Integrative and Comparative Biology

Integrative and Comparative Biology, volume 62, number 5, pp. 1448–1459
https://doi.org/10.1093/icb/icac038

Society for Integrative and Comparative Biology

SYMPOSIUM

Growth and Mortality as Causes of Variation in Metabolic Scaling Among Taxa and Taxonomic Levels

Tommy Norin [1]

DTU Aqua: National Institute of Aquatic Resources, Technical University of Denmark, Kemitorvet, Building 202, 2800 Kgs. Lyngby, Denmark

From the symposium "Causal mechanisms of interspecific metabolic scaling patterns" presented at the annual meeting of the Society for Integrative and Comparative Biology virtual annual meeting, January 3–February 28, 2022.

[1]E-mail: tnor@aqua.dtu.dk

Synopsis Metabolic rate (MR) usually changes (scales) out of proportion to body mass (BM) as $MR = aBM^b$, where a is a normalisation constant and b is the scaling exponent that reflects how steep this change is. This scaling relationship is fundamental to biology, but over a century of research has provided little consensus on the value of b, and why it appears to vary among taxa and taxonomic levels. By analysing published data on fish and taking an individual-based approach to metabolic scaling, I show that variation in growth of fish under naturally restricted food availability can explain variation in within-individual (ontogenetic) b for standard (maintenance) metabolic rate (SMR) of brown trout (*Salmo trutta*), with the fastest growers having the steepest metabolic scaling ($b \approx 1$). Moreover, I show that within-individual b can vary much more widely than previously assumed from work on different individuals or different species, from −1 to 1 for SMR among individual brown trout. The negative scaling of SMR for some individuals was caused by reductions in metabolic rate in a food limited environment, likely to maintain positive growth. This resulted in a mean within-individual b for SMR that was significantly lower than the across-individual ("static") b, a difference that also existed for another species, cunner (*Tautogolabrus adspersus*). Interestingly, the wide variation in ontogenetic b for SMR among individual brown trout did not exist for maximum (active) metabolic rate (MMR) of the same fish, showing that these two key metabolic traits (SMR and MMR) can scale independently of one another. I also show that across-species ("evolutionary") b for SMR of 134 fishes is significantly steeper (b approaching 1) than the mean ontogenetic b for the brown trout and cunner. Based on these interesting findings, I hypothesise that evolutionary and static metabolic scaling can be systematically different from ontogenetic scaling, and that the steeper evolutionary than ontogenetic scaling for fishes arises as a by-product of natural selection for fast-growing individuals with steep metabolic scaling ($b \approx 1$) early in life, where size-selective mortality is high for fishes. I support this by showing that b for SMR tends to increase with natural mortality rates of fish larvae within taxa.

Introduction

Metabolic rate is intimately related to body mass, but when mass increases, metabolic rate usually increases less. This disproportionate scaling relationship between metabolic rate (MR) and body mass (BM) can be described by a simple power function, $MR = aBM^b$, where a is a normalisation constant and b the scaling exponent that reflects how steep the change in metabolic rate with body mass is (the linear slope on logarithmic axes). This metabolic scaling relationship is fundamental to biology and widely applied, but despite more than a century of research on metabolic scaling we still know surprisingly little about why metabolic rate scales out of proportion to body mass, and why there appears to be variation in b among taxa and taxonomic levels (Glazier 2005, 2010, 2014, 2018; White et al. 2006; Capellini et al. 2010; DeLong et al. 2010; White & Kearney 2013, 2014; Harrison 2017; Hatton et al. 2019; Kozłowski et al. 2020). A better knowledge of metabolic scaling is fundamentally important for understanding biological patterns and ecosystem dynamics, as it allows us to predict at what rates individuals

Advance Access publication May 17, 2022

and species of different sizes expend energy and need to consume resources such as food and oxygen from the environment. For example, metabolic scaling is an integral part of influential growth models (von Bertalanffy 1957; West et al. 2001; Hou et al. 2008) and of size-based models predicting animal distributions and responses to climate change (Dillon et al. 2010; Cheung et al. 2013; Deutsch et al. 2015, 2020). The importance of understanding metabolic scaling is clear, yet it remains one of the most debated and controversial topics in biology (Glazier 2005, 2018; White 2010; Harrison 2017, 2018; Kozłowski et al. 2020).

Metabolic rate was initially thought to scale in geometric proportion to the surface area of animals, resulting in $b \approx \frac{2}{3}$ (Rubner 1883), a value of b that has been reiterated more recently for mammals and birds (White & Seymour 2003; White et al. 2006; Roberts et al. 2010), although not necessarily due to geometric constraints on heat loss over the body surface (White & Seymour 2004). Later, however, an empirical value of $b \approx \frac{3}{4}$ was repeatedly found and considered a scaling law in biology ("Kleiber's law") (Kleiber 1932, 1947; Brody 1945; Hemmingsen 1960), with later fractal network theory explaining this $\frac{3}{4}$-power scaling "law" as arising universally due to fixed physical constraints on the transport of resources (e.g., oxygen and nutrients) through branching vessels in the body (West et al. 1997, 1999). While canonical $\frac{3}{4}$-power scaling of metabolic rate is still widely promoted (e.g., Banavar et al. 2010; Sibly et al. 2012; Brown et al. 2018) and applied (e.g., Dillon et al. 2010; Deutsch et al. 2020), there is increasing awareness that b varies among different taxonomic groups (Glazier 2005; White et al. 2006; White et al. 2009; Capellini et al. 2010), among endo- and ectotherms (White et al. 2007), across life's evolutionary transitions (DeLong et al. 2010; Uyeda et al. 2017), with temperature and lifestyle (Killen et al. 2010; Glazier 2010, 2020), among different states of activity within the same animal (resting *vs*. active metabolic rates; Weibel & Hoppeler 2005; White et al. 2007; Glazier 2008, 2009, 2020), and with a suite of other physiological and ecological conditions (Glazier 2010, 2014, 2018; Harrison 2017; Kozłowski et al. 2020).

Variation in growth has also long been proposed as an important driver of variation in metabolic rate and its body-mass-scaling (Epp & Lewis 1980; Riisgaard 1998; Glazier 2005, 2015; Czarnołęski et al. 2008; Hatton et al. 2019), although this idea has been overshadowed by the dominating view in the very influential fractal network theory (West et al. 1997, 1999; Banavar et al. 2010), metabolic theory of ecology (Brown et al. 2004), and ontogenetic growth models (West et al. 2001; Hou et al. 2008), which is that growth adjusts to metabolic rate rather than metabolic rate adjusting to growth. Still, many researchers have embraced the variation that exists in b and are now trying to explain it for what it is, rather than explaining why it is not $\frac{2}{3}$ or $\frac{3}{4}$. Despite this, the causes of variation in b among taxa and taxonomic levels remain debated.

Variation in scaling relationships among taxonomic levels has received more attention in other branches of allometry (Gould 1975; Lande 1979; Cheverud 1982; Pélabon et al. 2013; Voje et al. 2013; Tsuboi et al. 2018). For example, Tsuboi et al. (2018) have shown that the scaling of mammalian and avian brain size with body size across species (termed "evolutionary" scaling) is steeper than scaling across individuals within species (so-called "static" scaling). They interpret this reduction in scaling exponents at lower taxonomic levels as a reduced allometric constraint on brain–body size evolution, which has facilitated the encephalisation of mammals and birds compared to other animal taxa (Tsuboi et al. 2018). Thus, understanding how evolutionary scaling of metabolic rate relate to metabolic scaling at static and ontogenetic levels may also provide important insight into constraints on the (co)evolution of metabolic rate and body mass. However, the evolution of metabolic scaling and its constraints have only been given attention relatively recently (Beaman et al., preprint; Czarnołęski et al. 2008; Glazier et al. 2011, 2020; Uyeda et al. 2017; White et al. 2019; Kozłowski et al. 2020), and scaling of metabolic rate over ontogeny has been investigated almost exclusively for different individuals at different life stages; variation between individuals in true ontogenetic scaling (scaling within an individual as it grows) has received virtually no attention (e.g., Beaman et al., preprint; Maxwell et al. 2003; Norin & Gamperl 2018; Ye et al. 2021), despite natural selection acting on variation between individuals.

Using published data on fish, I here investigate and compare how both the standard (maintenance) metabolic rate (SMR) and the aerobic maximum (active) metabolic rate (MMR) scale with body mass across species (evolutionary scaling), across individuals of the same age within species (static scaling), and within individuals as they grow (ontogenetic scaling). I do this by combining phylogenetically-informed data on the evolutionary scaling of SMR and MMR across 134 and 114 species of ray-finned fish, respectively, with data on static and ontogenetic scaling of metabolic rate from 68 and 33 individuals of two of these species (cunner, *Tautogolabrus adspersus*, and brown trout, *Salmo trutta*, respectively). I also examine if variation in ontogenetic metabolic scaling can be explained by variation in growth rate among individuals, and if ecological factors such as natural mortality rates early in life (where fish need to grow fast to escape size-selective mortality; Sogard 1997) can explain variation in metabolic scaling

later in life (of juveniles or adults for which metabolic scaling has primarily been investigated before).

Materials and methods

Data were analysed using R v. 4.0.2 (R Core Team 2020). Data and R script, including model structures and validations, were uploaded for reviewers and editors at manuscript submission, and are archived in a citable format on figshare: https://doi.org/10.6084/m9.figshare.19619895.

Evolutionary metabolic scaling

The literature-derived dataset used to assess evolutionary (across-species) scaling of SMR and MMR with body mass is an updated version of the dataset from Killen et al. (2016), where criteria for including metabolic rate data are described. The dataset contains SMR and MMR values from 140 and 115 species, respectively, of juvenile or adult ray-finned fish (class Actinopterygii).

I analysed evolutionary metabolic scaling relationships using phylogenetically-informed models. First, I extracted phylogenetic information from The Fish Tree of Life (Rabosky et al. 2018) and matched it to the species in the metabolic rate dataset using the package *fishtree* (Chang et al. 2019). There was no or dichotomous information available for six species, which were removed from the dataset, reducing it to 134 and 114 species for SMR and MMR, respectively. I then analysed these data using phylogenetic generalised least squares (PGLS) models in the package *ape* (Paradis & Schliep 2019). Each PGLS model had $\log_{10}$-transformed whole-animal SMR or MMR as a response variable, while $\log_{10}$-transformed body mass and the temperature at which metabolic rate was measured in the original study were included as fixed effects. Phylogenetic correlation was estimated using Pagel's λ (Freckelton et al. 2002).

For graphical presentation, metabolic rates of each species were adjusted to a common temperature of 15°C by adding residuals from the respective PGLS model for SMR or MMR to the metabolic rates predicted by the models when keeping temperature fixed at 15°C.

Since metabolic rate is most often estimated as oxygen uptake rate, and researchers tend to favour smaller individuals or species that are more easily accommodated in respirometry chambers, it is possible that this potential bias towards smaller-sized animals could affect metabolic scaling relationships. To assess this, I extracted estimated asymptotic and maximum observed lengths for the species in the dataset from FishBase (Froese & Pauly 2019) and converted these to body masses using the length–weight relationships also provided in FishBase. Since asymptotic and maximum observed sizes often differed, I took the mean of these two measures as a single measure of maximum body mass. I also calculated the relative body mass of each species in the dataset by dividing the body mass at which the species was measured for metabolic rate with its estimated maximum body mass. Maximum body mass estimates were only available from FishBase for a subset of the species in the metabolic rate dataset (121 species for SMR, 109 for MMR), so I ran separate PGLS models on this reduced dataset to assess the influence of species-specific maximum and relative body mass on metabolic scaling. These PGLS models were structured as the PGLS models already described above, except that either $\log_{10}$-transformed maximum body mass or $\log_{10}$-transformed relative body mass was included as an additional fixed effect.

Static and ontogenetic metabolic scaling

Data allowing for analyses of both static (across-individual) and ontogenetic (within-individual) scaling of metabolic rate were available for two fishes: cunner (*Tautogolabrus adspersus*) from Norin & Gamperl (2018) and brown trout (*Salmo trutta*) from Norin & Malte (2011). These cunner and brown trout were hatchery-reared fish from wild-caught parents, with individuals from each species kept in the same tank and being from the same respective cohorts and age. All fish were kept and measured at the same temperature (15°C) in the original studies.

Norin & Gamperl (2018) measured the metabolic rates and body masses of the same 68 individual cunner five times over a 10-month period. The full body mass range of the cunner during this period spanned from 0.45 g for the smallest individual at the first measurement to 19.5 g for the largest individual at the fifth measurement, with the mean body mass of the 68 fish spanning a 4.0-fold range, from 1.63 g at the first measurement to 6.46 g at the fifth measurement. The body masses of individuals within each of the five measurements spanned a 10.1-fold range on average across the five trials.

Norin & Malte (2011) measured the metabolic rates and body masses of the same 33 individual brown trout four times over a 15-week period. The full body mass range of individual trout during this period spanned from 20.7 g for the smallest individual at the first measurement to 68.2 g for the largest individual at the fourth measurement, with the mean body mass of the 33 fish spanning a 1.7-fold range, from 32.3 g at the first measurement to 54.0 g at the fourth measurement. The body masses of individuals within each of the four measurements spanned a 1.9-fold range on average across the four trials.

The data for brown trout were not analysed for scaling relationships in the original study, and all data, including those for cunner, were therefore re-analysed here. I analysed ontogenetic (within-individual) and static (across-individual) scaling simultaneously (in the same model) using linear mixed-effects (LME) models in the package *lme4* (Bates et al. 2015) and following the "within-subject centring" method recommended by van de Pol & Wright (2009). Each LME model had $\log_{10}$-transformed whole-animal SMR or MMR as a response variable, $\log_{10}$-transformed body mass as predictor variable (fixed effect), and individual fish ID as a random effect. Following the guidelines of van de Pol & Wright (2009), body mass was included twice in each model to tease apart within- *vs*. across-individual relationships: each individual's mean body mass across all five (cunner) or four (brown trout) trials was subtracted from its body mass at the time of each trial, and these mean-centred values were used as a fixed effect to express within-individual variation; the overall mean body mass of each individual (the same value repeated for each trial) was included as a separate fixed effect to express across-individual variation.

Comparing evolutionary, static, and ontogenetic metabolic scaling

Statistical differences between evolutionary, static, and ontogenetic metabolic scaling exponents (b; model-estimated slopes) were evaluated at the $P = 0.017$ level to account for multiple comparisons. Pairwise comparisons between b for SMR (b_{SMR}) and MMR (b_{MMR}) within each of evolutionary, static, and ontogenetic levels were evaluated at the $P = 0.05$ level. Comparisons between static and ontogenetic b were done directly within each LME model, as in van de Pol & Wright (2009). All other comparisons were done by evaluating if the confidence interval for the difference between estimates of b excluded zero.

Ontogenetic metabolic scaling and growth rates

Relationships between within-individual b and specific growth rates (% daily increase in body mass) from the original studies over the 10-month growth period for cunner (Norin & Gamperl 2018) and 15-week growth period for brown trout (Norin & Malte 2011) were evaluated using Pearson's correlation analyses. The cunner were maintained on an *ad libitum* feeding regime and fed ~2.5% of their mean body mass daily, while the trout were maintained on a restricted feeding regime and fed ~0.6% of their mean body mass daily.

Metabolic scaling and mortality rates

To assess the possible influence of natural selection during early life stages on metabolic scaling later in life, I extracted estimates of natural mortality rates of marine fish larvae from the literature (McGurk 1986; Pepin 1991; Davis et al. 1991; Houde & Zastrow 1993). As only few data existed on larval mortality rates and metabolic scaling of the same species, I performed these analyses at the order level. This was done for orders for which SMR or MMR data of at least six species were available in the multi-species (evolutionary) metabolic rate dataset (99 species for SMR, 83 for MMR). Metabolic scaling exponents for these orders were estimated using PGLS models with $\log_{10}$-transformed whole-animal SMR or MMR as a response variable, and $\log_{10}$-transformed body mass, the temperature at which metabolic rate was measured in the original study, and order as fixed effects, with an interaction between $\log_{10}$-transformed body mass and order.

Larval mortality rates of species from four orders matched the orders with six or more species in the metabolic rate dataset: Gadidae (n = 4 species), *Incertae sedis* in Eupercaria (n = 3 species), Pleuronectiformes (n = 2 species), and Scombriformes (n = 3 species). Larval mortality rates also existed in the literature for one species from the order Sparidae, which were included in the analyses to increase sample size due to Sparidae's close evolutionary proximity to *Incertae sedis* in Eupercaria (Supplementary Material Fig. S1A), despite metabolic rate data being available for only three species within Sparidae. The scaling exponents for SMR and MMR of Sparidae were therefore assumed to be the same as those for *Incertae sedis* in Eupercaria because of these taxa's close evolutionary proximity, which appears to be reasonable based on where the metabolic rates of species from Sparidae fall on the scaling relationships for *Incertae sedis* in Eupercaria (Fig. S1D). Mortality rates of species for which more than one estimate was available in the literature were averaged. Since very few mortality rate estimates existed for freshwater species, only data for marine species were included. Relationships between model-estimated b_{SMR} or b_{MMR} and mean larval mortality rates within orders were assessed using Pearson's correlation analyses.

Results

Comparing evolutionary, static, and ontogenetic metabolic scaling

Evolutionary metabolic scaling across the 134 (SMR) or 114 (MMR) fishes is steeper (higher b) than the

mean ontogenetic scaling of both cunner and brown trout (Fig. 1), although the difference is not significant for MMR of cunner ($P = 0.159$). Static scaling is also steeper than ontogenetic scaling, except for MMR of trout ($P = 0.381$; Fig. 1). Evolutionary scaling is different from (steeper than) static scaling only for SMR of cunner ($P = 0.014$; Fig. 1). Scaling exponents for SMR and MMR are significantly different within both static and ontogenetic levels for both cunner and brown trout (P 0.05), while b_{SMR} tends to be higher than b_{MMR} at the evolutionary level ($P = 0.077$) (Fig. 1).

Effect of species body size on evolutionary metabolic scaling

At the evolutionary level, there are no effects of either maximum body mass or relative body mass (i.e., at what body mass metabolic rate was measured, relative to the species' maximum body mass) on the scaling exponent for either SMR or MMR (PGLS, interactions between measured mass and estimated maximum or relative mass; $0.664 \geq P \geq 0.171$), or the scaling coefficient (intercept) for SMR or MMR (PGLS, effects of estimated maximum or relative mass; $0.104 \geq P \geq 0.095$).

Ontogenetic metabolic scaling and growth rates

Metabolic scaling exponents for SMR of individual brown trout, which were fed a restricted ration of ~0.6% of their body mass daily, correlate positively with the fish's growth rates ($r = 0.701$, P 0.0001; Fig. 2D). There are no significant correlations between b_{SMR} and growth rate for the *ad libitum* fed cunner, or for b_{MMR} and growth rate for either cunner or trout ($r = -0.003$ to 0.168, $P = 0.351$ to 0.983; Fig. 2A-C).

Metabolic scaling and mortality rates at the order level

While there are only little data available on natural larval mortality rates and metabolic scaling of the same taxa, the order-level scaling exponent for SMR tends to increase with the mean mortality rate of larvae from species within those orders ($r = 0.825$, $P = 0.086$; Fig. 3A). There is less indication of a relationship between b_{MMR} and mortality rate (Fig. 3B), although, if anything, this relationship appears to be negative ($r = -0.766$, $P = 0.131$).

Discussion

I find that evolutionary (across-species) metabolic scaling for more than 113 fishes is significantly steeper than ontogenetic (within-individual) scaling for the two fishes for which within-individual data on metabolic scaling were available (cunner and brown trout), in particular for SMR (Fig. 1). Within the two species, I further find that ontogenetic scaling is shallower than static (across-individual) scaling. I also show that variation in ontogenetic scaling of SMR, but not MMR, of the brown trout can be explained by variation in growth rate among individuals (Fig. 2). This strong relationship between metabolic scaling and growth rate existed only for the brown trout that were maintained on a restricted feeding regime, and not for the *ad libitum* fed cunner. While more data on within-individual ontogenetic scaling is needed to confirm if the relationship between metabolic scaling and growth is species-specific, or exists for all species when food availability is naturally restricted, my analyses have revealed that b can vary much more widely than previously assumed from work on different individuals or different species, from −1 to 1 for ontogenetic scaling of SMR (mean $b_{SMR} = 0.27$; Figs 1E and 2D); nearly all theories for metabolic scaling predict that b should fall between $\sim\frac{2}{3}$ and 1 (reviewed by Glazier 2005, 2018). The strong correlation between b_{SMR} and growth rate of individual brown trout indicates that the observed variation is real and biologically meaningful, and that negative scaling of metabolic rate (within individuals) is an overlooked phenomenon. Publication bias could have further accentuated our poor understanding of this phenomenon, because shallowly positive or negative scaling relationships may be more likely to be non-significant and therefore less likely to be published.

A reduction in SMR in response to reduced food availability, as observed here for some trout, is a common observation among fish and other animals (Mueller & Diamond 2001; Fu et al. 2005; Wang et al. 2006; Van Leeuwen et al. 2012; Auer et al. 2016); however, this has previously been thought to increase b, as an inverse relationship between metabolic rate and its scaling exponent is predicted when energy availability is low (*sensu* the "metabolic-level boundaries hypothesis"; Glazier 2005, 2010). While this is found to be true at the across-individual level where it has been studied in the past (reviewed by Glazier 2005), and confirmed here (high static b_{SMR} of brown trout; Fig. 1E–G), my extended focus on within-individual scaling has revealed biological patterns that would have been overlooked with a traditional species-level focus: metabolic scaling can be shallow and even negative at one level of biological organisation (within individuals) while being steep and positive at others [among (the same) individuals and across species; Fig. 1]. Based on these interesting findings, I hypothesise that the steeper evolutionary than ontogenetic scaling observed here for fishes could be a result of natural selection on growth rate; mortality is often very high in the early life stages of fishes

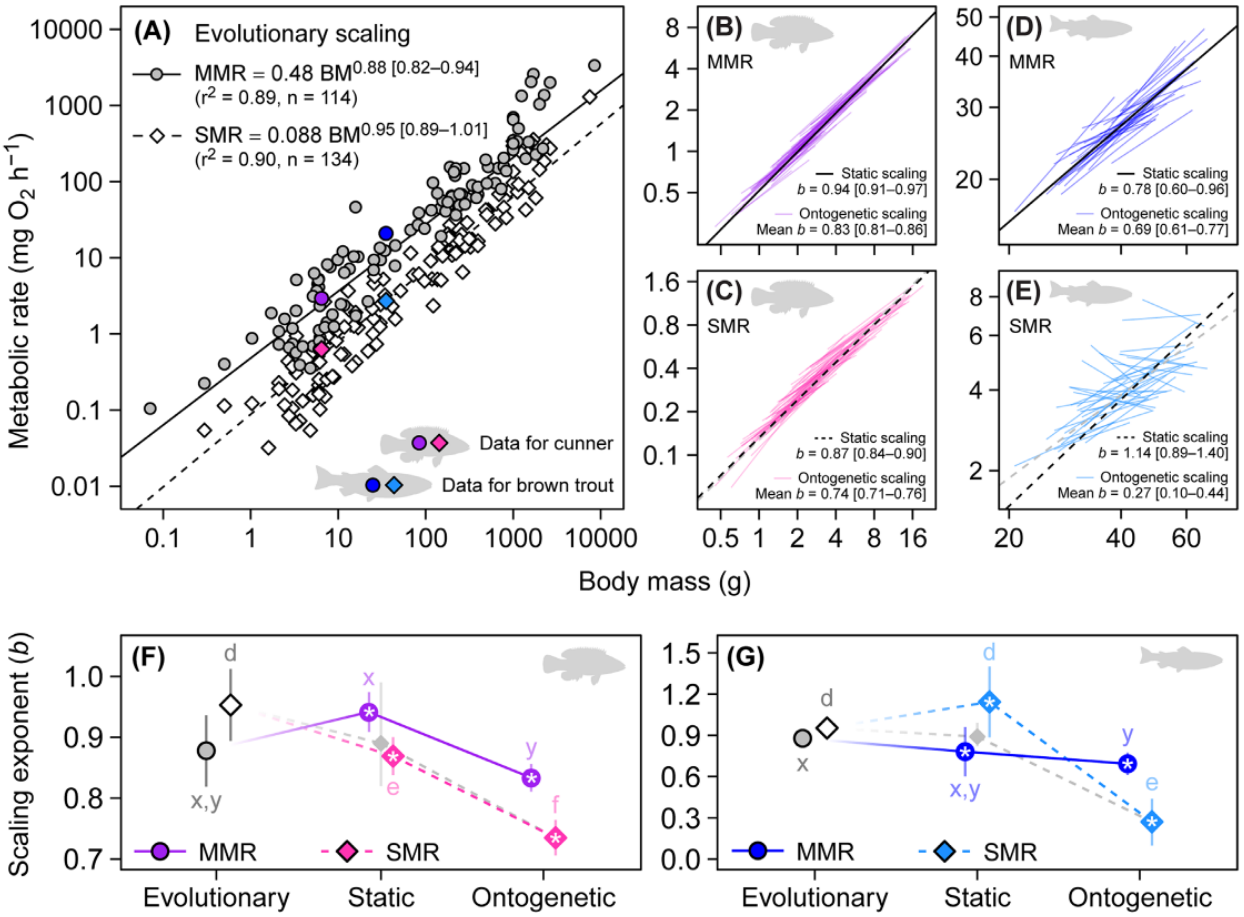

Figure 1. Scaling of metabolic rate with body mass at evolutionary, static, and ontogenetic levels in fishes. Evolutionary scaling (**A**) of metabolic rate with body mass (BM) is shown for standard metabolic rate (SMR) and maximum metabolic rate (MMR) of 134 and 114 species of ray-finned fish, respectively. Each data point represents a species, and the presented data have been adjusted to a temperature of 15°C using residuals from the PGLS regression for SMR ($\log_{10}$ SMR = −1.386 0.953 $\log_{10}$ BM 0.022 T; $r^2 = 0.893$, $\lambda = 0.869$) or MMR ($\log_{10}$ MMR = −0.624 0.878 $\log_{10}$ BM 0.020 T; $r^2 = 0.878$, $\lambda = 0.911$). The coefficients for temperature translate to thermal sensitivities (Q_{10}) of 1.66 for SMR and 1.60 for MMR. Scaling equations shown on the panel are for the temperature-adjusted data. Static and ontogenetic scaling (**B-E**) at 15°C are shown for cunner (*Tautogolabrus adspersus*) (B-C) and brown trout (*Salmo trutta*) (D-E). Mean scaling exponents (b) are noted on panels A-E and compared in **F-G** using the same colour coding. Different lower-case letters in F-G indicate significant differences (P 0.017) between exponents for either SMR or MMR, while pairs of asterisks indicate significant differences between b_{SMR} and b_{MMR} within each taxonomic level (P 0.05). Full LME regressions for panels B-E are: (B) $\log_{10}$ MMR = −0.286 0.833 $\log_{10}$ BM_{Ont} 0.941 $\log_{10}$ BM_{Stat} ($_m r^2 = 0.968$, $_c r^2 = 0.977$); (C) $\log_{10}$ SMR = −0.880 0.735 $\log_{10}$ BM_{Ont} 0.869 $\log_{10}$ BM_{Stat} ($_m r^2 = 0.949$, $_c r^2 = 0.956$); (D) $\log_{10}$ MMR = 0.181 0.693 $\log_{10}$ BM_{Ont} 0.781 $\log_{10}$ BM_{Stat} ($_m r^2 = 0.726$, $_c r^2 = 0.797$); and (E) $\log_{10}$ SMR = −1.260 0.271 $\log_{10}$ BM_{Ont} 1.143 $\log_{10}$ BM_{Stat} ($_m r^2 = 0.528$, $_c r^2 = 0.805$). The coefficients for BM_{Ont} and BM_{Stat} represent the ontogenetic (within-individual) and static (across-individual) scaling exponents, respectively, and $_m r^2$ and $_c r^2$ are marginal and conditional r^2, respectively. The mean within-species b_{SMR} of 0.89 recently reported for 16 fishes (Jerde et al. 2019) is included for comparison with the static b_{SMR} of cunner and brown trout (grey diamonds and grey dashed lines) and supports a general decrease in b_{SMR} from evolutionary across static to ontogenetic levels. Values in square brackets (A-E) and error bars (F-G) are 95% CIs. Data in A-E are presented on logarithmic axes to linearise the relationship between metabolic rate and body mass.

(Houde 1997; Almany & Webster 2006; Hatton et al. 2019), so strong natural selection for fast-growing individuals with steep metabolic scaling ($b_{SMR} \approx 1$; Fig. 2) could be driving the steep evolutionary scaling of SMR (b also ~1; Fig. 1). This has preliminary support from the data on natural mortality rates (Fig. 3), which indicate that b_{SMR} tends to be higher in fish taxa that experience high early-life mortality. But why then, is variation in metabolic scaling maintained within species? A possible explanation is that it is selection happening during the larval or early juvenile stages that shape evolutionary scaling relationships, due to a genetic correlation (pleiotropy) between metabolic rate and growth rate that exists only early in life (discussed further below). The relaxation of such a genetic correlation later in life could allow metabolic rate and body size to diverge due to differential effects of a heterogeneous environment on individual metabolic and growth rates (Norin

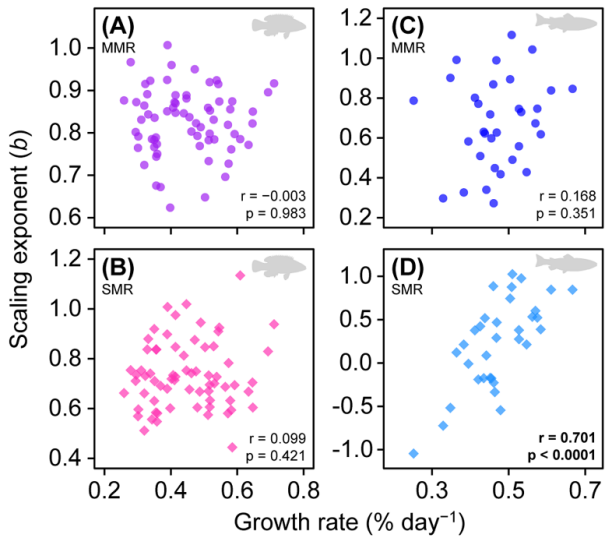

Figure 2. Relationships between metabolic scaling exponents and growth rates of individual fish. The ontogenetic scaling exponents (*b*) for MMR (**A, C**) and SMR (**B, D**) of individual fish (the same individuals as shown in Fig. 1B–E) are unrelated to growth rates (A-C), except for SMR under restricted food intake (D). Cunner (A, B) were fed an *ad libitum* ration of ~2.5% of their body mass daily over the 10-month study (Norin & Gamperl 2018), while brown trout (C, D) were fed a restricted ration of ~0.6% of their body mass daily over the 15-week study (Norin & Malte 2011). Note that 11 out of 33 individuals in panel D exhibit negative scaling whereby SMR is reduced despite the fish growing; this is likely caused by the energetic constraint resulting from the restricted feeding regime, which forced a reduction in SMR in some individuals (negative *b*) to maintain positive growth.

et al. 2016; Pettersen et al. 2020), thereby causing (more) variation in metabolic scaling in the life stages for which evolutionary (across-species) metabolic scaling is most often quantified (older juveniles or adults). In this context, it is important to also bear in mind that metabolic scaling is nearly always estimated for animals kept for different durations in the laboratory, usually in the absence of all of selection, mortality, and constraints on growth, meaning that the variation we observe may not always be representative of that in nature.

While the idea of metabolic scaling patterns being driven by variation in growth fits well with the findings for SMR of brown trout here, which were kept on a naturally realistic (restricted) food regime and were likely competing for food, *b* was unrelated to growth in the *ad libitum* fed cunner. It is possible that the release from an energetic constraint in the cunner (possibly due to relaxed competition for food) removed the relationship between ontogenetic b_{SMR} and growth rate in this species, such that all individuals were able to grow unhindered, with no need to adjust metabolic rate. That is, despite metabolic rate varying ~2-fold among same-sized individuals (Burton et al. 2011; Norin & Malte 2011), the *ad libitum* food regime allowed all individual cunner to cover their metabolic demands, regardless of having a relatively low or high metabolic rate for their size. Since growth of fishes is tightly related to SMR but not MMR (Auer et al. 2015, 2016; Norin et al. 2016), this could also explain why ontogenetic, static, and evolutionary scaling are more similar for MMR than for SMR (Fig. 1), and why evolutionary b_{SMR} tends to be higher than evolutionary b_{MMR} (i.e., the influence of growth on b_{SMR} does not affect b_{MMR}). In turn, the reversal of this pattern at the lowest taxonomic level, evidenced by steeper ontogenetic scaling of MMR than SMR (Fig. 1), could be explained by the fitness advantage of keeping maintenance costs relatively low throughout an individual's life (shallow ontogenetic scaling of SMR) while

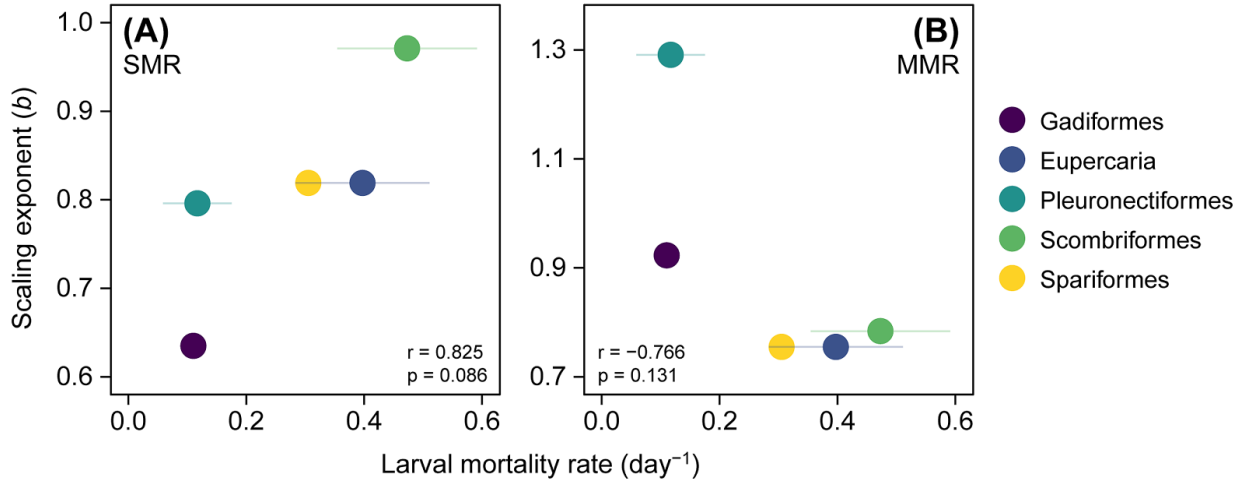

Figure 3. Relationships between metabolic scaling exponents and natural mortality rates of fish larvae. Model-estimated *b* for SMR (**A**) and MMR (**B**) of juvenile or adult fish from different bony fish (teleost) orders and their correlations with the mean (± SE error bars) natural larval mortality rates of different species within the orders, estimated in each species' native marine environment. See Fig. S1 for plots of the metabolic scaling relationships for each order.

maximising scope for aerobic activity (steep ontogenetic scaling of MMR relative to SMR) (Killen et al. 2016).

Given that the majority of fishes, along with most aquatic invertebrates, generally produce many (even millions) offspring that are spawned into the water or onto the substrate and left to fend for themselves, it is possible that the hypothesis of steep evolutionary metabolic scaling being driven by strong natural selection for fast ontogenetic growth out of the vulnerable early-life stage only applies to these aquatic ectotherms. Indeed, mortality rates in aquatic ectotherms scale steeper than for eukaryotes in general, with disproportionately high mortality rates of fish egg and larvae (Hatton et al. 2019). On the other hand, it is also possible that variation among taxa in natural selection pressures on growth systematically shapes evolutionary, static, and ontogenetic metabolic scaling patterns for all animals (and perhaps all organisms), such that evolutionary scaling within a taxonomic group reflects the average ontogenetic scaling of the surviving individuals within that group. For example, the progressively shallower scaling of maintenance metabolic rate (SMR in ectotherms; basal metabolic rate in endotherms) found for reptiles ($b_{SMR} \approx 0.8$) as well as mammals and birds ($b_{SMR} \approx \frac{2}{3}$), relative to fish and amphibians ($b_{SMR} \approx 0.9$–1) (White et al. 2006; Fig. 1), could be caused by a relaxation of the selection pressure on fast early-life growth, resulting in higher offspring survival in these lineages (e.g., due to parental care and relatively large offspring). This fits well with what is known from amphibians, which experience high natural larval (tadpole) mortality (~95%; Melvin & Houlahan 2012) and, like fishes, have steep metabolic scaling across species ($b_{SMR} \approx 0.9$; White et al. 2006). Nonetheless, more data on early-life mortality rates and among-individual variation in both within-individual metabolic scaling and growth (for selection to act upon) are needed to evaluate if size-selective mortality in early life could be a universal driver of (variation in) metabolic scaling.

A less exciting possibility is that the steeper evolutionary than ontogenetic metabolic scaling observed here for fishes is a statistical artefact of ontogenetic scaling being analysed over a much narrower body mass range than evolutionary scaling, which could statistically reduce the slope (scaling exponent), since the covariance between the independent (body mass) and dependent (metabolic rate) variables may decrease relatively more than the variance in the independent variable does when the range in the independent variable decreases (and the slope is calculated as the covariance between the independent and dependent variables, divided by the variance in the independent variable). This could potentially also explain why ontogenetic scaling of the cunner is shallower than static scaling, as the mean body mass range for ontogenetic scaling of the cunner (4.0-fold) is narrower than that for static scaling (10.1-fold). However, this is not the case for the brown trout, where ontogenetic and static scaling were evaluated over similar body mass ranges (on average 1.7-fold for ontogenetic and 1.9-fold for static scaling) but mean ontogenetic scaling of SMR ($b = 0.27$) was still (much) shallower than static scaling ($b = 1.14$). The less pronounced differences between ontogenetic and evolutionary scaling of MMR, compared to that of SMR, for both the cunner and brown trout also argue against a statistical artefact, as scaling of MMR was analysed over the same body mass ranges as for SMR. Moreover, a recent study on Nile tilapia (*Oreochromis niloticus*) has also found shallower ontogenetic (mean $b = 0.714$ [95% CI, 0.695–0.733]) than static ($b = 0.770$ [95% CI, 0.740–0.801]) scaling of resting metabolic rate over a much wider (32-fold) range in ontogenetic body mass (Ye et al. 2021) compared to the cunner and brown trout data analysed here. Thus, the differences in ontogenetic, static, and evolutionary metabolic scaling observed here for fishes could indeed be real biological differences.

That real differences exist for scaling at different taxonomic levels is also in agreement with early arguments that evolutionary brain size allometries arise from static or ontogenetic allometries when there is strong selection on body size (Gould 1975; Lande 1979; Riska & Atchley 1985). Steeper scaling of brain size at higher taxonomic levels has recently been confirmed in mammals and birds and attributed to selection on early growth periods (Tsuboi et al. 2018), with brain size co-evolving with body size likely because genes affecting both brain and body size are expressed simultaneously (under pleiotropic gene control) early in life, whereas genes affecting only body size dominate later in life where selection for fast growth is relaxed (Riska & Atchley 1985). If the same applies to metabolic rate, this provides a genetic mechanism for how the steep evolutionary metabolic scaling observed here can be a by-product of selection for fast ontogenetic growth (to achieve a relatively large body size) in the larval or juvenile stages. The existence of a strong genetic correlation between metabolic rate and body mass has indeed been found recently within three insect species, three bird species, and a mammal (White et al. 2019). If this genetic correlation between metabolic rate and body mass weakens during ontogeny, which is unknown, this allows metabolic rate and body mass to change independently of one another, causing more variable and lower b at lower taxonomic levels for older juveniles or adults for which metabolic scaling has predominantly been investigated. Thus, for organisms in general, it is possible that more or less selection for fast-growing individuals in early life simul-

taneously and indirectly selects for more or less steep metabolic scaling, and causes more or less steep evolutionary scaling, as both growth and body size are heritable traits (Beaman et al., preprint; Mousseau & Roff 1987) while metabolic rate and its scaling appear less so (Beaman et al., preprint; Pettersen et al. 2018; Fossen et al. 2019).

That high mortality in early life may select for fast growth and cause steep scaling of metabolic rate has also been proposed before (Epp & Lewis 1980; Glazier 1991, 2005), but with a focus on explaining static, species-level, scaling patterns. Glazier et al. (2011, 2020) have also found evidence of shallower scaling of metabolic rate in freshwater amphipod populations naturally selected for slow growth; amphipods from springs where adult mortality is high (due to size-selective predation on large amphipods by fish) exhibit lower b, relative to conspecifics from fishless springs. This is the opposite of what I propose for the metabolic scaling of fish, where larger and faster-growing individuals generally have higher survival (Hutchings 1991; Sogard 1997)—something that also applies to aquatic invertebrates (Moran & Emlet 2001, Marshall & Keough 2007, 2008)—but supports that size-selective mortality affects metabolic scaling exponents in the predicted direction (high mortality of slow growers = high b, low mortality of slow growers = low b).

A handful of earlier studies on primarily birds (Daan et al. 1989; Piersma et al. 1995; Scott et al. 1996; Lindström et al. 1999; Kvist & Lindström 2001), but also a bat (McLean & Speakman 2000) and a reptile (Maxwell et al. 2003), have also analysed within-individual metabolic scaling relationships but found mixed results when comparing with scaling across individuals; the studies on endotherms report mainly higher (Daan et al. 1989; Scott et al. 1996; McLean & Speakman 2000) but also lower (Kvist & Lindström 2001) scaling exponents within compared to across individuals, or did not compare within- and across-individual scaling (Lindström et al. 1999), while the reptile study finds within- and across-individual scaling to be the same (Maxwell et al. 2003). However, common for all these studies is that they had relatively low samples sizes in their analyses of within-individual scaling (two to nine individuals), and all the endotherm studies investigated scaling of primarily adults for which body mass changed due to changes in body composition related to season (migration or moulting) or to feeding, fasting, or flying. Thus, for all but the reptile study, these within-individual scaling relationships do not represent ontogenetic metabolic scaling of growing individuals that are developing during ontogeny, and more data are needed to evaluate if metabolic scaling differs systematically across taxonomic levels, from ontogenetic across static to evolutionary levels of biological organisation. If metabolic rate scales systematically steeper at higher taxonomic levels, as observed here for fishes, it can potentially also explain the continuing debate over $\frac{2}{3}$ *vs.* $\frac{3}{4}$-power scaling for mammals and birds: studies that have analysed metabolic scaling within these groups (i.e., at a relatively low taxonomic level) have found shallower, $\frac{2}{3}$-power scaling (e.g., White & Seymour 2003; White et al. 2006), while those that have combined mammals and birds (a higher taxonomic level) have found steeper, $\frac{3}{4}$-power scaling (e.g., Kleiber 1932; Gillooly et al. 2017).

In conclusion, the observed effects of growth (under restricted food availability) and natural mortality rates on metabolic scaling exponents support that metabolic scaling is shaped by ecological factors (Witting 1998; Glazier 2005, 2014, 2018; Killen et al. 2010; White & Kearney 2013, 2014; Harrison 2017; Uyeda et al. 2017; Hatton et al. 2019; Glazier et al. 2020), rather than being fixed by physical or geometric principles (West et al. 1997, 1999; Banavar et al. 2010) that, in turn, control ecological processes (Brown et al. 2004; Sibly et al. 2012). While others have also promoted an ecological view on metabolic scaling recently—and, among other things, theorised that b is shaped by optimisation of adult body size through species-specific strategies for allocation of energy to growth or reproduction (Kozłowski et al. 2020), or by life-history tradeoffs between fast-paced neurolocomotory performance in small-bodied species and slow-paced risk-reducing strategies in large species (Harrison 2017)—they have primarily focused on final (adult) body size, given little consideration to within-individual scaling, and one theory predicts shallower metabolic scaling across than within species [see Figure 5 in Kozłowski et al. (2020)], which is the opposite of what the empirical data on fish I present here indicate (Fig. 1). I hope that the thoughts and ideas I have expressed here will inspire more research on metabolic scaling across all taxonomic levels, including within individuals.

Funding

This work was supported by research grants from VILLUM FONDEN (40713) and the Independent Research Fund Denmark (1054–00020B).

Supplementary data

Supplementary data available at *ICB* online.

Conflict of interest

I have no conflicting interests with myself.

Data availability

Data and R script were uploaded for reviewers and editors at manuscript submission, and are archived in a citable format on figshare: https://doi.org/10.6084/m9.figshare.19619895.

References

Almany GR, Webster MS. 2006. The predation gauntlet: early post-settlement mortality in reef fishes. Coral Reefs 25:19–22.

Auer SK, Salin K, Rudolf AM, Anderson GJ, Metcalfe NB. 2015. Flexibility in metabolic rate confers a growth advantage under changing food availability. J Anim Ecol 84:1405–11.

Auer SK, Salin K, Rudolf AM, Anderson GJ, Metcalfe NB. 2016. Differential effects of food availability on minimum and maximum rates of metabolism. Biol Lett 12:20160586.

Banavar JR, Moses ME, Brown JH, Damuth J, Rinaldo A, Sibly RM, Maritan A. 2010. A general basis for quarter-power scaling in animals. Proc Natl Acad Sci 107:15816–20.

Bates D, Maechler M, Bolker B, Walker S. 2015. Fitting linear mixed-effects models using lme4. J Stat Softw 67:1–48.

Beaman JE, Ortiz-Barrientos D, Monro K, Hall MD, White CR (preprint) Metabolic scaling has diversified among species, despite an evolutionary constraint within species. bioRxiv 2020.05.26:117846, doi: https://doi.org/10.1101/2020.05.26.117846.

Brody S. 1945. Bioenergetics and growth. Reinhold, New York.

Brown JH, Gillooly JF, Allen AP, Savage VM, West GB. 2004. Toward a metabolic theory of ecology. Ecology 85:1771–89.

Brown JH, Hall CAS, Sibly RM. 2018. Equal fitness paradigm explained by a trade-off between generation time and energy production rate. Nat Ecol Evol 2:262–8.

Burton T, Killen SS, Armstrong JD, Metcalfe NB. 2011. What causes intraspecific variation in resting metabolic rate and what are its ecological consequences? Proc R Soc B 278:3465–73.

Capellini I, Venditti C, Barton RA. 2010. Phylogeny and metabolic scaling. Ecology 91:2783–93.

Chang J, Rabosky DL, Smith SA, Alfaro ME. 2019. An R package and online resource for macroevolutionary studies using the ray-finned fish tree of life. Methods Ecol Evol 10:1118–24.

Czarnołęski M, Kozłowski J, Dumiot G, Bonnet J-C, Mallard J, Dupont-Nivet M. 2008. Scaling of metabolism in *Helix aspersa* snails: changes through ontogeny and response to selection for increased size. J Exp Biol 211:391–400.

Cheung WWL, Sarmiento JL, Dunne J, Frölicher TL, Lam VWY, Palomares MLD, Watson R, Pauly D. 2013. Shrinking of fishes exacerbates impacts of global ocean changes on marine ecosystems. Nat Clim Change 3:254–8.

Cheverud JM. 1982. Relationships among ontogenetic, static, and evolutionary allometry. Am J Phys Anthropol 59:139–49.

Daan S, Masman D, Strijkstra A, Verhulst S. 1989. Intraspecific allometry of basal metabolic rate: relations with body size, temperature, composition, and circadian phase in the kestrel, *Falco tinnunculus*. J Biol Rhythms 4:155–71.

Davis TLO, Lyne V, Jenkins GP. 1991. Advection, dispersion and mortality of a patch of southern Bluefin tuna larvae *Thunnus maccoyii* in the East Indian Ocean. Mar Ecol Prog Ser 73:33–45.

DeLong JP, Okie JG, Moses ME, Sibly RM, Brown JH. 2010. Shifts in metabolic scaling, production, and efficiency across major evolutionary transitions of life. Proc Natl Acad Sci 107:12941–5.

Deutsch C, Ferrel A, Seibel B, Pörtner H-O, Huey RB. 2015. Climate change tightens a metabolic constraint on marine habitats. Science 348:1132–5.

Deutsch C, Pell JL, Seibel B. 2020. Metabolic trait diversity shapes marine biogeography. Nature 585:557–62.

Dillon ME, Wang G, Huey RB. 2010. Global metabolic impacts of recent climate warming. Nature 467:704–6.

Epp RW, Lewis WM, Jr.. 1980. The nature and ecological significance of metabolic changes during the life history of copepods. Ecology 61:259–64.

Fossen EIF, Pélabon C, Einum S. 2019. Genetic and environmental effects on the scaling of metabolic rate with body size. J Exp Biol 222:jeb193243.

Freckleton RP, Harvey PH, Pagel M. 2002. Phylogenetic analysis and comparative data: a test and review of evidence. Am Nat 160:712–26.

Froese R, Pauly D. 2019. FishBase. World Wide Web electronic publication. www.fishbase.org, version 12/2019.

Fu SJ, Xie XJ, Cao ZD. 2005. Effect of fasting on resting metabolic rate and postprandial metabolic response in *Silurus meridionalis*. J Fish Biol 67:279–85.

Gillooly JF, Gomez JP, Mavrodiev EV. 2017. A broad-scale comparison of aerobic activity levels in vertebrates: endotherms versus ectotherms. Proc R Soc B 284:20162328.

Glazier DS. 1991. Separating the respiration rates of embryos and brooding females of Daphnia magna: Implications for the cost of brooding and the allometry of metabolic rate. Limnol Oceanogr 36:354–61.

Glazier DS. 2005. Beyond the '3/4-power law': variation in the intra- and interspecific scaling of metabolic rate in animals. Biol Rev 80:11–662.

Glazier DS. 2008. Effects of metabolic level on the body size scaling of metabolic rate in birds and mammals. Proc R Soc B 275:1405–10.

Glazier DS. 2009. Activity affects intraspecific body-size scaling of metabolic rate in ectothermic animals. J Comp Physiol B 179:821–8.

Glazier DS. 2010. A unifying explanation for diverse metabolic scaling in animals and plants. Biol Rev 85:111–38.

Glazier DS. 2014. Metabolic scaling in complex living systems. Systems 2:451–540.

Glazier DS. 2015. Is metabolic rate a universal 'pacemaker' for biological processes? Biol Rev 90:377–407.

Glazier DS. 2018. Rediscovering and reviving old observations and explanations of metabolic scaling in living systems. Systems 6:4.

Glazier DS. 2020. Activity alters how temperature influences intraspecific metabolic scaling: testing the metabolic-level boundaries hypothesis. J Comp Physiol B 190:445–54.

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

Glazier DS, Borrelli JJ, Hoffman CL. 2020. Effects of fish predators on the mass-related energetics of a keystone freshwater crustacean. Biology 9:40.

Gould SJ. 1975. Allometry in primates, with emphasis on scaling and the evolution of the brain. In Approaches to primate paleobiology(Szalay F, ed). Karger, Basel, pp. 244–92.
Harrison JF. 2017. Do performance-safety tradeoffs cause hypometric metabolic scaling in animals? Trends Ecol Evol 32:653–64.
Harrison JF. 2018. Approaches for testing hypotheses for the hypometric scaling of aerobic metabolic rate in animals. Am J Physiol Regul Integr Comp Physiol 315:R879–94.
Hatton IA, Dobson AP, Galbraith ED, Loreau M. 2019. Linking scaling laws across eukaryotes. Proc Natl Acad Sci 116:21616–22.
Hemmingsen AM. 1960. Energy metabolism as related to body size and respiratory surfaces, and its evolution. Rep Steno Mem Hosp Nord Insulin Lab 9:1–110.
Hou C, Zuo W, Moses ME, Woodruff WH, Brown JH, West GB. 2008. Energy uptake and allocation during ontogeny. Science 322:736–9.
Houde ED. 1997. Patterns and trends in larval-stage growth and mortality of teleost fish. J Fish Biol 51:52–83.
Houde ED, Zastrow CE. 1993. Ecosystem- and taxon-specific dynamic and energetics properties of larval fish assemblages. Bull Mar Sci 53:290–335.
Hutchings JA. 1991. Fitness consequences of variation in egg size and food abundance in brook trout *Salvelinus fontinalis*. Evolution 45:1162–8.
Jerde CL, Kraskura K, Elison EJ, Csik SR, Stier A, Taper ML. 2019. Strong evidence for an intraspecific metabolic scaling coefficient near 0.89 in fish. Front Physiol 10:1166.
Killen SS, Atkinson D, Glazier DS. 2010. The intraspecific scaling of metabolic rate with body mass in fishes depends on lifestyle and temperature. Ecol Lett 13:184–93.
Killen SS, Glazier DS, Rezende EL, Clark TD, Atkinson D, Willener AST, Halsey LG. 2016. Ecological influences and morphological correlates of resting and maximal metabolic rates across teleost fish species. Am Nat 187:592–606.
Kleiber M. 1932. Body size and metabolism. Hilgardia 6:315–53.
Kleiber M. 1947. Body size and metabolic rate. Physiol Rev 27:511–41.
Kozłowski J, Konarzewski M, Czarnołęski M. 2020. Coevolution of body size and metabolic rate in vertebrates: a life-history perspective. Biol Rev 95:1393–417.
Kvist A, Lindström Å. 2001. Basal metabolic rate in migratory waders: intra-individual, intraspecific, interspecific and seasonal variation. Funct Ecol 15:465–73.
Lande R. 1979. Quantitative genetic analysis of multivariate evolution, applied to brain–body size allometry. Evolution 33:402–16.
Lindström Å, Klaassen M, Kvist A. 1999. Variation in energy intake and basal metabolic rate of a bird migrating in a wind tunnel. Funct. Ecol. 13:352–9.
Maxwell LK, Jacobson ER, McNab BK. 2003. Intraspecific allometry of standard metabolic rate in green iguanas, *Iguana iguana*. Comp Biochem Physiol A: Mol Integr Physiol 136:301–10.
Marshall DJ, Keough MJ. 2007. The evolutionary ecology of offspring size in marine invertebrates. Adv Mar Biol 53: 1–60.
Marshall DJ, Keough MJ. 2008. The relationship between offspring size and performance in the sea. Am Nat 171: 214–24.
McGurk MD. 1986. Natural mortality of marine pelagic fish eggs and larvae: role of spatial patchiness. Mar Ecol Prog Ser 34:227–42.
McLean JA, Speakman JR. 2000. Effects of body mass and reproduction on the basal metabolic rate of brown long-eared bats (*Plecotus auritus*). Physiol Biochem Zool 73:112–21.
Melvin SD, Houlahan JE. 2012. Tadpole mortality varies across experimental venues: do laboratory populations predict responses in nature? Oecologia 169:861–8.
Moran AL, Emlet RB. 2001. Offspring size and performance in variable environments: field studies on a marine snail. Ecology 82:1597–612.
Mousseau TA, Roff DA. 1987. Natural selection and the heritability of fitness components. Heredity 59:181–97.
Mueller P, Diamond J. 2001. Metabolic rate and environmental productivity: well-provisioned animals evolved to run and idle fast. Proc Natl Acad Sci 98:12550–4.
Norin T, Malte H. 2011. Repeatability of standard metabolic rate, active metabolic rate and aerobic scope in young brown trout during a period of moderate food availability. J Exp Biol 214:1668–75.
Norin T, Malte H, Clark TD. 2016. Differential plasticity of metabolic rate phenotypes in a tropical fish facing environmental change. Funct Ecol 30:369–78.
Norin T, Gamperl AK. 2018. Metabolic scaling of individuals vs. populations: evidence for variation in scaling exponents at different hierarchical levels. Funct Ecol 32:379–88.
Paradis A, Schliep K. 2019. ape 5.0: an environment for modern phylogenetics and evolutionary analyses in R. Bioinformatics 35:526–8.
Pélabon C, Bolstad GH, Egset CK, Cheverud JM, Pavlicev M, Rosenqvist G. 2013. On the relationship between ontogenetic and static allometry. Am Nat 181:195–212.
Pettersen AK, Marshall DJ, White CR. 2018. Understanding variation in metabolic rate. J Exp Biol 221:jeb166876.
Pettersen AK, Hall MD, White CR, Marshall DJ. 2020. Metabolic rate, context-dependent selection, and the competition-colonization trade-off. Evol Lett 4:333–44.
Pepin P. 1991. Effect of temperature and size on development, mortality, and survival rates of the pelagic early life history stages of marine fish. Can J Fish AquatSci 48:503–18.
Piersma T, Cadée N, Daan S. 1995. Seasonality in basal metabolic rate and thermal conductance in a long-distance migrant shorebird, the know (*Calidris canutus*). J Comp Physiol B 165:37–45.
R Core Team. 2020. R: a language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. URL: https://www.R-project.org/.
Rabosky DL, Chang J, Title PO, Cowman PF, Sallan L, Friedman M, Kaschner K, Garilao C, Near TJ, Coll M, Alfaro ME. 2018. An inverse latitudinal gradient in speciation rate for marine fishes. Nature 559:392–5.
Riisgård HU. 1998. No foundation of a "3/4 power scaling law" for respiration in biology. Ecol Lett 1:71–3.
Riska B, Atchley WR. 1985. Genetics of growth predict patterns of brain-size evolution. Science 229:668–71.
Roberts MF, Lightfoot EN, Porter WP. 2010. A new model for the body size–metabolism relationship. Physiol Biochem Zool 83:395–405.
Rubner M. 1883. Über den einfluss der körpergrösse auf stoff- und kraftwechsel. Zeitschr F Biol 19:535–62.

Scott I, Mitchell PI, Evans PR. 1996. How does variation in body composition affect the basal metabolic rate of birds? Funct Ecol 10:307–13.

Sibly RM, Brown JH, Kodric-Brown A. 2012. Metabolic ecology: a scaling approach. Wiley-Blackwell, John Wiley & Sons Ltd, West Sussex.

Sogard SM. 1997. Size-selective mortality in the juvenile stage of teleost fishes: a review. Bull Mar Sci 60:1129–57.

Tsuboi M, ven der B, Kopperud BT, Erritzøe J, Voje KL, Kotrschal A, Yopak KE, Collin SP, Iwaniuk AN, Kolm N. 2018. Breakdown of brain–body allometry and the encephalization of birds and mammals. Nat Ecol Evol 2:1492–500.

Uyeda JC, Pennell MW, Miller ET, Maia R, McClain CR. 2017. The evolution of energetic scaling across the vertebrate tree of life. Am Nat 190:185–99.

Voje KL, Hansen TF, Egset CK, Bolstad GH, Pélabon C. 2013. Allometric constraints and the evolution of allometry. Evolution 68:866–85.

van de Pol M, Wright J. 2009. A simple method for distinguishing within- versus between-subject effects using mixed models. Anim Behav 77:753–8.

Van Leeuwen TE, Rosenfeld JS, Richards JG. 2012. Effects of food ration on SMR: influence of food consumption on individual variation in metabolic rate in juvenile coho salmon (*Oncorhynchus kisutch*). J Anim Ecol 81:395–402.

von Bertalanffy L. 1957. Quantitative laws in metabolism and growth. Q Rev Biol 32:217–31.

Wang T, Hung CCY, Randall DJ. 2006. The comparative physiology of food deprivation: from feast to famine. Annu Rev Physiol 68:223–51.

Weibel ER, Hoppeler H. 2005. Exercise-induced maximal metabolic rate scales with muscle aerobic capacity. J Exp Biol 208:1635–44.

West GB, Brown JH, Enquist BJ. 1997. A general model for the origin of allometric scaling laws in biology. Science 276:122–6.

West GB, Brown JH, Enquist BJ. 1999. The fourth dimension of life: fractal geometry and allometric scaling of organisms. Science 284:1677–9.

West GB, Brown JH, Enquist BJ. 2001. A general model for ontogenetic growth. Nature 413:628–31.

White CR. 2010. There is no single *p*. Nature 464:691–3.

White CR, Seymour RS. 2003. Mammalian basal metabolic rate is proportional to body mass$^{2/3}$. Proc Natl Acad Sci 100: 4046–9.

White CR, Seymour RS. 2004. Does basal metabolic rate contain a useful signal? Mammalian BMR allometry and correlation with a selection of physiological, ecological, and life-history variables. Physiol Biochem Zool 77:929–41.

White CR, Phillips NF, Seymour RS. 2006. The scaling and temperature dependence of vertebrate metabolism. Biol Lett 2:125–7.

White CR, Cassey P, Blackburn TM. 2007. Allometric exponents do not support a universal metabolic allometry. Ecology 88:315–23.

White CR, Blackburn TM, Seymour RS. 2009. Phylogenetically informed analysis of the allometry of mammalian basal metabolic rate supports neither geometric nor quarter-power scaling. Evolution 63:2658–67.

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

White CR, Kearney MR. 2014. Metabolic scaling in animal: methods, empirical results, and theoretical explanations. Compr Physiol 4:231–56.

White CR, Marshall DJ, Alton LA, Arnold PA, Beaman JE, Bywater CL, Condon C, Crispin TS, Janetzki A, Pirtle E, Winwood-Smith HS, Angilletta MJ, Jr, Chenoweth SF, Franklin CE, Halsey LG, Kearney MR, Portugal SJ, Ortiz-Barrientos D. 2019. The origin and maintenance of metabolic allometry in animals. Nat Ecol Evol 3:598–603.

Witting L. 1998. Body mass allometries caused by physiological or ecological constraints? Trends Ecol Evol 13:25.

Ye X, Lu L, Jiang M, Jia J, Li W, Wu H, Liao Y, Li J. 2021. metabolic scaling: individual versus intraspecific scaling of Nile tilapia (*Oreochromis niloticus*). J Comp Physiol B 191: 721–9.