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Beyond the '3/4-power law': variation in the intra- and interspecific scaling of metabolic rate in animals

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ABSTRACT

In this review I show that the '3/4-power scaling law' of metabolic rate is not universal, either within or among animal species. Significant variation in the scaling of metabolic rate with body mass is described mainly for animals, but also for unicells and plants. Much of this variation, which can be related to taxonomic, physiological, and/or environmental differences, is not adequately explained by existing theoretical models, which are also reviewed. As a result, synthetic explanatory schemes based on multiple boundary constraints and on the scaling of multiple energy-using processes are advocated. It is also stressed that a complete understanding of metabolic scaling will require the identification of both proximate (functional) and ultimate (evolutionary) causes. Four major types of intraspecific metabolic scaling with body mass are recognized [based on the power function $R = aM^b$, where R is respiration (metabolic) rate, a is a constant, M is body mass, and b is the scaling exponent]: Type I: linear, negatively allometric ($b < 1$); Type II: linear, isometric ($b = 1$); Type III: nonlinear, ontogenetic shift from isometric ($b = 1$), or nearly isometric, to negatively allometric ($b < 1$); and Type IV: nonlinear, ontogenetic shift from positively allometric ($b > 1$) to one or two later phases of negative allometry ($b < 1$). Ontogenetic changes in the metabolic intensity of four component processes (i.e. growth, reproduction, locomotion, and heat production) appear to be important in these different patterns of metabolic scaling. These changes may, in turn, be shaped by age (size)-specific patterns of mortality. In addition, major differences in interspecific metabolic scaling are described, especially with respect to mode of temperature regulation, body-size range, and activity level. A 'metabolic-level boundaries hypothesis' focusing on two major constraints (surface-area limits on resource/waste exchange processes and mass/volume limits on power production) can explain much, but not all of this variation. My analysis indicates that further empirical and theoretical work is needed to understand fully the physiological and ecological bases for the considerable variation in metabolic scaling that is observed both within and among species. Recommended approaches for doing this are discussed. I conclude that the scaling of metabolism is not the simple result of a physical law, but rather appears to be the more complex result of diverse adaptations evolved in the context of both physico-chemical and ecological constraints.

Key words: allometry, animals, body size, constraints, evolution, metabolism, ontogeny, optimization, respiration, scaling.

CONTENTS

I. INTRODUCTION

A fundamental property of all organisms is their metabolism, which constitutes the collective biochemical processes by which they transform energy and materials to support various life functions and structures. The rate of these metabolic processes can be indirectly estimated by the rate of heat production, or in aerobic organisms by the rate of respiration, i.e. the rate of oxygen consumed or the rate of carbon dioxide released. Estimating metabolic rate is of fundamental importance because it is linked to the rates of many other biological activities at various hierarchical levels of organization, and as such represents a holistic measure of the 'pace of life'. Furthermore, metabolic rate does not vary willy-nilly in organisms, but shows highly regular relationships with other organismal properties, in particular body mass. These relationships are of interest because they suggest that at least some of the variation in metabolic rate that is observed may be explained by relatively simple mechanisms.

Typically the relationship between metabolic (respiration) rate (R) and body mass (M) is expressed as a power function, $R = aM^b$. It has been widely accepted that the scaling exponent b is 3/4, the so-called '3/4-power law' (Brody, 1945; Hemmingsen, 1960; Kleiber, 1961; McMahon & Bonner, 1983; Peters, 1983; Calder, 1984; Schmidt-Nielsen, 1984; Blaxter, 1989; Brown & West, 2000; Savage *et al.*, 2004*b*). However, this scaling law, which is relevant to many areas of biology, is currently the subject of two related controversies. First, is the scaling exponent for

metabolic rate universally 3/4, or may it have other values, such as 2/3 or even 1, in at least some organisms? Second, how is metabolic scaling to be explained?

This review has three major aims. First, I consider whether the scaling exponent of 3/4 is universally applicable to organisms both within and among species. My focus is on animals, though some reference to unicells and plants is also made. In so doing, I show that although 3/4-power scaling appears to be common, a variety of other scaling relationships have also been observed. I pay particular attention to intraspecific metabolic scaling because it has been relatively neglected in the metabolic scaling literature. Following the insightful, but largely forgotten work of Bertalanffy (1957), I propose a tentative classification of intraspecific patterns of metabolic scaling. Second, I review many of the most prominent models that have been offered recently to explain the scaling of metabolism in relation to body mass. In so doing, I advocate a synthetic 'boundary approach' that includes components from multiple existing models. All of these models are judged in terms of their match with existing empirical data. I especially focus on why metabolic scaling relationships may be linear or non-linear, and why the scaling exponent may be 3/4, 2/3 or some other value, both within and among species. Third, I discuss seven general approaches that may help us to advance our still incomplete understanding of the causes of metabolic scaling.

A review of this kind is urgently needed for three major reasons. First, variation in the metabolic scaling exponent has been underappreciated by many investigators, especially theoreticians. This problem has resulted probably because no comprehensive review of metabolic scaling data has appeared in nearly 50 years for intraspecific patterns (since Bertalanffy, 1957), and in 20 years for interspecific patterns (since Schmidt-Nielsen, 1984).

Second, several theoreticians have recently proposed new models without adequately considering or even being aware of the variety of other models or explanations that have been proposed. This 'isolationist' tendency has prevented a holistic understanding of metabolic scaling, and in some cases has even resulted in 're-inventing the wheel'. To counter this tendency, I have classified many of the most promising models in an effort to show how they are related, and to identify gaps in our understanding [see Section III (2)]. This classification reveals that all models proposed to date examine only one or at most a few of the multiple factors potentially affecting metabolic scaling, and thus are often complementary rather than mutually exclusive.

Third, few attempts have been made to compare metabolic scaling at several different scales of observation. Metabolic scaling relationships appear to depend on the range of taxa and body sizes sampled [see also Sections III (4) and IV (2)]. Here I clearly distinguish intra- *versus* interspecific scaling patterns, which is sometimes not done in the literature. This distinction is not only important for analyses of metabolic scaling (*cf.* Heusner, 1982; Wieser, 1984; Norris, 1998; Kooijman, 2000), but also for allometric studies in general (e.g. Gould, 1966; Harvey & Pagel, 1991). Intraspecific metabolic scaling patterns reported in the literature typically involve relationships between metabolic rate and body mass as an organism grows during its lifetime. However, interspecific metabolic scaling patterns involve relationships between metabolic rate and body mass as species transform into new species during evolutionary time. These two kinds of metabolic scaling may involve different mechanisms, and thus are treated separately. However, as I argue in Section IV (2), studies of one kind of scaling may illuminate our understanding of the other.

To avoid misunderstanding, I want to emphasize that this review is not intended to show that the 3/4-power law is completely invalid, but merely to show that other metabolic scaling relationships also occur and deserve our consideration. By finding exceptions, it becomes apparent that the 3/4-power law should at most be regarded as a useful statistical 'rule' or 'trend', rather than as an inviolable law. In addition, my intent is to use existing empirical data to critically assess the nature of metabolic scaling relationships and the models proposed to explain them. My frequent focus on empirical data should not be construed to mean that I oppose seeking general principles to explain metabolic scaling. My approach to generality follows the advice attributed to Albert Einstein: 'Make everything as simple as possible, but not simpler'. Successful general explanations of metabolic scaling may turn out to be quite complex, incorporating multiple mechanisms and constraints, and including the effects of both physical law and context-dependent contingencies. Furthermore, showing that metabolic scaling is variable and based on multiple mechanisms does not necessarily jeopardize the development of a general 'metabolic theory of ecology' that helps explain processes at higher levels of biological organization, including populations, communities and ecosystems, as envisioned by Brown *et al.* (2004*a*, *b*). As a case in point, Banavar *et al.* (2002*a*) have shown that substituting a metabolic scaling exponent of 2/3 in place of 3/4 has little effect on the fit of the growth model of West, Brown & Enquist (2001) to empirical data (see also West, Enquist & Brown, 2002*a*; Ricklefs, 2003). The importance of body-size scaling in understanding processes at many levels of biological organization remains regardless of what the scaling relationships may turn out to be (see Peters, 1983).

Some readers may question the value of arguing about what the metabolic scaling exponent(s) is (are) exactly and how it (they) should be explained. However, if one is interested in using scaling relationships as predictive tools, the exact value of a scaling exponent is critical because even slight differences in the exponent can greatly affect calculated metabolic rates predicted from body mass (Heusner, 1982; Hayssen & Lacy, 1985; Riisgård, 1998; Finn *et al.*, 2002). For example, arbitrarily choosing between slightly different scaling exponents of 0.75 (Kleiber, 1961) and 0.73 (Brody, 1945), as has been done for mammals, affects the predicted metabolic rate by as much as 15 % for a 1 kg animal and 26 % for a 100 kg animal (Hayssen & Lacy, 1985). Furthermore, I disagree with Peters (1983, 1991) who argued that the most profitable use of scaling relationships is as predictive tools, and that we should not waste time on trying to explain why their scaling exponent is 3/4 or some other value. Instead, I agree with Lawton (1991) and others

that a deep understanding of how life is 'designed', functions and evolves requires that we seek a conceptual and mechanistic understanding of the regularities that we observe in nature. Consequently, accurate descriptions of these regularities (including error estimates) are necessary if we are to build realistic theories about how nature works. This point is particularly relevant here, because some recent models attempting to explain metabolic scaling have been based on the assumption that the scaling exponent of 3/4 is universal or nearly so in the living world (e.g. West, Brown & Enquist, 1997, 1999*b*; Banavar, Maritan & Rinaldo, 1999; Dreyer & Puzio, 2001; Rau, 2002; Fujiwara, 2003; Santillán, 2003), which appears not to be true. In addition, the hypothetical formulation of underlying mechanisms increases our ability to make testable predictions, thus advancing our understanding of the natural world more than would otherwise be possible.

II. WHAT IS THE SLOPE OF METABOLIC SCALING RELATIONSHIPS?

(1) Methodological considerations

The metabolic rate of individual organisms can vary in response to many intrinsic and extrinsic factors, especially activity level and ambient temperature. Therefore, activity level and temperature are usually controlled when comparing metabolic rate among different individuals or species. Most analyses of metabolic scaling are based on estimates of maintenance metabolism, i.e. the minimal amount of energy needed to maintain the tissues and essential life functions of an organism without any loss of body mass. In endothermic birds and mammals, 'basal rates of metabolism' are measured in resting adults that are not digesting food, and that are in a thermal environment (neither too cold nor too hot) requiring minimal thermoregulatory demands. In ectothermic animals, 'standard rates of metabolism' are similarly measured in resting, postabsorptive adults at a specific temperature, but do not include a 'minimal cost of endothermy' (McNab, 2002). However, these 'maintenance' conditions are not strictly met in organisms that are growing, reproducing or continuously active. The metabolic rates of larval or juvenile animals include energy costs of growth and maintenance that are notoriously difficult to separate [see Section III (3*c*)]. Thus, all intraspecific analyses of metabolic scaling that are based on an ontogenetic series of body sizes include costs of growth. Other energy costs of development may also affect ontogenetic metabolic scaling (e.g. the development of heat production in immature birds and mammals). Even analyses of metabolic scaling based on adults only may be affected by costs of growth if the animals sampled exhibit indeterminate growth (i.e. growth after maturation). In addition, some healthy, well-fed adult animals (e.g. cladocerans) may be continuously in a reproductive state, which may affect their metabolic scaling as well [see Section III (3*c*)]. Furthermore, many animals that inhabit open-water environments must remain continuously active to prevent life-threatening sinking. Measures of 'routine metabolism' include these activities (Calow, 1985; Jobling, 1994). As will be seen, locomotor activity can significantly affect the scaling of metabolism, both within and among species. Therefore, the metabolic scaling relations examined in this review may not only include energy costs of maintenance, but also costs of growth, development, reproduction and locomotion.

Another important methodological consideration is that the metabolic scaling exponents reviewed here are based on least-squares regressions (LSR), which has been done for three reasons. First, LSR is the most common method used to calculate metabolic scaling exponents, primarily because it allows one not only to predict metabolic rate (dependent variable) based on body mass (independent variable), but also to 'subtract' the effect of body mass on metabolic rate in order to discern other influences (Harvey & Pagel, 1991; Sokal & Rohlf, 1995). Therefore, more data are available for scaling exponents based on LSR than on other regression techniques, e.g. reduced major axis (RMA) analyses. Second, although body mass is measured with error, a violation of one of the assumptions of LSR, it is typically 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. Third, most metabolic scaling relationships 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 ($b_{\mathrm{RMA}} = b_{\mathrm{LSR}}/r$; Calder, 1987). Further discussion of the relative merits of using LSR, RMA and other regression techniques in scaling studies can be found in Gould (1966), LaBarbera (1989), Harvey & Pagel (1991), Niklas (1994, 2004), Sokal & Rohlf (1995), McArdle (2003) and Kaitaniemi (2004).

An additional methodological problem in interspecific scaling studies arises when the body masses of the analyzed species are unevenly distributed with respect to taxonomic affiliation, geography, and/or environmental conditions. Most interspecific metabolic scaling studies have not corrected for the varying taxonomic (phylogenetic) relatedness of the species sampled, which can affect the scaling exponent that is observed (e.g. Symonds & Elgar, 2002). This is partly because many metabolic scaling studies were done before phylogenetic correction techniques had become available and widely appreciated, and partly because phylogenies for some taxa are not sufficiently well known. Therefore, I am currently unable to assess how important phylogenetic effects may be on most of the interspecific metabolic scaling exponents reviewed here. However, several recent studies on birds and mammals have shown little difference between metabolic scaling exponents based on traditional *versus* phylogenetically corrected analyses (e.g. Weathers & Siegel, 1995; Ricklefs, Konarzewski & Daan, 1996; Speakman, 2000; Tieleman & Williams, 2000; Frappell, Hinds & Boggs, 2001; White & Seymour, 2003; McKechnie & Wolf, 2004), as also appears to be true for many other allometric analyses (Ricklefs & Starck, 1996). Furthermore, phylogenetic correction techniques, such as that of phylogenetically independent contrasts, have been criticized for unjustifiably giving priority to effects attributed to 'phylogeny', for

which no clear causes are identified, over those of other biological and environmental factors for which mechanistic explanations can be provided (e.g. Westoby, Leishman & Lord, 1995; McNab, 2002, 2003).

The reader should also be cautioned that, since geography and ecology may affect metabolic rate and its scaling with body mass [see Section II (4*d*)], uneven representation of their effects in different body-mass intervals may also affect the scaling exponent observed. However, this has been little studied, despite recognition of its potential importance (e.g. McNab, 1988, 2002; Lovegrove, 2000).

(2) Is the '3/4-power scaling law' universal?

Two major kinds of evidence appear to support the universality, or near universality of the 3/4-power law. First, the interspecific scaling exponent of metabolic rate more often approximates 3/4 than any other value. Indeed, the mean scaling exponent [$b = 0.738 \pm 0.018$ (mean $\pm 95\%$ confidence limits, C.L.)] of 146 allometric relations for metabolic rate in various animal and unicellular taxa, compiled by Peters (1983), is not significantly different from 0.75 (for a similar result, see the compilation of Withers, 1992). Second, many other biological processes dependent on metabolic energy appear to obey the 3/4-power law or can be derived from it (including rates that scale with body mass to the 3/4-power and durations that scale with body mass to the 1/4-power: e.g. McMahon & Bonner, 1983; Peters, 1983; Calder, 1984; Schmidt-Nielsen, 1984; Niklas, 1994, 2004; Peters *et al.*, 1996; Brown & West, 2000; Brown *et al.*, 2003, 2004*b*; Dobson, Zinner & Silva, 2003; Enquist, 2003; Savage *et al.*, 2004*a*, *b*; Gillooly *et al.*, 2005). The common occurrence of 1/4- and 3/4-power scaling is striking, indeed. Thus, it is not surprising that many recent theories proposed to explain metabolic scaling have assumed that the scaling exponent is 3/4, which seems to be reasonable, at least as a first approximation (see Section III).

However, there are four problems with this evidence. First, although the mode (0.74) of the metabolic scaling exponents compiled by Peters (1983) is very nearly 3/4, the distribution of values is quite broad (<0.5 to >1.0) and includes values that have been shown to be significantly different from 0.75 (see also Kozłowski, Konarzewski & Gawelczyk, 2003*a*, *b*). Although a substantial percentage (49%) of the exponents is between 0.7 and 0.8, a slightly larger percentage lies outside this range (51%: see Fig. 4.1 in Peters, 1983). The distribution of interspecific metabolic scaling exponents is similarly broad in Withers' (1992) compilation, which has a mode (0.814) somewhat higher than 0.75.

Second, the compilation of Peters (1983) may be misleading because it is highly vertebrate-biased; 72% (117/162) of the allometric relations included in his Appendix III involve vertebrates, and 44% (72/162) involve birds and mammals, despite the fact that most animals are invertebrates. Furthermore, there is considerable redundancy in this compilation: multiple allometric relations are frequently included for the same taxon (e.g. 10 for passerine birds).

Third, the reviews of Peters (1983) and others emphasize interspecific scaling relations, and usually ignore intraspecific relations. Intraspecific metabolic scaling is highly variable, as will be seen. For example, Withers (1992) found that the intraspecific metabolic scaling exponent varied from 0.3 to 1.8 in his sample of 220 species. Although the mean scaling exponent (0.724) was similar to 0.75, the mode was 0.667.

Fourth, although 1/4- and 3/4-power scaling has been reported for many biological processes, other kinds of scaling have also been observed. For example, according to Calder (1984; Table 14-1), many biological processes in birds and mammals (e.g. respiration cycle duration, doubling time for individual and population growth rates, time to puberty, life expectancy, etc.) appear to scale with body mass (M) approximately as $M^{1/3}$. In addition, although theory based on 3/4-power scaling has predicted that animal home ranges should scale as $M^{3/4}$ and population density as $M^{-3/4}$, observed scaling exponents are often significantly steeper (e.g. Cyr, 2000; Haskell, Ritchie & Olff, 2002; Cyr & Walker, 2004; Makarieva, Gorshkov & Li, 2004*a*; but see Belgrano *et al.*, 2002; Dobson *et al.*, 2003; Jennings & Mackinson, 2003). In a study of 987 mammal species, Dobson *et al.* (2003) claim that mammalian population density appears to scale approximately as $M^{-3/4}$, but this conclusion depended on the taxa included [e.g. the scaling exponent for the whole sample was actually -0.642 ± 0.036 (mean $\pm 95\%$ C.L.), which is significantly different from $-3/4$, but not from $-2/3$].

(3) Intraspecific relationships

Some of the most telling evidence against the universality of 3/4-power scaling comes from studies of intraspecific (ontogenetic) metabolic scaling. After reviewing the literature on vertebrates and invertebrates, I offer a tentative classification of intraspecific metabolic scaling in animals, with the hope of encouraging further comparative research on the functional and evolutionary causes of variation in metabolic scaling at the individual species level. This classification may have to be modified when more data on a greater variety of taxa are collected, especially of unicells, algae, fungi and plants.

Intraspecific (ontogenetic) analyses of metabolic scaling avoid the problems of phylogenetic effects that plague interspecific analyses (Symonds & Elgar, 2002; Bokma, 2004; Van Voorhies, Khazaeli & Curtsinger, 2004) and offer greater opportunities for repeated experimental tests [see also Section IV (5)]. However, some investigators (e.g. Calder, 1987; Brown, Enquist & West, 1997) have discounted the value of intraspecific metabolic scaling relationships because they claim that the smaller body-size ranges observed within species cause estimates of scaling exponents to be more variable and unreliable. In fact, metabolic scaling exponents compiled by Withers (1992) do show a broader range of values for intraspecific *versus* interspecific analyses (see his Fig. 4–7). However, although some of this variation may be a sampling artifact, much of it also appears to be biologically meaningful. In particular, many animal species with indeterminate growth can

exhibit body-size ranges encompassing two or more orders of magnitude, and in some cases over five orders of magnitude, as in some fish. One striking example is the carp *Cyprinus carpio* L., which can grow over 100 000 times in mass from larva to adult. Interestingly, its metabolic scaling exponent is also significantly different from 3/4 (see Table 5 in Appendix).

Nevertheless, Brown *et al.* (1997) claim that in most intraspecific analyses of metabolic scaling '3/4 is within the statistical confidence intervals.' Since they did not present data to support this claim, I compiled data from the literature on 642 intraspecific metabolic scaling exponents ($\pm$95 % C.L.) for 218 animal species (Table 5 in Appendix). Unfortunately, many published studies do not provide sufficient statistical information to estimate confidence limits, but my compilation has a large enough sample size and includes a broad enough range of taxa and environments to provide some useful perspective on the variability of the metabolic scaling exponent. Five classes of vertebrates and ten phyla of invertebrates from a wide variety of aquatic and terrestrial environments are represented. This compilation reveals that 50.2 % (322) of the scaling exponents are significantly different from 3/4, and that 65.6 % (143) of the species sampled exhibited at least one scaling exponent under some specific condition that is significantly different from 3/4. Furthermore, fewer significant differences from 2/3 are seen for all regressions (294), though not for all species sampled (147). By contrast, more significant differences from 1 are observed (469 and 176, respectively). This analysis of intraspecific metabolic scaling not only contradicts the claim of Brown *et al.* (1997), but also suggests that 2/3-power scaling is slightly more common than 3/4-power scaling. This finding is consistent with the analysis of Withers (1992), which showed that the mode of intraspecific scaling exponents in animals is 2/3 rather than 3/4 [but see Sections II (3*a*, *b*)].

However, one might still argue that many of the observed differences from 3/4-power scaling are the result of chance or measurement error resulting from small sample sizes, narrow body-mass ranges and the lack of statistical corrections for multiple comparisons [which I did not make because my test of the claim of Brown *et al.* (1997) only required that I assess the frequency with which the sampled scaling exponents had individually calculated confidence intervals not including 3/4]. Consequently, 3/4-power scaling may still be the rule when only analyses involving large sample sizes and broad body-mass ranges are examined. I tested this hypothesis by examining the frequency with which metabolic scaling exponents significantly differed from 3/4 in two groups of data: the first including only regressions with sample sizes $\geqslant$50 and the second including only regressions with body-mass ranges spanning two or more orders of magnitude. Contrary to expectation, greater percentages of the scaling exponents significantly differed from 3/4 than that seen in the entire sample of regressions: 71.1 % (64/90) for the first group and 72.7 % (56/77) for the second. Therefore, it is clear that not only is 3/4-power scaling not universal at the intraspecific level, but also in studies with the most statistical power (because of large sample sizes and/or body-mass ranges) it is about 2.5 times more likely to be statistically rejected than provisionally accepted.

(*a*) *Vertebrates*

As seen for all sampled animals mentioned above, the intraspecific metabolic scaling exponents of vertebrates more often differ significantly from 3/4 than 2/3 (109 *versus* 94 out of 192 regressions, and 54 *versus* 51 out of 80 species; see Table 5 in Appendix). This finding is in agreement with studies that have shown that 2/3-power scaling is more common within species of mammals and reptiles than is 3/4-power scaling. The mean intraspecific scaling exponent is about 2/3 in the few mammals that have been examined (Brody, 1945; Thonney *et al.*, 1976; Heusner, 1982). Similarly, the scaling exponents for squamate reptiles average approximately 2/3 (Andrews & Pough, 1985; Dmi'el, 1986; Zari, 1993).

However, it would be misleading to say that the intraspecific allometry of metabolic rate typically follows a 2/3-power relationship (as claimed by Heusner, 1982) because the scaling exponent varies considerably among species and sometimes even among studies of the same species. For example, various studies of the laboratory rat *Rattus norvegicus* Berkenhout have reported scaling exponents ranging from 0.35 to 0.97 (Refinetti, 1989), and similarly wide ranges have been reported among other mammals (Brody, 1945; Thonney *et al.*, 1976; see Table 5 in Appendix). Some of this variation may be the result of sampling error, methodological differences and/or to the narrow body-mass ranges (all less than one order of magnitude) encompassed by the adults that were sampled (Table 5 in Appendix). However, I suspect that the considerable variation in b observed in mammals is unlikely to be merely the result of relatively narrow body-mass ranges because squamate reptiles exhibit a greater range of scaling exponents (0.27–1.26) than do mammals (0.38–1.11; see Table 5 in Appendix; and references in Maxwell, Jacobson & McNab, 2003), despite generally having broader adult body-mass ranges because of their indeterminate growth (75 % of the reptile regressions listed in Table 5 include body-mass ranges greater than or equal to one order of magnitude). In addition, much of the variation in scaling exponents in these and other vertebrates is statistically significant (as can be seen by comparing the 95 % confidence limits of the scaling exponents listed in Table 5). For example, varanid lizards often show relatively high scaling exponents that are closer to 1 than to 3/4 or 2/3 (Table 5 in Appendix). Although the scaling exponents vary widely (b = 0.43–1.26), the pooled means of nine species are relatively high (b = 0.89 and 0.97 at 25 and 35 °C, respectively; Thompson & Withers, 1997). Clarke & Johnston (1999) have similarly reported that, although the scaling exponents of teleost fish vary widely (b = 0.40–1.29), their mean scaling exponent [$b = 0.793 \pm 0.020$ (95 % C.L.)] is significantly greater than 3/4 and 2/3. In a more recent analysis of fish (taxa unspecified), Bokma (2004) reported a lower average scaling exponent of 0.715, but noted that the 198 estimates he analyzed were 'very unlikely to come from

populations with identical b' (ANOVA: $F_{197,1342}=7.33$, $P<0.001$), a conclusion that was upheld when only the 25 estimates based on relatively large sample sizes and body-mass ranges were examined. Other studies have shown significant differences in b among species of fish (Barlow, 1961; Du Preez, McLachlan & Marais, 1988; Clarke & Johnston, 1999), amphibians (Hillman & Withers, 1979), reptiles (Taylor & Davies, 1981; Andrews & Pough, 1985; Chappell & Ellis, 1987), and mammals (Thonney *et al.*, 1976) as well.

Significant variation in intraspecific scaling exponents may be seen even among different life stages of the same species. The scaling of metabolic rate is often steeper in larvae and juveniles (b approaching or exceeding 1) than in adults, as observed in several species of fish, birds and mammals [Table 5 in Appendix; also see Section II (3*c*)]. For example in the rainbow trout *Oncorhynchus mykiss* (Walbaum), $b=0.974\pm0.019$ (95% C.L.) in larvae and young juveniles, but $b=0.787\pm0.046$ in older juveniles and adults, a significant difference (Post & Lee, 1996). Similarly, in juveniles of the hispid cotton rat *Sigmodon hispidus* (Say & Ord) $b=1.11\pm0.09$, whereas in adults $b=0.48\pm0.05$, again a significant difference (McClure & Randolph, 1980).

(*b*) *Invertebrates*

As seen for vertebrates and all sampled animals mentioned above, the intraspecific metabolic scaling exponents of invertebrates more often differ significantly from 3/4 than 2/3 (213 *versus* 200) when all (450) regressions are considered individually, though slightly fewer differences from 3/4 than 2/3 are observed (89 *versus* 96) when the 138 sampled species are compared (see Table 5 in Appendix). Once again a wide range of scaling exponents is observed (-1.20 to 2.05), many of which are significantly different from one another (Table 5 in Appendix). Specific studies that have noted significant differences in scaling exponents among ecologically and/or taxonomically related species include Teal (1959), Prasada Rao & Ganapati (1969), Mason (1971), Sameoto (1976), Biggs (1977), Dye & McGwynne (1980), Larson (1987*b*), Jäger & Walz (2003), and Peck & Barnes (2004). Data for some invertebrates also reveal differences in scaling exponents between different life stages, as observed in some vertebrates (Table 5 in Appendix). For example, in nauplii of the copepod *Mesocyclops brasilianus* Kiefer, $b=1.08\pm0.20$ (95% C.L.), whereas in older copepodids and adults, $b=0.56\pm0.08$, a significant difference (Epp & Lewis, 1980).

As a further test of the universality of the 3/4-power scaling law, I compiled data on the intraspecific scaling exponents of 413 invertebrate species, regardless of whether confidence limits were supplied or could be calculated (based on reviews and original comparative surveys in Mann, 1956; Berg & Ockelmann, 1959; Conover, 1959; Teal, 1959; Wesemeier, 1960; Wolvekamp & Waterman, 1960; Farmanfarmaian, 1966; Altman & Dittmer, 1968, 1974; Mason, 1971; Bayne *et al.*, 1976; Sameoto, 1976; Biggs, 1977; Holopainen & Ranta, 1977*a*; Weber, 1978; Atkinson, 1980; Ivleva, 1980; Petersen, 1981; De Cuyper & Vanfleteren, 1982; Lawrence & Lane, 1982; Vidal & Whitledge, 1982; Bayne & Newell, 1983; Herman & Heip, 1983; Sutcliffe, 1984; Cetta, Madin & Kramer, 1986; Hughes, 1986; Kremer, Canino & Gilmer, 1986; Lynch, Weider & Lampert, 1986; Larson, 1987*b*; Pandian & Vernberg, 1987; Spicer & Taylor, 1987; Hamburger & Dall, 1990; Lam, Dudgeon & Ma, 1991; Shick, 1991; Patterson, 1992; Taylor & Carefoot, 1993; Arai, 1997; Riisgård, 1998; Yamada & Ikeda, 2003; Peck & Barnes, 2004; and many other sources too numerous to mention here). The median of these exponents is 0.770 and the mean is 0.772 ± 0.018 (95% C.L.), which is significantly greater than 3/4 (Table 1). Although the mean exponents of most invertebrate taxa are not significantly different from 3/4, nine of the 26 taxa examined with sample sizes >1 have mean exponents that are significantly different from 3/4 when analyzed individually, and these differences remain for three taxa (including the Insecta, the most species-rich taxon on earth) when significance levels are adjusted for multiple comparisons (Table 1). Furthermore, the mean exponents of 12 taxa are not significantly different from 2/3 when analyzed individually, and this is true for 21 taxa when corrections for multiple comparisons are made [nearly the same as the number (23) not significantly different from 3/4]. The mean exponents of seven taxa are also not significantly different from 1 when analyzed individually, and this is true for 11 taxa when corrections for multiple comparisons are made.

Three additional observations clearly show that the 3/4-power law is not universal. First, the frequency distribution of scaling exponents for all of the invertebrate species sampled displays significant kurtosis [kurtosis/(standard error of kurtosis)$=2.112/0.239$; $P<0.05$). The tails of the distribution are significantly longer than that expected from a normal distribution, thus indicating a very wide range in scaling exponents (see also Fig. 1). Second, the number of species (92) exhibiting a scaling exponent $\geqslant0.9$ (i.e. nearly proportional to body mass or greater) is nearly as great as the number (114) with an exponent similar to 0.75 (i.e. between 0.70 and 0.80). Third, although sampling error probably explains some of this variation, it also appears to reflect real biological differences worthy of further study. In particular, pelagic (open-water) species have a mean scaling exponent (0.947) significantly greater than that of non-pelagic species (0.744) ($t=8.686$; $d.f.=411$; $P<0.0000000001$; Fig. 1). Although the mean scaling exponent for non-pelagic species is not significantly different from 3/4, the mean scaling exponent for pelagic species is significantly different from 3/4, and just barely significantly different from 1 (95% C.L.$=0.901$–0.994: Table 1). This pattern is verified by the data presented in Table 5 (see Appendix): b is not significantly different from 1 in a greater proportion of pelagic *versus* benthic invertebrates [66.7% (26/39) *versus* 13.5% (37/275), a significant difference: $G_{adj}=46.583$, $P<0.001$; Sokal & Rohlf (1995); see also Section IV (7)]. Among the pelagic species included in this survey are some of the most abundant life forms on the planet, including jellyfishes, comb jellies, sea butterflies, squid, water fleas, krill, and salps.

Table 1. (a) Mean intraspecific scaling exponents (±95 % confidence limits, C.L.) for log respiration (metabolic) rate in relation to log body mass of 32 invertebrate taxa (following classification of Tudge, 2000). The exponents are based on resting or routine metabolic rates exhibited by animals showing minimal activity, though activity level was difficult to control in many studies (see text). Number of species sampled per taxon is indicated (multiple values of a scaling exponent for the same species were averaged). Significant differences ($P<0.05$) from proportionality to body-surface area (2/3), body mass (1.0), and the 3/4-power scaling law are indicated. Probability values with **bold** numbers are still valid when based on Student's *t*-distribution using Sidak's multiplicative inequality to correct for multiple comparisons (i.e. 26 taxa with sample sizes >1). (b) Mean intraspecific scaling exponents (±95 % confidence limits) for log respiration (metabolic) rate in relation to log body mass of pelagic species (including scyphozoan and hydrozoan medusae, ctenophores, pelagic copepods and cephalopods, heteropod and pteropod gastropods, cladocerans, euphausiiaceans, and salps), other chiefly non-pelagic species, and all species combined. Species with combined pelagic and benthic lifestyles (e.g. anostracan and mysid crustaceans) were included in the non-pelagic category

(a)

	Scaling exponent		Significantly different from		
Taxon	Mean ± 95 % C.L.	*N*	2/3	3/4	1.0
PORIFERA	0.927	1	—	—	—
CNIDARIA					
Anthozoa	0.732 ± 0.133	15	NS	NS	**<0.05**
Scyphozoa	0.948 ± 0.048	6	**<0.05**	**<0.05**	<0.05
Hydrozoa	0.951 ± 0.113	16	**<0.05**	**<0.05**	NS
CTENOPHORA	0.935 ± 0.172	6	<0.05	<0.05	NS
PLATYHELMINTHES	0.728 ± 0.097	8	NS	NS	**<0.05**
NEMERTEA	0.478	1	—	—	—
BRYOZOA	0.982 ± 0.188	4	<0.05	<0.05	NS
ANNELIDA	0.680 ± 0.084	26	NS	NS	**<0.05**
MOLLUSCA					
Bivalvia	0.781 ± 0.064	23	<0.05	NS	**<0.05**
Cephalopoda	0.915 ± 0.209	3	<0.05	NS	NS
Gastropoda	0.717 ± 0.036	75	<0.05	NS	**<0.05**
NEMATODA	0.677 ± 0.117	7	NS	NS	**<0.05**
ONYCHOPHORA	0.932	1	—	—	—
ARTHROPODA					
Chelicerata					
Arachnida	0.854 ± 0.076	14	**<0.05**	<0.05	**<0.05**
Crustacea					
Branchiopoda					
Anostraca	0.641 ± 0.306	3	NS	NS	<0.05
Conchostraca	0.840	1	—	—	—
Notostraca	0.803	1	—	—	—
Cladocera	0.863 ± 0.086	5	<0.05	<0.05	<0.05
Maxillopoda					
Ostracoda	0.746	1	—	—	—
Copepoda	0.845 ± 0.060	11	**<0.05**	<0.05	**<0.05**
Cirripedia	0.694 ± 0.114	5	NS	NS	**<0.05**
Malacostraca					
Eucarida					
Euphausiacea	0.859 ± 0.167	4	<0.05	NS	NS
Decapoda	0.718 ± 0.056	29	NS	NS	**<0.05**
Peracarida					
Mysida	0.686 ± 0.088	4	NS	NS	**<0.05**
Isopoda	0.723 ± 0.027	25	<0.05	NS	**<0.05**
Amphipoda	0.719 ± 0.085	24	NS	NS	**<0.05**
Myriapoda	0.756 ± 1.187	2	NS	NS	NS
Insecta	0.830 ± 0.036	54	**<0.05**	**<0.05**	**<0.05**
ECHINODERMATA	0.720 ± 0.078	26	NS	NS	**<0.05**
CHORDATA					
Urochordata					
Ascidiacea	0.775 ± 0.210	4	NS	NS	<0.05
Salpida	1.110 ± 0.233	8	<0.05	<0.05	NS

(b)

Pelagic species	0.947 ± 0.046	58	<0.001	<0.001	<0.05
Non-pelagic species	0.744 ± 0.018	355	<0.001	NS	<0.001
All species	0.772 ± 0.018	413	<0.001	<0.05	<0.001

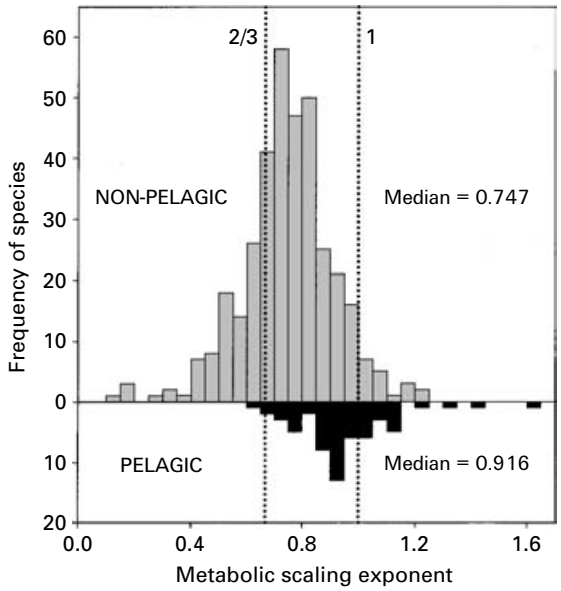

Fig. 1. Frequency of invertebrate species with different scaling exponents of log respiration (metabolic) rate in relation to log body mass. Note the higher median exponent (0.916) of pelagic species, as compared to that (0.747) of non-pelagic species. The vertical dotted lines denote scaling in proportion to body-surface area (2/3) and body mass (1). Data taken from references listed in the text.

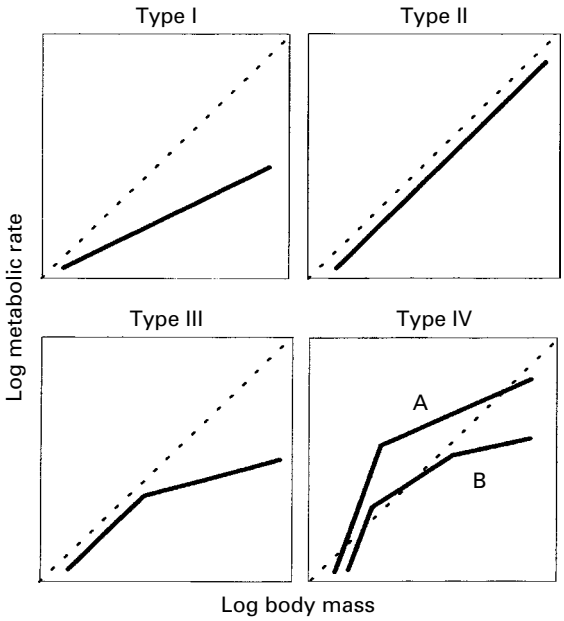

Fig. 2. Graphical representation of four major types of intraspecific (ontogenetic) metabolic scaling with body mass (on logarithmic axes). The dotted diagonal lines in each graph denote isometric relationships between log metabolic rate and log body mass (i.e. scaling exponent $b=1$). Type I: scaling is linear and negatively allometric ($b<1$). Type II: scaling is linear and isometric ($b=1$). Type III: scaling is nonlinear with an ontogenetic shift from isometric ($b=1$), or nearly isometric, to negatively allometric ($b<1$). Type IV: scaling is nonlinear with an ontogenetic shift from positively allometric ($b>1$) to one (A) or two (B) later phases of negative allometry ($b<1$).

(*c*) *A tentative classification of intraspecific metabolic scaling in animals*

Half a century ago, Bertalanffy (1951, 1957) proposed that animals show three metabolic types based on how their metabolic rate scaled to body mass [i.e. proportional to body-surface area ($M^{2/3}$), body mass (M^{1}), or intermediate (e.g. $M^{3/4}$]. He also suggested that these metabolic types were related to different patterns of growth, and concluded that his analysis heralded 'a new chapter of physiology, the comparative physiology of growth' (Bertalanffy, 1957, p. 229). Unfortunately however, although Bertalanffy's growth equations have received much attention, his notion of metabolic types has been largely forgotten. I suggest that Bertalanffy's classification should be resurrected and revised to accommodate recent data showing considerable variation in the intraspecific scaling of metabolism. A survey of the literature reveals four major types of metabolic scaling represented by log-log plots (Fig. 2):

TYPE I: Linear, negatively allometric ($b<1$). This type is common in many kinds of animals, especially those without major developmental shifts in physiology and/or morphology (e.g. Bertalanffy, 1957; Ultsch, 1973; Klekowski, Wasilewska & Paplinska, 1974; Piekarzewska, 1977; Dmi'el, 1986; Pandian & Vernberg, 1987; Cruz-Neto & Abe, 1994; Maxwell *et al.*, 2003).

TYPE II: Linear, isometric ($b=1$), or nearly isometric. This type is common in pelagic animals, including scyphozoan and hydrozoan medusae (Larson, 1987*b*; Arai, 1997), ctenophores (Kremer *et al.*, 1986), pteropod gastropods (Conover & Lalli, 1974), cephalopods (Johansen *et al.*, 1982; DeMont & O'Dor, 1984), cladocerans (Glazier, 1991), euphausiaceans (Paranjape, 1967; Small & Hebard, 1967; Kils, 1982) and salps (Cetta *et al.*, 1986). However, isometric scaling has been observed in a few non-pelagic anthozoans, bryozoans, annelids, molluscs, crustaceans, insects, echinoderms, tunicates, amphibians and reptiles as well (e.g. Mann, 1956; Bertalanffy, 1957; Berg & Ockelmann, 1959; Buikema & Armitage, 1969; Altman & Dittmer, 1974; Lawrence & Lane, 1982; Hughes & Hughes, 1986; Pandian & Vernberg, 1987; Shick, 1991; Patterson, 1992; Withers, 1992; Nielsen *et al.*, 1995; Thompson & Withers, 1997; Nakaya, Saito & Motokawa, 2003; see also Table 5 in Appendix).

TYPE III: Nonlinear, ontogenetic shift from isometric ($b=1$), or nearly isometric, to negatively allometric ($b<1$). This type is common in animals with distinct larval and adult stages, such as copepods (Epp & Lewis, 1980), insects (Muthukrishnan & Pandian, 1987) and many kinds of marine invertebrates (Zeuthen, 1953; Riisgård, 1998). It has also been observed in several fish species (Giguère, Côté & St-Pierre, 1988; Rombough, 1988; Kaufmann, 1990; Oikawa & Itazawa, 1993; Wieser, 1995; Post & Lee, 1996; Bochdansky & Leggett, 2001; Wuenschel, Werner & Hoss, 2004) and some mammals (Brody, 1945; Hill & Rahimtulla, 1965; Wilkie, 1977; McClure & Randolph, 1980; Hulbert, Mantaj & Janssens, 1991).

TYPE IV: Nonlinear, ontogenetic shift from positively allometric ($b>1$) to one or two later phases of negative allometry ($b<0$). This type has been frequently observed in endothermic birds and

mammals when juveniles, as well as adults, have been included in the analysis (e.g. Brody, 1945; Bertalanffy, 1957; Poczopko, 1979; Weathers & Siegel, 1995; Brück & Hinckel, 1996; Dietz & Drent, 1997; Székely, Pétervári & Andrews, 2001).

Other metabolic types can be envisioned, but are unlikely to be common because of physical and/or functional constraints: e.g. linear, positively allometric ($b>1$; as observed in some salps: Cetta *et al.*, 1986); no relationship between metabolic rate and body mass at all ($b=0$; as observed in the fruit fly *Drosophila melanogaster* Meigen: Van Voorhies *et al.*, 2004; but see Ellenby, 1953), or nonlinear, ontogenetic shift from negatively allometric ($b<1$) to isometric ($b=1$) or positively allometric ($b>1$). As far as I am aware, a significant shift from a negatively allometric scaling pattern in juveniles ($b=0.561$) to a nearly isometric pattern in adults ($b=0.909$) has been observed only in the orbatid mite *Steganacarus magnus* Nicolet (Webb, 1975). Similar patterns have been reported for a nematode and an oligochaete (Zeuthen, 1955), but with insufficient statistical analysis (Atkinson, 1980). Shifts from negative to positive allometry may also occur in some insect larvae as they grow (e.g. Kittel, 1941; Woodland, Hall & Calder, 1968; Table 5 in Appendix).

It is important to note that studies based on too narrow a range of ages and body sizes may result in species being incorrectly classified as Type I or II, when they are actually Type III or IV. For example, the laboratory rat actually follows Type III or IV scaling (Bertalanffy, 1957), but studies reviewed by Refinetti (1989) match Type I because they examined adults only. Studies examining all stages of the life cycle may also reveal more than two phases of metabolic scaling (e.g. Brody, 1945; Zeuthen, 1953, 1955; Wieser & Kanwisher, 1960; Hamburger *et al.*, 1983; Wieser, 1984; Rombough, 1988; Oikawa, Itazawa & Gotoh, 1991; Kamler, 1992; Dietz & Drent, 1997; Hulbert & Else, 2000; Finn *et al.*, 2002). For example, recently hatched larval fish still dependent on their yolk sac for sustenance may show a brief phase of positive allometry, followed by successive phases of isometry and negative allometry in actively feeding larvae and juveniles/adults, respectively (Oikawa *et al.*, 1991; Kamler, 1992).

Intermediate cases between the above modal types may be observed as well. For example, larvae of the blowfly *Lucilia illustris* Meigen appear to be intermediate between Types I and III; they show a shift from a steep, but negatively allometric exponent of 0.820 at small sizes (0.1 to 1 mg) to a shallower exponent of 0.692 at larger sizes (10 to 40 mg) (Hanski, 1976). Some precocial birds and mammals show a similar biphasic pattern (Brody, 1945; Weathers & Siegel, 1995). In addition, more than one metabolic type may be observed in the same species depending on environmental conditions. For example, the terrestrial isopod *Porcellio scaber* Latreille shows Type I metabolic scaling at 30 °C ($b=0.687$), but Type III at 20 °C ($b=0.917$: body mass <1–6 mg; $b=0.400$: body mass >6–100 mg; Wieser & Oberhauser, 1984).

Variations from the proposed metabolic types may occur due to special metabolic characteristics of specific life-history stages. For example, insect pupae may show decreases in metabolic rate relative to prior larval and/or later adult stages of similar body mass (Zwicky & Wigglesworth, 1956; Klekowski, Prus & Zyromska-Rudzka, 1967; Guerra & Cochran, 1970; Hanski, 1976; Muthukrishnan & Pandian, 1987). Changes in the scaling of metabolic rate may occur during aging (De Cuyper & Vanfleteren, 1982; Sutcliffe, 1984; Piers *et al.*, 1998; Hulbert & Else, 2000), moulting (Zwicky & Wigglesworth, 1956; Roberts, 1957; Wieser & Kanwisher, 1960; Zotin and Zotina, 1978; Taylor & Davies, 1981; Gromysz-Kałkowska, Oder & Szubartowska, 1984; Sutcliffe 1984), and shifts from free-living to parasitic stages, as in the intestinal nematode *Heligmosomoides polygyrus* (Dujardin) (=*Nematospiroides dubius*) (Bryant, 1974). Nonlinear metabolic scaling may involve developmental shifts in the elevation of a scaling relation, as well as of the slope, as observed in copepods (Epp & Lewis, 1980). Curvilinear scaling patterns have also been observed (e.g. Brody, 1945; Edwards, 1958; Wiegert, 1964; Hanski, 1976; Weathers & Siegel, 1995; Bochdansky & Leggett, 2001).

(*d*) *Other organisms besides animals*

Unfortunately, I know of no studies of intraspecific metabolic scaling that have been carried out in any algae or fungi, and only one published study that has been carried out in plants. Chen & Li (2003) have shown that the scaling exponents for the rate of photosynthesis in relation to total plant biomass differ significantly between two species of two-year-old tree seedlings [1.830 ± 0.611 *versus* 0.831 ± 0.466 (95 % C.L.)], one being significantly different from 3/4.

In addition, only a few studies have examined intraspecific metabolic scaling in protozoans, but these also suggest that the 3/4-power law is not universally applicable. In the two such studies that I was able to find, the scaling exponents were quite different from 3/4, though insufficient statistics were supplied to determine the significance of these differences [$b=0.601$ at 15 °C and 0.674 at 20 °C in *Stentor coeruleus* Ehrenberg (Laybourn, 1975); $b=0.96$ at 10 °C, 0.98 at 15 °C, and 1.00 at 20 °C in *Didinium nasutum* Müller (Laybourn, 1977)].

(*e*) *Intrinsic and extrinsic effects*

Various intrinsic (biological) and extrinsic (environmental) factors may affect the scaling of metabolic rate within a species. One intrinsic factor that has already been mentioned is age (or body-mass interval). In many animals, relatively small larvae and juveniles exhibit different (often higher) scaling exponents than larger adults. Gender may also be important. For example, metabolic scaling appears to be linear in female mole salamanders *Ambystoma talpoideum* (Holbrook), but nonlinear (biphasic) in males (Ryan & Hopkins, 2000). Other examples can be found in Table 5 of the Appendix (e.g. male humans have a significantly steeper scaling exponent than females).

Another major intrinsic factor that may affect intraspecific metabolic scaling is activity level. For example, Brett (1965) showed that the metabolic scaling exponent

of sockeye salmon (*Oncorhynchus nerka* Walbaum) becomes steeper at increasing levels of activity (approaching $b=1$ at maximal activity; see also Brett & Glass, 1973), a pattern also observed in other fishes (Farmer & Beamish, 1969; Rao, 1971; Webb, 1977; Brett & Groves, 1979; Wieser, 1985, 1995; Kaufmann, 1990; Goolish, 1991; Herrmann & Enders, 2000). Similarly, in many other organisms, the scaling of metabolism is steeper during activity than during rest, often being isometric or nearly so (e.g. Hughes *et al.*, 1971; Wallace, 1972; Prange & Jackson, 1976; Taigen & Pough, 1981; Pough & Andrews, 1984; Garland & Else, 1987; Refinetti, 1989; Rogers, Olson & Wilmore, 1995; Kotiaho *et al.*, 1998; Eisenmann, Pivarnik & Malina, 2001; Batterham & Jackson, 2003; Pis, 2003; but see Newell, 1973; Hillman & Withers, 1979; Garland, 1984; Walton, 1988). In addition, metabolic scaling in the colonial ascidean *Botrylloides simodensis* (Saito, Mukai & Watanabe) has been shown to depend on physiological state (Nakaya *et al.*, 2003), and that of the land planarian *Bipalium kewense* Moseley on reproductive state (Daly & Matthews, 1982).

A major extrinsic factor that may affect intraspecific metabolic scaling is ambient temperature. Many (but not all) experimental studies have shown that temperature can significantly alter the scaling exponent (selected examples are shown in Fig. 3; see also Rao & Bullock, 1954; Dehnel & Segal, 1956; Conover, 1960; Dehnel, 1960; Barlow, 1961; Armitage, 1962; Beamish, 1964; Farmanfarmaian, 1966; Åkerlund, 1969; Barnes & Barnes, 1969; Rising & Armitage, 1969; Petitpren & Knight, 1970; Bernice, 1971; Hughes, 1971; Lewis, 1971; Paine, 1971; Dame, 1972; Kennedy & Mihursky, 1972; Newell, 1973; Block & Tilbrook, 1975; Bayne *et al.*, 1976; Holopainen & Ranta, 1977*b*; Cairns, 1978; Young, 1979; Dye & McGwynne, 1980; Walsh & Somero, 1981; Armitage & Wall, 1982; Eccles, 1985; Hornbach, 1985; Hughes, 1986; Pandian & Vernberg, 1987; Rombough, 1988; Xiaojun & Ruyung, 1990; Shick, 1991; Hölker, 2003; Nespolo, Lardies & Bozinovic, 2003; Lardies, Catalán & Bozinovic, 2004). Therefore, the assumption that body size and temperature independently affect metabolic rate (e.g. Gillooly *et al.*, 2001) is often not correct. Other environmental factors may also be influential, such as salinity (Dehnel, 1960; Farmer & Beamish, 1969; Rao, 1971), oxygen availability (Atkinson, 1973), water availability (Chen & Li, 2003), light intensity (Buikema, 1972; Finn *et al.*, 2002), season (Berg & Ockelmann, 1959; Paloheimo & Dickie, 1966; Newell & Pye, 1971; Stickle, 1973; Knight & Simmons, 1975; Marsden, 1979; Dye & McGwynne, 1980; Van Senus, 1985), habitat (Navarro, Ortega & Iglesias, 1987), geographical location (Barlow, 1961; Walsh & Somero, 1981), nutritional level (Marsden, Newell & Ahsanullah, 1973; Newell, Roy & Armitage, 1976; Lane & Lawrence, 1979; Armitage & Wall, 1982), parasitic infection (Duerr, 1967), and exposure to air *versus* water, as in intertidal animals (Newell, Ahsanullah & Pye, 1972; Spicer & Taylor, 1987; Shick *et al.*, 1979; Shick, 1991). Oikawa *et al.* (1991) have also suggested that larval fish hatched from pelagic eggs exhibit higher metabolic scaling exponents than those hatched from demersal (benthic) eggs.

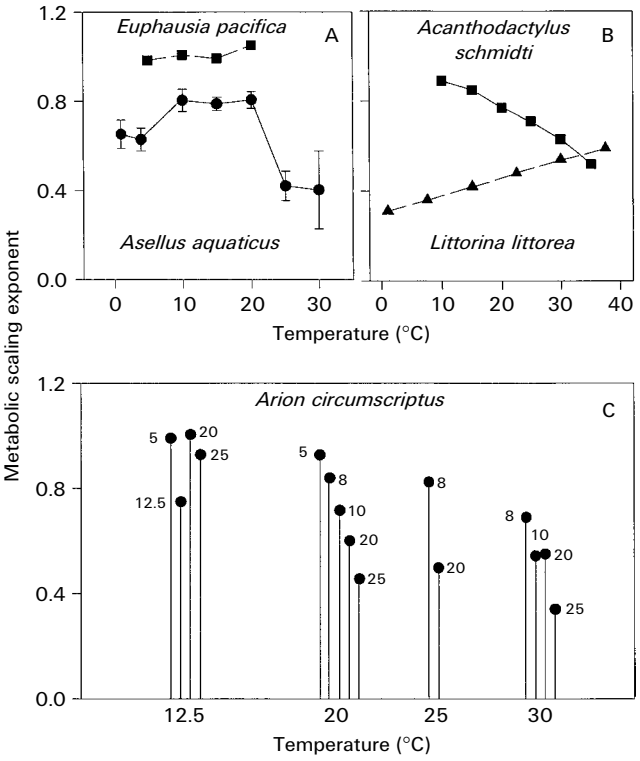

Fig. 3. Selected examples of the effect of ambient temperature on the mean scaling exponent (±95% confidence limits) of log respiration (metabolic) rate in relation to log body mass. (A) The krill *Euphausia pacifica* Hansen shows no significant effect (Paranjape, 1967), whereas the freshwater isopod *Asellus aquaticus* L. shows a humped relationship (Mladenova, 1993). (B) The lizard *Acanthodactylus schmidti* Haas shows a significantly negative relationship (Al-Sadoon & Abdo, 1991), whereas the marine gastropod *Littorina littorea* L. shows a significantly positive relationship (Newell, 1973). (C) The terrestrial gastropod *Arion circumscriptus* Johnston shows negative relationships between the metabolic scaling exponent and both experimental and acclimation temperatures (Roy, 1969). Experimental temperatures are on the x-axis, whereas acclimation temperatures are located near each point.

(4) Interspecific relationships

Metabolic scaling exponents show significant variation in interspecific analyses, as they do in intraspecific analyses. Furthermore, although 3/4-power scaling appears to be common, significant differences from 3/4 have been found in numerous animal taxa, including mammals (Hayssen & Lacy, 1985; McNab, 1988; Heusner, 1991*a*, *b*; Koteja & Weiner, 1993; Degen *et al.*, 1998; Speakman, 2000; Dodds, Rothman & Weitz, 2001; Kozłowski *et al.*, 2003*b*; Lovegrove, 2003; White & Seymour, 2003), birds (Bennett & Harvey, 1987; Ricklefs *et al.*, 1996; Tieleman & Williams, 2000; Dodds *et al.*, 2001; Frappell *et al.*, 2001; Schleucher & Withers, 2002), reptiles (Galvão, Tarasantchi & Guertzenstein, 1965; Bennett & Dawson, 1976; Andrews & Pough, 1985; Chappell & Ellis, 1987; Beuchat & Vleck, 1990; Thompson & Withers, 1997), amphibians (Feder,

1976*b*; Hillman & Withers, 1979), fishes (G.G. Winberg, 1960: cited in Peters, 1983; Brett & Groves, 1979), and invertebrates (Ivleva, 1980; Ikeda & Mitchell, 1982; Lighton *et al.*, 2001). The scaling exponent appears to depend on the taxa, body-size ranges and ecological types examined (Zeuthen, 1953; Vogel, 1976; Phillipson, 1981; Bartels, 1982; Prothero, 1984, 1986; Schmidt-Nielsen, 1984; Hayssen & Lacey, 1985; Bennett & Harvey, 1987; Elgar & Harvey, 1987; McNab, 1983, 1988, 2002; Jürgens, 1989; Heusner, 1991*a*, *b*; Lovegrove, 2000; Dodds *et al.*, 2001; Atanasov & Dimitrov, 2002; Kozłowski *et al.*, 2003*a*, *b*; White & Seymour, 2003). Both intrinsic and extrinsic factors may cause variation in interspecific metabolic scaling exponents. Here I focus on three intrinsic factors (mode of temperature regulation, body-size interval and activity level), and three extrinsic factors (geography, habitat, and temperature).

(*a*) *Effects of mode of temperature regulation*

Ectothermic animals primarily use external heat sources to regulate their body temperature, whereas endothermic animals primarily use internally generated heat. In his classic paper, Hemmingsen (1960) claimed that the metabolic scaling exponent is approximately 3/4 in both ectothermic and endothermic animals, a result later apparently confirmed by Robinson, Peters & Zimmerman (1983) and Gillooly *et al.* (2001). Robinson *et al.* (1983) found scaling exponents of 0.76 [± 0.017 (95% C.L.), $N=729$] and 0.79 (± 0.028, $N=89$) for ectotherms and endotherms, respectively. Both of these b values are near 3/4, though the latter is significantly different from 3/4. Unfortunately, however, adequate statistics were not supplied by Hemmingsen (1960) and Gillooly *et al.* (2001) to test for 3/4-power scaling (see also Dodds *et al.*, 2001). By contrast, Phillipson (1981) showed that the scaling exponent for endothermic birds and mammals [$b=0.69\pm0.0017$ (95% C.L.), $N=71$] is significantly less than 3/4, whereas the exponent for other ectothermic animals ($b=0.88\pm0.00002$, $N=470$) is significantly greater than 3/4. Many other studies based on relatively large samples of species have revealed similar differences between ectothermic and endothermic vertebrates. McNab's (2002) compilation of scaling exponents for various vertebrate taxa (in his Table 3.2) reveals that ectotherms generally have higher b values (0.71–1.06) than endotherms (0.67–0.75) (see also Withers, 1992). Numerous analyses suggest that the scaling exponent of resting metabolism in endothermic birds and mammals approximates 2/3 (Bartels, 1982; Hayssen & Lacy, 1985; Bennett & Harvey, 1987; Jürgens, 1989; Daan, Masman & Groenewold, 1990; Heusner, 1991*b*; Koteja & Weiner, 1993; Reynolds & Lee, 1996; Ricklefs *et al.*, 1996; Degen *et al.*, 1998; Lovegrove, 2000, 2003; Speakman, 2000; Tieleman & Williams, 2000; Dodds *et al.*, 2001; Frappell *et al.*, 2001; Schleucher & Withers, 2002; McNab, 2003; White & Seymour, 2003; McKechnie & Wolf, 2004), whereas the scaling of ectothermic vertebrates is usually steeper ($b\geqslant0.8$: Galvão *et al.*, 1965; Bennett & Dawson, 1976; Feder, 1976*b*; Hillman & Withers, 1979; Andrews & Pough, 1985; Chappell & Ellis, 1987; Gatten, Miller & Full, 1992; Thompson & Withers, 1997; Clarke & Johnston, 1999). Moreover, the approximately 2/3-power scaling in birds and mammals is confirmed in several recent studies that have used phylogenetic corrections (e.g. Ricklefs *et al.*, 1996; Degen *et al.*, 1998; Lovegrove, 2000, 2003; Speakman, 2000; Tieleman & Williams, 2000; Frappell *et al.*, 2001; White & Seymour, 2003, McKechnie & Wolf, 2004). Especially telling is the study of White and Seymour (2003), the most rigorous and comprehensive analysis of the scaling of basal metabolic rates in mammals yet undertaken. After controlling for differences in body temperature, digestive state and phylogeny in a sample of 619 species and subspecies, they show that b is significantly different from 3/4, but not from 2/3.

However, in a reanalysis of the mammalian data, Savage *et al.* (2004*b*) claim that the 3/4-power law is supported. They found that $b=0.737\pm0.016$ by employing a 'binning' procedure that gives equal weight to all body-size intervals. They claim that previous studies reporting 2/3-power scaling in mammals (e.g. Heusner, 1991*b*; Dodds *et al.*, 2001; White & Seymour, 2003) are biased by an overrepresentation of small-bodied species. However, the analysis of Savage *et al.* (2004*b*) has three problems. First, it ignores the fact that most mammals *are* small-bodied, and thus one could argue that a truly unbiased representation of mammalian metabolic scaling should actually include more small- than large-bodied species. Second, although Savage *et al.* (2004*b*) are aware of studies that have shown that metabolic scaling is significantly steeper in large *versus* small mammals (see next section), their regression analysis ignores this difference, as does that of White & Seymour (2003). These two problems together appear to have caused the b value reported by Savage *et al.* (2004*b*) to be biased toward that of large mammals because their binning procedure results in each species of large mammal having a larger effect on b than each small mammal. Although 50% (26/52) of their body-size classes are greater than 1000 g, they encompass only 24% (149/626) of the total number of species sampled. This problem gets worse toward the upper end of the body-mass scale: e.g. the uppermost 10 body-mass intervals (19% of the total) are represented by only 24 species (4% of the total), thus increasing the danger that idiosyncratic properties of the largest species may disproportionately affect the scaling relationship observed [see also Section III (4*b*)]. Thus, it is not surprising that the analysis of Savage *et al.* (2004*b*) yielded a b value higher than that of White & Seymour (2003) and others. Third, in their analysis Savage *et al.* (2004*b*) ignore the various corrections for body temperature, digestive state, and phylogeny made by White & Seymour (2003) because they regard them as 'questionable, *ad hoc* methods'. Although this is debatable, even without these corrections the b value (0.69 ± 0.01) found by White & Seymour (2003) is still significantly less than 3/4.

In any case, the weight of the published evidence suggests that ectothermic vertebrates usually exhibit higher interspecific scaling exponents than do endothermic vertebrates. To test this idea more carefully, I calculated b values for six orders of endotherms and three orders of ectotherms, as listed in Table 2. I chose orders for comparison to reduce the effect of phylogenetic differences within each group of

Table 2. Interspecific scaling exponents (±95 % confidence limits) for $\log_{10}$ resting respiration (metabolic) rate in relation to $\log_{10}$ body mass of selected orders of endothermic and ectothermic vertebrates. Only terrestrial orders with large sample sizes (>20) and comparable body-mass ranges were examined. Number of species sampled per order and the range of their body masses (median body mass in parentheses) are indicated. Scaling exponents (b) given in **bold** are included in Fig. 6. Significant differences ($P<0.05$) from proportionality to body-surface area (2/3), body mass (1.0), and the 3/4-power scaling rule are indicated. Probability values with **bold** numbers are still valid when based on Student's *t*-distribution using Sidak's multiplicative inequality to correct for multiple comparisons (i.e. 17 taxa)

	Scaling exponent			Significantly different from			
Taxon	b ± 95 % C.L.	Body mass range (g)	N	2/3	3/4	1.0	Sources
ENDOTHERMS							
Mammalia							
Chiroptera	**0.781** ± 0.056	3.7–1024.3 (75.6)	79	**<0.05**	NS	**<0.05**	1
Dasyuromorpha	**0.752** ± 0.031	7.1–6126.8 (100.0)	33	**<0.05**	NS	**<0.05**	1
Insectivora	**0.457** ± 0.080	2.4–1213.5 (21.9)	50	**<0.05**	**<0.05**	**<0.05**	1
Eulipotyphla[a]	0.472 ± 0.077	2.4–1213.5 (14.1)	38	**<0.05**	**<0.05**	**<0.05**	
Afrosoricida[a]	0.571 ± 0.247	6.9–650.0 (57.3)	12	NS	NS	**<0.05**	
Rodentia	0.669 ± 0.026	7.2–26 385 (69.3)	281	NS	**<0.05**	**<0.05**	1
(excluding spp. >500 g)[b]	**0.618** ± 0.041	7.2–498.0 (60.8)	249	<0.05	**<0.05**	**<0.05**	
(only spp. >500 g)[b]	0.711 ± 0.096	528.5–26 385 (1044.3)	32	NS	NS	**<0.05**	
Aves							
Columbiformes	**0.664** ± 0.083	36–2313 (219.0)	43	NS	<0.05	**<0.05**	2
Passeriformes							3
(including 2 *Pica* spp.)	0.711 ± 0.109	9–158.9 (14.1)	35	NS	NS	**<0.05**	
(excluding 2 *Pica* spp.)[c]	**0.661** ± 0.159	9–41.0 (13.9)	33	NS	NS	**<0.05**	
ECTOTHERMS							
Reptilia							
Squamata[d]							4
(all spp.)	0.702 ± 0.038	0.40–40 085 (48.5)	82	**<0.05**	<0.05	**<0.05**	
(excluding boids)	**0.807** ± 0.040	0.40–4410.0 (25.0)	68	**<0.05**	<0.05	**<0.05**	
(boids only)	1.179 ± 0.351	162–40 085 (1873)	14	<0.05	<0.05	NS	
Amphibia							
Anura[e]	**0.842** ± 0.103	0.28–562.3 (11.0)	48	**<0.05**	NS	**<0.05**	5
Urodela[f]							5
(all spp.)	0.807 ± 0.090	0.31–652.0 (3.6)	57	**<0.05**	NS	**<0.05**	
(excluding spp. >30 g)	**1.077** ± 0.139	0.31–19.7 (3.2)	50	**<0.05**	**<0.05**	NS	

[a] Recent phylogenetic analyses based on molecular data suggest that the order Insectivora is diphyletic and should be split into two independently evolved orders, as indicated (Springer *et al.*, 2004).

[b] Large rodents excluded because inspection of the overall regression suggested a break in scaling at about 500 g and the largest rodent (capybara) *Hydrochaeris hydrochaeris* (L.) had large leverage (0.060) as well.

[c] Two *Pica* (magpie) species had large leverage (0.269 and 0.279) on the regression, and thus were excluded.

[d] Regressions calculated from sources (chiefly Andrews & Pough, 1985; and Chappell & Ellis, 1987) using species averages at 30 °C (chosen because the largest sample size was at this temperature). Since the relatively large boid snakes ($N=14$) all had metabolic rates below that predicted by the overall regression line (boid metabolic rate was highly significantly different from that of other squamates of equivalent body mass, according to ANCOVA: $F_{1,79}=58.749$, $P<0.001$), three regressions lines were calculated: for all squamates, for squamates other than boids, and for boids only. Note that other non-boid snakes ($N=14$) do not significantly deviate from the regression for non-boid squamates ($F_{1,65}=1.487$, $P=0.227$).

[e] Regression calculated from source using species averages at 20 °C (chosen because the largest sample size was at this temperature).

[f] Regressions calculated from source using species averages at 15 °C (chosen because the largest sample size was at this temperature). Since six of seven metabolic rate values for animals with body mass >30 g had negative residuals in the regression for all species, a second regression was calculated with these values excluded. Sources: 1-White & Seymour (2003), Savage *et al.* (2004*b*); 2-Schleucher & Withers (2002); 3-McKechnie & Wolf (2004); 4-Andrews & Pough (1985), Chappell & Ellis (1987), Wood *et al.* (1978), Al-Sadoon (1991), Al-Sadoon & Abdo (1991), Zari (1991, 1993), Thompson & Withers (1992), DeMarco (1993), Cruz-Neto & Abe (1994), Maxwell, Jacobson & McNab (2003); 5-Gatten, Miller & Full (1992).

species, while simultaneously maintaining a relatively large sample size (>20 species). I also controlled for the effect of body size by only analyzing orders with broadly overlapping body-mass ranges. All of the scaling exponents for ectotherms are >0.8, whereas all of the exponents for endotherms are <0.8, after making some adjustments for heterogeneity of scaling effects within each of some of the orders (see bold values in Table 2). These differences are significant according to the binomial distribution ($P=0.037=[0.333]^3[0.667]^0$; Sokal & Rohlf, 1995). Moreover, when analyzed individually, three of the six orders of endotherms (Insectivora, Rodentia, and Columbiformes) have scaling exponents significantly less than 3/4, whereas two of the three orders of ectotherms (Squamata and Urodela) have scaling exponents significantly greater than 3/4. These significant differences remain even after making statistical corrections for multiple comparisons, except that for the Columbiformes and Squamata (Table 2).

Other effects of mode of temperature regulation on metabolic scaling have been observed in birds and mammals as well. In particular, McNab (1983, 1986, 1988, 2002) has shown that species with variable body temperatures (heterotherms) tend to exhibit higher b values than those with relatively constant body temperatures (endothermic homeotherms).

(*b*) *Effects of body-size interval*

Interspecific metabolic scaling may be nonlinear, as has been observed many times within species [see Section II (3)]. For example, Hayssen & Lacy (1985) reported that the scaling of basal metabolic rate in mammals was significantly better represented by a curvilinear regression (concave upward) than a linear one, a pattern also seen within some taxa of mammals (McNab, 1983, 1986, 1988). Several other studies have shown that the metabolic scaling exponent is greater in large *versus* small mammals (Zotin, Konoplev & Grudnitzky, 1978; Phillipson, 1981; Bartels, 1982; Heusner, 1991*a*; Lovegrove, 2000; Dodds *et al.*, 2001; Makarieva, Gorshkov & Li, 2003), and similar differences may also exist in birds (Zotin *et al.*, 1978). By contrast, Zeuthen (1953) reported that the scaling exponent is higher in small crustaceans (0.95: <0.1 mg body nitrogen) than in large crustaceans (0.80). My own analyses further show significant differences in metabolic scaling between large and small urodele amphibians, and between large boid snakes and other mostly smaller squamate reptiles (Table 2). The scaling exponent of large rodents (>500 g) also appears to be somewhat higher than that of smaller rodents (Table 2).

(*c*) *Effects of activity level*

Activity level can profoundly influence the scaling of metabolic rates in animals, both within and among species [see also Section II (3*e*)]. For example, the maximal rate of metabolism during activity tends to scale more steeply than basal (or resting) metabolic rate. The maximal metabolic rate of exercising birds and mammals, adjusted for heart mass and blood-haemoglobin concentration, scales as approximately 0.88 ± 0.02 (95% C.L.) power of body mass (Bishop, 1999), rather than as the 2/3 power often reported for basal metabolism [see Section II (4*b*)], or as the 3/4 power predicted by Kleiber (1961) and West *et al.* (1997, 1999*b*). Taylor *et al.* (1981), Koteja (1987), Weiner (1989), Weibel, Taylor & Hoppeler (1991), Hinds *et al.* (1993), Jones & Lindstedt (1993), Hoppeler & Flück (2002), and Weibel *et al.* (2004) have reported similarly high scaling exponents ($b=0.81$–0.92) for maximal metabolic rate in running mammals. In addition, relatively steep scaling of metabolism has often been reported for flying or swimming birds and mammals ($b=0.71$–1.05: Videler & Nolet, 1990; Voigt & Winter, 1999; Harrison & Roberts, 2000), as well as for active fish and reptiles [$b=0.87\pm0.078$: Robinson *et al.* (1983)].

Paradoxically, the scaling of metabolism in torpid or hibernating mammals with depressed body temperatures is often significantly steeper than that of basal metabolism as well, and may even approach 1 in states of deep torpor (French, 1985, 1992; Geiser, 1988, 2004; Weiner, 1989; Heldmaier & Ruf, 1992; Speakman & Thomas, 2004). For example, in 18 species of bats ranging in body mass between 4.9 and 30.0 g, the scaling exponent of torpid metabolic rate, corrected for body temperature, is 0.959 ± 0.356, which is not significantly different from 1 or 3/4 (calculated from data in Speakman & Thomas, 2004). Significant differences from 3/4 are seen when a broader range of body masses is examined. For example, in a survey of 16 mammals (body mass = 7.4–2650 g) hibernating at temperatures between 4.3 and 10.1 °C, Weiner (1989) reported a scaling exponent ($b=0.879\pm0.082$) significantly higher than 3/4. Using data on hibernating mammals (body mass = 6–80 000 g) given in Geiser (2004), I have calculated a scaling exponent of 0.830 ± 0.073 ($N=42$) for torpid metabolic rate (corrected for body temperature using multiple regression) that is also significantly greater than 3/4.

Savage *et al.* (2004*b*) report that the scaling of metabolic rates estimated in free-living mammals (so-called 'field metabolic rates') conforms to the 3/4-power law ($b=0.749\pm0.052$; see also Nagy, Girard & Brown, 1999). However, the scaling exponents of field metabolic rates are significantly different among various vertebrate taxa and often deviate significantly from 3/4 (e.g. birds: $b=0.681\pm0.036$; and reptiles: $b=0.889\pm0.059$: Nagy *et al.*, 1999). Bryant (1997) has also calculated a slope for the scaling of field metabolic rates in birds that is significantly less than 3/4 ($b=0.53\pm0.10$, $N=53$), as has Speakman (2000) for small mammals ($b=0.588$ for phylogenetically independent contrasts).

(*d*) *Environmental effects*

Many studies have examined the effects of environmental factors on the intensity of metabolism (e.g. see review in McNab, 2002), but relatively few on the scaling exponent of metabolism, which is often assumed to be a design constraint [see also Section III (2*d*)]. Nevertheless, Lovegrove (2000) found significant differences in metabolic scaling exponents for mammals from different geographical regions and habitats. For example, desert mammals show significantly higher b values than do mesic mammals. McNab

(1986, 1988) also suggested that diet may influence metabolic scaling in mammals because grouping species according to food habits increases the precision of the regression analysis. Scaling exponents for mammals grouped according to food habits vary from 0.451 to 0.873, though most of the 95% confidence limits on these values are extensively overlapping because of the relatively small sample sizes (N=4–21: McNab, 1988).

In addition, temperature may affect the interspecific scaling exponent of metabolism (e.g. Feder, 1976*b*; Ivleva, 1980; MacNally, 1981; Pandian & Vernberg, 1987), as observed in several intraspecific analyses [see Section II (3*e*)]. For example, in the crustacean study of Ivleva (1980), the most extensive survey yet done of temperature effects on the interspecific allometry of metabolism, the scaling exponent decreases significantly with increasing habitat temperature ($b=0.732\pm0.024$, 0.673 ± 0.018, and 0.630 ± 0.024 at temperatures of 20, 25 and 29 °C and N=247, 249 and 212, respectively).

(*e*) *Other organisms besides animals*

Enquist, Brown & West (1998) suggested that metabolic rate scales to the 3/4 power in plants, but this claim was based on calculations derived from xylem-transport rates, rather than on direct measurements of metabolic rate. Insufficient data are currently available to test adequately the 3/4-power law in plants (Reich, 2001). Hemmingsen (1960) claimed that unicellular organisms follow the 3/4-power law, but more recent analyses have provided mixed results. Phillipson (1981) found that the metabolic scaling exponent of 21 unicellular species was closer to 2/3 than 3/4 [$b=0.66\pm0.092$ (95% C.L.)], though not significantly different from either. However, both Hemmingsen (1960) and Phillipson (1981) lumped together prokaryotes and eukaryotes, which are very different metabolically. Prothero (1986) suggested that the 3/4-power scaling reported by Hemmingsen (1960) may be an artifact of lumping heterogeneous taxa, including bacteria, protists, fungi and animal zygotes. The scaling exponent is sensitive to which taxa are included in the analysis. For example, if bacteria, flagellates and zygotes are omitted, the exponent becomes 0.608 ± 0.051, which is significantly less than 0.75 (Prothero, 1986). Tang & Peters (1995) reported significant taxonomic differences in the allometry of unicellular metabolism as well. Phytoplankton have a metabolic scaling slope (proportional to $V^{0.88}$, where V is cell volume) significantly different from 3/4 and from that of protozoans (proportional to $V^{0.69}$). Furthermore, chlorophytes scale as $C^{1.09}$ (where C is cell-carbon content), whereas non-chlorophytes scale as $C^{0.92}$.

III. HOW IS METABOLIC SCALING TO BE EXPLAINED?

(1) Some key ideas from a historical perspective

The simplest conceivable kind of metabolic scaling would involve a one-to-one relationship between metabolic rate and body mass ($b=1$). This isometric relationship should follow if metabolic rate is simply dictated by the energy demand of maintaining body tissue, assuming that this cost is a constant per unit mass. Therefore, as body tissue increases in mass either through ontogeny or evolution, so should metabolic rate with a slope of 1. However, although the scaling of metabolic rate with body mass may be isometric, it is usually negatively allometric ($b<1$) and more rarely positively allometric ($b>1$) on a log-log scale (see Section II; Figs 1 and 2; and Tables 1, 2 and 5 in Appendix). Why is this so?

According to their frequency of occurrence, negatively allometric relationships have received the most attention in the theoretical literature on metabolic scaling, whereas positively allometric relationships have received hardly any attention at all, and have even been regarded by some as impossible, or at least highly improbable, because of physical constraints (e.g., Prigogine & Wiame, 1946; Zeuthen, 1953; Barenblatt & Monin, 1983; Kozłowski *et al.*, 2003*b*; but see Günther, 1975). Many of the explanations for the negative allometry of metabolic rate have invoked physical limits associated with the supply of metabolites to the body tissues or to the release of metabolically generated heat or waste products from the body (Table 3; and see below). By contrast, positively allometric relationships have been explained chiefly as the result of body-size related changes in the energy demand of maintaining or developing various body tissues or functions, as will be seen. As metabolic scaling theory has developed, some investigators have suggested that mechanisms controlling both energy supply and demand must be considered to gain an adequate understanding of the diverse scaling relationships that exist.

Nevertheless, the theory of metabolic scaling started with and has continued to focus on explaining why metabolic rate often shows negative allometry. Rubner (1883) was the first to provide reliable empirical data showing that metabolic rate may scale with an exponent <1. He observed that in dogs of different body size, b approximated 2/3. Based on earlier theoretical work of Sarrus & Rameaux (1839: cited in McNab, 2002), his explanation was that for endothermic mammals to maintain a constant body temperature they must produce enough body heat through metabolic processes in their body tissues to exactly balance the amount of heat lost through their body surface. Therefore, since body surface scales as $M^{2/3}$, so should metabolic rate. Since Rubner's (1883) seminal work, the metabolic rate of many species of animals has been shown to scale as $M^{\sim 2/3}$, but other scaling relations (e.g. $b<2/3$, $b\sim3/4$ or 1, and $b>1$) have been observed as well (see Fig. 1; and Tables 1 and 5 in Appendix). Furthermore, Rubner's (1883) hypothesis cannot explain why the metabolic rate of ectotherms often shows negative allometry, as does that of endotherms (but see Bejan, 2001). To cope with this problem, some biologists suggested that ectotherms may show 2/3-power scaling like endotherms because of surface-area constraints associated with nutrient uptake and gas exchange that occur in both kinds of animals (e.g. Ellenby, 1937; Bertalanffy, 1957; Edwards, 1958; Whitford & Hutchison, 1967; Hutchison, Whitford & Kohl, 1968; Anderson, 1970; Ultsch, 1973, 1974*a*, 1976). Since

Table 3. Selected types of models and hypotheses used to explain the scaling of metabolism with body mass, along with predicted scaling exponents. Models that focus on evolutionary optimization of the whole organism (in terms of reproductive fitness) are distinguished from those that focus on optimization of specific functional systems and/or on the effects of specific external/internal constraints

Type	Authors	Predicted metabolic scaling exponent(s)
WHOLE-ORGANISM OPTIMIZATION		
Maximization of reproductive fitness	Kozłowski & Weiner (1997)	Variable (includes 2/3 and 3/4)
EXTERNAL (ECOLOGICAL) CONSTRAINTS		
Resource acquisition (maximization of foraging efficiency)	Witting (1995)	3/4 (two-dimensional foragers) 5/6 (three-dimensional foragers)
EXTERNAL & INTERNAL (CHEMICAL) CONSTRAINTS		
External resource constraints & quantum mechanical constraints on flow of protons & electrons in metabolic pathways	Demetrius (2003)	2/3 (abundant, unstable resources) 3/4 (scarce, stable resources)
EXTERNAL & INTERNAL (PHYSICAL) CONSTRAINTS		
Resource uptake through body surfaces	Patterson (1992)	1/2 to 5/4 (depends on effects of body shape and ambient flow conditions)
	Makarieva *et al.* (2003)	2/3 (small mammals) 1 (large mammals)
INTERNAL (CHEMICAL) CONSTRAINTS		
Thermodynamic constraints at molecular level	Fugiwara (2003)	3/4
INTERNAL (PHYSICAL) CONSTRAINTS		
Transport of resources (including oxygen and nutrients) to tissues	West *et al.* (1997)	2/3 (two-dimensional organisms) 3/4 (three-dimensional organisms)
(minimization of transport cost)	Dodds *et al.* (2001)	5/6 or 1 (based on modifications of model of West *et al.*, 1997)
	Santillán (2003)	7/9
(maximization of areas of internal resource-exchange surfaces)	West *et al.* (1999*b*)	3/4
(minimization of transport-fluid volume)	Banavar *et al.* (1999)	3/4
(minimization of mass of transport system & even distribution of resources to all parts of body)	Dreyer & Puzio (2001)	3/4
(physical constraints on transport-fluid flow)	Rau (2002)	2/3 to 3/4
Resource uptake and use at cell surfaces	Davison (1955)	2/3 to 1 (depends on relative importance of cell size *versus* number in body-size differences)

Table 3 (*cont.*)

Type	Authors	Predicted metabolic scaling exponent(s)
Tissue demand for resources	McMahon (1973)	3/4
	Darveau *et al.* (2002)	Variable (includes 3/4; depends on physiological state: e.g. resting *versus* active)
Waste (heat) loss through body surfaces (minimization of heat-flow resistance within body)	Rubner (1883)	2/3 (based on heat loss in endotherms)
	Bejan (2001)	Variable (based on relative importance of internal *versus* body-surface heat transfer), including 1/3 (small endotherms), 2/3 (ectotherms) and 3/4 (large endotherms)
MULTIPLE INTERNAL (PHYSICAL) CONSTRAINTS		
Resource supply (exchange) and demand	Kooijman (2000) and others, including present work (e.g. 'metabolic-level boundaries' hypothesis: see text)	2/3 to 1 [depends on relative importance of surface-area limits on resource/waste exchange rates *versus* mass (volume) limits on rates of resource demand (power production)]
	Banavar *et al.* (2002*b*)	2/3 (ontogenetic scaling) 3/4 (interspecific scaling)
Resource demand and heat loss	Sibly & Calow (1986)	2/3 (in resting endotherms) 9/11 (in maximally active endotherms)
WHOLE-ORGANISM OPTIMIZATION AND CONSTRAINTS		
Evolution of resource demand and metabolic scaling in the context of cellular and demographic constraints	Kozłowski *et al.* (2003*a*, *b*)	Variable (includes 2/3, 3/4 and 1, depending on size-specific mortality and relative contributions of changes in cell size and number to differences in body size within evolutionary lineages)
Evolution of resource demand and metabolic scaling in the context of physical and ecological constraints	Present work (see text & Fig. 5)	Variable, linear or nonlinear ontogenetic scaling (includes 2/3, 3/4 and $\geqslant 1$ depending on timing and relative magnitude of energy demand of growth, reproduction, locomotion and/or heat production). Age (size)-specific mortality provides selective impetus and surface-area limits on resource/waste exchange processes and mass/volume limits on power production act as constraints

nutrients and oxygen fuel metabolism, and since they are absorbed through body surfaces, then 2/3-power scaling should also apply to ectotherms. Deviations from 2/3-power scaling were explained as the result of nutrient/gas-exchange surfaces scaling with body mass with powers different (often greater) than 2/3 (e.g. Ellenby, 1951; Bertalanffy, 1957; Hemmingsen, 1960; Szarski, 1964; Gould, 1966; Hughes & Hughes, 1986; Post & Lee, 1996; Vollmer & Edmunds, 2000), though matches between the scaling of respiratory surface areas and rates of oxygen consumption have not always been found (e.g. Feder, 1976*a*; Babula, Bielawski & Sobieski, 1978; Hillman & Withers, 1979; Hughes, 1984; Oikawa & Itazawa, 1985; Johnson & Rees, 1988; Rombough, 1988). In any case, explanations based on simple Euclidean geometry (surface area/volume ratios) fell into disfavour when it became commonly accepted that metabolic rate scales as $M^{3/4}$, not as $M^{2/3}$, among species of different body size. An approximately 3/4-power relationship was first reported in mammals by Kleiber (1932), Brody & Proctor (1932) and Benedict (1938), and claimed to be generalizable to other animals (both endotherms and ectotherms) and unicells by Hemmingsen (1960). The predominance of the 3/4-power law was later proclaimed in major reviews of allometric relationships in animals, most notably by Peters (1983), Calder (1984) and Schmidt-Nielsen (1984). As a result, during the last several decades, most theoretical work on metabolic scaling focused on 3/4-power relationships. Many attempts were made to explain this relationship, but none were widely accepted because they lacked generality or testability, made imprecise predictions, were phenomenological rather than being mechanistically based on first

principles, or were simply inconsistent with available data (see Peters, 1983; Schmidt-Nielsen, 1984, McNab, 2002; Agutter & Wheatley, 2004). As a result, the quest for an explanation of the 3/4-power law, a 70+ year mystery, has been called the 'Holy Grail in comparative biology' (C. Beuchat cited in Norris, 1998).

This quest continues, though recent models based on the branching, space-filling structure of resource-supply networks within organisms have provided especially interesting insights. The well publicized model of West *et al.* (1997) has even been hailed by some as the 'long-awaited explanation' for why the pace of various life processes scales with body mass to the 3/4 power (Hunter, 2003; *cf.* Whitfield, 2004). This model takes a new approach by focusing on the fractal geometry of internal exchange surfaces and distribution networks in organisms, rather than on the Euclidean geometry of external surfaces, as emphasized by Rubner (1883) and his followers. A system following fractal geometry is made up of self-repeating parts that at various spatial scales resemble the whole. The branching networks of the circulatory and pulmonary systems in mammals appear to be good examples of fractal geometry. Other investigators have recognized a possible connection between the fractal geometry of these systems and the 3/4-power law of metabolism (e.g. Barenblatt & Monin, 1983; Sernetz, Willems & Bittner, 1989; Spatz, 1991), but West *et al.* (1997, 1999*b*) were the first to derive a quantitative fractal-based model of the 3/4-power law from fundamental physical and biological principles. According to West *et al.* (1999*b*), metabolism scales to the 3/4 power because the areas of internal resource-exchange surfaces also scale to the 3/4 power.

Although insightful and compelling in many ways, the model of West *et al.* (1997) has been controversial. Criticisms of the model can be grouped into three major categories: (1) its apparently restricted range of applicability, (2), apparent problems with some of its assumptions, and (3) its current inability to explain the diversity of metabolic scaling relationships that have been observed.

Although West *et al.* (1997, 1999*b*) claim that their model can potentially be applied to all or nearly all of life, it flies in the face of the observation that biological distribution systems are often not fractal, at least apparently so. Cited exceptions include the open (nontubular) circulatory systems of many invertebrates (Beuchat, 1997; Alexander, 1999; Kooijman, 2000), the nonfractal vasculature of plant leaves (Kozłowski & Konarzewski, 2004) and perhaps that of fish gills and book lungs of spiders and land crabs as well (Santillán, 2003), and the absence of any anatomical circulatory system at all in some organisms (Alexander, 1999; Kooijman, 2000). Brown *et al.* (1997, 2004*a*) have responded to this criticism by suggesting that fractal-like resource distribution networks may be more widespread than currently realized, a matter deserving further empirical study. They also suggest that fractal-like networks may be 'virtual' (functional) rather than 'hard-wired' (structural) in some organisms, though this possibility is difficult to test. In any case, Banavar *et al.* (1999) have developed a simpler model that shows that general properties of three-dimensional transportation networks can explain 3/4 power scaling, regardless of whether they are fractal or not (see also Dreyer & Puzio, 2001; Banavar *et al.*, 2002*b*; Santillán, 2003; and criticism by Dodds *et al.*, 2001).

Various assumptions of the model of West *et al.* (1997) have also been criticized as being unrealistic. For example, the model assumes that the terminal units of resource transport to the tissues should have a constant size, independent of body size. However, in mammals the radius and length of the capillaries (the terminal units of the circulatory system) change with body mass, scaling as $M^{1/12}$ and $M^{5/24}$, respectively (Dawson, 2003). In addition, resting metabolic rate is assumed to be proportional to capillary number, but direct measurements indicate that the scaling of capillary number to mammal body mass depends on the tissue examined (e.g. $b = 0.62$ in kidney tissue: Dawson, 2003; and $b = 0.79$–0.93 in various skeletal muscle tissues: Hoppeler *et al.*, 1981). The scaling of capillary density in muscle tissue appears to be more related to maximal metabolic rate than to resting metabolic rate [Dodds *et al.*, 2001; see also Section III (4*c*)]. Still other criticisms of various assumptions and calculations made in the model of West *et al.* (1997) can be found in Alexander (1999), Dodds *et al.* (2001), Agutter & Wheatley (2004), and Kozłowski & Konarzewski (2004). However, according to G.B. West (personal communication), many of these criticisms are the result of misinterpretations of the model. Moreover, Brown *et al.* (1997) defend their model by stating that: 'Like any model, it is a deliberate oversimplification that can serve as a point of departure for understanding a much more complicated reality. It should be useful if it captures the essence of the mechanisms that underlie biological scaling and if, by making theoretical predictions, it helps to explain observed deviations from these values.' According to West (1999), the model is only 'a beginning rather than an end'. The question remains whether the model captures enough of the essential properties of real biological systems that it should continue to be given prominence in the theory of metabolic scaling, at least in modified form or as part of more complex models.

The final category of criticism has been directed towards the predictive power of the model of West *et al.* (1997). Although the model certainly seems to be highly successful in explaining many kinds of allometric relationships, especially in mammals and plants (Enquist, 2003; Brown *et al.*, 2004*a*, *b*; Niklas, 2004; Savage *et al.*, 2004*b*), it fails to explain adequately the great diversity of metabolic scaling exponents that have been observed both within and among species (*cf.* Riisgård, 1998; Dodds *et al.*, 2001; Hochachka *et al.*, 2003; Kozłowski *et al.*, 2003*b*; Bokma, 2004; Kozłowski & Konarzewski, 2004).

Whatever the fate of the model of West *et al.* (1997), it has helped stimulate both theoretical and empirical work on the nature of metabolic scaling. Next I classify many of the major theoretical models that have been proposed, especially during the last two decades, before examining their ability to explain the patterns of metabolic scaling reported in Section II. Older unsuccessful explanations not considered here are discussed in reviews by Peters (1983), Calder (1984), Schmidt-Nielsen (1984), Niklas (1994), McNab (2002) and Agutter & Wheatley (2004).

(2) Major types of explanatory models

Metabolic scaling explanations can be grouped according to several interrelated features, such as locus of metabolic control, scale of application, and relative emphasis on single *versus* multiple scaling relationships, single *versus* multiple causes, internal (design) *versus* external (environmental) causes, and physico-chemical constraints *versus* adaptive optimization. However, a classification using these features is not always clear-cut because the domain of some explanations is not always obvious, even to their authors, as will be seen. This problem has led to considerable confusion and sometimes fruitless, overheated debate between proponents of different explanations. Excessive contrasts between different points of view have also created needless dissension. Although I have sincerely tried to be as even-handed as possible in considering many different, often opposing views, I apologize to workers in the field of metabolic scaling and to my readers in general for any inadvertent mistakes that I might have made.

(*a*) *Locus of metabolic control*

Energy flow through an animal involves several steps, including (1) energy acquisition *via* foraging and thermal uptake, (2) energy uptake into the body interior, (3) energy transport to individual cells, (4) energy use in the cells, and (5) loss of energy (heat and other waste products) from the cells to the environment. Gas (oxygen and carbon dioxide) exchange that supports aerobic metabolism also occurs at many steps, as reviewed in detail by Weibel (2000). Metabolic rate and its relation to body size may potentially be controlled or at least influenced by energy and gas fluxes at any one or more of these steps. However, no current model of metabolic scaling has adequately considered all of these steps in a holistic way. As we have seen in the last section, some of the most publicized recent models of metabolic scaling (e.g. West *et al.*, 1997, 1999*b*, Banavar *et al.*, 1999) have focused mainly on only one or two steps (i.e. uptake of oxygen and energy/nutrients into the body interior and/or the transport of these resources to the cells). Rubner's (1883) surface law focused chiefly on loss of heat from the body, whereas modifications of this law have focused on surface-related uptake of resources into the body. Other metabolic scaling explanations have concentrated on resource acquisition *via* foraging (Witting, 1995, 1997), resource uptake into the body (Hutchison *et al.*, 1968; Patterson, 1992; Makarieva *et al.*, 2003), resource transport within the body (Dreyer & Puzio, 2001; Rau, 2002, Santillán, 2003), rate of metabolism at the level of individual cells (Weymouth *et al.*, 1944; Bertalanffy, 1957; Else & Hulbert, 1985; Porter & Brand, 1995*a*; Porter, 2001; Hulbert & Else, 1999, 2000; Else *et al.*, 2004) or different tissues (Bertalanffy, 1957; Oikawa & Itazawa, 1984, 1993; Itazawa & Oikawa, 1986; Even *et al.*, 2001; Wang *et al.*, 2001; Darveau *et al.*, 2002; Weibel *et al.*, 2004), transport of heat within the body to the outside environment (Bejan, 2001), or combinations of these steps (e.g. most notably some combination of supply and demand constraints: e.g. Hemmingsen, 1960; Jobling, 1985; Sibly & Calow, 1986; Weiner, 1989; Withers, 1992; Hulbert & Else, 2000; Kooijman, 2000; Banavar *et al.* 2002*b*; Hochachka *et al.* 2003; Weibel *et al.*, 2004; see also Table 3).

A major question concerns how many and which of the steps in energy flow and gas exchange are most importantly responsible for the scaling of metabolic rate with body mass. There are three major possibilities. First, a single step may have been set by physical constraints and/or by adaptive evolution to be the 'master limit' or 'ultimate regulator', and other steps have optimally co-adapted. Second, all steps may be equally important limits or regulators. Third, some intermediate situation may apply – e.g. some or all steps may be more or less important limits or regulators depending on physiological and/or environmental conditions. Unfortunately, however, these three possibilities are difficult to disentangle in tightly regulated physiological systems. Moreover, even if only one step is the master limit, it is difficult to determine which is that step (Spatz, 1991; Clarke & Johnston, 1999).

West *et al.* (1997) and Banavar *et al.* (2002*b*) favour the first possibility. They claim that the master limit for metabolic rate occurs at the level of resource transport within the body and that other steps have evolved to conform to it. Witting (1995, 1998) and Makarieva *et al.* (2003) similarly claim that one step acts as the primary limit for metabolic rate, but they argue that it is the upstream process of energy acquisition or assimilation, rather than the downstream process of resource transport, which occurs only if energy and materials are already available. According to these workers, metabolic scaling is more likely to be constrained by physical or ecological factors that cannot be changed by the organism because they lie outside its phenotype, than by factors such as the branching pattern of vascular systems that are evolutionarily malleable, phenotypic traits.

Weibel (2000) has discussed evidence concerning the second possibility. According to the 'symmorphosis' concept of Taylor & Weibel (1981), all steps involved in gas exchange and aerobic metabolism have co-evolved in such a way that no step is more limiting than another.

Darveau *et al.* (2002) favour the third possibility. They claim that multiple steps act as master regulators, whose importance varies with physiological state (e.g. resting *versus* active).

Following the concept of a single master limit, the model of West, Woodruff & Brown (2002*b*) predicts that the metabolic rate per cell in intact organisms should scale as $M^{-1/4}$ because of resource-supply constraints imposed by 'macrosopic transport networks.' However, the metabolic rate per cell of isolated, cultured cells freed from such constraints should scale independently of body mass, i.e. as M^0 (assuming that cell size is also independent of body size). West *et al.* (2002*b*) collected data from the literature in support of their prediction, but these data are questionable because they are based on oxygen-consumption rates determined in tumor cells taken from a variety of tissues cultured under varying conditions by different investigators. Studies of 'normal' cells from the same tissue cultured under the same conditions in the same laboratory do not support the prediction of West *et al.* (2002*b*). For example, the mass-specific scaling exponent for isolated, cultured liver cells is significantly less than 0 in both mammals [-0.18 ± 0.11

(95 % C.L.): my calculations from data in Porter & Brand (1995*b*)] and birds (-0.10 ± 0.06: Else *et al.*, 2004). Similar negative allometry of metabolic rate has also usually been observed in cultured preparations of various tissues or organs in a variety of animals, both within and among species (mass-specific $b = 0.05$ to -0.74: Kleiber, 1941; Weymouth *et al.*, 1944; Krebs, 1950; Bertalanffy & Pirozynski, 1953; Davison, 1958; Newell & Pye, 1971; Marsden *et al.*, 1973; Oikawa & Itazawa, 1984, 2003; Else & Hulbert, 1985; Couture & Hulbert, 1995; Singer, Ince & Hallmann, 2002; Singer *et al.*, 2004). These findings suggest that body-size-related differences in cell metabolic rate cannot be entirely attributed to transport-network properties (*cf.* Kleiber, 1961; Schmidt-Nielsen, 1984; Suarez, Darveau & Childress, 2004), at least in a proximate (immediate functional) sense. However, these findings do not exclude the possibility that, in an ultimate (evolutionary) sense, the intrinsic metabolic rate of cells has been adjusted, at least in part, to limits set by transport networks or some other systemic property of the organism.

(*b*) *Scale of application*

Some metabolic scaling explanations appear to focus on broad comparisons encompassing many orders of magnitude in body size (e.g. West *et al.* 1997, 1999*b*, 2002*b*; Banavar *et al.*, 1999, 2002*b*; Dreyer & Puzio, 2001), whereas others appear to be most applicable to narrower body-size ranges within specific species or higher taxa (e.g. Heusner, 1982, 1991*a*, *b*; Itazawa & Oikawa, 1986; Post & Lee, 1996; Riisgård, 1998; Kozłowski *et al.*, 2003*a*). However, the domain for some models is not always clear. For example, West *et al.* (1997) claim that the 3/4-power law, and their model proposed to explain it, best apply to broad comparisons over many orders of magnitude of body mass, whereas any deviations from the law are more likely to occur in specific species or groups of species with relatively narrow body-mass ranges (see also Brown *et al.* 1997, 2004*a*; Banavar *et al.*, 2002*b*). They cite three reasons for the deviations. First, the scaling exponent is more prone to measurement error when small *versus* large ranges in body size are considered. Second, the effect of body size is less likely to predominate over the effects of other factors acting orthogonally along the overall regression line when small ranges of body size are considered (*cf.* Calder, 1987; Tilman *et al.*, 2004). Third, adaptive 'escape' from the constraints of quarter-power scaling is more likely to occur for small than large changes in body size. However, West *et al.* (2001) do not appear to follow this broad-scale view consistently because in their own model of growth they assume that 3/4-power scaling universally applies to relatively small ontogenetic changes in body mass as well. As I have shown in Section II (3), ontogenetic metabolic scaling is highly variable and often significantly deviates from the 3/4-power law. It is thus not surprising that several investigators have criticized the 3/4-power law and the associated models of West *et al.* (1997, 2001) by using data from ontogenetic metabolic scaling (e.g. Beuchat, 1997; Riisgård, 1998; Ricklefs, 2003; Bokma, 2004; Makarieva, Gorshkov & Li, 2004*b*). Problems with indiscriminately applying the 3/4-power law to both intra- and inter-species comparisons have been noted by many others as well (e.g. Thonney *et al.*, 1976; Brown & Mount, 1982; Heusner, 1982; Wieser, 1984; Dmi'el, 1986; Kozłowski & Weiner, 1997; Witting, 1998; Kooijman, 2000; Kvist & Lindström, 2001; Wuenschel *et al.*, 2004).

(*c*) *Single versus multiple causes and scaling relationships*

Most models of metabolic scaling have attempted to explain a single relationship (most often the 3/4-power law) based chiefly on a single mechanism. Notable recent examples include the models of West *et al.* (1997, 1999*b*) and Banavar *et al.* (1999). Others have accepted the commonness of a particular relationship, i.e. $\sim$3/4-power scaling, but have regarded it as merely a statistical result of lumping together ecologically and physiologically heterogenous taxa (e.g. Heusner, 1982, 1991*b*; Prothero, 1986; Fenchel, 1987; Withers, 1992; Feldman, 1995; Dodds *et al.*, 2001; Hochachka & Somero, 2002; McNab, 2002). According to this view, although the 3/4-power relationship is a useful approximation, it is the result of multiple underlying factors, rather than being a universal law. Still others have proposed models that predict multiple, distinctly different scaling relationships, often resulting from varying relative effects of multiple causes or constraints (e.g. Günther, 1975; Sibly & Calow, 1986; Patterson, 1992; Witting, 1995; Kozłowski & Weiner, 1997; Kooijman, 2000; Bejan, 2001; Darveau *et al.*, 2002; Demetrius, 2003; Hochachka *et al.*, 2003; Kozłowski *et al.*, 2003*a*, *b*; Makarieva *et al.*, 2003; Suarez *et al.*, 2004; see also Table 3).

Although some investigators have given the impression that they are quite certain that their view is correct and that others are wrong, misguided or misinformed, the alternative views just mentioned are actually difficult to separate cleanly and test. For example, all three views can explain, at least in part, the observation that metabolic scaling commonly follows an approximately 3/4 exponent, but also exhibits a range of other exponents, often significantly different from 3/4 (as shown in Section II). Which view one adopts depends on the significance that one attaches to the variation that one sees. Is this variation relatively minor noise that is obscuring the signal of a single primary mechanism, or is it indicative of multiple mechanisms or influences of major effect?

The position that some investigators take on the above question appears to be influenced by their academic background and philosophical worldview. For example, physical scientists have tended to seek single unifying mechanisms based on physical constraints (e.g. Banavar *et al.* 1999; West, 1999; Bejan, 2001; Dreyer & Puzio, 2001; Fujiwara, 2003), whereas many (but not all) biologists appear to be more attracted to multiple-cause explanations that include the effects of various biological factors (e.g. Phillipson, 1981; Sibly & Calow, 1986; McNab, 1988; Kooijman, 2000; Darveau *et al.*, 2002; Demetrius, 2003; Hochachka *et al.*, 2003; Kozłowski *et al.*, 2003*b*; Suarez *et al.*, 2004; but see Calder, 1984; Brown *et al.*, 2004*a*, *b*). As a result, the controversy about metabolic scaling has sometimes been characterized as a debate between those seeking unifying

principles and those focusing on diversity and complexity (e.g. Whitfield, 2001, 2004; Brown *et al.*, 2004*a*). However, these need not be opposing approaches (*cf.* Brown *et al.*, 2004*a*). For example, many workers who have criticized the 3/4-power law because of the diversity of scaling relationships that have been found, are seeking general principles of metabolic scaling (e.g. Kozłowski, 2000; Hochachka *et al.*, 2003; Kozłowski *et al.*, 2003*a*, *b*; Makarieva *et al.*, 2003, 2004*a*; Suarez *et al.*, 2004), just as much as those who are proponents of the 3/4-power law. General theories need not only explain unity, but also may explain diversity as well (e.g. the theory of natural selection).

One's emphasis on unity *versus* diversity may also affect how one treats the empirical data on metabolic scaling. On one hand, some proponents of the universality of the 3/4-power law, and a single primary mechanism causing it, note the many cases where the law seems to apply, but downplay significant deviations (e.g. Brown *et al.*, 2004*b*; Savage *et al.* 2004*b*; West, Brown & Enquist, 2004). Overall 'average' tendencies are of interest, rather than 'idiosyncratic' patterns observed in specific cases (*cf.* Marquet, Labra & Maurer, 2004). However, metabolic scaling exponents that are significantly different from 3/4 have been observed at a variety of scales, from individual species to all of life. Section II in this review describes the great diversity of b values seen both within and among species, including samples spanning more than two orders of magnitude (see also Tables 2 and 5 in Appendix). Even at the broadest scales significant deviations may be seen. For example, Brown *et al.* (2004*b*) report that the overall scaling exponent [$b = 0.71 \pm 0.02$ (95 % C.L.)] of temperature-corrected metabolic rate for organisms spanning 16 orders of magnitude in body mass is actually significantly different from 3/4. It is also approximately equidistant between two major theoretical values, 2/3 and 3/4, as noted by Horn (2004).

On the other hand, some proponents of the view that multiple mechanisms or influences are involved in metabolic scaling focus heavily on the diversity of scaling exponents that have been reported for metabolic allometry (e.g. Riisgård, 1998; Darveau *et al.*, 2002; Hochachka *et al.*, 2003; Bokma, 2004; Kozłowski & Konarzewski, 2004; Suarez *et al.*, 2004), but appear to ignore the ability of the 3/4-power law and single-mechanism models such as that of West *et al.* (1997) to predict successfully numerous other allometric relationships, at least in some taxa [see Brown *et al.*, 2004*a*; Savage *et al.*, 2004*b*; and Section II (2)].

Another problem in the debate about metabolic scaling and its causes is that it has sometimes been characterized as mainly a disagreement over whether the scaling exponent is universally 2/3 *versus* 3/4 (e.g. Heusner, 1982, 1991*b*; Dodds *et al.*, 2001; Brown *et al.*, 2004*b*; Savage *et al.*, 2004*b*). This attitude appears to be a holdover from the long history of influence that both the 2/3- and 3/4-power laws have had in the field of metabolic scaling [see Section III (1)]. However, many investigators currently believe that not only may both scaling relationships apply under specific circumstances, but also other kinds of scaling relationships as well (e.g. Phillipson, 1981; Feldman & McMahon, 1983; Sibly & Calow, 1986; Withers, 1992; Feldman, 1995; Kooijman, 2000; Kozłowski *et al.*, 2003*a*, *b*).

(*d*) *Internal versus external causes*

Another distinguishing feature of various metabolic scaling models is their relative emphasis on causes that are internal *versus* external to an organism's body (Table 3). Most models have focused on internal mechanisms, especially in relation to physical or geometric aspects of organismal design. These include Rubner's (1883) surface law, McMahon's (1973) biomechanical model, and various models that focus on the physics of transport of nutrients, gases and/or heat within the body (e.g. West *et al.*, 1997, 1999*b*; Banavar *et al.*, 1999; Bejan, 2001; Dreyer & Puzio, 2001; Santillán, 2003). Other internalistic models or explanations include those that focus on the effects of cell surface area and volume (e.g. Davison, 1955, 1956; Newell & Pye, 1971; Kozłowski *et al.*, 2003*a*, *b*) or on intracellular biochemical mechanisms (e.g. Weymouth *et al.*, 1944; Bertalanffy, 1957; Else & Hulbert, 1985, 2003; Else, 1991; Porter, Hulbert & Brand, 1996; Hulbert & Else, 1999; Porter, 2001; Brand *et al.*, 2003; Demetrius, 2003; Fujiwara, 2003; Else *et al.*, 2004; Turner *et al.*, 2005). Relatively few models include ecological relationships between an organism and its environment (e.g. Patterson, 1992; Witting, 1995; Kozłowski & Weiner, 1997; Demetrius, 2003; Hochachka *et al.*, 2003; Kozłowski *et al.*, 2003*b*).

The emphasis on internal causes stems from the long-standing, widespread belief that metabolic scaling follows a single universal relationship, the 3/4-power law, in virtually all organisms and environments. However, a growing awareness of the diversity of metabolic scaling relationships, often in association with specific environmental factors (as reviewed in Section II), will hopefully increase interest in exploring external causes. As a result, we should then be able to establish a more balanced understanding of metabolic scaling based on both internal and external causes [see also Section IV (7)]

(*e*) *Physico-chemical constraints versus adaptive optimization*

Some models are based solely on physical, chemical or geometric constraints (e.g. Rubner, 1883; McMahon, 1973; Patterson, 1992; Rau, 2002; Fujiwara, 2003), whereas others are based on adaptive optimization in the context of implicitly or explicitly recognized constraints (e.g. Kozłowski & Weiner, 1997; West *et al.*, 1997, 1999*b*; Kozłowski *et al.*, 2003*b*). The optimization models differ in terms of the focal point at which optimization occurs. Most of these models focus on optimization of a specific body system or process: e.g. minimization of the energy cost (resistance) of fluid transport in a resource-distribution network (West *et al.*, 1997; Santillán, 2003); minimization of the fluid volume in a transport network (Banavar *et al.*, 1999); minimization of heat-flow resistance within the body (Bejan, 2001); maximization of the area of resource-exchange surfaces (West *et al.*, 1999*b*); or maximization of foraging efficiency (Witting, 1995, 1997). In these 'specific optimization' models, body size is viewed as a constraint on metabolic scaling relationships. By contrast, the model of Kozłowski & Weiner (1997) focuses on optimization at the level of whole-organism reproductive fitness (in particular, optimal allocation between growth and reproduction; see

also Kozłowski, 2000; Kozłowski *et al.*, 2003*b*). In this 'general optimization' model, both body size and metabolic scaling relationships are viewed as the result of adaptive optimization in the context of various patterns of size-specific mortality.

If metabolic scaling is the result of adaptive optimization and not just physico-chemical constraints, a critical question, similar to that noted in Section III (2*a*), needs to be asked: 'At which focal point(s) is optimization most important for determining the scaling that is observed?' Since evolutionary success is ultimately a function of reproductive fitness, it seems reasonable to suppose that whole-organism, life-history optimization approaches, such as that of Kozłowski & Weiner (1997), may be fundamentally more important than those based on the optimization of specific organismal processes. After all, optimization of a specific behavioral or physiological process is only important evolutionarily if it increases reproductive fitness. In addition, optimization of a specific process may not be reached if it compromises the effectiveness of other vital processes (e.g. Dudley & Gans, 1991; Garland, 1998). However, the optimization approach of Kozłowski & Weiner (1997) assumes that allometric metabolic relationships already exist within the species in their model simulations of interspecific allometries (see also Kozłowski *et al.*, 2003*b*). Thus, their model leaves open the possibility that optimization of specific functional systems may also be important in causing intraspecific (and interspecific) metabolic scaling (*cf.* Norris, 1998; Witting, 1998).

In considering the above problem, it is useful to distinguish two basic kinds of causation: proximate and ultimate (Mayr, 1997). Proximate causes refer to functional mechanisms that physically or chemically produce the biological property of interest (in this case, metabolic scaling). Ultimate causes refer to the adaptive value of a biological property, i.e. the selective vectors causing its evolutionary origin in the context of specific environments. Explanations based solely on physical or chemical mechanisms usually focus on proximate causes, though physico-chemical constraints may act as boundary conditions that restrict the directions of the evolutionary process, and thus can be important at the level of ultimate causation as well (see also next section). By contrast, optimization approaches focus primarily on ultimate causes. Several recent discussions of metabolic scaling have focused mainly on one type of cause or approach over the other, which can lead to needless dissension (*cf.* Cates & Gittleman, 1997; Daan & Tinbergen, 1997; Norris, 1998). A complete understanding of metabolic scaling will require the exploration of both physico-chemical mechanisms and selective optimization in an environmental context, and thereby the explication of both proximate and ultimate causes [*cf.* Hochachka *et al.*, 2003; see also Section IV (7)].

(*f*) *Boundary approaches*

One potentially fruitful way to explain the variety of metabolic scaling relationships that have been observed, both within and among species, is to invoke multiple constraints that act as limiting boundaries enveloping an 'option set' (*sensu* Sibly & Calow, 1986) of scaling relations that can potentially evolve depending on specific circumstances. For example, Kooijman (2000) has argued that the scaling of metabolism is due to two boundary constraints: surface-area limits on resource/waste exchange processes and mass/volume limits on power production (*cf.* Hemmingsen, 1960; Gould, 1966; Jobling, 1985; Weiner, 1989; Withers, 1992; Hulbert & Else, 2000). When surface-area processes predominate, metabolic rate should scale as $M^{\sim 2/3}$, whereas when volume-related processes predominate, metabolic rate should scale as $M^{\sim 1}$. This view is consistent with the observation that most metabolic scaling exponents are between 2/3 and 1 (Tables 1, 2 and 5 in Appendix; Fig. 1; see also Peters, 1983; Withers, 1992). Others have similarly proposed that specific physical constraints act as boundaries delimiting the metabolic scaling relationships that are possible evolutionarily (e.g. Günther, 1975; McNab, 1983; Sibly & Calow, 1986; Reich, 2001; Finkel, Irwin & Schofield, 2004). These physical constraints may involve the energy demands of continuous endothermy (scaling as $M^{\sim 1/3}$), surface-related processes of heat loss or nutrient uptake (scaling as $M^{\sim 2/3}$), network-related processes of resource transport (scaling as $M^{\sim 3/4}$), or biomechanical processes of power generation for locomotor activity (scaling as $M^{\sim 5/6\text{-}7/6}$). The boundary approach is useful in showing that, although a single constraint cannot explain all metabolic scaling relationships, it may still have an overt influence in specific cases [e.g. the Euclidean geometry of heat loss may strongly influence the scaling of resting metabolism in endotherms, but not in ectotherms: see also Sections III (4*a*, *c*)]. As Reich (2001) writes: '… natural selection operates within the boundaries placed by physico-chemical constraints, and … both operate together to lead to the strong scaling relationships observed in nature'.

Here I propose an extension of Kooijman's (2000) boundary approach – the 'metabolic-level boundaries' hypothesis – that successfully predicts numerous patterns of metabolic scaling, as will be seen. Assume that when the energy demands of maintenance are relatively high, the rate of resting metabolism is mainly constrained by surface area, which limits the uptake of oxygen and nutrients fueling metabolism, as well as the loss of heat and wastes resulting from metabolism. However, when the energy demands of maintenance are low and can be amply met by surface-dependent exchange processes, the rate of resting metabolism is more nearly a function of the mass/volume of metabolizing tissue. Given these assumptions, one can predict that metabolism should scale approximately in proportion to external (Euclidean) surface area ($M^{2/3}$) or internal (fractal) surface area ($M^{3/4}$) in animals with high-energy maintenance demands. By contrast, metabolism should scale more steeply, approaching direct proportionality with body mass (M^{1}), in animals with relatively low-energy maintenance demands. Further development and discussion of this hypothesis are provided in Sections III (3*b*, 4*a*, *c*).

(3) Intraspecific relationships

Most theoretical models of metabolic scaling have been proposed to explain interspecific relationships, rather than

those seen within species. Furthermore, of the few explanations of intraspecific scaling that have been proposed, it has been commonly assumed that the scaling exponent is 2/3 or nearly so. For example, Rubner's (1883) surface law has been used to explain 2/3-power scaling seen in mammal species at different stages of growth (Heusner, 1982; Feldman & McMahon, 1983; McMahon & Bonner, 1983). The model of Banavar *et al.* (2002*b*) also predicts that 2/3-power scaling should occur within species. When resource delivery (supply) and consumption (demand) rates are equal, 3/4 power scaling should apply, as often observed in interspecific comparisons. However, when rates of resource delivery and consumption are not in balance, as observed during ontogenetic growth, 2/3-power scaling should occur. Others have assumed that 3/4-power scaling applies both within and among species (e.g. Kleiber, 1961; West *et al.*, 2001).

Although metabolic rate often scales as $M^{\sim 2/3}$ or $M^{\sim 3/4}$ within species, many other scaling relationships are observed as well [see Section II (3); Tables 1 and 5 in Appendix; Fig. 1]. No theoretical model currently gives a realistic explanation of the full range of scaling exponents that have been observed. The hydrodynamic model of Patterson (1992) may explain some of the variation in scaling exponents observed in aquatic organisms, but it is not applicable to terrestrial organisms. Differences in metabolic scaling relationships observed during ontogeny have been related to variation in growth rates (e.g. Bertalanffy, 1957; Epp & Lewis, 1980; Muthukrishnan & Pandian, 1987; Riisgård, 1998), but these relationships cannot explain all of the variation that has been observed either, as will be seen. The models of West *et al.* (1997) and Banavar *et al.* (1999) predict that three-dimensional organisms should show 3/4-power scaling, whereas nearly two-dimensional organisms (e.g. flatworms and bryozoans) should exhibit 2/3-power scaling, but limited available data do not provide good support for this prediction. For example, the mean intraspecific scaling exponent of eight species of flatworms is 0.728, which is unexpectedly closer to 3/4 than 2/3, though it is not significantly different from 2/3, as predicted (see Table 1). The metabolic scaling exponent (0.76) of flat-bodied adults of the cestode *Taenia taeniaeformis* Batsch is also unexpectedly most similar to 3/4, but again is not significantly different from 2/3 (Brand & Alling, 1962). Even more problematic is the finding that the metabolic scaling of the bryozoan *Electra pilosa* (L.) is isometric ($b \sim 1$) (Hughes & Hughes, 1986). Similarly high scaling exponents have been found in other '2D bryozoans' (Peck & Barnes, 2004; Table 1). The metabolic scaling of the thin and flat colonies of the tunicate *Botrylloides simodensis* is much steeper ($b = 0.80$–0.95) than predicted as well (Nakaya *et al.*, 2003).

(*a*) *Cellular models*

Another approach deserving further attention is to consider that the scaling of metabolic rate during ontogeny may be a function of changes in cell size and number. Davison (1955) proposed that metabolic rate should be proportional to total cell-surface area because many important metabolic processes (e.g. maintenance of ionic gradients and transport of metabolites) occur at cell surfaces. He reasoned that cell-surface area and thus metabolic rate should scale as $M^{2/3}$ if growth is entirely due to cell enlargement, but as M^{1} if growth is entirely due to an increase in cell number. Intermediate scaling relations should apply if growth involves increases in both cell size and number. As evidence, Davison (1955, 1956) showed that the scaling of metabolic rate in crayfish and anuran larvae matched closely the scaling of total cell-surface area in muscle tissue. However, Ellenby (1953) found no effect of cell size on oxygen-consumption rate in diploid and triploid prepupae of the fruit fly *Drosophila melanogaster*.

Unfortunately, the range of application of Davison's cell-surface hypothesis is not yet known. A comparison of metabolic scaling in animals that utilize different proportions of cell proliferation *versus* cell enlargement during growth could be revealing. Although most animals use both cell proliferation and enlargement during growth, some use mainly cell enlargement (e.g. rotifers, nematodes, tardigrades, appendicularians, and some insects: Ellenby, 1953; Calow, 1978). If the cell-surface hypothesis is correct, the latter animals should exhibit metabolic scaling exponents approximating 2/3. In fact, b values (0.64–0.72) reported for four species of nematodes are not significantly different from 2/3, though a significantly lower value has been found for the parasitic *Ascaris lumbricoides* L. (0.43), and a significantly higher value for *Plectus palustris* de Man at low food levels (0.77; see Table 5 in Appendix). The mean intraspecific scaling exponent for seven species of nematodes (0.677) is also not significantly different from 2/3 (Table 1). By contrast, squid make use of cell proliferation to support continual growth throughout their lives (Jackson & O'Dor, 2001; Moltschaniwskyj, 2004), and as expected, they exhibit scaling exponents approaching 1 [see Section II (3*c*); Table 1]. In addition, early stages of mammalian growth and development appear to involve increases in cell number, whereas later stages involve increases in cell size (Goss, 1966; Winick & Noble, 1966; Cheek, 1975; Riska & Atchley, 1985), a pattern that may help explain why metabolic scaling is often steeper early *versus* late in the ontogeny of mammals (*cf.* Davison, 1955). Similar shifts from cell proliferation to cell enlargement observed during growth in fishes (Weatherley, 1990) may help explain the biphasic metabolic scaling often observed in these animals as well. However, although this hypothesis may help explain Type III scaling, it cannot explain Type IV scaling where $b > 1$ during early life stages.

A deeper look at ontogenetic changes in metabolically important intracellular processes would be worthwhile as well. However, few studies have done this. Ontogenetic scaling of cell metabolic rates in various tissues has been documented in the rat (Bertalanffy & Pirozynski, 1953; Else, 1991; Singer *et al.*, 2004), mouse (Singer *et al.*, 2002, 2004), and some fish (Oikawa & Itazawa, 1984, 1993, 2003) and invertebrates (Weymouth *et al.*, 1944; Newell & Pye, 1971). Much of the scaling of metabolic rate in rat liver cells appears to be a function of parallel changes in Na^+-pump activity (Else, 1991), but we know little else. Hulbert & Else (2000) review the metabolically significant

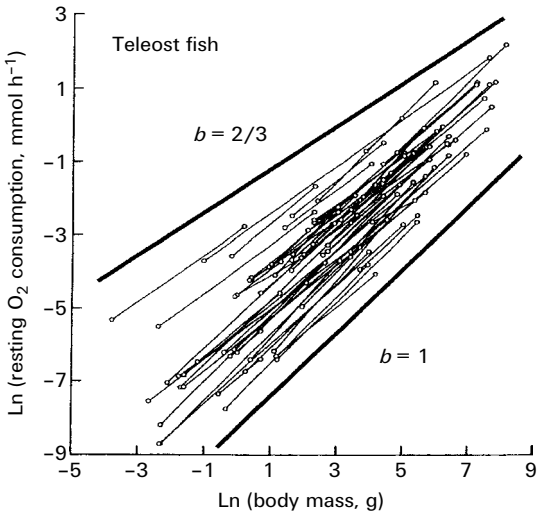

Fig. 4. Relationships between ln resting oxygen consumption (metabolic rate) and ln body mass of 69 species of teleost fish [modified from Fig. 1a in Clarke & Johnston (1999) with permission]. Each line tipped with small circles (maximal and minimal body masses) represents an individual species. The thick boundary lines represent scaling in proportion to body-surface area ($b = 2/3$) and body mass ($b = 1$).

biochemical changes that are known to occur during the ontogeny of mammals.

(*b*) *Boundary approaches*

Boundary approaches like those described in Section III (2*f*) may also be helpful, but they require further development. For example, the metabolic-level boundaries hypothesis predicts that the scaling exponent should be an inverse function of metabolic intensity. The intraspecific scaling of metabolism in teleost fish, as studied by Clarke & Johnston (1999), appears to support this hypothesis. Species with high metabolic rates tend to have scaling exponents approaching 2/3, whereas those with low metabolic rates tend to have scaling exponents approaching 1 (Fig. 4). As a result, the interspecific range in predicted metabolic rate is greater at small body sizes (about four orders of magnitude) than at larger sizes (about two orders of magnitude). Similarly, as expected, the scaling exponent of the ascidean *Botrylloides simodensis* increases to near 1 when its resting metabolic rate shifts from a high- to a low-intensity state (Nakaya *et al.*, 2003).

The metabolic-level boundaries hypothesis further predicts that decreases in metabolic rate caused by low energy availability or low environmental temperature should be accompanied by increases in the scaling exponent. As expected, many animals exhibit increased metabolic scaling exponents when their rate of metabolism is reduced by fasting or reduced food availability (significant increases: e.g. Marsden *et al.*, 1973; Newell *et al.* 1976; Lane & Lawrence, 1979; non-significant increases: e.g. Klekowski, Schiemer & Duncan, 1979; Carefoot, 1987; Glazier & Calow, 1992; but see Paloheimo & Dickie, 1966). Low temperatures are often associated with steeper metabolic scaling relations in a variety of animals as well (e.g. Rao & Bullock, 1954; Barlow, 1961; Farmanfarmaian, 1966; Åkerlund, 1969; Roy, 1969; Hughes, 1971; Lewis, 1971; Paine, 1971; Dame, 1972; Kennedy & Mihursky, 1972; Tilbrook & Block, 1972; Holopainen & Ranta, 1977*a*; Armitage & Wall, 1982; Xiaojun & Ruyung, 1990; Al-Sadoon & Abdo, 1991; Shick, 1991; Jobling, 1994). In fact, Wieser & Oberhauser (1984) anticipate the metabolic-level boundaries hypothesis by explaining the high-temperature-caused decrease of the scaling exponent (from 0.917 to 0.687) in young isopods (*Porcellio scaber*) as the result of surface-area constraints on nutrient and oxygen exchange being more important when energy demands are higher. Xiaojun & Ruyung (1990) have also suggested that surface-related processes may predominate over mass-related processes in fish, as temperature increases. However, many exceptions exist [see Section II (3*e*); Fig. 3]. In a few cases, opposite effects from that predicted by the metabolic-level boundaries hypothesis have even been observed (e.g. Conover, 1960; Dehnel, 1960; Newell, 1973; Nespolo *et al.*, 2003; Lardies *et al.*, 2004). The effect of temperature on ontogenetic metabolic scaling appears to be quite complex and requires further study. Some of the observed variation in temperature effects (negative, absent, and sometimes even positive) may be the result of small sample sizes and body-size ranges (Bayne *et al.*, 1976; Xiaojun & Ruyung, 1990; Al-Sadoon & Abdo, 1991; Shick, 1991), lack of control of extraneous environmental effects related to temperature (e.g. oxygen availability), lack of control of size-related activity levels, energy-storage levels or other non-maintenance energy-using processes (*cf.* Buikema & Armitage, 1969; Newell, 1973; Newell *et al.*, 1976; Lane & Lawrence, 1979; Al-Sadoon & Abdo, 1991), unpredictable effects of temperature at the molecular level on biological processes at higher levels of organisation (Koch, Wieser & Niederstätter, 1992; Chaui-Berlinck *et al.*, 2004), and/or species differences in age- and size-specific adaptive optimization of temperature effects (*sensu* Clarke, 2004; Clarke & Fraser, 2004).

In any case, boundary approaches may be less effectively applied to intra- than inter-specific analyses [*cf.* Section III (4)]. This is because effects on metabolism unrelated to body size will be more evident when body-size ranges are relatively small, and because comparisons of different life stages within a species involve more complex differences in various metabolic processes than do comparisons of different species at comparable life stages.

(*c*) *Effects of ontogenetic changes in various energy-using processes*

To understand fully ontogenetic metabolic scaling, one must recognize that whole-organism metabolism is a composite of several processes occurring at different rates in different tissues, each of which may scale differently to body size (*cf.* Davison, 1958; Oikawa & Itazawa, 1984, 1993, 2003; Itazawa & Oikawa, 1986; Kooijman, 2000; Wang *et al.*, 2001; Hochachka & Somero, 2002; Hochachka *et al.*, 2003; Suarez *et al.*, 2004). Here I show that ontogenetic changes in the relative magnitude of four component processes – growth, reproduction, locomotion, and heat

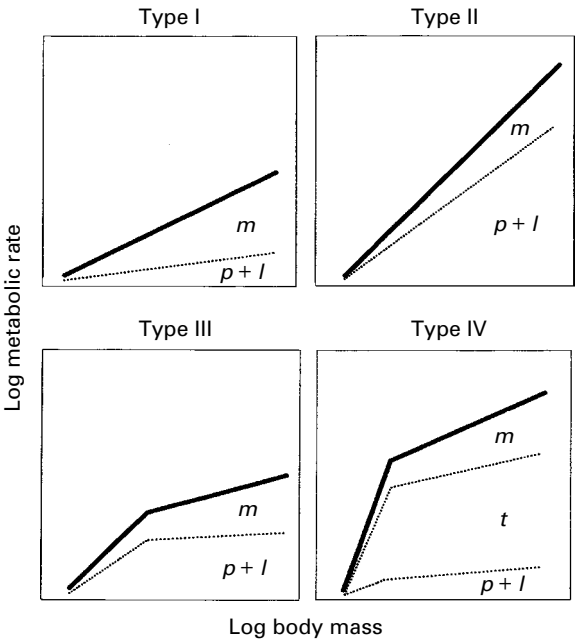

Fig. 5. Idealized graphical representation of how proportional energy expenditures for body maintenance (*m*), production (*p* = growth and reproduction), locomotion (*l* = traveling or other activities related to feeding and movement, including buoyancy maintenance), and thermogenesis (*t*) may hypothetically change with body mass during the ontogeny of animals following four different major types of metabolic scaling (see also Fig. 2).

production – may help explain the four major types of intraspecific metabolic scaling identified in Section II (3*c*) (Fig. 5). First, let us consider the effects of the energetic cost of growth.

Bertalanffy (1951, 1957) originally suggested that differences in metabolic scaling explain differences in growth trajectories observed among animal species. Conversely, it is also possible that patterns of growth may determine patterns of metabolic scaling (Parry, 1983; Riisgård, 1998). Growth has a metabolic cost that includes increases in respiration rate (Parry, 1983; Jobling, 1985; Jørgensen, 1988; Wieser, 1994; Riisgård, 1998; Peterson, Walton & Bennett, 1999), unless the costs of growth are compensated by decreases in other component processes, such as locomotor activity or specific components of maintenance metabolism (see Wieser, 1989, 1994, 1995). Moreover, experimental manipulations in animal embryos have shown that, although an increase in the rate of growth (cell division) causes an increase in respiration rate, the reverse is not observed (Tyler, 1942). In addition, during growth it is difficult to separate the energetic costs of growth and basal (maintenance) metabolism (Brody, 1945; Millward, Garlick & Reeds, 1976; Parry, 1983; Riisgård, 1998; Ricklefs, 2003; Makarieva *et al.*, 2004*b*). Therefore, ontogenetic changes in the energetic cost of growth may help explain why the scaling of metabolic rate may shift as an animal develops, as seen especially in Type III metabolic scaling.

A key example discussed by Riisgård (1998) is the mussel *Mytilus edulis* L., which shows Type III metabolic scaling ($b \sim 0.9$ in pelagic larvae and juveniles, but becomes ~ 0.7 in adults; see also Zeuthen, 1953; Hamburger *et al.*, 1983). Riisgård (1998) suggests that the early rapid growth of larvae and juveniles requires a high, nearly isometric metabolic rate, because of the high-energy cost of growth. Adults grow more slowly, and thus their metabolism slows down with increasing size. This hypothesis is consistent with the finding of Clausen & Riisgård (1996) that mass-specific respiration rate is highly correlated with mass-specific growth rate in *M. edulis*. Similar relationships have been found in many other animals as well (e.g. Jobling, 1985; Jørgensen, 1988; Blaxter, 1989; Wieser, 1994). Furthermore, since growth of new tissue is expected to be proportional to existing tissue mass, the energy cost of growth should scale as M^1 (Jobling, 1985; Wieser, 1994). Therefore, the metabolic scaling exponent should 'approach 1 when metabolic costs of growth predominate' (Jørgensen, 1988), as is observed in larval mussels.

A similar biphasic pattern is seen in copepods, which Epp & Lewis (1980) claim is the result of selection for rapid growth and maturation during larval (naupliar) stages highly vulnerable to predation. Isometric metabolic scaling has also been observed in other planktonic larvae subject to high predation, such as larvae of starfish (Zeuthen, 1953), oysters (Gerdes, 1983), sea urchins (McEdward, 1984) and fish (Wieser, 1995). Kamler (1992) has noted that the exponential pattern of growth in body mass of larval carp (*Cyprinus carpio* L.) is associated with isometric metabolic scaling, whereas the sigmoidal pattern of growth of older juveniles is associated with negatively allometric metabolic scaling, just as Bertalanffy (1957) predicted. Furthermore, larvae of the mouthbrooding fish *Oreochromis niloticus* (L.), which enjoy parental protection from predation, exhibit a shallower metabolic scaling exponent ($b = 0.42$: De Silva, Premawansa & Keembiyahetty, 1986) than other fish larvae without such protection (see Rombough, 1988; Oikawa *et al.*, 1991), as expected. In short, high juvenile mortality and growth rates may select for Type III metabolic scaling.

Ontogenetic changes in growth costs help to explain Type III metabolic scaling, but can they also explain the other types? Perhaps relatively low juvenile (and adult) mortality and growth rates select for Type I metabolic scaling, whereas relatively high mortality and growth rates during both juvenile and adult stages select for Type II metabolic scaling. Indeed, it may be worthwhile to explore to what extent metabolic types I–III are respectively associated with the classic survivorship curves Types I–III, proposed by Pearl (1928) and discussed in detail by Deevey (1947).

Interestingly, many pelagic species, which are exposed to high predation, and thus typically have short lives and undergo rapid rates of growth and maturation, exhibit isometric (Type II), or nearly isometric metabolic scaling (Fig. 1; Tables 1 and 5 in Appendix). It is probably no coincidence that pelagic salps, whose growth rates are among the highest known in animals (Heron, 1972; Fiala-Medioni, 1987; Madin & Deibel, 1998), and which continue to grow rapidly throughout their lifetimes (Hirst, Roff &

Table 4. Mean mass-specific respiration (oxygen consumption) rates (μl O_2 mg dry mass^{-1} h^{-1}), growth rates (μg dry mass μg dry mass^{-1} day^{-1}), and production (growth and brood production) rates ([μg egg dry mass + grown maternal dry mass] μg maternal dry mass^{-1} day^{-1}) of two clones (S-1, F) of *Daphnia magna* Straus at different ages and ration levels (data from Glazier & Calow, 1992). Oxygen consumption was recorded in animals starved for 24 h. High ration = 1.5 μg C ml^{-1}; low ration = 0.3 μg C ml^{-1} of the alga *Chlorella vulgaris* Beijerink

		High ration			Low ration		
Clone	Rate	Instars 1–4	5–6	7–9	Instars 1–4	5–6	7–9
S-1	Respiration	6.02	6.23	5.20	4.44	5.70	4.10
	Growth	0.336	0.085	0.041	0.247	0.028	0.024
	Production	0.379	0.492	0.371	0.263	0.151	0.141
F	Respiration	5.54	5.08	4.25	4.32	5.32	3.78
	Growth	0.351	0.141	−0.009	0.250	0.030	0.015
	Production	0.372	0.405	0.388	0.265	0.208	0.148

Lampitt, 2003), have the highest metabolic scaling exponents of the invertebrates surveyed here (Table 1). Pelagic cnidarians, ctenophores, pteropods, squids, and euphausiaceans, which also often follow Type II metabolic scaling, typically have very high rates of sustained growth during relatively short life spans as well (Lasker, 1966; Mauchline & Fisher, 1969; Reeve, Walter & Ikeda, 1978; Frandsen & Riisgård, 1997; Jackson & O'Dor, 2001; Hirst *et al.*, 2003; Arkhipkin, 2004; Jackson, 2004; Moltschaniwskyj, 2004), though we have much to learn about the growth rates of many of these animals (e.g. Nicol, 2000).

Age- and size-specific patterns of growth may even explain some unusual patterns of metabolic scaling, such as shifts from negative to positive allometry, as observed in some insect larvae. For example, in the cockroach *Blattella germanica* (L.), the rate of female growth declines during instars 1–4, but then resurges to the 1st instar level during instars 5–6. As expected by the growth hypothesis, the metabolic scaling exponent parallels these changes in growth rate ($b = 0.712$ during instars 1–4; and $b = 1.285$ during instars 4–5: Woodland *et al.*, 1968; Table 5 in Appendix).

However, other processes besides growth must also be involved in causing some of the variation in metabolic scaling that is observed, especially in Types II and IV. For example, some animals show Type II (isometric) metabolic scaling despite exhibiting a slowing down of growth as they develop into adults. This has been clearly demonstrated in a study of two parthenogenetic clones (S-1 and F) of the water flea *Daphnia magna* Straus raised at two ration levels (Glazier, 1991; Glazier & Calow, 1992; Table 4).

Respiration rate is nearly isometric in *D. magna*, not only in juveniles, but also in adults showing little or no growth, over approximately a 70-fold body-mass range (5.5–397.2 μg dry mass) (Glazier, 1991). Many studies have reported that $b < 1$ in cladocerans, but they did not properly factor out the relatively low respiration rate of eggs or embryos carried by brooding females. This is important because healthy, adult, parthenogenetic cladocerans almost always carry young. In clone S-1 of *D. magna*, raised at high ration, the respiration rate of eggs and early embryos is only approximately 1/3 of that of juveniles and adults (see also Bohrer & Lampert, 1988). If the brood-respiration rate is factored out, either by egg removal or calculation, b increases from 0.86 to 0.95. By applying this correction, essentially isometric respiration rates have also been observed in clone S-1 at low ration ($b = 1.0$) and in clone F at both high ($b = 0.92$) and low rations ($b = 0.94$) (Glazier & Calow, 1992). The scaling exponent tends to be slightly less than 1 probably, in part, because the proportion of body mass that is relatively metabolically inert cuticle increases as *Daphnia* spp. grow (Lynch *et al.*, 1986; Glazier, 1991, Glazier & Calow, 1992). Essentially isometric metabolic rates have also been observed in adult *Daphnia* spp. in other studies (Richman, 1958; Schindler, 1968; Bohrer & Lampert, 1988).

What other process or processes, then, contribute to isometric metabolic rates even in slowly growing adult *Daphnia* spp.? Likely candidates include reproduction and locomotor activity (see Fig. 5). Reproduction requires more than 50 % of the total energy budget in well-fed, adult female *D. magna* (Glazier & Calow, 1992), and stays at a high level through several successive clutches (Glazier, 1992). Furthermore, as one clutch of offspring is being released from the brood pouch, another is already being provisioned in the ovary (Zaffagnini, 1987; Bradley, Baird & Calow, 1991). Perhaps in *Daphnia* spp., respiration rate is more closely linked to the rate of total production (growth and egg production), than to growth rate alone (see also below; and Table 4).

Isometric metabolic scaling in *Daphnia* spp. and many other pelagic species (and larvae) may also be the result of continually high, energy-expensive levels of locomotor activity, including the essentially continuous swimming required to stay afloat in open water and frequent feeding to support high production costs. Anaesthesia studies have shown that the cost of swimming and/or collecting food can account for a major proportion of the metabolic rate in scyphozoans (Davenport & Trueman, 1985; Arai, 1997), cladocerans (Philippova & Postnov, 1988; Glazier & Calow, 1992), krill and pelagic amphipods and shrimp (Davenport & Trueman, 1985), pteropod and heteropod molluscs (Davenport & Trueman, 1985), and salps (Trueman, Bone

& Braconnot, 1984; Cetta *et al.*, 1986). The Antarctic krill *Euphausia superba* Dana, which is non-buoyant and thus constantly susceptible to sinking, uses a considerable proportion of its metabolism to fuel its 'hovering' activity, which increases by up to 60% as an individual grows and becomes even less buoyant (Kils, 1982). Based on energy-budget calculations, Kils (1982) suggested that the ontogenetic increase in the mass-specific energy costs of hovering may cause the metabolic scaling exponent of krill to be 1 rather than <1, as is typical of benthic crustaceans. A similar explanation has been given for isometric metabolic scaling in salps (Madin & Deibel, 1998). During growth, increasing weight (especially of the test) may require increased swimming effort, which may in turn require relatively high metabolic rates at all body sizes.

Consider that muscle power (rate of doing work) = (work per unit volume of muscle per contraction) × (contraction frequency) (Sibly & Calow, 1986). Thus, since muscle work scales as $M^{1.0}$, and contraction frequency must scale as $M^{\geqslant 0.0}$ to counteract an increased tendency to sink as an animal grows, then according to dimensional analysis, muscle metabolism during hovering should scale as $M^{1.0+\geqslant 0.0} = M^{\geqslant 1.0}$. Helicopter-based theory also predicts that 'the power required for hovering in geometrically similar animals of equal density' should scale as $M^{7/6}$ (Alexander, 1990). A similar conclusion can be derived by using dimensional analysis based upon a mechanical (dynamic) similarity criterion (Günther, 1975). In fact, the rate of metabolism during hovering of flower-visiting moths, bats and hummingbirds scales as $M^{0.88–0.95}$, the scaling exponents not being significantly different from 1, which is close to that predicted (Voigt & Winter, 1999; Harrison and Roberts, 2000). Even higher scaling exponents for hovering metabolism have been observed within species of flower-visiting bats, as predicted by aerodynamic theory (Voigt & Winter, 1999; Voigt, 2000).

However, isometric metabolic scaling is also found in pelagic animals that are neutrally buoyant or nearly so (Aleyev, 1977; Lalli & Gilmer, 1989; Alexander, 1990, 1992; Arai, 1997), thus presumably reducing locomotor costs required for maintaining position in the upper levels of the open ocean and other bodies of water. Indeed, some water-filled, gelatinous forms, such as many ctenophores and cnidarian medusae, are quite sluggish and spend considerable periods of time drifting (Harbison, Madin & Swanberg, 1978). Despite having a tendency to sink, pelagic snails (heteropods and pteropods) are not continuous swimmers either (Davenport & Trueman, 1985; Lalli & Gilmer, 1989). Some of the shell-less or almost shell-less heteropods are nearly neutrally buoyant, and thus can conserve energy by motionless floating. Shelled pteropods are heavier and must swim more often, but even they may 'spend most of their time passively floating' (Lalli & Gilmer, 1989, p. 76). Similarly, buoyant species of squid are less active than non-buoyant species (O'Dor & Webber, 1986). Furthermore, in cnidarian medusae, pulsation rate (and presumably the relative energy cost of swimming) decreases with increasing body size (Larson, 1987*a*; Arai, 1997; McHenry & Jed, 2003). And the water flea *Daphnia magna* shows no detectable increase in metabolic rate even when it is carrying eggs/embryos weighing as much as its own mass (Glazier, 1991). Thus, the explanations given by Kils (1982) and Madin & Deibel (1998) for isometric metabolic scaling in krill and salps may not be applicable to cnidarian medusae and other buoyant animals of open waters.

Nevertheless, other costs associated with maintaining buoyancy, such as continual ionic regulation and the frequent production of anti-sinking mucus strands, as observed in pelagic gastropods (Lalli & Gilmer, 1989), may be importantly involved in isometric metabolic scaling. To reduce sinking, many gelatinous zooplankton continuously expel heavy sulphate ions from their body fluids (Bidigare & Biggs, 1980; Bone, 1998), but the energy costs of such active transport are unknown. Other mechanisms that pelagic animals have used to become buoyant are reviewed by Alexander (1990) and Schmidt-Nielsen (1990).

Further evidence that steep metabolic scaling is not necessarily tied to energetically costly locomotion can be observed in some copepods with Type III scaling. Small, young nauplii, which swim very little, have unexpectedly steeper scaling relations than larger older, more active copepodid and adult stages (Epp & Lewis, 1980).

By contrast, Wuenschel, Werner & Hoss (2000, 2004) have suggested that in some marine fish the ontogenetic shift from isometric to negatively allometric metabolic scaling may be the result of a maturational change to a more energy-efficient swimming mode (*cf.* Post & Lee, 1996). In support, they note that the phase shift in metabolic scaling of the spotted seatrout *Cynoscion nebulosus* (Cuvier) occurs at the same body size at which movement becomes governed more by inertial *versus* viscous forces in an aquatic environment. As a result, the larvae switch from using continuous eel-like locomotion, involving flexing of the whole body, to using more energy-efficient 'beat and glide' locomotion, involving bending movements concentrated in the tail region (for further information on this shift in swimming mode, see Rombough, 1988; Kaufmann, 1990; Osse & van den Boogart, 2000; and references therein).

However, this explanation is not generally applicable to fish because some species (e.g. salmonids) show isometric scaling beyond this transitional stage (Wuenschel *et al.*, 2000). Other developmental changes (such as shifts in the scaling of gill-surface area and body storage) may be related to the Type III scaling of salmonids and other fish (see Oikawa & Itazawa, 1985; Hughes & Al-Kadhomiy, 1988; Kamler, 1992; Post & Lee, 1996; Post & Parkinson, 2001). Nevertheless, regardless of whether or not ontogenetic shifts in metabolic scaling coincide with shifts in swimming mode, locomotor costs still appear to be involved in the metabolic scaling of these animals. As reviewed in Section II (3*e*), several studies have shown that the metabolic scaling exponent increases with increasing swimming activity in salmonids and other fishes.

All in all, although anti-gravity locomotor activity may contribute to isometric metabolic scaling in at least some pelagic species (and larvae), other factors such as high rates of sustained production may be generally more important. High rates of production require high rates of ATP use, not only for biosynthesis, but also for high rates of feeding, which may be energetically expensive as well (see also Kils,

1982; Glazier, 1991; Wieser, 1995; Madin & Deibel, 1998). Therefore, high sustained production costs at all ages and sizes may require a mass-specific metabolic rate that does not decrease with increasing body mass, i.e. isometric metabolic scaling. A high rate of sustained production and associated isometric metabolic scaling may, in turn, have evolved as an adaptive response to high levels of mortality (predation) in exposed open-water environments. Since pelagic animals are generally short-lived, they must rapidly expend energy to grow and reproduce before they die. The importance of predation in the life of pelagic animals is indicated by the fact that many of them have evolved to be transparent (Johnsen, 2001).

Other explanations have been given for isometric metabolic scaling in specific taxa, but they are not generally applicable (e.g. Rombough, 1988; Madin & Deibel, 1998), or require further development before they can be properly evaluated (e.g. the 'mutual interaction model' of Nakaya *et al.*, 2003).

Yet another process that appears to play an important role in Type IV scaling is heat production (see Fig. 5). The steep scaling exponent ($b \sim 1$ or $b > 1$) for metabolic rate in many young birds and mammals appears to be largely the result of escalating energy costs associated with the development of increasingly effective endothermy (Brody, 1945; Bryant & Hails, 1975; McNab, 1983, 2002; Dietz & Drent, 1997; Nichelmann & Tzschentke, 2002). The importance of ontogenetic changes in thermoregulatory capacity in causing age-related shifts in the allometry of metabolic rate is supported by two rodent studies. Chappell & Bachman (1995) observed an inflection in the scaling of resting metabolic rate in the ground squirrel *Spermophilus beldingi* Merriam that occurred at nearly the same body mass as the inflection for the scaling of maximal thermogenesis. In addition, McClure & Randolph (1980) showed that the preweaning relationship between average daily metabolic rate and body mass is steeper in the precocial hispid cotton rat, *Sigmodon hispidus* Say & Ord, which has a faster rate of thermoregulatory development, than that of the altricial eastern wood rat, *Neotoma floridana* (Ord). Further support comes from their observation that the shift in slope of the metabolic scaling relationship occurs earlier in the cotton rat than in the wood rat, which matches the relative ages at which they attain homeothermy. Similar patterns are seen in comparisons of precocial and altricial birds (Ricklefs, 1974; McNab, 2002). However, although the development of thermogenesis may contribute importantly to the steep metabolic scaling of growing young birds and mammals, other costs of growth and development may be importantly involved as well (see Brody, 1945; Loudon, Rothwell & Stock, 1985; Chappell & Bachman, 1995; Székely *et al.*, 2001).

(*d*) *Effects of ontogenetic changes in body composition and tissue metabolic rates*

Lastly, intraspecific variation in metabolic scaling may also be influenced by ontogenetic changes in biochemical composition and by the differential growth of tissues and organs with varying metabolic intensity. For example, changes in water content have been related to ontogenetic shifts of metabolic scaling in some altricial birds (e.g. Weathers & Siegel, 1995; West *et al.*, 2004), but it seems doubtful that such changes can completely explain all cases of Type III and IV scaling that have been observed in many kinds of animals. Changes in the 'proportion of metabolically active mass', alluded to by Weathers (1996), may be more generally significant in causing various patterns of ontogenetic metabolic scaling (see also Kvist & Lindström, 2001). Several investigators have related decreases in mass-specific metabolic rate during ontogeny to disproportionate increases in metabolically inert materials (e.g. skeletal and/or reserve materials, such as fat and inorganic substances: Wiegert, 1964; Dahlman & Herald, 1971; Knight & Simmons, 1975; Herman & Heip, 1982; Vidal & Whitledge, 1982; Muthukrishnan & Pandian, 1987; Glazier, 1991; Elia, 1992). Consider the marine fish *Pagrus major* (Temminck & Schlegel), which follows Type III scaling. During early larval stages, the metabolic rate and growth in mass of various organs scale isometrically with increasing body size. However, during juvenile and later stages, metabolic rate shows negative allometry ($b = 0.82$), possibly in part because the growth of organs with high metabolic activity (e.g. brain, fins and gastrointestinal tract) also show negative allometry ($b < 1$), whereas the growth of organs with low metabolic activity (e.g. trunk muscles) show positive allometry ($b > 1$) (Oikawa & Itazawa, 1993, 2003). Itazawa & Oikawa (1986) reported a similarly close match between the scaling of whole-body metabolism ($b = 0.83$–0.91) of the common carp (*Cyprinus carpio*) and that of its individual tissue types summed according to their individual masses ($b = 0.87$).

(*e*) *Summary*

Several component processes may contribute to variation in intraspecific (ontogenetic) metabolic scaling in animals. However, the significance of these processes in affecting metabolic scaling requires further testing before we can have much confidence in their importance. In making such tests, attention should be given to the possibility that various energy-using processes may interactively affect metabolic scaling. The components of metabolism are often considered to be additive (e.g. Calow, 1985; Lucas, 1996; Weathers, 1996), but they may also be interactive and overlapping (*cf.* Ricklefs *et al.*, 1996). For example, maintenance metabolism may be higher during episodes of high activity or production because of 'overhead costs' (enhanced metabolic machinery) required for sustaining these activities (e.g. enlarged guts, muscles, and/or other organs involved in resource acquisition or the performance of a specific activity: Piersma & Lindström, 1997). Consideration of such interactive effects (both positive and negative) may explain why simple correlations between rates of growth and metabolism may not always be found, as discussed here in the context of Type II and III metabolic scaling.

For example, although fasting metabolic rate is not related to growth rate in *Daphnia magna* ($r = 0.295$, $P = 0.35$, $N = 12$; based on data for two clones fed at two different ration levels during three different instar classes, as given

in Table 4), it appears to be related to growth and reproduction together, though this relationship is not quite significant ($r=0.527$, $P=0.07$, $N=12$; Table 4). Negative interactions between functions, such as between growth and developmental maturation may also influence associations with maintenance metabolism. In hatchlings of turkeys and guinea fowl, the development of thermogenesis appears to conflict with growth, thus causing a negative association between the rates of growth and metabolism. However, after the development of thermogenesis is essentially completed in older chicks, the rates of growth and metabolism are positively associated (Dietz & Drent, 1997).

Ultimately a general synthetic explanation of ontogenetic metabolic scaling will require determination of not only the functional mechanisms underlying the relationships of metabolic rate with age and body mass (e.g. changes in cell metabolic rates, in cell size and number, in body composition, and in rates of various organismal processes), but also the selective factors favoring the evolution of differences in these relationships under different ecological conditions [see also Sections III (2*e*) and IV (7)]. Ecological factors may select for age-specific rates of growth, reproduction, locomotion and heat production, which in turn may affect the scaling of metabolism with body size. In addition, physico-chemical constraints limiting the rates of supply of metabolites to the tissues and of consumption of metabolites by the tissues may act as boundary conditions for the kinds of metabolic scaling that can potentially evolve.

(4) Interspecific relationships

Many of the models that have been offered to explain interspecific metabolic scaling have focused chiefly on the 3/4-power law (e.g. McMahon, 1973; West *et al.*, 1997; 1999*b*; Banavar *et al.*, 1999, 2002*b*; Dreyer & Puzio, 2001; Fujiwara, 2003; Santillán, 2003). However, interspecific metabolic scaling exponents often differ significantly from 3/4, as reviewed in Section II (4). Here I emphasize models that have been devised to explain at least some of this variation.

First, I briefly discuss a few models that may have some merit, but that do not fit the available evidence very well, that appear to have limited applicability, or that have yet to be adequately tested. Second, I evaluate the effectiveness of various models in accounting for variation in metabolic scaling exponents related to mode of temperature regulation, body-size interval and activity level. Third, I briefly discuss some models offered to explain metabolic scaling exponents observed in unicells and plants.

Witting (1995, 1997) proposed a metabolic scaling model based on constraints associated with foraging in mobile organisms. His model predicts that the metabolic scaling exponent should be 3/4 when foraging occurs in two dimensions, and 5/6 when foraging occurs in three dimensions. The evidence presented by Witting (1995) is only partly satisfactory, and further empirical research is needed to test both the assumptions and predictions of his model. Witting's categorization of animals as two-dimensional (2D) *versus* three-dimensional (3D) foragers is problematic (e.g. bats and birds are considered 2D foragers, whereas lizards and salamanders are considered 3D foragers). In addition, Ikeda (1985) has reported a metabolic scaling exponent (0.789) for 143 species of pelagic zooplankton (3D foragers) that is unexpectedly closer to 3/4 than to 5/6. However, if metabolic rate is regressed against other measures of body size besides dry mass, scaling exponents closer to 5/6 (0.833) are obtained ($b=0.835$ for carbon mass; $b=0.851$ for nitrogen mass; $b=0.817$ for phosphorus mass: Ikeda, 1985) (see also Vidal & Whitledge, 1982).

The model of Demetrius (2003) predicts that the metabolic scaling exponent should be 3/4 when resources are stable and relatively scarce, but should be 2/3 when resources are unstable and relatively high. However, these predictions are based on the assumption that metabolic rate should be higher when resources are scarce than when they are abundant, which is the opposite of that generally seen (e.g. McNab, 1980, 2002; Hoffmann & Parsons, 1991; Degen, 1997).

Several investigators have attempted to explain the negative allometry of metabolic rate as being the result of disproportionate increases in the mass of metabolically inert materials in larger animals [e.g. Benedict, 1915; Calder, 1984; Blaxter, 1989; Spaargaren, 1994; Burton, 1998; Kooijman, 2000; Wang *et al.*, 2000; McNab, 2002; see also Section III (3*d*)]. Although size-related changes in body composition may partially explain the negative allometry of metabolic rate, it is doubtful that they provide a complete explanation. For example, Ultsch (1974*b*) has calculated that size-related increases in relative skeletal mass account for only about 7 % of the decrease in mass-specific metabolic rate with increasing body size observed in mammals (but see Spaargaren, 1994).

Another factor that may contribute to variation in the metabolic scaling exponent is the relative importance of cell size *versus* cell number in accounting for species differences in body size. As Davison (1955) originally proposed, if body-size differences are mainly due to differences in cell size, metabolic rate should scale as $M^{2/3}$, but if they are mainly due to differences in cell number, metabolic rate should scale as M^{1} [see also Section III (3*a*)]. This hypothesis assumes that cell-surface area is related to metabolic rate because of its effect on the supply of metabolites to cells and/or because important metabolic activities occur at cell-surface membranes (Davison, 1955; Günther, 1975; Kozłowski *et al.*, 2003*a*, *b*). As expected, Kozłowski *et al.* (2003*a*) have found that the metabolic scaling exponent is inversely correlated with the scaling exponent for cell size (as estimated by genome size) among eight orders of birds and six orders of mammals.

The cell-surface hypothesis is worth further testing, but may only be able to explain relatively minor differences in the interspecific scaling of metabolic rate (*cf.* Bligh, 1998). This is because cell number almost always contributes much more to species differences in body size than cell size does (Rensch, 1960; Peters, 1983; Calder, 1984; Schmidt-Nielsen, 1984; Riska & Atchley, 1985; Niklas, 1994). For example, mammalian cell size scales as $M^{\sim 0.03-0.05}$ (Günther, 1975; Peters, 1983; Calder, 1984), such that, although an elephant weighs about 100 000 times more

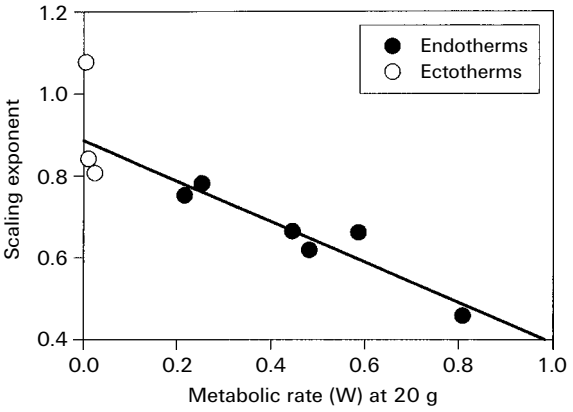

Fig. 6. Metabolic scaling exponent (b) in relation to expected metabolic rate (Watts) of a 20 g animal calculated from regressions of $\log_{10}$ metabolic rate *versus* $\log_{10}$ body mass (g) of six orders of endothermic vertebrates (●) and three orders of ectothermic vertebrates (○) (see also Table 2 and text). The line indicates the significant negative relationship between b and expected metabolic rate (R) for the endotherm orders only [$b=0.886$–0.495 (R); $r=-0.945$; $P=0.005$]. This line predicts higher values of b for the ectotherms, as shown. The correlation coefficient for all nine orders is significant as well ($r=-0.892$; $P=0.001$).

than a mouse, its cells are only about twice as large (Berrill, 1955).

(*a*) *Effects of mode of temperature regulation*

Several recent analyses suggest that the scaling of basal (resting) metabolism in endothermic birds and mammals is more shallow (b often $\sim 2/3$) than that of ectothermic vertebrates [b often $\geqslant 0.8$; see Section II (4*a*); Table 2]. These differences are unexpected for various models that assume that the 3/4-power law is universal. However, they are predicted by the metabolic-level boundaries hypothesis proposed in Section III (2*f*). The maintenance metabolism of high-energy endothermic animals is expected to be more limited by surface-dependent processes, and thus scale as $M^{2/3}$ or $M^{3/4}$. However, the maintenance metabolism of low-energy ectothermic animals is expected to be more limited by volume-dependent processes, and thus show a steeper scaling relation approaching a slope of 1.

In fact, the scaling exponent is highly significantly inversely correlated with metabolic rate, adjusted to the body mass of a 20 g animal, among nine orders of vertebrates (Fig. 6). This result is not simply a statistical artifact of the intercept and slope of log–log regressions tending to be negatively correlated [as shown for birds and mammals by Peters (1983) and McNab (1988)], because similar patterns are observed if metabolic level is calculated at other body sizes. In fact, I would suggest that the statistical patterns reported by Peters (1983) and McNab (1988) may not be entirely artifactual, but may actually be biologically significant, at least in part, as predicted by the metabolic-level boundaries hypothesis.

Several other observations are predicted by the metabolic-level boundaries hypothesis as well [see also Sections III (3*b*) and III (4*c*)]. First, the scaling exponent for field metabolic rates of free-living vertebrates is inversely correlated with metabolic level, as expected ($b=0.681$, 0.734, and 0.889, respectively in birds, mammals and reptiles: see Nagy *et al.*, 1999). Second, the scaling exponent of maintenance metabolism in torpid or hibernating mammals (which have very low energy demands) should approach 1, which it does [see Section II (4*c*)]. Third, birds and mammals that are essentially ectothermic (or poikilothermic), either as adults or during immature stages, often show isometric or nearly isometric metabolic scaling. For example, African mole rats (Bathygeridae), which have relatively low, poorly regulated body temperatures, exhibit nearly isometric metabolic rates (Lovegrove & Wissel, 1988). Ectothermic mammalian foetuses and newborns also often show isometric or nearly isometric metabolic scaling within and among species (Brody, 1945, Adolph, 1983; Hulbert *et al.*, 1991; Meschia, 1994). Bird hatchlings, which have relatively weak thermoregulatory capacities compared to adults, have significantly higher interspecific scaling exponents ($b\sim 0.85$–0.90) than do adults ($b\sim 0.67$) as well (Klaassen & Drent, 1991; Dol'nik, 1995; Weathers, 1996). Fourth, heterothermic mammals with relatively low metabolic rates show steeper scaling exponents than continuous endotherms with relatively high metabolic rates [Section II (4*a*)]. Fifth, small mammals in deserts tend to have lower metabolic rates than those from more mesic habitats (Degen, 1997; Lovegrove, 2003), and as expected they also exhibit steeper metabolic scaling relationships (Lovegrove, 2000). Sixth, the scaling exponent is expected to become steeper as ambient temperature (and thus energy demand) declines, which has been observed in crustaceans (Ivleva, 1980) and anurans (MacNally, 1981) [see also Section II (4*d*)], though not in salamanders (Feder, 1976*b*) or squamate reptiles (McNab, 2002). Perhaps the ability of relatively slender salamanders to 'breathe' through their skin has nullified surface-area constraints on oxygen uptake in these low-energy animals, even at high ambient temperatures. However, this explanation does not apply to squamates, which depend entirely on pulmonary respiration. Temperature effects on interspecific metabolic scaling appear to be complex, as also observed within species [Section III (3*b*)].

Although the metabolic-level boundaries hypothesis yields many interesting, often confirmed predictions, it is obviously oversimplified. For example, it ignores the possibility that an animal could evolve increases in surface area to accommodate higher energy demands. Indeed, to support their high metabolic rates, endothermic vertebrates have evolved significantly greater respiratory and intestinal surface areas than ectothermic vertebrates of equivalent size (Karasov & Diamond, 1985; Bennett, 1991). Nevertheless, there are limits on how much surface area can be increased, especially externally in endothermic animals for which conservation of body heat is important. As a result, heat loss through a limiting body surface may still be important in the scaling of metabolism in birds and mammals, as originally proposed by Rubner (1883) and supported by recent statistical analyses.

In future tests of the metabolic-level boundaries hypothesis, it may be worthwhile to distinguish the effects of external and internal surface areas on metabolic scaling. External surface areas usually follow Euclidean geometry ($M^{2/3}$), whereas internal surface areas often follow fractal geometry ($M^{3/4}$) (see West *et al.*, 1999*b*). Therefore, endothermic animals that maintain a constant body temperature (i.e. homeotherms) may often follow 2/3-power metabolic scaling because of external surface-related rates of heat loss that must be exactly compensated by rates of heat production. By contrast, ectothermic and endothermic animals that do not maintain a constant body temperature (i.e. poikilotherms) may often follow 3/4-power or isometric metabolic scaling, or something between, depending on the relative importance of internal surface-related processes *versus* volume-related processes in affecting maintenance costs (*cf.* Kooijman, 2000).

Other hypotheses may be offered to explain differences in metabolic scaling between endothermic and ectothermic vertebrates. For example, many ectothermic vertebrates show indeterminate growth (i.e. they continue to grow after maturation), whereas most endothermic vertebrates are essentially determinate growers (i.e. they grow little or not at all after maturation). These different growth patterns may cause differences in metabolic scaling because the energetic cost of growth increases metabolic rate [see Section III (3*c*)]. Perhaps, metabolic scaling is relatively steep in many continually growing ectotherms because the energy cost of growth increases the metabolic rate of large species above that which would be expected if they were not continuous growers (*cf.* Parry, 1983). The metabolic rate of larger species may be affected more than that of smaller species because larger species not only have more tissue mass to grow, but also relatively more of their growth may be post-maturational because their longer lives would allow them to benefit more from this additional growth in terms of increased evolutionary fitness (e.g. increased fecundity associated with larger body size). Studies of the allometry of growth-related energy use and its relation to metabolic rate are required to evaluate this hypothesis. Another possibility is that indeterminate growth may result in species differences in adult size being more related to differences in cell number than cell size, thus causing the scaling of metabolic rate in ectotherms to be steeper, as predicted by the cell-surface hypothesis (Davison, 1955), than that seen in endotherms with determinate growth. These hypotheses may have merit, but they do not seem to have the same predictive power as the metabolic-level boundaries hypothesis already outlined.

Bejan's (2001) theoretical arguments (based on the dynamics of convective heat transfer within animal bodies) predict that the metabolic rate of endotherms should scale to the 3/4 power, whereas that of ectotherms should scale to the 2/3 power. However, these predictions are the opposite of what has usually been observed, as described above.

In addition, Atanasov & Dimitrov (2002) claim that the metabolic scaling exponent is a function of the complexity of animal organisation, and that this is why birds and mammals have lower scaling exponents than snakes and lizards. This may be true, but a mechanistic explanation for such an association has not yet been provided.

(*b*) *Effects of body-size interval*

Another empirical pattern not explained by models focusing on the 3/4-power law is the observation that small mammals show significantly shallower scaling relationships than large mammals [see Section II (4*b*)]. Savage *et al.* (2004*b*) claim that the model of West *et al.* (1997) predicts this difference, but unfortunately it does so incorrectly. It predicts that the smallest mammals should show metabolic scaling exponents $\geqslant 3/4$, but actually they show scaling exponents $< 3/4$ (nearer to 2/3 or even 1/3). Dawson (2003) has also shown that this 'transition' cannot be explained by the scaling of capillary dimensions and number, which appear to be similar in small and large mammals. He suggests that other differences in the oxygen-delivery system may be responsible, but these have not yet been identified.

Heat-transfer theory predicts that heat loss (and thus metabolic rate) should scale less steeply at small *versus* large body sizes in endothermic animals (Bejan, 2001). This prediction follows from the assumption that body thickness scales as $V_b^{1/3}$ (where V_b is body volume), and thus as body size decreases, conductive resistance to heat loss [which scales as $V_b^{1/3}/$ (body-surface area $\sim V_b^{2/3}) = V_b^{-1/3}$] becomes increasingly important relative to resistance to heat loss via internal tree-like convective currents (which scales as $V_b^{-3/4}$) (Bejan, 2001). This theory predicts metabolic scaling exponents of 1/3 and 3/4 at the extreme limits of small *versus* large body size, respectively.

Interestingly, McNab (1983, 2002) has shown that at small body masses (< 50 g) the maintenance of a high body temperature by continuous endothermy requires that metabolic rate scale as $M^{1/3}$. Moreover, according to McNab (1983), the metabolism of larger birds and mammals tends to scale as $M^{3/4}$. These empirically based scaling relations exactly match the theoretical predictions derived by Bejan (2001) using heat-transfer theory. However, some small birds and mammals show metabolic scaling exponents steeper than 1/3 apparently because they are heterothermic, rather than strictly endothermic. These animals (e.g. pocket mice and hummingbirds) have lower metabolic rates than strict endotherms of equivalent size (e.g. voles and some shrews and passerine birds); and they readily become torpid by allowing their body temperature to drop under energetically stressful conditions. Therefore, metabolic rate may scale differently in small *versus* large endotherms because the capacity for effective endothermy and temperature regulation itself scales with body mass (McNab, 1983, 2002).

The analyses of Bejan (2001) and McNab (1983) raise interesting questions about the different metabolic scaling relations observed in endothermic *versus* ectothermic vertebrates, as already discussed. Perhaps the key difference is not between endotherms and ectotherms *per se*, but between small strictly endothermic animals that exhibit relatively shallow metabolic scaling, and the rest of the animal world, which includes large endotherms, small heterotherms, and

small and large ectotherms. If this is true, then the 2/3-power scaling frequently observed in endothermic birds and mammals may be the result of the shallow scaling of strict endotherms at the small end of the body-size range tilting down what would otherwise have been a steeper scaling relation similar to that of ectothermic fish, amphibians and reptiles (*cf.* McNab, 1983). This effect may be relevant, but it is probably not the whole story because many ectothermic taxa show steeper metabolic scaling relations ($b > 0.8$) than that observed in large endotherms (presumed to be ~ 0.75 by McNab, 1983). In addition, phylogenetic effects should be examined to see whether the higher metabolic scaling exponent observed for large mammals is the result of examining a taxonomically more heterogeneous group of species. In McNab's (1983, 1988) analyses, only five orders of mammals were included in the small-bodied group (< 50 g), but 21 orders were included in the large-bodied group ($\geqslant 50$ g). The effect of taxonomic heterogeneity on the scaling of metabolism may be especially significant if metabolically different taxa are unevenly represented in different regions of the sampled body-size range. For example, the relatively large scaling exponent for large mammals may be artificially inflated by the heavy representation of artiodactyls at the high end of the body-size range. Microbial fermentation in the ruminant stomach of artiodactyls may prevent these animals from becoming postabsorptive, thus elevating their metabolic rates above basal levels (White & Seymour, 2003). A further complication is that differences between the metabolic scaling of small and large mammals appear to depend on geographical region (Lovegrove, 2000).

The model of Makarieva *et al.* (2003) predicts that the metabolic scaling exponent of endothermic vertebrates should be 2/3 at body masses < 20 kg, but should approach 1 at body masses > 20 kg. They provide data on mammals that seem to support their model, but they did not test for statistical differences between the scaling of small *versus* large species. Furthermore, their model surprisingly predicts that the scaling of metabolism should be steeper in large mammals because they have greater problems of overheating than smaller mammals. However, if overheating is a greater problem in larger mammals, then their metabolism should scale less steeply with increasing body mass, rather than more steeply. This confusion may have arisen because Makarieva *et al.* (2003) focus on mass-specific metabolic rates, which appear to scale ($b - 1$) less steeply in large *versus* small mammals, rather than on total metabolic rates, which show the opposite scaling (b) differences.

(*c*) *Effects of activity level*

Still another empirical pattern unexplained by models focusing on the 3/4-power law is that the scaling of metabolic rate varies with activity level. For example, maximal metabolic rate of exercising animals scales more steeply than resting metabolic rate [see Section II (4*c*)]. The model of West *et al.* (1997) assumes that resting metabolic rate is set by the rate of oxygen or fuel delivery, which is dictated by an optimally designed network system. However, the physical properties of this delivery system (including the respiratory and cardiovascular systems) should have evolved to accommodate the highest aerobic demands, and thus should be more involved in the scaling of maximal metabolic rate (which does not follow the 3/4-power law) than of resting metabolic rate (Bishop, 1999). If rate of resource supply limits resting metabolic rate, as West *et al.* (1997) claim, then 'there can be no room left for scope for activity: higher metabolic rates would be blocked by this one process because it would already be rate-limiting even at rest' (Darveau *et al.*, 2002). Tissue demand for fuel and oxygen must also be important [Darveau *et al.*, 2002; Hochachka & Somero, 2002; Weibel, 2002; Hochachka *et al.*, 2003; Kozłowski & Konarzewski, 2004; Suarez *et al.*, 2004; see also Sections III (2, 3*c*)].

West *et al.* (2004) have responded to this criticism by arguing that although the scaling of metabolic rate is limited by the supply rate of the resource-distribution network, the metabolic rate of an individual animal is not. An individual animal can vary its metabolic rate 'on demand by varying fuel supply'. However, this reply does not explain why the scaling of maximal metabolic rate is steeper than that of resting metabolic rate. Moreover, it does not explain how the resource-supply network could cause an elephant to have a lower mass-specific rate of basal metabolism than that of a mouse, when supply limits have clearly not been reached (*cf.* Suarez *et al.*, 2004). Capacities for supplying fuel and oxygen to the cells are much higher than that required in the resting state (Weibel, 2000).

The boundary approach proposed by Sibly & Calow (1986) explains both the 2/3-power scaling of basal metabolism in endothermic vertebrates, as well as the steeper scaling of maximal metabolic rate during activity. At rest, metabolic rate is largely set by the need to maintain a constant body temperature. Since heat loss is proportional to $M^{2/3}$, heat production (and thus metabolic rate) should also scale as $M^{2/3}$. However, during heavy exercise, metabolic rate is chiefly set by the need to fuel muscular activity. According to dimensional analysis, since maximal work per muscle volume per contraction scales as $M^{1.0}$ and muscle-contraction frequency scales as $M^{-0.18}$ in running birds and mammals, then maximal metabolic rate should scale as $M^{1.0-0.18} = M^{0.82}$, a prediction close to that actually observed. Kooijman (2000), Hulbert & Else (2000) and Marden & Allen (2002) have also argued that muscular power production should be more related to body mass/volume than to surface area. Larger mammals have more muscle mass (scaling as $M^{1.0}$: Calder, 1984; Hoppeler & Flück, 2002), which means more cell fibres for causing stronger contractions, more mitochondria for supplying energy, and more stored fuel (lipid) to help sustain a high frequency of contractions for a longer period of time (all scaling as $M^{0.91-0.99}$: Hoppeler & Flück, 2002).

Darveau *et al.* (2002) have also presented a 'multiple-causes model of allometry' in which whole-organism metabolism is considered to be a 'weighted average' of multiple processes, each with their own scaling exponents. Their model predicts that when an animal is at rest, maintenance costs (e.g. protein synthesis and ion transport) should dominate, with metabolic rate scaling as $M^{0.76-0.79}$. However, when an animal is exercising vigorously, the costs

of muscular work should dominate, with metabolic rate scaling as $M^{0.82-0.92}$. However, although this approach has merit, it is not completely novel as the authors claim (many other workers have suggested that multiple mechanisms or constraints may underlie metabolic scaling [e.g. Economos, 1982; Jobling, 1985; Sibly & Calow, 1986; Weiner, 1989; Withers, 1992; Hulbert & Else, 2000; Kooijman, 2000; see also Section III (2*c*)]. Nor is it the first to explain differences between the scaling of resting and maximal metabolic rates (see Sibly & Calow, 1986; Hulbert & Else, 2000). Furthermore, their specific model has several problems. First, it incorrectly predicts that resting metabolic rate in birds and mammals should scale as $M^{\sim 0.78}$, when in fact it appears to scale often as $M^{\sim 0.67}$. Second, the model has serious mathematical flaws; and its basic parameters, including the scaling exponents themselves, are purely empirically based, rather than being derived from fundamental physical or biological principles (Banavar *et al.*, 2003; West *et al.*, 2003; but see Darveau *et al.*, 2003; Hochachka *et al.*, 2003; Suarez *et al.*, 2004). Third, although the model reasonably and probably correctly assumes that the scaling of metabolic rate represents the weighted average of the different scaling exponents of several component biochemical processes, this assumption seems to be largely inconsequential given that recent research suggests that most of these processes actually scale very similarly with body mass anyway (Rolfe & Brown, 1997; Hulbert & Else, 2000). In a major review of the biochemical basis for metabolic rate in mammals, Rolfe & Brown (1997) conclude: 'it appears that differences in SMR [standard (resting) metabolic rate] seen in animals of different body mass and phylogeny are the result of a proportional change in the activity of most cellular processes' (p. 751). Hulbert & Else (2000) similarly state that: 'Although BMR [basal metabolic rate] varies considerably, an intriguing finding is that the percentage contribution of various processes to BMR appears to be relatively constant irrespective of the actual level of BMR' (p. 211; see also Else *et al.*, 2004).

Nevertheless, Hochachka & Somero (2002) point out that the activity of some enzymes does not scale as $M^{2/3}$ or $M^{3/4}$, which they say supports their view that the scaling exponent of whole-body metabolic rate is a composite of the scaling exponents of different biochemical processes. However, all of the scaling differences that they mention involve enzymes whose functioning is critical during exercise when different scaling relations apply than those observed at rest. All of the enzyme activities with scaling exponents $>3/4$ that they cite were analyzed in the gastrocnemius muscle of 10 mammal species (see Emmett & Hochachka, 1981). Since muscular work scales more steeply than maintenance metabolism, it is not surprising that muscle-enzyme activities also scale more steeply as well. For example, the activity of citrate synthase and other enzymes involved in oxidative metabolism scale as $M^{0.79-0.94}$, which is similar to the scaling of maximal metabolic rate during exercise ($M^{0.81-0.92}$). Muscle enzymes functioning in anaerobic metabolism scale even more steeply ($b \geqslant 1$), but they catalyze glycogen catabolism mainly during brief episodes of 'burst' locomotion, and thus contribute little to the fueling of sustained muscular work (see also Childress & Somero, 1990; Withers, 1992).

Thus, a fourth problem with the model of Darveau *et al.* (2002) is that it assumes that the scaling of enzyme activities is a cause rather than a result of whole-body metabolic scaling. This may be true, but they provide no convincing evidence for this assumption. Plausible arguments can be made for an opposite causal pathway or a process of co-adaptation as well [see also Section III (2*a*)]. Perhaps enzyme activities have been adaptively adjusted to match metabolic requirements that are controlled by other physiological and ecological factors. In line with this latter view, the scaling exponents for the activities of anaerobic (glycolytic) enzymes are >1 only in tissues that rely heavily on anaerobic metabolism (e.g. muscle), and not in those relying entirely on aerobic metabolism (e.g. brain) (Somero & Childress, 1980; Withers, 1992). Similarly, pelagic fish that are active swimmers exhibit steeper scaling exponents for muscle-enzyme activities than do relatively sluggish benthic fish (Childress & Somero, 1990; Somero & Childress, 1990).

However, Darveau *et al.* (2002) are correct in pointing out that metabolic scaling may be affected by the varying contributions that different tissues and organs make to whole-organism metabolism. Some organs (such as brain, heart, kidney and liver) have much higher metabolic rates than others (such as skin, muscle, and connective and white adipose tissues) (e.g. Krebs, 1950; Itazawa & Oikawa, 1986; Elia, 1992; Scott & Evans, 1992; Rolfe & Brown, 1997; Alexander, 1999; Hulbert & Else, 2000; Oikawa & Itazawa, 2003). Consequently, changes in the relative sizes and/or metabolic intensity of these organs with changes in body mass should and apparently do affect the scaling of metabolism with body mass [see also Section III (3*d*)].

Therefore, the relatively steep scaling of maximal metabolic rate in exercising birds and mammals may be, at least partly, the result of enhanced metabolic activity of tissues that have relatively low metabolic activity in the resting state (e.g. muscle). This effect is large, because muscle makes up a large proportion of the body mass of an adult animal (e.g. skeletal muscle constitutes 27–68 % of the total mass of vertebrates: Blaxter, 1989; Calder, 1984). In fact, in mammals over 90 % of the oxygen consumed during maximal metabolism is used for muscular work (Weibel, 2000; Weibel *et al.*, 2004). Data on the scaling of tissue metabolism in five mammals (Wang *et al.*, 2001) are consistent with this hypothesis of differential tissue effects. The metabolism of organs that are involved mainly in homeostatic activities scales similarly to $M^{2/3}$ [liver: $M^{0.60}$; brain: $M^{0.62}$; and kidney: $M^{0.77}$; which scale collectively as $M^{0.63}$; and similarly, in another study of eight mammals (Porter, 2001), liver metabolism scaled as $M^{0.64}$], whereas the metabolism of organs critically involved in supporting locomotor activity scales more steeply (heart muscle: $M^{0.86}$; skeletal muscle and other 'residual' tissues: $M^{0.84}$; which collectively scale as $M^{0.84}$).

The importance of muscle metabolism in affecting maximal metabolic rate during activity is further supported by the study of Hoppeler & Flück (2002), who show that muscle mitochondrial volume ($b=0.91$) scales similarly to maximal metabolic rate ($b=0.92$) in their sample of mammals (see also Wieser, 1995; Weibel, 2000; Weibel

et al., 2004). In addition, capillary density in muscle scales similarly to maximal metabolic rate ($b = 0.79$–0.93: Hoppeler *et al.*, 1981), whereas capillary density in homeostatic organs (e.g. kidney) scales more in line with resting metabolic rate ($b = 0.62$: Dawson, 2003). Perhaps a shift from metabolism driven by homeostasis to that driven by muscular work explains why the scaling of other estimates of active (sub-maximal) metabolic rates is often steeper than that of resting metabolic rates as well [see Sections II (3*e*) and II (4*c*)]. However, energetic costs of growth may also contribute to the relatively steep scaling of active metabolic rates in ectothermic animals with indeterminate growth (Peterson *et al.*, 1999).

A further prediction of the boundary approach is that, in birds and mammals, maximal metabolic rates associated with locomotion should scale more steeply (as $M^{0.82–1.0}$) than maximal metabolic rates associated with cold-induced thermogenesis (as $M^{2/3}$). This is because muscle metabolism is expected to be a function of muscle power (scaling as $M^{0.82}$) or muscle mass (scaling as $M^{1.0}$), whereas thermogenesis is expected to be a function of rate of heat loss (scaling as $M^{2/3}$). Data assembled by Weiner (1989) for mammals support this prediction: exercise-induced maximal metabolic rate scales significantly more steeply (as $M^{0.86}$) than cold-induced maximal metabolic rate ($M^{0.67}$). Similar marginally significant differences ($P < 0.10$) are seen within specific mammalian taxa, including rodents ($M^{0.77}$ *versus* $M^{0.66–0.68}$: Bozinovic & Rosenmann, 1989; Bozinovic, 1992; Hinds & Rice-Warner, 1992; *cf.* Rezende, Bozinovic & Garland, 2004) and marsupials ($M^{0.88}$ *versus* $M^{0.77}$: Hinds *et al.*, 1993). However, more data in other mammals and birds are needed to test this hypothesis adequately.

At this point, it is important to clear up a possible misunderstanding. According to the metabolic-level boundaries hypothesis [proposed in Section III (2*f*) and discussed in Sections III (3*b*) and III (4*a*)], animals with relatively high rates of resting metabolism should have less steep scaling relationships than those with lower rates of resting metabolism. However, in this section I appear to be advocating a contradictory view that maximal metabolic rates during activity should scale more steeply than resting metabolic rates. In the first case, higher metabolic rates are associated with shallower scaling, whereas in the latter they are associated with steeper scaling. How can I have it both ways? This apparent inconsistency can be resolved as follows.

Resting metabolic rates involve long-term homeostatic functions requiring a balance between supply and demand, whereas maximal metabolic rates during activity involve short-term muscular work that can temporarily operate with demand exceeding supply (*cf.* Hammond & Diamond, 1997; Speakman, 2000). Two different processes, involving different constraints, are at work. Consider first the process of homeostasis. When homeostatic energy requirements are low, metabolic rate should be directly proportional to the mass of tissues that is being maintained. However, when homeostatic energy requirements are high, it is more likely that surface-area constraints on the delivery of nutrients and release of wastes will become important, thus resulting in metabolic rate being proportional to $M^{2/3}$ (if external surface area is most important, as in endothermic animals), or $M^{3/4}$ (if internal surface area is most important). However, during heavy muscular work, demand is temporarily independent of supply (until muscle fatigue sets in), and thus at short-term maximal levels metabolic rate is again more nearly proportional to M^1 than to $M^{2/3}$ or $M^{3/4}$.

The above explanatory scheme circumvents the argument of Darveau *et al.* (2002) that resting metabolic rate cannot be controlled by surface-dependent exchange of nutrients/wastes because then there would be 'no room left' for supplying sufficient resources to support maximal metabolic rates. In addition, consider that homeostatic energy demands increased by high temperatures or other stresses may very well be constrained by supply limits in ectothermic animals that have evolved resource-supply systems adapted to low-energy lifestyles. Furthermore, in high-energy endothermic birds and mammals, surface area may act as a lower limit on the scaling of metabolic rate, rather than as an upper limit (*cf.* Sibly & Calow, 1986), except during thermal stress, though even in this case the maximum limit of metabolic rate has not been reached (see below). At rest, endotherms must at minimum produce enough heat to replace the heat lost through their body surfaces. This may be why the resting metabolism of endotherms tends to scale as $M^{2/3}$. However, this constraint does not prevent birds and mammals from attaining higher metabolic rates during times of activity.

Fig. 7 summarizes predictions of the metabolic level-boundaries hypothesis as they pertain to the scaling of metabolic rates in endothermic birds and mammals. Tissue maintenance is the lowest boundary constraint and it scales as M^1, as seen in torpid/hibernating mammals with depressed body temperatures. Resting metabolic rate includes thermal homeostasis, and thus scales as $M^{2/3}$. It acts as the lower boundary constraint in non-torpid birds and mammals. Maximal thermogenesis also scales as $M^{2/3}$, and it acts as the upper boundary constraint in non-active, thermally challenged birds and mammals. Maximal metabolic rate during activity scales as $M^{0.82–1.0}$ and is the highest boundary constraint in strenuously exercising birds and mammals. In both birds and mammals, exercise-induced maximal metabolic rates exceed cold-induced maximal metabolic rates (Hinds *et al.*, 1993).

(*d*) *Other organisms besides animals*

Indirect estimates suggest that metabolic scaling in plants may follow the 3/4-power law, and if so could be explained by the fractal geometry of nutrient-transport networks (Niklas, 1994, 2004; Enquist *et al.*, 1998; Savage *et al.*, 2004*b*). However, the fractal nature of the plant vascular system has been questioned by Kozłowski & Konarzewski (2004). They point out that leaf vasculature is a mesh of interconnected vessels, as in dicotyledons, or is a system of parallel vessels without interconnections, as in monocotyledons. However, the model of West *et al.* (1997) strictly applies to the nutrient-transport system connecting the base of the trunk to the petioles, and not to the leaves themselves (see West, Brown & Enquist, 1999*a*; Enquist, West & Brown, 2000). Further work is needed to resolve

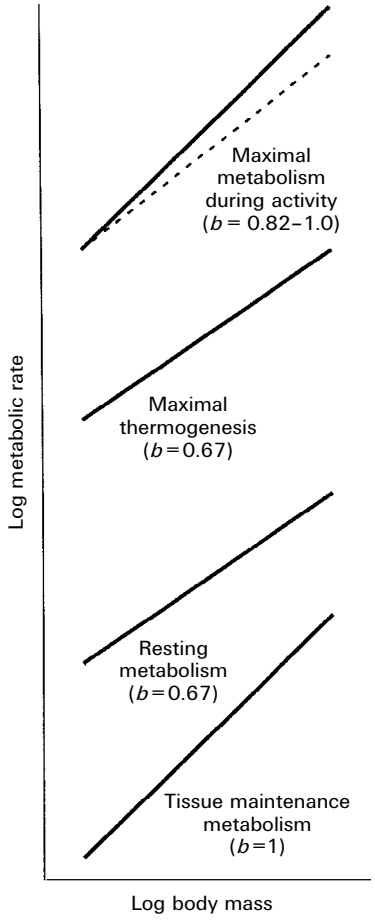

Fig. 7. Scaling of metabolism in endothermic birds and mammals, as predicted by the metabolic-level boundaries hypothesis. Tissue-maintenance metabolism is the lowest level of metabolism allowable for life and is observed in torpid/hibernating mammals. Resting metabolism occurs in non-active, non-torpid animals that are not thermally challenged. Maximal thermogenesis occurs in thermally challenged (cold-exposed) animals. Maximal metabolism during activity occurs during peak levels of exercise. The elevations of the scaling relations depicted are qualitatively correct, but are not meant to be quantitatively accurate.

how important the non-fractal transport networks of leaves are to metabolic scaling in plants. Presumably, they would be most important in plants that have relatively large ratios of leaf to (stem + root) biomass.

Phillipson (1981) suggested that the metabolic rate of unicells should scale to the 2/3 power, because cell-surface area, across which nutrients and metabolic waste products are exchanged, scales as $M^{2/3}$. His data support this suggestion, but other studies have revealed a variety of scaling exponents for unicells [see Section II (4*e*)].

Apparently the scaling of photosynthetic rate in phytoplankton depends on light intensity (Finkel *et al.*, 2004). Finkel *et al.* (2004) use a boundary approach to explain this finding. They assume that under conditions of light saturation, metabolic (photosynthetic) rate will be mainly influenced by the rate of transport of resources within the cell, and thus will scale as $V^{3/4}$ (where V is cell volume), following the model of West *et al.* (1997). However, when light is limited, metabolic rate will be primarily influenced by the rate of supply of resources to the cell, and thus will scale as $V^{2/3}$. Similar to expectations, $b = 0.79 \pm 0.04$ (± 2 SE) in phytoplankton under light saturation, but $b = 0.60 \pm 0.08$ under light limitation (Finkel, 2001; Fujiki & Taguchi, 2002). However, further research is needed to determine whether phytoplankton cells have the fractal nutrient-transport system required by the model of West *et al.* (1997).

IV. RECOMMENDATIONS FOR FURTHER RESEARCH

Seven major approaches may advance our understanding of the functional and evolutionary causes of various patterns of metabolic scaling, as follows.

(1) Employing rigorous statistical tests

Future empirical analyses should provide enough statistical information (e.g. confidence limits) so that it can be determined whether metabolic scaling exponents differ significantly from 3/4 or some other theoretical value. Tests should also be employed to determine whether scaling relations are linear or non-linear (e.g. Chappell, 1989). Controls for possible phylogenetic, ecological and/or geographic effects acting differentially across the sampled body-size range should also be attempted [see also Section II (1)].

(2) Exploring metabolic scaling at different scales

Most theoretical analyses of metabolic scaling have focused on broad-scale interspecific relationships. More attention should be given to relatively narrow-scale, intraspecific relationships, which have been relatively neglected. Even more neglected have been studies of intra-individual metabolic scaling, which have not been considered in this review. As observed in a few birds and mammals, the intra-individual metabolic scaling exponent ($b = 0.92$–1.88) is usually higher than that of intra- and interspecific scaling exponents (Daan *et al.*, 1989; Piersma, Cadée & Daan, 1995; Scott, Mitchell & Evans, 1996; Lindström, Klaassen & Kvist, 1999; McLean & Speakman, 2000; Kvist & Lindström, 2001), though Maxwell *et al.* (2003) reported an intra-individual scaling exponent ($b = 0.777$) in the iguanid lizard *Iguana iguana* (L.) that is not significantly different from the intraspecific scaling exponent ($b = 0.734$). Within specific animal taxa, scaling exponents for individual species (means and/or individual values) often differ significantly from interspecific exponents as well (e.g. Feldman & McMahon, 1983; Wieser, 1984; Andrews & Pough, 1985; Chappell & Ellis, 1987; Zari, 1993; Thompson & Withers, 1997; Clarke & Johnston, 1999; Kvist & Lindström, 2001). Further research is needed to explain these differences.

They are worth our attention because they suggest that no single universal physical law determines metabolic scaling at all scales (*cf.* Bokma, 2004; Clarke, 2004).

Nevertheless, studies of the intraspecific allometry of metabolic rate may illuminate our understanding of interspecific relationships and *vice versa*. I have three reasons for saying this. First, the optimization model of Kozłowski & Weiner (1997) has shown that intraspecific scaling relationships may significantly affect the scaling relationships that evolve among species (see also Kozłowski, 2000; Kozłowski *et al.*, 2003*b*). Second, the metabolic-level boundaries hypothesis successfully predicts several metabolic scaling patterns seen both within and among species [see Sections III (3*b*) and III (4*a*, *c*)], thus implying that both are affected by at least some of the same mechanisms or constraints. Although it is doubtful that a single physical law can account for both intra- and interspecific metabolic scaling, it is still possible that similar structural/physiological constraints may act as boundary limits on what is evolvable at both levels. Third, certain key processes appear to provide important insight into the mechanisms underlying both intra- and interspecific metabolic scaling [e.g. allometric scaling of various intracellular biochemical processes and of respiration rates at the cell and tissue levels; changes in body composition and cell-surface area/volume relationships during ontogenetic and evolutionary changes in body size; and the apparent influence of muscular energy expenditure on isometric or nearly isometric metabolic scaling observed within and among species engaged in active locomotion: see Sections III (3*c* & 4*c*)]. Future research exploring possible connections between intra- and interspecific metabolic scaling is much needed if we are to develop a truly general theory of metabolic scaling.

(3) Undertaking quantitative genetic studies

Quantitative genetic studies are needed to determine whether metabolic scaling is genetically variable and thus evolutionarily malleable. Glazier & Calow (1992) found that the scaling exponent was not significantly different between two genetic clones of *Daphnia magna* with different metabolic rates and life-history traits, but further work on other clones and species is needed to test the generality of this result. A lack of genetic variation in the scaling exponent would support the view that the scaling of metabolism is constrained both within and among species. By contrast, the existence of genetic variation in the scaling exponent would suggest that metabolic scaling can be modified by natural selection, with possible consequences for both intra- and interspecific allometries. Wu *et al.* (2002) present a statistical model for identifying the number and location of genes that affect allometric relationships. This model may prove useful in exploring the genetic basis of metabolic scaling.

(4) Undertaking comparative studies of little-studied taxa

Comparative studies of the ecological and physiological correlates of different types of metabolic scaling, both within and among species, may significantly improve our understanding of not only proximate causes (functional mechanisms), but also ultimate causes (adaptive values) of the allometry of metabolism. Data on metabolic scaling are still unavailable for many taxa. Studies of these taxa may not only serve as useful tests of patterns and hypotheses offered here and elsewhere, but also reveal new patterns worthy of explanation. For example, to my knowledge, no studies have been done on the metabolic scaling of any species or group of species of fungi, an entire kingdom of life! This could be very enlightening because surface area/volume constraints may not be important in these filamentous organisms, thus permitting their metabolic rate to scale isometrically with body mass. In addition, it would be interesting to compare the metabolic scaling of aerobic and anaerobic organisms. Aerobic metabolism is more likely to depend on geometric limits of exchange surfaces and transport networks than anaerobic metabolism, which appears to depend more on mass-related factors such as fuel (glycogen) supply (Childress & Somero, 1990; Goolish, 1991). Further detailed comparisons of pelagic and benthic forms within specific taxa may also be especially useful in helping us to decipher the various factors affecting metabolic scaling. Pelagic forms occur in 11 different phyla (May, 1994; Johnsen, 2001), though I have reviewed metabolic scaling data for only five.

(5) Employing experimental manipulations

The study of metabolic scaling has been largely based on comparative and theoretical approaches. These approaches are necessary and important, but they are poor at establishing cause and effect relationships. The experimental approach is best at doing this, and thus should be used more often in the study of metabolic scaling. Data from intraspecific experimental manipulations, in conjunction with relevant interspecific comparative analyses, may help illuminate the mechanisms underlying metabolic scaling both within and among species. Manipulations of many kinds of extrinsic (environmental) and intrinsic (biological) factors could be useful in this respect. For example, temperature manipulations are needed to test the metabolic-level boundaries hypothesis discussed in Sections III (2*f*, 3*b*, 4*a*, *c*). Numerous studies have already examined the effect of temperature on metabolic scaling, both within and among species [see Sections II (3*e*) and II (4*d*); Fig. 3; Table 5 in Appendix], but more carefully controlled experiments are now needed to make sense of the complex results that have been observed. Several investigators have also employed varying gas mixtures to explore the effects of surface area on oxygen-consumption rates (e.g. Beckenbach, 1975; Holloway & Geiser, 2001). Ultsch (1976) has demonstrated that lunged salamanders permitted to breathe air and water exhibit steeper metabolic scaling relationships than those allowed to breathe water only. These data and the higher scaling exponents observed in active lunged *versus* lungless salamanders (Whitford & Hutchison, 1967; Feder, 1977) provide strong evidence that respiratory surface area can significantly influence metabolic scaling.

An intrinsic factor that is especially easy to manipulate is activity level. As discussed in Sections II (3*e*, 4*c*) and

III (3*c*, 4*c*), metabolic scaling varies in relation to activity level, and in so doing provides insight into the mechanisms involved (*cf.* Suarez *et al.*, 2004; Weibel *et al.*, 2004). Remarkably, one can also manipulate body size, surface area, and modes of gas exchange in some organisms. For example, Nakaya *et al.* (2003) have shown that colonies of colonial tunicates can be cut into smaller unharmed colonies or fused into larger colonies. Studies of metabolic scaling in colonial organisms may be relevant to unitary organisms that are, in effect, colonies of cells. Some unitary organisms that tolerate body re-sizing (e.g. flatworms: Oviedo, Newmark & Sánchez Alvarado, 2003) may also be useful systems for the experimental study of metabolic scaling. In addition, sea anemones naturally alter their surface area through contraction and expansion of their tentacles, which results in significant changes in the allometry of metabolic rate (Shick *et al.*, 1979). Surgical or chemical procedures can also be used to block gas exchange in specialized respiratory organs, thus shifting all gas exchange to the skin, as in amphibians (e.g. Bentley & Shield, 1973; Shield & Bentley, 1973; Heath, 1976) and holothurians (Brown & Shick, 1979). Such manipulations may lead to significant changes in metabolic scaling as well (Brown & Shick, 1979). Furthermore, genetic engineering and developmental/hormonal manipulations could be used to alter other intrinsic factors, such as the sizes and/or metabolic intensities of specific organs or tissues, the areas of resource-exchange surfaces, the branching pattern of resource-supply networks, and the magnitude of various biological processes affecting metabolic rate, such as growth, reproduction, locomotion, and heat production.

(6) Testing both assumptions and predictions of theoretical models

Several intriguing models have recently been proposed to explain the scaling of metabolism, especially the 3/4-power law (see Table 3). However, testing of the assumptions and predictions of these models has barely begun. This is especially critical given the fact that metabolic scaling is highly variable and often significantly different from 3/4 (see Section II). It remains to be determined whether models designed to explain 3/4-power scaling are fatally flawed and should be discarded, or whether they can be modified in such a way to permit successful prediction of the diverse array of metabolic scaling relationships that actually exist. For example, Dodds *et al.* (2001) have shown that one can alter critical assumptions of the model of West *et al.* (1997) in reasonable ways to predict scaling exponents of 6/7 or 1, instead of 3/4. West *et al.* (2004) also show how including additional assumptions related to water balance in their model can explain isometric metabolic scaling in growing altricial chicks.

Other models that predict variable scaling relationships require similar scrutiny. For example, the optimization model of Kozłowski & Weiner (1997) explicitly shows how changes in the mean values and level of variation of intraspecific scaling parameters can affect the scaling exponent that evolves among species. In particular, the interspecific scaling exponent is predicted to increase as the variation in intraspecific scaling exponents and/or elevations increases, but this has yet to be tested.

(7) Synthesizing physiological and ecological explanations

Numerous biological phenomena, besides metabolic rate, are related to body size (Peters, 1983; Calder, 1984; Schmidt-Nielsen, 1984; Brown & West, 2000). These include heart-beat frequency, growth and reproductive rates, lifespan, activity level, home-range size and population density, to name a few. Within specific taxa many of these allometries show similar scaling exponents, suggesting that they are functionally linked. Typically, physical constraints associated with body size are regarded as the primary causes of the scaling of metabolism, which in turn, is thought to influence the allometries of other organism-, population- and community-level traits. This 'bottom-up' approach is promoted by West *et al.* (1997), who argue that physiological constraints, arising from the fractal nature of branching vascular networks, are the primary causes of the scaling of metabolism, and that ecological allometries are secondary results of this physiological scaling (see also Brown & West, 2000; Belgrano *et al.*, 2002; Brown *et al.*, 2003, 2004*b*; Dobson *et al.*, 2003; Enquist, 2003; Savage *et al.*, 2004*a*, *b*).

However, a 'top-down' approach is also possible. For example, Witting (1995, 1997, 1998) has claimed that ecological (foraging) constraints can explain the interspecific scaling of metabolism, and that physiological scaling is the secondary result, rather than the primary cause, of ecological allometries (see also Daan *et al.*, 1990). My survey of invertebrate scaling exponents also supports the importance of ecological factors in causing intraspecific metabolic scaling. For example, pelagic species with many different body sizes and structural designs (representing five different phyla) show isometric, or nearly isometric scaling, in contrast to the negatively allometric scaling of most non-pelagic species (Table 1; Fig. 1). This ecological effect can also be seen within many of these phyla. For example, in the Cnidaria, pelagic scyphozoans and hydrozoans have significantly steeper scaling relations than sessile anthozoans (ANOVA: $F_{2,34}=4.88$; $P=0.014$); and in the Urochordata, pelagic salps have significantly steeper scaling relations than sessile ascidians ($F_{1,10}=5.02$; $P=0.049$). Pelagic *versus* benthic differences in metabolic scaling can be observed at lower taxonomic levels as well. For example, the mean metabolic scaling exponent of four species of pelagic amphipods [$b=0.900\pm0.159$ (95 % C.L.); data from Yamada & Ikeda, 2003] is significantly steeper ($F_{1,22}=4.55$; $P=0.044$) than that shown by non-pelagic amphipods ($b=0.683\pm0.092$). In addition, many individual species of marine invertebrates have planktonic larvae showing isometric metabolic scaling, in contrast to the negatively allometric scaling shown by benthic adults (e.g. Zeuthen, 1953; Riisgård, 1998). Therefore, it is unlikely that metabolic scaling can be explained solely in terms of functional/physiological constraints associated with specific body designs.

There is, of course, a third possibility, and that is that causality has occurred in both directions (an 'up-and-down'

effect). In other words, physiological and ecological allometries may have co-evolved. Both constraints and selection are probably involved in the scaling of metabolism [see also Section III (2*e*)]. Furthermore, body size is not merely a constraint, but also is a variable subject to selection (*cf.* Clutton-Brock & Harvey, 1983; Daan & Tinbergen, 1997; Kozłowski & Weiner, 1997). Following this view, life-history theory can be used to increase our understanding of how body size and the rates of various correlated biological processes (e.g. metabolism, growth and reproduction) have evolved together as adaptive complexes. Size-related life-history variation – as embodied in the concepts of *r versus K*, wasteful *versus* frugal, and fast *versus* slow strategies (see MacArthur & Wilson, 1967; Szarski, 1983; Weiner, 1989; Kozłowski *et al.*, 2003*b*; Lovegrove, 2003) – may not simply be the result of size-related physical constraints (as argued by Brown *et al.*, 2004*b*), nor simply the result of ecological adaptation (as emphasized by Kozłowski & Weiner, 1997), but rather the result of both. Added insight into the causes of biological scaling may be acquired through multi-pronged analyses of the covariance of metabolic rate with other physiological, morphological, behavioural and ecological traits (e.g. by using multivariate comparative analyses, artificial selection and genetic engineering approaches, and other kinds of manipulative experiments; e.g. Moruppa, 1990; Rollo, 1995; Moody, Pomp & Nielsen, 1997; Selman *et al.*, 2001).

V. CONCLUSIONS

(1) To advance our understanding of the scaling of metabolism with body size, we should expand our focus beyond the 3/4-power scaling law, which is not universal within or among species. Rather than one scaling relation, there is a diversity of scaling relations that requires explanation.

(2) Existing theoretical models cannot explain the full range of variation in metabolic scaling that is observed at a variety of scales from individual species to all of life.

(3) This problem may be solved, at least partially, by explanatory schemes that recognize multiple boundary constraints and the differential scaling of the several component processes making up whole-organism metabolism.

(4) New insights about metabolic scaling may be acquired by exploring possible relationships between intra- and interspecific scaling patterns, by employing manipulative experiments and other under-utilized methodological approaches, by searching for both proximate (functional) and ultimate (evolutionary) causes, by considering the effects of evolutionary optimization in the context of multiple, rather than single constraints, and by viewing body mass and metabolic rate as features that have co-evolved with many other interrelated physiological and ecological traits.

(5) The scaling of metabolism – the 'fire of life', as Kleiber (1961) called it – is not the simple result of a physical law, but rather appears to be the more complex result of diverse adaptations evolved in the context of both physicochemical and ecological constraints.

VI. APPENDIX

Because of insufficient space here, Table 5 is online in the 'Selected Publications' section of the author's home page at http://faculty.juniata.edu/glazier/#PUBLICATIONS. Click on 'Table 5 of Appendix here' next to a citation of this review under the heading of 'Ecology of metabolic scaling'. Any reader who does not have access to the internet may request a copy of this table from the author.

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