RESEARCH ARTICLE

The Effects of Temperature and Activity on Intraspecific Scaling of Metabolic Rates in a Lungless Salamander

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ABSTRACT

The scaling of metabolic rate with body mass holds substantial predictive power as many biological processes depend on energy. A significant body of theory has been developed based on the assumption that metabolic rate scales with body mass as a power function with an exponent of 0.75, and that this scaling relationship is independent of temperature. Here we test this hypothesis at the intraspecific level in a lungless salamander using data on both standard and maximal metabolic rates (SMR and MMR, respectively). We also address a recently proposed alternative explanation that predicts systematic variation in this mass-scaling exponent, the metabolic level boundaries hypothesis (MLB). Consistent with predictions of the metabolic theory of ecology the mass scaling of SMR and MMR were independent of temperature, however, we find evidence that the mass-scaling exponent for SMR and MMR differ significantly from 0.75. Further, our data do not provide strong support for MLB. Mass-scaling exponents for MMR generally exceed those for SMR, although these differences are rarely statistically significant. *J. Exp. Zool. 319A:230–236, 2013.* © 2013 Wiley Periodicals, Inc.

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Energy fuels all biological processes, thus biologists have sought general theories to explain variation in energy expenditure. The scaling of metabolic rate with body mass is broadly relevant because this relationship holds substantial predictive power as many biological processes depend on energy. The relationship between metabolic rate, M_R, and mass, W, is often described as the power function:

$$M_R = aW^b$$

where a is the mass-scaling coefficient and b is the mass-scaling exponent (i.e., the slope of a log–log plot). A significant body of theory assigns $b \approx 0.75$ (Kleiber, '32), independent of temperature and metabolic intensity (Hemmingsen, '60; Gillooly et al., 2001; Brown et al., 2004; Allen and Gillooly, 2007). The proposed universality of this scaling exponent stems from studies on resource transport network models. In these models, metabolic rate is constrained by the flux of resources and materials across surfaces and through fractally branching networks, which scale to the 1/4-power with body mass or volume (West et al., '97, '99a,b). Despite the promise of this theory for explaining biological phenomena spanning multiple levels of biological organization (e.g., Ernest et al., 2003; Savage et al., 2004a,b; Gillooly

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et al., 2005), the mass-scaling exponent often differs from 0.75 (Glazier, 2005 and citations therein) and is not independent of other factors such as temperature and activity.

One alternative explanation is the "metabolic level boundaries" hypothesis (MLB), which states that the metabolic scaling exponent varies between boundary constraints defined by surface area and volume ($b = 0.67$ and 1, respectively) with the relative importance of these constraints dependent on the metabolic intensity (metabolic level, L; Glazier, 2005, 2008, 2009a,b). The metabolic level (=metabolic intensity) represents the magnitude of the estimated metabolic rate standardized at a common mass. This hypothesis predicts that for resting animals, b should be negatively related to L, because high metabolic rates should be most limited by surface related fluxes of resources (which scale as $W^{0.67}$); whereas low metabolic rates should be released from this surface-related constraint and scale with energy necessary for tissue maintenance (which scales as W^{1}). During activity, however, MLB predicts that b should be positively related to L because increased activity shifts the energetic demand towards fueling muscle tissues, the mass of which scales isometrically with body mass. Thus, as metabolic level increases from very low to high, the metabolic scaling exponent should vary in a U- or V-shaped pattern (Glazier, 2008).

Data published over the last four decades have generally supported predictions of MLB at the interspecific level (Wood and Lawton, '73; Withers, '80; Robinson et al., '83; Gatten et al., '92; Weibel et al., 2004; Nagy, 2005; Niven and Scharlemann, 2005; Glazier, 2008, 2009a; Killen et al., 2010). However, the intraspecific applicability of this hypothesis has only recently been explicitly addressed (Glazier, 2009b; Ohlberger et al., 2011). Here, we test whether the intraspecific scaling of metabolic rate with body mass is dependent on temperature and activity in a lungless salamander, *Plethodon albagula*. In particular, we test both predictions of MLB: (1) that the scaling of standard metabolic rate (SMR) is negatively associated with metabolic level; and (2) that scaling exponents during activity exceed those at rest. For the first prediction, we explicitly test the competing hypotheses of a temperature-independent and a temperature-dependent (linear and nonlinear) mass-scaling exponent using a model selection approach (Ohlberger et al., 2011). For the second prediction, we test whether the mass-scaling exponent for maximal metabolic rate (MMR) exceeds that for SMR, at each temperature. Finally, we use SMR and MMR data measured at different temperatures to determine whether the mass-scaling exponent exhibits a U- or V-shaped pattern over a broad range of metabolic levels as observed among species (Glazier, 2008).

MATERIALS AND METHODS

Collection and Husbandry

Salamanders in the genus *Plethodon* have a direct developing life history (i.e., completely lacking a larval stage) and rely exclusively on cutaneous respiration for gas exchange (i.e., they are lungless). We collected 19 juvenile and adult *P. albagula* from a site in central Missouri. Animals varied sevenfold in mass, spanning a range of sizes (0.7–4.7 g) that is characteristic of the species from this location (Peterman, unpublished data). Animals were housed in rectangular plastic containers (16.5 cm × 10.5 cm × 6 cm) lined with moist paper toweling and a second crumpled moist paper towel for a refuge. We placed all containers in a temperature controlled incubator set at 15°C on a 14L:10D photoperiod. Animals were offered approximately 75 fruit flies (*Drosophila hydei*) once per week, at which time we recorded body mass. Prior to metabolic measurements, we fasted all salamanders for 7 days so that they were post-absorptive during measurement.

Metabolic Rate Measurements

For metabolic measurements, we placed individual salamanders inside 60 mL cylinders, which served as metabolic chambers. Each chamber contained a small length of moist sponge to prevent desiccation during measurement. Metabolic chambers were housed in a temperature-controlled cabinet set at the target test temperature. Animals were allowed a minimum of 3 hr to acclimate to test conditions. Prior testing indicated that 3 hr was sufficient to obtain metabolic rates of these salamanders at rest. We measured SMR for 5 min after the initial acclimation period. After this 5-min period a custom-built motorized rotating device was switched on that rotated the metabolic chamber at a slow rate (~4 revolutions min^{-1}) forcing the salamander to continually right itself. We continuously recorded metabolic rate until the trace of O_2 consumption stabilized, at which time the motor was switched off. Metabolic rate was recorded for an additional 3 min to confirm that there was no additional increase. SMR and MMR for each animal represent lowest and highest stable 2-min intervals, respectively.

We measured SMR and MMR at each of four temperatures (10, 15, 20, and 25°C) using an automated flow-through respirometry system (Qubit Systems, Inc., Kingston, Ontario, Canada). Each salamander rested for 10 days between temperature treatments. These temperatures represent ecologically relevant conditions that salamanders experience in the field. All measurements were taken between 1300 and 1900 hr to reduce temporal effects. We randomized temperature order; however, we tested all salamanders in the same sequence. Source gas was pushed through Drierite and soda lime columns prior to entering a mass flow controller (G246, Qubit Systems, Inc.), which regulates the flow rate through metabolic chambers. Flow rates during measurement varied between 50 and 100 mL min^{-1} depending on the size of the animal and temperature. The air stream exiting the chambers flowed into a gas switcher (G244, Qubit Systems, Inc.), which directed the stream from a focal chamber through the gas analyzers. The effluent gas stream was sub-sampled in parallel through H_2O scrubbers prior to entering an O_2 (S104 [DOX], Qubit Systems, Inc.) and a CO_2 (S157, Qubit Systems, Inc.) analyzer.

Oxygen and carbon dioxide traces were phased matched visually. We quantified gas exchange rates using the equations of Withers (2001) to account for dilution and concentration effects. These calculations were performed in the Multi Channel Gas Exchange Software (C950, Qubit Systems, Inc.). For all analyses we report SMR and MMR as rates of oxygen consumption ($\mu L\ hr^{-1}$).

Hypothesis Testing

We tested for temperature-dependence of the mass-scaling coefficient, *b*, using a model-selection approach. The first model assumes the mass-scaling exponent is independent of temperature and is described by the following equation (Gillooly et al., 2001):

$$R = R_0 M^b e^{\left(\frac{E_i(T-T_0)}{kTT_0}\right)} \quad \text{(Model 1)}$$

Following Ohlberger et al. (2011) we incorporated temperature-dependence into the model by adding a temperature term to the scaling exponent, *b*; and by incorporating a nonlinear term via an optimality function. The equations used for tests of temperature dependence are as follows:

$$R = R_0 M^{(b+c(T-T_0))} e^{\left(\frac{E_i(T-T_0)}{kTT_0}\right)} \text{(linear)} \quad \text{(Model 2)}$$

$$R = R_0 M^{(b+c(T-T_0)+d(T-T_0)^2)} e^{\left(\frac{E_i(T-T_0)}{kTT_0}\right)} \text{(nonlinear)} \quad \text{(Model 3)}$$

where *M* is body mass (g), *T* is temperature (K), T_0 is the freezing point of water (273.15 K), *k* is the Boltzmann constant ($eV\ K^{-1}$), and E_i is activation energy (eV). We used nonlinear least-squares regression with the Guass–Newton algorithm to estimate the remaining parameters in the models (R_0, *b*, *c*, *d*, and E_i). Following Ohlberger et al. (2011), we statistically estimated E_i, as this parameter is not known for *P. albagula*.

We tested the nested competing models against one another with likelihood-ratio tests. The linear model (Model 2) was first tested against the temperature independent model (Model 1), and then the nonlinear model (Model 3) was tested against the previously selected model. Superior models were identified by statistically significant likelihood-ratio tests ($P \leq 0.05$, via a χ^2 distribution with df = 1) and comparison of AIC values (Akaike, '74). We also tested for differences in mass-scaling exponents using repeated measures ANCOVA. For this analysis we $\log_{10}$-transformed metabolic rate, examined temperature as a repeated factor, and $\log_{10}$-transformed body mass as a covariate.

The scaling of MMR is predicted to exceed that for SMR at the same temperature because of a shift from surface related constraints to a dependence on fueling muscle activity, which scales isometrically with body mass (M^1). We calculated *b* and *a* using nonlinear models (metabolic rate as a power function of body mass) for each temperature. We tested whether the scaling exponents for MMR and SMR at each temperature differed using repeated measures ANCOVA with log-transformed body mass as a covariate, and response type (SMR vs. MMR) as a repeated fixed factor (i.e., we tested whether the body mass/response type interaction term was statistically significant). We excluded sex from these analyses because it was not a significant predictor at any temperature. Finally, we used *b* and *a* values calculated for MMR and SMR versus body mass measured at four different ecologically relevant temperatures to test for the predicted U- or V-shaped relationship between *b* and *L*. For this analysis, *L* was standardized at the mean body mass. We conducted all statistical tests in the statistical computing environment R version 2.14.1 (R Development Core Team, 2011).

RESULTS

Both SMR and MMR scaled significantly with body mass, although mass-scaling exponents showed little variation over temperatures (Fig. 1, Table 1). Our results provide conflicting evidence with regard to the central prediction of the metabolic theory of ecology. We found that the mass-scaling exponent of SMR is independent of temperature. The model selection approach revealed that the model 1 provided the best fit to our data for SMR (Table 2). Therefore, as temperature increased, the mass-scaling exponent remained relatively constant (Fig. 2A). All coefficients in this model differed significantly from zero (Table 3). However, a likelihood-ratio test favors a model with the estimated scaling exponent ($b = 0.621$) over a model with this value fixed at 0.75, as assumed by MTE ($\chi^2 = 6.487$, df = 1, $P = 0.01$). Similarly, model 1 provided the best fit to the data for MMR; thus, the mass-scaling exponent for MMR appears invariant to temperature (Table 2). All estimated coefficients in this model deviate significantly from zero (Table 3). The estimated scaling exponent for MMR ($b = 0.666$) provides a better fit to the data than one assuming $b = 0.75$ ($\chi^2 = 5.733$, df = 1, $P = 0.02$).

Results from repeated measures ANCOVA agree with the above analyses. As expected metabolic rates were significantly correlated with salamander body mass (SMR, $F_{1,13} = 16.21$, $P = 0.003$; MMR, $F_{1,13} = 42.17$, $P < 0.001$). Neither SMR nor MMR differed significantly between sexes ($F_{1,13} = 0.059$, $P = 0.81$, $F_{1,13} = 0.004$, $P = 0.98$). The scaling of SMR and MMR with body mass did not differ significantly between sexes as indicated by the sex by mass interaction terms (SMR, $F_{1,13} = 1.32$, $P = 0.28$; MMR, $F_{1,13} = 1.15$, $P = 0.31$). Furthermore, the mass-scaling exponents did not differ significantly among temperatures as indicated by the temperature by mass interaction terms (SMR, $F_{3,11} = 1.51$, $P = 0.29$; MMR, $F_{3,11} = 0.53$, $P = 0.68$).

The mass-scaling exponents for MMR were generally higher than those for SMR as predicted by the MLB hypothesis (Table 1); however, these differences were only statistically significant at 15 and 20°C (repeated measures ANCOVA: 15°C, $F_{1,34} = 5.88$, $P = 0.02$; 20°C, $F_{1,34} = 4.36$, $P = 0.04$; respectively). The MLB hypothesis also predicts that as metabolic intensity (*L*) increases, *b* should vary in a U- or V-shaped pattern (i.e., should be "concave-up"). Our data do not support this prediction. For salamanders in

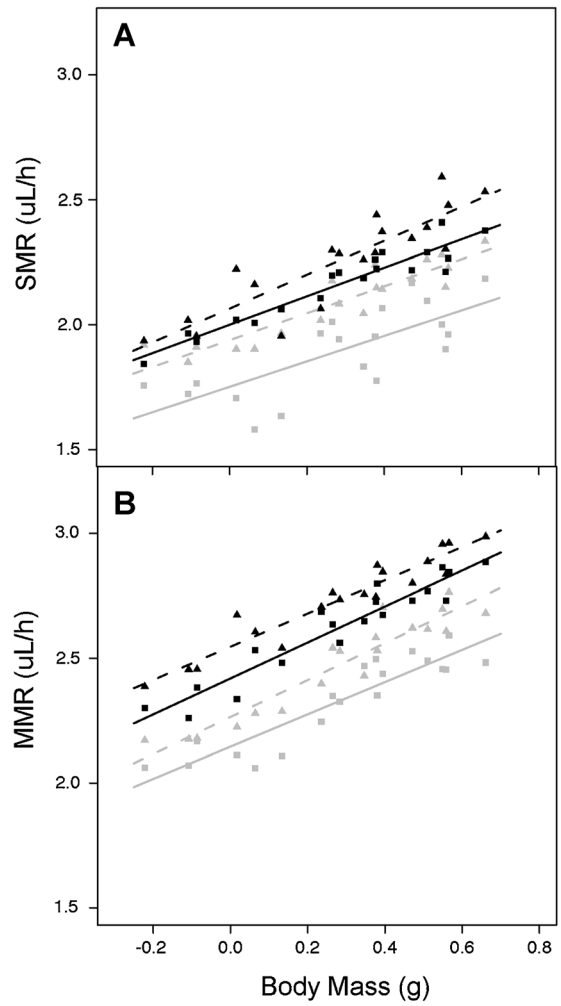

Figure 1. Relationships between standard (A) and maximal (B) rates of oxygen consumption (μL hr^{-1}; SMR and MMR, respectively) measured at four different temperatures. Plots represent least-squares regression of log_{10}-transformed values. Symbols represent measurements at different temperatures as follows: Gray squares and gray solid line (10°C), gray triangles and gray dashed line (15°C), black squares and black solid line (20°C), and black triangles and black dashed line (25°C).

Table 1. Allometric equations relating standard metabolic rate (SMR) and maximal metabolic rate (MMR) to body mass in salamanders.

	b	*a*	R^2
SMR			
10	0.549 ± 0.131	56.455 ± 7.789	0.557
15	0.567 ± 0.067	85.541 ± 6.059	0.838
20	0.544 ± 0.066	102.797 ± 7.105	0.837
25	0.707 ± 0.115	115.772 ± 14.615	0.744
MMR			
10	0.608 ± 0.088	146.613 ± 13.751	0.791
15	0.697 ± 0.090	193.341 ± 19.073	0.830
20	0.684 ± 0.069	272.758 ± 20.479	0.887
25	0.654 ± 0.061	356.910 ± 23.602	0.898

Equations were estimated using nonlinear least-squares regression ($M_R = a \times \text{Mass}^b$). Metabolic rates were estimated from rates of oxygen consumption (μL hr^{-1}). All $P < 0.001$.

this study, *b* and *L* are not significantly correlated with one another ($R^2 = 0.38$, $P = 0.11$; Fig. 3) and show a slight positive trend. We recognize that these data suffer from pseudo-replication; we simply use this analysis to illustrate a pattern.

DISCUSSION

One of the main assumptions of the metabolic theory of ecology is ¾-scaling between metabolic rate and body mass and that this scaling relationship is independent of body temperature (Gillooly et al., 2001; Brown et al., 2004). Numerous studies have examined patterns of metabolic scaling in various organisms; some finding that metabolic rate often does not scale in the predicted fashion. This discord appears to be found most often in ontogenetic data sets (i.e., within species, Withers, '92; Glazier, 2005). The metabolic scaling exponents for SMR and MMR are significantly lower than 0.75 when estimated using the model of Gillooly et al. (2001; Model 1, above) taking temperature into account. In addition, we cannot reject the hypothesis that the scaling exponent is temperature independent.

Thermal dependence of allometric scaling of SMR and MMR is predicted by the metabolic level boundaries hypothesis (MLB, Glazier, 2005). This hypothesis posits that the particular scaling relationship expressed is dependent on metabolic intensity and is bounded by surface-area and volume constraints. Recent research within several fish species provided mixed support for the MLB hypothesis at the intraspecific level and suggested that both plastic and adaptive responses may be responsible for the observed variation in intraspecific scaling (Ohlberger et al., 2011). Our data might be consistent with the MLB hypothesis in that MMR tends to scale more steeply with body mass than SMR. By contrast, our data for SMR are inconsistent with MLB. Specifically, we found that *b* is not correlated with temperature, rather than decreasing with increasing temperature as predicted by MLB. Our results also disagree with MLB when scaling exponents for SMR and MMR are combined with metabolic level, *L*. Rather than exhibiting a "U-shaped" pattern, in our dataset, *b* shows little relationship to *L*.

Intraspecific scaling relationships tend to be more variable than interspecific ones. Although several studies identify patterns of metabolic scaling that are consistent with MLB (i.e., negative

Table 2. Model comparisons for both standard and maximal metabolic rate (SMR and MMR, respectively).

		SMR			MMR		
Model	*K*	AIC_c	Log L	*P*	AIC_c	Log L	*P*
1	3	733.18	−362.31		840.16	−415.80	
2	4	733.94	−361.54	0.216	842.45	−415.79	0.931
3	5	736.15	−361.47	0.701	841.33	−414.06	0.175

Metabolic rates were estimated from rates of oxygen consumption (μL hr^{-1}). Models are ordered by the number of parameters (*K*) and include log-likelihoods (log L) and AIC values corrected for small sample size (AICc). Statistical significance of likelihood ratio tests indicates the preferred model. Model numbers represent variation in the thermal dependence of the mass-scaling exponent (1 = temperature independent, 2 = linear, 3 = nonlinear). Details of each model are given in the text.

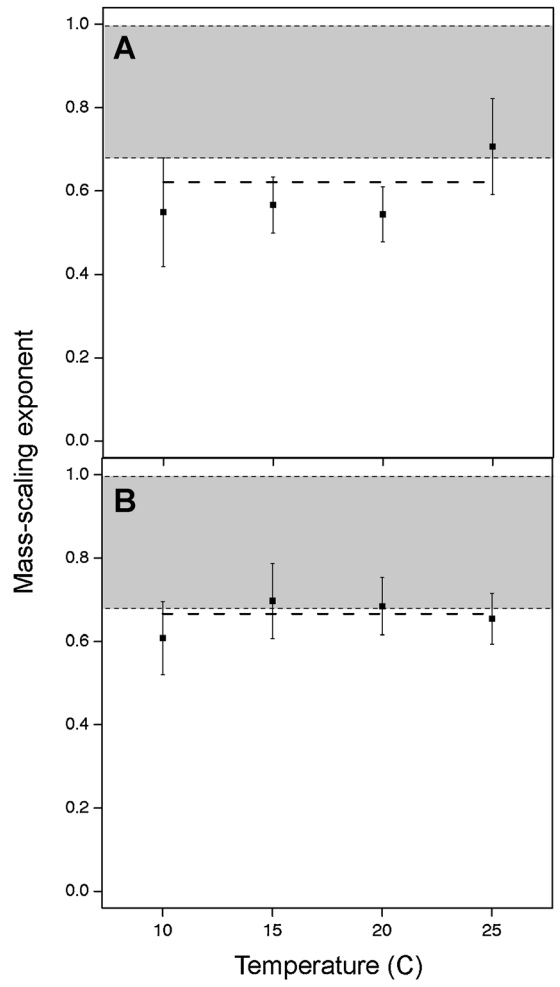

Figure 2. Thermal sensitivity of the mass-scaling exponent, *b*, for standard (**A**) and maximal (**B**) metabolic rates (μL O_2 hr^{-1}). The shaded area in each panel denotes mass-scaling exponents of 0.67 and 1.0, boundaries predicted by the metabolic level boundaries hypothesis.

relationship between *b* and *L* [or temperature]), a few have reported positive relationships (Newell, '73; Nespolo et al., 2003; Lardies et al., 2004). Variation in intraspecific metabolic scaling relationships has been attributed to several factors, both biological and methodological. Moses et al. (2008) found that substantial variation in metabolic scaling exponents (variation outside the 0.67 to 1) might derive from examination of patterns in samples that vary over a narrow range of body masses (less than 2 orders of magnitude). When body mass ranges are smaller, residual variation may have a proportionately larger influence on estimates of *b*. The body mass range examined here varies by less than one order of magnitude (sevenfold), so we cannot reject the possibility that the relatively low mass-scaling exponents reported here are a consequence of additional sources of residual variation. Some possible sources of residual variation include variation in body condition, gender, or reproductive state. If larger animals have disproportionately larger fat stores (positive

Table 3. Parameter estimates, standard errors (SE), and statistics from nonlinear least squares regressions of model 1 for standard and maximum metabolic rates (SMR and MMR, respectively; df = 91, *n* = 19).

Parameter	Estimate	SE	*t*	*P*
SMR				
R_0	31.818	3.411	9.328	<0.001
b	0.621	0.049	12.780	<0.001
E_i	0.387	0.032	12.236	<0.001
MMR				
R_0	75.417	5.918	12.744	<0.001
b	0.666	0.035	19.086	<0.001
E_i	0.437	0.023	19.147	<0.001

Metabolic rates were estimated from rates of oxygen consumption (μL hr^{-1}). Details of this model are provided in the text.

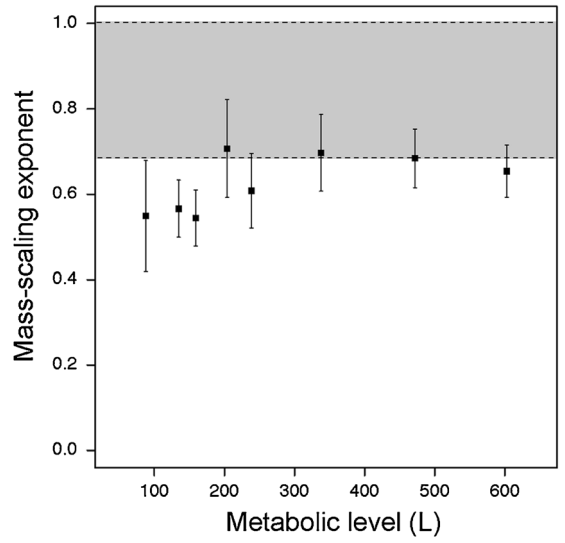

Figure 3. The relationship between mass-scaling exponents, *b*, and metabolic level, *L*, for standard and maximal metabolic rates (μL O_2 hr^{-1}) combined. *L* is standardized to the mean body mass for each.

allometry, $b > 1$), the metabolically sluggish fat might cause scaling exponents for metabolic rate to be low. Salamanders in this study differed in body condition, although there were no consistent differences between large and small individuals and no consistent correlations between residual metabolic rate and body condition (10°C, $R^2 = 0.004$, $P = 0.81$; 15°C, $R^2 = 0.33$, $P = 0.01$; 20°C, $R^2 = 0.008$, $P = 0.72$; 25°C, $R^2 = 0.08$, $P = 0.23$). It is possible that variation in body condition could account for a low scaling exponent at 15°C, but appears a less viable explanation at the other temperatures. If males and females do not differ in size, sex-specific variation in metabolic rates could also account for relatively low scaling exponents. Our sample included both unsexed juveniles and sexed adults, and between the sexes (adults) body size did not differ ($F_{1,11} = 0.26$, $P = 0.62$). Thus, it is possible that sex-specific variation in metabolic rate could account for additional residual variation (see Ryan and Hopkins, 2000) and influence the estimated mass-scaling exponents. However, in this study, metabolic rates did not differ significantly between adult males and females. Finally, reproductive state could have an influence on metabolic scaling. Our sample contained no gravid females, thus we feel that this explanation is unlikely for our data. Other factors such as a lack of control of activity levels, energy storage, or other non-maintenance energy-using processes could also influence scaling relationships. Although our acclimation period (3 hr) was relatively short, we controlled for activity by video-recording all measurements to ensure that metabolic rates at rest were obtained. However, it is possible that some variance in our measurements could result from stress. Additional measurements after a longer acclimation period could test this possibility. Finally, we made sure to measure post-absorptive metabolic rates to control for additional energy-using processes (i.e., Specific Dynamic Action).

Our results are mixed with respect to the predictions of MTE and MLB. We find that the metabolic scaling exponents are significantly different than 0.75, but do not differ from 0.67. Finally, our results are potentially consistent with the prediction of MLB that the scaling exponent for MMR should be higher than for SMR, although the differences we observe are rarely statistically significant. Furthermore, we find little relationship between scaling exponents and metabolic level, *L*. Our results highlight some of the difficulties in testing metabolic scaling theories. Many metabolic scaling studies focus on patterns at the interspecific level, however, a more complete understanding of the scaling of metabolic rate with body mass and the myriad factors that might influence it will require additional studies of ontogenetic scaling. At the intraspecific level, many factors can introduce residual variation in metabolic rate measurements that might influence patterns of metabolic scaling. Therefore, carefully planned manipulative experiments or careful control of these factors will be critical in future empirical tests of these theories.

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