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A unifying explanation for diverse metabolic scaling in animals and plants

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ABSTRACT

The scaling of metabolic rate with body mass has long been a controversial topic. Some workers have claimed that the slope of log-log metabolic scaling relationships typically obeys a universal 3/4-power law resulting from the geometry of resource-transport networks. Others have attempted to explain the broad diversity of metabolic scaling relationships. Although several potentially useful models have been proposed, at present none successfully predicts the entire range of scaling relationships seen among both physiological states and taxonomic groups of animals and plants. Here I argue that our understanding may be aided by three shifts in focus: from explaining average tendencies to explaining variation between extreme boundary limits, from explaining the slope and elevation (metabolic level) of scaling relationships separately to showing how and why they are interrelated, and from focusing primarily on internal factors (e.g. body design) to a more balanced consideration of both internal and external (ecological) factors. By incorporating all of these shifts in focus, the recently proposed metabolic-level boundaries hypothesis appears to provide a useful way of explaining both taxonomic and physiological variation in metabolic scaling relationships. This hypothesis correctly predicts that the scaling slope should vary mostly between 2/3 and 1 and that it should be related to metabolic (activity) level according to an approximately U-shaped function. It also implies that the scaling of other energy-dependent biological processes should be related to the metabolic level of the organisms being examined. Some data are presented that support this implication, but further research is needed.

Key words: activity level, allometric scaling, animals, body mass, constraints, ecology, metabolism, plants, respiration

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I. INTRODUCTION

Understanding the factors affecting the rate of respiratory metabolism is of fundamental importance because it is linked to the rates of many other activities at various levels of biological organization, and thus represents a useful measure of the 'pace of life'. One of the most important of these factors is body size. Within specific taxonomic groups, respiratory metabolic rate (R) often varies with body mass (M) in a highly regular way that can be described by the power function

$$R = aM^b, \quad (1)$$

where a is the scaling coefficient (or proportionality constant) and b is the scaling exponent (or log-log slope). Historically b has been thought to approximate 3/4 in a variety of animals (Kleiber, 1932; Hemmingsen, 1960; Peters, 1983; Calder, 1984; Schmidt-Nielsen, 1984); and recently it has been argued that this apparent trend represents a '3/4-power law' that applies to virtually all organisms, including both animals and plants, and that it can be explained by universal properties of resource-transport networks (West, Brown & Enquist, 1997, 1999*a*, *b*; Banavar, Maritan & Rinaldo, 1999; Banavar *et al.*, 2002; Savage *et al.*, 2004). Furthermore, approximately 3/4-power scaling appears to apply to the rates of many kinds of biological processes, at molecular to ecological levels, and over physiological to evolutionary time frames, and thus has been considered to be a fundamental component of a general theory of life (e.g. Brown *et al.*, 2004*b*; Niklas, 2004; Savage *et al.*, 2004; West & Brown, 2005; Allen *et al.*, 2006; Whitfield, 2006; Brown, Allen & Gillooly, 2007; Cable, Enquist & Moses, 2007; Enquist, Tiffney & Niklas, 2007*c*; Gillooly, McCoy & Allen, 2007; Marbà, Duarte & Agusti, 2007; McCoy & Gillooly, 2008; but see Hirst & Bunker, 2003; Li, Han & Wu, 2005; Muller-Landau *et al.*, 2006; Atkinson & Hirst, 2007; Duncan, Forsyth & Hone, 2007; Russo, Wiser & Coomes, 2007; de Castro & Gaedke, 2008; Spence, 2009; and Section VII).

II. BEYOND THE 3/4-POWER LAW

Many investigators have recently questioned the universality of the 3/4-power law and the theoretical models underpinning it (reviewed in Agutter & Wheatley, 2004; Glazier, 2005; da Silva, Garcia & Barbosa, 2006). After surveying numerous data sets, several workers concluded that the body-size scaling of metabolic rate is highly variable and that there is no single universal scaling exponent. Substantial variation in b has been observed both among taxa (Glazier, 2005, 2006, 2009*b*, *c*; Reich *et al.*, 2006; White, Phillips & Semour, 2006; White, Cassey & Blackburn, 2007*b*) and physiological states (Bishop, 1999; Weibel *et al.*, 2004; Glazier, 2005, 2008, 2009*a*; Niven & Scharlemann, 2005; White & Seymour, 2005*a*; White *et al.*, 2007*b*). Furthermore, many log-log metabolic scaling relationships are nonlinear and cannot be represented by a simple power function (eq. 1) (Glazier, 2005; Kozłowski & Konarzewski, 2005; Painter, Edén & Bengtsson, 2006; Packard & Birchard, 2008).

In addition, several critiques have argued that the models of West *et al.* (1997, 1999*b*) and Banavar *et al.* (1999, 2002) proposed to explain the 3/4-power law are based on questionable or unsubstantiated assumptions and (or) are mathematically and logically inconsistent (e.g. Kozłowski & Konarzewski, 2004; Makarieva, Gorshkov & Li, 2005*c*; Painter 2005*b*, *c*; Chaui-Berlinck, 2006; Apol, Etienne & Olff, 2008). Critical analyses of these network models show that they may not predict a scaling slope of 3/4 as claimed, but other slopes such as 2/3 (Makarieva *et al.*, 2005*c*), 0.81 (Savage, Deeds & Fontana, 2008), 6/7 (Dodds, Rothman & Weitz, 2001), or 1 (Dodds *et al.*, 2001; Kozłowski & Konarzewski, 2004, 2005; Makarieva *et al.*, 2005*c*; Painter, 2005*c*; Chaui-Berlinck, 2006; Apol *et al.*, 2008). The proponents of these models have objected to some of these criticisms, saying that they are incorrect, misguided, or inconsequential and that their models remain fundamentally sound (Brown, West & Enquist, 2005; West & Brown, 2005; Banavar *et al.*, 2006; Savage, Enquist & West, 2007). However, several critics have been unsatisfied with these responses, claiming that their most serious criticisms have not been fully or directly addressed (Kozłowski & Konarzewski, 2005; Makarieva, Gorshkov & Li, 2006*a*; Chaui-Berlinck, 2007).

One possible solution to this controversy is to modify or further develop the resource-transport network models in ways that will allow them to predict the diversity of scaling relationships seen in nature. Recently, this approach has been taken by Gillooly & Allen (2007), Price, Enquist & Savage (2007) and Savage *et al.* (2008), but the results of these analyses have not resolved the controversy. Gillooly & Allen (2007) claim that adding a size-dependent effect of exercise on body temperature enables the model of West *et al.* (1997) to explain, at least partially, the relatively steep scaling of maximal metabolic rate ($b \sim 7/8$) in exercising mammals, but their view is contradicted by several lines of evidence (Glazier, 2008, 2009*a*; White *et al.*, 2008). Price *et al.* (2007) show how alterations of the geometry of resource-transport networks may affect allometric scaling in plants, but their analysis primarily focused on morphological traits,

rather than on metabolic scaling. Savage *et al.* (2008) explore several consequences and modifications of the assumptions of the model of West *et al.* (1997) and find that this model makes predictions that are often "at odds with trends detectable in empirical data".

The original model of West *et al.* (1997) predicted that small mammals should show steeper metabolic scaling slopes than larger mammals (*cf.* Savage *et al.*, 2004, 2008; West & Brown, 2005), but actually the opposite frequently has been observed (e.g. Heusner, 1991; Lovegrove, 2000; Dodds *et al.*, 2001; Kozłowski & Konarzewski, 2005; Painter *et al.*, 2006; Packard & Birchard, 2008). In addition, the observed change in slope occurs at a body mass much larger than that predicted by this model (Kozłowski & Konarzewski, 2005). Enquist *et al.* (2007*a*) have further claimed that the model of West *et al.* (1997, 1999*a*) predicts that the metabolic scaling slope of very small plants (e.g. seedlings) should be 1, but should approach 3/4 in larger plants (e.g. trees). However, the study of Reich *et al.* (2006, 2007) revealed that the metabolic scaling slope is near 1 over the entire body-mass range of their plant samples spanning over five orders of magnitude, comparable to a size range encompassing mice and elephants. As shown in Table 1, the reduced major axis scaling exponent used by Reich *et al.* (2006) is near 1 in all three major size groupings: very small seedlings (0.967 to 1.058), small to medium sized tree saplings (1.117) and relatively large trees (0.968). In addition, the model of West *et al.* (1997) predicted that the b value of flat, nearly two-dimensional organisms such as flatworms and bryozoans should be approximately 2/3, but available data do not provide strong support for this prediction either (e.g. bryozoans tend to show significantly higher b values often approximating 1: Peck & Barnes, 2004; Glazier, 2005).

An additional fundamental problem with the resource-transport-network models is that there is no evidence that they can be applied to organisms lacking vascular circulatory systems or other kinds of anatomical transport networks. These models, as presently formulated, cannot explain the extensive variation in metabolic scaling exponents that has been observed in organisms with or without physically observable resource-transport networks. For example, Glazier (2005, 2006) reported significant differences in metabolic scaling exponents between pelagic and benthic animals in four different animal phyla with very different body designs, including closed (tubular) or open (nontubular) circulatory systems, or none at all. Furthermore, these ecological differences in metabolic scaling are seen within groups of related species or even within species, where differences in resource-distribution systems are expected to be minimal.

Another possible solution to this controversy is to pursue entirely different approaches to the problem of metabolic scaling, as advocated by several workers (e.g., Kooijman, 2000; Darveau *et al.*, 2002; Kozłowski, Konarzewski & Gawelczyk, 2003*a*, *b*; Glazier, 2005, 2008; Chaui-Berlinck, 2006, 2007; da Silva *et al.*, 2006; Demetrius, 2006; van der Meer, 2006; Chown *et al.*, 2007; Apol *et al.*, 2008).

III. ALTERNATIVE MODELS AND APPROACHES

A variety of alternative models has been proposed to explain the diversity of metabolic scaling relationships that have been observed (reviewed in Agutter & Wheatley, 2004; Glazier, 2005; da Silva *et al.*, 2006; O'Connor *et al.*, 2007). Some of these models show promise and deserve further exploration, but in their present form, none (excepting the one featured here) appears to be capable of explaining the wide variation in b seen both among taxa and among physiological states. For example, the models of Patterson (1992), Witting (1995), Bejan (2001), Kozłowski *et al.* (2003*a*), Demetrius (2006) and Ginzburg & Damuth (2008) potentially provide insight into why b may vary among taxonomic lineages. However, their models, as currently developed, do not explain why b may also vary with physiological state (activity level). By contrast, the models of Sibly & Calow (1986), Darveau *et al.* (2002), Painter (2005*a*) and Barbosa, Garcia & da Silva (2006) attempt to explain variation in b with physiological state, but they have yet to be developed sufficiently to explain taxonomic variation in b. A truly unifying theory of metabolic scaling should be able to explain both. Further development of the above approaches singly or in combination may eventually lead to a unifying theory, but here I argue that another approach based on ecological effects, operating within the context of simple, generally applicable physical constraints, can already explain much of the variation that has been observed both among taxa and physiological states. This approach may help us to construct a unifying theory, if we are willing to make three shifts in focus.

(1) Adopting a focus on boundary conditions, rather than on average tendencies

Most theoretical models proposed to date have strived to explain some average value (or set of modal values) of the metabolic scaling slope (e.g. 2/3 or 3/4). Therefore, it should not be surprising that such models have failed to explain much of the variation around these predicted values. Here I propose that this variation can be understood better by focusing on the boundary constraints delimiting the range of metabolic scaling relationships that are possible, rather than focusing on average tendencies. Others have speculated about the importance of boundary conditions in metabolic scaling (reviewed in Glazier, 2005), but none have developed a general, testable model using this approach. Furthermore, most discussion of boundary constraints has focused on the origin of 3/4-power scaling, rather than on the diversity of scaling relationships that actually exist (but see Sibly & Calow, 1986; Weiner, 1989; Finkel, Irwin & Schofield, 2004). For example, Hemmingsen (1960) remarked that 3/4-power scaling appears to be the result of a "struggle between proportionality of metabolism to body weight and proportionality to surface functions" (p. 94), and this view has been echoed by Gould (1966), Kooijman (2000), and others. By contrast, the models of Davison (1955) and Kozłowski *et al.* (2003*a*) consider how extreme conditions of body-size change–either by changing cell size or changing cell

Table 1. Metabolic scaling data used in this paper. Values are normalization constants (antilog intercepts, *a*, ml O_2 h^{-1} at 1 g wet body mass, according to eq. 1 in text), scaling exponents (slopes, $b \pm 95\%$ confidence limits), sample sizes (N), correlation coefficients (r), and minimum and maximum wet body masses (g) of least squares regressions (LSR) and reduced major axis (RMA) analyses for various kinds of metabolic rate in different taxa of animals and plants. Scaling parameters for RMA analyses were taken from the original sources or calculated following Niklas (1994). Temperatures at which metabolic rates were made, or adjusted to (see Section V.1), are indicated. Wet body masses of gastropods and bivalves pertain to soft tissues only. Plant data include five sample groups: relatively large trees from Japan, field grown tree saplings, tree and herb seedlings grown in a greenhouse (GH) and tree seedlings grown in a growth chamber (GC) (see Reich *et al.*, 2006). The sample size equals number of species, except for the plants and polychaetes where N equals number of measurements. The total number of species of plants and polychaetes studied was 43 and 29, respectively

Resting metabolic rates

	LSR		RMA				Log_{10} body mass range (g)		
Taxon and temperature	*a*	$b \pm 95\%$ CL	*a*	*b*	N	r	Min.	Max.	Source(s)
Vertebrates									
Birds (38°C)	6.07	0.64 ± 0.03	5.36	0.66	83	0.97	0.740	3.588	1
Mammals (38° C)	4.98	0.676 ± 0.013	5.10	0.69	456	0.98	0.699	5.140	1
Reptiles (20° C)	0.11	0.76 ± 0.04	0.09	0.80	159	0.95	−0.398	4.457	1
Fishes (20° C)	0.09	0.88 ± 0.06	0.08	0.91	82	0.96	−0.658	3.362	1
Amphibians (20° C)	0.08	0.88 ± 0.05	0.07	0.94	158	0.97	−0.812	2.754	1
Invertebrates									
Winged insects (22° C)	0.399	0.662 ± 0.058	0.42	0.70	61	0.95	−3.310	0.716	2
Winged insects (25° C)	0.378	0.717 ± 0.048	0.433	0.788	204	0.91	−4046	0.731	3
Non-winged insects (25° C)	0.212	0.821 ± 0.065	0.302	0.912	158	0.90	−4.000	0.862	3
Crustaceans (20° C)	0.171	0.74 ± 0.06			225		−5.699	3.00	4
Spiders (22° C)	0.138	0.781 ± 0.117	0.17	0.87	46	0.90	−2.523	−0.072	5–7
Gastropods (20° C)	0.13	0.78 ± 0.04			268		−3.886	2.044	8
Bivalves (20° C)	0.09	0.76 ± 0.06		0.78	77	0.97	−3.638	2.230	9
Polychaetes (20° C)	0.068	0.850 ± 0.106	0.07	0.90	37	0.94	−2.799	1.271	10
Plants (24° C)									
Field/US tree saplings	0.004	1.062 ± 0.067	0.003	1.117	119	0.95	0.675	3.558	11
GH/tree seedlings	0.027	1.012 ± 0.058	0.028	1.037	165	0.98	−2.585	1.929	11
GC/tree seedlings	0.035	1.027 ± 0.036	0.037	1.058	190	0.97	−1.789	2.142	11
GH/herb seedlings	0.032	0.942 ± 0.038	0.034	0.967	62	0.97	−1.084	0.979	11
Trees (Japan sample)	0.005	0.858 ± 0.367	0.002	0.968	10	0.89	3.473	4.813	11[a]

Field metabolic rates

	LSR		RMA				Log_{10} body mass range (g)		
Taxon	*a*	$b \pm 95\%$ CL	*a*	*b*	N	r	Min.	Max.	Source(s)
Birds	21.66	0.681 ± 0.036		0.70	95	0.97	0.568	4.946	12
Mammals	9.94	0.734 ± 0.038		0.75	79	0.97	0.863	4.996	12
Reptiles	0.404	0.889 ± 0.059		0.91	55	0.97	0.041	4.655	12

Maximal or near-maximal metabolic rates during exercise

	LSR		RMA				Log_{10} body mass range (g)		
Taxon	*a*	$b \pm 95\%$ CL	*a*	*b*	N	r	Min.	Max.	Source(s)
Running mammals	17.42	0.870 ± 0.060	14.79	0.89	34	0.98	0.857	5.677	13
Flying Insects (22° C)	59.7	1.080 ± 0.062	60.7	1.09	56	0.99	−3.31	0.716	2

Table 1. (*Continued*)

Minimal or near- minimal metabolic rates during dormancy, torpor or hibernation									
Taxon and	LSR		RMA				Log_{10} body mass range (g)		
physiological state	*a*	*b* ± 95% CL	*a*	*b*	*N*	*r*	Min.	Max.	Source(s)
Shallowly hibernating mammals (20° C)	0.373	0.794 ± 0.064			36		0.716	3.602	14
Deeply hibernating mammals (5° C)	0.047	0.941 ± 0.086			29		0.716	3.602	14
Immobile insect pupae (25° C)	0.186	0.939 ± 0.136	0.229	1.00	28	0.94	−3.699	0.699	15–34
Diapausing insect pupae (25° C)	0.021	1.013 ± 0.182	0.022	1.04	10	0.98	−1.161	0.699	15, 21, 27, 35–37

[a]Calculated from supplementary data of Reich *et al.* (2006). Sources: 1, White *et al.* (2006); 2, Niven & Scharlemann (2005); 3, Chown *et al.* (2007); 4, Alekseeva & Zotin (2001); 5, Anderson (1994); 6, Lighton & Fielden (1995); 7, Anderson (1996); 8, Vladimirova (2001); 9, Vladimirova *et al.* (2003); 10, Cammen (1987); 11, Reich *et al.* (2006); 12, Nagy *et al.* (1999); 13, Weibel *et al.* (2004); 14, Geiser (1988); 15, Keister & Buck (1974); 16, Taylor (1927); 17, Dobzhansky & Poulson (1935); 18, Klekowski *et al.* (1967); 19, Guerra & Cochran (1970); 20, Kono (1970); 21, Denlinger *et al.* (1972); 22, Hågvar (1975); 23, Hanski (1976); 24, Bauman *et al.* (1978); 25, Prakash & Pandian (1978); 26, Baker *et al.* (1979); 27, Denlinger (1979); 28, Cairns (1982); 29, Gromysz-Kałkowska & Hubicka (1988); 30, Vogt & Appel (1999); 31, Mbata *et al.* (2000); 32, Fielden *et al.* (2001); 33, Sláma & Neven (2001); 34, Kramarz & Kafel (2003); 35, Crozier (1979); 36, Varjas & Sáringer (1998); 37, Kemp *et al.* (2004)

number–may help explain extreme metabolic scaling slopes of 2/3 and 1, and values between, but unfortunately their models cannot explain variation in scaling slopes that arises from different activity states. To develop a truly general, unifying model of metabolic scaling based on boundary conditions, I argue that the elevation (metabolic level) of a scaling relationship must also be considered.

(2) Considering both the slope and elevation of metabolic scaling relationships and their covariation

Most recent discussions of metabolic scaling have focused on the slopes of these relationships and ignored their elevations, which are often considered of secondary importance. This is a major oversight, as emphasized by Heusner (1991) who argued for the importance of understanding the vertical location of a metabolic scaling relationship in a "mass/power plane" (see also Kozłowski & Konarzewski, 2005; Demetrius, 2006; O'Connor *et al.*, 2007; Seibel, 2007; Glazier, 2008, 2009*b*, *c*; McNab, 2008, 2009). A biased focus on the slope of metabolic scaling relationships may in part stem from the common belief that metabolic rate scales to the 3/4-power, regardless of the elevation (metabolic level) of that relationship. This belief originated with the classic work of Hemmingsen (1960) who reported 3/4-power relationships in unicells and multicellular ectotherms and endotherms, despite their very different metabolic levels. Gillooly *et al.* (2001) claimed to show a similar pattern, but a reanalysis of their data shows that the scaling slope for endotherms is significantly different from 3/4, and nearly so for plants and unicells (Downs, Hayes & Tracy, 2008). Others have also found significant heterogeneity in slopes among the above groups by using larger, more comprehensive data sets (Phillipson, 1981; Robinson, Peters & Zimmerman, 1983; Glazier, 2005; Reich *et al.*, 2006; White *et al.*, 2007*b*).

When attempts are made to explain both the slopes and elevations of metabolic scaling relationships, usually separate mechanisms are invoked for each (but see Demetrius, 2006; McNab, 2008, 2009). For example, some proponents of the model of West *et al.* (1997) have claimed that the slope is universally 3/4 or nearly so because of internal design constraints, whereas the elevation is affected independently by various taxon-specific or environmental factors (Savage *et al.*, 2004; West & Brown, 2005; Kerkhoff *et al.*, 2005; Gillooly *et al.*, 2006; Allen & Gillooly, 2007; Price *et al.*, 2007). However, this view raises conceptual difficulties and cannot explain why the slope and elevation of metabolic scaling relationships often covary in systematic, biologically significant ways (Glazier, 2005, 2008, 2009*b*, *c*; see also Section V). A unifying theory of metabolic scaling should be able to explain this covariation.

(3) Considering ecological effects on both the slope and elevation of metabolic scaling relationships

The slopes of metabolic scaling relationships have often been considered to be the result of universal physical constraints, whereas the elevations of these relationships, and individual species deviations from them, have been attributed to various taxon-specific or ecological factors (but see Daan & Tinbergen, 1997; Kozłowski *et al.*, 2003*b*). According to this common view, ecological factors have little influence on the slope of metabolic scaling, which follows naturally from a belief in a universal 3/4-power law that applies to virtually all organisms regardless of their phylogeny or environment. However, the possibility that ecological factors may affect both the slope and elevation of metabolic scaling relationships deserves our attention because of the great diversity in metabolic scaling slopes that exists, some of which has already been linked to ecological differences (Glazier, 2005; Killen, Atkinson & Glazier, 2008). For example, Glazier (2006) has shown that pelagic invertebrates generally

exhibit significantly steeper intraspecific scaling slopes than related benthic species, a robust pattern that can be seen in four different animal phyla and at lower taxonomic levels as well. McNab (2008, 2009) has also shown how various ecological factors can affect both the slope and elevation of interspecific metabolic scaling in birds and mammals.

IV. THE METABOLIC-LEVEL BOUNDARIES HYPOTHESIS

The metabolic-level boundaries (MLB) hypothesis first presented in nascent form by Glazier (2005) incorporates all of the three shifts in focus described above. According to this model, which is more fully developed here, ecological lifestyle and activity level influence the overall metabolic intensity of a specific group of organisms (i.e. the level or elevation of its metabolic scaling relationship), which in turn affects the relative influence of basic physical boundary constraints on the metabolic scaling slope b (Fig. 1).

The two most extreme idealized boundary constraints are considered to be surface-area limits on fluxes of metabolic resources, wastes and (or) heat (scaling allometrically as $M^{2/3}$), and volume limits on energy use or power production (scaling isometrically as M^{1}) (*cf.* Kooijman, 2000; van der Meer, 2006). The relative influence of these boundary constraints depends on metabolic (activity) level L. In resting organisms, b should be negatively related to L because when maintenance costs are high, metabolic scaling should be primarily limited by fluxes of resources, wastes and (or) heat across surfaces (scaling as $M^{2/3}$), whereas when they are low and amply met by surface-dependent processes, metabolic scaling should be more related to the energy demand required to sustain the tissues, which is directly proportional to tissue mass or volume (scaling as M^{1}). This negative correlation between b and L should extend to dormant organisms with very low L, where b should be near 1, and to cold-exposed endothermic animals with relatively high L, where b should be near 2/3. Resting metabolic rates are usually estimated in the laboratory, but a negative correlation between b and L should also be seen in field animals and those engaged in minimal (routine) activities, as long as maintenance costs remain a large proportion of the energy budget.

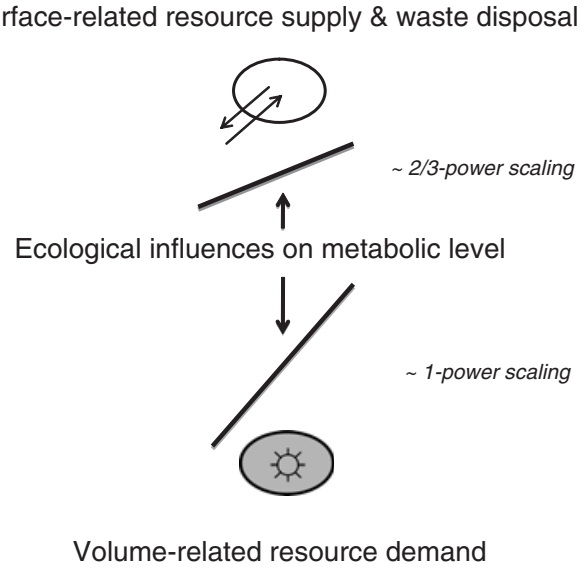

Fig. 1. A schematic representation of the metabolic-level boundaries hypothesis, as it applies to variation in the scaling of resting metabolic rate. Various ecological factors affect metabolic level, which, in turn, affects the relative influence of two boundary constraints (surface-area limits on fluxes of resources, wastes and heat, and volume limits on energy use and power production) on the slope of the scaling of log metabolic rate with log body mass.

However, in actively moving animals, b should be positively related to L because as activity increases metabolic rate is increasingly dominated by the energy demand of muscular tissue, which scales in direct proportion to muscle mass, which in turn scales as M^{1} (Calder, 1984; Darveau *et al.*, 2002; Weibel *et al.*, 2004; Glazier, 2005). During bursts of maximal activity, b should approach 1 because metabolic rate is primarily driven by the resource demand of metabolizing tissues, rather than by surface-dependent resource supply or waste removal (*cf.* Hammond & Diamond, 1997; Weibel & Hoppeler, 2005). These temporary episodes of high metabolic demand are made possible by stored oxygen and energy in the muscle tissues and their temporary tolerance to accumulation of wastes (e.g. lactic acid). Oxygen-carrying myoglobin and energy-carrying glycogen and phosphagens permit metabolic energy expenditure to exceed that which is immediately supplied by the resource-transport system (Sacktor & Wormser-Shavit, 1966; Suarez, 1996; Chung *et al.*, 2004; Riggs & Gorr, 2006). Exercise is also accommodated by increases in the supply of nutrients and oxygen to muscle tissues resulting from increased rates of respiration, heart beat and blood circulation (Weibel *et al.*, 2004). In addition, muscle activity enhances local blood flow by stimulating the opening and synthesis of increased numbers of intramuscular capillaries (Andersen & Henriksson, 1977; Ingjer & Brodal, 1978; Dawson, 2003; Bloor, 2005), thus further showing how resource demand dictates resource supply during exercise (*cf.* Lane, 2005; Weibel & Hoppeler, 2005), rather than the reverse assumed by resource-supply network models (e.g. West *et al.*, 1997; see also Section VII).

A corollary prediction is that the scaling exponent for maximal metabolic rate during exercise should become closer to 1, as relative muscle mass (athleticism) increases, and as the metabolic demand of muscular activity (scaling as M^{1}) increases relative to that of other demands (e.g. thermoregulatory heat production, scaling as $M^{2/3}$).

Overall, the MLB hypothesis predicts a U-shaped (or V-shaped) relationship between b and L at different activity levels (Fig. 2).

Mathematically,

$$b = f(L) = 1(p) + 2/3(1 - p) = (p + 2)/3 \qquad (2)$$

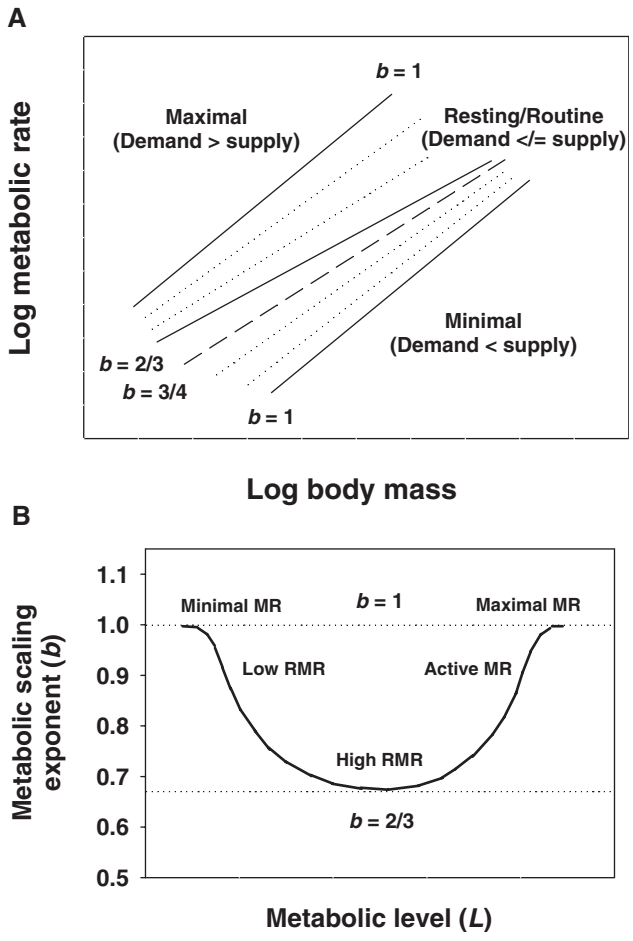

Fig. 2. Graphical models of how the slope (b) of the scaling of log metabolic rate with log body mass should vary with the elevation of the scaling relation (i.e. metabolic level, L), which are meant to be qualitatively correct, but not quantitatively accurate. (A) Maximal metabolic rate during exercise and minimal metabolic rate during dormancy are predicted to scale as M^1 (depicted by top and bottom solid lines), whereas relatively high rates of resting metabolism are predicted to scale as $M^{2/3}$ or $M^{3/4}$ (depicted by central solid and dashed lines, respectively). For levels of metabolism that are intermediate between resting and deeply dormant states and between resting and maximally active states, intermediate scaling relations should apply (depicted by dotted lines). (B) Over the full range of physiological states from minimal to maximal rates of metabolism, the metabolic scaling exponent (slope, b) should vary with metabolic level (L) according to an approximately U-shaped (or V-shaped) relation, as indicated by the concave solid line (see text for more details). Extreme values of b should be 2/3 and 1, as indicated by the dotted lines. MR = metabolic rate; RMR = resting metabolic rate.

and thus

$$R = aM^{f(L)} = aM^{(p+2)/3}, \quad (3)$$

where p and $1-p$ are the proportions of influence of the volume and surface-area boundary constraints represented by the scaling exponents 1 and 2/3, respectively. Thus when $p = 1$ (at minimal inactive and maximal active metabolic rates), $b = 1$, whereas when $p = 0$ (at maximal inactive metabolic rates), $b = 2/3$. Accordingly, when $1 > p > 0$, $1 > b > 2/3$. The exact shape of the predicted function [$f(L)$] should depend on the relative range of metabolic levels over which each constraint has a predominant influence (see also Section VI).

Other noteworthy views and observations complement this hypothesis (see also Section VI). For example, Makarieva, Gorshkov & Li (2005b) and Makarieva *et al.* (2006*b*) present a boundary approach that makes predictions that match some of those given by the MLB hypothesis. They argue that mass-specific metabolic rates should scale as M^0 at both maximal and minimal metabolic levels because these represent extreme mass-independent limits (*cf.* Singer *et al.*, 1993). This scaling parallels that predicted by the MLB hypothesis for whole-body maximal active and minimal inactive metabolic rates (M^1; see also Section VI). In addition, Lane (2005) argued that, since muscular power production has significant fitness consequences (e.g. it can increase rates of foraging, mate-finding and escape from predators), during maximal activity animals of all sizes should expend energy at or close to full capacity, thus causing the metabolic scaling exponent to approach 1. In other words, large animals that use less of their power capacity than small animals (as would occur if the scaling exponent for maximal metabolic rate was <1) should be selected against. Otherwise larger animals would be both weaker and less capable of bearing and transporting their heavier mass than smaller animals. On the other hand, the isometric scaling of maximal active metabolic rate may be the result of a universal scaling principle that transcends biological systems. A proportional (1:1) relationship between maximum force output and mass is exhibited by both exercising animals and human-made motors (Marden & Allen, 2002). In flying animals, maximum power production also scales isometrically with whole body mass (Voigt & Winter, 1999; Askew, Marsh & Ellington, 2001).

The potential limiting effect of internal surfaces and transport systems on metabolic scaling may also be incorporated into the MLB hypothesis. However, presently there is uncertainty about precisely what boundary constraint(s) would result. In Fig. 2, this possible boundary constraint is indicated by a scaling slope of 3/4, following the resource-transport-network models of West *et al.* (1997, 1999*b*) and Banavar *et al.* (1999, 2002); but this value is controversial: it may be 2/3, 0.81, 6/7 or 1 according to others who have scrutinized these models (see Section II).

V. EMPIRICAL TESTS OF THE MLB HYPOTHESIS

Given space limitations, the tests of the MLB hypothesis described here are generally based on only the most recent and comprehensive data sets on metabolic scaling that I could find. Interspecific scaling relationships are emphasized,

whereas intraspecific relationships are mainly considered elsewhere (e.g., Glazier, 2009*a*, *c*). Additional relevant empirical observations from various published studies, some of which are described in Glazier (2005, 2008, 2009*b*) are also briefly mentioned.

(1) Methods

(*a*) *Sources of data*

Sources and regression statistics of the data sets used here are given in Table 1. To test the prediction that the metabolic scaling slope b should be negatively related to metabolic level L in resting organisms, I used data sets for major taxa (e.g. orders and classes) of animals and plants selected on the basis of large sample size ($N = 29–456$ species) and body-mass range (2.5 to 8.7 orders of magnitude). Winged insects were analyzed separately from wingless insects because they are known to exhibit significantly different metabolic intensities and scaling slopes (Reinhold, 1999; Addo-Bediako, Chown & Gaston, 2002), differences corroborated by this study (see Section V.3; and Table 1]. This prediction was also tested for field metabolic rates, using the large data sets collected by Nagy, Girard & Brown (1999) for reptiles, birds and mammals. To test the prediction that increasing muscular activity, and associated L, should cause increases in b, comparisons were made between the scaling slopes of active *versus* inactive birds, mammals and insects, the only taxa for which sufficient interspecific data were available. To test the prediction that b should be related to L according to a concave function for all activity states, insects and mammals were used, not only because sufficient metabolic scaling data ($N = 10–56$ species) were available for both of these taxa in a variety of activity states, but also because they have largely non-overlapping body-mass ranges. The latter circumstance allowed for a robust statistical test of the MLB hypothesis, as will be seen. Minimal metabolic rates were estimated in diapausing insect pupae and hibernating mammals because they represent the lowest metabolic levels known in these taxa (Schneiderman & Williams, 1953; Singer *et al.*, 1993). Near-maximal rates of metabolism were estimated in actively flying insects (Niven & Scharlemann, 2005) and running mammals (Weibel *et al.*, 2004).

Respiratory metabolism was expressed as ml O_2 h^{-1}, and body size as g wet body mass. Conversions were made for some of the data from the original sources that were not given in these units of measurement (following Elliott & Davison, 1975; Peters, 1983). For example, dry body mass was converted to wet body mass assuming that 1 g dry mass = 6.5 g wet mass; a Watt (J s^{-1}) was converted to ml O_2 h^{-1} assuming 1 Joule = 0.0495 ml O_2, and therefore 1 Watt = 178 ml O_2 h^{-1}; and 1 mg O_2 h^{-1} was assumed to be equal to 0.699 ml O_2 h^{-1}. For plant metabolic rates originally expressed as CO_2 released (Reich *et al.*, 2006), a respiratory quotient of 1 was assumed (Platenius, 1942; Effer & Ranson, 1967).

Within each scaling relation, every value of metabolic rate was measured at the same ambient or body temperature or adjusted to the same temperature, as described in each source, or by using the van't Hoff normalization method (Schmidt-Nielsen, 1990) with a Q_{10} value given in the original source or assumed to be 2. In most cases the temperature used was the one closest to that at which the majority of measurements of metabolic rate were made (e.g., 38°C in endothermic vertebrates and 20°C in ectothermic vertebrates; see also Table 1). Therefore, when temperature adjustments were made, they were usually relatively minor. Moreover, the exact temperature used had little effect on the results. For example, inverse correlations between the scaling exponent (b) and metabolic level (log a) were found among the vertebrate classes regardless of whether all of the data were adjusted to 38°C ($r = -0.957$, $P = 0.011$) *versus* the data adjusted to 38°C and 20°C for endotherms and ectotherms, respectively ($r = -0.914$, $P = 0.030$; data from White *et al.*, 2006). White *et al.* (2006) also found that temperature corrections did not significantly affect the metabolic scaling slopes that they found in various vertebrate groups.

For each interspecific scaling relation, individual data points were different species (multiple values for the same species were averaged), except for the relations for plants (Reich *et al.*, 2006) and polychaetes (Cammen, 1987). In these cases, multiple data points for the same species were included because body-size differences between conspecific samples were too great to be adequately summarized as an average.

(*b*) *Statistical analyses*

Most scaling relations were examined using least-squares regressions (LSR), rather than alternative methods such as reduced major axis (RMA) analyses, for four reasons. First, most metabolic scaling relations reported in the literature are based on LSR, primarily because it allows metabolic rate to be predicted directly from body mass; and it also allows the effect of body mass on metabolic rate to be 'subtracted' in order to detect other influences (Harvey & Pagel, 1991; Sokal & Rohlf, 1995). Second, although body mass is measured with error, a violation of one of the assumptions of LSR, it is usually measured with much less error than metabolic rate. Therefore, I follow Calder (1987) in focusing on LSR, rather than on RMA analyses, which assume that the error variance of the X and Y variables is the same relative to the total variance on each axis (but see Warton *et al.*, 2006). Third, LSR permits relatively easy calculation of confidence limits for the scaling parameters, whereas other methods such as RMA analyses are more complicated or depend on LSR analyses (Sokal & Rohlf, 1995; Warton *et al.*, 2006). Fourth, most metabolic scaling relations have a high correlation coefficient (r often greater than 0.9), which results in scaling exponents based on LSR and RMA being very similar anyway ($b_{RMA} = b_{LSR}/r$) (Calder, 1987; Niklas, 1994). In Table 1, I report scaling parameters based on both LSR and RMA, which are usually similar. Using one over the other has no significant effect on the patterns reported herein. For example, RMA analyses reveal that

the inverse correlation between the scaling exponent (b) and metabolic level ($\log a$) among the various animal and plant taxa examined ($r = -0.927$, $P < 0.001$, $N = 13$) is very similar to that found for LSR analyses ($r = -0.870$, $P < 0.001$, $N = 16$; see also Fig. 5A).

The scaling relationships examined here were not corrected for phylogenetic effects (i.e. differential relatedness of the sampled species, and uneven distribution of body masses with respect to taxonomic affiliation). This was because the scaling parameters of most of these relationships were taken from studies that did not make these corrections. This is not a significant problem for the present work because my primary focus is on the relative magnitude of the slope of a scaling relationship in relation to the elevation (metabolic level) of that relationship. Phylogenetic corrections may alter the scaling slope somewhat (e.g. Symonds & Elgar, 2002; Chown *et al.*, 2007; White *et al.*, 2007*b*), but in no case have such changes been great enough to affect the general patterns observed herein. For example, in several studies the estimated scaling slope for resting metabolic rate in high-energy, endothermic birds and mammals is relatively low ($<3/4$) compared to that of many low-energy ectothermic organisms ($>3/4$), regardless of whether these slopes were based on traditional *versus* phylogenetically corrected analyses (McKechnie & Wolf, 2004; Glazier, 2005, 2008; White & Seymour, 2005*a*; White *et al.*, 2007*b*; Wiersma *et al.* 2007; Kabat *et al.*, 2008; Lovegrove, 2009; White, Blackburn & Seymour, 2009). Furthermore, the patterns reported here for interspecific scaling relationships are also seen for comparisons based on intraspecific scaling relationships that largely avoid the problems of phylogenetic effects (see Fig. 7; Glazier, 2005, 2009*a*, *c*; D. S. Glazier, unpublished data; Killen *et al.*, 2008).

It is also possible that the scaling relationships examined herein may be affected by uneven distribution of body masses of the analyzed species with respect to ecological lifestyle and environmental conditions (McNab, 1988, 2002; Lovegrove, 2000; Glazier, 2005). However, corrections for various ecological effects (McNab, 2008, 2009) show that the metabolic scaling slopes of birds (0.689) and mammals (0.694) are similar to the exponents used in this study (see Figs 3 and 6, and Table 1) and elsewhere (Glazier, 2008).

To test the MLB hypothesis rigorously, the scaling exponent b was compared to two complementary estimates of metabolic level L, each with offsetting advantages and disadvantages. The first estimate of L ($= L_a$) was the conventional use of the intercept ($\log a$; Prosser, 1973; Zotin, 1990; McCarthy & Enquist, 2005). This method has the advantage of using a single standard body mass (0 log unit mass or 1 unit mass) for comparison. The common mass was chosen to be 1 g because it is conveniently unitary, and approximately midway between the extreme body masses examined herein (ranging from ~0.0001 to 100,000 g). A disadvantage of this method is that the intercept ($\log a$) may depend not only on biological factors affecting the metabolic intensity of an organism (e.g. endothermy *versus* ectothermy), but also on the slope (b) of the regression line. A dimensional analysis of eq. 1 (see Section I) reveals that a has units of energy consumed per time per unit body mass^b (Xiao, 1998), and thus depends on both b and the units chosen to represent body mass [but note that herein (e.g. Table 1 and Figs 3, 5–8) a is represented simply by energy or oxygen consumed per unit time for a 1-g organism so as to match the actually measured values of metabolic rate (R) used to determine a scaling relationship, and to facilitate comparisons among scaling relationships]. An autocorrelation between the slope and intercept (see Gould, 1966; Peters, 1983; McNab, 1988) may confound attempts to determine relationships between b and L_a ($= \log a$) that are biologically significant, and not merely statistical or mathematical artifacts. The relative influence of this autocorrelation effect on the relationship between b and $\log a$ should be a negative function of the vertical distance between two regression lines, but a positive function of the distance between each intercept and the midpoint of each regression line. Thus, observed relationships between b and $\log a$ may be largely a result of the autocorrelation effect when two or more regression lines are close and cross one another. However, this statistical artifact should be negligible when large, over-riding biological effects cause the regression lines to be far apart over the entire range of body masses that exist for each taxonomic sample. In addition, if each intercept ($\log a$) is close to the midpoint of each regression line, the autocorrelation effect should be minimal, because changing the slope has no effect on the value of an intercept located at the pivotal midpoint of the regression line (at the midpoint, a is represented by energy used per unit time per 1 mass unit, and thus $L_a = L_m$, another measure of L discussed below). However, if each intercept is near the lower end of each regression line, the autocorrelation effect should bias the relationship between b and $\log a$ towards being negative, whereas, if each intercept is near the upper end of each regression line, the relationship between b and $\log a$ should be biased towards being positive (Peters, 1983), an important distinction that will become relevant later. This argument strictly applies to only the actual body-mass ranges examined in a given taxonomic group, and not to extended hypothetical extrapolations of scaling relationships to non-sampled or nonexistent organisms with extremely small or large body masses. If one extends scaling relationships with different slopes far enough they will cross, and thus negate or even reverse the autocorrelation effects just described, but this is a not a problem in this study because most scaling relationships did not cross within the actually sampled body-mass ranges.

To overcome the above statistical problem, a second method was used that estimated the metabolic level (L) of each scaling relation as the predicted mass-specific metabolic rate at the midpoint of the regression line for each taxonomic sample. This method has the advantage that the estimated L ($= L_m$) is completely independent of the slope (b), and thus any observed relationship between b and L_m cannot be considered a result of statistical autocorrelation. However, this method has the disadvantage of not being

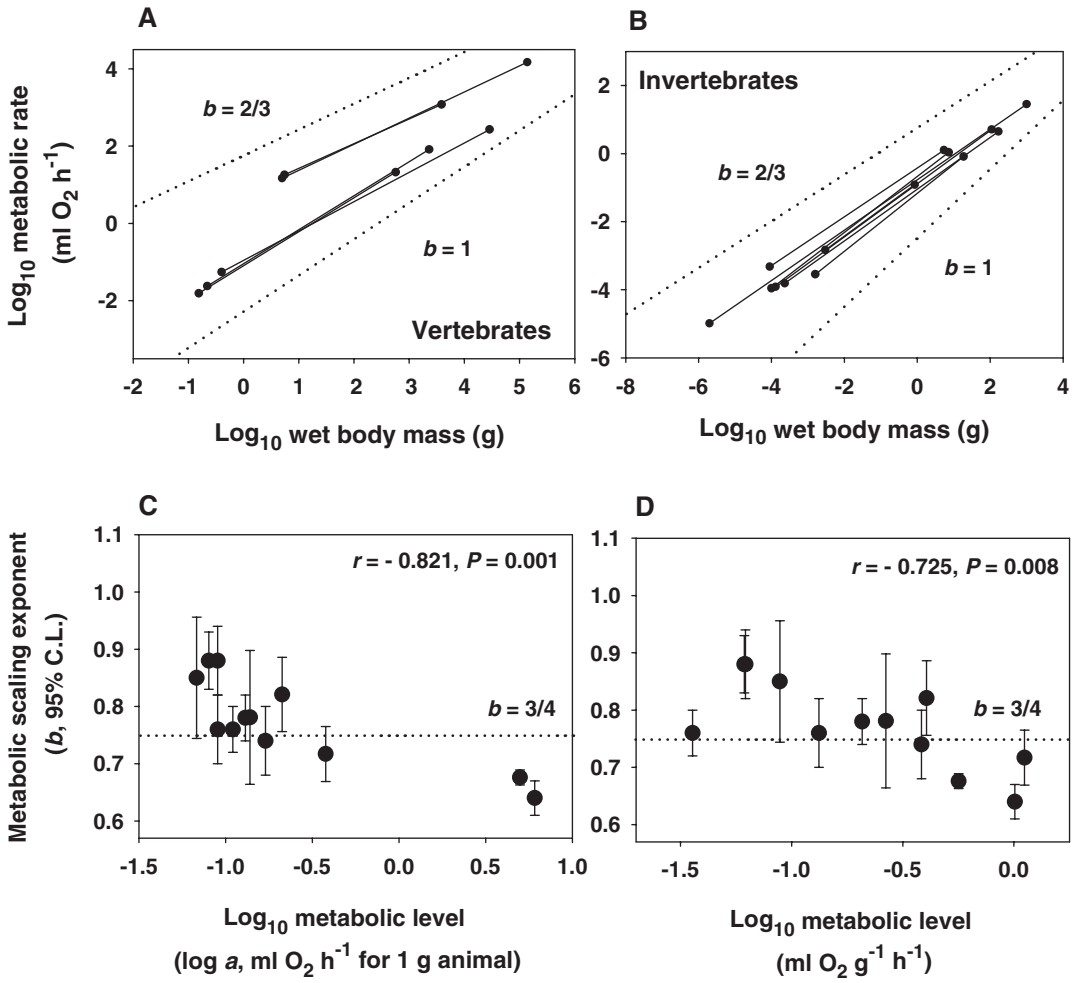

Fig. 3. Scaling of resting metabolic rate in relation to wet body mass in major taxa of animals (data from Table 1). The symbols at the ends of each log-log least-squares regression line denote the minimum and maximum body masses for each sample. Dotted lines represent scaling in proportion to body-surface area ($b = 2/3$) and body mass ($b = 1$), and according to the 3/4-power law. (A) Scaling for five vertebrate classes. (B) Scaling for six invertebrate orders or classes (winged and wingless insects are represented by separate lines because they have significantly different b and L values). (C, D) Scaling exponents ($b \pm 95\%$ confidence limits) *versus* metabolic level estimated as the metabolic rate at the intercept ($\log a$) and as the mass-specific metabolic rate at the midpoint of each regression line, respectively, among all of the 12 animal groups (left to right in C: polychaetes, amphibians, fishes, bivalves, reptiles, gastropods, spiders, crustaceans, wingless insects, winged insects, mammals, and birds; and in D: reptiles, fishes, amphibians, polychaetes, bivalves, gastropods, spiders, crustaceans, wingless insects, mammals, birds, winged insects). Correlation coefficients (r) and associated probability values (P) are shown.

standardized to the same body mass. Therefore, this method is more reliable when the body-mass ranges of the regression lines being compared are similar and largely overlapping, than when they are dissimilar and largely or wholly non-overlapping.

Fortunately, these two methods of comparing L and b are complementary: the L_a method uses a single reference body mass, but may be influenced by an autocorrelation with b, whereas the L_m method suffers no autocorrelation effects, but may be influenced by comparisons involving different body masses. Therefore, if these methods reveal similar patterns, it is likely that the results are biologically significant and not merely statistical artifacts (see also Sections V.3 and V.5).

(2) Metabolic scaling slopes vary mostly between 2/3 and 1

As predicted by the MLB hypothesis, the exponents of the metabolic scaling relations presented in Figs 3–7 vary mostly between 2/3 and 1 (see also Table 1). The extensive survey by Peters (1983) of resting and routine metabolic rates of animals and unicells also reveals that most interspecific scaling exponents vary between these values (85%; range = 0.2–1.0; $N = 162$). In addition, the large invertebrate survey of Glazier (2005) shows that most exponents for intraspecific metabolic scaling relations lie between 2/3 and 1 (70%; range = 0.2–1.2; $N = 413$), but a greater proportion of values occur outside this range than that

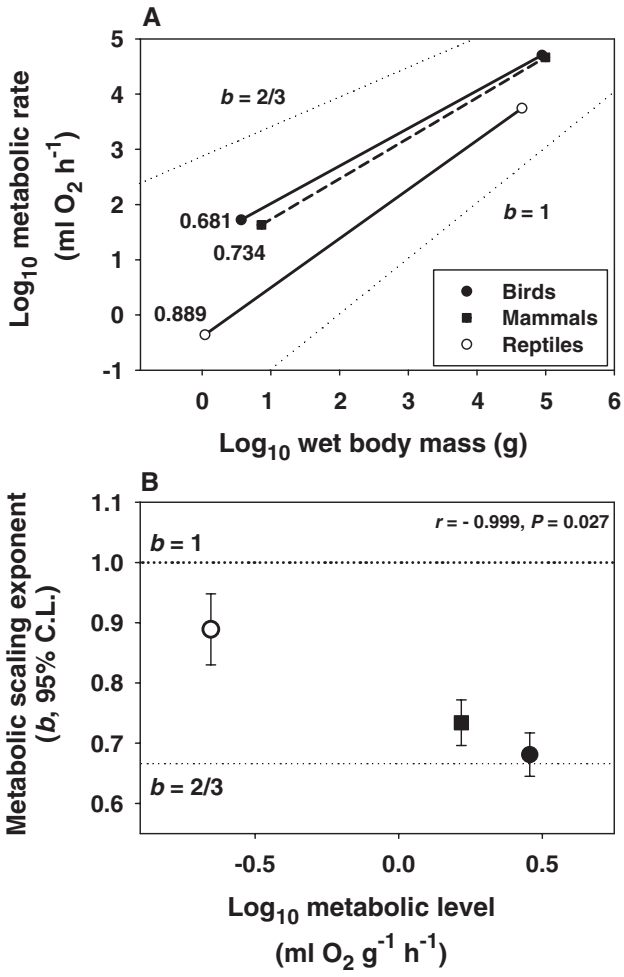

Fig. 4. Scaling of field metabolic rate in relation to wet body mass in three vertebrate classes (data from Table 1). Dotted lines represent scaling in proportion to body-surface area ($b = 2/3$) and body mass ($b = 1$). (A) The symbols at the ends of each log-log least-squares regression line denote the minimum and maximum body masses for each sample. The slope (scaling exponent) for each line is given. The slopes for the solid lines are significantly different from 3/4, whereas the slope of the dashed line is not significantly different from 3/4. (B) Scaling exponents ($b \pm 95\%$ confidence limits) *versus* metabolic level estimated as the mass-specific metabolic rate at the midpoint of each regression line. The correlation coefficient (r) and associated probability value (P) are shown. A similar negative relationship is observed when metabolic level is estimated as the metabolic rate at the intercept ($\log a$) ($r = 0.998$, $P = 0.039$).

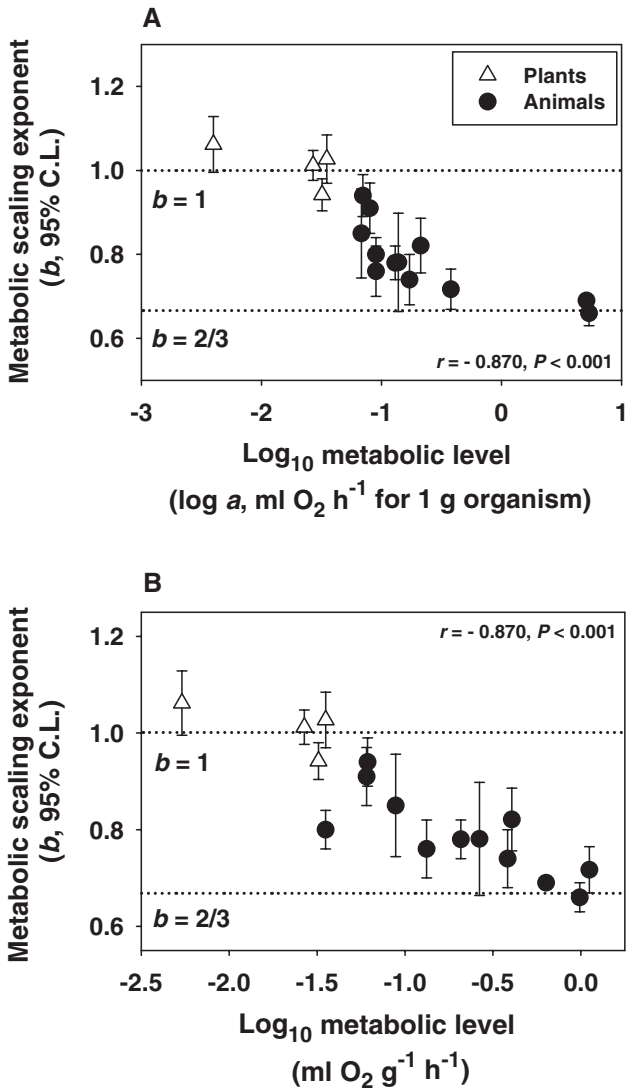

Fig. 5. Scaling exponents in relation to resting metabolic level in plants and animals. Scaling exponents ($b \pm 95\%$ confidence limits) and metabolic level [estimated as the metabolic rate at the intercept ($\log a$) in A, and as the mass-specific metabolic rate at the midpoint of each regression line in B] are based on log-log least-squares regressions for four experimental groups of plants and 12 taxonomic groups of animals (data from Fig. 3 and Table 1). Correlation coefficients (r) and associated probability values (P) are shown. Dotted lines represent scaling in proportion to body-surface area ($b = 2/3$) and body mass ($b = 1$).

observed for interspecific relations. Inspection of Fig. 4–7 in Withers (1992) similarly reveals that most metabolic scaling exponents for animals and unicells occur between 2/3 and 1 (see also White *et al.*, 2007*b*), and that more variation outside these values is observed for intraspecific relations (range = 0.3–1.8; $N = 220$) than for interspecific relations (range = 0.4–1.1; N = 107).

If the error in estimating the scaling exponents is taken into account, a match between the observed range of exponent values and the hypothetical boundaries of 2/3 and 1 becomes even closer. For example, if the average 95% confidence limits (±0.092) of the interspecific exponents surveyed ($N = 80$) by Peters (1983) is used to extend the lower and upper limits of the predicted range to 0.575–1.092, virtually all (97%) of the exponents reported are thereby bracketed. A similar procedure applied to the survey of Glazier (2005) also results in nearly all (96%) of the intraspecific exponents being bracketed by the error-extended range of 0.467–1.200 (based on average 95% confidence limits of ±0.200; $N = 642$;

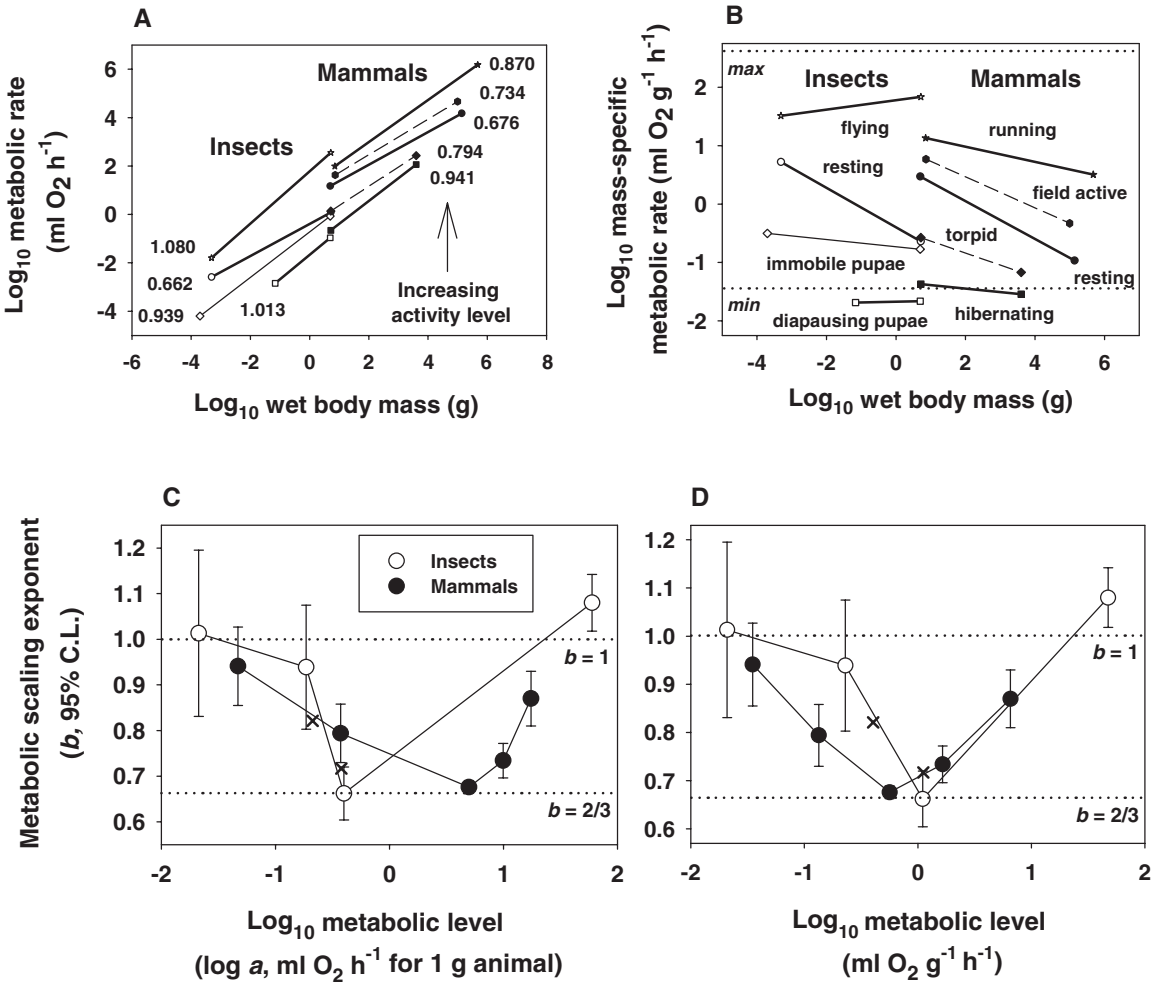

Fig. 6. Scaling of metabolic rate for different activity states in relation to wet body mass in mammals and insects (data from Table 1). The symbols at the ends of each log-log least-squares regression line denote the minimum and maximum body masses for each sample. (A) The slope (scaling exponent) for each line is given. The scaling exponents of solid lines are significantly different from 3/4, whereas the slopes of dashed lines are not significantly different from 3/4. (B) Scaling of log mass-specific metabolic rate in relation to log wet body mass. Activity states are indicated (note that "torpid" and hibernating" refer to hibernating mammals with body temperatures of 20°C and 5° C, respectively; see Table 1). The scaling exponents of solid lines are significantly different from -1/4, whereas the slopes of dashed lines are not significantly different from -1/4. The dotted lines denote the maximum (*max*) and minimum (*min*) metabolic rates estimated for living matter (Makarieva *et al.*, 2005*a*; see also Section VI). (C, D) Scaling exponents ($b \pm 95\%$ confidence limits) *versus* metabolic level [estimated as the metabolic rate at the intercept ($\log a$) and as the mass-specific metabolic rate at the midpoint of each regression line, respectively] among insects and mammals in different activity states. Note the approximately U-shaped relationship shown for each taxon with extreme values of the scaling exponents near 2/3 and 1 (indicated by dotted lines), as predicted by the MLB hypothesis (Fig. 2B). The *b* value for insect resting metabolic rate represents that for only the winged species also used to estimate *b* for maximal metabolic rate during flying. However, if this value is replaced by other somewhat different *b* values that have been reported (as indicated by the X symbols) the U-shaped pattern between *b* and *L* remains [the left X indicates the *b* value for wingless insects, and the right X value the *b* value for winged insects, using data from Chown *et al.* (2007); see also Table 1]. The U-shaped pattern for mammals also occurs regardless of which *b* values are chosen from the literature (see Glazier, 2008).

Table 5 of Glazier, 2005). This exercise also helps explain why *b* values are more variable for intra- *versus* interspecific relationships: the former are usually estimated with more error (broader confidence limits), probably because they often encompass narrower body-size ranges. The variance of *b* tends to be inversely correlated with the body-mass range examined (Bokma, 2004; White & Seymour, 2005*b*; Moses *et al.*, 2008). Nevertheless, an analysis of the extensive intraspecific data compiled by Glazier (2005) demonstrates that almost all of the *b* values are between 2/3 and 1, or nearly so, when the body-mass range exceeds two orders of magnitude (see Fig. 1 in Moses *et al.*, 2008).

The MLB hypothesis allows for the possibility that 3/4-power scaling may represent one extreme boundary

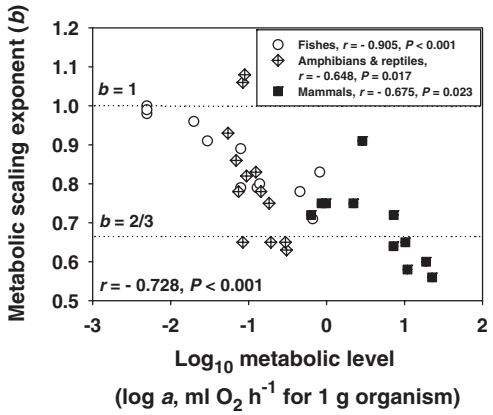

Fig. 7. Intraspecific scaling exponents in relation to resting metabolic level of vertebrate animals. Scaling exponents (b) and resting metabolic level (log a) are based on log-log least-squares regressions for each of 36 vertebrate species (data from Withers, 1992). Metabolic rates of mammals were adjusted to 38°C, whereas those of fishes, amphibians and reptiles were adjusted to 20°C (see also Section V.1*a*). Correlation coefficients (r) and associated probability values (P) are shown for all of the vertebrates, fishes alone, amphibians and reptiles alone, and mammals alone. Dotted lines represent scaling in proportion to body-surface area ($b = 2/3$) and body mass ($b = 1$).

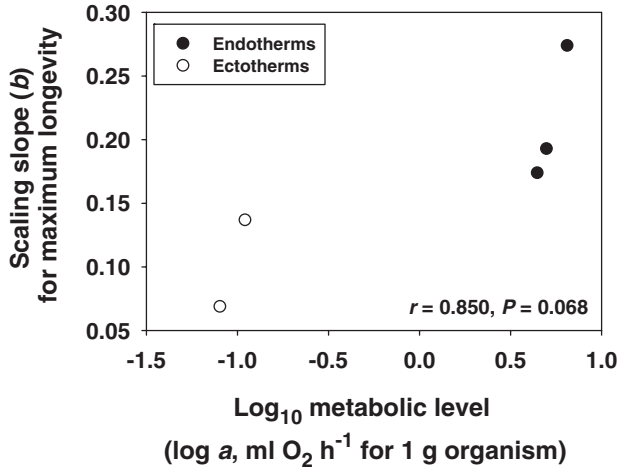

Fig. 8. Scaling slope (b) of maximum longevity of five vertebrate groups (see also text) in relation to their metabolic level (log a) (data from Table 1; de Magalhães, Costa & Church, 2007; McNab, 2009). The mammal sample excluded bats and cetaceans because these groups have strongly divergent lifestyles, and different methods were used to estimate their longevity (see de Magalhães *et al.*, 2007). The correlation coefficient (r) and associated probability value (P) are shown. Note that although the correlation between b (maximum longevity) and log a (metabolic level) is only marginally significant ($P < 0.10$), the correlation between b and a is significant ($r = 0.906$, $P = 0.034$).

constraint, rather than 2/3-power scaling. However, the scaling relations displayed in Figs 3–7 appear to be bounded by extreme scaling exponents of ~2/3 and ~1, rather than by ~3/4 and ~1. In addition, the surveys of Peters (1983) and Glazier (2005) reveal that far fewer scaling exponents vary between 3/4 and 1 for both interspecific (46%) and intraspecific relations (48%) than between 2/3 and 1 (see above).

(3) Negative relationships between *b* and *L* for resting or routine metabolic rates

The MLB hypothesis predicts that the scaling exponent b for resting metabolic rate should not only vary between 2/3 and 1, but also should be inversely related to metabolic level L. As expected, birds and mammals with relatively high-energy maintenance demands have scaling exponents not significantly different from 2/3 (but significantly <3/4), whereas relatively low-energy fishes and amphibians have exponents approaching 1 (and significantly >3/4) (Fig. 3, Table 1). Animals with intermediate metabolic intensities (e.g., reptiles, spiders, crustaceans and gastropods) exhibit scaling exponents that are between 2/3 and 1 and not significantly different from 3/4 (Fig. 3, Table 1). Furthermore, as predicted, among all of the sampled taxa, b is highly significantly inversely correlated with L, estimated as log a (L_a: Fig. 3C), or as the mass-specific metabolic rate at the midpoint of each regression line (L_m: Fig. 3D). Neither of these relationships is merely an autocorrelation effect because relationships between b and L_m are independent of such effects, and L_a (intercept at log 0) is located near the high end of the body-mass range for seven of the 12 taxonomic groups, which should have caused a net positive autocorrelation between b and L, rather than the negative one observed (see also Section V.1*b*). In addition, b appears to be inversely related to L (regardless of the body mass at which L is calculated) not only for resting metabolic rates estimated in the laboratory, but also for field metabolic rates in birds, mammals and reptiles (Fig. 4).

This negative pattern appears to be related to the varying ecological lifestyles and mobility of the sampled taxa. Relatively active endothermic birds and mammals have high L and low b values, whereas less active ectothermic amphibians and fish have low L and high b values. Highly mobile winged insects also show a significantly higher L and lower b than less mobile wingless insects (Figs 3, 6C, D; see also Section V.5, Table 1, and Addo-Bediako *et al.*, 2002). Indeed, Niven & Scharlemann (2005) reported that a sample of high-energy winged insects exhibited a scaling exponent that was nearly 2/3 (and significantly <3/4), like that of birds and mammals (Fig. 6; Table 1). The mobility and routine activity level of animals may be positively linked to the amount of 'metabolic machinery' that is maintained and thereby the level of resting metabolic rate (e.g. Bennett, 1991; Reinhold, 1999), which in turn is negatively related to b, according to the MLB hypothesis. These relationships are further corroborated by b being near 1 in plants (Fig. 5) and relatively sedentary scorpions ($b = 0.90$; Lighton *et al.*, 2001), araneid spiders ($b = 0.96$; Anderson & Prestwich, 1982), turtles ($b = 1.06$; Trullas, Spotila & Paladino, 2006) and boid snakes ($b = 1.18$; Glazier, 2005) and in dormant insects, mammals (Fig. 6) and birds (Glazier, 2008), all of which have relatively low metabolic levels. The nearly

isometric metabolic scaling of plants represents a nearly linear continuation of the inverse relationship between b and L seen in animals (Fig. 5). Low availability of environmental resources may also select for a low L, which, according to the MLB hypothesis, should be linked to a relatively high b. As predicted, desert mammals with relatively low resting and field metabolic rates exhibit higher scaling exponents than do mammals from more mesic habitats (Nagy *et al.*, 1999; Glazier, 2005).

Additional data support the MLB hypothesis. For example, ectothermic mammal newborns and bird hatchlings, which have relatively low levels of maintenance metabolism, exhibit isometric or nearly isometric metabolic scaling ($b \sim 1$), as predicted (Glazier, 2005). The MLB hypothesis further predicts that higher ambient temperatures causing higher metabolic rates in ectothermic organisms should be associated with lower scaling exponents. As expected, in a crustacean study that is the most extensive survey yet done of temperature effects on the interspecific allometry of metabolism (Ivleva, 1980), the scaling exponent (based on wet body mass) decreases significantly with habitat temperature ($b = 0.781 \pm 0.016$, 0.725 ± 0.086, 0.664 ± 0.026 at temperatures of 20, 25 and 29°C and $N = 247$, 249 and 212, respectively, means ±95% C.L.). Furthermore, the scaling exponent at the highest temperature and level of resting metabolic rate is not significantly different from 2/3, as predicted. However, mixed results have been found for other less extensive studies of the effects of temperature on interspecific and intraspecific metabolic scaling relations (Glazier, 2005, 2009*b*; see also Section VI).

(4) Positive relationships between b and L for metabolic rates during activity

As predicted by the MLB hypothesis, insects, birds, and mammals show significantly higher b values when flying or running ($b = 1.08$, 0.88, and 0.87, respectively) than when resting ($b = 0.66$, 0.64 and 0.68, respectively: Fig. 6A, C, D; Table 1; Glazier, 2008). Also as predicted, the b value during exercise is closer to 1 in ectothermic insects than in endothermic birds and mammals, presumably because they have lower surface-related thermoregulatory costs (scaling as $M^{2/3}$) that would lower b (see Section IV). The temporary endothermy of some insects during flight does not invalidate this conclusion because heat production in insects is a byproduct of muscular activity rather than being an independent, competing energy cost (see Heinrich, 1993). A further observation corroborating the MLB hypothesis is that the b value of maximal active metabolic rate is higher in athletic *versus* non-athletic mammals (Weibel *et al.*, 2004) and fishes (Killen *et al.*, 2007). Contrary to Gillooly & Allen (2007), the relatively high b value of exercising animals appears not to be a simple temperature effect for several reasons (Glazier, 2008, 2009*a*; White *et al.*, 2008), including that ectothermic animals (e.g. fish) that show little change in body temperature during exercise still show a significant increase in b with increased muscular activity (Glazier, 2005, 2009*a*).

(5) Overall U-shaped relationship between b and L for all levels of activity

As predicted, both insects and mammals show approximately U-shaped (or possibly V-shaped) relations between the scaling exponent b and metabolic level L over all activity states from minimal (dormancy) to maximal (strenuous exercise) (compare Fig. 2 with Fig. 6A, C, D; based on data in Table 1). These highly regular relationships are seen whether L is estimated as log a (Fig. 6C), or as the mass-specific metabolic rate at the midpoint of each regression line (Fig. 6D). Also in both taxa, b varies between approximately 2/3 (for resting metabolic rates) and 1 (for minimal and maximal metabolic rates), as predicted. Diapausing insect pupae and deeply hibernating mammals with body temperatures of 5°C both have extremely low metabolic rates, which, as expected, scale nearly in direct proportion to body mass ($b = 1.013$ and 0.941, respectively). Actively flying insects and running mammals have very high metabolic rates that also scale with a power of almost 1 ($b = 1.08$ and 0.87, respectively), as predicted. Intermediate scaling exponents are observed for animals that have metabolic rates between those in the resting and deeply dormant states, as expected (e.g. $b = 0.939$ for resting insect pupae; $b = 0.794$ for shallowly hibernating mammals with body temperatures of 20°C). Note that these U-shaped patterns remain even if somewhat different b values given in the literature for resting metabolic rates of insects and mammals are used (see Fig. 6 legend). Indeed, the b value (0.717) for winged insects based on data in Chown *et al.* (2007) is within the 95% confidence limits of the value (0.662) based on data of Niven & Scharlemann (2005) that is featured in Fig. 6. Furthermore, the b values for winged and wingless insects derived from the data of Chown *et al.* (2007) (see X symbols in Fig. 6C, D) are negatively related to metabolic level, and fit in with the overall U-shaped pattern, as predicted by the MLB hypothesis (see also Section V.3 and Table 1).

These U-shaped patterns are clearly not the result of an autocorrelation effect because b and L_m are not autocorrelated at all, and because the autocorrelation effect incorrectly predicts that b and L_a should be negatively and monotonically correlated for the mammals, but positively and monotonically correlated for the insects because the intercepts are at the bottom end of the size range for the mammals, and at the upper end of the body size range for the insects (see Fig. 6A and Section V.1*b*). Contrary to these supposed autocorrelation effects, b and L_a show an approximately U-shaped relationship in both insects and mammals, as predicted by the MLB hypothesis. Thus, the position of the intercept with respect to the body-size range does not significantly affect the observed dependence of b on L_a (as claimed by Peters, 1983). That the autocorrelation effect has an insignificant influence on the relationships observed here between b and L is not surprising because most of the scaling lines examined here do not cross, and thus have distinctly different elevations regardless of what body mass is used for estimating metabolic level (see Figs 3A, B, 4, 6A, B; see also Glazier, 2008, 2009*b*).

Birds exhibit a similar U-shaped relationship between b and L (Glazier, 2008). Therefore, the concave function between b and L that is predicted by the MLB hypothesis is supported by data from three independently evolved, ecologically dominant taxa, thus suggesting that it may be widely applicable. However, this pattern is expected to be less pronounced or not seen at all in organisms that show little or no activity (e.g. plants and highly sedentary animals). In these organisms, b should always be relatively high (approaching 1).

VI. THE MLB HYPOTHESIS REQUIRES FURTHER DEVELOPMENT AND TESTING

Although the MLB hypothesis appears to have considerable predictive power, it still requires further development and testing. The MLB hypothesis represents a general conceptual framework that needs to be fleshed out in several ways. Here some potentially useful areas for future research regarding the MLB hypothesis, and metabolic scaling in general, are posed as questions.

First, what are the various ecological (ultimate, evolutionary) factors that affect the overall metabolic level (L), and in turn the metabolic scaling exponent (b), of various groups of organisms? Mobility, activity level, temperature and resource availability have been implicated in this paper (see Sections V.3–5), but many others may also be involved (*cf.* Speakman, 2000; McNab, 2002, 2008, 2009; Lovegrove, 2003, 2004; White & Seymour, 2004; Enquist, Tiffney & Niklas, 2007*c*; Seibel & Drazen, 2007; White *et al.*, 2007*a*; Killen *et al.*, 2008). For example, frequency of feeding has been shown to be important in snakes because it is positively correlated with the masses of metabolically expensive visceral organs, and thus resting metabolic rate (Secor & Diamond, 2000). Furthermore, the resting metabolic level of boid snakes has been shown to be negatively related to their metabolic scaling slope, as predicted by the MLB hypothesis (Glazier, 2009*c*). Temperature may also be an especially interesting factor to investigate because the literature reveals diverse kinds of associations between b and temperature (reviewed by Glazier, 2005), possibly because temperature affects multiple components of metabolic rate that may scale differently with body mass (*cf.* Darveau *et al.*, 2002). For example, increasing temperature may increase maintenance costs, thus causing the scaling exponent to approach 2/3, but it may also increase growth and activity, thus causing the scaling exponent to approach 1 (*cf.* Section IV and below). Other possible causes of variable effects of temperature on the metabolic scaling exponent are discussed by Glazier (2005).

Second, what are the molecular, physiological and (or) structural (proximate, functional) mechanisms underlying variation in metabolic level? Biochemical properties of cell membranes may influence the metabolic rates of cells and whole organisms (Hulbert & Else, 2005; Demetrius, 2006; Hulbert *et al.*, 2007; but see Valencak & Ruf, 2007; Haggerty *et al.*, 2008), as may the size and surface-area to volume ratio of cells (Davison, 1955; Kozłowski *et al.*, 2003*a*). Variation in resting metabolic rates of various vertebrate animals has been linked to the relative proportion of body mass that consists of metabolically expensive tissues (e.g. Daan, Masman & Groenewold, 1990; Konarzewski & Diamond, 1995; Secor & Diamond, 2000; McNab, 2002; Nespolo *et al.*, 2002; Song & Wang, 2006). In addition, some investigators have claimed that metabolic level or scaling is related to the structural complexity of organisms (e.g. Zotin, 1990; Atanasov & Dimitrov, 2002; McCarthy & Enquist, 2005).

Third, how does metabolic level precisely affect the scaling slope? At which metabolic levels should the influence of one of the postulated physical boundary constraints predominate over the other? Why does this happen at a specific metabolic level? And why is the relationship between b and L continuous and not sharply divided between 2/3- and 1-power scaling? A quantitative, mechanistic theory is needed that explains how the influences of volume and surface-area constraints (as represented by p and $p-1$ in eq. 2), and (or) other possible constraints (e.g. those related to transport networks) interact at different metabolic levels to produce the varying metabolic scaling relationships that are observed. This theory should also be able to predict the relative influences of surface-dependent fluxes of resources, wastes and heat on metabolic scaling, which may be taxon-specific. For example, surface-related heat loss appears to play an important role in the near-2/3-power scaling of metabolic rate in resting and cold-exposed endothermic birds and mammals (Glazier, 2008), as postulated long ago by Rubner (1883). In both birds and mammals, external surface area scales to the 2/3-power, as expected (Calder, 1984; Reynolds, 1997). However, fluxes of nutrients, respiratory gases and (or) waste materials across internal and (or) external surfaces probably play a more important role in ectothermic organisms (Glazier, 2005).

Future theory should also predict why the empirical relationships between b and L (Figs 3, 5, 6) suggest that volume-related (M^1) scaling predominates at metabolic levels below approximately 0.05 ml O_2 h^{-1} and above approximately15 ml O_2 h^{-1} (standardized to 1 g body mass), whereas surface-area-related scaling ($M^{2/3}$) appears to predominate from near 0.5 to 5 ml O_2 h^{-1} (Figs. 3C, 5A, 6C; Table 1). Something fundamental appears to be occurring because intraspecific metabolic scaling relationships show a similar pattern. For example, b is significantly negatively related to L for the intraspecific scaling of vertebrate resting metabolic rate (Fig. 7), and the L values at which b approaches 1 and 2/3 are similar to those observed for interspecific relationships in animals and plants. Moreover, the empirically determined linear regression equations are remarkably similar for the vertebrate intra- and interspecific resting metabolic rate relationships [intraspecific: $b = 0.737 - 0.102(\log a)$; interspecific: $b = 0.733 - 0.105(\log a)$]. Similar equations are seen for all of the animal interspecific scaling relationships studied here [$b = 0.716 - 0.093(\log a)$ (Fig 5A)] and for all the animals and plants taken together [$b = 0.707 - 0.140(\log a)$]. Both the intercepts and slopes of all of these regressions have broadly overlapping 95% confidence limits,

indicating that they are not significantly different [log *a*: 0.700−0.774, 0.664−0.802, 0.676−0.756, 0.654−0.760; *b*: −0.134(−0.070), −0.180(−0.030), −0.137(−0.049), −0.185(−0.095), respectively]. For all of these organisms at rest, both within and across species, the scaling exponent decreases approximately $1/10^{th}$ of a power for every 10-fold increase in metabolic level. These quantitative similarities suggest that the constraints identified by the MLB hypothesis appear to have very regular and possibly ubiquitous influences on metabolic scaling.

To understand further why there appears to be a continuous relationship between *b* and *L*, it may be helpful to realize that species in a taxon (or individuals in a species) represent a cloud of different metabolic rates, rather than adhering strictly to a linear metabolic level calculated by statistical regression. When most species in a taxon (or individuals in a species) have metabolic rates that place them close to a boundary constraint, the overall scaling slope will be mainly influenced by that constraint. However, intermediate scaling relationships are expected when relatively few species have metabolic rates close to either boundary, or when there are several species close to both boundaries. The relative influence of a boundary constraint on a scaling relationship may thus depend on the proportion of species (or individuals) that have metabolic rates near that boundary limit.

Fourth, how do resource demand and supply interact mechanistically to produce the diverse metabolic scaling relationships predicted by the MLB hypothesis (Fig. 2) or other models (e.g. Darveau *et al.*, 2002)? This interaction may depend critically on whether a specific level of metabolic activity is being sustained in the long-term, thus requiring continual energy uptake (income), or is operating in the short-term, thus requiring only temporary energy stores (capital). If metabolic activity largely depends on stored capital, as during bouts of dormancy or strenuous exercise, demand may predominate, thus causing volume-related isometric scaling (as M^1), whereas if it largely depends on continual energy income, as during sustained routine activity, supply may predominate, thus causing surface-area-related or resource-transport-network-related allometric scaling (as $M^{<1}$, such as $M^{2/3}$ or $M^{3/4}$).

The relative influences of demand and supply may also depend on the surface area to volume ratio of an organism, either internally or externally. For example, plants may exhibit metabolic scaling slopes near 1, not only because of their low metabolic levels, but also because their leafiness gives them relatively high external surface areas that make it less likely that supply rates are limiting. However, if this is true, why do growth (production) rates in plants appear to scale allometrically, often near the 3/4-power (Niklas, 1994, 2004; Enquist *et al.*, 1999, 2007*b*, *c*; Brown *et al.*, 2004*b*; Muller-Landau *et al.*, 2006), whereas their respiratory metabolic rates appear to scale nearly isometrically (Reich *et al.*, 2006)? Perhaps maintenance metabolism is volume-related in plants, whereas growth-related photosynthesis is more a function of fluxes of nutrients and solar energy across surfaces or through transport networks (see also Section VII; *cf.* West *et al.*, 1999*a*; de los Santos, Pérez-Lloréns & Vergara, 2009; Koyama & Kikuzawa, 2009; but see Jin *et al.*, 2008).

Fifth, during strenuous activity, does resource demand actually dictate resource supply (as assumed by the MLB hypothesis), rather than the reverse (as claimed by Darveau *et al.*, 2002; Agutter & Wheatley, 2004; Painter, 2005*a*; Turner, Hulbert & Else, 2006)? In mammals, the similar scaling of maximal active metabolic rate ($b = 0.87$) and that of various supply factors during exercise [e.g. heart-beat frequency per unit mass ($b = 0.85$: Weibel & Hoppeler, 2005), muscle capillary number ($b = 0.79–0.93$: Hoppeler *et al.*, 1981), total volume of muscle capillaries ($b = 0.98$: Hoppeler & Flück, 2002; Weibel & Hoppeler, 2005), volume of intramuscular energy-supplying mitochondria ($b \sim 0.9$: Hoppeler & Flück, 2002; Weibel & Hoppeler, 2005), and the total volume of stored high-energy lipid in muscle tissue ($b = 0.96$: Hoppeler & Flück, 2002)] may be the result of supply having adjusted to demand, but it is difficult to determine direction of causality from correlations only. Any attempts to resolve this problem should distinguish the aerobic and anaerobic components of maximal metabolic rate (*cf.* Goolish, 1991). The MLB hypothesis predicts that the scaling exponent of total metabolic rate during maximal activity should approach 1, but *b* for the aerobic component may be more related to the surface-area-related uptake of oxygen than is *b* for the anaerobic component. This may be why the activity of muscle enzymes involved in oxidative metabolism scale as $M^{0.79–0.94}$ (Emmett & Hochachka, 1981), whereas the activity of muscle enzymes functioning in anaerobic metabolism scale as $M^{\geq 1}$ (Childress & Somero, 1990; Withers, 1992).

Sixth, how hard are the boundary constraints of the MLB hypothesis? The existence of some metabolic scaling slopes above and below the predicted 2/3–1 range (see Section V) may be the result of sample or measurement error, the taxon-specific alteration of a boundary constraint, or the adaptive bypassing of a constraint. For example, scaling as $M^{2/3}$ may be altered if the surface area of a set of organisms scales differently than expected. In fact, Seibel (2007) has suggested that differences in the scaling of surface area have affected the metabolic scaling exponent in cephalopods (see also Rosa, Trueblood & Seibel, 2009). The current version of the MLB hypothesis assumes that the 2/3-power scaling of surface area represents an extreme, idealized lower limit for the metabolic scaling exponent, but it can be modified to include other scaling relations for surface area, as may be pertinent for specific taxa. Scaling as M^1 may also be altered if body composition and (or) the proportion of metabolically active tissues or energy-expensive processes vary with body size (*cf.* Glazier, 2005).

Seventh, can the MLB hypothesis be applied to other organisms besides animals and plants? The MLB hypothesis may explain why low-energy prokaryotic unicells tend to have metabolic scaling exponents near 1, whereas high-energy eukaryotic unicells have exponents significantly less than 1 (Makarieva *et al.*, 2005*b*) and that are negatively related to metabolic level (Glazier, 2009*b*). The MLB hypothesis also

predicts that b should be near 1 in low-energy, filamentous fungi, but unfortunately no data are currently available to test this hypothesis (Glazier, 2005).

Eighth, can the MLB hypothesis explain log-log metabolic scaling that is nonlinear, as occurs for many intraspecific relationships and some interspecific relationships as well (reviewed in Glazer, 2005)? The MLB hypothesis may provide a useful general theoretical framework for explaining many nonlinear scaling patterns, provided that they involve either changes in metabolic level that, in turn, affect the scaling exponent, or changes in the relative contribution of volume- *versus* surface-area-related processes to metabolic rate. For example, during ontogeny many birds and mammals shift from being low-energy ectotherms to high-energy endotherms. Therefore, according to the MLB hypothesis, these increases in L should be accompanied by decreases in b, which is what is typically observed (see Glazier, 2005). Ontogenetic changes in the contribution of various volume-related processes (e.g. growth) to the total metabolic rate may also cause shifts in metabolic scaling (*cf.* Riisgård, 1998; Glazier, 2005; Czarnołęski, *et al.*, 2008; see also below). In addition, Glazier (2008) has suggested that large mammals may have steeper metabolic scaling slopes than smaller mammals because their more effective insulation may reduce the effect of surface-area-related heat loss relative to that of volume-related tissue demand for energy. However, other plausible explanations are possible (Bejan, 2001; Makarieva, Gorshkov & Li, 2003; Glazier, 2005; White & Seymour, 2005*a*; Painter *et al.*, 2006; da Silva, Barbosa & Silva, 2007; Clauss *et al.*, 2008; Packard & Birchard, 2008).

Ninth, how is the MLB hypothesis related to other models and approaches? How are they similar and how are they different? Further development of metabolic scaling theory may benefit from integrating the most useful parts of various models, including the MLB hypothesis. For example, the MLB hypothesis appears to complement the boundary approach of Makarieva *et al.* (2005*b*, 2006*b*) because both approaches predict that minimal inactive and maximal active metabolic rates should scale as $M^{\sim 1}$ (or as $M^{\sim 0}$ on a mass-specific basis), as is observed (Fig. 6; see also Section IV). The MLB hypothesis also predicts that the metabolic scaling slope should vary between 2/3 and 1, as do some other models (e.g. Kooijman, 2000; Kozłowski *et al.*, 2003*a*; Demetrius, 2006; van der Meer, 2006). The models of Kozłowski *et al.* (2003*a*) and Demetrius (2006) focus on the body-size scaling of cell size and number and of energy transduction rates in cell membranes, respectively, both of which may contribute to the variation in b that has been observed. By contrast, the theory of dynamic energy budgets (DEB) posits that the scaling of metabolic rate depends on the relative importance of physiological processes that are proportional to surface area or volume (Kooijman, 2000; van der Meer, 2006), as does the MLB hypothesis. However, DEB theory and other models do not examine how the effect of these processes should vary with metabolic level L, though some models predict that b should be higher during exercise than during rest (see Section III). Consequently, the MLB hypothesis makes further successful predictions that are not explicitly stated by the above models: e.g. that the metabolic scaling slope b should vary with metabolic level L according to a concave function, that L and thus b are related to various ecological factors, and that b for maximal active metabolic rate should depend on athleticism (muscularity) and relative maintenance costs, as observed here and elsewhere (Glazier, 2008; Killen *et al.*, 2007, 2008).

The MLB hypothesis also predicts that b should approach 1 whenever volume-related resource demand becomes more important than surface-area-related resource supply (or waste disposal), and not just at the lowest and highest metabolic levels that life can achieve, as argued by Makarieva *et al.* (2005*b*, 2006*b*). For example, growth and heat production associated with feeding (the specific dynamic action, SDA) appear to involve metabolic processes that pervade all of the tissues of the body, and thus should be strongly volume-related. As expected, the scaling of metabolism associated with growth and SDA both tend to be nearly isometric (Glazier, 2005, 2006; Czarnołęski *et al.*, 2008; Secor, 2008). Furthermore, the scaling exponent of maximal metabolic rate in endotherms need not always be near 1, as predicted by Makarieva *et al.* (2005*b*, 2006*b*), but rather should depend on whether it is the result of exercise ($b \sim 1$, as a result of volume-related costs of muscular activity) or cold exposure ($b \sim 2/3$, as a result of surface-related heating costs), as has been observed (Glazier, 2008).

By focusing on how boundary constraints delimit the diversity of metabolic scaling slopes, the MLB hypothesis may also complement other models that focus on the average tendencies of these slopes. Proponents of the resource-transport network model of West *et al.* (1997) have emphasized that this model is primarily intended to explain the "central tendency" of 3/4-power scaling (e.g. Marquet, Labra & Maurer, 2004; Allen & Gillooly, 2007; Enquist *et al.*, 2007*a*, *c*). After all, the mean slope of both inter- and intraspecific metabolic scaling is often near 3/4 (e.g., Peters, 1983; Withers, 1992; Savage *et al.*, 2004; Glazier, 2005; Moses *et al.*, 2008). Perhaps 3/4-power scaling represents some kind of intermediate optimum that is bounded by the physical constraints specified by the MLB hypothesis. However, Makarieva *et al.* (2005*a*, 2008) have claimed that the "optimal" mass-specific metabolic rate of animals, plants and microbes, encompassing 20 orders of magnitude in body mass, varies over a much narrower range than that expected from the 3/4-power law. In addition, an analysis of the extensive compilation of intraspecific metabolic scaling relationships provided by Glazier (2005) reveals that, although the mean exponent for studies with body-mass ranges (M_R) exceeding two orders of magnitude is not significantly different from 3/4 (0.79 ± 0.07; Moses *et al.*, 2008), over 70% of the individual b values are significantly different from 3/4 (Glazier, 2005). Indeed, significant deviations from 3/4-power scaling become relatively more frequent as the reliability (M_R) of the scaling relationships increases (50.2% for all studies; 71.1% for only the studies

with $M_R > 1.5$ orders of magnitude; 88.5% for only the studies with $M_R > 2.5$ orders of magnitude). This pattern does not provide strong support for the optimality of 3/4-power scaling, either.

Nevertheless, the MLB hypothesis can explain why the mean *b* value may be near 3/4 without invoking any central optimal tendency (at least for *b*, though there may be one for *L*: *cf.* Makarieva *et al.*, 2005*a*, 2008). Since most taxa exhibit an intermediate metabolic level (as shown by Makarieva *et al.*, 2005*a*, 2008; and as supported by the normal distribution of L_a values compiled by Peters, 1983), then the MLB hypothesis predicts that most taxa should also show a scaling exponent between 2/3 and 1, which, of course, includes 3/4. In short, the MLB hypothesis can explain both the average tendency of near-3/4-power scaling and deviations from it.

More precisely the MLB hypothesis predicts that, for a sample of scaling relationships including the full range of existing metabolic levels, the mean scaling exponent should actually be near 0.833 (half-way between the postulated extreme exponents of 2/3 and 1). This prediction is supported by the linear regression equation reported above for the correlation between *b* and log *a* among the interspecific scaling relationships for resting metabolic rate of all of the animals and plants examined in this study, which include minimum and maximum scaling exponents of 0.640 and 1.062. The mean log *a* of all 16 of these scaling relationships is -0.898, which according to the *b versus* log *a* regression equation, predicts a slope of 0.833 (which corresponds with the calculated mean exponent $\pm$0.067 95% confidence limits), exactly as expected. The predicted mean *b* value of 0.833 is also within the 95% C.L. of the mean slope calculated for the Glazier (2005) sample of intraspecific scaling relationships with a mass range over two orders of magnitude (see above). However, the 'average' slope that is observed will depend on the distribution of metabolic levels exhibited by the sampled taxa. This may explain why Withers (1992) reported a modal exponent (0.814) in his diverse compilation of interspecific metabolic scaling relationships that is somewhat higher than the value (0.74) reported by Peters (1983) in his more frequently cited, but biased analysis, which was based on a sample that included a large proportion (44%) of high-energy endotherms that are expected to have relatively low exponents near 2/3, according to the MLB hypothesis (see also Glazier, 2005).

VII. IMPLICATIONS OF THE MLB HYPOTHESIS

If true, the MLB hypothesis has several implications for our understanding of allometric scaling relationships and their application to a variety of biological problems. First, biologists should no longer assume that metabolic rate scales universally as $M^{3/4}$. Many theories and empirical analyses that have made this assumption should be re-evaluated (see also below). Nutritional requirements of animals and humans are frequently estimated using a 'metabolic weight' of $M^{3/4}$ (e.g. Kleiber, 1961; Balnave, Farrell & Cumming, 1978; Vander Tuig, Romsos & Levielle, 1980; Baker *et al.*, 1996; Rucker, 2007; Knap, 2009). Similarly, to discern the effects of various factors (e.g. temperature) on a biological process, the effect of body size is often 'removed' by assuming 3/4-power scaling (e.g. Ørskov & MacLeod, 1982; Gillooly *et al.*, 2001, 2006, 2007; Brown *et al.*, 2004*b*; Brougher, Douglass & Soares, 2005; West & Brown, 2005; Allen *et al.*, 2006; Enquist *et al.*, 2006). However, since metabolic scaling is highly variable, it would be better to use analysis of covariance (Thonney *et al.*, 1976; White *et al.*, 2007*b*), or an empirically determined metabolic scaling relationship to control for the effect of body size (e.g. Robinson *et al.*, 1983; O'Connor *et al.*, 2007; Pennington & Meehan, 2007; de Castro & Gaedke, 2008; Downs *et al.*, 2008). Furthermore, although developing models based on 3/4-power scaling may give adequate first approximations of large-scale phenomena, they may be incapable of making precise predictions at smaller scales (*cf.* Brown *et al.*, 2004*a*; O'Connor *et al.*, 2007; Martinez del Rio, 2008; Allen & Gillooly, 2009). These models may be improved by using a range of scaling exponents from 2/3 to 1 for organisms with different metabolic (activity) levels, as predicted by the MLB hypothesis.

Second, the slope and elevation of metabolic scaling relationships should no longer be studied in isolation. As predicted by the MLB hypothesis, *b* and *L* covary in a highly regular way within broad boundary limits, and there is growing evidence that *L* is, in turn, related to a variety of biochemical, structural, and ecological factors. This systematic, predictable variation demonstrates that there is no single universal scaling law, but rather that metabolic scaling is highly malleable both physiologically and evolutionarily, albeit within the boundaries of multiple, generally applicable, physical constraints.

Focus on both *b* and *L* as interrelated scaling parameters may also help to eliminate some conceptual difficulties associated with the application of some theoretical models of metabolic scaling. For example, many workers have interpreted the model of West *et al.* (1997) as predicting that metabolic rate should scale to the 3/4 power, regardless of *L* (see e.g. Gillooly *et al.*, 2001, 2006; Savage *et al.*, 2004; West, Brown & Enquist, 2004; Kerkhoff *et al.*, 2005; Price *et al.*, 2007; and Section III.2). However, this model assumes that metabolic rate is constrained by an optimal nutrient-supply network, which cannot be simultaneously true (at least in the same optimal way) at different metabolic levels. For example, if the scaling of resting metabolic rate is the result of resource-supply limits, as often assumed, the original model of West *et al.* (1997) cannot also be used to explain the scaling of maximal or minimal metabolic rates that exceed or are below these postulated supply limits (*cf.* Darveau *et al.*, 2002; Kozłowski *et al.*, 2003*b*; Agutter & Wheatley, 2004; Glazier, 2005; Suarez & Darveau, 2005; O'Connor *et al.*, 2007). For the same reason, it cannot be used, without major modification, to explain the scaling of taxa with different metabolic levels (e.g. endo- *versus* ectotherms). The MLB hypothesis avoids this conceptual problem by allowing the

relative influences of resource supply and demand to vary with L (see also Glazier, 2005).

Third, the MLB hypothesis implies that a 'symmorphosis' or co-adapted matching of resource demand with the capacities of resource-supply and waste removal (Weibel, 2000) is not a universal, always-present property of life, but varies with physiological state and metabolic level. This is because organisms may at times have resource demands that are temporarily below (e.g. during dormancy) or above (e.g. during bursts of maximal activity) the capacity of the body to deliver resources or remove wastes. As noted in Sections IV and VI, these episodes of 'asymmorphosis' are permitted by accumulated body stores of resources and temporary tolerance to waste accumulation.

The relative importance of symmorphosis *versus* asymmorphosis may also vary among different kinds of organisms with different lifestyles and overall metabolic intensities. In low-energy organisms, such as many sedentary ectothermic animals that spend most of their time at rest (in maintenance mode), asymmorphosis (resource demand < resource-supply and waste-removal capacities) may predominate over symmorphosis because resource-supply and waste-removal capacities are geared for supporting relatively infrequent high-energy non-maintenance activities, such as locomotion and food-processing. Similarly in plants, asymmorphosis may often apply to their low-energy respiratory metabolism, whereas symmorphosis may be more important for relatively high-energy growth-related photosynthesis (see also Section VI). By contrast, in high-energy organisms, such as mobile winged insects and endothermic birds and mammals, symmorphosis (resource demand ~ resource-supply or waste-removal capacities) should predominate over asymmorphosis because the more frequently or continually high metabolic activity of these animals is more likely to be limited by rates of resource supply or waste removal. This may be why the concept of symmorphosis works relatively well in high-energy mammals, the test group for this concept (Weibel, 2000), but its applicability to low-energy organisms has not yet received sufficient attention. The above view is also consistent with the heavier reliance of low-energy ectothermic animals and plants on stored resources (capital) to support their metabolic needs, as compared to high-energy birds and mammals that depend more on relatively frequent, higher levels of resource uptake (income) and waste removal (*cf.* Section VI; Bonnet, 1998).

Fourth, since all biological processes depend on metabolic energy, the MLB hypothesis implies that the body-size scaling of these various processes should also depend on metabolic level and the kind of metabolic rate (i.e. resting, routine, field, or maximal) that is most relevant to them. As demonstrated here and elsewhere, the scaling of metabolic rate varies with physiological state, environmental conditions and taxonomic affinity (Glazier, 2005, 2008, 2009*a*, *b*, *c*; White & Seymour, 2005*a*; White *et al.*, 2006, 2007*b*), and so should that of other energy-dependent processes. This implication could have repercussions in many areas of biological research that make use of scaling relationships, including physiology, developmental biology, gerontology, toxicology, nutrition, medicine, ecology, and evolutionary biology.

Although the application of the MLB hypothesis to the scaling of various biological processes will be examined more fully elsewhere, I mention here a few relevant observations to show that research along these lines should be worthwhile. Although near-3/4 or 1/4-power scaling appears to be common for a variety of biological phenomena, and has been claimed as support for the model of West *et al.* (1997) (see Section I), this pattern is also consistent with the MLB hypothesis and other models (e.g. Kozłowski *et al.*, 2003*a*, *b*; Demetrius, 2006; van der Meer, 2006; see also Section VI; O'Connor *et al.*, 2007). More importantly, several data sets suggest that the scaling of various biological processes may vary in accordance with variation in the scaling of metabolic rate, as predicted by the MLB hypothesis. For example, as expected from the near-2/3-power scaling of their resting metabolic rate (Glazier, 2005, 2008; White *et al.*, 2006, 2007*b*; Downs *et al.*, 2008), several analyses for birds and mammals have shown that foraging rate, ingestion rate, defecation rate, respiration frequency, respiration-cycle duration, dive duration and depth, time to puberty, life expectancy, home-range size, population density, birth rate, production rate, and doubling time for individual and population growth rates scale approximately as multiples of a third-power (1/3, 2/3), or can be derived from such scaling (e.g. Western & Ssemakula, 1982; Peters, 1983; Calder, 1984; Maurer, 1996; Charnov, 2001; Frappell, Hinds & Boggs, 2001; Dobson, Zinner & Silva, 2003; White & Seymour, 2003; Economo, Kerkhoff & Enquist, 2005; Halsey, Butler & Blackburn, 2006; Sibly & Brown, 2007). Other processes that show approximately 3/4-power scaling in mammals may be more related to the scaling of field metabolic rate ($b \sim 3/4$) than to the scaling of resting metabolic rate (*cf.* Savage *et al.*, 2004; Carbone, Teacher & Rowcliffe, 2007*b*), a hypothesis requiring testing.

The scaling of various traits that are likely linked to metabolic rate also differs between resting and active animals, and between high-energy endotherms and low-energy ectotherms, as predicted by the MLB hypothesis. For example, in birds and mammals, mass-specific heartbeat frequency scales more steeply during exercise (birds: $b = 0.84–0.85$; mammals: $b = 0.81–0.85$) than during rest (birds: $b = 0.77–0.79$; mammals: $b = 0.73–0.75$) (Peters, 1983; Weibel & Hoppeler, 2005), as does the scaling of metabolic rate (see Section V.4). In addition, the scaling slope of ingestion rate in carnivorous vertebrates is significantly lower in endotherms ($b = 0.692 \pm 0.024$ 95% C.L.) than in ectotherms ($b = 0.820 \pm 0.062$), and both are significantly different from 3/4 (Farlow, 1976). A similar difference exists for egestion rate ($b = 0.79$ for birds and mammals; $b = 1.18$ for reptiles and amphibians: Peters *et al.*, 1996). The scaling of offspring investment (expressed as mass or volume per clutch or litter) is also less steep in endothermic birds ($b = 0.52–0.74$) and mammals ($b = 0.70–0.84$) than in reptiles ($b = 0.88$) and other aquatic ectotherms ($b = 0.92$) (Blueweiss *et al.*, 1978; Reiss, 1989; Dol'nik, 2000; Hendriks &

Mulder, 2008). In addition, annual reproductive investment scales significantly less steeply in mammals ($b = 0.685$) than in plants ($b = 0.834$) (Falster, Moles & Westoby, 2008), in accordance with the higher metabolic level and reproductive power of mammals, as predicted by the MLB hypothesis.

By contrast, the scaling exponent for dive duration is higher in endothermic birds and mammals than in ectothermic turtles and other reptiles ($b \sim 1/3$ *versus* $b \sim 0$: Halsey *et al.*, 2006; Brischoux *et al.*, 2007). This pattern is consistent with the MLB hypothesis because dive duration is expected to scale inversely with metabolic rate, which scales approximately as $M^{2/3}$ in endotherms (see above) and nearly as M^1 in low-energy ectotherms (especially turtles: Trullas *et al.*, 2006). Similarly, reproductive rate scales more steeply in mammals ($b = -0.39$) than in ectothermic vertebrates ($b = -0.19$) and invertebrates ($b = -0.15$) (Hendriks & Mulder, 2008). In addition, the scaling exponent of maximum longevity is higher in endothermic passerine birds (0.274), nonpasserine birds (0.174) and mammals (0.193) than in ectothermic reptiles (0.137) and amphibians (0.069), and is positively related to the metabolic level of these taxa (Fig. 8). This pattern is also predicted by the MLB hypothesis, assuming that the mass-independent maximum longevity of a taxon is negatively related to its metabolic level, as can be seen by comparing the proportionality constants (a) of the scaling relationships of maximum longevity and metabolic rate ($r = -0.907$, $P = 0.033$; data from Table 1; de Magalhães, Costa & Church, 2007; McNab, 2009). Differences between endotherms and ectotherms for other scaling relationships are described by Hendriks (2007) and Hendriks & Mulder (2008).

Although more data are needed, the above observations show that the scaling of various biological processes does not universally follow a 3/4-power law, in accordance with that also seen for the scaling of metabolic rate. Furthermore, this variation in the scaling exponent does not appear to be merely the result of statistical error or idiosyncratic effects because it is related to other biologically significant factors in an orderly way (e.g. physiological or taxonomic differences in metabolic intensity, as predicted by the MLB hypothesis). Realizing this may not only enhance our general understanding of biological scaling, but also help to improve the precision and range of applicability of specific scaling models. For example, the predictive accuracy of the offspring-investment model of Charnov & Ernest (2006) improves significantly if one replaces their assumption that resting metabolic rate in mammals scales as $M^{3/4}$ with an apparently more realistic scaling in relation to $M^{2/3}$ (following White & Seymour, 2003, 2005*a*; Glazier, 2008) or $M^{0.68-0.74}$ (following White *et al.*, 2009). It can now better explain why offspring number per year scales with neonate mass ($b = -0.30$) and weaning mass ($b = -0.32$) more nearly as a $-1/3$ power than as a $-1/4$ power. Similarly, White & Seymour (2003) show that predictions of home-range size in mammals are markedly improved, if one assumes that resting metabolic rate scales as $M^{2/3}$, and not as $M^{3/4}$. Assuming that b is always 3/4 may produce misleading analyses of metabolic scaling with temperature as well (Downs *et al.*, 2008). In addition, Weitz & Levin (2006) and Carbone *et al.* (2007*a*) have increased the robustness of their models on the scaling of predator-prey dynamics and of animal abundance, respectively, by including the possibility that the metabolic scaling exponent may differ from 3/4, ranging between 2/3 and 1.

Fifth, the MLB hypothesis shows that it may be possible to construct a general, unifying theory of biological scaling that explains the entire diversity of existing scaling relationships, rather than only an average or idealized scaling relationship. This is made possible by adopting an approach that is in some ways intermediate between two of the most extreme positions taken in the current debate about the theory of metabolic scaling. At one extreme, some workers have advocated the development of a general theory to explain the "central tendency" of metabolic scaling, which is considered to be a universal law (e.g. West *et al.*, 1997, 1999*b*; Banavar *et al.*, 1999, 2002; Savage *et al.*, 2004; West & Brown, 2005; Allen & Gillooly, 2007; Enquist *et al.*, 2007*c*; Moses *et al.*, 2008). Deviations from the central tendency (quarter-power scaling) are then explained by attributing them to statistical error or idiosyncratic adaptive variation, by including additional factors, or by modifying the theory in relatively minor ways (see Section II; Brown, Enquist & West, 1997; Allen & Gillooly, 2007; Enquist *et al.*, 2007*a*, *c*; Moses *et al.*, 2008). At the other extreme, some have advocated the development of multiple models, specific to particular taxa or conditions, to explain the diversity of metabolic scaling (e.g. Reich *et al.*, 2006; O'Connor *et al.*, 2007; Martinez del Rio, 2008). Alternatively I suggest that a single multiple-factor model, based upon the MLB hypothesis and (or) possibly the further development of other approaches (e.g. Kooijman, 2000; Darveau *et al.*, 2002; Kozłowski *et al.*, 2003*a*, *b*; Suarez & Darveau, 2005; Demetrius, 2006) may provide a suitable theoretical framework for explaining much of the diversity that we see. General theory need not focus only on universal, invariant, or idealized laws of nature, but may also successfully embrace the diverse, variable, actually observed aspects of nature.

VIII. CONCLUSIONS

(1) Contrary to conventional belief, there is no universal 3/4-power law for the body-mass scaling of metabolic rate. The slopes of metabolic scaling vary substantially from approximately 2/3 to 1.

(2) Current theoretical models, as now formulated, cannot explain the substantial variation in metabolic scaling exponents that exists both among taxonomic groups and physiological (activity) states.

(3) A new approach is advocated that involves three major shifts in emphasis: a focus on boundary conditions delimiting the range of possible metabolic scaling relationships, rather than on average tendencies; a focus on how the elevation (metabolic level) and slope (exponent) of these relationships are related, rather than considering them as independent scaling

parameters requiring wholly separate explanations; and a focus on how both external (ecological) and internal (e.g. biochemical, cellular, physiological, and anatomical) factors may affect metabolic scaling, rather than an emphasis only on internal factors.

(4) The metabolic-level boundaries (MLB) hypothesis presented here incorporates all of these shifts in emphasis, and as a result successfully predicts most of the diversity of metabolic scaling relations that has been observed among physiological states and among various taxa of animals and plants. Volume and surface-area constraints (scaling respectively as M^1 and $M^{2/3}$) act as boundary limits on the metabolic scaling slope, and metabolic level determines the relative importance of these constraints. The correctly predicted concave relationship between the scaling slope and metabolic level shows that the scaling of metabolic rate with body mass itself scales with the overall metabolic level of the organisms being examined.

(5) Although further theoretical development and empirical testing are required, the MLB hypothesis may help explain not only variation in metabolic scaling, but also variation in the scaling of other biological processes that depend on metabolic energy.

(6) Numerous lines of evidence supporting the MLB hypothesis suggest that variation in the metabolic scaling exponent is not merely the result of statistical error or secondary causes that are obscuring the primary 'signal' of a single universal scaling law, but rather indicates the contingent operation of multiple boundary constraints that are themselves of primary importance. Recognition of diversity may aid rather than impede the development of a general metabolic theory of biology, as has sometimes been implied in the recent scaling literature.

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