

1

2 **The Space-Lifetime Hypothesis: viewing organisms in four**
3 **dimensions, literally**

4

5 Lev Ginzburg¹ and John Damuth²

6 ¹ Department of Ecology and Evolution, Stony Brook University, Stony Brook,

7 N. Y. 11794 USA (lev@ramas.com)

8 ² Department of Ecology, Evolution and Marine Biology, University of California, Santa

9 Barbara, CA 93106 USA (damuth@lifesci.ucsb.edu)

10

10

Abstract

11 Much of the debate about alternative scaling exponents may result from
12 unawareness of the dimensionality appropriate for different data and questions: in some
13 cases analysis has to include a fourth temporal dimension and in others it does not.
14 Proportional scaling simultaneously applied to an organism and its generation time,
15 treating the latter as a natural fourth dimension, produces a simple explanation for the $\frac{3}{4}$
16 power in large-scale interspecies comparisons. Analysis of datasets of reduced
17 dimensionality (e.g., ones constructed such that one or more of the four dimensions are
18 fixed), results in predictably lower metabolic exponents of $\frac{2}{3}$ and $\frac{1}{2}$, under one and two
19 constraints, respectively. Our space-lifetime view offers a predictive framework mutually
20 consistent with much of the content of existing “3-dimensional” theories, but does not
21 currently offer an alternate mechanism. Our view is useful as a step in developing a more
22 complete mechanistic theory of metabolic scaling.

23

23

Introduction

24 The $\frac{3}{4}$ power scaling of metabolism with animal body mass (Kleiber's
25 Law; Kleiber 1932) generalized to all forms of life (Brown et al. 2004; Hemmingsen
26 1960; Savage et al. 2004), has been not unlike Fermat's Theorem in the theory of
27 integers: it is an observation that has been relatively easy to see but hard to explain.

28 From the beginning, dimensional arguments have played an important role in
29 attempts to account for metabolic scaling. Before Kleiber, metabolism was thought to
30 scale as the $\frac{2}{3}$ power of mass, since organisms metabolize through two-dimensional
31 surfaces but supply a three-dimensional body (Rubner 1883). Recent work has produced
32 a largely satisfactory general explanation of the observed tendency for metabolic rates to
33 scale interspecifically according to Kleiber's law (instead of $\frac{2}{3}$) by focusing on the
34 geometry of organisms' internal distribution networks for metabolites or nutrients
35 (Banavar et al. 2002; Banavar et al. 1999; West et al. 1997; West et al. 1999). In this
36 theoretical approach the $\frac{3}{4}$ exponent results because the network scales as if it has a
37 metaphorical "extra" spatial dimension, related to the extra distances that a functional
38 network requires as it increases in size (but for different reasons, depending on the
39 models of different research groups). This characteristic of networks has been dubbed the
40 "fourth dimension of life" (West et al. 1999). However, here we discuss something
41 different: we argue that there is a distinct and literal sense in which the conventional
42 fourth dimension — time — may be profitably incorporated into biological scaling
43 theory. Our goal here is to adopt this literal (rather than metaphorical) four-dimensional
44 view of organismic scaling and explore novel predictions arising from it.

45 Part of our motivation is that even if network geometry explains the prevalence of
46 Kleiber's law, there is considerable variation in the degree to which different subsets of
47 organisms and taxa conform to it (Glazier 2005; White et al. 2007). The field of
48 Metabolic Ecology, recently "baptized" by Brown and colleagues (Brown et al. 2004),
49 has developed quickly over the last decade and incorporates many previously discovered
50 $\pm 1/4$ power allometries, including those for generation time (Bonner 1965), rate of
51 population increase (Fenchel 1974), population density (Damuth 1987; Damuth 2007)
52 and many others discovered and summarized by **earlier workers** (Calder 1984; Peters
53 1983; Savage et al. 2004). All exhibit variation and most are interrelated, such that
54 articulating an adequate theoretical account of the empirical complexity of metabolic
55 ecology appears to be a daunting task (Glazier 2005). A four-dimensional approach
56 reveals order and simplicity not readily apparent in the traditional three-dimensional
57 view.

58 Our point of departure is a well-known observation: With respect to body mass
59 (M) in a wide range of taxa, most life history traits scale either as approximately $M^{-1/4}$
60 (rates of physiological processes, and reproduction) or as $M^{1/4}$ (various times, including
61 generation time and lifespan, (Brown et al. 2004; Calder 1984)). It is striking that when
62 combined with the $3/4$ interspecific scaling of metabolism, such life history scaling gives
63 rise to a host of invariants or isometries with respect to body mass (Calder 1984; Charnov
64 1993). For example, lifetime metabolism scales as $M^{3/4} \times M^{1/4} = M^1$ and thus is
65 proportional (isometric, not allometric) to body size. As a consequence, since mass-
66 specific metabolism scales as $M^{-1/4}$, the lifetime metabolism of each gram of an
67 organism is independent of body size. Though frequently remarked upon, this

68 characteristic of the lifespan is usually considered an outcome of other scaling
69 relationships (Brown et al. 2004; Lindstedt and Calder 1981) and has not been treated as
70 a primary principle of scaling theory — although it has formed the basis of a theory of
71 aging (Pearl 1928). To us, these observations suggest that, instead, the scaling of
72 lifetimes may reflect a fundamental manner in which organisms of all body masses are
73 ecologically and evolutionarily functionally similar. Thus, we would expect that adding
74 time to scaling theory would simplify the theory with no loss of explanatory power.

75 Here we build forcefully on this suggestion by defending a simple proposition: it
76 is productive to view organisms as four-dimensional objects with three spatial
77 dimensions and one temporal dimension that is equal to the generation time. This *space-*
78 *lifetime* hypothesis has immediate implications. Scaling now has to be thought of as
79 simultaneous proportional change in all linear dimensions and in generation time. On this
80 view, $\frac{3}{4}$ scaling of metabolism is not at all surprising since the exchange of energy with
81 the environment takes place through a three-dimensional surface (two spatial and one
82 temporal) and expenditures are correspondingly four dimensional (three spatial and one
83 temporal). All the $\frac{1}{4}$ -power allometries for linear dimensions and life history follow
84 simultaneously from this simple view.

85 Blum (1977) reasoned similarly that if organisms were literally four-dimensional
86 then the $\frac{3}{4}$ exponent follows easily, but he did not suggest what that fourth dimension
87 should be. Time associated with physiological processes has been treated as an explicit
88 dimension in some physiological models of metabolism (da Silva et al. 2006; Heusner
89 1982b) and of course plays a key role in many others (e.g., Banavar et al. 2002).
90 However, in this paper we are concerned with ecological time, and specifically

91 **generation times.** Ecological time-related characters have been mentioned in the literature
92 as candidates for a fourth dimension, but this topic has not been explored further (Calder
93 1984; Hainsworth 1981).

94 It is a straightforward observation that, to a first approximation, the power of
95 unity in the lifetime metabolic-expenditure isometry ($M^{3/4} \times M^{1/4} = M^1$) is subdivided
96 into approximately equal quarters among the four total temporal and spatial dimensions:
97 lifespan scales as $M^{1/4}$ and metabolic rate per chronological unit of time as $M^{3/4}$. Purely
98 equal subdivision among the dimensions does not have to occur, and in fact there may be
99 many exceptions. For example, using the database of Froese and Pauly (2000) we
100 determined (Ginzburg, unpublished ms.) that the slope of metabolic rate of fishes, after
101 adjusting for temperature, is 0.84, higher than $3/4$. We found that at the same time fish
102 generation time scales with the exponent of 0.16, so the lifetime metabolism scales again
103 as power 1. In contrast, mammals show a more even distribution between temporal and
104 spatial dimensions (Calder 1984).

105 **Generation time as a dimension**

106 Why should generation time be so significant that it forms a fourth dimension for
107 organisms? Time units driven by astronomical events do not form a natural timescale for
108 biology. Although organisms may respond to various astronomical cycles, the periodicity
109 of such cycles depends upon accidental properties of the solar system and not the
110 functional requirements of biological systems. When we adopt a timescale more suitable
111 for organisms we would expect it to exhibit a clear relationship to processes important for
112 organismic function and fitness.

113 Since populations of established species tend to be roughly stable over the long
114 run, the per-capita rate of survival to the next generation has to be approximately unity.
115 That is, one surviving daughter of a size equal to its mother has to replace each mother
116 per generation. This is a requirement for ecological and evolutionary success.
117 Constructing one viable and reproductively capable daughter requires a certain duration
118 (a generation time) that is conveniently viewed as an organism's fourth dimension. So, on
119 average, it takes a generation time of metabolism for a mother to guarantee the existence
120 of her replacement. On this basis we deduce that the generation-time (and correlated
121 lifetime) metabolism should be isometric to body size, as described above. Thus
122 generation time is a plausible constraint inseparably linked to the size dimensions of an
123 organism through metabolism. Generation time is the fundamental timescale in studies of
124 evolution and in much of population dynamics, because of the obvious importance of
125 reproductive rates (Ginzburg and Colyvan 2004).

126 It is the average metabolic rate under natural conditions — the field metabolic
127 rate (FMR) — that is most relevant to this four-dimensional view, since organisms do not
128 typically live their entire lives at basal or standard metabolic rates. However, our analyses
129 are necessarily restricted to using basal rates, since currently there are too few species for
130 which both published FMR and life history data are available (Anderson and Jetz 2005;
131 Nagy et al. 1999). In any case, FMR scales roughly parallel to basal rates in vertebrate
132 taxa, and is close to $\frac{3}{4}$ in placental mammals (Nagy 2005). We expect that the results of
133 using basal rates will thus be comparable to use of FMR directly.

134 We have further found that the residuals of the scaling of basal metabolism and
135 the scaling of maximum lifespan covary negatively (226 species shared by datasets of

136 Savage, et al. 2004 and Ernest 2003; correlation coefficient -0.25 , $p < 0.0002$), although
137 the scatter is large. That is, a species that is overmetabolic with respect to the metabolism
138 line has a tendency to be below the line for generation-time allometry, and vice-versa.

139 We venture below to make some specific predictions based on our four-
140 dimensional view. We have been able to test some of them with satisfactory results;
141 others remain conjectures for future testing.

142 **Predicted and actual allometries for subsets of reduced dimensionality**

143 First, consider a set of organisms of different sizes that all share the same
144 generation time. This means that one dimension out of four is fixed and the organisms
145 differ only in three dimensions rather than four. Metabolism in a three-dimensional
146 system would be expected to scale not as $\frac{3}{4}$, but as $\frac{2}{3}$, consistent with the reasoning of
147 Rubner (1883) and other pre-Kleiber workers. However, from our four-dimensional view
148 the reason that the slope will be different is simply that one dimension has been removed.

149 An important special case of such three-dimensional sets is that members of a
150 single species have essentially the same generation time. Thus we would predict that
151 intraspecific metabolism would scale with a lower exponent, ideally $\frac{2}{3}$. This prediction
152 is in complete agreement with the well-known observation that intraspecific scaling
153 exponents for metabolism are often different than interspecific exponents and tend to be
154 closer to $\frac{2}{3}$ than to $\frac{3}{4}$ (Chown et al. 2007; Feldman and McMahon 1983; Glazier 2005).

155 Secondly, note that if, in a three-dimensional set of organisms, we standardize an
156 additional dimension (for example, one of the three spatial dimensions, say, body length),

157 we effectively remove two of the four dimensions and, by the foregoing reasoning,
158 expect the slope to be $\frac{1}{2}$ (i.e., the remaining variability is two-dimensional).

159 Substantial data are available to test these predictions for *Homo sapiens*. As a
160 single species it is three-dimensional and thus should exhibit a metabolic scaling
161 exponent of $\frac{2}{3}$; in fact, the data we have analyzed show the exponent equal to 0.63 with
162 a 95% confidence interval of 0.59 to 0.67 (Fig. 1A). We can further reduce the
163 dimensionality by performing a multiple regression of metabolic rate on both mass and
164 height, in which case we would expect a value of $\frac{1}{2}$ for the partial regression coefficient
165 associated with mass. In agreement with the prediction the observed value is 0.47 (0.43 –
166 0.51; Fig. 1 B). If, equivalently, we bin the individuals into groups of equal heights (0.01
167 log height [cm]), the mean slope for the scaling of metabolism within groups gives the
168 same result: 0.47 (0.42 – 0.52). Standard textbook formulas used in human physiology
169 that regress surface area for humans on their height and weight have the exponents of
170 weight varying between 0.43 and 0.54, in agreement with our own estimate (Dubin and
171 Zietz 1996; Dubois and Dubois 1916; Verbraecken et al. 2006).

172 We can perform the same test on an interspecific scale across placental mammal
173 species, with some caveats. The mammal data certainly incorporate a wider range of
174 variation in ecological and physiological constraints than do intraspecific data. In
175 particular, it is known that metabolism in small mammals (< 50g) scales with a much
176 shallower slope than it does in large mammals (Glazier 2005; McNab 1988) — see
177 below. Accordingly, we will restrict our analysis to species > 100g in body mass, among
178 which the allometric relationship is relatively uniform. We have also perforce used
179 maximum recorded lifespan to represent generation time; though an imperfect proxy,

180 lifespan does scale similarly to the other life history characters that jointly determine
181 actual generation times (Lindstedt and Calder 1981). Finally, we have not investigated
182 whether phylogenetic non-independence affects our estimates of slopes. Our interest here
183 is in a direct comparison with the human data for which no comparable genealogical
184 information is available. Moreover, published phylogenetically-based and non-
185 phylogenetic studies tend to yield similar exponents for the relevant allometries in
186 mammals, though some life history traits may be exceptions (Duncan et al. 2007; Martin
187 et al. 2005; Nagy 2005). We expect that the results of a phylogenetically-based analysis
188 would be qualitatively the same as ours, but an exploration of this additional complexity
189 is beyond the scope of the present work.

190 Table 1 shows that the results for mammals are similar to those for humans. In the
191 four-dimensional (unconstrained) case, the metabolic exponent is not different from $\frac{3}{4}$
192 and the 95% confidence interval does not include $\frac{2}{3}$. In the three-dimensional case
193 (controlling for lifespan), the exponent is lower, but variation is such that it is consistent
194 with either $\frac{2}{3}$ or $\frac{3}{4}$. In the two-dimensional case (controlling for both lifespan and
195 length), the exponent is 0.46, not significantly different from $\frac{1}{2}$ and almost exactly the
196 value that we obtained in the intraspecific case.

197 The focal values of $\frac{3}{4}$, $\frac{2}{3}$ and $\frac{1}{2}$ correspond to integer reductions in
198 dimensionality, and they seem to represent the modal values seen widely in metabolic
199 scaling (Glazier 2005). However, we can easily imagine fractional dimension reduction,
200 which would produce metabolic scaling exponents of various intermediate values. For
201 example, mammals are not perfect cubes, and the slope of the regression of body mass to
202 length tends to be slightly larger (up to 3.6) than the expected 3.0 in most orders (Damuth

203 1990; Silva 1998). The same exponent is closer to 2.8 for fishes (this paper) and for
204 mammalian carnivores (Van Valkenburgh 1990). Thus, constraining by body length
205 would be expected to have different effects in different groups, because slightly more or
206 slightly less than a full spatial dimension contributing to body mass is being standardized.

207 Actual morphological, developmental, or temporal constraints (as opposed to
208 those imposed statistically by the investigator) may also cause observed metabolic
209 allometries with powers outside of this simple set of $\frac{n-1}{n}$ fractions or with powers
210 unexpected from the apparent dimensionality of the system. For example, the low
211 exponents for metabolic scaling observed in small (< 50g) mammals ($\frac{1}{2}$ or even $\frac{1}{3}$; ref.
212 (Glazier 2005) immediately suggest to us that small mammal species form effectively at
213 most a two-dimensional set. We conjecture that small mammals experience constraints in
214 *both* spatial and temporal dimensions. At present we have no suggestions for the source
215 of the apparent reduction by an additional dimension. Nevertheless, the four-dimensional
216 view allows us to frame a novel question about the system that may lead to further
217 understanding. Likewise, some researchers argue that true basal metabolic rates of birds
218 and mammals scale with an exponent near $\frac{2}{3}$ (Glazier 2005; Heusner 1982a; White and
219 Seymour 2003). Should this turn out to be the case, it would suggest to us that under
220 basal conditions mammals experience some constraints that have the effect of reducing
221 the dimensionality by approximately one, in contrast to the $\frac{3}{4}$ scaling observed for FMR
222 and predicted by our four-dimensional perspective.

223

223

Discussion

224 The space-lifetime view predicts the $\frac{3}{4}$ exponent for metabolic scaling across
225 species. Significantly, it also successfully predicts the exponents of metabolic scaling in
226 sets of organisms of progressively lower dimensionality, and further correctly predicts
227 that intraspecific metabolic slopes will tend to be lower than interspecific slopes — and
228 ordinarily closer to $\frac{2}{3}$. Considering these observations and other conjectures discussed
229 above, we suggest that our proposed four-dimensional view of metabolic scaling is in
230 many ways simpler than the conventional 3-D view but with a similar and, in some cases,
231 superior predictive power.

232 We are aware that there are multiple explanations within the 3-D framework for
233 many of the same patterns that we address (Glazier 2005). Perhaps surprisingly, we
234 would argue that our theory is not likely to be a competing causal theory nor does it
235 necessarily contradict existing 3-D theories. We rely, informally, on the concept of
236 “duality” to suggest how this can be so.

237 Duality is a widely used concept in modern physics. The two dual theories
238 describe the same facts in different ways, typically by differing by one dimension. In a
239 sense they are the same theory, but distinct formulations that emphasize different aspects
240 or package the ingredients differently (Randall 2005). Neither 3-D nor 4-D metabolic
241 theory has yet been developed sufficiently to determine whether the theories are formally
242 dual. But it is in the spirit of such a possible duality that we offer our 4-D view. The fact
243 that we do not have a mechanistic 4-D model, yet see predictable relationships from that

244 perspective, strongly suggests duality with 3-D mechanistic theory rather than an
245 alternative or replacement.

246 We thus present our view at this time without a mechanistic underpinning.
247 Knowledge of regular patterns in nature without a concurrent understanding of their
248 underlying mechanisms is more common (and useful) in science than people often think
249 (Greene 2001). Darwin's lack of knowledge of the mechanisms of heredity (which we
250 now understand), or physics' lack of a mechanism for gravity (which we still do not
251 understand) are just two examples. Our presentation of a non-mechanistic framework
252 means only that this represents less of an intellectual advance than one would strive for. It
253 is in this spirit of stepwise progress that we offer our views.

254 When we add generation time to scaling theory as an organism's fourth
255 dimension, we see order involving metabolic exponents that was previously obscured.
256 The exponents depend in a simple way on the dimensionality of the set of organisms
257 being considered: $\frac{1}{2}$ for two dimensions, $\frac{2}{3}$ for three, $\frac{3}{4}$ for four. We believe that our
258 view can serve as a general organizing framework, within which various theories and
259 mechanisms may coexist peacefully, occupying their own (sub)space of correctly
260 identified dimensionality. Instead of expecting universal applicability of one of the
261 exponents (e.g., $\frac{3}{4}$, $\frac{2}{3}$ or $\frac{1}{2}$), we expect to see various exponents based on variation in
262 dimensionality. The four-dimensional view thus embraces network theory, aimed at
263 explaining the central tendencies of interspecific scaling, and simultaneously other
264 approaches, including those involving multiple constraints (e.g., Demetrius 2006; Glazier
265 2005; Kooijman 2000) that seek to explain much of the variation in metabolic scaling at
266 various scales and in particular groups. At the same time, the scaling patterns predicted

267 and successfully explained by the four-dimensional view offer a challenge to traditional
268 theories, which must account for them.

269 Including the temporal dimension as an integral part of the organism's phenotype
270 may have broader applications in ecology than just those involved with metabolism and
271 scaling. If organisms are considered to occupy a 4-dimensional space, then time, like the
272 dimensions of 3-D space, can be considered a resource. Where time for growth and
273 reproduction is in short supply there are fewer resources to be divided, with implications
274 for diversity, resource partitioning, and biogeography. Other ecological processes
275 ultimately depending on reproductive rates (such as population fluctuations and local
276 extinction probability) must depend partly on generation time. We speculate that an
277 extended four-dimensional view, if confirmed by additional studies, may provide similar
278 clarification of theoretical areas of ecology currently based in three dimensions.

279 Generation time has always been the fundamental unit of time for understanding
280 evolution. Our suggested view of metabolic ecology is that a generational time scale is
281 equally fundamental for ecology. A well-known metaphor by Hutchinson (1965) sets
282 ecology as a theater and evolution as a play. We believe that the theatre's clock ticks at
283 the same rate that the play is being performed. The coincidence of the basic time scale of
284 ecology to that of evolution is another confirmation of the unity of the two fields of
285 biology.

286 **Acknowledgments**

287 Our special acknowledgment is to Jim Brown, who encouraged us to develop this
288 idea further when Lev Ginzburg presented it at a seminar in New Mexico in May 2006.
289 Discussions with Geoffrey West, Douglas Glazier, Richard Sibly, and Francisco

290 Bozinovic helped in focusing on the appropriate tests. Emil Aalto worked on the fish
291 data and Roberta Harnett analyzed some of the mammal and all of the human data.
292 Marina Silva graciously provided mammalian length data and Dr. Steven Heymsfield
293 supplied human metabolism, body size and height data. Oskar Burger, Christopher Jensen
294 and Jeffrey Yule made useful comments on the manuscript.
295

295

References

296 Anderson, K. J., and W. Jetz. 2005. The broad-scale ecology of energy expenditure of
297 endotherms. *Ecology Letters* 8:310-318.

298 Banavar, J. R., J. Damuth, A. Maritan, and A. Rinaldo. 2002. Supply-demand balance and
299 metabolic scaling. *Proceedings of the National Academy of Science, USA* 99:10506-
300 10509.

301 Banavar, J. R., A. Maritan, and A. Rinaldo. 1999. Size and form in efficient transportation
302 networks. *Nature* 399:130-131.

303 Blum, J. J. 1977. Geometry of 4-dimensions and relationship between metabolism and body-
304 mass. *Journal of Theoretical Biology* 64:599-601.

305 Bonner, J. T. 1965. *Size and cycle*. Princeton, NJ, Princeton University Press.

306 Brown, J. H., J. F. Gillooly, A. P. Allen, V. M. Savage, and G. B. West. 2004. Toward a
307 metabolic theory of ecology. *Ecology* 85:1771-1789.

308 Calder, W. A., III. 1984. *Size, function, and life history*. Cambridge, Massachusetts, Harvard
309 University Press.

310 Charnov, E. L. 1993. *Life history invariants: some explorations of symmetry in evolutionary*
311 *ecology: Oxford series in ecology and evolution*. Oxford, Oxford University Press.

312 Chown, S. L., E. Marais, J. S. Terblanche, C. J. Klok, J. R. B. Lighton, and T. M. Blackburn.
313 2007. Scaling of insect metabolic rate is inconsistent with the nutrient supply network
314 model. *Functional Ecology* 21:282-290.

315 da Silva, J. K. L., G. J. M. Garcia, and L. A. Barbosa. 2006. Allometric scaling laws of
316 metabolism. *Physics of Life Reviews* 3:229-261.

317 Damuth, J. 1987. Interspecific allometry of population density in mammals and other animals:

318 the independence of body mass and population energy-use. *Biological Journal of the*
319 *Linnean Society* 31:193-246.

320 —. 1990. Problems in estimating body masses of archaic ungulates using dental measurements,
321 Pages 229-253 *in* J. Damuth, and B. J. MacFadden, eds. *Body size in mammalian*
322 *paleobiology: estimation and biological implications*. New York, Cambridge University
323 Press.

324 —. 2007. A macroevolutionary explanation of energy equivalence in the scaling of body size and
325 population density. *American Naturalist* 169:621-631.

326 Demetrius, L. 2006. The origin of allometric scaling laws in biology. *Journal of Theoretical*
327 *Biology* 243:455-467.

328 Dubin, S., and S. Zietz. 1996, Body surface estimation: a critical valuation. P. K. Bajpai, ed.
329 *Proceedings of the 1996 Fifteenth Southern Biomedical Engineering Conference*:397-
330 400.

331 Dubois, D., and E. F. Dubois. 1916. A formula to estimate the approximate surface area if height
332 and weight be known. *Archives of Internal Medicine* 17:863-871.

333 Duncan, R. P., D. M. Forsyth, and J. Hone. 2007. Testing the metabolic theory of ecology:
334 allometric scaling exponents in mammals. *Ecology* 88:324-333.

335 Ernest, S. K. M. 2003. Life history characteristics of placental non-volant mammals. *Ecology*
336 84:3402-3402.

337 Feldman, H. A., and T. A. McMahon. 1983. The 3/4 mass exponent for energy metabolism is not
338 a statistical artifact. *Respiratory Physiology* 52:149-163.

339 Fenchel, T. 1974. Intrinsic rate of natural increase: the relationship with body size. *Oecologia*
340 14:317-326.

341 Froese, R., and D. Pauly. 2000. FishBase 2000: concepts, design and data sources: ICLARM,
342 Los Baños, Laguna, Philippines. <http://www.fishbase.org>.

343 Ginzburg, L. R., and M. Colyvan. 2004. Ecological orbits: how planets move and populations
344 grow. New York, Oxford University Press.

345 Glazier, D. S. 2005. Beyond the '3/4-power law": variation in the intra- and interspecific scaling
346 of metabolic rate in animals. *Biological Reviews* 80:611-662.

347 Greene, M. 2001. A tool, not a tyrant. *Nature* 410:875.

348 Hainsworth, F. R. 1981. *Animal Physiology: Adaptations in Function*. Reading, MA, Addison-
349 Wesley.

350 Hemmingsen, A. M. 1960. Energy metabolism as related to body size and respiratory surfaces,
351 and its evolution. *Reports of the Steno Memorial Hospital and the Nordisk*
352 *Insulinlaboratorium* 9:1-110.

353 Heusner, A. A. 1982a. Energy metabolism and body size. I. Is the 0.75 mass exponent of
354 Kleiber's equation a statistical artifact? *Respiration Physiology* 48:1-12.

355 —. 1982b. Energy metabolism and body size. II. Dimensional analysis and energetic non-
356 similarity. *Respiration Physiology* 48:13-25.

357 Hutchinson, G. E. 1965. *The ecological theater and the evolutionary play*. New Haven, Yale
358 University Press.

359 Kleiber, M. 1932. Body size and metabolism. *Hilgardia* 13:315-353.

360 Kooijman, S. A. L. M. 2000. *Dynamic energy and mass budgets in biological systems*.
361 Cambridge, Cambridge University Press.

362 Lindstedt, S. L., and I. Calder, W. A. 1981. Body size, physiological time, and longevity of
363 homeothermic mammals. *Quarterly Review of Biology* 56:1-16.

364 Martin, R. D., M. Genoud, and C. K. Hemelrijk. 2005. Problems of allometric scaling analysis:
365 examples from mammalian reproductive biology. *Journal of Experimental Biology*
366 208:1731-1747.

367 McNab, B. K. 1988. Complications inherent in scaling the basal rate of metabolism in mammals.
368 *Quarterly Review of Biology* 63:25-54.

369 Nagy, K. A. 2005. Field metabolic rate and body size. *Journal of Experimental Biology*
370 208:1621-1625.

371 Nagy, K. A., I. A. Girard, and T. K. Brown. 1999. Energetics of free-ranging mammals, reptiles,
372 and birds. *Annual Review of Nutrition* 19:247-277.

373 Pearl, R. 1928. *The rate of living: being an account of some experimental studies on the biology*
374 *of life duration.* New York, Alfred A. Knopf.

375 Peters, R. H. 1983. *The ecological implications of body size.* Cambridge, Cambridge University
376 Press.

377 Randall, L. 2005. *Warped passages: unraveling the mysteries of the universe's hidden*
378 *dimensions.* New York, Harper-Collins.

379 Rubner, M. 1883. Über den Einfluss der Körpergrösse auf Stoff- und Kraftwechsel. *Zeitschrift*
380 *für Biologie* 19:535-562.

381 Savage, V. M., J. F. Gillooly, W. H. Woodruff, G. B. West, A. P. Allen, B. J. Enquist, and J. H.
382 Brown. 2004. The predominance of quarter-power scaling in biology. *Functional Ecology*
383 18:257-282.

384 Silva, M. 1998. Allometric scaling of body length: elastic or geometric similarity in mammalian
385 design. *Journal of Mammalogy* 79:20-32.

386 Van Valkenburgh, B. 1990. Skeletal and dental predictors of body mass in carnivores, Pages

387 181-205 in J. Damuth, and B. J. MacFadden, eds. Body size in mammalian paleobiology:
388 estimation and biological implications. New York, Cambridge University Press.

389 Verbraecken, J., P. Van de Heyning, W. De Backer, and L. Van Gaal. 2006. Body surface area in
390 normal-weight, overweight, and obese adults. A comparison study. *Metabolism Clinical
391 and Experimental* 55:515-524.

392 West, G. B., J. H. Brown, and B. J. Enquist. 1997. A general model for the origin of allometric
393 scaling laws in biology. *Science* 276:122-126.

394 —. 1999. The fourth dimension of life: fractal geometry and allometric scaling of organisms.
395 *Science* 284:1677-1679.

396 White, C. R., P. Cassey, and T. M. Blackburn. 2007. Allometric exponents do not support a
397 universal metabolic allometry. *Ecology* 88:315-323.

398 White, C. R., and R. S. Seymour. 2003. Mammalian basal metabolic rate is proportional to body
399 mass^{2/3}. *Proceedings of the National Academy of Science, USA* 100:4046-4049.