characterized as "live fast, die young"), is based on oxygen radicals produced as byproducts of metabolism that are known to accelerate senescence and thus reduce life span (2, 9, 29). Whereas H2 applies to average ecological lifetimes, this hypothesis relates to the maximum physiological lifetime. While prior work has shown mixed support for this hypothesis (9, 29), data remain limited for fully rejecting this hypothesis within groups (*SI Appendix*, Fig. S7), but across groups, our data do not support this hypothesis (combining Fig. 2 *A* and *D* gives *H*).

In summary, our data support 2 previously proposed equivalence relationships connected to the population energetics (H1) and steady-state dynamics (H2) of abundance but are inconsistent with 2 other equivalence hypotheses often used to argue for a metabolic basis for growth and mortality scaling (H3 and H4). This raises the question of the ultimate basis for the scaling of these variables and in particular the origin of $k \sim 3/4$ growth scaling across eukaryotes.

Linking Growth and Metabolism

Many of the theories for body mass scaling are based on structural dimensions of the body thought to limit the flux of raw materials or products of metabolism (3, 9, 24, 25). The flux of energy needed to support all life processes is in turn assumed to dominate the scaling of other characteristics (1, 2, 7, 11, 15, 18, 20, 26). While our results are indeed consistent with a metabolic basis for abundance both across and within groups (H1), it is only within groups that metabolism can account for the allometric scaling of other variables, such as growth and mortality (H3 and H4).

Basal metabolism exhibits 2 scaling regimes: an allometric regime within groups ($k < 1$, and typically $k \sim 3/4$) and a near-isometric regime across groups ($k \sim 1$). These 2 scaling regimes are also observed in mammals in different activity states. Basal metabolism across mammals scales near 3/4, as has long been known (1, 2, 7, 21), while maximum (35) and minimum (torpor) (36) metabolic rates scale as $k \sim 1$ (Fig. 3*B*). Mass-specific metabolic rates across eukaryotes and across activity levels in mammals are thus limited over the same 3 orders of magnitude. This isometric regime suggests the existence of strict metabolic boundaries above which eukaryote cells may be damaged by metabolites or unable to pack mitochondria and below which tissues cease to maintain function or become unviable (4, 5, 8, 25). Within this broad 3 orders of magnitude scope, basal metabolism exhibits a tendency to scale near 3/4 within groups but is systematically staggered in elevation across groups so as to remain within near-isometric metabolic boundaries (Fig. 3*C*). These 2 distinct scaling regimes are suggestive of multiple underlying processes.

Growth, on the other hand, appears to exhibit a single scaling regime ($k \sim 3/4$) both within and across species, with no systematic shifts in elevation, which is consistent with a single underlying process. Similar growth scaling is also observed in mammals at different life stages. Maximum reproductive growth (mammals in red in Fig. 1*C*) (15, 16) scales very similarly to maximum ontogenetic (14) and prenatal (19) growth (Fig. 3*B*). We also observe similar ~3/4 scaling in the growth of whole communities across large biomass gradients in grasslands, forests, lakes, and oceans (16). These community-level growth relationships represent a higher level of organization that cannot be accounted for by any strictly individual-level constraint. This single scaling regime, describing growth across and within groups and across life stages and levels of organization, eludes any single metabolic explanation and instead may point to a more general underlying process.

Several authors have suggested that basal metabolic scaling is rather underpinned by growth scaling and not the reverse (9, 14, 23, 27, 30–32). The idea that metabolism adjusts to growth factors rather than growth being limited by structural constraints on metabolism has been proposed for a variety of different reasons. In *SI Appendix*, section 9, we summarize some of the different lines of evidence for this proposal. In addition, we list growth phenomena at different levels of organization and stages of development, be they normal, abnormal, or experimentally induced, that are known to have downstream effects on metabolism. Extreme limits on metabolism can always limit growth, but except in some cancers (e.g., the Warburg effect) (37), normal or experimental changes in metabolism rarely cause downstream changes in growth. Growth is often seen to be regulated upstream of metabolism, which adjusts to fuel protein synthesis and cell replication and ensures coregulation in both variables (*SI Appendix*, Fig. S8). Despite the many indications that metabolism adjusts to growth, so far no quantitative theory for ~3/4 growth scaling has been proposed that encompasses the numerous instances in which such scaling is observed.

Outlook. We believe there may be a universal process underlying the ubiquitous tendency for growth to scale near 3/4. This single growth scaling regime suggests something fundamental in the dynamics of how mass changes over time across very different kinds of living systems. The idea that metabolism adjusts to fuel growth within groups and adheres to metabolic boundaries across groups appears to provide a more parsimonious explanation for the link between the growth and metabolism relationships shown in Fig. 3 and is supported by multiple lines of evidence (*SI Appendix*, section 9 and Fig. S8). We propose that basal metabolism in part reflects ontogenetic adjustments and evolutionary adaptations to efficiently fuel growth and turnover within groups, and that groups are staggered to remain within energetic limits. This could account for why basal metabolism and maximum growth within groups have such similar scaling (Fig. 3*A*), but it does not solve the problem of how this scaling arises.

We are not prepared to speculate as to what general process might underpin growth scaling, but in many of the instances in which such scaling is observed, we are essentially seeking an explanation for the relationship between mass m and its derivative, $\frac{dm}{dt} = cm^{\alpha}$, where $\alpha \sim 3/4$ and c is a constant (33). For $\alpha < 1$, this relationship implies a continually diminishing growth rate and, more specifically, a characteristic scale-free form of sub-exponential growth. Integrating this relationship, we see how

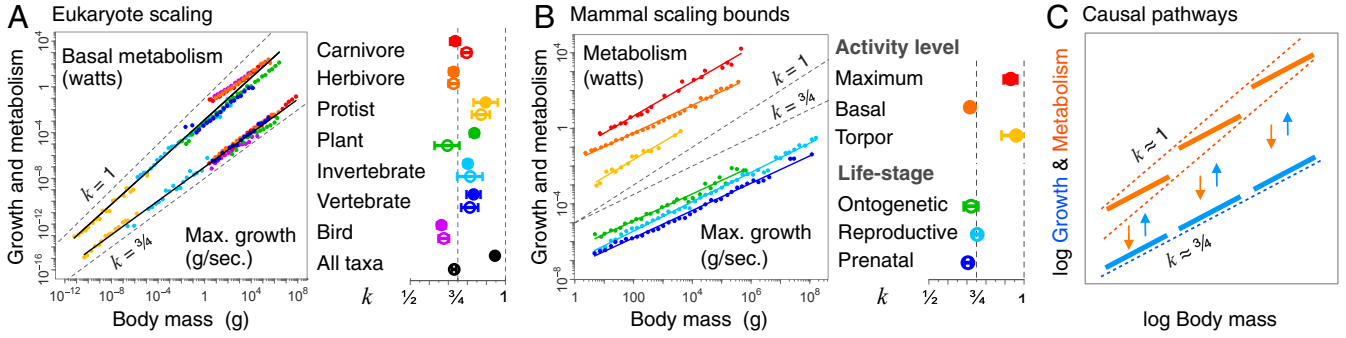


Fig. 3. Growth and metabolism size class scaling. In *A* and *B*, original data from Fig. 1 *A* and *C* are binned into logarithmic body size classes to highlight the principal relationships and give equal representation to different size classes. Exponents and 95% CIs are shown to the right of each plot for the nonbinned data, with filled circles for metabolism (above) and empty circles for growth (below). (*A*) Scaling relationships for basal metabolism (W) and maximum growth (g/s) from Fig. 1 *A* and *C* (with additional taxonomic groups) reveal scaling similarities for most groups but a systematic divergence at larger body mass across groups. (*B*) Mammal scaling relationships for metabolism across activity levels and maximum growth across life stages shows the boundaries in which these characteristics vary. (*C*) The prevailing view is that metabolism determines growth scaling (orange arrows of causality) on the basis that within a given major group, both variables scale as $k \sim 3/4$ (parallel thick lines). The metabolic view, however, cannot explain why growth follows the same universal $k \sim 3/4$ scaling both within and across groups, given that metabolism often shows shifts in elevation between groups. A more parsimonious view is that across groups, metabolism adheres to isometric bounds ($k \sim 1$), but that within major groups, metabolism adjusts to growth scaling of $k \sim 3/4$ (blue arrows of causality).

mass changes over time and approaches $m(t) \sim t^{\frac{1}{1-\alpha}}$. Thus, this solution predicts how the timing of life history events will scale with body mass as $t \sim m^{1-\alpha}$, consistent with the timing of gestation, maturity, and life span (1, 2, 20, 38) (Fig. 2*D*). Understanding the origin of this simple growth relationship may shed light on a number of other linked characteristics and represents a critical area for further research.

Conclusion

The data that we report here provide a comprehensive view of the most general boundaries within which life exists. Although the data are of varying quality, gathered over many decades using different methods and for different objectives, the patterns exhibit remarkable regularity and surprising connections between core species characteristics.

Population abundance is known to be highly variable, but when viewed at the largest scales reveals a reciprocal pattern with metabolism, mirroring both the slopes and shifts in elevation within and across major groups. This supports an equivalence in population-level energy use, consistent with a fundamental energetic basis to abundance that spans all eukaryotes (H1). The maintenance of steady state in abundance is necessarily mediated by the dynamics of birth and death, which also results in an equivalence in lifetime growth (H2). These relationships are intriguing given that so many presumably adaptive traits scale with body size, and yet across eukaryotes, their combined influence has a relatively neutral net effect on population energy use (H1) and individual lifetime growth potential (H2). These 2 broadly supported equivalence relationships, in connection with other known abundance patterns (Box 1), suggest the possibility for a more synthetic understanding of the different aspects of abundance in ecology.

The widely held view that a metabolic basis underpins the allometric scaling of growth and mortality is not supported across eukaryotes (H3 and H4). Most importantly, the consistent ~3/4 scaling of growth found across groups cannot be explained by a single set of metabolic constraints, given that metabolic scaling exhibits different scaling regimes within and across groups. In contrast to metabolic scaling, the striking similarities in growth scaling within and across groups, and also across life-stages and levels of organization, are indicative of a single generating process, begging further understanding. We propose considering the dynamics of growth as presenting a general basis for biological scaling.

Methods

Additional details for all data sources, methods, and limitations, along with regression statistics for more than 200 major and minor group relationships, are provided in *SI Appendix*. Further analysis is possible using the raw data and source code available at https://zenodo.org/record/3145281.

Metabolism measurements were normalized to 20 °C for all taxa except mammals and birds, using both published values of Q_{10} and the Arrhenius factor with standard activation energies, revealing only slight differences among methods that do not alter our conclusions (*SI Appendix*, Fig. S4). All abundance data were gathered over a relatively large area and originally reported in aerial units (e.g., m^2), including aquatic species, normalized over multiple depths in the water column. The mammal growth shown in Fig. 3*B* was calculated for each life stage as follows: (*i*) maximum reproductive growth is from population time series data or, more commonly, calculated from life history measurements, integrated over the entire life cycle; (*ii*) maximum ontogenetic growth is obtained from near the inflection point of somatic growth curves of different mammal species (14); and (*iii*) prenatal growth is calculated as birth mass divided by gestation period (19) (*SI Appendix*).

We used ordinary least squares (OLS; type I regression) to calculate regression statistics, which is the standard approach in bivariate regression when the dependent variable is measured with greater error than the independent variable (1) (*SI Appendix*). Exponents from reduced major axis (type II regression) are similar to OLS for all cross-taxa regressions, although differences are apparent among more resolved groups, and can be obtained from *SI Appendix*, Tables S1–S4. The binning of data shown in Fig. 3 was achieved by taking the geometric mean value in each logarithmic size class, which allows equal representation for different size classes when data are not evenly distributed across the size range (7).

We combined variables to test equivalence hypotheses (Table 1) using several taxonomic levels to ensure that residual variability in combined variables is largely preserved. When direct species matches could not be made in both datasets, we combined measurements from the more-dispersed variable with order-level regression predictions from the less-dispersed variable, which preserves the majority of residual variation while ensuring that regression equations are comparatively reliable. In the relatively few cases where limited taxonomy precluded order-level regressions, we then combined estimates with regression predictions for major groups, and we linked all variables using the same 3 taxonomic levels for all hypotheses in Table 1. More specific treatments for particular groups and paired variables are described in *SI Appendix*.

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1. R. H. Peters, *The Ecological Implications of Body Size* (Cambridge Univ Press, ed. 1, 1983).
2. J. H. Brown, J. F. Gillooly, A. P. Allen, V. M. Savage, G. B. West, Toward a metabolic theory of ecology. *Ecology* **85**, 1771–1789 (2004).
3. G. B. West, J. H. Brown, B. J. Enquist, A general model for the origin of allometric scaling laws in biology. *Science* **276**, 122–126 (1997).
4. A. M. Makarieva, V. G. Gorshkov, B. L. Li, Energetics of the smallest: Do bacteria breathe at the same rate as whales? *Proc. Biol. Sci.* **272**, 2219–2224 (2005).
5. A. M. Makarieva *et al*., Mean mass-specific metabolic rates are strikingly similar across life's major domains: Evidence for life's metabolic optimum. *Proc. Natl. Acad. Sci. U.S.A.* **105**, 16994–16999 (2008).
6. C. R. White, N. F. Phillips, R. S. Seymour, The scaling and temperature dependence of vertebrate metabolism. *Biol. Lett.* **2**, 125–127 (2006).
7. V. M. Savage *et al*., The predominance of quarter-power scaling in biology. *Funct. Ecol.* **18**, 257–282 (2004).
8. D. S. Glazier, Beyond the "3/4-power law": Variation in the intra- and interspecific scaling of metabolic rate in animals. *Biol. Rev. Camb. Philos. Soc.* **80**, 611–662 (2005).
9. D. S. Glazier, Is metabolic rate a universal "pacemaker" for biological processes? *Biol. Rev. Camb. Philos. Soc.* **90**, 377–407 (2015).
10. J. Damuth, Interspecific allometry of population density in mammals and other animals: The independence of body mass and population energy-use. *Biol. J. Linn. Soc. Lond.* **31**, 193–246 (1987).
11. A. Belgrano, A. P. Allen, B. J. Enquist, J. F. Gillooly, Allometric scaling of maximum population density: A common rule for marine phytoplankton and terrestrial plants. *Ecol. Lett.* **5**, 611–613 (2002).
12. S. Nee, A. F. Read, J. J. Greenwood, P. H. Harvey, The relationship between abundance and body size in British birds. *Nature* **351**, 312–313 (1991).
13. R. F. Hechinger, K. D. Lafferty, A. P. Dobson, J. H. Brown, A. M. Kuris, A common scaling rule for abundance, energetics, and production of parasitic and free-living species. *Science* **333**, 445–448 (2011).
14. T. J. Case, On the evolution and adaptive significance of postnatal growth rates in the terrestrial vertebrates. *Q. Rev. Biol.* **53**, 243–282 (1978).
15. S. K. M. Ernest *et al*., Thermodynamic and metabolic effects on the scaling of production and population energy use. *Ecol. Lett.* **6**, 990–995 (2003).
16. I. A. Hatton *et al*., The predator-prey power law: Biomass scaling across terrestrial and aquatic biomes. *Science* **349**, aac6284 (2015).
17. J. H. Brown, C. A. S. Hall, R. M. Sibly, Equal fitness paradigm explained by a trade-off between generation time and energy production rate. *Nat. Ecol. Evol.* **2**, 262–268 (2018).
18. G. B. West, J. H. Brown, B. J. Enquist, A general model for ontogenetic growth. *Nature* **413**, 628–631 (2001).
19. R. Tacutu *et al*., Human ageing genomic resources: Integrated databases and tools for the biology and genetics of ageing. *Nucleic Acids Res.* **41**, D1027–D1033 (2013).
20. M. W. McCoy, J. F. Gillooly, Predicting natural mortality rates of plants and animals. *Ecol. Lett.* **11**, 710–716 (2008).
21. M. Kleiber, Body size and metabolism. *Hilgardia* **6**, 315–353 (1932).
22. D. S. Glazier *et al*., Ecological effects on metabolic scaling: Amphipod responses to fish predators in freshwater springs. *Ecol. Monogr.* **81**, 599–618 (2011).
23. H. U. Riisgard, No foundation of a "3/4 power scaling law"for respiration in biology. *Ecol. Lett.* **1**, 71–73 (1998).

24. J. R. Banavar *et al*., A general basis for quarter-power scaling in animals. *Proc. Natl. Acad. Sci. U.S.A.* **107**, 15816–15820 (2010).
25. S. A. L. M. Kooijman, *Dynamic Energy and Mass Budgets in Biological Systems* (Cambridge Univ Press, 2000).
26. C. Hou *et al*., Energy uptake and allocation during ontogeny. *Science* **322**, 736–739 (2008).
27. W. Wieser, Cost of growth in cells and organisms: General rules and comparative aspects. *Biol. Rev. Camb. Philos. Soc.* **69**, 1–33 (1994).
28. A. Clarke, Energy flow in growth and production. *Trends Ecol. Evol.* **34**, 502–509 (2019).
29. J. R. Speakman, Body size, energy metabolism and lifespan. *J. Exp. Biol.* **208**, 1717–1730 (2005).
30. M. Czarnołeski *et al*., Scaling of metabolism in Helix aspersa snails: Changes through ontogeny and response to selection for increased size. *J. Exp. Biol.* **211**, 391–400 (2008).
31. R. E. Ricklefs, Is rate of ontogenetic growth constrained by resource supply or tissue growth potential? A comment on West et al.'s model. *Funct. Ecol.* **17**, 384–393 (2003).
32. G. D. Parry, The influence of the cost of growth on ectotherm metabolism. *J. Theor. Biol.* **101**, 453–477 (1983).
33. E. L. Charnov, R. Warne, M. Moses, Lifetime reproductive effort. *Am. Nat.* **170**, E129–E142 (2007).
34. L. Von Bertalanffy, Quantitative laws in metabolism and growth. *Q. Rev. Biol.* **32**, 217–231 (1957).
35. E. R. Weibel, L. D. Bacigalupe, B. Schmitt, H. Hoppeler, Allometric scaling of maximal metabolic rate in mammals: Muscle aerobic capacity as determinant factor. *Respir. Physiol. Neurobiol.* **140**, 115–132 (2004).
36. F. Geiser, Metabolic rate and body temperature reduction during hibernation and daily torpor. *Annu. Rev. Physiol.* **66**, 239–274 (2004).
37. M. G. Vander Heiden, L. C. Cantley, C. B. Thompson, Understanding the Warburg effect: The metabolic requirements of cell proliferation. *Science* **324**, 1029–1033 (2009).
38. J.-M. Gaillard *et al*., An analysis of demographic tactics in birds and mammals. *Oikos* **56**, 59–76 (1989).

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Supplementary Information for

Linking scaling laws across eukaryotes

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This PDF file includes:

Figs. S1 to S9
Tables S1 to S8
References for SI reference citations (39-132)

Other supplementary materials for this manuscript include the following:

Data files: S1 Data.xls
Analysis source code: "Link-scaling.Rproj"
(https://zenodo.org/record/3145281)

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Sections: Page

In this paper, we compile data across all major eukaryote taxa for four basic ecological variables: metabolism, abundance, growth and mortality (Table 1 A). For each variable, we have collated data for >2000 species (section 1). Our analysis summarizes the body mass scaling of each variable across eukaryotes, and then pairs variables through multiplication or division to obtain compound variables that have previously been hypothesized to be invariant with body mass (Table 1 C; Figs. 2 E-H). We test these hypotheses by evaluating whether combined variables exhibit any trend with mass to offer insight into how variables are mechanistically linked. The patterns we show are robust to different taxonomic treatments for aggregating species measurements, regression methods, combining variables and temperature corrections (described in sections 2 to 4).

More specific considerations for particular variables are included in sections 5 to 8, and further discussion of the physiological links between growth and metabolism in section 9. The data file includes all 22,761 original measurements over all four variables, mammal data in Fig. 3 B, and all 2791 published sources from which data were obtained (section 10). Our analysis can be reproduced from the source code including temperature corrections, species aggregation, variable combination and regression methods (section 11). Tables S1 to S8 list regression statistics for 216 body mass scaling relations for basic and combined variables down to the level of taxonomic order (section 12).

1. Data sources

Metabolism

Basal metabolism (watts) is the basic processing of energy and materials in an individual. It is measured as the amount of O_2 consumed or CO_2 produced in a variety of units that were converted to watts (J/s) (Fig. 1A). Data includes 8098 measurements across 230 taxonomic orders, obtained from a number of meta-analyses (e.g. (5, 6, 19, 39–42)). We used a conversion factor of 1 watt = 20 J per ml O_2 consumed to convert O_2 or CO_2 to energy consumption (1, 5). Measurements were normalized to 20 °C for all taxa except endotherms (mammals and birds), and were excluded if temperature information was not included with original data. We did not temperature correct

endotherm (mammal and bird) metabolic rate to 20 °C given that the normal range of temperature is considerably higher, and we seek realistic levels of energy use (5, 6) (temperature-corrections for all species including endotherms lowers the cross-taxa metabolic exponent from $k = 0.95$ to 0.92). We used both published values of Q_{10} and the Arrhenius factor with standard activation energies to correct for temperature, finding only slight differences among methods that do not alter our conclusions. To compare mammal metabolism across activity levels (maximum, basal and minimum torpor; Fig. 3 B; (35, 36)), we did not correct for temperature (36), but show such corrections in Fig. S4. Further information on temperature corrections is in Section 4, below.

Abundance

Abundance is the population density (individuals/m^2) of a species in its natural habitat over a relatively large spatial extent, such as a lake or protected area (Fig. 1 B). We only included aquatic data that were originally reported in aerial spatial units (rather than volumetric units), obtained over multiple depths in the water column. Data includes 5985 measurements (101 orders), and were obtained primarily from a number of meta-analyses (e.g. (10, 11, 16, 43–48)). The scaling of population density with mass was first observed in mammals, and found to be near −¾ though with a considerable 3 to 4 orders of magnitude residual variation about the line. Similar abundance-mass scaling was found in other taxa (11, 12, 43), but studies considering larger size ranges tend to show near inverse scaling ($k \approx -1$; Refs: (10, 44–46). Given the large residual variation, other studies over more limited size ranges have reported various other types of relations (49, 50). Plant density data are primarily mono-culture stands, and more diverse natural systems have densities that are at least an order of magnitude lower. Bird densities are difficult to estimate over the vast areas they can occupy, and most existing data are given as relative abundance rather than absolute abundance in space. Finally, the grey points in Fig. 1 B with the highest densities are for both bacteria and algae from a single study (43), and along with plants and birds should be treated with caution. Removing these groups, we find $k = -1.03$ (n=2056), and ranges from −0.86 to −1.09, depending on which groups are included or excluded. Human densities are shown ranging across the 1000 largest cities and 300 hunter-gatherer communities, but are otherwise excluded from analysis. Information on the residual variation and outliers in the abundance-mass relation is in section 5, below.

Growth

Maximum productivity or growth (g/yr) is the maximum mass produced per unit time by an individual through ontogeny and/or reproduction. This definition is equivalent to the maximum population growth rate, termed the intrinsic growth rate (r_{max}) multiplied by adult body mass (Fig. 1 C). Data includes 3812 estimates (176 orders), and were obtained primarily from several meta-analyses (e.g. (15, 16, 47, 51–58)). The individual productivity was supplemented with calculated productivity from life-history characteristics reported in (19) (AnAge data build 14; October 2017). Mammal growth data shown in Fig. 3 B were obtained from several meta-analyses at different life-stages as follows: prenatal growth (19, 59), ontogenetic growth (14) and reproductive growth (15, 16). Further information on calculating growth rate at different life stages is provided in section 6, below.

Mortality

Mortality rate (1/yr) is the inverse of lifespan, and can be defined to include either intrinsic factors of senescence (measured as maximum lifetime in captivity) or extrinsic factors such as disease,

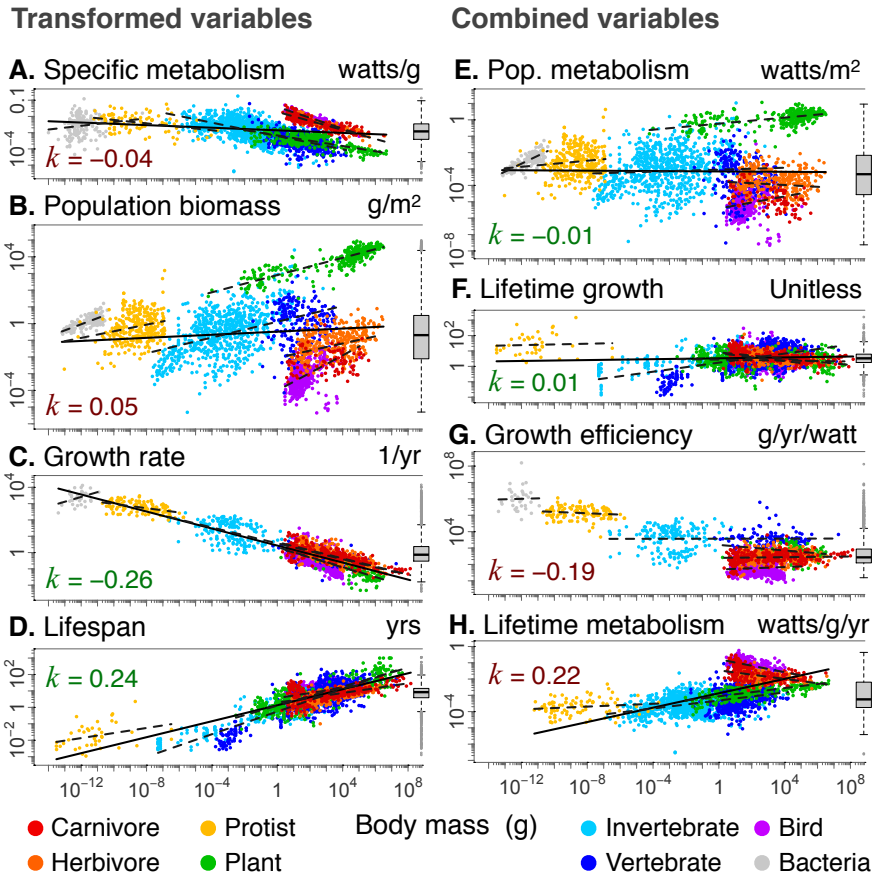

Fig. S1. Scaling at higher taxonomic resolution. Data are the same as Fig. 2, transformed as per Table 1 B (**A** to **D**), and combined as per Table 1 C (**E** to **H**), and showing several additional groups over Fig. 2. The dashed lines show major taxa regressions, while the solid line shows cross-taxa scaling with exponent *k* (exponent colors correspond to boxes in Table 1). X- and Y-axes have equal order of magnitude spacing. Boxplots are shown for each variable.

predation, food availability, and other factors (measured as average lifetime in the wild) (20). Our data include both average lifetime in the wild and maximum lifetime in captivity (Fig. 1 D). Among birds and mammals, it is thought that maximum lifespan is about 2.5 times the average lifespan in the wild (20), though this is not well supported by our data. often increasing disproportionately with adult size ($k > 1$; (60)), but most of which die in early ontogeny (many of the blue dots well above the line in Fig. 1 D are juvenile fish). Mortality data includes 4866 measurements (199 orders) and were obtained primarily from four meta-analyses (19, 20, 61, 62). Data are limited for protists and invertebrates, and so we only used species average values for endotherms to increase representation among smaller size classes. These data limitations preclude a robust description of lifetime metabolism scaling across the size spectrum (Table 1 C iv), further information on which is included in section 7, below.

Mammal metabolic scope

Maximum mammal metabolism come from two meta-analyses (35, 63) (Fig. 3 A). We did not aggregate species values in (35), and so our regression slope for whole-organism metabolism is

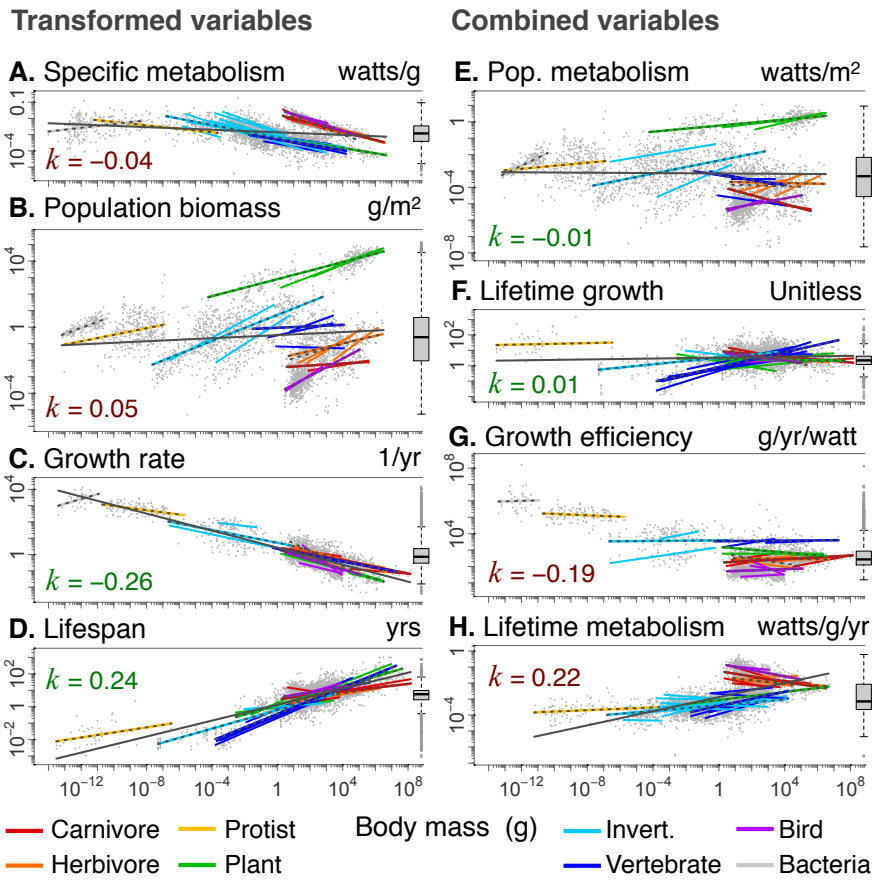

Fig. S2. Scaling of taxonomic orders. Data are the same as Fig. 2 and Fig. S1. Dotted lines show major taxa regressions (as in Fig. S1), while the solid colored lines show order level regressions extending over at least two orders of magnitude with at least 20 data points. Regressions are listed in Tables S1 to S8. See Fig. S1 legend for further details.

steeper ($k = 0.93$) than that reported in (35) ($k = 0.87$). Minimum torpor metabolism was considered from mammals that enter deep torpor (hibernation), derived principally from one meta-analysis (64), and expanded with data in (36), the latter of which did not distinguish extended torpor from daily heterotherms, and so we only include a few larger animals from this list that are known to enter deep hibernation. We excluded mammals with minimum metabolism estimates that were near an order of magnitude higher than other estimates for the same species, in order to estimate the true minimum value that smaller body sizes are capable of achieving.

2. Species aggregation and regression

Measurements of different individuals of the same species were aggregated to obtain a single variable estimate for each species, where multiple estimates for the same species were available. For basal metabolism and maximum growth, we obtained the minimum and maximum values, respectively, and used the corresponding body mass associated with that value. For abundance and mortality, we obtained the mean value and mean body mass for each species. We excluded plants

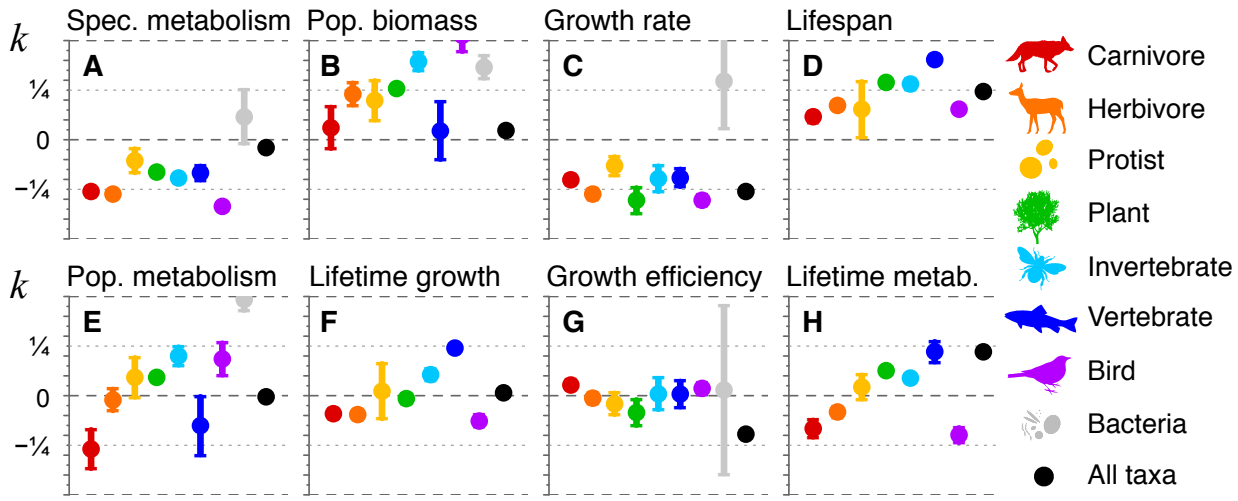

Fig. S3. Scaling exponents and 95% CI. Transformed (**A** to **D**) and combined (**E** to **H**) variable exponents (*k*) derive from the same data as Fig. 2, but mammals are split into herbivores and carnivores, and ectotherms are split into vertebrates and invertebrates, with birds and bacteria also included, as shown in Figs. S1 and S2.

from all aggregation given that they can vary in body size and other variables by many orders of magnitude through their life-cycle. We only aggregated endotherm species for mortality, due to data limitations among smaller size classes. For all variables, we tested whether different aggregation functions (minimum, maximum, mean or geometric mean) altered our broad scale r esults, and find almost no detectable differences among these functions for cross-taxa regressions, and only small differences for most within-group predictions. We also find very similar results without species aggregation, or else aggregation into different logarithmic size classes (binning), which gives more equal representation across the size spectrum (e.g. Fig. 3). Different species aggregation functions can be further examined from the source code (section 11). One of the limitations of aggregating multiple species measurements is that a single species can vary a great deal in abundance and mortality. On the other hand, not aggregating data at the species level will further bias the already unequal representation of data across the size spectrum. An average adult mass for a species can also be problematic since species grow through different sizes in ontogeny. We converted all mass units to fresh or wet mass in grams, with the dry mass to wet mass ratio assumed to be 0.3 (Refs: (1, 5).

We used ordinary least squares (OLS; type I regression) to calculate regression statistics, which is the standard approach in bivariate power law regression (1, 65), but assumes that all error is in the Y-axis variable. This tends to underestimate the slope *k* as error in the X-axis variable increases. Alternative regression approaches such as reduced major axis (RMA; type II regression) partition variation equally among both axes but tend to overestimate slopes as error increases (e.g. if x- and y-variables are uncorrelated and range over similar extents, OLS gives a slope near zero, while RMA gives a slope near ±1). Given the greater potential for measurement error and for natural variability in the Y-axis for all basic variables relative to body mass, OLS is considered a less biased slope estimator than many type II regression methods, though likely underestimates the steepness of all slopes to some degree. Exponents from reduced major axis, are similar to OLS for all cross-taxa regressions of basic variables, and can be obtained by dividing reported OLS derived slopes by the square root of the coefficient of determination ($\sqrt{R^2}$; Tables S1 to S4).

3. Combining variables

By combining variables through multiplication or division, we obtain compound variables that may reveal an equivalence across species, as shown in Table 1 C. By 'equivalence' we do not mean that residual variance is small, but rather that a variable does not change systematically with body mass. A stronger version of equivalence is that the variability within a species is not significantly different from variability among all species, and can be tested for some species where sufficient data are available. Combining variables can be facilitated by transforming basic variables by multiplying or dividing by body mass, or taking their inverse, as shown in Table 1 B. Transformed variables shown in Figs. 2 A-D are also shown in Figs. S1 and S2 A-D at higher taxonomic resolution.

In order to gain insight into whether variability within a species is similar to all others across hypothesized equivalence relations (Table 1 C i to iv), we combined variables using several taxonomic levels to ensure that residual variability is largely conserved. There are approximately 500 to 1000 species matches between datasets, mostly among mammals and birds, requiring the use of regression predictions to obtain estimates for the remainder of species which could not be matched in both datasets. For example, multiplying metabolism and abundance to give population metabolism (Table 1 C i), is first undertaken on the basis of species matches, where available. This is followed by matches among taxonomic orders, preserving the residual variation from the more scattered regression (i.e. population density) and using the less scattered order-level regression prediction (i.e. metabolism) to estimate the composite variable (i.e. population metabolism; Table 1 C i). This ensures that regression predictions are relatively well behaved. In cases where order level regressions could not be constructed due to limited taxonomic information, we then matched major taxonomic groups and used respective regression predictions at that level. The majority of matches were made at the order level, and we matched all variables using the same three-levels of taxonomy for all combined variables in Table 1 C. Combined variables plotted against body mass shown in Figs. 2 E-H are also shown in Figs. S1 and S2 E-H at higher taxonomic resolution. Combining variables in reverse; i.e. using the less scattered estimate and more scattered regression prediction is less ideal but tends to give quite similar results to those reported. The exception is for lifetime metabolism (H4), which is sensitive to the order in which variables are combined (section 7). Alternative ways of combining variables can be further analyzed using the source code (section 11).

For illustration, we have split eukaryotes into four major groups: mammals, protists, plants and ectotherms in Figs. 1 and 2. Bird and bacteria data are shown in grey in Fig. 1, but the abundance data for these groups should be treated with caution (this caution applies to bacteria data in all plots). Analysis among more resolved taxonomic groups were also undertaken, which broadly supports our overall conclusions, albeit with larger confidence intervals (e.g. Fig. S3). Finally, we report order level regression predictions in the accompanying Tables S1 to S4, and further, more specific, analyses are facilitated from the Supplementary source code and data file (sections 10 to 12).

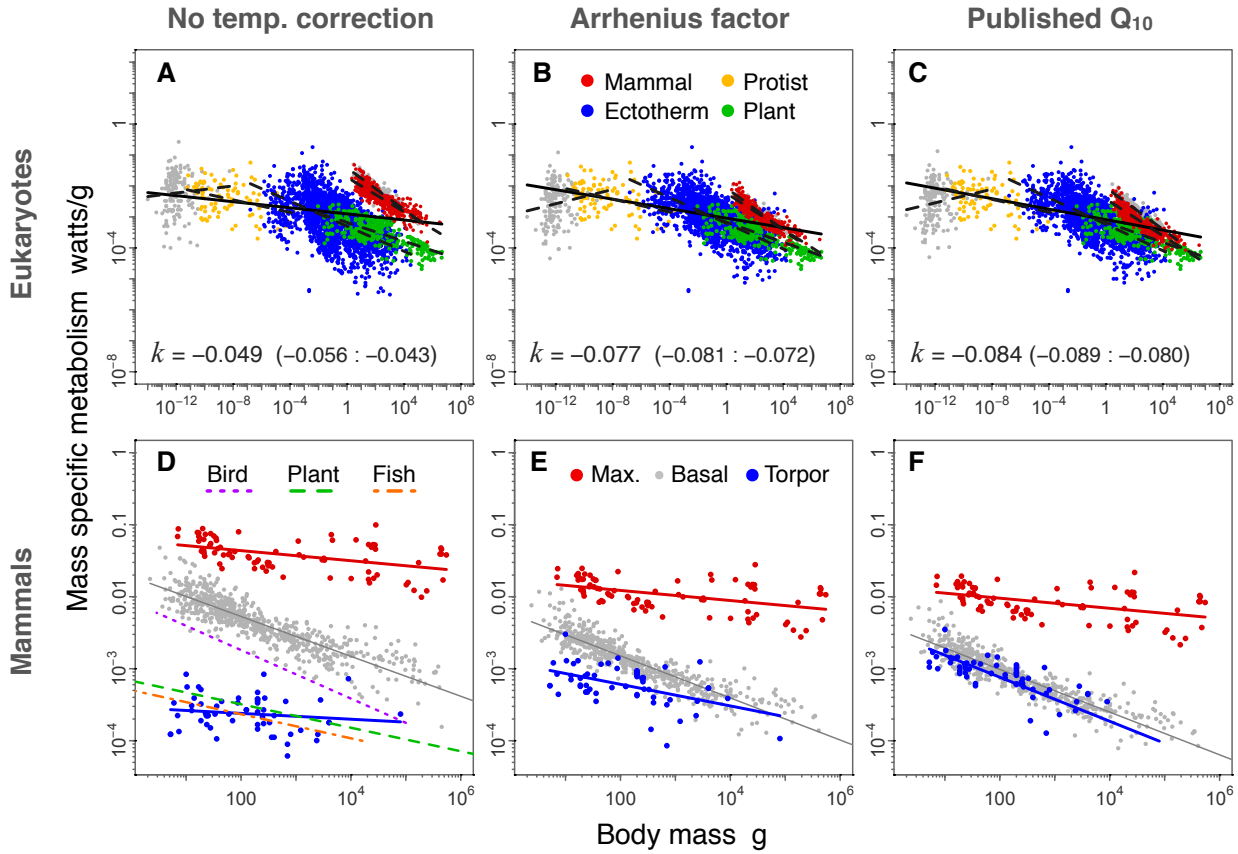

Fig. S4. The effect of different temperature corrections on mass-specific metabolism. The top panels show basal mass-specific metabolism across all taxa (as in Fig. 2 A), while the bottom panels show maximum, basal and minimum mass-specific metabolism for mammals (as in Fig. 3 A). All temperature corrections are to 20°C, and for the Arrhenius correction make use of a generic activation energy of 0.6 eV (in B and E). **A.** Without temperature correction. These differ from Fig. 2 A, in which only endotherms are not temperature corrected. **B.** Temperature correction for all species using an exponential Arrhenius factor. **C.** Temperature correction for all species using published Q_{10} values. **D**. Without temperature correction, as in Fig. 3 A. Temperature corrected regression lines are shown for other taxa. **E.** Temperature correction using an Arrhenius factor. **F**. Temperature correction using a generic Q_{10} of 2.5 for basal and maximum rates, and individually measured Q_{10} for torpor rates, the latter of which reveal the circularity of using Q_{10} across activity states.

4. Metabolic temperature corrections

Basal metabolism in Fig. 1 A was corrected to 20°C for all species except birds and mammals, which regulate their temperature well above this value, and for which temperature corrections would represent unrealistic values of basal metabolism (5, 6). Likewise, we did not temperature correct any of the mammal metabolic data in Fig. 3 B.

Temperature corrections of metabolism (W_T) measured at temperature T (in °C) were corrected to 20°C, or equivalently 293 Kelvin ($W_{20°C}$), using the Boltzmann-Arrhenius factor with a generic activation energy of E=0.6 eV, and the Boltzmann constant of κ=8.62×10^{-5} eV, and estimated as follows (66),

$$W_{20°C} = W_T * e^{-\frac{E}{\kappa}\left(\frac{1}{293} - \frac{1}{273+T}\right)}$$

Temperature corrections to 20°C using published Q_{10} values were estimated as follows,

$$W_{20°C} = W_T * Q_{10}{}^{(20-T)/10}$$

Previously published Q_{10} values are included in the Supplementary Data file. Our results are robust to these different methods and parameters (activation energies or Q_{10} values), though are more sensitive to whether or not temperature corrections are applied to endotherms. Temperature-corrections for endotherm metabolism lowers the cross-taxa exponent from $k = 0.95$ to 0.92, and thus alters hypothesized equivalence relations in Table 1 C (e.g. the population metabolism hypothesis, H1, exponent changes from $k = -0.014$ to $+0.045$.

Despite not temperature correcting metabolism in endotherms, we show in Fig. S4 the effect of correcting for temperature for both basal metabolism in endotherms, and maximum and torpor metabolism in mammals. Although the scaling in maximum and basal rates remain largely unchanged with different corrections, the scaling in torpor metabolism changes dramatically. However, we do not believe these corrections are meaningful across metabolic activity levels (5, 6, 36). In particular, using measured Q_{10} values, which are calibrated to basal metabolism implies a circularity in comparing across these physiological states (Fig. S4 F). Moreover, torpor is unrealistic for mammals at temperatures of basal or maximum activity levels, and in contrast with daily heterotherms, hibernators are known to use metabolic inhibition to achieve lower body temperatures rather than responding solely to ambient temperature (9, 36, 67). Given these reasons, and that we seek the range in actual metabolism in the wild, correcting for temperature in endotherms across activity levels is not justified (5, 6, 36).

5. Abundance-mass residual variation

There are a number of notable outliers ing the abundance-mass scaling relation (Fig. 1 B). The plant density data is for mono-culture stands and should be considered an upper limit that is not representative of natural and diverse communities (11, 47, 55, 56). We attempted to calculate density in more natural mixed forest systems such as Barro Colorado Island, where some 250,000 stems from some 300 species have been documented over 50 hectares of old growth forest, allowing a mean species density to be obtained for all species in a community. These diverse forest data reveal species densities more than an order of magnitude lower than the mono-culture stands, but ranging over three orders of magnitude in density, and so overlapping with the highest mammal densities. We did not include these data, however, given the difficulties of assigning a mean body mass to trees that follow indeterminate growth, varying over many orders of magnitude through their life-span, and are largely composed of non-metabolically active tissue. These limitations apply to the monoculture stands shown in Fig. 1 B, but have previously been reported to give a regular abundance-mass relation (11, 47), and so are included for comparison in our analysis.

Humans in the largest cities (>500 000 residents) have biomass densities in excess of any other animal on Earth (Fig. 1 B; Fig. S5), and it is not meaningful to estimate an average human

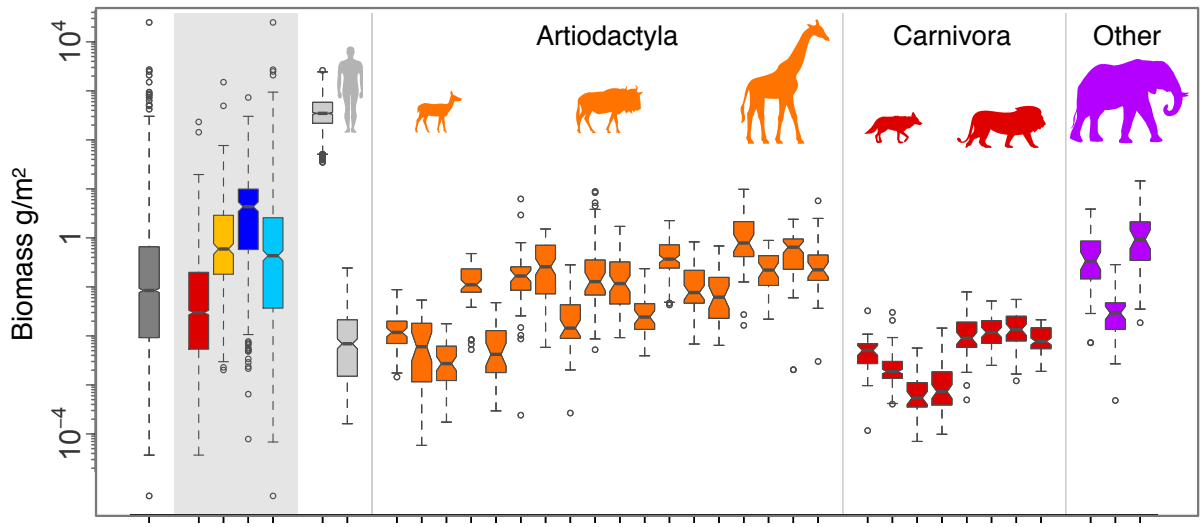

Fig. S5. Boxplots of biomass across major taxa and individual species. Biomass of eukaryotes (excluding plants and birds) compared with major taxa, and 30 individual mammal species, each with > 30 estimates of biomass density in different locations. The boxplot at left includes all data (*n*=4335), while the shaded region next to it shows four major taxa: mammals (red), protists (yellow), vertebrate ectotherms (dark blue) and invertebrates (light blue). This is followed by humans in grey (assumed to be 60 kg) in the 1000 largest cities and among 300 hunter gatherer communities. The remainder of boxplots are separate species in the order Artiodactyla and Carnivora (sorted by increasing size), and three other species (zebra, langur and elephant).

population density given the range from hunter gatherers to urban residents, over nearly seven orders of magnitude (Fig. S5).

Excluding these outliers, there remains significant residual variation in population abundance amounting to three to four orders of magnitude (Fig. 1 B). A large part of this residual variation is the natural variability through time or along an environmental gradient in the populations of any given species, which are known to vary over a similar range of residual variation as all other species. Another part of the residual variation is due to trophic level losses of energy up the food chain. The large residual variability in abundance means it is difficult to make the case for 'strong' equivalence (variabilities within and across species are similar; see section 3, above). In particular, we do not have sufficient data for the same species in different environments and at different times to test whether their population biomass may vary similarly to all other species. For some species of large mammals, for example, we have at least 30 estimates of the same species in different environments, and can compare the within-species variabilities to that of all other species. Removing plants, birds and bacteria due to data limitations, as discussed below, the inter-quartile range in biomass is < 2 orders of magnitude. Fig. S5 shows that species within the mammal orders of Artiodactyla and Carnivora have comparable variabilities to most other species and span the interquartile range of all eukaryote species. Population biomass across all taxa looks to be log-normally distributed, as does mass-specific metabolism, population metabolism and lifetime growth (Fig. S6).

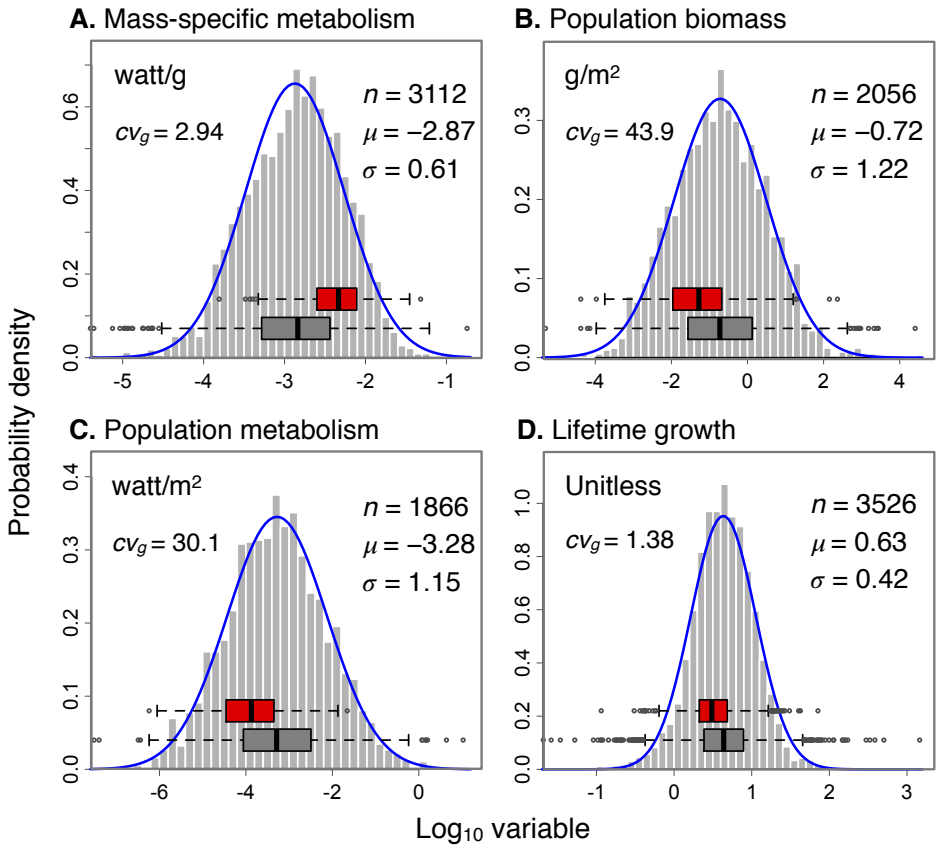

Fig. S6. Log-normal probability distributions of four equivalence relations. Histograms of $\log_{10}$-transformed data and a normal distribution fit to the histograms shown by the blue line, whose mean μ and standard deviation σ of the $\log_{10}$ transformed data (n) is shown. The boxplot in grey is for all the same data, and the boxplot in red shows just mammals. The 'geometric coefficient of variation' was calculated as $cv_g = \sqrt{e^{\sigma^2} - 1}$, (where σ is, in this case, the standard deviation of the *natural* log (base e) transformed data). Population metabolism in C has a lower cv_g (and lower arithmetic coefficient of variation) than population biomass in B, suggesting less relative dispersion and greater regularity. Plants, birds and bacteria are excluded in plots A to C for reasons outlined in section 5.

6. Estimating growth and efficiency

There are many ways of estimating maximum individual growth, which refer to maximum gains in mass per unit time associated with an individual. Fig. 1 C includes data from multiple sources, all of which sought to estimate maximum growth, though often using quite different measurement techniques. For data to be comparable across very different species, estimates should encompass all sources contributing to a change in mass per time, including both post-natal and reproductive growth integrated over the entire life-history of an individual. Many estimates of productivity in the literature are specific to a given life-stage in particular taxonomic groups, be it egg, offspring or weaning mass production (15, 58, 60, 68, 69) and are thus not generally appropriate for comparative purposes across very different major groups. Nonetheless, these different measures

tend to converge on similar values, and we have included many such estimates in our growth dataset.

A more integrated estimate of maximum reproductive growth can be estimated in mammals and birds from life-history characteristics including maximum litter or clutch size (s), minimum age of maturity (a), maximum reproductive lifespan (z), and a characteristic interbirth interval (i), assuming there is no extrinsic mortality of juveniles and an even sex ratio (s/2). The number of females born at the next time step ($N_{age=0,\,t+1}$) can be estimated as the litter of females per birth interval i, multiplied by the sum of all reproductive females, as follows,

$$N_{age=0,t+1} = \frac{s}{2i}\sum_{a}^{z} N_{age,t}$$

$$N_{age=1,t+1} = N_{age=0,t}$$

$$\vdots$$

$$N_{age=z,t+1} = N_{age=z-1,t}$$

This age-structured model supposes that all females born into the age=0 class derive from the maximum reproductive output of all reproductively active females in a population, and that all such juveniles survive to reproduce at age=a, and do so until they reach age=z. After iterating this model, an invariant growing population structure will converge on a particular exponential distribution (70), regardless of starting values. The total number of females in one year (or age class) divided by the total number in the previous will give the finite growth rate (λ), which can be converted to instantaneous growth rate, $r_{max} = \ln(\lambda)$. This allows commonly reported life-history observations to be used to estimate maximum reproductive growth rate, r_{max}. Multiplying r_{max} and adult body mass gives an estimate of maximum growth that is comparable across species.

Maximum growth estimates in Fig. 3 B were estimated as follows: i) Maximum reproductive growth was obtained from the maximum increase in population time-series data, or more commonly, calculated from life-history measurements, and is described further below; ii) Maximum ontogenetic growth was obtained from the linear phase (inflection point) of ontogenetic growth curves up to maturity between 5% and 30 to 50% of adult body weight (data from (14)); and, iii) Prenatal growth was calculated as average birth mass divided by gestation period, given that mammal ova are typically similar in size (data from (19)). Although this does not give a true maximum for prenatal growth, mammal foetal growth does not tend to follow a sigmoid growth curve, and more typically follows sub-exponential growth, with an exponent that approaches near ¾ (59, 71). Given that such power law growth does not have an inflection point, the maximum growth rate occurs very early in development, where few data are available. Relatively few foetal timeseries data are available to calculate maximum pre-natal growth in the same way as ontogenetic growth, and so the use of birth divided by gestation provides a broadly comparable measure of growth across many species (59). Prenatal growth is lower than reproductive growth because mammals tend to have about 2.5 offspring per litter (20).

Growth efficiency (H3) was calculated as maximum growth divided by basal metabolism, and therefore represents an upper limit, since in many taxa resting metabolism is not sufficient to fuel

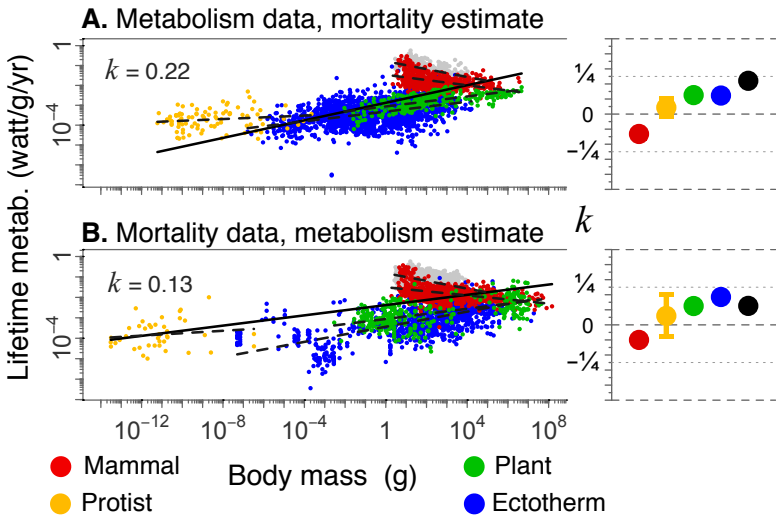

Fig. S7. Lifetime metabolism showing two ways of combining variables. Because of limited lifespan data among smaller size classes, the lifetime metabolism hypothesis (H4) is sensitive to the way in which variables are combined. Where direct species matches cannot be made between metabolism and lifespan, lifetime metabolism can be calculated using metabolism estimates and lifespan regression predictions (**A**), or vice versa: mortality estimates and metabolism regression predictions (**B**). These methods give similar results within taxa, but cross-taxa scaling is quite different (compare k = 0.22 to k = 0.13). Nonethelss, binning the data and taking averages, or randomly sampling across size classes or taxonomic groups always yields significant positive scaling in H4.

maximum growth. Our estimate of growth efficiency should be considerably higher than most field estimates that measure and relate average growth and field respiration (27, 28, 57).

7. Lifetime metabolism limitations

All combined variables are somewhat sensitive to the way in which basic variables are combined, but this is particularly true for the lifetime metabolism hypothesis (H4). This hypothesis multiplies mass-specific basal metabolism by the maximum physiological lifespan to give the amount of energy fluxed in a given lifetime (29, 72–74). There are only 792 species matches and we have few mortality data among smaller sized species (protists and invertebrates), so that the cross-taxa regression depends on how we combine metabolism and mortality variables to calculate lifetime metabolism for those species that are not present in both datasets (Fig. S7). In particular, if we combine metabolism measurements with regression predictions for mortality at the order or major taxa level, we obtain a cross-taxa exponent of $k = 0.22$. On the other hand, if we use mortality estimates with regression predictions for metabolism, we obtain $k = 0.13$. Nonetheless, using only direct species matches (n=792), we find that lifetime metabolism varies over 5 orders of magnitude with overall significant positive scaling. Moreover, sub-sampling the data equally across size classes, or logarithmic binning of the data to achieve equal data representation across the eukaryote size range also results in significant positive scaling, suggesting poor support for this hypothesis. Ectotherms tend to have positive lifetime metabolism scaling while endotherms tend to have negative scaling with mass, as shown in Fig. S7.

8. Linking abundance patterns

Abundance-mass scaling represents one of several ways of viewing population abundance (Fig. S8), an understanding of which is central to much of ecology. In Box 1, we sub-sample the abundance-mass data shown in Fig. 1 B, and using simple functions, we calculate different metrics of abundance to show that the scaling and residual variation of the abundance-mass data is broadly consistent with four other well-known abundance patterns. We sub-sample only those taxonomic groups in which the relevant abundance pattern is typically observed, and for which we can make direct comparisons with independent data, as shown in Fig. S8. The four patterns we consider are as follows: home-range area scaling with mass of different species across systems (often $k \approx 1$); size spectra, which is the size-frequency distribution of total abundance of all species across different logarithmic size classes within a particular community (often $k \approx -1$); mean-variance scaling in population abundance between sites or through time (Taylor's law, often $1.5 < k < 2$); and the species abundance distribution within a particular community (often log-normal). The first two patterns deal with the scaling exponent of the abundance-mass relation, while the latter two deal with its residual variation.

A) *Home range scaling*

Home range area (A) was initially thought to scale with body mass (m) near $k = ⅔$ or ¾ (Ref: (75)), but further work showed near linear scaling ($k \approx 1$) (Refs: (1, 76–84)). The data for home range area has focused on mammals, birds and some other vertebrates, but overall are less extensive than the data for population density shown in Fig. 1 B. If there is no systematic change across species in the average encounter rates of individuals of the same species inside a given territory, then encounter rate is simply a multiplier of the coefficient and does not change the scaling exponent (84). Population abundance is then the inverse of home range area, multiplied by the average number of other members of the same species within an average home range area at any given time.

We randomly sample the abundance-mass data for mammals, on which many home range area studies cited above have focused. If we assume individuals of any species encounter other members of their population within their home range with similar frequency, then the inverse of population density ($1/N$; $(\text{individuals/area})^{-1}$) is proportional to home range area (A). On average, this gives home range area scaling with mass near ¾ across all mammals ($A \sim m^{¾}$). For illustration, we compare to data from Pantheria (85) (Fig. S8 A; right hand side plot), which exhibits a home range area vs. mass slope near one, similar to what is commonly reported (1, 76–83), but differing significantly from the near ¾ mass exponent obtained from sub-sampling the abundance data. This suggests that encounter rates or group sizes increase with body mass (84). Carnivores tend to have steeper scaling than herbivores (76, 82), which is also the case for abundance-mass scaling (1, 10) (Table S2), suggesting a reciprocal connection between these ways of viewing abundance.

B) *Size spectra scaling*

This has also been called the Sheldon spectra (86) or abundance-spectra, and is the size-frequency distribution of individuals in a community across logarithmic size bins. It has most commonly been observed in aquatic systems, and represents the total abundance of all individuals,

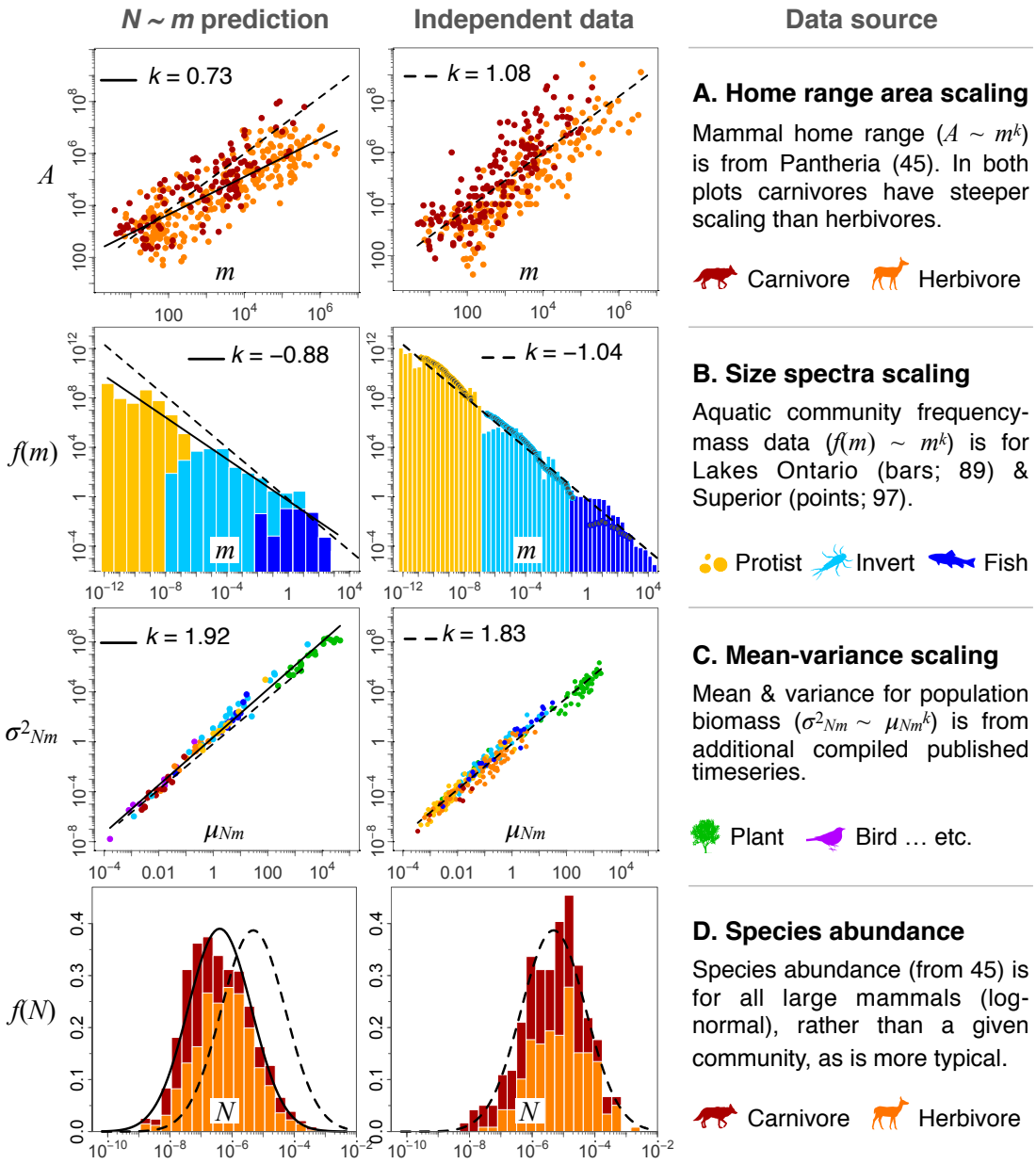

Fig. S8. Comparing abundance sub-sample predictions with direct measures. Abundance and mass ($N \sim m$) data from Fig. 1 B were sampled and simple functions applied to obtain the plots and solid line predictions, as described in Box 1 and reproduced in the plots on the left. These are compared with direct measurements of these quantities obtained from independent data sources, shown at right, with their best fit predictions shown by the dashed line in both plots. The taxonomic groups sub-sampled at left were chosen based on the data available to compare against at right. Further discussion of the discrepancy between sub-sample predictions (left) and known abundance patterns (right) is in section 8.

regardless of species identity, in each log size bin. This pattern is known to follow a power law size-frequency distribution with near inverse scaling ($f(m) \sim m^{-1}$) (Refs: (1, 46, 86–93)). The size

spectra scaling and abundance-mass scaling should be the same if the distribution of species body masses and diversity is roughly constant across logarithmic size classes (1, 94).

We randomly sample abundance-mass data among protists and ectotherms (thus ignoring mammals and birds, which are mostly terrestrial). We then sum the abundances of all species within a size-class, which gives a size-frequency distribution of different species within our hypothetical random community. We find an approximate power law relation between size and frequency, with average scaling near $k = -0.88$, regardless of how we sample the abundance-mass data, provided our sample is evenly distributed across size classes. This is largely consistent, though somewhat shallower than what is commonly observed empirically. For illustration, we compare our sub-sample to data from Lake Ontario with $k = -1.04$ (87) and Lake Superior with $k = -1.1$ (95) (Fig. S8 B; right hand side plot).

There are, however, some important differences between abundance-mass scaling and size-spectra scaling. The size spectra refers to a single community such as a lake or a patch of ocean, while the abundance-mass relation encompasses very different terrestrial and aquatic communities. Moreover, the size spectra is the sum of all abundances of all species within a size class, while the abundance-mass relation is concerned with the abundance of a single species of a given adult size. The average adult size and abundance of a species of fish, for example, is also quite different from the actual sizes and abundances of larvae, juveniles, and adults of that same species residing in a community, and though large fish are comparatively very rare, they produce a disproportionately large number of small offspring (60), most of which are consumed as juveniles. Some communities may also not have equal diversity across logarithmic size classes (96). The connections between the within-community size-spectra and the cross-community abundance-mass relation is more complex than the simple assumptions we have employed, and may account for the differences we observe in exponent estimates.

C) *Mean-variance scaling*

This is also called Taylor's law or fluctuation scaling, and is a power law relation between the variance σ^2 and mean μ in population abundance. This relation holds for a single species through time or between different sites at a given time, as well as a wide variety of other biological and even non-biological phenomena (97–101). The relation $\sigma^2 \sim \mu^k$ is widely observed in every kind of population, often scaling as $1.5 < k < 2$. Mean-variance scaling as the square is equivalent to the standard deviation scaling proportional with the mean ($k = 1$), or a constant coefficient of variation (CV) across different mean population sizes ($k = 0$).

Since we do not have sufficient data on the fluctuations of individual species across the size range in our abundance dataset, we assume a population varies over the same range as other species within its size class, as suggested by mammal variations in Fig. S5. We sample the abundance-mass data across the size range, and relate the mean and variance of population biomass (Nm; g/m^2) in different log size classes. We find a power law relation with an exponent that is on average near $k = 1.9$, regardless of how we sample or bin samples across the size range. This is consistent with observed mean-variance scaling. For illustration, we compare this to time-series data that we compiled for $n = 320$ species spanning the eukaryote size range, showing a nearly identical scaling pattern (Fig. S8 C; right hand side plot). A similar pattern extending over many more orders of magnitude is obtained if we relate the mean and variance of population density (N; ind./m^2) rather than population biomass. The difference between mean-variance scaling between abundance (N)

and biomass (*Nm*) is that taxonomic groups are shifted dramatically in their position on the line, but the exponent remains very similar in both cases.

D) *Species abundance distribution*

This has also been called relative abundance, and is the frequency distribution of the number of species in different abundance classes (102–107). This pattern of commonness and rarity ('most species are rare, but a few are common, and a few are very rare') is widely observed in different communities, though the precise distribution is often debated (102, 106). Many of the best-sampled communities, however, tend to exhibit a log-normal distribution (103, 105, 107). Most species abundance distributions are observed from a sample of a community of species that typically range in body mass over fewer than three orders of magnitude. These distributions tend to focus on particular taxonomic groups, and as such, are more focused community studies than the aquatic size-spectra discussed above.

We sample the abundance-mass data for large mammals in the size range of 1 to 1000 kg, where we have numerous individual measurements for multiple species (Fig. S5), and find the sample exhibits a log-normal distribution, consistent with many species abundance distributions. For illustration, we compare this to the distribution of mammal abundance data in Pantheria (85) (Fig. S8 D; right hand side plot), which reports a single estimate of abundance for each mammal species, and which were not included in our abundance dataset.

We do not yet fully understand the mechanistic basis for the connections between these relations. These different four different ways of considering abundance are broadly consistent with the functional form of the near inverse scaling (Fig. S8 A and B) and the residual variability (Fig. S8 C and D) in the abundance-mass relation. Further work is needed to understand the mismatches in the scaling in A and B, how cross-system pattern in A and C are related to within-community patterns in B and D.

9. Control of growth and metabolism

The regulation of growth is complex, integrating local and systemic signals to achieve strict size targets across multiple tissues and organs, through highly characteristic development trajectories (108). These targets and trajectories are often stable under a range of perturbations, such as compensatory growth in organs and tissues, or catch-up growth of juveniles to the normal growth curve following growth suppression (108). These growth corrections are known to occur only when energy is available, but suggest acute fidelity and non-linear negative feedback control rather than a passive response to energy supply (31, 108).

Physiological data are consistent with a complex genetic program regulating growth that operates across multiple organs during juvenile development (108, 109). This program involves the up- or down-regulation of hundreds of genes with size increases, and suggests that growth is fundamentally regulated with downstream effects on metabolism. A more mechanistic understanding of the links between the scaling of growth and metabolism can be gained by considering their regulation. By observing how qualitative changes in one variable, be they normal, abnormal, or experimental, correlate to changes in the other variable, we can better untangle the

order of their control. Although a lack of energy will limit growth, an oversupply rarely induces a proportionate response in growth. This includes normal changes in metabolic activity state, abnormal conditions such as hyperthyroidism, and force-feeding or selection experiments (9, 31). Conversely, in many cases, changes in growth are correlated to changes in metabolism (9, 14, 23, 27, 30–32). As discussed further below, this includes i) normal changes in growth rate associated with particular developmental stages and growth rate adaptations to different selection pressures (with corresponding changes in metabolic scaling) (8, 9, 22, 23, 30); ii) abnormal changes to growth regulation in many cancers (with corresponding promotion of angiogenesis and ATP-generating processes) (110, 111); and, iii) experimental injections of growth hormone or selection for faster growth rates (with downstream effects on greater feeding rates and digestive efficiency) (14, 30, 31, 112). Below we consider cases where this order of causality between growth and metabolism is most evident.

Normal ontogenetic growth

The growth rate in early ontogeny in most vertebrates and plants is high in early ontogeny and slows as an organism approaches maturity. Consistent with these temporal changes are shifts in metabolic exponents, which tend to be higher in early development stages than later in development (23, 30). These correlations hold for pelagic animals with high growth throughout their lives, where metabolic scaling is approximately linear with mass through the lifespan. Moreover, the correlation also holds for insects such as cockroaches, that have slow growth in early development (instars 1-4) and more rapid growth later on (instars 5 and 6; Refs: (8, 9, 23, 30)). In comparisons of the same species of amphipod that grow at varying rates based on their exposure to predation, it has been shown that metabolic scaling is influenced by the needs of growth (22). In many fish species, the evolution of differential growth rates cause the variation in the scaling of metabolic rate (113). Intra-specific studies on invertebrates, such as snail (30), blue mussel (23) and amphipod (22), have shown that selection for rapid growth is associated with an increase in metabolic scaling.

Compensatory growth

Compensatory growth can refer to a variety of observations including catch-up growth in juvenile individuals, the accelerated growth of transplanted juvenile organs into adult hosts, or of regenerating tissues. Catch-up growth is the observation that a juvenile whose growth is suppressed through lack of food or disease, can recover its original growth trajectory if normal conditions resume in time (108, 114). Such stunted juveniles exhibit gains in size above statistical limits of normality for age, returning them to the normal growth curve (114–116). This indicates that the regulation of growth has much greater adaptive fidelity than simply responding to energy supply. Moreover, this is known to have downstream effects on metabolism into adulthood (117), possibly through increased insulin production (118). Energy limited conditions can thus limit growth, but when conditions resolve, metabolism can be induced to fuel growth acceleration to return to the normal growth curve.

Another form of compensatory growth concerns juvenile organ transplants into adult hosts, which can often accumulate mass faster than they would in smaller juvenile individuals. Juvenile organ transplants of intestine (119), heart (120) and other organs into adult hosts cause compensatory growth of the organ (108), and also induce changes to energetics (14, 119). Finally, at the tissue level, damage can be repaired through cell division by less metabolically active, fully

differentiated cells in at least kidney (121) and pancreas (122). These phenomena suggest that the regulation of cell division can exceed expectations based on normal energy supply, by inducing associated changes in metabolism.

Abnormal regulation of growth

In a great variety of cancers, oncogene and tumor-suppressor gene mutations all operate to increase tumor cell number through cell division or the inhibition of apoptosis (123). Cancer is thus considered as an abnormal change in the regulation of growth, which typically has attendant secondary effects on metabolism (123, 124). At various stages cancer mutations can cause a switch to aerobic glycolysis and/or tumors can induce angiogenesis to create the vasculature to supply the growing tumor's increased need for energy (110, 111, 125). Where it is unable to do so, the tumor often becomes benign, showing how limits to energy can halt growth. While there are still complex feedbacks, such as the fact that many cancers require certain metabolites for growth (e.g. the Warburg effect (37, 126)), there is rarely direct causality from metabolic supply to cancer growth. If cancer were merely the result of an oversupply of energy, we should expect highly active tissues such as skeletal muscle and brain to exhibit higher incidence of cancer, which they do not (9). Instead, energy supply is known to often adjust to the needs of growing tumors.

Experimental alterations to growth

A variety of experimental alterations to growth result in downstream effects on metabolism. Growth hormone deficiencies or injections can inhibit or stimulate growth, respectively, and this is often accompanied by correlated downstream changes in feeding rates or energetic efficiency (14, 127). The same phenomenon occurs with targeted ablations of a number of growth-regulating genes (14). Artificial selection experiments with domesticated animals reveal that selecting for more rapid mass-specific growth also cause more efficient utilization of food consumed and promote greater feeding rates (14, 128). Manipulations of animal embryos for increased cell division also increase oxygen consumption rates, but increases in metabolic rate have little effect on growth rate (129). Moreover, transgenic individuals with elevated growth hormone have increased growth rates, and higher metabolic rates (30, 112).

Endocrine control of growth and metabolism

One alternative way to account for many of these observations is if the regulation of metabolism is partly nested within the regulation of growth. An example is provided by endocrine signaling whereby the pituitary release of thyroid stimulating hormone controls metabolism needed for growth, but growth is also regulated directly by pituitary secretion of growth hormone and other factors (Fig. S9). This scheme accounts for limits to energy limiting growth, but also how growth can be regulated upstream of metabolism to ensure co-regulation in both variables.

The hypothalamic-pituitary-thyroid axis in many vertebrates exemplifies the regulation of growth and metabolism at the whole-organism level. The anterior pituitary regulates growth, while the thyroid primarily regulates metabolism, both of which in turn have consequences for growth and development. The secretion of hormones by the thyroid is itself controlled by thyroid stimulating hormone released by the anterior pituitary (130). The anterior pituitary thus regulates growth directly through the production of growth hormone and other factors, but also regulates the thyroid's control on metabolism, which fuels protein synthesis necessary for growth (131). If growth has feedbacks on pituitary function, it represents a nested form of regulation whereby

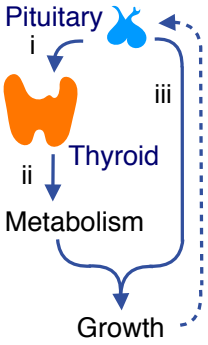

Fig. S9. Endocrine regulation of growth and metabolism. The hypothalamic-pituitary-thyroid axis represents an example of the regulation of metabolism nested within that of growth. The pituitary releases thyroid stimulating hormone (TSH; i) which controls metabolism (ii) in multiple organs and supplies the energy needed for growth, reproduction and turnover. Limits to the raw materials of metabolism will thus limit growth. But growth is also regulated directly by the pituitary release of growth hormone and other factors (iii). Any feedback from growth to the pituitary (dashed line) will result in growth being co-regulated with metabolism.

metabolism has proximal effects on growth, but growth can ultimately control metabolism through the function of the pituitary (Fig. S9).

Abnormal regulation of metabolism can have adverse effects on growth, but rarely in ways that would be expected if growth responded passively to the supply of energy. Hyper- or hypo-thyroidism are abnormalities in the ability of the thyroid to regulate metabolic rate throughout the body. Hypothyroidism lowers metabolism and in some cases can restrict growth, but rarely in any predictable way. Conversely, hyperthyroidism does not result in any predictable weight gain, and is often associated with weight loss.

The function of these glands is often related to their size, with abnormalities in the size of the pituitary and thyroid correlated with abnormalities in the regulation of growth and metabolism respectively. Hyperthyroidism (excess production of thyroid hormones) is often associated with an enlarged thyroid, while hypothyroidism (inadequate secretion of thyroid hormones) can be associated with thyroidectomy (partial removal of the thyroid). Pregnancy also results in an enlarged thyroid, which results in an increased production of thyroxine. Hyperpituitarism (excess production of pituitary hormones) is most often caused by hormone secreting pituitary adenomas, which can also cause hyperthyroidism. As adenomas enlarge, they can compress cells of the normal pituitary, and if the adenoma is not functional, it can lead to hypopituitarism.

These physiological observations of how natural or induced changes in growth have correlated downstream impacts on metabolism make the case that size scaling may be based in growth dynamics rather than metabolic constraints, as a number of other authors have suggested (9, 14, 23, 27, 30–32, 132).

10. Data file

The Supplementary Data includes all data presented in Figs. 1 and 3. While the figures show average values for a particular species, where multiple estimates were made, the Data file include all original individual estimates, conversion factors, taxonomic information where available, notes and referenced sources.

The data is an Excel file (.xls) with six worksheets. Below, we describe each worksheet:

Refs: This worksheet contains background information about the data file and a list of all primary references.

Metabolism. This worksheet contains all basal metabolism data in units of watts, temperature in °C, where available, and published Q_{10} values.

Abundance. This worksheet contains all population density data in units of individuals/m^2 and location description, where available.

Growth. This worksheet contains all maximum individual productivity data in units of grams/year.

Mortality. This worksheet contains all mortality rates data in units of 1/yr, as well as noting whether lifespan is measured in captivity or the wild.

MammalRange. This worksheet contains all data for mammals from Fig. 3 B, including maximum and minimum metabolism, and ontogenetic and prenatal growth across mammal species. These are arranged one after another as separate datasets with different column headings.

The worksheets describing the four basic variables (described above) are structured similarly for the first eight columns of data, after which additional columns are more variable-specific. The first eight columns allow a consistent work flow across variables and are described below:

UniqueID: This is a unique number for each row of data.

Plot: This lists integers from 1 to 6, specifying the major taxonomic group as follows: 1-mammal; 2-protist; 3-plant; 4-ectotherm; 5-bird; 6-prokaryote (where available). The number 9999 is used to identify which rows of data were excluded from analysis, with the reason given under the "Notes" column.

Plot2: This lists integers from 1 to 9, specifying more resolved taxonomic groups as follows: 1-herbivore mammal; 2-carnivore mammal; 3-protist; 4-plant; 5-invertebrate; 6-vertebrate ectotherm; 7-bird; 8-bacteria; 9-omnivore mammal. The number 9999 is used to identify data excluded from analysis, with the reason given under the "Notes" column.

Major_taxa: This lists the major taxonomic group, corresponding to the Plot and Plot2 columns.

Order: This lists the taxonomic order of the species.

Species: This lists the species binomial, where available.

Mass_g: This is the body mass in grams.

The column after Mass_g includes the data for the variable of interest and has different name headings and units in different worksheets (units are specified in Table 1 A).

In addition to these eight columns and additional variable specific columns, a number of other columns are similar across datasets, but are not always completely populated. They are as follows: **Trophic** refers to the primary trophic level of the species and is mostly only populated for mammals; **Group** refers to arbitrary high-level taxonomic classification used to build regression Tables S1 to S8; **Genus** refers to taxonomic genus; **Reference** refers to the reference code (usually first author and year) that serves to locate the reference (on the Refs worksheet) for the row of data; and, **Notes** refers to additional information that cannot be accommodated other columns.

11. Source code

Source code is available as a supplementary RStudio Project called “Link-scaling.Rproj”, available at (https://zenodo.org/record/3145281). This code reproduces Tables S1 to S8. By opening the "Link-scaling.Rproj" file and running all the "Analysis.R" code, the analysis described in the paper can be reproduced, or altered in various ways. The "Analysis.R" code reads in the following data files: “Metabolism.csv”, “Abundance.csv”, “Growth.csv” and “Mortality.csv”. These data files are located in the "Data" folder in the "Link scaling" folder along with the Excel file that reproduces these data and includes additional data and original references, described in Section 10, above.

The “Analysis.R” code calls a number of functions (from "Funx.R"). The tempcorr() function allows the specification for how basal metabolism is temperature corrected to a particular temperature, using either published Q10 values or the Arrhenius equation with a standard activation energy. The aggre() function allows specifying how multiple measures of a species are aggregated (using functions of min, max, mean or geometric mean), as described in Section 2, above. For both of these functions we can also specify which major groups the functions apply to, indicated at the parameter plot2. Plot2 lists numbered major groups in each dataset as follows: 1 - Herbivore mammal; 2 - Carnivore mammal; 3 – Protist; 4 – Plant; 5 – Invertebrate; 6 - Ectotherm vertebrate; 7 – Bird; 8 – Bacteria; 9 - Omnivore mammal.

The combVar() function allows basic variables (metabolism, abundance, growth and mortality) to be combined through multiplication, either through direct species matches, or if matches are not available, at the order-level, and then followed by major group-level using regression predictions, as described in Section 3, above. The regtable() function outputs regression summary statistics equivalent to Tables S1 to S8 in the Supplementary Data file (for the default function parameters). The taxonomic order-level regressions are returned based on a regression meeting the minimum number of user-specified data points (e.g. len=15) and the minimum mass range of two orders of magnitude (e.g. rang=100). Order-level regressions can be excluded from results by setting orderadd=FALSE. A single regression on user-specified data can be returned from the segslope() function, which can also draw the regression line on plotted data. More details on the functions are included in "Funx.R".

12. Regression tables

Tables S1 to S4 list basic variable regressions and Tables S5 to S8 list combined variable regressions. All regressions have body mass in grams on the *x*-axis, and are species’ aggregate values, except for plants and the relatively few data that were not resolved to species level. The exception is for mortality, given the limited available data among smaller size classes (protists and invertebrates). In order to increase representation among these smaller sized groups, we did not aggregate their mortality estimates, and thus only aggregated mammals and birds. “All measurements” shows regressions for the entire relevant dataset without any species level aggregation. Taxonomic order level regressions are shown dashed (e.g. - Artiodactyla), where data extend over at least two orders of magnitude and have $n > 15$ species.

Column headings to Tables S1 to S8 are as follows:

x-ref: cross-reference figure that displays the data;

n: number of data points (usually number of species);

k: scaling exponent;

95% CI (*k*): 95% confidence interval on the exponent *k*;

R^2: coefficient of determination;

c: regression coefficient (*y* value at *x*=1);

Range mass: g: range in body mass in grams;

Sy•x: Standard deviation of the log residual variation of the regression;

***p*-value**: Probability value (only in Tables S5 to S8).

All original data and references for these regressions are included in the Supplementary data file. The source code (section 11) allows all regression tables to be reproduced with the default settings, and alternative regressions can be generated for different user-specified parameters. Data and source code reproducing these tables is available at (https://zenodo.org/record/3145281).

Table S1. Basal metabolism (watts) to body mass (g) regressions.

Metabolism is temperature corrected to 20 °C, except in birds and mammals.

x-ref:	Fig. 1 A	*n*	*k*	95% CI (*k*)	R^2	*c*	Range mass: g	*Sy·x*
S1 .1	All species	3965	**0.96**	0.95 : 0.97	0.96	0.0014	1.0E-14 : 4.7E+6	0.64
S1 .2	Eukaryotes	3816	**0.95**	0.94 : 0.95	0.94	0.0014	6.0E-12 : 4.7E+6	0.64
S1 .3	Mammals	698	**0.72**	0.71 : 0.73	0.96	0.019	2.2 : 3.7E+6	0.17
S1 .4	Carnivore	236	**0.74**	0.72 : 0.76	0.96	0.018	2.2 : 3.2E+6	0.2
S1 .5	Omnivore	200	**0.64**	0.62 : 0.67	0.93	0.027	7.4 : 6.0E+4	0.14
S1 .6	Herbivore	262	**0.73**	0.71 : 0.74	0.97	0.02	7.3 : 3.7E+6	0.15
S1 .7	Eutherian mammal	613	**0.73**	0.72 : 0.74	0.96	0.019	2.2 : 3.7E+6	0.17
S1 .8	- Artiodactyla	24	**0.75**	0.69 : 0.8	0.97	0.021	1600 : 4.1E+5	0.085
S1 .9	- Carnivora	67	**0.76**	0.7 : 0.82	0.92	0.016	77 : 3.9E+5	0.19
S1 .10	- Chiroptera	92	**0.78**	0.73 : 0.83	0.92	0.014	3.7 : 1000	0.12

Table S1 continued. Basal metabolism (watts) to body mass (g) regressions.
Metabolism is temperature corrected to 20 ℃, except in birds and mammals.

x-ref:	Fig. 1 A	*n*	*k*	95% CI (*k*)	R^2	*c*	Range mass: g	*Sy·x*
S1 .11	- Eulipotyphla	34	**0.52**	0.41 : 0.64	0.72	0.052	2.2 : 750	0.17
S1 .12	- Primate	33	**0.78**	0.71 : 0.85	0.95	0.012	62 : 6.0E+4	0.15
S1 .13	- Rodentia	295	**0.67**	0.65 : 0.7	0.91	0.026	7.3 : 2.6E+4	0.14
S1 .14	Marsupial mammal	81	**0.71**	0.68 : 0.73	0.98	0.016	7.1 : 4.5E+4	0.1
S1 .15	- Dasyuromorphia	22	**0.74**	0.7 : 0.78	0.99	0.014	7.1 : 5000	0.077
S1 .16	- Diprotodontia	28	**0.71**	0.67 : 0.74	0.98	0.017	10 : 2.9E+4	0.096
S1 .17	Protist	95	**0.89**	0.84 : 0.95	0.92	5.3E-4	6.0E-12 : 2.2E-4	0.49
S1 .18	- Ciliophora	18	**0.82**	0.37 : 1.3	0.48	2.1E-4	1.5E-8 : 1.2E-5	0.65
S1 .19	Plant	337	**0.84**	0.82 : 0.85	0.97	6.8E-4	0.0093 : 4.7E+6	0.28
S1 .20	Ectotherm animal	2314	**0.79**	0.79 : 0.8	0.92	6.3E-4	1.6E-7 : 1.6E+4	0.43
S1 .21	Invertebrate	1719	**0.81**	0.79 : 0.82	0.88	6.7E-4	1.6E-7 : 1.2E+4	0.43
S1 .22	- Amphipoda	52	**0.97**	0.74 : 1.2	0.59	0.0015	4.3E-4 : 1.6	0.68
S1 .23	- Araneae	112	**0.6**	0.53 : 0.66	0.75	3.7E-4	7.2E-4 : 1	0.26
S1 .24	- Calanoida	17	**0.56**	0.22 : 0.9	0.45	2.3E-4	7.7E-6 : 0.019	0.57
S1 .25	- Cephalopoda	37	**0.85**	0.69 : 1	0.77	7.4E-4	0.0012 : 1.2E+4	0.67
S1 .26	- Coleoptera	373	**0.78**	0.74 : 0.81	0.84	8.5E-4	7.7E-5 : 7.3	0.32
S1 .27	- Collembola	30	**0.83**	0.74 : 0.92	0.93	6.8E-4	3.1E-6 : 0.0048	0.22
S1 .28	- Copepoda	115	**0.66**	0.6 : 0.72	0.82	2.5E-4	2.6E-6 : 0.06	0.38
S1 .29	- Decapoda	88	**0.68**	0.61 : 0.76	0.79	6.8E-4	3.0E-4 : 950	0.41
S1 .30	- Diptera	23	**0.64**	0.5 : 0.79	0.80	0.0017	4.9E-4 : 1.7	0.3
S1 .31	- Hemiptera	20	**0.7**	0.6 : 0.8	0.92	0.0015	1.6E-4 : 2.8	0.24
S1 .32	- Hymenoptera	55	**0.8**	0.69 : 0.92	0.79	9.0E-4	1.0E-4 : 1.3	0.37
S1 .33	- Isopoda	120	**0.69**	0.62 : 0.76	0.76	3.6E-4	6.4E-4 : 33	0.28
S1 .34	- Oribatida	45	**0.7**	0.54 : 0.86	0.63	6.2E-5	1.7E-6 : 3.3E-4	0.27
S1 .35	- Orthoptera	35	**0.65**	0.49 : 0.81	0.66	0.0013	0.018 : 2.9	0.31
S1 .36	Vertebrate	595	**0.83**	0.8 : 0.86	0.83	5.0E-4	0.02 : 1.6E+4	0.4
S1 .37	- Anura	95	**0.8**	0.73 : 0.87	0.85	5.6E-4	0.27 : 560	0.25
S1 .38	- Caudata	67	**0.82**	0.73 : 0.9	0.85	2.8E-4	0.15 : 650	0.26
S1 .39	- Cypriniformes	32	**0.86**	0.74 : 0.97	0.89	7.6E-4	0.3 : 1800	0.32
S1 .40	- Perciformes	87	**0.87**	0.75 : 1	0.70	5.8E-4	0.02 : 2600	0.58
S1 .41	- Squamata	152	**0.79**	0.75 : 0.83	0.91	5.2E-4	0.4 : 1.6E+4	0.27
S1 .42	Bird	372	**0.66**	0.65 : 0.68	0.95	0.039	2.9 : 1.0E+5	0.13
S1 .43	Passerine	181	**0.74**	0.7 : 0.77	0.93	0.034	5.2 : 1200	0.09
S1 .44	Non passerine	191	**0.7**	0.68 : 0.72	0.94	0.029	2.9 : 1.0E+5	0.14
S1 .45	Bacteria	149	**1.1**	0.99 : 1.2	0.66	0.064	1.0E-14 : 5.6E-9	0.61

Table S2. Population density (count/m^2) to body mass (g) regressions.

x-ref:	Fig. 1 B	*n*	*k*	95% CI (*k*)	R^2	*c*	Range mass: g	*Sy·x*
S2 .1	All species	3051	**-0.95**	-0.97 : -0.94	0.81	0.33	6.6E-14 : 3.2E+6	2.3
S2 .2	Eukaryotes	2880	**-0.91**	-0.93 : -0.89	0.73	0.29	9.3E-14 : 3.2E+6	2.3
S2 .3	Mammals	608	**-0.79**	-0.83 : -0.74	0.63	0.009	3.8 : 2.8E+6	0.8
S2 .4	- Carnivore	90	**-0.94**	-1 : -0.84	0.80	0.0036	3.8 : 3.9E+5	0.65
S2 .5	- Omnivore	180	**-0.71**	-0.83 : -0.6	0.47	0.0068	6.4 : 1.3E+5	0.74
S2 .6	- Herbivore	338	**-0.77**	-0.82 : -0.72	0.72	0.013	5 : 2.8E+6	0.69
S2 .7	Eutherian mammal	578	**-0.8**	-0.85 : -0.75	0.65	0.01	3.8 : 2.8E+6	0.8
S2 .8	- Artiodactyla	103	**-0.28**	-0.5 : -0.056	0.06	3.3E-5	2600 : 1.4E+6	0.64
S2 .9	- Carnivora	61	**-0.83**	-1 : -0.64	0.57	0.0011	79 : 3.9E+5	0.58
S2 .10	- Eulipotyphla	17	**0.27**	-0.2 : 0.74	0.09	1.9E-4	3.8 : 800	0.5
S2 .11	- Lagomorpha	21	**-0.5**	-0.86 : -0.14	0.31	0.0015	20 : 3.2E+4	0.58
S2 .12	- Primate	118	**-0.38**	-0.56 : -0.21	0.14	5.3E-4	60 : 1.3E+5	0.6
S2 .13	- Rodentia	227	**-0.62**	-0.75 : -0.48	0.27	0.0072	5 : 1.0E+5	0.73
S2 .14	Marsupial mammal	30	**-0.25**	-0.47 : -0.033	0.17	2.3E-4	15 : 4.1E+4	0.58
S2 .15	Protist	301	**-0.8**	-0.9 : -0.71	0.48	33	9.3E-14 : 1.3E-7	0.87
S2 .16	Plant	412	**-0.74**	-0.76 : -0.73	0.96	810	6.0E-5 : 3.2E+6	0.34
S2 .17	- Fagales	73	**-0.6**	-0.66 : -0.54	0.85	150	160 : 3.2E+6	0.19
S2 .18	- Malpighiales	26	**-0.65**	-0.99 : -0.31	0.39	340	2700 : 2.1E+6	0.61
S2 .19	- Pinales	174	**-0.62**	-0.67 : -0.57	0.79	200	1.7 : 2.2E+6	0.26
S2 .20	Ectotherm animal	957	**-0.76**	-0.79 : -0.74	0.75	1.3	2.3E-8 : 1.2E+4	1.2
S2 .21	Invertebrate	739	**-0.61**	-0.65 : -0.57	0.55	5.5	2.3E-8 : 630	1.1
S2 .22	- Calanoida	15	**-0.33**	-0.83 : 0.16	0.14	1000	1.2E-5 : 0.002	0.61
S2 .23	- Cladocera	15	**-0.06**	-0.83 : 0.71	0.00	2900	4.8E-6 : 5.8E-4	0.79
S2 .24	- Diptera	61	**-0.48**	-0.64 : -0.31	0.35	29	2.6E-7 : 0.6	0.71
S2 .25	- Haplotaxida	15	**-0.86**	-1.2 : -0.49	0.66	4.6	8.2E-6 : 1.9	0.86
S2 .26	- Heterobranchia	32	**-0.37**	-0.75 : 0.0053	0.12	1.7	1.9E-4 : 5.8	1.2
S2 .27	Vertebrate	218	**-0.96**	-1.1 : -0.82	0.46	0.92	0.033 : 1.2E+4	1.1
S2 .28	- Cypriniformes	32	**-0.78**	-1.1 : -0.49	0.50	0.32	0.29 : 2400	0.86
S2 .29	- Perciformes	23	**-0.63**	-1.2 : -0.09	0.22	0.28	6 : 2600	0.95
S2 .30	- Squamata	30	**-1**	-1.3 : -0.74	0.66	0.068	0.84 : 1.2E+4	0.75
S2 .31	Bird	602	**-0.47**	-0.55 : -0.4	0.19	9.9E-5	2.9 : 1.1E+5	0.64
S2 .32	Carnivore	391	**-0.7**	-0.82 : -0.58	0.26	1.8E-4	3.4 : 4600	0.6
S2 .33	Omnivore	45	**-0.24**	-0.51 : 0.037	0.07	4.3E-5	12 : 3100	0.52
S2 .34	Herbivore	66	**-0.32**	-0.46 : -0.17	0.24	5.9E-5	2.9 : 1.1E+5	0.63
S2 .35	Bacteria	171	**-0.63**	-0.69 : -0.58	0.77	2.2E+4	6.6E-14 : 3.9E-11	0.26

Table S3. Maximum individual productivity (g/yr) to body mass (g) regressions.

x-ref:	Fig. 1 C	*n*	*k*	95% CI (*k*)	R^2	*c*	Range mass: g	*Sy·x*
S3 .1	All species	2729	**0.74**	0.73 : 0.74	0.97	2.7	4.0E-14 : 1.4E+8	0.43
S3 .2	Eukaryotes	2692	**0.73**	0.73 : 0.74	0.96	2.8	2.0E-11 : 1.4E+8	0.42
S3 .3	Mammals	982	**0.75**	0.74 : 0.76	0.92	4.3	2.1 : 1.4E+8	0.34
S3 .4	Carnivore	332	**0.8**	0.78 : 0.82	0.96	2.8	2.1 : 1.4E+8	0.31
S3 .5	Omnivore	221	**0.53**	0.49 : 0.58	0.70	14	6 : 1.4E+5	0.34
S3 .6	Herbivore	429	**0.73**	0.71 : 0.75	0.92	6	8 : 4.8E+6	0.31
S3 .7	Eutherian mammal	878	**0.75**	0.74 : 0.77	0.92	4.2	2.1 : 1.4E+8	0.35
S3 .8	- Artiodactyla	126	**0.82**	0.77 : 0.87	0.88	2.4	1500 : 3.8E+6	0.18
S3 .9	- Carnivora	146	**0.76**	0.72 : 0.8	0.90	4.5	60 : 1.0E+6	0.23
S3 .10	- Cetacea	31	**0.92**	0.85 : 0.98	0.97	0.31	3.2E+4 : 1.4E+8	0.21
S3 .11	- Chiroptera	94	**0.92**	0.8 : 1	0.73	1.3	3 : 800	0.29
S3 .12	- Eulipotyphla	25	**0.73**	0.49 : 0.97	0.64	7.6	2.1 : 770	0.42
S3 .13	- Primate	114	**0.66**	0.61 : 0.71	0.86	3.4	64 : 1.4E+5	0.19
S3 .14	- Rodentia	268	**0.72**	0.67 : 0.77	0.76	7	6 : 5.5E+4	0.31
S3 .15	- Xenarthra	15	**1**	0.75 : 1.3	0.83	0.27	220 : 4.5E+4	0.25
S3 .16	Marsupial mammal	104	**0.75**	0.71 : 0.8	0.91	5.1	6.1 : 5.5E+4	0.27
S3 .17	- Dasyuromorphia	36	**0.85**	0.77 : 0.93	0.94	3	6.1 : 8000	0.18
S3 .18	- Diprotodontia	51	**0.78**	0.71 : 0.84	0.92	3.5	9.5 : 5.5E+4	0.2
S3 .19	Protist	124	**0.87**	0.83 : 0.91	0.93	47	2.0E-11 : 1.8E-6	0.3
S3 .20	Plant	132	**0.69**	0.64 : 0.75	0.80	2.1	1.7 : 3.2E+6	0.33
S3 .21	- Fagales	42	**0.77**	0.66 : 0.88	0.83	0.74	2.6E+4 : 3.2E+6	0.19
S3 .22	- Pinales	68	**0.74**	0.67 : 0.81	0.86	1.3	1.7 : 2.2E+6	0.29
S3 .23	Ectotherm animal	306	**0.76**	0.74 : 0.77	0.96	3.7	2.2E-7 : 1.9E+7	0.5
S3 .24	Invertebrate	187	**0.8**	0.74 : 0.86	0.79	5.3	2.2E-7 : 5.8	0.58
S3 .25	- Diptera	49	**0.79**	0.63 : 0.95	0.66	2.5	2.6E-7 : 0.6	0.58
S3 .26	- Hemiptera	25	**0.88**	0.72 : 1	0.85	34	2.7E-4 : 0.053	0.25
S3 .27	Vertebrate	119	**0.81**	0.77 : 0.85	0.93	2.3	0.54 : 1.9E+7	0.32
S3 .28	- Carcharhiniformes	27	**0.84**	0.73 : 0.94	0.92	1.6	730 : 4.4E+5	0.17
S3 .29	- Cypriniformes	23	**0.82**	0.68 : 0.97	0.87	2	1.8 : 2400	0.35
S3 .30	- Perciformes	16	**0.91**	0.8 : 1	0.96	2.2	0.54 : 3.3E+4	0.25
S3 .31	Bird	1148	**0.69**	0.67 : 0.72	0.76	2	3.1 : 1.1E+5	0.34
S3 .32	Passerine	461	**0.77**	0.71 : 0.83	0.58	1.6	5.2 : 1000	0.26
S3 .33	Non passerine	687	**0.68**	0.64 : 0.72	0.62	2.2	3.1 : 1.1E+5	0.38
S3 .34	- Gruiformes	27	**0.24**	0.13 : 0.35	0.45	56	35 : 8800	0.21
S3 .35	- Pelecaniformes	37	**0.51**	0.26 : 0.75	0.33	8.7	86 : 9500	0.4
S3 .36	- Procellariiformes	70	**0.76**	0.68 : 0.83	0.85	0.66	23 : 8900	0.21
S3 .37	Bacteria	37	**1.3**	1.1 : 1.5	0.79	9.0E+6	4.0E-14 : 1.2E-11	0.36

Table S4. Natural mortality rate (1/yr) to body mass (g) regressions.

x-ref:	Fig. 1 D	*n*	*k*	95% CI (*k*)	R^2	*c*	Range mass: g	*Sy·x*
S4 .1	Eukaryotes	3798	**-0.24**	-0.25 : -0.24	0.60	0.74	3.0E-14 : 1.5E+8	0.48
S4 .2	Mammals	1079	**-0.15**	-0.16 : -0.13	0.32	0.51	1.9 : 1.5E+8	0.31
S4 .3	- Carnivore	365	**-0.12**	-0.14 : -0.094	0.23	0.34	1.9 : 1.5E+8	0.38
S4 .4	- Omnivore	241	**-0.31**	-0.34 : -0.27	0.58	1.3	6 : 4.7E+5	0.24
S4 .5	- Herbivore	473	**-0.17**	-0.19 : -0.16	0.51	0.77	5 : 4.8E+6	0.22
S4 .6	Eutherian mammal	952	**-0.14**	-0.15 : -0.12	0.31	0.45	1.9 : 1.5E+8	0.31
S4 .7	- Artiodactyla	149	**-0.15**	-0.18 : -0.12	0.41	0.65	2200 : 1.8E+6	0.1
S4 .8	- Carnivora	182	**-0.13**	-0.16 : -0.11	0.39	0.44	47 : 2.4E+6	0.14
S4 .9	- Cetacea	42	**-0.17**	-0.24 : -0.1	0.39	0.51	3.1E+4 : 1.5E+8	0.24
S4 .10	- Chiroptera	88	**0.15**	0.067 : 0.24	0.13	0.053	4.2 : 1200	0.26
S4 .11	- Eulipotyphla	24	**-0.25**	-0.39 : -0.12	0.40	2.2	1.9 : 1000	0.19
S4 .12	- Primate	159	**-0.18**	-0.21 : -0.15	0.46	0.36	60 : 4.7E+5	0.13
S4 .13	- Rodentia	244	**-0.2**	-0.23 : -0.16	0.31	0.95	5 : 5.5E+4	0.24
S4 .14	Marsupial mammal	124	**-0.22**	-0.26 : -0.18	0.51	1.3	5.3 : 4.8E+4	0.23
S4 .15	- Dasyuromorphia	27	**-0.17**	-0.27 : -0.066	0.32	1.1	5.3 : 6500	0.21
S4 .16	- Diprotodontia	58	**-0.17**	-0.23 : -0.11	0.38	0.83	9 : 4.8E+4	0.18
S4 .17	Protist	42	**-0.15**	-0.29 : -0.019	0.12	1	3.0E-14 : 3.3E-7	0.69
S4 .18	Plant	335	**-0.29**	-0.31 : -0.27	0.71	0.78	0.0029 : 4.4E+7	0.53
S4 .19	- Alismatales	100	**-0.46**	-0.6 : -0.32	0.30	0.57	0.028 : 10	0.41
S4 .20	- Ericales	34	**-0.41**	-0.51 : -0.3	0.66	3.8	0.6 : 5.6E+6	0.5
S4 .21	- Malpighiales	28	**-0.39**	-0.47 : -0.31	0.80	1.3	1.5 : 9.2E+6	0.47
S4 .22	Ectotherm animal	1092	**-0.34**	-0.36 : -0.33	0.76	1.8	5.0E-8 : 1.9E+7	0.52
S4 .23	Invertebrate	220	**-0.28**	-0.3 : -0.26	0.78	1.6	5.0E-8 : 3500	0.46
S4 .24	- Euphausiacea	15	**-0.36**	-0.69 : -0.035	0.31	0.71	5.0E-5 : 0.079	0.5
S4 .25	- Veneroida	28	**-0.072**	-0.21 : 0.069	0.04	0.99	0.0095 : 660	0.36
S4 .26	Vertebrate	872	**-0.4**	-0.42 : -0.39	0.72	2.8	1.7E-4 : 1.9E+7	0.51
S4 .27	- Acipenseriformes	16	**-0.25**	-0.39 : -0.11	0.53	0.31	28 : 1.1E+6	0.26
S4 .28	- Clupeiformes	77	**-0.44**	-0.47 : -0.41	0.91	3.4	3.7E-4 : 3000	0.33
S4 .29	- Cypriniformes	36	**-0.2**	-0.31 : -0.094	0.30	0.38	8.2 : 2.5E+4	0.23
S4 .30	- Gadiformes	59	**-0.38**	-0.44 : -0.32	0.74	3.4	9.0E-4 : 5.3E+4	0.41
S4 .31	- Perciformes	266	**-0.43**	-0.46 : -0.39	0.70	4.9	1.7E-4 : 3.8E+5	0.52
S4 .32	- Pleuronectiformes	80	**-0.41**	-0.45 : -0.37	0.86	3.6	2.0E-4 : 2.0E+5	0.43
S4 .33	- Salmoniformes	35	**-0.37**	-0.54 : -0.2	0.36	2.4	27 : 3.8E+4	0.41
S4 .34	- Scorpaeniformes	91	**-0.42**	-0.5 : -0.33	0.49	1.4	0.013 : 3.8E+4	0.5
S4 .35	Bird	1250	**-0.15**	-0.17 : -0.14	0.19	0.27	2.7 : 1.1E+5	0.27
S4 .36	Passerine	479	**-0.18**	-0.25 : -0.12	0.06	0.29	5.3 : 1100	0.28
S4 .37	Non passerine	771	**-0.15**	-0.18 : -0.12	0.14	0.27	2.7 : 1.1E+5	0.27
S4 .38	- Gruiformes	18	**-0.34**	-0.53 : -0.14	0.46	1.3	34 : 8700	0.26
S4 .39	- Procellariiformes	45	**-0.28**	-0.42 : -0.13	0.25	0.45	25 : 8600	0.34

Table S5. H1 - Population metabolism (watts/m^2) to body mass (g) regressions.

x-ref:	Fig. 2 E	*n*	*k*	95% CI (*k*)	R^2	*c*	Range mass: g	*Sy·x*	*p*-value
S5 .1	All species	3051	**-0.012**	-0.025 : 0.00062	0.00	7.3E-4	6.6E-14 : 3.2E+6	1.8	0.062
S5 .2	Eukaryotes	2880	**0.014**	-0.0021 : 0.03	0.00	6.7E-4	9.3E-14 : 3.2E+6	1.8	0.09
S5 .3	Mammals	608	**-0.054**	-0.1 : -0.0062	0.01	1.8E-4	3.8 : 2.8E+6	0.8	0.027
S5 .4	Carnivore	90	**-0.27**	-0.36 : -0.17	0.27	1.2E-4	3.8 : 3.9E+5	0.61	1.8E-7
S5 .5	Omnivore	180	**-0.03**	-0.15 : 0.089	0.00	1.6E-4	6.4 : 1.3E+5	0.78	0.62
S5 .6	Herbivore	338	**-0.02**	-0.07 : 0.03	0.00	2.3E-4	5 : 2.8E+6	0.68	0.43
S5 .7	Eutherian mammal	578	**-0.068**	-0.12 : -0.02	0.01	2.0E-4	3.8 : 2.8E+6	0.8	0.006
S5 .8	Marsupial mammal	30	**0.42**	0.2 : 0.65	0.35	4.7E-6	15 : 4.1E+4	0.59	6.2E-4
S5 .9	Protist	301	**0.088**	-0.0072 : 0.18	0.01	0.018	9.3E-14 : 1.3E-7	0.87	0.07
S5 .10	Plant	412	**0.094**	0.08 : 0.11	0.30	0.58	6.0E-5 : 3.2E+6	0.33	7.2E-34
S5 .11	Ectotherm animal	957	**0.032**	0.0024 : 0.061	0.00	9.0E-4	2.3E-8 : 1.2E+4	1.2	0.034
S5 .12	Invertebrate	739	**0.2**	0.16 : 0.24	0.11	0.0044	2.3E-8 : 630	1.2	3.4E-20
S5 .13	Vertebrate	218	**-0.15**	-0.29 : -0.0087	0.02	5.7E-4	0.033 : 1.2E+4	1.2	0.038
S5 .14	Bird	602	**0.18**	0.1 : 0.26	0.03	3.9E-6	2.9 : 1.1E+5	0.64	4.6E-6
S5 .15	Bacteria	171	**0.47**	0.41 : 0.52	0.65	1400	6.6E-14 : 3.9E-11	0.26	2.6E-40

Table S6. H2 - Lifetime growth (unitless) to body mass (g) regressions.

x-ref:	Fig. 2 F	*n*	*k*	95% CI (*k*)	R^2	*c*	Range mass: g	*Sy·x*	*p*-value
S6 .1	Eukaryotes	3798	**0.014**	0.0077 : 0.021	0.00	3.3	3.0E-14 : 1.5E+8	0.51	2.1E-5
S6 .2	Mammals	1079	**-0.1**	-0.11 : -0.09	0.21	8.1	1.9 : 1.5E+8	0.28	2.4E-57
S6 .3	Carnivore	365	**-0.091**	-0.11 : -0.072	0.21	8.7	1.9 : 1.5E+8	0.31	5.2E-20
S6 .4	Omnivore	241	**-0.2**	-0.23 : -0.16	0.35	13	6 : 4.7E+5	0.25	9.4E-24
S6 .5	Herbivore	473	**-0.095**	-0.11 : -0.078	0.20	7.4	5 : 4.8E+6	0.25	1.4E-24
S6 .6	Eutherian mammal	952	**-0.11**	-0.12 : -0.097	0.26	8.8	1.9 : 1.5E+8	0.27	2.5E-63
S6 .7	Marsupial mammal	124	**-0.032**	-0.086 : 0.022	0.01	3.8	5.3 : 4.8E+4	0.31	0.25
S6 .8	Protist	42	**0.024**	-0.11 : 0.16	0.00	45	3.0E-14 : 3.3E-7	0.69	0.72
S6 .9	Plant	335	**-0.018**	-0.038 : 0.0018	0.01	2.7	0.0029 : 4.4E+7	0.53	0.074
S6 .10	Ectotherm animal	1092	**0.15**	0.13 : 0.16	0.32	1.8	5.0E-8 : 1.9E+7	0.57	7.4E-95
S6 .11	Invertebrate	220	**0.1**	0.081 : 0.13	0.28	3.3	5.0E-8 : 3500	0.5	3.4E-17
S6 .12	Vertebrate	872	**0.24**	0.22 : 0.26	0.47	0.82	1.7E-4 : 1.9E+7	0.52	7.1E-121
S6 .13	Bird	1250	**-0.13**	-0.15 : -0.11	0.08	7.2	2.7 : 1.1E+5	0.37	8.6E-25
S6 .14	Passerine	479	**-0.021**	-0.097 : 0.054	0.00	5.1	5.3 : 1100	0.33	0.58
S6 .15	Non passerine	771	**-0.15**	-0.19 : -0.11	0.07	8	2.7 : 1.1E+5	0.39	2.2E-13

Table S7. H3 - Growth efficiency (g/yr/watt) to body mass (g) regressions.

x-ref:	Fig. 2 G	*n*	*k*	95% CI (*k*)	R^2	*c*	Range mass: g	*Sy·x*	*p*-value
S7 .1	All species	2729	**-0.19**	-0.2 : -0.18	0.43	610	4.0E-14 : 1.4E+8	0.75	0.0E+0
S7 .2	Eukaryotes	2692	**-0.17**	-0.18 : -0.17	0.33	550	2.0E-11 : 1.4E+8	0.74	1.0E-239
S7 .3	Mammals	982	**0.0094**	-0.0048 : 0.024	0.00	250	2.1 : 1.4E+8	0.35	0.19
S7 .4	Carnivore	332	**0.053**	0.034 : 0.072	0.08	170	2.1 : 1.4E+8	0.32	9.6E-8
S7 .5	Omnivore	221	**-0.16**	-0.21 : -0.11	0.15	660	6 : 1.4E+5	0.37	2.9E-9
S7 .6	Herbivore	429	**-0.014**	-0.036 : 0.0066	0.00	330	8 : 4.8E+6	0.31	0.18
S7 .7	Eutherian mammal	878	**0.012**	-0.0027 : 0.027	0.00	240	2.1 : 1.4E+8	0.35	0.11
S7 .8	Marsupial mammal	104	**0.041**	-0.0052 : 0.086	0.03	310	6.1 : 5.5E+4	0.26	0.082
S7 .9	Protist	124	**-0.037**	-0.087 : 0.013	0.02	6.3E+4	2.0E-11 : 1.8E-6	0.36	0.15
S7 .10	Plant	132	**-0.087**	-0.15 : -0.026	0.06	1600	1.7 : 3.2E+6	0.34	0.0055
S7 .11	Ectotherm animal	306	**0.002**	-0.02 : 0.024	0.00	3800	2.2E-7 : 1.9E+7	0.64	0.86
S7 .12	Invertebrate	187	**0.0065**	-0.069 : 0.082	0.00	3900	2.2E-7 : 5.8	0.72	0.86
S7 .13	Vertebrate	119	**0.014**	-0.047 : 0.075	0.00	3400	0.54 : 1.9E+7	0.49	0.66
S7 .14	Bird	1148	**0.043**	0.02 : 0.066	0.01	48	3.1 : 1.1E+5	0.34	2.8E-4
S7 .15	Passerine	461	**0.034**	-0.026 : 0.094	0.00	48	5.2 : 1000	0.26	0.27
S7 .16	Non passerine	687	**0.01**	-0.031 : 0.051	0.00	61	3.1 : 1.1E+5	0.39	0.63
S7 .17	Bacteria	37	**0.077**	-0.37 : 0.52	0.00	6.8E+6	4.0E-14 : 1.2E-11	0.7	0.72

Table S8. H4 - Lifetime metabolism (watts/g/yr) to body mass (g) regressions.

x-ref:	Fig. 2 H	*n*	*k*	95% CI (*k*)	R^2	*c*	Range mass: g	*Sy·x*	*p*-value
S8 .1	Eukaryotes	3816	**0.22**	0.21 : 0.23	0.35	0.0013	6.0E-12 : 4.7E+6	0.77	0.0E+0
S8 .2	Mammals	698	**-0.13**	-0.16 : -0.11	0.19	0.035	2.2 : 3.7E+6	0.32	1.7E-33
S8 .3	Carnivore	236	**-0.17**	-0.21 : -0.13	0.23	0.057	2.2 : 3.2E+6	0.4	3.8E-15
S8 .4	Omnivore	200	**-0.11**	-0.15 : -0.063	0.10	0.024	7.4 : 6.0E+4	0.26	4.0E-6
S8 .5	Herbivore	262	**-0.087**	-0.11 : -0.065	0.19	0.024	7.3 : 3.7E+6	0.2	1.7E-13
S8 .6	Eutherian mammal	613	**-0.13**	-0.15 : -0.11	0.19	0.038	2.2 : 3.7E+6	0.31	3.4E-30
S8 .7	Marsupial mammal	81	**-0.071**	-0.12 : -0.022	0.09	0.012	7.1 : 4.5E+4	0.22	0.0054
S8 .8	Protist	95	**0.039**	-0.017 : 0.096	0.02	4.5E-4	6.0E-12 : 2.2E-4	0.49	0.17
S8 .9	Plant	337	**0.13**	0.11 : 0.14	0.43	8.8E-4	0.0093 : 4.7E+6	0.28	2.4E-43
S8 .10	Ectotherm animal	2314	**0.12**	0.11 : 0.13	0.18	4.8E-4	1.6E-7 : 1.6E+4	0.49	3.1E-101
S8 .11	Invertebrate	1719	**0.088**	0.073 : 0.1	0.08	4.1E-4	1.6E-7 : 1.2E+4	0.44	1.2E-31
S8 .12	Vertebrate	595	**0.22**	0.17 : 0.27	0.13	3.7E-4	0.02 : 1.6E+4	0.61	5.3E-19
S8 .13	Bird	372	**-0.2**	-0.23 : -0.17	0.27	0.16	2.9 : 1.0E+5	0.29	6.5E-27
S8 .14	Passerine	181	**-0.1**	-0.2 : -0.0091	0.03	0.14	5.2 : 1200	0.28	0.032
S8 .15	Non passerine	191	**-0.13**	-0.18 : -0.085	0.14	0.098	2.9 : 1.0E+5	0.27	9.9E-8

References

1. Peters RH (1983) *The ecological implications of body size* (Cambridge University Press, Cambridge). 1st Ed.
2. Brown JH, Gillooly JF, Allen AP, Savage VM, West GB (2004) Toward a metabolic theory of ecology. *Ecology* 85(7):1771–1789.
3. West GB, Brown JH, Enquist BJ (1997) A general model for the origin of allometric scaling laws in biology. *Science* 276(5309):122–126.
4. Makarieva AM, Gorshkov VG, Li B (2005) Energetics of the smallest: do bacteria breathe at the same rate as whales? *Proceedings of the Royal Society B: Biological Sciences* 272(1577):2219–2224.
5. Makarieva AM, et al. (2008) Mean mass-specific metabolic rates are strikingly similar across life's major domains: Evidence for life's metabolic optimum. *Proceedings of the National Academy of Sciences* 105(44):16994–16999.
6. White CR, Phillips NF, Seymour RS (2006) The scaling and temperature dependence of vertebrate metabolism. *Biology Letters* 2(1):125–127.
7. Savage VM, et al. (2004) The predominance of quarter-power scaling in biology. *Functional Ecology* 18(2):257–282.
8. Glazier DS (2005) Beyond the '3/4-power law': variation in the intra-and interspecific scaling of metabolic rate in animals. *Biological Reviews* 80(4):611–662.
9. Glazier DS (2015) Is metabolic rate a universal 'pacemaker' for biological processes? *Biol Rev* 90(2):377–407.
10. Damuth J (1987) Interspecific allometry of population density in mammals and other animals: The independence of body mass and population energy-use. *Biological Journal of the Linnean Society* 31(3):193–246.
11. Belgrano A, Allen AP, Enquist BJ, Gillooly JF (2002) Allometric scaling of maximum population density: a common rule for marine phytoplankton and terrestrial plants. *Ecology letters* 5(5):611–613.
12. Nee S, Read AF, Greenwood JJ, Harvey PH (1991) The relationship between abundance and body size in British birds. *Nature* 351(6324):312–313.
13. Hechinger RF, Lafferty KD, Dobson AP, Brown JH, Kuris AM (2011) A common scaling rule for abundance, energetics, and production of parasitic and free-living species. *Science* 333(6041):445–448.
14. Case TJ (1978) On the evolution and adaptive significance of postnatal growth rates in the terrestrial vertebrates. *Quarterly Review of Biology* 53:243–282.
15. Ernest SKM, et al. (2003) Thermodynamic and metabolic effects on the scaling of production and population energy use. *Ecology Letters* 6(11):990–995.
16. Hatton IA, et al. (2015) The predator-prey power law: Biomass scaling across terrestrial and aquatic biomes. *Science* 349(6252):aac6284.
17. Brown JH, Hall CA, Sibly RM (2018) Equal fitness paradigm explained by a trade-off between generation time and energy production rate. *Nature ecology & evolution*:1.
18. West GB, Brown JH, Enquist BJ (2001) A general model for ontogenetic growth. *Nature* 413(6856):628–631.
19. Tacutu R, et al. (2012) Human Ageing Genomic Resources: integrated databases and tools for the biology and genetics of ageing. *Nucleic acids research* 41(D1):D1027–D1033.

20. McCoy MW, Gillooly JF (2008) Predicting natural mortality rates of plants and animals. *Ecology letters* 11(7):710–716.
21. Kleiber M (1932) Body size and metabolism. *Hilgardia* 6:315–353.
22. Glazier DS, et al. (2011) Ecological effects on metabolic scaling: amphipod responses to fish predators in freshwater springs. *Ecological Monographs* 81(4):599–618.
23. Riisgard HU (1998) No foundation of a'3/4 power scaling law'for respiration in biology. *Ecology Letters* 1:71–73.
24. Banavar JR, et al. (2010) A general basis for quarter-power scaling in animals. *Proceedings of the National Academy of Sciences* 107(36):15816–15820.
25. Kooijman SALM (2000) *Dynamic energy and mass budgets in biological systems* (Cambridge University Press).
26. Hou C, et al. (2008) Energy uptake and allocation during ontogeny. *Science* 322(5902):736–739.
27. Wieser W (1994) Cost of growth in cells and organisms: general rules and comparative aspects. *Biological Reviews* 69(1):1–33.
28. Clarke A (2019) Energy flow in growth and production. *Trends in ecology & evolution*.
29. Speakman JR (2005) Body size, energy metabolism and lifespan. *Journal of Experimental Biology* 208(9):1717–1730.
30. Czarnoleski M, et al. (2008) Scaling of metabolism in Helix aspersa snails: changes through ontogeny and response to selection for increased size. *Journal of Experimental Biology* 211(3):391–400.
31. Ricklefs RE (2003) Is rate of ontogenetic growth constrained by resource supply or tissue growth potential? A comment on West et al.'s model. *Functional Ecology* 17(3):384–393.
32. Parry GD (1983) The influence of the cost of growth on ectotherm metabolism. *Journal of Theoretical Biology* 101(3):453–477.
33. Charnov EL, Warne R, Moses M (2007) Lifetime reproductive effort. *The American Naturalist* 170(6):E129–E142.
34. Bertalanffy LV (1957) Quantitative laws in metabolism and growth. *The Quarterly Review of Biology* 32(3):217–231.
35. Weibel ER, Bacigalupe LD, Schmitt B, Hoppeler H (2004) Allometric scaling of maximal metabolic rate in mammals: muscle aerobic capacity as determinant factor. *Respiratory physiology & neurobiology* 140(2):115–132.
36. Geiser F (2004) Metabolic rate and body temperature reduction during hibernation and daily torpor. *Annu Rev Physiol* 66:239–274.
37. Vander Heiden MG, Cantley LC, Thompson CB (2009) Understanding the Warburg effect: the metabolic requirements of cell proliferation. *science* 324(5930):1029–1033.
38. Gaillard J-M, et al. (1989) An analysis of demographic tactics in birds and mammals. *Oikos*:59–76.
39. Reich PB, Tjoelker MG, Machado JL, Oleksyn J (2006) Universal scaling of respiratory metabolism, size and nitrogen in plants. *Nature* 439(7075):457–461.
40. Mori S, et al. (2010) Mixed-power scaling of whole-plant respiration from seedlings to giant trees. *PNAS* 107(4):1447–1451.
41. Ehnes RB, Rall DC, Brose U (2011) Phylogenetic grouping, curvature and metabolic scaling in terrestrial invertebrates. *Ecology Letters* 14(10):993–1000.
42. McNab BK (2008) An analysis of the factors that influence the level and scaling of mammalian BMR. *Comp Biochem Physiol, Part A Mol Integr Physiol* 151(1):5–28.

43. Li WKW (2002) Macroecological patterns of phytoplankton in the northwestern North Atlantic Ocean. *Nature* 419(6903):154–157.
44. Peters RH, Wassenberg K (1983) The effect of body size on animal abundance. *Oecologia* 60(1):89–96.
45. Currie DJ, Fritz JT (1993) Global patterns of animal abundance and species energy use. *Oikos*:56–68.
46. Cyr H, Peters RH, Downing JA (1997) Population density and community size structure: comparison of aquatic and terrestrial systems. *Oikos*:139–149.
47. Niklas KJ, Enquist BJ (2004) Biomass allocation and growth data of seeded plants. Data set. Available online from Oak Ridge National Laboratory Distributed Active Archive Center, Oak Ridge, TN, USA [http://www. daac. ornl. gov].
48. Juanes F (1986) Population density and body size in birds. *The American Naturalist* 128(6):921–929.
49. Marquet PA, Navarrete SA, Castilla JC (1995) Body size, population density, and the energetic equivalence rule. *Journal of Animal Ecology*:325–332.
50. White EP, Ernest SK, Kerkhoff AJ, Enquist BJ (2007) Relationships between body size and abundance in ecology. *Trends in Ecology & Evolution* 22(6):323–330.
51. Fagan WF, Lynch HJ, Noon BR (2010) Pitfalls and challenges of estimating population growth rate from empirical data: consequences for allometric scaling relations. *Oikos* 119(3):455–464.
52. Pereira HM, Daily GC (2006) Modeling biodiversity dynamics in the countryside landscapes. *Ecology* 87(8):1877–1885.
53. Duncan RP, Forsyth DM, Hone J (2007) Testing the metabolic theory of ecology: allometric scaling exponents in mammals. *Ecology* 88(2):324–333.
54. Rose JM, Caron DA (2007) Does low temperature constrain the growth rates of heterotrophic protists? Evidence and implications for algal blooms in cold waters. *Limnology and Oceanography* 52(2):886–895.
55. Cannell MGR (1982) *World Forest Biomass and Primary Production Data* (Academic Press, London, UK).
56. Huston MA, Wolverton S (2009) The global distribution of net primary production: resolving the paradox. *Ecological Monographs* 79(3):343–377.
57. DeLong JP, Okie JG, Moses ME, Sibly RM, Brown JH (2010) Shifts in metabolic scaling, production, and efficiency across major evolutionary transitions of life. *Proceedings of the National Academy of Sciences* 107(29):12941–12945.
58. Sibly RM, et al. (2012) Energetics, lifestyle, and reproduction in birds. *Proceedings of the National Academy of Sciences* 109(27):10937–10941.
59. Ricklefs RE (2010) Embryo growth rates in birds and mammals. *Functional Ecology* 24(3):588–596.
60. Barneche DR, Robertson DR, White CR, Marshall DJ (2018) Fish reproductive-energy output increases disproportionately with body size. *Science* 360(6389):642–645.
61. McGurk MD (1986) Natural mortality of marine pelagic fish eggs and larvae: role of spatial patchiness. *Marine ecology progress series*:227–242.
62. Carey JR, Judge DS (2000) Longevity records: life spans of mammals, birds, reptiles, amphibians and fish. *Odense Monographs on Population Aging (ed B Jeaune and JW Vaupel) Odense: Odense University Press*.

63. Bozinovic F, Rosenmann M (1989) Maximum metabolic rate of rodents: physiological and ecological consequences on distributional limits. *Functional Ecology*:173–181.
64. Geiser F (1988) Reduction of metabolism during hibernation and daily torpor in mammals and birds: temperature effect or physiological inhibition? *Journal of Comparative Physiology B* 158(1):25–37.
65. Sibly RM, Brown JH, Kodric-Brown A (2012) *Metabolic ecology: a scaling approach* (Wiley-Blackwell).
66. Gillooly JF, Brown JH, West GB, Savage VM, Charnov EL (2001) Effects of size and temperature on metabolic rate. *Science* 293(5538):2248–2251.
67. Heldmaier G (2011) Life on low flame in hibernation. *Science* 331(6019):866–867.
68. Hamilton MJ, Davidson AD, Sibly RM, Brown JH (2011) Universal scaling of production rates across mammalian lineages. *Proc R Soc B* 278(1705):560–566.
69. Sibly RM, Brown JH (2007) Effects of body size and lifestyle on evolution of mammal life histories. *Proceedings of the National Academy of Sciences* 104(45):17707–17712.
70. Lotka AJ (1925) *Elements of physical biology* (Williams & Wilkins Company).
71. Ricklefs RE (2006) Embryo development and ageing in birds and mammals. *Proceedings of the Royal Society of London B: Biological Sciences* 273(1597):2077–2082.
72. Pearl R (1928) *The rate of living* (University Press London).
73. Sohal RS, Weindruch R (1996) Oxidative stress, caloric restriction, and aging. *Science* 273(5271):59–63.
74. Speakman JR, et al. (2015) Oxidative stress and life histories: unresolved issues and current needs. *Ecology and Evolution* 5(24):5745–5757.
75. McNab BK (1963) Bioenergetics and the determination of home range size. *The American Naturalist* 97(894):133–140.
76. Kelt DA, Van Vuren DH (2001) The ecology and macroecology of mammalian home range area. *The American Naturalist* 157(6):637–645.
77. Makarieva AM, Gorshkov VG, Li B-L (2005) Why do population density and inverse home range scale differently with body size?: Implications for ecosystem stability. *Ecological Complexity* 2(3):259–271.
78. Harestad AS, Bunnel FL (1979) Home Range and Body Weight–A Reevaluation. *Ecology* 60(2):389–402.
79. Mace GM, Harvey PH (1983) Energetic constraints on home-range size. *The American Naturalist* 121(1):120–132.
80. Lindstedt SL, Miller BJ, Buskirk SW (1986) Home range, time, and body size in mammals. *Ecology* 67(2):413–418.
81. Haskell JP, Ritchie ME, Olff H (2002) Fractal geometry predicts varying body size scaling relationships for mammal and bird home ranges. *Nature* 418(6897):527.
82. Kelt DA, Van Vuren D (1999) Energetic constraints and the relationship between body size and home range area in mammals. *Ecology* 80(1):337–340.
83. Ofstad EG, Herfindal I, Solberg EJ, S\a ether B-E (2016) Home ranges, habitat and body mass: simple correlates of home range size in ungulates. *Proc R Soc B* 283(1845):20161234.
84. Jetz W, Carbone C, Fulford J, Brown JH (2004) The scaling of animal space use. *Science* 306(5694):266–268.
85. Jones KE, et al. (2009) PanTHERIA: a species-level database of life history, ecology, and geography of extant and recently extinct mammals. *Ecology* 90(9):2648–2648.

86. Sheldon RW, Prakash A, Sutcliffe WH (1972) The size distribution of particles in the ocean. *Limnology and oceanography* 17(3):327–340.
87. Sprules WG, Barth LE (2015) Surfing the biomass size spectrum: some remarks on history, theory, and application. *Canadian Journal of Fisheries and Aquatic Sciences* 73(4):477–495.
88. Parsons TR (1969) The use of particle size spectra in determining the structure of a plankton community. *Journal of the Oceanographical Society of Japan* 25(4):172–181.
89. Ahrens MA, Peters RH (1991) Patterns and limitations in limnoplankton size spectra. *Canadian Journal of Fisheries and Aquatic Sciences* 48(10):1967–1978.
90. Sprules WG, Munawar M (1986) Plankton size spectra in relation to ecosystem productivity, size, and perturbation. *Canadian Journal of Fisheries and Aquatic Sciences* 43(9):1789–1794.
91. Kerr SR, Dickie LM, Kerr SR (2001) *The biomass spectrum: a predator-prey theory of aquatic production* (Columbia University Press).
92. Trebilco R, Baum JK, Salomon AK, Dulvy NK (2013) Ecosystem ecology: size-based constraints on the pyramids of life. *Trends in ecology & evolution* 28(7):423–431.
93. Blanchard JL, Heneghan RF, Everett JD, Trebilco R, Richardson AJ (2017) From bacteria to whales: using functional size spectra to model marine ecosystems. *Trends in ecology & evolution* 32(3):174–186.
94. Zaoli S, Giometto A, Maritan A, Rinaldo A (2017) Covariations in ecological scaling laws fostered by community dynamics. *Proceedings of the National Academy of Sciences* 114(40):10672–10677.
95. Yurista PM, et al. (2014) A new look at the Lake Superior biomass size spectrum. *Canadian Journal of Fisheries and Aquatic Sciences* 71(9):1324–1333.
96. Reuman DC, Gislason H, Barnes C, Mélin F, Jennings S (2014) The marine diversity spectrum. *Journal of Animal Ecology* 83(4):963–979.
97. Taylor LR (1961) Aggregation, Variance and the Mean. *Nature* 189(4766):732–735.
98. Cohen JE, Xu M, Schuster WS (2013) Stochastic multiplicative population growth predicts and interprets Taylor's power law of fluctuation scaling. *Proceedings of the Royal Society B: Biological Sciences* 280(1757):2012–2955.
99. Taylor LR, Woiwod IP, Perry JN (1978) The density-dependence of spatial behaviour and the rarity of randomness. *The Journal of Animal Ecology*:383–406.
100. Xiao X, Locey KJ, White EP (2014) A process-independent explanation for the general form of Taylor's Law. *arXiv:14107283 [q-bio]*. Available at: http://arxiv.org/abs/1410.7283 [Accessed April 7, 2015].
101. Cohen JE, Poulin R, Lagrue C (2017) Linking parasite populations in hosts to parasite populations in space through Taylor's law and the negative binomial distribution. *Proceedings of the National Academy of Sciences* 114(1):E47–E56.
102. McGill BJ, et al. (2007) Species abundance distributions: moving beyond single prediction theories to integration within an ecological framework. *Ecology letters* 10(10):995–1015.
103. Shoemaker WR, Locey KJ, Lennon JT (2017) A macroecological theory of microbial biodiversity. *Nature ecology & evolution* 1(5):0107.
104. Fisher RA, Corbet AS, Williams CB (1943) The relation between the number of species and the number of individuals in a random sample of an animal population. *The Journal of Animal Ecology*:42–58.
105. Preston FW (1948) The commonness, and rarity, of species. *Ecology* 29(3):254–283.

106. Baldridge E, Harris DJ, Xiao X, White EP (2016) An extensive comparison of species-abundance distribution models. *PeerJ* 4:e2823.
107. Ulrich W, Ollik M, Ugland KI (2010) A meta-analysis of species–abundance distributions. *Oikos* 119(7):1149–1155.
108. Lui JC, Baron J (2011) Mechanisms limiting body growth in mammals. *Endocrine reviews* 32(3):422–440.
109. Finkielstain GP, et al. (2009) An extensive genetic program occurring during postnatal growth in multiple tissues. *Endocrinology* 150(4):1791–1800.
110. Hicklin DJ, Ellis LM (2005) Role of the vascular endothelial growth factor pathway in tumor growth and angiogenesis. *Journal of clinical oncology* 23(5):1011–1027.
111. Wullschleger S, Loewith R, Hall MN (2006) TOR signaling in growth and metabolism. *Cell* 124(3):471–484.
112. McKenzie DJ, et al. (2003) Effects of growth hormone transgenesis on metabolic rate, exercise performance and hypoxia tolerance in tilapia hybrids. *Journal of Fish Biology* 63(2):398–409.
113. Killen SS, Atkinson D, Glazier DS (2010) The intraspecific scaling of metabolic rate with body mass in fishes depends on lifestyle and temperature. *Ecology letters* 13(2):184–193.
114. Boersma B, Wit JM (1997) Catch-up Growth. *Endocrine Reviews* 18(5):646–661.
115. Osborne TB, Mendel LB (1916) Acceleration of Growth After Retardation. *Am J Physiol* 40(1):16–20.
116. Kay's SK, Hindmarsh PC (2006) Catch-up growth: an overview. *Pediatric endocrinology reviews: PER* 3(4):365.
117. Criscuolo F, Monaghan P, Nasir L, Metcalfe NB (2008) Early nutrition and phenotypic development:'catch-up'growth leads to elevated metabolic rate in adulthood. *Proceedings of the Royal Society of London B: Biological Sciences* 275(1642):1565–1570.
118. Delemarre EM, Rotteveel J, Delemarre-van de Waal HA (2007) Metabolic implications of GH treatment in small for gestational age. *European Journal of Endocrinology* 157(suppl 1):S47–S50.
119. Cooke PS, Yonemura CU, Russell SM, Nicoll CS (1986) Growth and differentiation of fetal rat intestine transplants: dependence on insulin and growth hormone. *Neonatology* 49(4):211–218.
120. Crickmore MA, Mann RS (2008) The control of size in animals: insights from selector genes. *Bioessays* 30(9):843–853.
121. Humphreys BD, et al. (2008) Intrinsic epithelial cells repair the kidney after injury. *Cell stem cell* 2(3):284–291.
122. Dor Y, Brown J, Martinez OI, Melton DA (2004) Adult pancreatic β-cells are formed by self-duplication rather than stem-cell differentiation. *Nature* 429(6987):41–46.
123. Vogelstein B, Kinzler KW (2004) Cancer genes and the pathways they control. *Nature medicine* 10(8):789–799.
124. Jones RG, Thompson CB (2009) Tumor suppressors and cell metabolism: a recipe for cancer growth. *Genes & development* 23(5):537–548.
125. Kerbel R, Folkman J (2002) Clinical translation of angiogenesis inhibitors. *Nature Reviews Cancer* 2(10):727–739.
126. Agathocleous M, Harris WA (2013) Metabolism in physiological cell proliferation and differentiation. *Trends in cell biology* 23(10):484–492.

127. Brown PS, Giuliano R, Hough G (1974) Pituitary regulation of appetite and growth in the turtles Pseudomys scripta elegans and Chelydra serpentina. *Journal of Experimental Zoology Part A: Ecological Genetics and Physiology* 187(2):205–215.
128. Dickerson GE (1954) Hereditary mechanisms in animal growth. *In B. J. Boell (Ed.) Dynamics of Growth Processes* (Princeton University Press. Princeton), pp 242–276.
129. Tyler A (1942) Developmental processes and energetics. *The Quarterly Review of Biology* 17(3):197–212.
130. Zoeller RT, Tan SW, Tyl RW (2007) General background on the hypothalamic-pituitary-thyroid (HPT) axis. *Critical reviews in toxicology* 37(1–2):11–53.
131. Thompson DW (1942) On growth and form. *On growth and form*.
132. Tilman D, et al. (2004) Does metabolic theory apply to community ecology? It's a matter of scale. *Ecology* 85(7):1797–1799.