Response to Comments on "Energy Uptake and Allocation During Ontogeny"

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Our extended ontogenetic growth model is a theoretical model based on conservation of energy and general biological mechanisms underlying ontogenetic growth. We do not believe that the comments of Makarieva *et al*. and Sousa *et al*. expose substantive problems with our model. Nevertheless, they raise interesting, still unresolved questions and point to philosophical differences about the role of theory and of simple, general models as opposed to complicated, specific models.

We presented a model for energy uptake and allocation over an organism's growth and development that reconciles rates of food assimilation with rates of allocation to maintenance, biosynthesis, activity, and storage (*1*). Makarieva *et al*. (*2*) and Sousa *et al*. (*3*) raise concerns about our model, which we address here.

Makarieva *et al*. (*2*) take issue with the assumption of our extended ontogenetic growth model that resting metabolic rate scales as $M^{3/4}$ throughout postembryonic growth and development, similar to the scaling across adult animals of different species. They claim that "young animals have elevated metabolic rates compared with what is predicted for their body mass from interspecific scaling." This further implies that metabolic rate as a function of mass throughout ontogeny cannot be a simple power function. So, instead of plotting as a straight line on logarithmic axes, the relationship must be biphasic or curvilinear, with the slope initially steeper and subsequently shallower than the interspecific $M^{3/4}$ scaling [as shown in figure 1B in (*2*)]. The scaling of metabolic rate during ontogeny is ultimately an empirical issue, albeit one with important conceptual and methodological implications.

So what do the data say? In their figure 1A, Makarieva *et al*. present data for metabolic rates "measured at rest in the postabsorptive state" for eight species of birds and mammals during postembryonic growth [data from (*4*)]. Makarieva *et al*. selected the "eight largest values of metabolic rates in early ontogeny," which would tend to bias their results in the direction of their claim, and show that the metabolic rate early in ontogeny is relatively higher than predicted by our model (*1*). We have compiled and plotted in Fig. 1 all the data from (*4*), including a ninth species, quail, which deviates from predictions in Makarieva *et al*. (*2*) and was not included in their analysis. Our analysis (see also Table 1) offers some support for Makarieva *et al*.'s claim that metabolic rates, relative to the predicted mass to the 3/4 power, are consistently and substantially higher during early ontogeny than at adulthood. Although the data for the individual species are well described by a power law (see Table 1 and Fig. 1), in most species there are times during early ontogeny when metabolic rates are higher than what would be predicted for an adult of the same size. Makarieva *et al*., following the model proposed by Wieser (*5*), claim that the peak metabolic rate occurs at approximately 10% of adult mass, but this is true for only four of the nine species. The other five species have peak values at relative sizes that range from 1% to 48% of adult mass [see data and figure 3 in (*4*)], and the rabbit has two distinct peaks.

Our analysis highlights two important points. First, there are necessarily deviations between detailed observations of individual growth curves and the predictions of a parsimonious model such as ours, and it is valuable for such deviations to be recognized in the literature. Second, the value of our model is that it provides a baseline to which data can be compared, so that the causes of any observed deviations can be revealed. Makarieva *et al*. point out such a systematic deviation from the model. However, they provide only a statistical description of these deviations, and one that does not capture the measurements for the majority of the species. Moreover, they do not provide any mechanistic explanation for these deviations. We suggest that these periods of peak metabolism may correspond with "growth spurts" that have been observed in a number of species and are also deviations from predictions of our model.

More generally, the comment of Makarieva *et al*. raises important, still unanswered questions about the energetics of growth and development. Our model was intended primarily to focus on the consumption of food and the allocation of the assimilated energy between maintenance of existing biomass and synthesis of new biomass. It does indeed imply that the total metabolic rate of a growing animal should be the same as that of an adult of the same mass. In doing so, it assumes that a constant fraction of this total metabolic rate is due to resting metabolism, B_{rest}, and a constant fraction to "activity," B_{act}, so that $B_{tot} = B_{rest} + B_{act} = B_{rest} + fB_{rest}$, where B_{rest}, B_{act}, and B_{tot} all scale as $M^{3/4}$, and f is the activity scope. In adult animals, the total metabolic rate is the field metabolic rate, where $f \approx 3$. To apply our model to the metabolic rate measured early in ontogeny clearly depends on how "activity" is defined. At present, however, it is not clear even whether "rest" and "activity" represent comparable states for very young animals and adults. Most crucial for the realism of our model is not how the total metabolic rate is partitioned between rest and activity, but whether B_{tot} scales as $\sim M^{3/4}$ throughout postembryonic ontogeny, the same as across adult animals of different size. This appears to be true across a wide variety of species (*6*).

Sousa *et al*. (*3*) make several specific criticisms to support their claim that only the dynamic energy budget (DEB) theory of Kooijman (*7*) provides a complete theoretical and mechanistic treatment of growth. We respond by emphasizing that our model is much simpler and

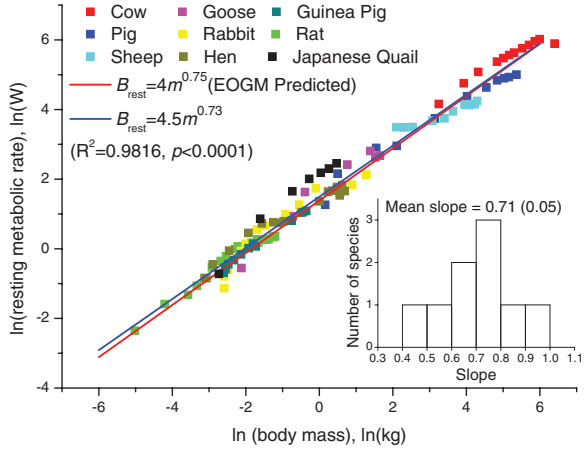

Fig. 1. Scaling of resting metabolic rate with body mass over ontogeny for nine species from (*4*). The red line is predicted by our extended ontogenetic growth model. The blue line is fitted to the data. The histogram shows the frequency distribution of the scaling exponents for these nine species.

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Table 1. The scaling exponent over ontogeny, peak normalized metabolic rate [$K \equiv B_g(m)/(B_0 m^{0.75})$] (*2*), and the relative body mass ($\mu \equiv m/M$) of this peak for each species, from (*4*).

Species	Slope	R^2	K	μ
Rat	0.71	0.9631**	1.41	0.36
Guinea pig	0.75	0.9901**	1.12	0.23
Hen	0.60	0.9643**	1.68	0.07
Rabbit§	0.77	0.905**	1.55	0.05
Goose	0.86	0.9517*	1.71	0.13
Sheep	0.46	0.9185**	1.71	0.11
Pig	0.66	0.9777**	1.49	0.01
Cow	0.59	0.9452**	1.57	0.13
Japanese quail	0.96	0.9858**	2.29	0.48
Mean	0.71		1.61	0.17

*$P < 0.001$ **$P < 0.0001$ §Rabbit shows another peak at $\mu = 0.25$

more parsimonious than DEB. DEB requires 17 variables and 18 parameters to be measured for every species (*8*), whereas our model contains only 2 variables (m and t) and 5 parameters (M, B_0, f, E_m, and E_c). There is nothing inherently wrong with the DEB approach, but for many theoretical and practical purposes it is desirable to have models that are as simple as possible as long as they still capture the essence of the phenomenon and give reasonably accurate predictions. As a particular case in point, Sousa *et al*. correctly point out that our model does not contain "an explicit chemical description of metabolism." From this they correctly conclude that the model we proposed "cannot explain the variable chemical composition of organisms growing with variable food." We note, however, that they present no data on how the chemical composition of the diet affects body composition or growth trajectory. In fact, as long as diets are relatively standard, the effects of such "variable food" are minor (*9*). Sousa *et al*. also claim that because our model does not explicitly incorporate the chemistry of metabolism, it cannot be applied to the growth of anaerobic organisms. This is incorrect. The fact that it deliberately ignores the details of biochemistry allows our model to provide a general quantitative accounting for the allocation of energy and biomass during postembryonic growth of diverse animals with different diets and biochemical pathways.

A critical difference between our model (*1*) and DEB is whether assimilation rate scales as a power law of body mass as a growing animal increases in size. The original DEB assumes that rate of food assimilation, because it is proportional to gut surface area, scales as $M^{2/3}$ (*7*, *10*). We are unaware of any evidence supporting this assumption. Indeed, our model would suggest that the highly elaborated fractal-like surface of the gut actually scales approximately as $M^{3/4}$ across adult animals of different species, so that the rate of energy uptake from food closely matches the rate of metabolic energy expenditure. Sousa *et al*. (*3*) suggest that DEB can account for changes in assimilation rate with increasing mass during growth "because the ratio of reserve to structure is not constant." Again, DEB introduces a level of detail and associated problems of defining and measuring "reserve" and "structure" that are peripheral to the central issue of how energy assimilation and allocation change with mass during ontogeny.

Sousa *et al*.'s characterization of our model as "demand" limited is misleading. DEB assumes that metabolic rate is limited by the availability and assimilation of food. Our model (*1*) is based on the original ontogenetic growth model of West *et al*. (*11*), which assumes that the scaling of metabolic rate with body mass is due to functional and geometric constraints on the capacity of the vascular network to supply energy to the body. So, like DEB, our model matches supply and demand, imposing mass and energy balance to require that uptake from food equals metabolic expenditure plus biomass storage.

Finally, we note that the comments of both Makarieva *et al*. (*2*) and Sousa *et al*. (*3*) expose fundamental philosophical differences about the role of models in biology. DEB is a single, very detailed model. It can potentially describe nearly all aspects of the metabolic basis of growth and account for variation within and across species due to variation in such factors as food supply, diet composition, and environmental conditions; to do so, however, requires measuring all 17 variables and 18 parameters. Our model, by contrast, is a very simple one that aims to quantify only the most essential features of energy acquisition and allocation. It is indeed similar to the model of Bertalanffy (*12*), as the comment authors and we ourselves have emphasized. Our model differs from Bertalanffy's model chiefly in its more explicit treatment of rates of assimilation and metabolism, including whether the latter scales as $M^{3/4}$ or $M^{2/3}$. Such simple models can provide a point of departure for incorporating complexity due to factors such as food restriction, temperature, or biochemical pathways of metabolism and for exploring additional phenomena such as energy allocation during embryonic development and tradeoffs between growth and reproduction in animals with indeterminate growth. We suggest that the complexity of DEB is the primary reason that, although the theory is frequently cited, the complete model is rarely implemented and applied to particular organisms. In most cases, the details and complexity of DEB are not required and a much simpler model, such as Bertalanffy's model or our model (*1*), will suffice.

Both Makarieva *et al*. and Sousa *et al*. imply that models such as ours and Bertalanffy's are flawed because the small number of variables and parameters do not include an explicit treatment of the chemistry of the diet and metabolic pathways. We emphatically disagree that more general models cannot "shed new light on the fundamentals of ontogenetic growth." Two parameters in our model, f and E_m, are difficult to assess quantitatively with data currently available. f, the "activity scope," is discussed above. E_m, the quantity of energy used to synthesize a quantity of biomass, is a fundamental biological parameter. We find it surprising that even today there are few data that can be used to estimate the value of E_m, let alone to assess how it may vary with diet, type of tissue being synthesized, taxon of organism, and environmental conditions. Indeed, most of the data used to inspire and evaluate our models of growth and by Makarieva *et al*. in their critique come from studies conducted decades ago. Without models that call attention to fundamental features of biological energetics, additional decades likely will pass before biologists are motivated to make the relevant measurements.

References and Notes

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