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SHORT COMMUNICATION

Nest predation and adult mortality relationships with post-natal metabolic rates and growth among songbird species

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ABSTRACT

Metabolism is thought to mediate the connection between environmental selection pressures and a broad array of life history tradeoffs, but tests are needed. High juvenile predation correlates with fast growth, which may be achieved via fast juvenile metabolism. Fast offspring metabolism and growth can create physiological costs later in life that should be minimized in species with low adult mortality. Yet, relationships between juvenile metabolism and mortality at offspring versus adult stages are unexplored. We found that post-natal metabolism was positively correlated with adult mortality but not nest predation rates among 43 songbird species on three continents. Nest predation, but not adult mortality, explained additional variation in growth rates beyond metabolism. Our results suggest that metabolism may not be the mechanism underlying the relationships between growth and mortality at different life stages.

KEY WORDS: Offspring mortality, Longevity, Metabolism, Growth rate, Songbird nestlings

INTRODUCTION

Investigating the reciprocal influences between physiological variation and sources of environmental mortality can be essential for our understanding of evolved differences in life history tradeoffs (Ricklefs and Wikelski, 2002). For example, metabolic rates are thought to underlie the slow–fast continuum in important life history traits such as offspring growth and adult longevity (Brown et al., 2004). Yet, a slow pace of life is not always accompanied by low metabolism (Bech et al., 2016). The contrasting evidence may arise from the fact that the connection between physiology and life history is interactive and that variation in metabolic rates is exposed to constraints and selection pressures among life stages (Pettersen et al., 2018). On the one hand, intrinsic processes such as metabolism and growth may impact mortality and longevity (Metcalfe and Monaghan, 2003). On the other hand, extrinsic mortality exerts selection on the evolution of phenotypic expression such as metabolism, with potential consequences for growth rates and longevity (Lovegrove, 2000; White and Kearney, 2013; Martin et al., 2015). Yet, the relationships of evolved variation among species in post-natal metabolism and growth with sources of mortality at different life stages are unexplored. Here, we document interspecific relationships of mortality during juvenile and adult life stages with resting post-natal metabolic rates (hereafter referred to as metabolism or metabolic rate) and their association with growth rates of diverse songbirds.

Predation is a major ecological source of offspring mortality, and higher predation rates underlie faster post-natal growth to reduce exposure to this risk (Remeŝ and Martin, 2002). High metabolism may evolve in species with faster growth because metabolism provides the building blocks for growth (West et al., 2001; Ton and Martin, 2016). Alternatively, fast growth that is associated with high protein synthesis may yield high metabolism (Rosenfeld et al., 2015). Independently from the direction of causality, a positive correlation is expected between high nest predation, post-natal metabolism and faster growth. Experiments within species indicate that increased predation rates trigger proximate increases in metabolism in developing stages and adults among taxa (Woodley and Peterson, 2003; Steiner and Van Buskirk, 2009; Hawlena and Schmitz, 2010). However, comparisons of metabolic rates with juvenile mortality rates across species are lacking. Examination of such relationships is important because other physiological mechanisms such as assimilation efficiency and variation in wing development may also evolve in response to predation risk during the post-natal stage (Cheng and Martin, 2012; Martin, 2014, 2015; Martin et al., 2018). These mechanisms may influence growth rate variation without necessarily affecting metabolism (McPeek, 2004; Thaler et al., 2012), but this prediction needs testing.

If predation were to increase rates of post-natal metabolism and growth in the early life stages, this may impose physiological costs that also increase adult mortality (Hulbert et al., 2007). This intrinsic component of adult mortality that arises as the consequence of physiological damages is only a part of total mortality, which also includes extrinsic elements. Interestingly, these environmental (extrinsic) sources of mortality themselves can be causes of variation in metabolism and growth (Martin et al., 2015). Indeed, long-lived species with low rates of extrinsic adult mortality should evolve strategies that minimize risks to survival (Charlesworth, 1994; Ghalambor and Martin, 2001). As a result, increases in offspring metabolic and growth rates in response to high predation should be disfavored by selection in species with low extrinsic adult mortality rates. In contrast, such physiological costs should be less important to species experiencing high extrinsic adult mortality and short longevity anyway (Martin et al., 2015).

Thus, theoretical predictions and available evidence suggest that an evolutionary conflict may arise between two sources of mortality, both acting on physiological traits at different life stages. Yet, the reciprocal influences between adult mortality, predation on juveniles, post-natal metabolic rate and growth are unexplored and the solution of this possible conflict remains unclear. Here, we provide a comparative test of the hypotheses outlined above on 43 songbird species across three continents showing remarkable interspecific variation in life history strategies along a slow–fast gradient.

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MATERIALS AND METHODS

Study area and species

We measured metabolic rates, growth rates, predation rates and probability of adult mortality in songbirds (order Passeriformes) at three sites (Table S1) encompassing substantial variation in post-natal metabolic rates. We studied 16 species in high-elevation (2300 m) mixed forest in Arizona, USA (34°N latitude), 14 species in tropical mid-elevation forest (1450–1950 m) in Malaysia (6°N), and 13 species in coastal shrubland at sea level in South Africa (described in Martin et al., 2015).

All measurements were conducted in accordance with relevant guidelines and regulations under the auspices of the University of Montana IACUC (protocol no. 059 10TMMCWRU).

Resting metabolic and growth rate measurements

We measured resting metabolic rate (RMR). We chose RMR because it represented the situation for the major portion of time for nestlings; they rest overnight, and are relatively inactive during the day. We recorded oxygen consumption ($\dot{V}_{O_2}$; ml h^{-1}) at 39.0°C, the typical body temperature of songbird offspring in the field (Ton and Martin, 2016). Recordings were performed on 361 nestlings in an open flow respirometry system using a Foxbox field gas analyzer (Sable Systems, Las Vegas, NV, USA). Our sample size varied between 2 and 13 (mean±s.e.m.=8.4±0.45 nestlings; see Table S1). We exposed all our samples to metabolic recordings along a 2°C step gradient (31–41°C) and found that minimal oxygen consumption occurred between 37 and 39°C. Because our recordings of inner body temperature for our nestlings averaged 38.95±0.06°C when they were extracted from their nest (Ton and Martin, 2016), we selected 39.0°C as the appropriate temperature for comparison.

Nestlings were removed from their nest, inserted in a jar filled with cotton, and transported to station within the next 5 to 40 min. Measurements occurred between 11:00 and 17:00 h to limit variation in metabolic rates owing to possible influences of circadian rhythms, such as ambient temperature and rates of food delivery. Measurements were made on only one nestling per clutch at pin break, a shared developmental stage when primary feathers break their sheaths. This stage is ideal for standardization because it is when growth rate is maximal and thermoregulatory capacities are achieved and comparable among species (Marsh, 1979; Sogge et al., 1991; Pereyra and Morton, 2001; Cheng and Martin, 2012). After being weighed, nestlings were placed in a cup-shaped mesh simulating a nest that allowed airflow while also constraining the mobility of the sample. The cup was inserted in a 2.3 liter stainless-steel airtight chamber within a dark temperature-controlled cabinet. The chamber was flushed with 200–300 ml min^{-1} flow of atmospheric air scrubbed of CO_2 and water vapor using soda lime, magnesium perchlorate and drierite (Lighton, 2008). Chicks were exposed to temperatures from 31 to 41°C in 2°C steps starting at 31°C as part of a broader examination of temperature effects not reported here. Consequently, $\dot{V}_{O_2}$ was typically measured for 39.0°C at approximately 120 to 180 min after the nestling was removed from the nest. Given the timing of our measurements, and the fast energy turn over typical of these early stages, we assumed our samples were post-absorptive when oxygen was recorded. At the end of the process, each nestling was fed commercial food for growing birds and returned to the original nest.

$\dot{V}_{O_2}$ was measured every 0.5 s until a plateau (most stable oxygen consumption) was reached and maintained for at least 10 min. A plateau was normally achieved between 12 and 31 min after exposure to 39°C (mean±s.e.m.=15.28±0.29 min). We also recorded oxygen baseline values for approximately 5 min before, during and after the experiment by temporarily disconnecting the chamber from the oxygen analyzer. These baselines were later used to correct for potential drift in ambient O_2 during measurements and thus maximize the accuracy of our estimates.

Because RMR needs to be recorded at a resting state, we monitored the regularity of the oxygen consumption trace on a laptop to assess activity level and stress. Samples that showed frequent spikes in oxygen consumption owing to movement or heat stress were excluded from analyses, but this occurred very rarely at 39°C. $\dot{V}_{O_2}$ (ml O_2 h^{-1}) was calculated as the O_2 concentration value observed during the most stable 5 min of oxygen consumption within the plateau using ExpeData software (version 1.3.2) from Sable Systems. We used the formula $\dot{V}_{O_2}=FR_i(F_{IO_2}-F_{EO_2})/(1-F_{EO_2})$, where FR_i is the incurrent mass flow rate scrubbed from water vapor and CO_2, F_{IO_2} is the incurrent fractional concentration of oxygen, and F_{EO_2} is the excurrent fractional concentration of oxygen (Lighton, 2008; see Ton and Martin, 2016 for more details).

Growth rates were estimated for all species by measuring nestling body mass at the same time (±1 h) every day for the first 3 days after hatch and then every other day until fledging (Martin et al., 2015). We used a GemPro 250 portable electronic scale (MyWeigh, Phoenix, AZ, USA) with an accuracy of ±0.001 g. We calculated the growth rate constant (k) for each species using the logistic curve (Ricklefs, 1968; Remeŝ and Martin, 2002). Sampling was extensive and lasted between 5 and 15 years depending on geographic site (Martin et al., 2015).

Predation rates and adult mortality probability

We located and monitored large numbers of nests (Table S1) for 28 years in Arizona (1987–2014), 6 years (2009–2014) in Malaysia and 5 years (2000–2004) in South Africa to obtain robust estimates of juvenile mortality rates owing to nest predation. We calculated daily predation rates during the post-natal period with the logistic exposure method (Shaffer and Burger, 2004).

In Arizona and Malaysia, we obtained annual adult mortality probability estimates by deploying 10 or 12 nets for 6 h starting at dawn as subplots within and across all nest-searching plots. These subplots were sampled three times per season, with 20–25 days between visits (Martin et al., 2015). Descriptions of netting methods for the South Africa site are available in Lloyd et al. (2014). All birds captured were banded with numbered metal rings and three color bands, resulting in unique combinations of two bands per leg. Color-bands were used for re-sighting by nest-searchers visiting their plots daily, or by netters in case of re-captured birds. We obtained estimates of annual survival probability (Φ) using capture–recapture and re-sighting data in program RMARK (Laake, 2013; see Martin et al., 2015).

Statistical analysis

We obtained mean estimates of metabolic rate for each species from a linear mixed effects model that used our $\dot{V}_{O_2}$ measurements at 39°C as the dependent variable, species as a fixed factor and individual nest identity as a random effect. To account for phylogenetic effects, we sampled 1000 trees from www.birdtree.org (Jetz et al., 2012) using the Hackett backbone (Hackett et al., 2008) and produced a majority-rules consensus tree (see Fig. S1) using the program Mesquite (http://www.mesquiteproject.org/). We conducted phylogenetic generalized least squares (PGLS) analyses using the caper package in R (Orme, 2018).

We used PGLS to test the ability of predation and adult mortality probabilities, along with mean body mass at the time of metabolic measurements and growth rate (k) to explain variation in mean

Journal of Experimental Biology (2020) 223, jeb226563. doi:10.1242/jeb.226563

$\log_{10}$-transformed post-natal metabolic rates ($\dot{V}_{O_2}$ at 39°C). Similarly, we tested whether predation rate, adult mortality probability, post-natal metabolic rate, and body mass explained variation in offspring growth rates. All combinations of two- and three-way interactions were tested among our factors, but were dropped because they were not significant in the full model. We did not include site as a factor because it did not improve the fit of any of our models. Also, including site would control average geographic differences in adult mortality and metabolism among our species and prevent us from testing correlations of adult mortality and metabolic rate across sites, which was one of the main goals of our study. All statistical analyses were performed in R v.3.0.3 for Macintosh (R Development Core Team 2014, Vienna, Austria). R codes for all our models are reported in Script 1.

RESULTS

Post-natal metabolic rates among species exhibited a strong allometric relationship with nestling mass (Fig. 1A) and growth (Fig. 1B), even after accounting for predation and adult mortality (Table 1). Nonetheless, predation showed no correlation with metabolism (Fig. 1C), while adult mortality probabilities were positively correlated with post-natal metabolic rates after correcting for mass and growth (Fig. 1D, Table 1).

Post-natal metabolism was positively correlated with post-natal growth as expected, but predation and body mass explained additional variation (Fig. 2, Table 1). In contrast, adult mortality did not explain additional variation in growth beyond metabolism, mass and predation (Table 1).

DISCUSSION

The relationships among ecological sources of mortality at different life stages and variation in metabolism and growth are undocumented. Here, we found the well-established results that mass and growth rates are strong correlates of metabolic rates (Brown et al., 2004; Ton and Martin, 2016). However, adult mortality explained additional variation in offspring resting metabolism after controlling for mass (Fig. 1). Nest predation instead showed no correlation with metabolism (Fig. 1C, Table 1), in contrast to experiments reporting proximate metabolic increases within species in response to increased predation (Woodley and Peterson, 2003; Steiner and Van Buskirk, 2009; Hawlena and Schmitz, 2010). Given that offspring predation rates are strongly

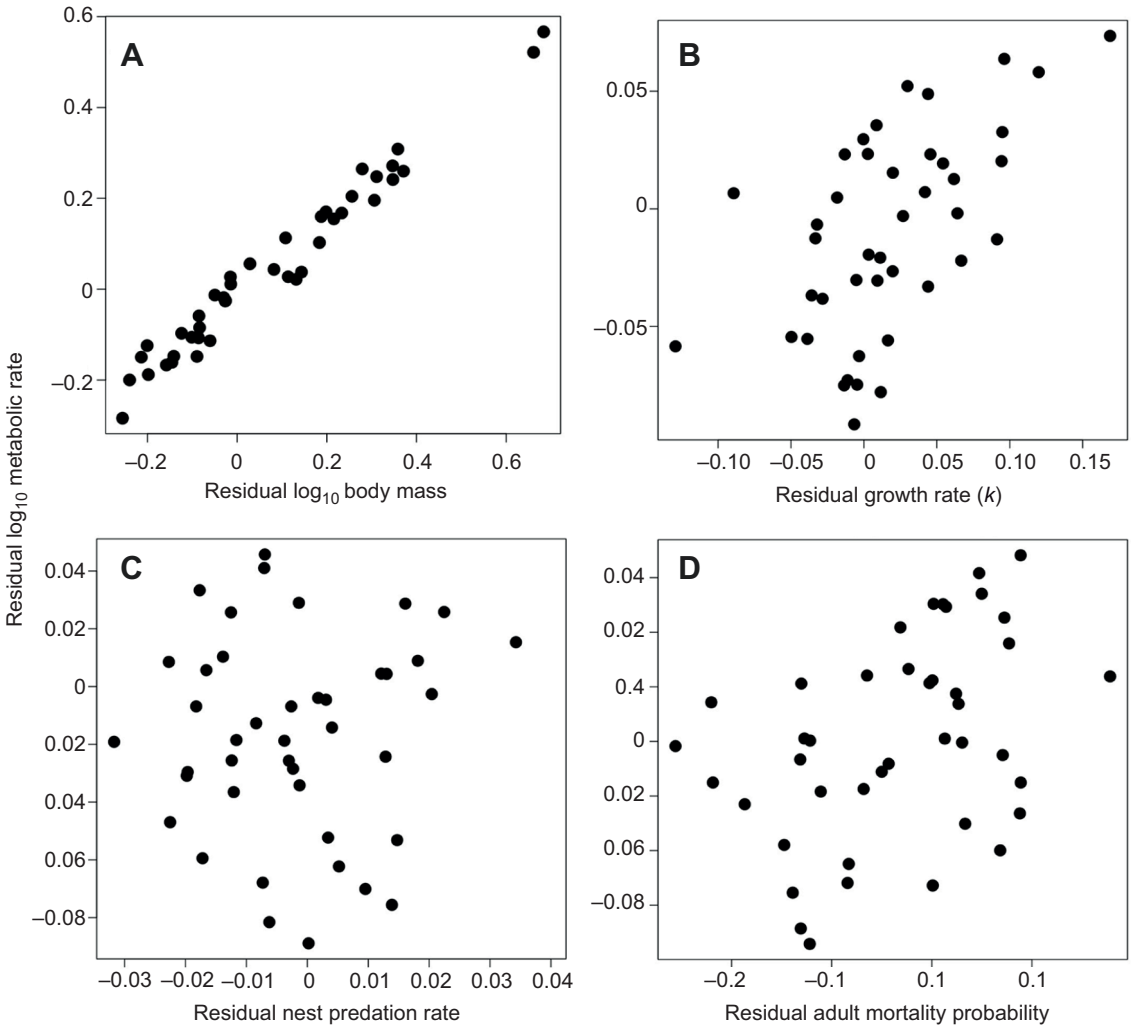

Fig. 1. Partial regression plots of metabolic rate (ml O_2 h^{-1} at 39°C) as a function of life history tradeoffs. (A) Body mass after accounting for growth rate, nest predation and adult mortality probability, (B) growth rate after accounting for adult mortality probability nest predation and body mass, (C) nest predation after accounting for growth rate, body mass and adult mortality probability and (D) adult mortality probability after accounting for growth rate, nest predation and body mass. Each point represents a species.

Journal of Experimental Biology (2020) 223, jeb226563. doi:10.1242/jeb.226563

Table 1. Linear models representing the relationship between post-natal metabolic rate (ml O_2 h^{-1} at 39°C) and nest predation, adult mortality probability, body mass and growth rate, and between offspring growth rate (*k*) and nest predation, adult mortality probability, metabolic rate and body mass for 43 species of songbirds

Dependent variable	Independent variable	β (s.e.)	*F*	d.f.	*P*
Post-natal metabolic rate (R^2=0.95)	Body mass	0.821 (0.023)	1211	1	<0.001
	Nest predation	0.058 (0.341)	0.028	1	0.865
	Adult mortality	0.139 (0.057)	5.9	1	0.019
	Growth rate	0.445 (0.098)	20.52	1	<0.001
	Error			38	
Offspring growth rate (R^2=0.56)	Body mass	−0.665 (0.144)	21.16	1	<0.001
	Metabolic rate	0.738 (0.175)	17.55	1	<0.001
	Nest predation	0.959 (0.42)	5.19	1	0.028
	Adult mortality	0.074 (0.081)	0.828	1	0.367
	Error			38	

predictive of growth rates across songbird species (Remeŝ and Martin, 2002; Martin et al., 2015), and that offspring metabolism is a reasonably strong predictor of growth rates (Fig. 2B), the absence of a relationship between offspring predation rates and post-natal metabolic rates (Fig. 1B, Table 1) was surprising. These results suggest that proximate and ultimate dynamics linking predation and metabolism do not necessarily mirror each other. Our findings also suggest that adult mortality may have an overriding effect on selection by predation targeting metabolism. Additionally, they may indicate that predation acts on growth via other pathways for which metabolism may be simply a correlate.

We found that nest predation explained additional variation in growth rates beyond metabolism and mass (Fig. 2, Table 1). Differential development of other traits such as endothermic

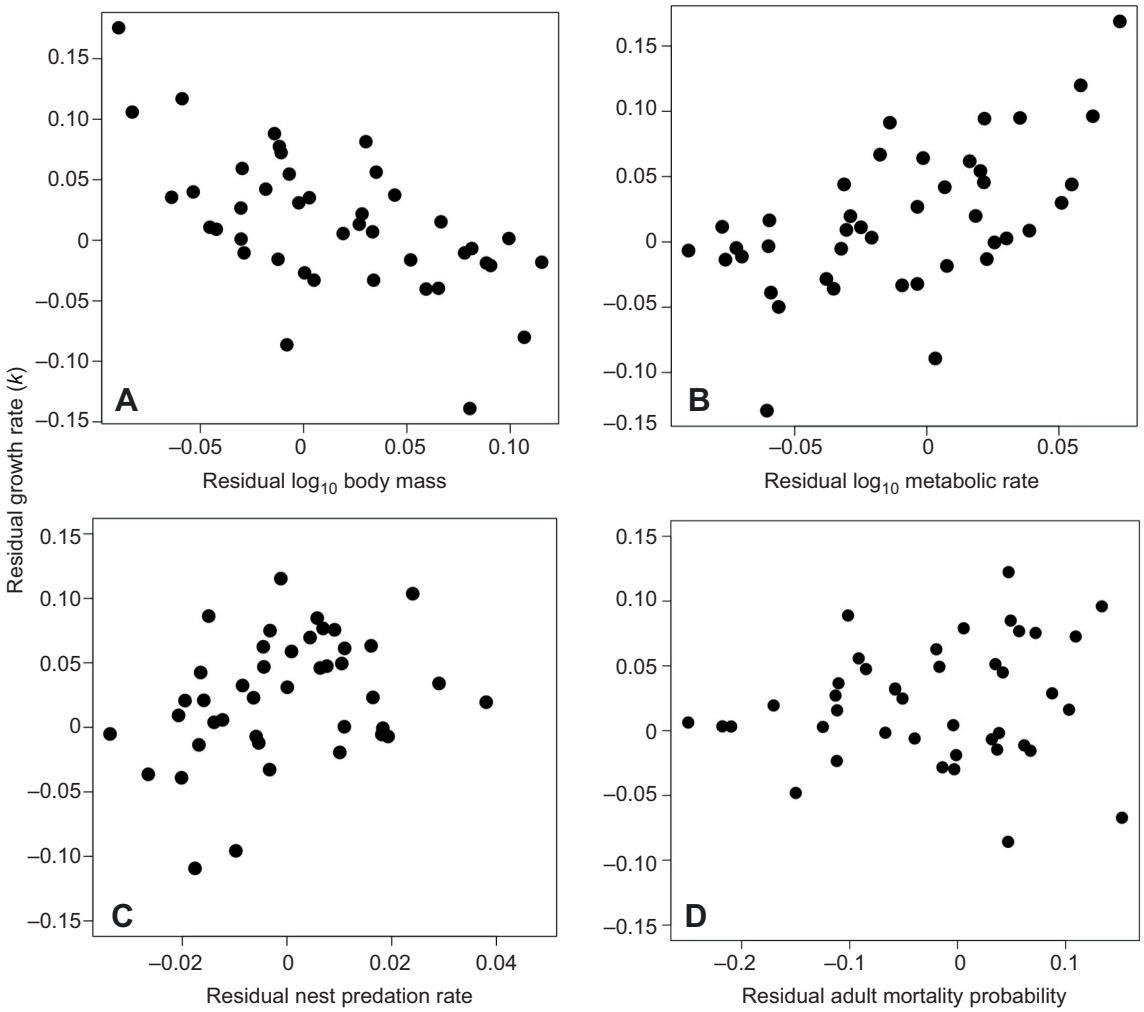

Fig. 2. Partial regression plots of growth rate (*k*) as a function of life history tradeoffs. (A) Body mass after accounting for nest predation, adult mortality probability and metabolic rate, (B) metabolic rate after accounting for nest predation, adult mortality probability and body mass, (C) nest predation after accounting for adult mortality probability, metabolic rate and body mass, and (D) adult mortality probability after accounting for nest predation, metabolic rate and body mass. Each point represents a species.

capacity and wings vary with offspring predation risk and explain variation in growth rates (Cheng and Martin, 2012). In addition, differential assimilation efficiency also can vary with predation risk and influence growth rates (McPeek, 2004; Thaler et al., 2012). Such alternative developmental pathways may provide an important response to nest predation because they also influence offspring survival after leaving the nest (Martin, 2014, 2015; Martin et al., 2018), whereas metabolic responses may have detrimental consequences for adult survival.

Higher metabolic rates have long been thought to reduce survival and longevity (Rubner, 1908; Hulbert et al., 2007). Classic studies on this subject normally measure metabolism in domestic organisms under laboratory settings and consider maximum longevity, raising questions about applicability to wild organisms, but they suggest intrinsic (physiological) processes can contribute to adult mortality (Hulbert et al., 2007). Any such intrinsic costs of metabolism may provide a possible explanation for our results, as found for adult birds of the same species (Boyce et al., 2020). In addition to this classic view, life history theory and experimental evidence show that long-lived species experiencing low levels of extrinsic adult mortality should evolve strategies to limit mortality costs to longevity (Charlesworth, 1994; Ghalambor and Martin, 2001; Martin, 2015). As such, long-lived species, which clearly experience low levels of environmental mortality, should minimize any intrinsic costs from metabolism to adult survival even if offspring predation is high. Under this scenario, benefits from increased adult mortality may override costs of juvenile mortality on metabolism, especially given alternative mechanisms (i.e. assimilation efficiency, wing growth tradeoffs) available to offspring for growth rate variation. Thus, the strong metabolic relationship with adult but not juvenile mortality (Fig. 1) follows from traditional life history theory (e.g. Charlesworth, 1994).

Adult mortality was not related to offspring growth despite the relationship between adult mortality and post-natal metabolic rate (Fig. 1C), and an influence of post-natal metabolic rate on growth (Fig. 2B; also Ton and Martin, 2016). This result was surprising for two reasons. First, we thought that the inclusion of metabolic rate in the model might unveil a correlation between growth and adult mortality that was not found in a previous study (Martin et al., 2015). Second, this result diverges from some within-species studies and theory that predict higher adult mortality with faster growth (Lee et al., 2013; Stier et al., 2015). However, these latter studies were based on proximate variation within species, which may differ substantially from evolved variation among species (Martin et al., 2015). Species with low adult mortality may evolve other mechanisms to offset the physiological costs of growth rates (Hulbert et al., 2007). Additionally, variation in parental care, such as food provisioning or warming, to influence growth may overshadow the correlation with adult mortality, and may instead reflect predation pressures (Martin et al., 2011).

In conclusion, our results suggest that there is not an evolutionary conflict mediated by metabolism between mortality at different ages. Indeed, predation at the nest appears to play a strong role in growth rate variation, but through alternative mechanisms beyond metabolism. Adult mortality instead showed a correlation with metabolism, with low mortality species exhibiting low offspring metabolism that potentially reduces future intrinsic costs. These previously unappreciated relationships raise questions on the role of metabolism as functional link underlying life history tradeoffs. Moreover, even after accounting for interspecific differences in metabolism, adult mortality was not related to growth rate contrary to intraspecific studies (Lee et al., 2013) and suggests that our understanding of the relationship between rate of growth and longevity may benefit from more tests in a comparative evolutionary framework. Further studies are needed to better understand how mortality sources at different life stages interplay to shape broad phenotypic variation in physiological and life history traits.

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Competing interests
The authors declare no competing or financial interests.

Author contributions
Conceptualization: T.E.M.; Methodology: R.T.; Validation: T.E.M.; Formal analysis: R.T.; Resources: T.E.M.; Data curation: R.T., T.E.M.; Writing - original draft: R.T.; Writing - review & editing: T.E.M.; Supervision: T.E.M.; Project administration: T.E.M.; Funding acquisition: R.T., T.E.M.

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Data availability
The data underlying this study are available in Martin et al. (2015).

Supplementary information
Supplementary information available online at
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