Co-variation and trade-offs in ontogenetic scaling of growth and metabolic rates

Alexander Rosén[1], Anna Andreassen[2], Zoe Storm[2], Suzanne Raqbi[1], Axel Moesby[2], Julius Exsteen[2], Ricardo Beldade[3], Suzanne Mills[2], and Tommy Norin[4]

[1]Technical University of Denmark
[2]Affiliation not available
[3]CNRS
[4]Technical University of Denmark National Institute of Aquatic Resources

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Abstract

How steeply metabolic rate and growth rate change (scale) with body mass, and their causal relationship, has been hotly debated for over a century. Prevailing theory argues that metabolic and growth rates are physically constrained to scale in the same way for all organisms, with body mass$^{0.75}$. Rivalling life-history optimisation theory suggests that scaling can vary and is shaped by evolutionary optimisation of energy allocation among metabolic rate, growth, and reproduction. However, past research has almost exclusively investigated metabolic scaling *across* individuals or species, not *within* individuals as they grow through ontogeny. This is critical oversight, as body mass is gained through growth, and bodily growth occurs *within* individuals. Here, we longitudinally measured body mass and both standard (maintenance) and maximum metabolic rates (and thus metabolic scope) on average 6.6 times within the same 389 individuals from seven fish species. We uncover previously unrecognized co-variation in the within-individual (ontogenetic) scaling of metabolic and growth rates: scaling of standard metabolic rate correlates positively with scaling of growth rate, while scaling of metabolic scope corelates negatively with scaling of growth rate. This indicates a trade-off whereby accelerating ontogenetic growth comes at a cost of reduced metabolic scope to support functions beyond maintenance. Our results challenge traditional 0.75-power scaling and new life-history optimisation theory that predicts a negative correlation between ontogenetic scaling of metabolic rate and growth. Our findings also suggest that unaccounted variation in growth is a likely explanation for the century-long debate about whether, and to what extent, metabolic scaling varies.

Dear Editor,

We are pleased to submit our manuscript titled "**Co-variation and trade-offs in ontogenetic scaling of growth and metabolic rates**" for consideration as a Research Report in *Ecology letters*.

Our study assesses the important link between whole-organism growth and metabolic rate, specifically how these traits change (scale) together with body mass. Understanding these fundamental biological relationships is key for predicting how populations respond to selection pressures such as size-selective harvesting or climate change.

Growth and metabolic rates have long been assumed to be connected, but to what extent, and the directionality of the relationship, has been hotly debated. However, the links between metabolic and growth rate scaling have never been examined within individuals as they grow through ontogeny; all existing metabolic theories are built on empirical data on metabolic scaling relationships *across* different individuals or species. This is a critical oversight in the work on the immensely influential theories to date, as it is at the level of the individual that bodily growth and selection occur. The lack of work on the individual level is presumably because it is very time consuming and logistically cumbersome to do the longitudinally repeated measurements of metabolic rates on the same individuals as they grow through life.

Our study tackles this challenging endeavour and investigates the link between scaling of metabolic rate and growth rate at the individual level. We have collected a uniquely large data set containing longitudinally repeated growth and metabolic rate measurements of 389 individuals across seven fish species. With an average of 6.6 repeated measurements per individual, our study contains 4324 paired measurements of body mass and metabolic rates (maintenance and maximum, and metabolic scope). We find hitherto overlooked co-variation in the ontogenetic scaling of metabolic and growth rates, with body mass: there is a positive correlation between the mass-scaling of maintenance metabolic rate and that of growth, while there is a negative relationship between the mass-scaling of metabolic scope and growth. This indicates a trade-off where accelerating ontogenetic growth comes at the cost of elevated maintenance metabolic demands and declining metabolic scope to support other tasks than maintenance. Such a trade-off is likely to have fitness consequences (e.g. fast growth allows individuals to escape size selective mortality, while reduced metabolic scope can compromise reproduction and other energy demanding tasks), which we discuss in our manuscript.

Our novel findings at the within-individual level contrast with and challenge existing metabolic scaling theories, including: (1) fixed 0.75-power scaling, as predicted by fractal network theory and the metabolic theory of ecology (West et al. 1997,1999; Brown et al. 2004); (2) new life-history optimisation theory, which predicts a negative relationship between the ontogenetic scaling of maintenance metabolic rate and growth based on scaling relationships across species (White et al. 2022), the opposite of what we find here for scaling of individuals; and (3) broad analyses of eukaryote scaling relationships, which argue that growth is constrained to scale to the 0.75-power of body mass independently of metabolic scaling (Hatton et al. 2019).

Our individual-based approach provides new insight into biological scaling relationships, and a novel understanding of size-related energetic trade-offs between growth and metabolic rates that we believe will inspire and guide biologists from a diverse array of fields in their future research. Therefore, we believe that *Ecology letters* would be the perfect outlet for our new findings. We suggest the following reviewers, who we feel will provide a critical but balanced and unbiased evaluation of our manuscript:

- Craig White, professor at Monash University, Australia: craig.white@monash.edu
- Giulia Ghedini, group leader at Gulbenkian Institute, Portugal: giulia.ghedini@gimm.pt
- Douglas Glazier, professor at Juniata College, USA glazier@juniata.edu

We hope you find our manuscript suitable for *Ecology letters* and recognize its substantial contribution to linking metabolism and growth and the broader field of biological scaling.

Sincerely,

Alexander Rosén and Tommy Norin (on behalf of all authors)

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[1] DTU Aqua: National Institute of Aquatic Resources, Technical University of Denmark, Henrik Dams Allé 202, 2800 Kongens Lyngby, Denmark

[2] PSL Université Paris: EPHE-UPVD-CNRS, UAR 3278 CRIOBE, BP 1013, 98729 Papetoai, Moorea, French Polynesia

[3] College of Science and Engineering and ARC Centre of Excellence for Coral Reef Studies, James Cook University, Townsville, 4811, Australia

[4] Facultad de Ciencias Biológicas, Pontificia Universidad Católica de Chile, Bernardo O'Higgins 340, Santiago de Chile, Chile

[5] Laboratoire d'Excellence "CORAIL", France

[6] Institut Universitaire de France (IUF), France

*Corresponding authors: Alexander Rosén (alero@aqua.dtu.dk, +45 22 48 86 14)

Author contributions

AR: Conceptualisation, data collection, data analysis, writing (initial draft and revisions), co-supervision

AHA: Data collection, writing (revisions)

ZS: Data collection, data analysis, writing (revisions)

JWE: Data collection, data analysis

AFM: Data collection, data analysis

SR: Data collection, data analysis, writing (revisions)

RB: Facilities, equipment, co-supervision, writing (revisions)

SCM: Facilities, equipment, co-supervision, writing (revisions)

TN: Conceptualisation, data collection, data analysis, writing (revisions), funding, equipment, facilities, primary supervision

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Abstract

How steeply metabolic rate and growth rate change (scale) with body mass, and their causal relationship, has been hotly debated for over a century. Prevailing theory argues that metabolic and growth rates are physically constrained to scale in the same way for all organisms, with body mass$^{0.75}$. Rivalling life-history optimisation theory suggests that scaling can vary and is shaped by evolutionary optimisation of energy allocation among metabolic rate, growth, and reproduction. However, past research has almost exclusively investigated metabolic scaling *across* individuals or species, not *within* individuals as they grow through ontogeny. This is critical oversight, as body mass is gained through growth and bodily growth occurs within individuals. Here, we longitudinally measured body mass and both standard (maintenance) and maximum metabolic rates, and thus metabolic scope, on average 6.6 times within the same 389 individuals from seven fish species. We uncover previously unrecognized co-variation in the within-individual (ontogenetic) scaling of metabolic and growth rates: scaling of standard metabolic rate correlates positively with scaling of growth rate, while scaling of metabolic scope corelates negatively with scaling of growth rate. This indicates a trade-off whereby accelerating ontogenetic growth comes at a cost of reduced metabolic scope to support functions beyond maintenance. Our results challenge traditional 0.75-power scaling and new life-history optimisation theory that predicts a negative correlation between ontogenetic scaling of metabolic rate and growth. Our findings also suggest that unaccounted variation in growth is a likely explanation for the century-long debate about whether, and to what extent, metabolic scaling varies.

Significance statement

It remains unknown how growth and metabolic rates (co)vary with body size. Understanding these fundamental biological scaling relationships is key for predicting size-dependent energy requirements and growth of individuals and populations. However, past work has overlooked how growth and metabolic rates scale within individuals as they grow through ontogeny, hampering our understanding of size-dependent ecological and evolutionary responses to both natural and human-induced selection. We show that variation in within-individual ontogenetic metabolic scaling among both individuals and species can be explained by variation in the ontogenetic scaling of growth rate. We also identify a trade-off whereby accelerating ontogenetic growth comes at the cost of both elevated maintenance metabolic demands and reduced metabolic scope that is likely to have fitness consequences.

Introduction

Body size and organismal biomass are the main drivers of biological patterns in nature, influencing metabolic rate, growth, reproduction, and ecological interactions (Brown et al., 2018; Burger et al., 2019; Hatton et al., 2015, 2019; Schmidt-Nielsen, 1984). Most biological rates change (scale) out of proportion with body mass following an allometric power function, rate = $a \cdot \text{mass}^b$, where a is the scaling coefficient and b is the scaling exponent (Schmidt-Nielsen, 1984). Of all biological rates, the scaling of metabolic rate with body mass has received the most attention since Kleiber (1932, 1947) produced his

famous “mouse-to-elephant curve”, showing that metabolic rates of endotherms scaled with body mass with an exponent (*b*) of approximately 0.75. This ¾-power scaling relationship has since been proposed to be a universal law in biology (Brown et al., 2004; Gillooly et al., 2001), explained by the fractal nature of resource delivery networks, such as the vascular system in vertebrates and xylem in trees (West et al., 1997, 1999). This has, in turn, laid the foundation for the metabolic theory of ecology (Brown et al., 2004), which posits that the canonical ¾-power scaling relationship between metabolic rate and body mass governs the scaling of all ecological processes, because metabolic rate controls how energy is allocated to growth, reproduction, and maintenance (Brown et al., 2004; Sibly et al., 2012). The metabolic theory of ecology has had an immense influence on metabolic and life history theory (White & Marshall, 2023). However, the notion that metabolic rate should scale equally steeply for all organisms (be constrained to a fixed value of *b*) has been repeatedly challenged, as multiple physiological, ecological, and taxonomic factors have been proposed to influence the steepness of metabolic scaling (Bertalanffy, 1957; Darveau et al., 2002; Glazier, 2005, 2022; Hatton et al., 2019; Norin, 2022; White et al., 2005; White & Marshall, 2023). Among these, the role of growth rate in metabolic scaling has been much debated (Glazier, 2015; Kearney, 2021; Marshall & White, 2019; Norin, 2022; Ricklefs, 2003; Riisgård, 1998).

Scaling of metabolic and growth rates within and across individuals

Growth requires metabolic energy and proximally determines body size. Growth could thus be an obvious driver of variation in metabolic scaling, but the prevailing view in highly influential metabolic growth models has been that it is metabolic scaling and variation therein that governs growth, rather than the other way around (Bertalanffy, 1957; Hou et al., 2008; West et al., 2001). Others have, however, proposed that metabolic rates adjust to growth, with the costs and regulation of growth driving metabolic rate and its scaling (Glazier, 2015; Hatton et al., 2019; Parry, 1983; Ricklefs, 2003). Alternatively, life-history theory suggests that the mass-scaling of metabolic and growth rates has co-evolved to maximise fitness (lifetime reproductive success) under different selection pressures (Harrison, 2017; Kozłowski et al., 2020; Kozłowski & Weiner, 1997; Marshall & White, 2019; White et al., 2022), such that one trait does not govern the other (White et al., 2022). Evidence supporting co-evolution includes artificial selection experiments on insects, which suggests evolutionary adaptability of allometric scaling between morphological traits (Bolstad et al., 2015; Frankino et al., 2005). Selection for body size has also been found to affect the scaling of metabolic rate in snails (Czarnołęski et al., 2008). However, the causal relationship between the scaling of metabolic rate and growth rate remains unknown and highly debated despite a century of work on metabolic theories (Kearney, 2021).

A possible explanation for the lack of consensus is that almost all previous work on metabolic scaling has investigated how metabolic rate changes with body mass across species (‘evolutionary’ scaling) or across individuals of different sizes within species (‘static’ scaling) (Harrison et al., 2022; Norin, 2022). How metabolic rate scales within individuals as they grow (ontogenetic scaling) has been almost entirely overlooked (but see Barneche et al., 2019; Norin, 2022; Norin & Gamperl, 2018; Ye et al.,

2021). This is critical oversight, as body growth is fundamentally a within-individual process and because selection acts at the level of the individual. Indeed, work from the related field of morphological allometry has shown how variation in individual growth trajectories and ontogenetic size-scaling of morphological traits can shape static and evolutionary scaling relationships (Pélabon et al., 2013; Riska & Atchley, 1985; Shingleton et al., 2007; Shingleton & Frankino, 2018). The oversight of ontogenetic metabolic scaling likely stems from the temporal, logistical, and technical challenges associated with conducting the longitudinally repeated measurements of metabolic rates and body mass on the same individuals that are required to quantify ontogenetic scaling. This can be especially challenging when wanting to evaluate scaling relationships from early in ontogeny, where small and fragile life stages such as fish larvae are difficult to handle and weigh.

To further our understanding of how the ontogenetic (within-individual) scaling of metabolic and growth rates co-vary, we measured oxygen uptake rates (as estimates of metabolic rates) and body mass longitudinally within 389 individuals from seven fish species: brown trout (*Salmo trutta*), rainbow trout (*Oncorhynchus mykiss*), cunner (*Tautogolabrus adspersus*), blue-green damselfish (*Chromis viridis*), orange-fin anemonefish (*Amphiprion chrysopterus*), Trinidadian guppy (*Poecilia reticulata*), and zebrafish (*Danio rerio*). As each individual fish was repeatedly measured for metabolic rate and body mass on average 6.6 times (ranging between four and twelve times), our dataset contains 2449 and 1989 measurements of standard (maintenance) metabolic rate (SMR) and growth rate, respectively, with growth rate being the absolute increase in body mass over time between two consecutive measurements. In addition to our measurements of SMR, we also measured maximum (active) metabolic rate (MMR; 1875 measurements) of individuals from all species except zebrafish. This further allowed us to calculate aerobic metabolic scope, the difference between MMR and SMR, which represents the total capacity to allocate oxygen and energy to activities other than maintenance. We calculated metabolic scope as both absolute aerobic scope (AS; MMR–SMR) and factorial (relative) aerobic scope (FAS; MMR/SMR). To examine variation in the ontogenetic scaling of metabolic traits (SMR, MMR, AS, and FAS) and their relationships with the ontogenetic scaling of growth rate, we correlated ontogenetic scaling exponents (*b*) for metabolic traits with those for growth rate both within and across species.

While our primary focus is on variation in ontogenetic (within-individual) scaling, our data also allowed us to quantify scaling relationships at the static level (across individuals within a species) and compare these to ontogenetic scaling. Our focus on within-individual ontogenetic scaling provides a new and interesting comparison to the more traditional work that has investigated the scaling of metabolic rate (mainly maintenance metabolic rate) and growth rate across broad organismal groups (e.g. DeLong et al., 2010; Hatton et al., 2019).

Ontogenetic scaling of metabolic rates

The overall ontogenetic scaling of metabolic rate across species was similar for SMR, MMR, and AS, with average scaling exponents (*b*) of approximately 0.9 for all traits (Fig. 1; Table 1).

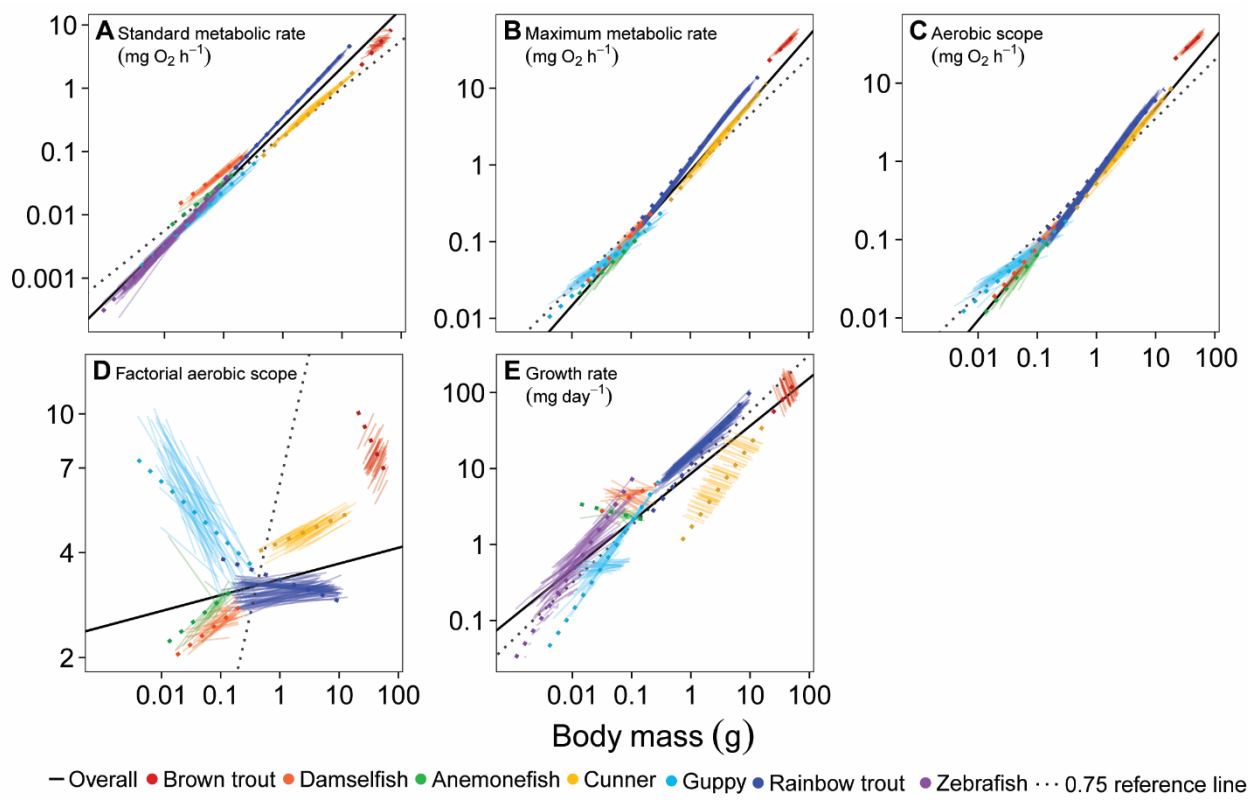

Figure 1. Body mass-scaling of (**A**) standard metabolic rate (SMR), (**B**) maximum metabolic rate (MMR), (**C**) aerobic scope (AS; MMR–SMR), (**D**) factorial aerobic scope (FAS; MMR/SMR), and (**E**) growth rate. Scaling within individuals (ontogenetic scaling) is presented as thin, solid, coloured lines; scaling across individuals within species (static scaling) is presented as dotted coloured lines; and the overall mean ontogenetic scaling across species is represented by the solid black line. The dotted black line is a reference line with a slope (scaling exponent, *b*) of 0.75. Mean scaling exponents for each species are presented in Fig. 2 and Table 1. MMR was not measured for zebrafish, wherefore AS and FAS could not be calculated. Metabolic and growth rates have been adjusted to a common temperature of 20°C for all species for these graphical presentations (which changes the intercept but not the slope, *b*).

Within species, the mean ontogenetic scaling exponents for SMR, MMR, and AS of all individuals generally ranged from ~0.5 to 1.1 for the different species (Fig. 2, Table S1). An exception to this was the ontogenetic scaling of brown trout SMR, with a mean ontogenetic scaling exponent (b_{SMR}) of 0.27 for the species, ranging broadly from -0.35 to 0.80 for the different individuals (Fig. 3). This broad variation in ontogenetic scaling of SMR among brown trout individuals was likely caused by the restricted food regime they were maintained on, resulting in SMR being down-regulated more in individuals that initially had a relatively high SMR for their size, causing the low and even negative scaling exponents for those individuals (Norin, 2022; Norin & Malte, 2011). Such a down-regulation of SMR is unlikely to be sustainable over time, making this pattern in brown trout a potential exception among the other species. Nevertheless, the brown trout data aligns well with the overall scaling patterns observed across species (Fig. 1). Notably, of the species' 19 scaling exponents for metabolic rates (seven for SMR, six for MMR,

and six for AS), only four significantly overlapped with the proposed canonical value of 0.75 (Brown et al., 2004; Hatton et al., 2019), while the majority exceeded 0.75 (Fig. 2, Table S1).

Table 1. Mean (inverse variance weighted) ontogenetic and static scaling exponents (with 95% CIs in square brackets) for standard metabolic rate (SMR), maximum metabolic rate (MMR), aerobic scope (AS), factorial aerobic scope (FAS), and growth rate (GR) for all species. Mean scaling exponents for individual species can be found in supplementary Table S1.

Trait	Scaling exponent (*b*)	
	Ontogenetic	Static
SMR	0.87 [0.85, 0.88]	0.89 [0.87, 0.92]
MMR	0.89 [0.88, 0.91]	0.90 [0.87, 0.92]
AS	0.90 [0.88, 0.92]	0.90 [0.87, 0.94]
FAS	0.03 [0.01, 0.05]	0.03 [0.00, 0.06]
GR	0.60 [0.56, 0.65]	1.02 [0.95, 1.10]

While limited research exists on within-individual ontogenetic metabolic scaling (see Barneche et al., 2019; Norin, 2022; Norin & Gamperl, 2018; Ye et al., 2021), the ontogenetic scaling exponents reported here are generally higher than those found for SMR by Barneche et al. (2019) for zebrafish (b_{SMR} = 0.65) and by Ye et al. (2021) for Nile tilapia (b_{SMR} = 0.71). However, our ontogenetic metabolic scaling exponents align well with the relatively high static and evolutionary scaling exponents observed in other studies on fish (Clarke & Johnston, 1999; Jerde et al., 2019; White et al., 2005). The lower ontogenetic scaling exponents reported by Barneche et al. (2019) and Ye et al. (2021) could be explained by methodological differences. Where we used intermittent-flow respirometry to estimate metabolic rates, Ye et al. (2021) used continuous-flow respirometry and Barneche et al. (2019) used closed respirometry. Given the known limitations of continuous-flow and closed respirometry (Killen et al., 2021; Steffensen, 1989), we believe our approach provides a more accurate representation of the true ontogenetic scaling exponents for SMR.

Aside from our previous research on the ontogenetic scaling of metabolic rates in brown trout (Norin, 2022; Norin & Malte, 2011) and cunner (Norin & Gamperl, 2018), both of which are included in the present dataset, we are unaware of any past studies quantifying the ontogenetic scaling of MMR, and thus AS and FAS. Interestingly, while the ontogenetic scaling of SMR, MMR, and AS were generally steep, the ontogenetic scaling of FAS across species was close to horizontal (b_{FAS} = 0.03; Fig. 1, Table 1), indicating that relative aerobic metabolic scope remains constant across a wide body mass range, although with pronounced variation between species (Figs. 1D, 2). Our finding of shallow ontogenetic scaling of FAS aligns with the shallow evolutionary (across-species) scaling of FAS for both ectotherms (b_{FAS} = 0.04) and endotherms (b_{FAS} = -0.02) reported by Gillooly et al. (2017).

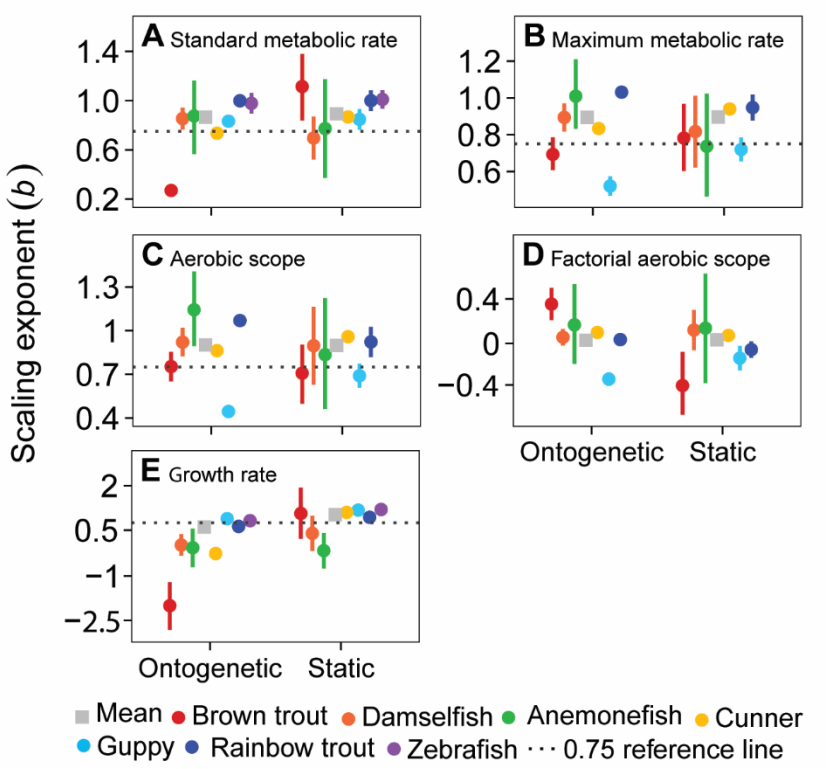

Figure 2. Mean ontogenetic and static scaling exponents (with 95% CI error bars) for (**A**) standard metabolic rate (SMR), (**B**) maximum metabolic rate (MMR), (**C**) aerobic scope (AS), (**D**) factorial aerobic scope (FAS), and (**E**) growth rate for the six or seven species (coloured circles) and their overall means (grey squares; CIs are small and hidden behind symbols). MMR (and thus AS and FAS) was not measured for zebrafish. Scaling exponents for individual species are in Table S1. The dotted black line is a reference line with a scaling exponent (*b*) of 0.75.

Ontogenetic scaling of growth rate and co-variation with metabolic scaling

The mean ontogenetic scaling of growth rate (GR) across fishes (b_{GR} = 0.60) was significantly shallower than the mean ontogenetic scaling of metabolic rates across fishes ($b \approx 0.9$; Fig. 1, Table 1), with nearly all individuals exhibiting steeper ontogenetic increases (higher *b*) in all of SMR, MMR, and AS with increasing body mass compared to the ontogenetic increase in growth rate (female guppies being the only exception; Fig. 3). The mean ontogenetic scaling of growth rate (b_{GR}) of 0.60 is also significantly shallower than the canonical 0.75-value (¾-power law) invoked by fractal geometry (West et al., 1997, 1999) but consistent with the findings of Case (1978), who reported an evolutionary (across-species) scaling exponent of 0.61 for growth rates of ten freshwater fishes. Similarly, Hatton et al. (2019) found shallower scaling of growth rate (b_{GR} = 0.74) compared to maintenance metabolic rate (b_{SMR} = 0.96) across diverse groups of eukaryotes. They further found that growth rate consistently scaled with an exponent of ~0.75 both within and across eukaryote groups, whereas maintenance metabolic rate did not, and they interpreted this consistent ¾-power scaling of growth rate as indicative of growth rate scaling governing metabolic rate scaling (Hatton et al., 2019). Our findings, on the other hand, demonstrate a strong positive relationship between the ontogenetic scaling of SMR and the ontogenetic scaling of growth rate, especially across species (Fig. 3) but also within them (Fig. S1), although not in a one-to-one

relationship; a linear regression between the ontogenetic scaling exponents for SMR (b_{SMR}) and those for growth rate (b_{GR}) across species has a slope of 0.22, meaning that the ontogenetic increase in SMR with increasing body mass is only around a fifth of the corresponding ontogenetic increase in growth rate (Fig. 3). Moreover, the intercept of the linear regression between the ontogenetic scaling exponents for SMR and those for growth rate across the seven species is 0.81, meaning that when growth rate is constant across ontogeny ($b_{GR} = 0$), SMR scales with an exponent of approximately 0.8 ($b_{SMR} = 0.81$). A decrease in growth rate as individuals grow larger across ontogeny ($b_{GR} < 0$) is thus associated with ontogenetic scaling of SMR that is shallower than $b_{SMR} \approx 0.8$, while an increasing ontogenetic growth rate ($b_{GR} > 0$) is associated with steeper scaling of SMR. Unaccounted variation in growth rate is thus a likely explanation for the now century-long debate about whether and by how much metabolic scaling varies for organisms in general and for fishes specifically. For example, large comparative studies on the static and evolutionary scaling of fishes have found mean scaling exponents (b) for SMR in the range 0.72 to 0.95, with an overall average around 0.88 (Clarke and Johnston 1999; Bokma 2004; White et al. 2006; Killen et al. 2016; Jerde et al. 2019; Hatton et al. 2019; Norin 2022). Interestingly, while we find here that SMR scales steeper than growth rate across most of the range of scaling exponents (higher b_{SMR} than b_{GR}; Fig. 3), they intersect (scale equally steeply) at $b = 1$. Thus, when growth rate scales isometrically (linearly) with body mass so does SMR (Fig. 3A). A doubling in mass would thus also entail a doubling of both SMR and growth rate. While this could be a coincidence, it could also imply that there is a hard ceiling to biological scaling where neither metabolic rate nor growth rate can increase faster with increasing body mass. An upper limit to metabolic scaling at $b = 1$ has also been suggested by others (Glazier, 2005, 2010, 2015).

A positive relationship among individuals between the ontogenetic scaling exponents for SMR (b_{SMR}) and those for growth rate (b_{GR}) existed within five of the seven species (with one of the five species showing borderline significance, $p = 0.053$ for rainbow trout; Fig. S1). Four of these five species with significant correlations (including rainbow trout) all had variation in the ontogenetic scaling of growth rate (b_{GR}) introduced through either strong sex-specific variation in growth rate (guppies) or through variation in food availability (damselfish, zebrafish) or early-life high temperature exposure before metabolic rate measurements (rainbow trout) with comparatively little effect on the ontogenetic scaling of SMR (b_{SMR}; see supplementary material for details). The fifth species (brown trout) had variation in the ontogenetic scaling of SMR introduced through restricted feeding and associated adjustment of SMR, as already detailed above, with little effect on the ontogenetic scaling of growth rate. These observations suggest that the ontogenetic development (scaling) of SMR as organisms grow larger co-vary with the ontogenetic development of growth rate whenever variation is generated in one or the other, either experimentally or due to inherent variation between species (Fig. 3). This also means that studies on cohorts of fish from the same treatment group might not detect small differences in scaling of biological rates. As all fish used in our study were fasted prior to respirometry measurement, for at least the 12-24 h sufficient to halt new tissue production (Wieser, 1994), the positive correlations between the

ontogenetic scaling of SMR (b_{SMR}) and that of growth rate (b_{GR}) cannot simply be attributed to the direct cost of growth being inadvertently included in measurements of SMR.

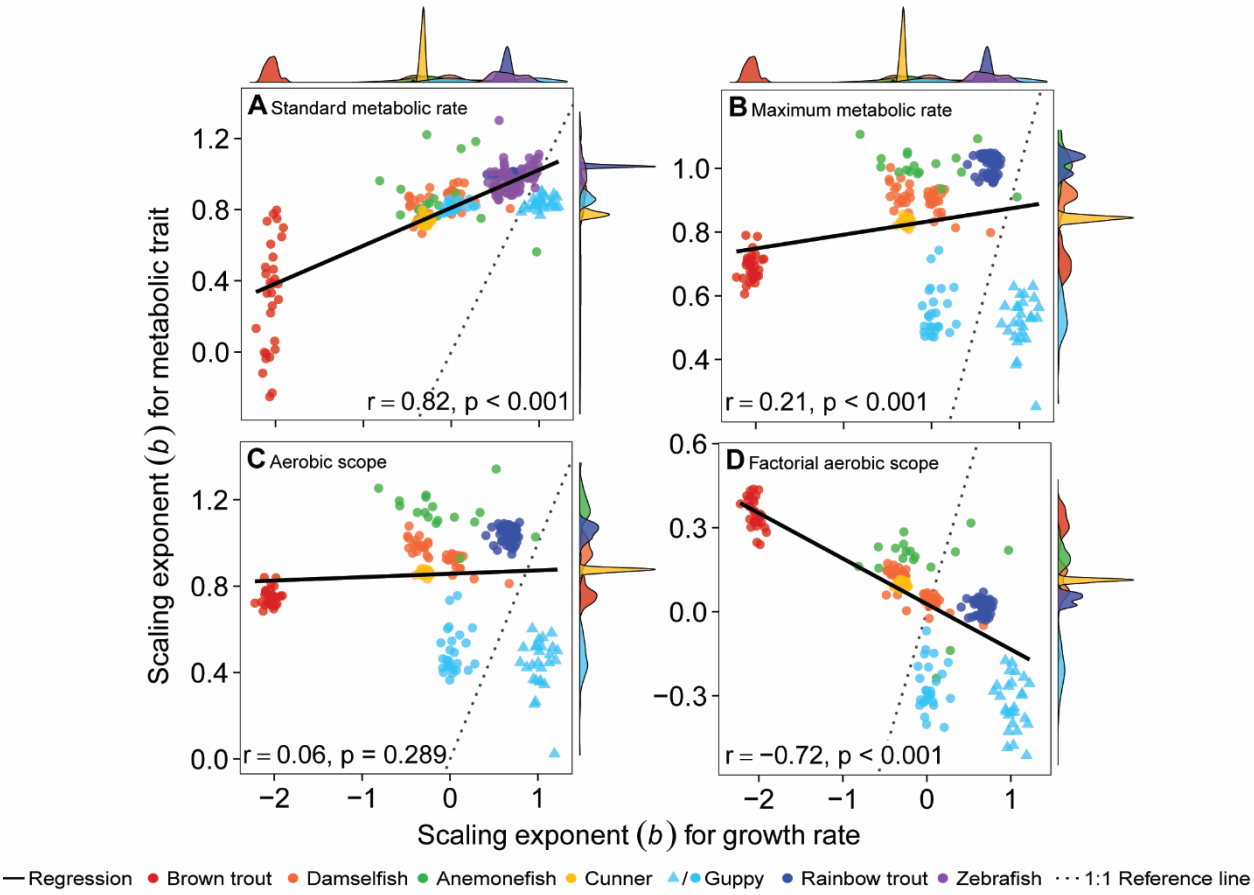

Figure 3. Correlations between ontogenetic (within-individual) scaling exponents (*b*) for metabolic traits and those for growth rate (GR); (**A**) standard metabolic rate (SMR), (**B**) maximum metabolic rate (MMR), (**C**) aerobic scope (AS), and (**D**) factorial aerobic scope (FAS) of up to seven fish species (MMR, and thus AS and FAS, was not measured for zebrafish). Each point is the model-predicted ontogenetic scaling exponent of an individual fish. The dotted grey line is a 1:1 reference line. The solid black lines are overall linear regressions for (A) $b_{SMR} = 0.81 + 0.22 \cdot b_{GR}$, (B) $b_{MMR} = 0.84 + 0.04 \cdot b_{GR}$, (C) $b_{AS} = 0.86 + 0.02 \cdot b_{GR}$, and (D) $b_{FAS} = 0.03 - 0.16 \cdot b_{GR}$. If brown trout are excluded, correlations become r = 0.67 ($p < 0.001$) for b_{SMR} *vs.* b_{GR}, r = -0.05 ($p = 0.453$) for b_{MMR} *vs.* b_{GR}, r = -0.142 ($p = 0.02$) for b_{AS} *vs.* b_{GR}, and r = -0.52 ($p < 0.001$) for b_{FAS} *vs.* b_{GR}. The marginal distribution plots on the sides show the density distribution of each species for a given scaling exponent. The overall coefficients of variation (CV) are 27.2% for SMR, 21.4% for MMR, 25.4% for AS, 575% for FAS, and 611% for GR. Plots and correlations for each individual species can be found in the supplementary material (Table S1 and Figs. 1S-5S). Guppies show strong sex-specific differences and are therefore highlighted as circles for males and triangles for females.

The positive relationships between the ontogenetic scaling of SMR and the ontogenetic scaling of growth rate supports the notion that biological scaling is malleable and not constrained by geometry or physics to a canonical value of 0.75 (or any other specific value). Additionally, the greater variation we observe in the ontogenetic scaling of growth rate compared to that for metabolic rates (Fig. 3) challenges the prevailing assumption in influential growth models (Bertalanffy, 1957; Hou et al., 2008; West et al.,

2001) and the metabolic theory of ecology (Brown et al., 2004) that growth is governed by metabolic rate and the two should scale the same. While we are hesitant to draw a conclusion regarding causality, variation in the ontogenetic scaling of SMR (b_{SMR}) appear more constrained than that for growth rate (b_{GR}). This constraint is further supported by the substantially higher variance in the ontogenetic scaling exponents for growth rate compared to those for SMR, MMR, and AS (Fig. 3).

Contrary to the patterns observed for the ontogenetic scaling of SMR, we find a strong negative correlation between the ontogenetic scaling of factorial aerobic scope (b_{FAS}) and that of growth rate (b_{GR}), with an intercept close to zero (Fig. 3D). Thus, when growth rate remains constant as body mass increases during ontogeny ($b_{GR} = 0$), relative metabolic scope also remains constant ($b_{FAS} = 0.03$). However, when growth rate decreases during ontogeny ($b_{GR} < 0$) relative metabolic scope increases ($b_{FAS} > 0$), while increasing growth rate decreases relative metabolic scope. As FAS is the factorial difference between MMR and SMR, this means that the relative increase in SMR out-scales that of MMR when growth rate increases with increasing body mass during ontogeny, and *vice versa* when growth rate decreases. This negative relationship between the ontogenetic scaling of FAS (b_{FAS}) and that of growth rate (b_{GR}) reveals a trade-off between growth and metabolic scope across fishes, with faster growing individuals experiencing a shallower increase in relative aerobic capacity with increasing body mass compared to their slower-growing counterparts. Interestingly, if brown trout are disregarded (because of their unsustainable temporal down-regulation of SMR under restricted food availability), the correlations between the ontogenetic scaling of growth rate and the ontogenetic scaling of both SMR and FAS (Figs. 3A and 3D) remain strong and maintain direction (positive and negative, respectively). However, the correlation between the ontogenetic scaling of AS and that of growth rate changes from being positive and non-significant to being significantly negative, whereas the correlation between the ontogenetic scaling of MMR and that of growth rate becomes non-significantly negative (Fig. 3). Similar patterns also exist within species, where the ontogenetic scaling of growth rate either shows no or significantly negative correlations with both the ontogenetic scaling of MMR and the ontogenetic scaling of AS (Figs. S2 and S3). Overall, these generally negative relationships between the ontogenetic scaling of growth rate and those of MMR, AS, and FAS underscore a trade-off between growth and the development of overall aerobic metabolic capacity during ontogeny, both within and across fish species. The trade-off between accelerating ontogenetic growth (steep ontogenetic scaling of growth rate) and reduced metabolic scope with increasing size is likely to have fitness consequences, as metabolic scope has been positively linked to offspring size and lifetime reproductive success (Dunsworth et al., 2012; Eliason et al., 2011).

Static scaling of metabolic and growth rates and relationships with ontogenetic scaling

We did not find an overall difference between static and ontogenetic scaling of any of the metabolic traits (SMR, MMR, AS and FAS) for all species together (Table 1). This contrasts with earlier findings by Norin (2022) for the brown trout and cunner, which have steeper static than ontogenetic scaling of SMR (Table S1). Norin (2022) proposed that selection for faster-growing individuals could result in populations with steeper static compared to ontogenetic scaling of SMR, due to the positive correlation

between the ontogenetic scaling of SMR and growth rate also found in the present study. The lack of consistently steeper static compared to ontogenetic scaling across all seven species investigated here may reflect differences in historical selection pressures, as some species were wild-caught while others originated from hatcheries or laboratory populations (see supplementary material).

The scaling of growth rate, on the other hand, was significantly different between the ontogenetic and static levels both overall and within several species, with generally (much) shallower ontogenetic than static scaling (lower ontogenetic than static *b*; Fig. 1, Table S1). These differences between ontogenetic and static scaling reveal that growth rate can increase steeply with increasing body mass *across* individuals within a given cohort or age class (positive static scaling) while increasing shallowly or even decreasing with increasing body mass (negative ontogenetic scaling) *within* the same individuals across ontogeny. This highlights how completely different and even opposing conclusions about scaling relationships can be reached depending on the level of investigation.

Conclusions

We find that the ontogenetic development (scaling) of growth rate co-varies positively with the ontogenetic development of maintenance metabolic demands (SMR) and negatively with the ontogenetic development of metabolic scope (FAS). The severity of these trade-offs between fast growth and both elevated maintenance costs and reduced metabolic scope likely depends on the environment; a steeply increasing SMR with increasing body mass may be benign or even advantageous in resource-rich and predictable environments, where higher food intake could compensate for the increased maintenance energy expenditure and allow for faster meal processing (Millidine et al., 2009) and more rapid growth (present study; Metcalfe et al., 2016). Such rapid ontogenetic growth would allow individuals to escape size-selective mortality (Sogard, 1997) but the associated cost of a dwindling metabolic scope could have critical negative consequences for fitness of fish and other animals alike, as metabolic scope has been tied to swimming performance (Pang et al., 2021), resistance to a changing environment (Fu et al., 2022), lifetime reproductive success (Eliason et al., 2011), and offspring size (Dunsworth et al., 2012). Accelerating growth also commonly occur as a compensatory response in individuals having experienced a period of slow growth caused by unfavourable environmental conditions (Metcalfe & Monaghan, 2001). In addition to escaping size-selective mortality, compensatory growth allows small individuals to catch up in size with conspecifics that have been less affected by the environmental dearth, but it decreases later-life health and lifespan (Barker, 2004; Lee et al., 2013; Metcalfe & Monaghan, 2001). Differences in environmental selection pressures could thus drive scenarios where accelerating ontogenetic growth (steeper scaling of growth rate) would be favourable even at the cost of increased maintenance demands (SMR) and reduced metabolic scope (FAS).

Our findings challenge the very influential idea of universal ¾-power scaling (West et al. 1997, 1999), as well as the equally influential idea outlined in the metabolic theory of ecology that metabolic rate governs growth such that the two should scale the same (Brown et al., 2004, 2018; Hou et al., 2008; West et al., 2001). Instead, we find both broad and more pronounced variation in the ontogenetic scaling

of growth rate compared to that of metabolic rates, especially SMR. Thus, we show that neither metabolic nor growth rate scaling is fixed but rather covaries with each other. The positive relationship between ontogenetic scaling of SMR and that of growth rate also contrasts with recent life-history optimisation theory, which predicts a negative relationship between ontogenetic metabolic scaling and growth (White et al., 2022). However, while the prediction by White et al. (2022) is made at the ontogenetic level, it is based on empirical data at the evolutionary (across-species) level, further highlighting how different and even opposing conclusions can be reached depending on the biological level under investigation. Finally, our findings indicate that previously unaccounted variation in growth could help explain the century-long debate about whether, and to what extent, metabolic scaling varies.

Materials and methods

Fish species and holding conditions

We collected empirical data on body mass and oxygen uptake rates as estimates of metabolic rates from 389 individuals across seven fish species. All individuals were kept at constant temperatures within the normal range for their species: brown trout (*Salmo trutta*) at 15°C, rainbow trout (*Oncorhynchus mykiss*) at 10°C, cunner (*Tautogolabrus adspersus*) at 15°C, blue-green damselfish (*Chromis viridis*) at 29°C, orange-fin anemonefish (*Amphiprion chrysopterus*) at 28°C, Trinidadian guppy (*Poecilia reticulata*) at 25°C, and zebrafish (*Danio rerio*) at 28°C. Data for brown trout and cunner were originally collected by Norin and Malte (2011) and Norin and Gamperl (2018), respectively. For these species, individuals were housed in a common tank and tagged for individual identification with either passive integrated transponders (brown trout) or elastomers (cunner). Individuals from the other five species were housed separately in individual tanks or containers.

Data collection and preparation

Each individual fish was measured repeatedly for metabolic rate and body mass up to twelve times during ontogeny. Individuals with fewer than four repeated measurements of both metabolic rate and body mass were excluded from analysis. This produced a final dataset comprising 4324 paired measurements of body mass and metabolic rates, with 2449 measurements of standard metabolic rate (SMR) and 1875 measurements of maximum metabolic rate (MMR), from which scaling relationships were derived as outlined below.

Metabolic rates were measured as oxygen uptake rates (mg O_2 h^{-1}) using respirometry. Prior to the start of every respirometry trial, fish were fasted for 12-24 h to ensure digestion would not interfere with metabolic rate measurements. SMR was measured for all species by continuously recording oxygen uptake rates overnight in an intermittent-flow respirometer, as described in Killen et al. (2021). The mean of the lowest 10% of these oxygen uptake rates was considered SMR. For guppies, MMR was measured as the highest oxygen uptake rate during exhaustive swimming in a small swim flume respirometer, as in Hejlesen et al. (2024). For the remaining species, MMR was measured as the oxygen uptake rate immediately after an exhaustive chase using a standard chase protocol, followed by transferring fish to an

intermittent-flow respirometer (Norin and Clark, 2016). We did not measure MMR for zebrafish. Aerobic scope (AS) was calculated as the absolute difference between MMR and SMR for each fish, while factorial aerobic scope (FAS) was calculated as the ratio of MMR to SMR.

Growth rate (mg day^{-1}) was calculated for each individual fish between each pair of two consecutive body mass measurements as the increase in body mass (mg) divided by the time interval (days) between measurements. Each body mass measurement was performed by placing the fish on a fine mesh and lightly dapping them with absorbent paper to remove adhering water. To quantify the ontogenetic scaling of growth rate, each growth rate value was associated with the mean body mass between each consecutive pair of measurements.

Statistical analysis

All data analyses were performed in R v 4.2.2 (R Core Team, 2021). Data and R script will be provided in the supplementary material upon publication.

Ontogenetic and static scaling of each trait for each species were analysed using MCMCglmm models (Hadfield 2010) as $\log_{10}(Trait) \sim \log_{10}(MassCent) + \log_{10}(MassMean) + (\log_{10}(MassCent)|FishID)$, where $Trait$ is metabolic or growth rates, $MassCent$ is the centred body mass of each individual, $MassMean$ is the mean body mass of each individual repeated the number of times that individual was measured, and $FishID$ is a unique ID for each fish. This model structure allowed us to analyse the ontogenetic (within-individual) and static (across-individual) scaling relationships simultaneously (van de Pol & Wright, 2009) and extract model-predicted scaling exponents (b) for individuals and species for further analysis and graphical presentation. For species where a treatment was included in the experiment (see supplementary material), this was accounted for with fixed-effects interactions between Treatment and both $\log_{10}(MassCent)$ and $\log_{10}(MassMean)$. The average scaling exponent between the treatments was then calculated using inverse variance weighting, taking into account the covariation between treatments. Mean ontogenetic or static scaling exponents for all species were calculated as inverse variance weighted averages of model-predicted values.

To account for differences in temperature between species for graphical presentations (Fig. 1), all measurements were standardised to a common temperature of 20°C. This was done by running an MCMCglmm model containing all measurements for a given trait and including temperature as a fixed effect, as $\log_{10}(Trait) \sim \log_{10}(Mass) + Temperature + (\log_{10}(Mass)|FishID)$. The effect of temperature was then calculated as Q_{10}, and all measurements were up- or down-regulated using the Q_{10} for that given trait to the common temperature of 20°C.

Comparisons between scaling exponents

Statistical comparisons between average scaling exponents were done by evaluating if there was overlap between the 95% CIs. Covariation between ontogenetic scaling exponents were evaluated using Pearson's correlation coefficients (r), with p-values below 0.05 considered significant.

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Supplementary methods

Fish origins

The fish used in this study originated from different sources. Damselfish were wild-caught as juveniles on the coral reef around the island of Moorea, French Polynesia. Among the anemonefish, three were wild-caught juveniles from the coral reefs around Moorea, while the remaining fifteen were lab-reared from eggs collected in the lagoons of Moorea or Tahiti, French Polynesia. The cunner and brown trout were first-generation hatchery-raised offspring of wild-caught adults from Canada (Newfoundland) and Denmark, respectively. Zebrafish and guppies were originally wild-caught in India and Trinidad, respectively, but kept in the lab for several generations. Rainbow trout were obtained from a commercial hatchery in Denmark as eggs.

Experimental treatments

Damselfish and zebrafish were maintained at different food levels. Damselfish were split into two groups, one fed twice as much as the other at each feeding. Zebrafish were split into three groups fed once, twice, or three times a day with the same amount at each feeding. Rainbow trout were exposed to different temperature treatments in the egg or yolk sac stage, with either a constant 10°C, 14°C during the egg stage only, or 14°C during the yolk sac stage only; all fish were subsequently kept at 10°C and metabolic and growth rates measured at that temperature. Guppies did not receive any experimental treatment but show strong sex-specific growth differences and thus cluster in two groups (males and females). Cunner, brown trout, and anemonefish were not exposed to different experimental treatments, although the brown trout were maintained at a restricted food ration at 34-39% of satiation rations (Norin and Malte 2011).

Table S1. Species-specific ontogenetic ('Onto') and static ('Static') scaling exponents (with 95% CIs in square brackets) for standard metabolic rate (SMR), maximum metabolic rate (MMR), aerobic scope (AS), factorial aerobic scope (FAS), and growth rate (GR). MMR (and thus AS and FAS) was not measured for zebrafish.

		Species							
Trait	Level	Zebrafish	Rainbow trout	Guppy	Damselfish	Brown trout	Cunner	Anemonefish	Overall
SMR	Onto	0.98 [0.89, 1.06]	1.00 [0.97, 1.03]	0.83 [0.80, 0.87]	0.85 [0.76, 0.94]	0.27 [0.09, 0.45]	0.74 [0.70, 0.77]	0.88 [0.57, 1.17]	0.87 [0.85, 0.88]
SMR	Static	1.01 [0.93, 1.08]	1.00 [0.91, 1.08]	0.85 [0.76, 0.93]	0.70 [0.52, 0.87]	1.12 [0.84, 1.38]	0.87 [0.83, 0.09]	0.78 [0.38, 1.17]	0.89 [0.87, 0.92]
MMR	Onto		1.03 [1.01, 1.05]	0.52 [0.47, 0.57]	0.89 [0.82, 0.97]	0.69 [0.60, 0.78]	0.83 [0.81, 0.86]	1.01 [0.82, 1.20]	0.89 [0.88, 0.91]
MMR	Static		0.95 [0.88, 1.02]	0.72 [0.65, 0.79]	0.82 [0.62, 1.01]	0.78 [0.61, 0.97]	0.94 [0.91, 0.97]	0.74 [0.46, 1.02]	0.90 [0.87, 0.92]
AS	Onto		1.07 [1.04, 1.10]	0.45 [0.38, 0.51]	0.92 [0.82, 1.02]	0.75 [0.65, 0.85]	0.86 [0.83, 0.89]	1.14 [0.89, 1.40]	0.90 [0.88, 0.92]
AS	Static		0.92 [0.82, 1.03]	0.69 [0.61, 0.77]	0.90 [0.63, 1.16]	0.71 [0.49, 0.91]	0.96 [0.92, 1.00]	0.83 [0.46, 1.22]	0.90 [0.87, 0.94]
FAS	Onto		0.03 [0.01, 0.06]	-0.32 [-0.38, -0.26]	0.06 [-0.02, 0.13]	0.35 [0.21, 0.05]	0.10 [0.06, 0.13]	0.17 [-0.17, 0.52]	0.03 [0.01, 0.05]
FAS	Static		-0.06 [-0.13, 0.02]	-0.13 [-0.24, -0.02]	0.12 [-0.06, 0.29]	-0.38 [-0.65, -0.08]	0.07 [0.04, 0.11]	0.13 [-0.34, 0.61]	0.03 [0.00, 0.06]
GR	Onto	0.82 [0.63, 1.01]	0.63 [0.58, 0.67]	0.89 [0.71, 1.06]	-0.01 [-0.38, 0.37]	-2.06 [-2.88, -1.26]	-0.29 [-0.53, -0.06]	-0.09 [-0.77, 0.54]	0.60 [0.56, 0.65]
GR	Static	1.19 [0.99, 1.4]	0.94 [0.82, 1.05]	1.18 [0.98, 1.37]	0.39 [-0.21, 0.99]	1.06 [0.21, 1.93]	1.10 [0.96, 1.25]	-0.19 [-0.79, 0.39]	1.02 [0.95, 1.09]

Table S2. Details about fish body masses and respirometry setup at the first (Mass start) and last (Mass end) of the longitudinally repeated measurements used to quantify scaling relationships. Respirometry chamber size is the volume of the respirometers used to estimate standard metabolic rate (SMR) and maximum metabolic rate (MMR). Open / closed respirometry time is the duration of the flush (open) and measurement (closed) periods of the intermittent-flow respirometry technique.

Species	Mass start (g), mean (and range)	Mass end (g), mean (and range)	Respirometry chamber size (mL)	Open / closed respirometry time (min)
Brown trout	32.3 (20.7-45.7)	54.0 (38.4-68.2)	540 (SMR, MMR)	2 / 3.5
Rainbow trout	0.268 (0.103-1.843)	7.119 (1.744-13.97)	8.2, 22, 100.5, or 116.6 (SMR, MMR)	3 / 3-5
Cunner	1.619 (0.451-4.606)	6.383 (1.455-19.455)	65, 145, or 400 (SMR, MMR)	5 / 10-12
Zebrafish	0.004 (0.0009-0.0174)	0.0385 (0.0015-0.1387)	2.18 or 4.68 (SMR)	2 / 3
Anemonefish	0.046 (0.013-0.078)	0.1246 (0.077-0.172)	2.4, 3.14, or 11.52 (SMR, MMR)	2 / 3
Damselfish	0.0586 (0.018-0.12)	0.1705 (0.074-0.265)	2.44 or 11.8 (SMR, MMR)	2 / 1.5
Guppy	0.0174 (0.004-0.0417)	0.1188 (0.0288-0.3379)	1.9, 4.2, or 10.8 (SMR) and 27 or 78 (MMR)	4 / 5

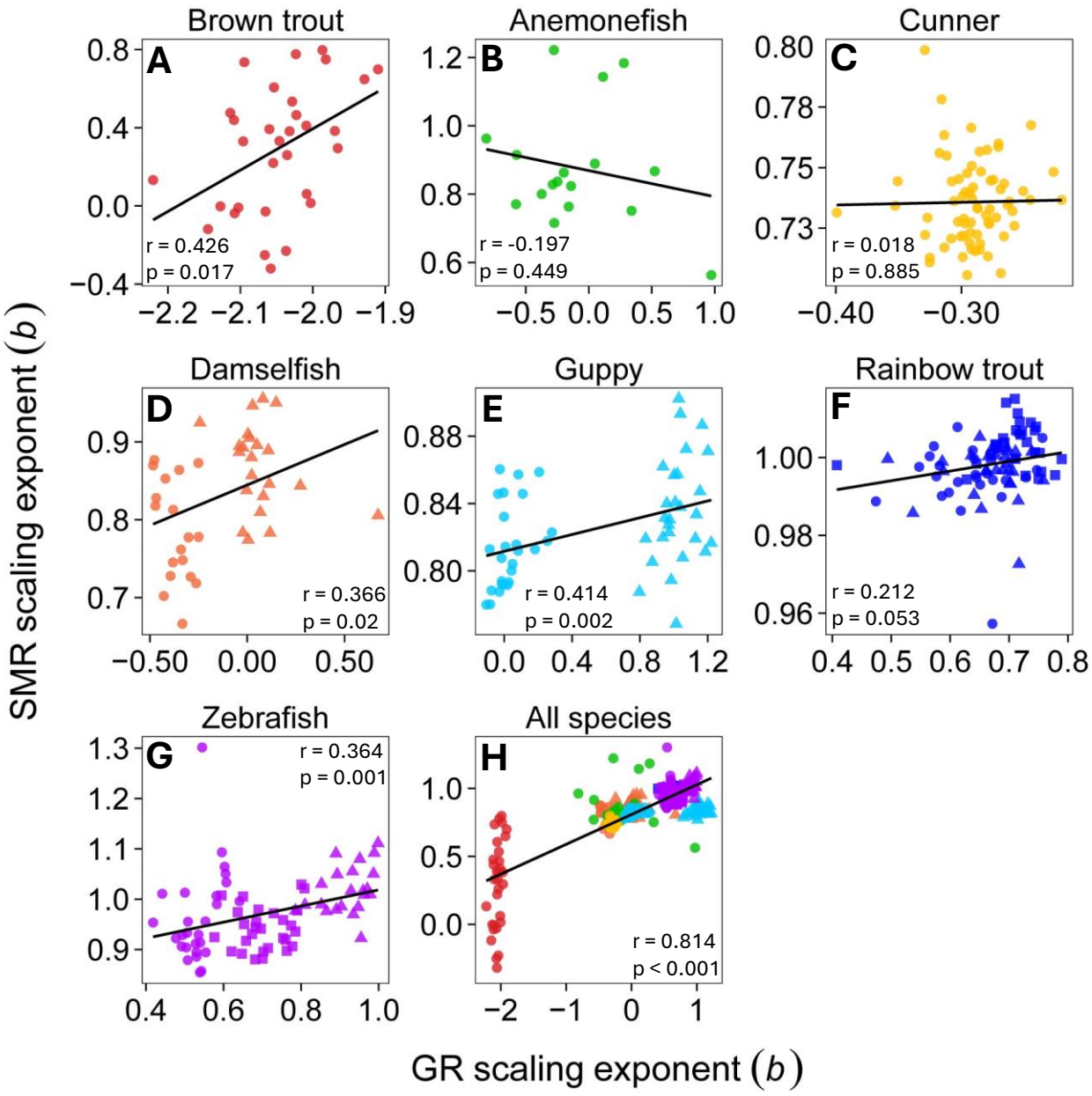

Figure S1. Correlations between ontogenetic scaling exponents (*b*) for standard metabolic rate (SMR) and those for growth rate (GR). Each point represents an individual's ontogenetic *b*. Panel **A**-**G** show correlations within species, while panel **H** shows it for all species combined, using the same colours from the species-specific plots. The solid black line is a regression line. Correlation coefficients (r) and associated p-values are Pearson's correlations. Damselfish and zebrafish were maintained at different feeding levels (circles = low, squares = moderate, triangles = high). Rainbow trout had different warming treatments during different early life stages (circles = constant 10°C, triangles = 14°C during egg stage only, squares = 14°C during yolk-sac stage only; all metabolic and growth rates were measured after the yolk-sac stage with all fish kept at a common 10°C). Guppies show strong sex-specific differences (circles = males, triangles = females).

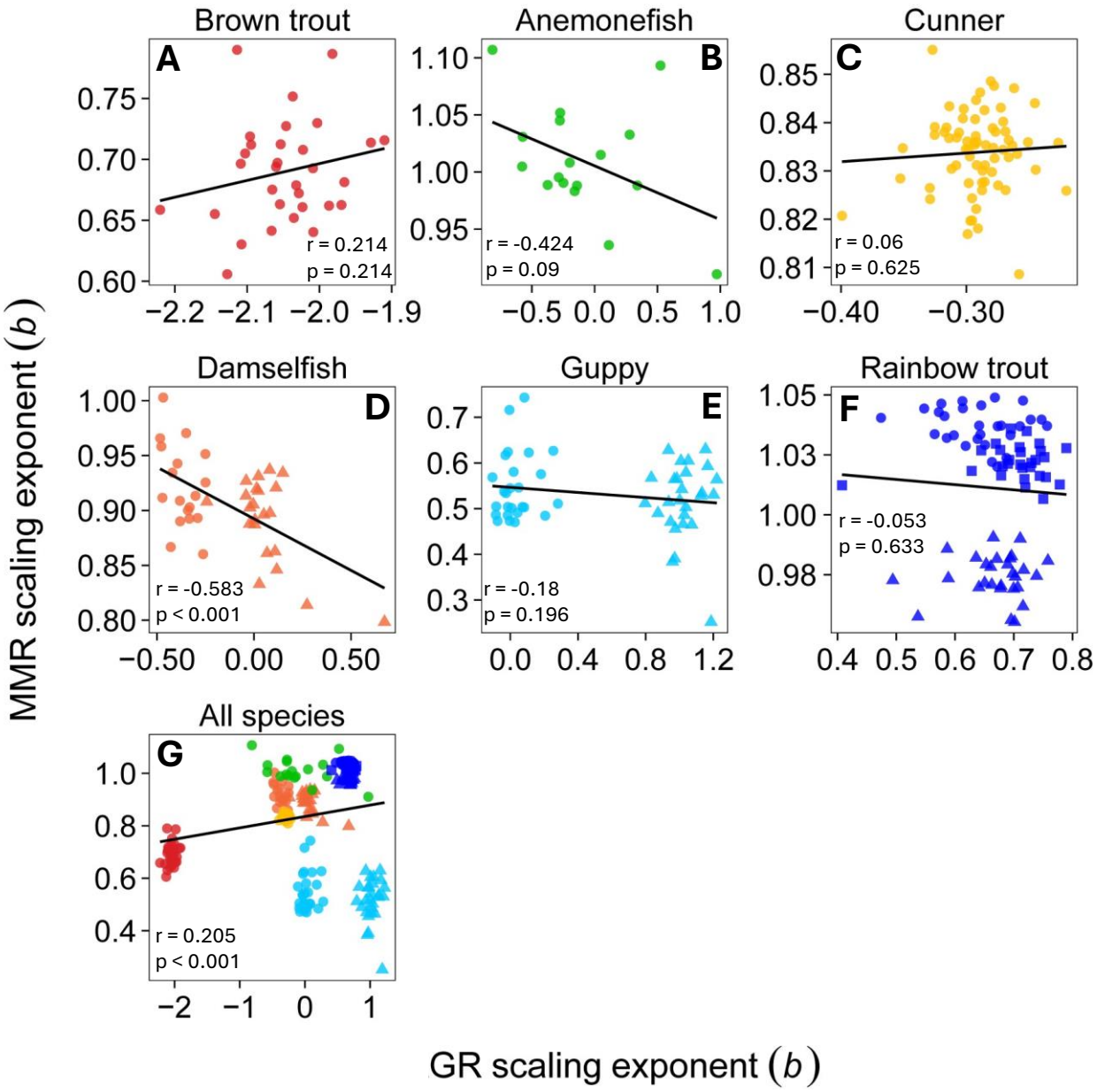

Figure S2. Correlations between ontogenetic scaling exponents (*b*) for maximum metabolic rate (MMR) and those for growth rate (GR). Each point represents an individual's ontogenetic *b*. Panel **A-F** show correlations within species, while panel **G** shows it for all species combined, using the same colours from the species-specific plots. The solid black line is a regression line. Correlation coefficients (r) and associated p-values are Pearson's correlations. Damselfish and zebrafish were maintained at different feeding levels (circles = low, squares = moderate, triangles = high). Rainbow trout had different warming treatments during different early life stages (circles = constant 10°C, triangles = 14°C during egg stage only, squares = 14°C during yolk-sac stage only; all metabolic and growth rates were measured after the yolk-sac stage with all fish kept at a common 10°C). Guppies show strong sex-specific differences (circles = males, triangles = females).

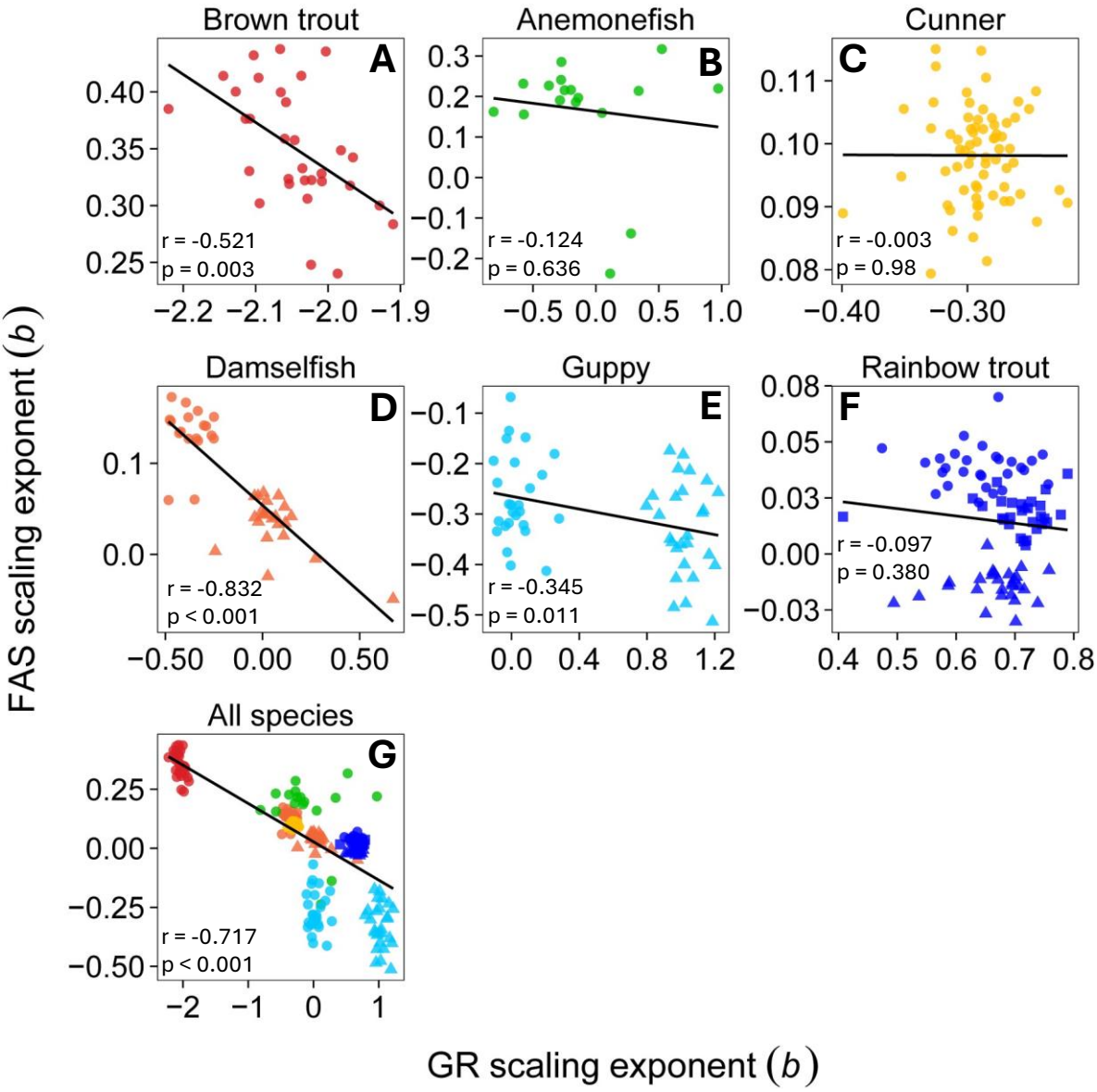

Figure S3. Correlations between ontogenetic scaling exponents (*b*) for factorial aerobic scope (FAS) and those for growth rate (GR). Each point represents an individual's ontogenetic *b*. Panel **A-F** show correlations within species, while panel **G** shows it for all species combined, using the same colours from the species-specific plots. The solid black line is a regression line. Correlation coefficients (r) and associated p-values are Pearson's correlations. Damselfish and zebrafish were maintained at different feeding levels (circles = low, squares = moderate, triangles = high). Rainbow trout had different warming treatments during different early life stages (circles = constant 10°C, triangles = 14°C during egg stage only, squares = 14°C during yolk-sac stage only; all metabolic and growth rates were measured after the yolk-sac stage with all fish kept at a common 10°C). Guppies show strong sex-specific differences (circles = males, triangles = females).

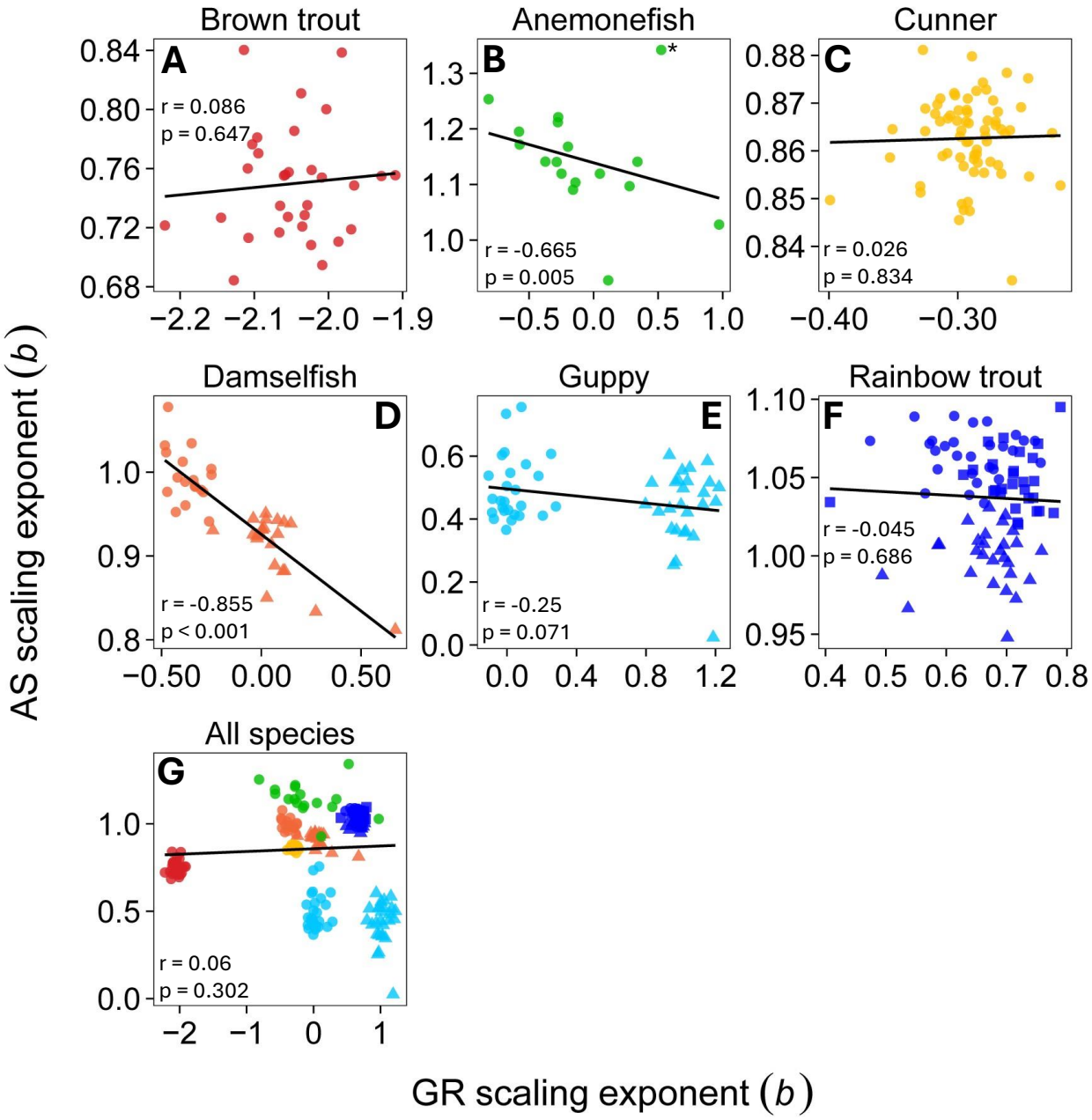

Figure S4. Correlations between ontogenetic scaling exponents (*b*) for aerobic scope (AS) and those for growth rate (GR). Each point represents an individual's ontogenetic *b*. Panel **A-F** show correlations within species, while panel **G** shows it for all species combined, using the same colours from the species-specific plots. The solid black line is a regression line. Correlation coefficients (r) and associated p-values are Pearson's correlations. Damselfish and zebrafish were maintained at different feeding levels (circles = low, squares = moderate, triangles = high). Rainbow trout had different warming treatments during different early life stages (circles = constant 10°C, triangles = 14°C during egg stage only, squares = 14°C during yolk-sac stage only; all metabolic and growth rates were measured after the yolk-sac stage with all fish kept at a common 10°C). Guppies show strong sex-specific differences (circles = males, triangles = females). For anemonefish, one point (*) was excluded from the correlation analysis following an outlier test (Cook's distance with a threshold of 0.726).