

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)

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/ $m²$) 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 $-³/₄$ though with a considerable 3 to 4 orders of magnitude residual variation about the line. Similar abundancemass scaling was found in other taxa (11, 12, 43), but studies considering larger size ranges tend to show near inverse scaling (*k* ≈ −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 metaanalyses (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 $(1/\gamma r)$ 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 Q₁₀ 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 (*WT*) 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 $\kappa = 8.62 \times 10^{-5}$ eV, and estimated as follows (66) ,

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

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

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

Previously published Q10 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. Temperaturecorrections 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

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 lognormally 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 log₁₀transformed data and a normal distribution fit to the histograms shown by the blue line, whose mean μ and standard deviation σ of the log₁₀ 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_q (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, $r_{\text{max}} = \ln(\lambda)$. 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 $\frac{3}{4}$ (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* ≈ −1); mean-variance scaling in population abundance between sites or through time (Taylor's law, often $1.5 \le k \le 2$); 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 $k = \frac{2}{3}$ or $\frac{3}{4}$ (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; (individuals/area)⁻¹) is proportional to home range area (*A*). On average, this gives home range area scaling with mass near $\frac{3}{4}$ across all mammals ($A \sim m^{\frac{3}{4}}$). 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 $\frac{3}{4}$ 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 sizefrequency 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

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 sizespectra 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 $1.5 \leq k \leq 2$. 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 abundancemass data across the size range, and relate the mean and variance of population biomass (*Nm*; $g/m²$) 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 $(N; \text{ind./m}^2)$ 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 ATPgenerating 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 hypothyroidism 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 \degree C, where available, and published Q_{10} values.

Abundance. This worksheet contains all population density data in units of individuals/m² 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*;

R2: 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 $(\frac{https://zenodo.org/record/3145281})$.

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

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

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

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

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
S ₂ .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
S ₂ .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
S ₂ .9 - Carnivora	61	-0.83	$-1: -0.64$	0.57	0.0011	79:3.9E+5	0.58
S ₂ 10 - Eulipotyphla	17	0.27	$-0.2:0.74$	0.09	1.9E-4	3.8:800	0.5
S ₂ .11 - Lagomorpha	21	-0.5	$-0.86: -0.14$	0.31	0.0015	$20:3.2E+4$	0.58
S ₂ 12 - Primate	118	-0.38	$-0.56: -0.21$	0.14	5.3E-4	$60:1.3E+5$	0.6
S ₂ 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
S ₂ .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
S ₂ .17 - Fagales	73	-0.6	$-0.66 : -0.54$	0.85	150	$160:3.2E+6$	0.19
S ₂ .18 - Malpighiales	26	-0.65	$-0.99 : -0.31$	0.39	340	$2700:2.1E+6$	0.61
S ₂ .19 - Pinales	174	-0.62	$-0.67 : -0.57$	0.79	200	$1.7:2.2E+6$	0.26
S ₂ .20 Ectotherm animal	957	-0.76	$-0.79 : -0.74$	0.75	1.3	$2.3E-8:1.2E+4$	1.2
S ₂ .21 Invertebrate	739	-0.61	$-0.65: -0.57$	0.55	5.5	2.3E-8:630	1.1
S ₂ .22 - Calanoida	15	-0.33	$-0.83:0.16$	0.14	1000	$1.2E-5:0.002$	0.61
S ₂ .23 - Cladocera	15	-0.06	$-0.83:0.71$	0.00	2900	4.8E-6: 5.8E-4	0.79
S ₂ .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
- Heterobranchia S ₂ .26	32	-0.37	$-0.75:0.0053$	0.12	1.7	$1.9E-4:5.8$	1.2
S ₂ .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
S ₂ .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
S ₂ 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 S2. Population density (count/m2) to body mass (g) regressions.

Fig. 1 C x-ref:	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
S ₃ .2 Eukaryotes	2692	0.73	0.73:0.74	0.96	2.8	$2.0E-11:1.4E+8$	0.42
S ₃ 3 Mammals	982	0.75	0.74:0.76	0.92	4.3	$2.1:1.4E+8$	0.34
S ₃ 4 Carnivore	332	0.8	0.78:0.82	0.96	2.8	$2.1:1.4E+8$	0.31
S ₃ .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
S ₃ .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	$\overline{7}$	$6:5.5E+4$	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
S ₃ .21 - Fagales	42	0.77	0.66:0.88	0.83	0.74	$2.6E+4:3.2E+6$	0.19
S ₃ .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
- Carcharhiniformes S3.28	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	$\overline{2}$	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	$\overline{\mathbf{c}}$	$3.1:1.1E+5$	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
- Pelecaniformes S3 .35	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 S3. Maximum individual productivity (g/yr) to body mass (g) regressions.

Fig. 1 D x-ref:	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	$\mathbf{1}$	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
- Acipenseriformes S4 .27	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
- Cypriniformes S4 29	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
- Perciformes S4 31	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
- Procellariiformes S4.39	45	-0.28	$-0.42 : -0.13$	0.25	0.45	25:8600	0.34

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

x-ref:	Fig. 2 E	n	k	95% CI (k)	R ²	c	Range mass: q	$S_V: x$	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
S53	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	$6.2E - 4$
.9 S5	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
S ₅ .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 S5. H1 - Population metabolism (watts/m²) to body mass (g) regressions.

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	$S_V: x$	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
S ₆ .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
S ₆ 8	Protist	42	0.024	$-0.11:0.16$	0.00	45	$3.0E-14:3.3E-7$	0.69	0.72
S ₆ .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	$2.2E-13$

x-ref:	Fig. 2 G	n	k	95% CI (k)	R^2	C	Range mass: g	$Sv \cdot x$	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 S7. H3 - Growth efficiency (g/yr/watt) to body mass (g) regressions.

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

x-ref:	Fig. 2 H	$\mathbf n$	k	95% CI (k)	R ²	C	Range mass: g	$Sy \cdot x$	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	$9.9E - 8$

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