

Chapter 2

Adaptive Aspects of Development: A 30-Year Perspective on the Relevance of Biomechanical and Allometric Analyses

Karl J. Niklas

Any discussion of adaptation is in danger of going around in circles unless the inherently comparative nature of adaptation is fully recognized at the outset

— Horn et al. (1982)

2.1 Introduction

The biologists contributing to the 1982 group report on the *Adaptive Aspects of Development* described themselves as so “dazzled by the variety of developmental patterns displayed by . . . animals and plants” that their “discussions were severely hampered at the outset by a lack of the most basic information” and called for “more critically comparative reviews about the constraints on development and possible adaptive patterns of development” (Horn et al. 1982, p. 217, pp. 230–1). They ended their report with a wish list of research goals, each ultimately requiring an understanding of the limits of developmental variation and a careful assessment of whether this variation results from natural selection, the operation of physical forces, historical accident, or some combination of all three (Horn et al. 1982). To achieve these goals, the participants repeatedly alluded to how biophysical and allometric analyses could help biologists understand the interactions among (and the constraints imposed by) size, shape, and development—a sentiment that resurfaces in many of the other conference group reports and papers (e.g., Bonner and Horn 1982; Gould 1982).

The conviction that biophysics and allometry could provide insights into adaptive evolution by natural selection (or any other evolutionary phenomenon) is understandable. Biophysical analyses quantify the extent to which physical laws and processes set specific kinds of limits on growth, development, and the final appearance of organic structures. They offer one way to understand the functional

K.J. Niklas (✉)

Section of Plant Biology, School of Integrative Plant Biology, Cornell University,
Ithaca, NY 14853, USA

e-mail: kjn2@cornell.edu

relationship between organic form and the abiotic environments in which it operates. In turn, the effects of physical laws are size-dependent and allometric analyses offer an important tool with which to evaluate size-dependent effects. In addition, biophysical and allometric analyses can be applied to extinct as well as extant organisms. They therefore provide a comparative approach that is fully capable of spanning all of evolutionary history.

The goal of this paper is to review the status of biophysical (*sensu* biomechanical) and allometric analyses in 1981 and to see if these disciplines have matured in ways that can achieve the aims of the 1981 Dahlem conference. The topic of network theory in the context of developmental biology is also treated. However, my disciplinary focus is more on allometric than biomechanical analyses. In part this is because biomechanics *sensu stricto* is grounded in physics and engineering, which by their nature have not fundamentally changed in ways that would alter our perception of evolution or adaptation. But, more importantly, I focus on allometric analyses because of recent (and controversial) attempts to formulate explanations for allometric trends, which have a direct bearing on whether evolutionary history largely reflects the operation of natural selection, physical forces, historical accidents, or some combination of all three (e.g., West et al. 1997, 1999; Savage et al. 2004).

The organismic focus of my review is primarily on plants for four reasons. First, the scope of biodiversity is so vast that no *experimentum crucis* exists. Because every lineage has a unique history, even if debates about evolutionary patterns are resolved for one lineage, their resolution remains problematic for other lineages in the absence of detailed study. Second, plants (here defined broadly as eukaryotic photoautotrophs) comprise over 90 % of all visible living matter. Therefore, if generalizations about evolution are based on the relative frequency of phenomena, plants provide an opportunity to formulate broad statements about evolutionary phenomena. Third, plant development and structure are simple compared to those of animals. It is easier therefore to analyze them. Fourth, I am a botanist.

Regardless of this phytocentric bias, my thesis is that the participants of the 1981 Dahlem conference knew that neither biomechanics nor allometry *sensu stricto* could provide mechanistic explanations for the phenomena that occupied their attention because these disciplines lacked mathematical formulations that could make their observational consequences explicit. The 1981 Dahlem attendees however did know that both disciplines were indispensable tools for formulating quantitatively rigorous hypotheses about how physical laws and processes affect development and evolution. Thus, even in the complete absence of an explanatory foundation, biomechanical analyses can be used to quantify how well a specific structure or organism performs a particular function or set of functions, even though it cannot explain why that structure evolved and survived natural selection. In this manner, biomechanics can help to explain why some combinations of structures occur rarely or not at all, while other combinations are more commonplace, especially when biophysical analyses are wedded to theoretically constructed morphospaces (e.g., Raup 1962; Niklas 1997; McGhee 1999). By the same token, allometric analyses can identify size-dependent or size-covariant trends during an organism's ontogeny, or across a spectrum of different species differing in size, without recourse to any underlying theory (Niklas 1994).

Another issue is whether allometric or biophysical analyses have evolved sufficiently to address some of the major questions raised by the 1981 Dahlem conference. It is true that both approaches have become more sophisticated technologically. It is also true that attempts have been made to formulate comprehensive theories about size-dependent trends (e.g., West et al. 1997, 1999; Savage et al. 2004). However, technological sophistication does not translate automatically into explanatory insight, nor can a theory be said to exist until its principal assumptions are tested and confirmed experimentally. Seen in this light and based on the available evidence, I believe that little has actually changed conceptually since 1981 and that mechanistic explanations for the phenomena discussed in the Dahlem proceedings continue to be elusive. Attempts to answer these questions must rely on additional disciplines, such as genomics and molecular developmental biology, although these also present their own set of conceptual challenges.

2.2 Biomechanics and Allometry in 1981

The state of biomechanical research around the time of the 1981 Dahlem conference is effectively summarized in the seminal book entitled *Mechanical Design in Organisms* (Wainwright et al. 1976). This book was cited in three separate contributions to the 1982 published proceedings. It therefore undoubtedly served as the then current model for what biomechanics could bring to a discipline that was to become known as evolutionary developmental biology (evo-devo).

Mechanical Design in Organisms reviews the fundamental principals of engineering theory clearly, perhaps even brilliantly. It also shaped the course of subsequent research in the areas of animal biomechanics and biomimetics. However, compared to more recent treatments, its scope was limited in a number of ways that must have caused the 1981 Dahlem participants some consternation. First, although it treats the mechanical properties of bones and other parts of the mammalian body plan, it deals largely with the physical properties of invertebrates; plants are mentioned four times but only in terms of the material properties of tissues and materials, such as wood and cellulose, neither of which are sufficient to cover even the basic aspects of plant biomechanics (see Niklas 1992). It therefore lacked the broad comparative phyletic scope the 1981 participants called for. Second, *Mechanical Design in Organisms* touches on the ecological aspects of animal biomechanics only briefly and on evolution (adaptive or otherwise) not at all.¹ Third, even though allometry is an integral part of biomechanical analyses, the effect of size is considered only in the context of defining stress, flexural stiffness, and other engineering size-dependent properties. Fourth, it provides only a cursory treatment of fluid mechanics—a topic treated at length (but removed from virtually any consideration of solid mechanics) by Steven Vogel in his

¹ These topics were treated later by Wainwright and Reilly (1994).

extremely influential and important 1981 book *Life in Moving Fluids*. The lack of a more extensive treatment of fluid mechanics in *Mechanical Design in Organisms* is curious for at least two reasons: many of the co-authors of *Mechanical Design in Organisms* are well known for their research on marine invertebrates and most of the organisms used as mechanical examples are aquatic.

Biomechanics came of age post-1981 Dahlem. However, seen from the perspective of 1981, the discipline as summarized by *Mechanical Design in Organisms* and *Life in Moving Fluids* must have appeared quite fragmented with an almost idiosyncratic fixation on the mechanical properties of the hard parts of marine invertebrates. Perhaps for this reason, the word “biomechanics” never actually appears in the 1982 published Dahlem proceedings. Arguably, this state of affairs very likely contributed to the participants’ desire for more data drawn from a broader ecological and phyletic spectrum of organisms.

The esteem in which allometry was held at the time can be gleaned from reading Stephen J. Gould (1977) who, as a participant of the 1981 conference, opined that “Although allometric cases could be catalogued by the hundreds, it is not clear that they qualify . . . as a ‘mechanism of macroevolution.’ They arise as a result of change in developmental timing, but they produce a small quantitative output for a small quantitative input” (Gould 1982, pp. 336–7). Nevertheless, Gould added “. . . allometric variants, although small and graded as their inputs, might still have relevance to macroevolution . . . if they constrain available variation and impart a preferred direction to evolutionary change not based upon natural selection (but upon systems of covariation in development)” (Gould 1982, p. 337).

Even though he used allometry as an adaptationist early on in his career (Gould 1966), Gould’s subsequent distaste for adaptationist explanations (or unquantifiable speculations about adaptation) is clearly outlined in his later publications. However, there are three additional reasons why he may have underplayed the usefulness or relevance of allometry other than the absence of evidence for the covariance of developmental phenomena. First, no credible explanation for size-dependent phenomena existed at the time. Even J.S. Huxley (the co-inventor of the word “allometry”) failed to identify a rational and objective explanation for his now famous formula (i.e., $Y = bX^a$) in his 1932 book *Problems of Relative Growth*. Second, statisticians and biologists even now vigorously disagree as to which among competing regression protocols are the most appropriate for size-dependent analyses (e.g., Smith 1980; Harvey 1982; Seim 1983; Ratner 1985; Prothero 1986; McArdle 1988; Jolicoeur 1990; Riska 1990). And, third, the very need for these protocols in allometric analyses had been called into question by no less a personage than D’Arcy Thompson—to whom Huxley dedicated *Problems of Relative Growth* (Thompson 1942).

Given this state of affairs, it is reasonable to ask why the 1981 Dahlem participants gave biomechanical (or more accurately “biophysical”) and allometric analyses as much attention as they did. There are at least two plausible reasons. First, any effort to determine whether physical laws and processes influence evolutionary history, development, or biological form-function relationships has to allude at the very least to a biophysical approach—no organism, plant or animal,

can obviate the laws of physics or chemistry. Second, the focus of many of the participants (particularly Gould) on heterochrony necessitated a discussion of allometry as a descriptive tool at the very least. Indeed, a careful reading of the 1982 conference proceedings suggests that the participants were interested in biomechanics or allometry only to the extent that these disciplines could be applied to resolving the relative importance of adaptive evolution by means of natural selection versus physical constraints and to assess the effects of size and developmental timing on organic shape.

2.3 Progress Since 1981?

Ironically perhaps, the faint hopes raised by the 1981 Dahlem participants that biophysical and allometric approaches could answer some of their questions stimulated other researchers, such as myself, to adopt a biophysical/mathematical approach to understanding development and evolution. Although my undergraduate degree was in mathematics, my Ph.D. thesis dealt with the morphology and chemistry of early Paleozoic, taxonomically problematic plant fossils. In 1982, I was using organic geochemistry to help resolve the phylogenetic affinities of these and other enigmatic plant fossils. However, after reading the 1982 Dahlem publication, I was inspired to return to my love of mathematics and physics and use these tools to quantify form-function relationships for both extinct and extant plants. Indeed, part of my frustration as a trained paleobotanist was my inability to actually experiment on the plants that I was studying. After reading the conference proceedings, *The Mechanical Design of Organisms*, and *Life in Moving Fluids*, I learned how to do the experiments I had only dreamed of before. This necessitated familiarizing myself with basic engineering, delving more deeply into statistics, and learning how to build wind tunnels. But a wonderful door had opened in my life. And I happily walked through it.

Biomechanical and allometric practice and theory have seen considerable progress since 1982. In biomechanics, new technologies have expanded the experimental repertoire of testing organic materials and structures even at the cellular and subcellular levels (for a review, see Niklas 1992). Nonlinear viscoelastic theory has advanced to a level where we can predict the behavior of fluid-like, organic materials. Indeed, the conceptual and practical problems that once separated the study of fluid mechanics from solid mechanics in books like *The Mechanical Design of Organisms* and *Life in Moving Fluids* (Vogel 1981) have been nearly eliminated. Genetically modified organisms have also been developed to test theories about the mechanical roles of particular materials, e.g., tobacco plant mutants lacking the ability to synthesize lignin have been used to determine whether lignin strengthens stems and leaves. In the field of allometric analyses and statistics, large data sets spanning thousands of species have been assembled for detailed analyses, and consensus has been reached (more or less) on the statistical protocols and computer software required for these analyses, e.g., reduced major

axis (RMA—otherwise known as standardized major axis) regression analysis has emerged as the “standard” protocol (Niklas 1994, 2004).

The real issue however is not whether progress has been made in the *general* advance of studying biomechanical and allometric phenomena, but rather whether progress has been made in providing mechanism-based explanations for phenomena that intrigued the 1981 Dahlem conference participants. Put differently, have these disciplines advanced beyond merely quantifying phenomenology? This question is addressed in the following section in the context of allometry.

2.4 Theories Versus Phenomenological Descriptions

J.S. Huxley was not the first to notice that much of the organic variation attending ontogenetic changes in size can be described by a simple formula. However, much like Charles Darwin, Huxley was the first to demonstrate convincingly that a very large body of data collected from diverse organisms could be described under the rubric of an intellectual construct and to publish this finding in the form of a book. Largely through Huxley’s efforts and the publication of his *Problems of Relative Growth* (Huxley 1932), the allometric formula $Y = bX^a$ gradually became an accepted analytical tool.

Nevertheless, the absence of an explanation for this formula was painfully noticeable from its inception. In his otherwise very favorable review of *Problems of Relative Growth*, the polymath C.F.A. Pantin wrote that Huxley’s

formula is necessarily empirical. Of the causes of differential growth we have little knowledge; their investigation is the problem at issue. A variety of possible relations might, in fact, reduce approximately to this formula. But it is not the object of the formula to establish the correctness of a particular hypothesis as to the cause of differential growth; it merely expresses the observed facts with considerable accuracy in a simple way, so that many very significant features emerge which would not otherwise do so. (Pantin 1932, p. 7760)

Like many others since his time, Pantin drew a distinction between a description of a phenomenon and an explanation for the phenomenon. Indeed, he was certainly aware that, from a purely mathematical perspective, no theory can ever emerge from a formula like $Y = bX^a$ because it sets no boundary conditions on the numerical values of a or b and because it makes no statement regarding the nature of Y and X . Indeed, the fractal relationship between the length of a coastline and the length of a stick used to measure it conforms to this equation. Logically, therefore, if there is nothing to predict, there can exist no theory to explain it. The allometric formula is nothing more than a mathematical statement about virtually any fractal relationship.

What is meant by a “theory” to explain size-dependent trends in biology is an explanation of the numerical values of the fractal-like *scaling exponents* that reappear when organisms differing in size are compared to one another using the formula $Y = bX^a$. Perhaps the most famous of these exponents is the $3/4$ scaling

exponent that describes the proportional relationship between basal metabolic rate B and total body mass M_T of vastly different animal and plant species (i.e., $B \propto M_T^{3/4}$). This proportional relationship is known as Kleiber's "rule" (Kleiber 1947). It has inspired many theoretical explanations (e.g., McMahon 1973; Blum 1977; Gray 1981). However, none is as far-reaching or as controversial as the theory proposed by G. West, J. Brown, and B. Enquist (West et al. 1997, 1999; see also Savage et al. 2004). West, Brown, and Enquist (henceforth WBE) argue that all biological scaling relationships are governed by $1/4$ (or multiples of $1/4$) power laws because all organisms, even unicellular ones, have internal fractal-like energy/mass delivery networks that have evolved by natural selection to minimize the energy and time required to absorb, distribute, and deliver resources internally. Although criticized on empirical and theoretical grounds, and challenged by alternative conceptual approaches (e.g., Banavar et al. 1999; Dodds et al. 2001; Darveau et al. 2002; Weibel 2002; Solow 2005; Kolokotronis et al. 2010), the WBE theory currently remains the most comprehensive allometric "theory," so much so that in the context of macroecology it can be called "the theory for everything."

Unfortunately, the basic assumptions upon which the WBE theory rests have not been tested directly. Support for this theory has thus far come exclusively from the concordance between the predicted and observed numerical values of scaling exponents and from computer simulations showing that an idealized WBE plant, i.e., a hypothetical plant that manifests all predicted $1/4$ (or multiple $1/4$) scaling relationships rapidly outcompetes all other simulated plants, even those that deviate in only one scaling exponent (Hammond and Niklas 2012).² However, any theory that stipulates $1/4$ (or multiples of $1/4$) power rules will necessarily agree with empirical observations, regardless of whether the basic assumptions of the theory are correct or false (Niklas 1994, 2004). The only real test of a theory is whether its fundamental postulates are shown to be correct. To date, no such test has been devised or implemented for the WBE theory.

2.5 Can Allometry Answer Size-Shape-Development Questions?

In the absence of a *bona fide* allometric theory, can allometric analyses still be useful in answering some of the questions raised during the 1981 Dahlem conference? I believe that they can. Here, I set up an experimental design with three components to illustrate how allometric analyses can be used to assess the prevalence of adaptive versus contingent evolution.

² A core assumption of the WBE theory is that $1/4$ scaling exponents result from natural selection operating to maximize the supply of nutrients and minimize the time of delivery. If true, an idealized WBE plant ought to outcompete all 'non-optimal' plants.

The first component of this demonstration is a data set for the dry biomass of the leaves, stems, and roots of seed plants (angiosperms and conifers) that either lack or possess very little secondary growth (data from Niklas and Enquist 2001, 2002; Niklas 1994, 2004, 2005).³ Across a broad spectrum of these species, the scaling exponents governing the size-dependent relationship among the dry mass of leaves, stems, and roots (denoted by M_L , M_S , and M_R , respectively) are statistically indistinguishable from one (i.e., $M_L \propto M_S \propto M_R$). It therefore follows that the mass of all above ground body parts (i.e., $M_A = M_L + M_S$) scales one-to-one (isometrically), or nearly so, with respect to below ground biomass (i.e., $M_A \propto M_B = M_R$) (Niklas 2004, 2005). Inserting the requisite allometric constants (with successive numerical subscripts) into these proportionalities gives

$$M_L = b_0 M_S = b_1 M_R \quad (2.1)$$

$$M_A = M_L + M_S = (1 + 1/b_0) b_1 M_B = R \quad (2.2)$$

The second component of this demonstration is a data set for the dry mass of the structural analogues of leaves, stems, and roots among non-seed plants including green and brown algae, mosses, and representatives of every extant seedless vascular plant lineage (Table 2.1). The juxtaposition of these data with those from seed plants provides an opportunity to determine whether a single partitioning pattern for biomass holds true across all plant lineages.

The third component of my demonstration is the argument that, if a single “canonical” biomass partitioning pattern exists across all polyphyletic plant lineages, it provides evidence for adaptive (functional) equivalence and detracts from the hypothesis that developmental constraints are responsible (Harvey and Pagel 1991; Niklas 1994). The “developmental constraint” hypothesis posits that natural selection acts on different body parts in opposing directions (gauged by biomass or some linear dimension), and that developmental covariance among these parts limits the extent to which a body plan can change evolutionarily. The “functional equivalence” hypothesis argues that particular body parts must change in size with respect to changes in the size of other body parts to maintain comparable functional levels of performance dictated by biophysical or physiological (invariant) “rules.”

These two hypotheses are not mutually exclusive in all respects. Natural selection operating on how organs perform certain biological tasks can act indirectly on the developmental patterns that give rise to organ structure, shape, size, etc. Put differently, if function-function covariants are the objects of selection, the developmental variations that give rise to them will also be the objects of selection. Certainly, organisms can neither obviate the laws of physics and chemistry nor the principles of engineering and mathematics. Thus, constraints on development have existed since the dawn of life.

³ Dry biomass is used rather than fresh mass because the water content of plant tissues can change diurnally dramatically and because water content tells us little about the actual biomass or metabolic “investment” involved in the construction of a plant.

Table 2.1 Species-groupings used to evaluate biomass partitioning patterns. For taxonomic relationships among these taxa, see Bold (1967), Bierhorst (1971), Bremer et al. (1987), Lewis and McCourt (2004)

Algae (polyphyletic)	
<i>Brown algae (Laminariales)</i>	
	<i>Alaria</i> sp.
	<i>Costaria costata</i>
	<i>Laminaria agardhii</i>
	<i>Nereocystis luetkeana</i>
	<i>Postelsia palmaeformis</i>
	<i>Pterygophora californica</i>
	<i>Saccorhiza dermatodea</i>
Sihonous (unicellular) chlorophycean algae	
	<i>Culerpa prolifera</i>
Charophcean algae (sister group to the land plants)	
	<i>Chara contraria</i>
	<i>Nitella flexilis</i>
Land plants (monophyletic embryophytes)	
Non-vascular plants (mosses)	
	<i>Dawsonia superba</i>
	<i>Funaria hygrometrica</i>
	<i>F. flavicans</i>
	<i>Polytrichum commune</i>
Vascular, seedless plants (pteridophytes)	
“Microphyllous”	
	<i>Equisetum arvense</i> (a horsetail)
	<i>Hupertizia lucidium</i> (a lycopod)
	<i>Selaginella krausianna</i> (a lycopod)
“Megaphyllous”	
	<i>Botrychium virginianum</i>
	<i>Marsilea quadrifolia</i>
	<i>Polytrichum acrosticoides</i>
	<i>Psilotum nudum</i>
	<i>Regnellidium diphyllum</i>
	<i>Salvina natans</i>

However, the concepts of “developmental constraints” *sensu stricto* and “constraints on development” are different; the first posits that developmental repertoires are “internally” regulated and limited genomically, whereas the second argues that the external environment limits which among possible developmental patterns persist in evolutionary time (Amundson 1994). Therefore, the “developmental constraint” hypothesis *sensu stricto* can be rejected if the biomass partitioning patterns observed for phyletically unrelated lineages are statistically and allometrically concordant, because it is known that the plants in my two data sets have vastly different developmental choreographies. Indeed, the brown algae (here limited to members of the Laminariales) and the chlorophycean algae provide critical tests. All of the evidence indicates that the ontogeny of brown algae is

radically different from that of vascular plants, and that the chlorophycean algae represent a clade distinct from the green algal lineage most closely related to the land plants, i.e., the charophycean algae (see Bremer et al. 1987; Gifford and Foster 1988; Graham and Wilcox 2000).

Therefore, the allometric trends of the seed plants can be used as a null hypothesis and the allometric trends of the brown and the chlorophycean algae serve as the phyletic “out-groups” to test whether “developmental constraint” or “functional (adaptive) equivalence” is predominant. The tactic is to (1) quantify the biomass partitioning pattern for all paired variables of interest across ecologically diverse seed plants, (2) superimpose the same kinds of data obtained from the other plant lineages, and (3) determine which (if any) taxa emerge as statistical outliers with respect to the allometry of seed plants (i.e., data that fall outside the 95 % confidence intervals for the RMA regression estimates of seed plant allometry). Three paired (and biologically interdependent) variables of interest are identified by the null hypothesis. These are M_L vs. M_S , M_L vs. M_R , and M_S vs. M_R (Niklas 2000).

My statistical test is intentionally conservative for three reasons: (1) the 95 % confidence intervals of regression curves tend to contract as both the sample size and correlation coefficient increase across different data sets; (2) the seed plant data set is especially robust in both respects; and, (3) the 95 % confidence intervals are thus comparatively easily trespassed by other plant life-forms, i.e., “outliers,” are not only readily apparent, they are “encouraged.”

2.6 Analyses of the Data Sets

Across all possible comparisons, the biomass partitioning patterns of the different plant lineages are isometric and statistically indistinguishable as gauged by their scaling exponents (Table 2.2). Likewise, bivariate plots of all of the paired variables of interest reveal few if any brown algal outliers, and, those that do occur are outnumbered by seed plant outliers (Fig. 2.1). For example, five outliers are observed for the scaling of M_L with respect to M_S . However, three are a fern (*Botrychium virginianum*), a lycopod (*Hupertzia lucidulum*) and a horsetail (*Equisetum arvense*) and only two are brown algae (Fig. 2.1). Like all extant horsetails and the majority of lycopods, *E. arvense* and *H. lucidulum* have small “microphyllous” leaves; those of *E. arvense* are vestigial and non-photosynthetic. Thus, a single, interspecific partitioning pattern for biomass appears to exist and lends support for convergent (adaptive) evolution among the various plant lineages.

The existence of natural functional organ-categories resonates reasonably well with the functional obligations that have been traditionally ascribed to each of the analogous body parts assigned to one of the three functional organ-categories. The foliose structures of marine green, red, or brown algal macrophytes intercept sunlight and exchange gasses with the fluid that surrounds them in physical and chemical ways that are not fundamentally dissimilar from those influencing the exchange of mass and energy between the air and the foliage leaves of

Table 2.2 Summary statistics of reduced major axis (RMA) regression of $\log_{10}$ -transformed data for seed plant leaf, stem and root dry mass (original units in kg; denoted by M_L , M_S , M_R , respectively), above- and below-ground body parts (denoted by M_A and $M_B = M_R$, respectively), and data for analogous body parts of seedless vascular plants and charophycean algae and brown algae (see Table 2.1 for species listing; for analyses, see Niklas 2000)

	a (95 % CIs)	$\log b$ (95 % CIs)	r^2	n
Seed plants				
$\log M_L$ vs $\log M_S$	0.90 (0.88, 0.92)	-0.19 (-0.25, -0.13)	0.91	862
$\log M_L$ vs $\log M_R$	0.93 (0.91, 0.96)	-0.03 (-0.11, -0.06)	0.87	668
$\log M_S$ vs $\log M_R$	1.01 (0.98, 1.04)	0.10 (-0.01, 0.20)	0.87	673
$\log M_A$ vs $\log M_B$	1.04 (1.02, 1.06)	0.38 (0.31, 0.45)	0.88	1,223
Seedless vascular plants and charophycean algae				
$\log M_L$ vs $\log M_S$	0.91 (0.70, 1.13)	-0.05 (-0.83, 0.73)	0.86	16
$\log M_L$ vs $\log M_R$	0.78 (0.51, 1.06)	-0.66 (-1.58, 0.25)	0.84	16
$\log M_S$ vs $\log M_R$	0.96 (0.68, 1.24)	-0.26 (-1.22, 0.70)	0.89	16
$\log M_A$ vs $\log M_B$	0.85 (0.64, 1.07)	-0.31 (-1.05, 0.43)	0.91	20
Brown algae (laminariales)				
$\log M_L$ vs $\log M_S$	0.83 (0.47, 1.20)	0.20 (-0.65, 1.06)	0.92	8
$\log M_L$ vs $\log M_R$	0.87 (0.58, 1.16)	0.52 (-0.22, 1.26)	0.95	8
$\log M_S$ vs $\log M_R$	1.04 (0.57, 1.51)	0.38 (-0.83, 1.58)	0.91	8
$\log M_A$ vs $\log M_B$	0.89 (0.80, 1.03)	0.59 (0.32, 0.86)	0.99	8

tracheophytes. Likewise, just as roots and holdfasts provide anchorage to a substrate, the mechanical functionality of the aerial stems of vascular plants, the stem-like axes of mosses, and the stipes of many brown algae is nearly identical from an engineering perspective, despite differences in the hydraulic (and thus anatomical) obligations of these otherwise very different structures.

That the size-dependent (scaling) requirements of developmentally different structures (which appear to share the same or very similar functional obligations) are not truly “invariant” is evident from the inspection of the absolute rather than the proportional biomass relationships among leaves, stems, root, and their corresponding analogs. Note that the scaling exponent describes the proportional relationship between two variables, whereas the allometric constant defines the absolute values of the variable of interest. Table 2.2 shows that these constants can have numerically broad 95 % confidence intervals, which indicates that some species groupings occupy a wide range of body part sizes.

No theory yet explains how or why allometric constants vary across species or lineages. However, the numerical “latitude” of these “constants” indicates *a posteriori* the “permissible variation” in the range of biomass occupied by each functional organ-category, which is otherwise confined by the operation of physical and chemical laws or processes. Metaphorically speaking, the data-scatter observed for all of the biomass scaling relationships reflect the size corridors through which plants have evolved as their size range expanded or contracted over evolutionary history. That these corridors have well defined limits attests to the operation

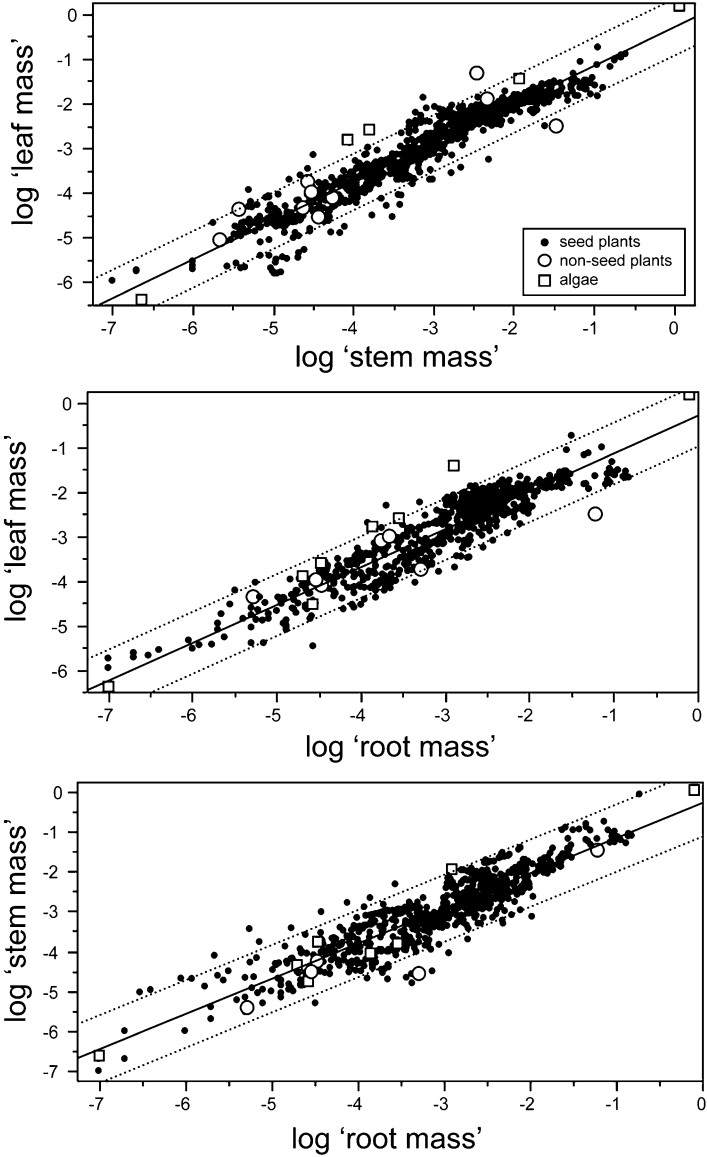


Fig. 2.1 Bivariate plots of $\log_{10}$ -transformed data for the dry mass of the leaves, stems, and roots of seed plants and the dry mass of the structural equivalents of these organ types in other species-groupings (see Table 2.1). *Solid and dashed lines are the respective reduced major axis regression curve and its 95 % CIs for the seed plant data*

of physico-chemical laws and processes, the objects of biophysical enquiry. However, it is also clear that some taxa “pushed these limits” (and are thus statistical outliers), which illustrates life’s remarkable ability to generate innovative solutions to biotic and abiotic challenges.

2.7 Developmental Networks

The 1981 Dahlem conference participants discussed developmental constraints at great length and how molecular biology could unravel the relationship between development and evolution. This optimism permeates much of the current literature, the majority of which is devoted to identifying the genes and gene networks underlying targeted aspects of morphogenesis and development. Although this approach is technologically very sophisticated, it currently has little or no explanatory power (other than that gene networks exist). I want to explore this aspect further by showing that our understanding of genetic networks is always incomplete unless the entire gene-network is mapped as an integrated system.

A synoptic treatment of Boolean logic circuits is beyond my scope (for classic references, see Halmos 1963; Harrison 1965; in terms of plant development, see Stein 1998). However, there are only two basic kinds: combinatorial circuits, in which the output signal depends exclusively on the near instantaneous value of the input signal, and sequential circuits, in which the output signal also depends on the history of previous inputs. Both types depict a signaling pathway as an electrical circuit containing one or more switches. The “logic” of a circuit is the algorithm that describes the conditions (logical propositions) dictating whether a signal passes through a circuit. Parallel and serial circuits exist. The former provides multiple responses to the same signal depending on instantaneous conditions, because parallel circuits allow an initial input signal to flow through two or more pathways, permitting two or more output signals at each terminus. Generally, the number of responses is given by 2^N , where N is the number of switches. Responses coordinated by parallel logic circuits can achieve seemingly continuous variation in response to the passage of a single input signal if they contain even a modest number of switches (e.g., $2^{N=10} = 1,024$), if some switches activate or suppress other switches in the circuit, if the circuit has two or more input signals, or if the output signals interact combinatorially. Also, if switches respond to more than one signal, the number of possible output signals is $S = 2^N$, where S is the number of input signals to which switches respond. Note that when $N = 5$, $S = 4.29 \times 10^9$.

Although a complex logic circuit can be simplified mathematically, four caveats are evident when biological systems are approached in this way (Niklas 2003): (1) there is no *a priori* method to determine which among logically equivalent circuits is biologically real, i.e., oversimplification can produce false circuit diagrams; (2) incomplete signaling pathways may appear to ‘work’ when diagrammed, i.e., missing components are not invariably obvious; (3) parallel logic circuits may obtain invariant output signals that give the appearance that input signals pass through serial switches, i.e., a bifurcating signal transduction pathway is more readily misdiagnosed than a serial pathway; and, (4) nothing in a logic circuit per se indicates when and how long a switch is turned on or off or how long a genomic or metabolic product lasts, i.e., the temporal components of signaling can be lost.

To be useful, logic circuits must be wedded to the subsystems they supervise. There are a variety of signal-activated subsystem configurations. The simplest is

error-activated, i.e., the output signal is used to modulate the input signal. This configuration has four essential components (see Harrison 1965; Hill and Peterson 1968): (1) a comparator to measure the difference (error) between the actual and the desired output of the subsystem; (2) an actuator/suppressor to convert the error-signal into an internal signal; (3) the actual machinery or assemblage that is controlled (the subsystem assembly); and, (4) a feedback element to direct the immediate output signal of the assemblage back to the comparator (Fig. 2.2a).

Feedback is defined as the property of a closed-loop system that permits the comparison of the output signal (or some other variable controlled by the subsystem) to the input of the subsystem (or an input to some other internal component) so that the control action is some function of the input-to-output ratio. In many ways, the feedback element is the most important of the four components because it confers four characteristics: (1) an increased range of input signals over which the subsystem responds satisfactorily; (2) reduced sensitivity to variations in the output to input signal ratio; (3) reduced effects of nonlinear distortions; and, (4) a tendency toward initial oscillatory behavior. Negative and positive feedback loops exist and a single loop can serve in both capacities, especially in the case of a subsystem hot-wired by a sequential (history-dependent) logic circuit (for an interesting example, see Bhalla et al. 2002).

By definition, subsystems are networked to other subsystems by shared circuits. This feature reduces the erratic behavior of the system as a whole. The linkage of two or more subsystems results in two important properties: (1) the ability to achieve global stability; and, (2) the feedback signaling of numerous components (a phenomenon called “recursive combinatorial regulation”). Global behavior confers homeostasis, whereas recursive combinatorial regulation permits a network to repeatedly cycle through a programmed series of transformations.

2.8 The Incompleteness Theorem

No single actuator/suppressor switch exists in isolation because each subsystem circuit requires an activation or suppression signal. One subsystem must receive a signal and temporarily function as an epistatic actuator. When switched on or off, this subsystem sparks the operation of the entire network, suppressing or activating one or more of the networked subsystems. However, once the entire system is set into operation, no “master” switch exists. If a developmental master switch ever existed, it was turned on when the first living cell evolved during the Precambrian.

This feature governs the working of *any* networked system and instantiates what I called the biological “incompleteness theorem,” i.e., the operation of *any* biological subsystem cannot be fully diagnosed in isolation of the operations of the other subsystems to which it is networked (Niklas 2003). This theorem, which is an analog to Kurt Gödel’s (1931) incompleteness theorems, can be proved mathematically, but it is easily illustrated by a hypothetical example consisting of two nuclear genes (*G1*, *G2*), their enhancer-promoters (EP1, EP2), a cell membrane docking

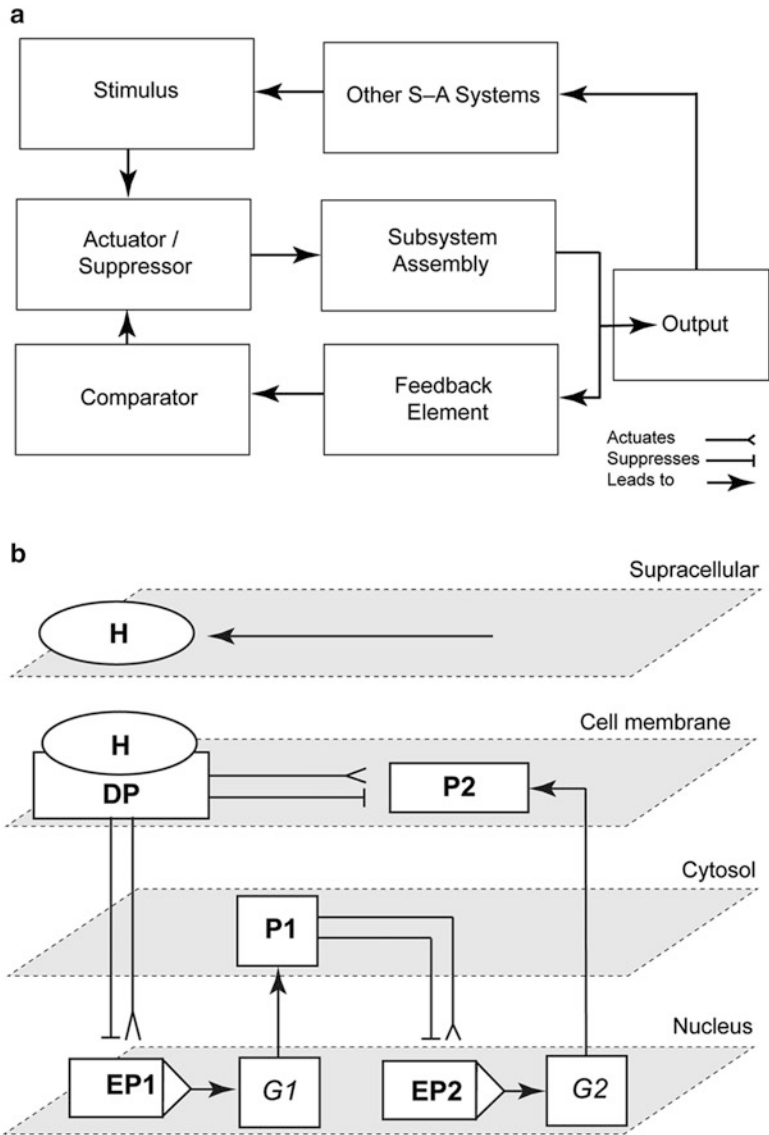


Fig. 2.2 Schematics of a simple, signal-activated subsystem and the “incompleteness theorem” (Adapted from Niklas 2003). (a) A signal-activated (S-A) subsystem with an actuator/suppressor, a subsystem assembly, a feedback element, and a comparator. The output signal (response to an input stimulus) feeds into other signal-activated (S-A) systems. (b) Schematic of the “Incompleteness theorem” for a circuit/subsystem consisting of a docking protein *DP* activated by a hormone *H* that mediates signalling to a cell membrane-bound protein *P2*, a cytoplasmic protein *P1*, and the enhancers/promoters (EP) of two genes *G1*–*2*. The network is structurally self-contained, but its operation depends on the delivery of the external signal (*H*)

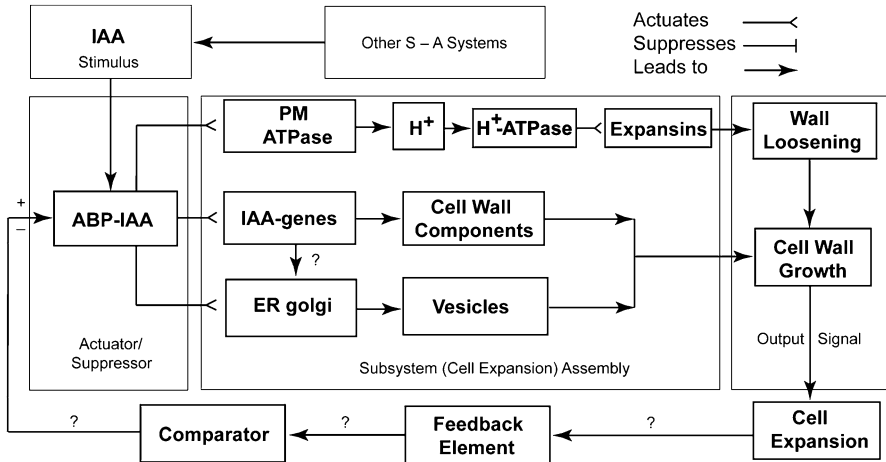


Fig. 2.3 Circuit/subsystem diagram for cell expansion mediated by IAA and the IAA binding protein ABP1. *PM* plasma membrane, *ER* endoplasmic reticulum, *CWC* cell wall components. See text for additional details

protein (DP) activated or suppressed by a hormone (H), and two regulatory proteins (P1, P2) (Fig. 2.2b). At the *structural* level, this network is a self-contained system regardless of the presence of H. However, in terms of its *operation*, this network is suppressed or activated by H, which is delivered from an *external* source that is regulated by one or more other network systems.

Consider now the logic circuit for the regulation of cell wall loosening as mediated by indole-3-acetic acid (IAA), wherein the plasmalemma-bound auxin binding protein 1–IAA conjugate (ABP1-IAA) is diagrammed as the actuator/suppressor switch for ATPases (Fig. 2.3). Once activated, the cell wall is acidified, cell wall bonds are broken (expansin proteins have been implicated in this process), and turgor pressure drives cell expansion (not shown). The ABP1-IAA switch also triggers delayed cytoplasmic and genomic responses involving the synthesis and delivery of cell wall components. This logic circuit diagram shows that sustained osmoregulation and cell wall loosening are required for continued cell expansion. The diagram also shows that the feedback loop and comparator for the output signal of the cell expansion machinery are unknown and must be sought experimentally. The IAA degradation, the down-regulation of solute concentrations, the synthesis of new cell-wall-binding polymers, the reorientation of cellulose microfibrils, the deposition of secondary wall layers, and the degradation of wall-loosening enzymes are among the many viable candidates for these missing network components. However, it is clear that cell wall loosening involves numerous other suppressor/actuator subsystems, many of which remain poorly understood, and largely ignored despite claims that this developmental system is fully diagnosed (e.g., Liepman et al. 2010).

The logic circuit for the regulation of cell wall loosening reveals the extent to which our understanding of an important and intensively studied developmental

subsystem is limited. It also illustrates the extent to which this developmental “subroutine” must be integrated with numerous other developmental subsystems (such as those regulating polar auxin transport and osmoregulation) before its role in plant development is completely comprehended. No subsystem functions in isolation. Each is integrated with the operation of all other developmental systems. Consequently, regardless of how well a subsystem is dissected and manipulated experimentally, its role will always be incompletely understood if isolated from an organism’s entire spatiotemporal developmental repertoire. This ‘incompleteness theorem’ presents a daunting intellectual challenge, given the extraordinary complexity of the developmental biology of even so seemingly simple things as unicellular plants, animals, and fungi. Nevertheless, it provides a perspective that resonates with many of the participants of the 1981 Dahlem conference.

2.9 Plato’s Cave and the 1981 Dahlem Conference

The 1981 Dahlem conference participants pondered the usefulness of network theory as well as biophysical and allometric tools to identify constraints on development. In general, they felt the available data were inadequate to resolve this question and called for a broader, more comprehensive *Weltanschauung*, a quest that is still justified. I have tried to illustrate how tools such as allometric analyses, biomechanics, and network theory can be brought into service to answer some of the important questions raised during the 1981 conference. I have also tried to expose some of the intrinsic limitations of these tools. In the context of allometric analyses, the numerical values of the scaling exponents used to describe any allometric trend depend on the phylogenetic composition of the data and these values will progressively differ numerically among data sets as the sample sizes of these data sets decrease (see Table 2.2). In the context of network theory, the piecemeal dissection of any system, even one as comparatively simple as the expansion of the plant cell wall, can lead to completely erroneous interpretations of experimental results (see Fig. 2.3).

These and other concerns draw attention to one interpretation of Plato’s allegory of the cave, i.e., we acquire concepts by our perceptual experiences of physical objects, but we are deluded if we believe that these concepts are truly on the same level of reality as the things we perceive. This allegory is particularly important when biology is reduced to molecular biology, physics, mathematics, or statistical inference. For example, even if we assume that a one-to-one pattern of biomass partitioning is not a statistical artifact, can we really evaluate whether it is biologically important? Mathematically, isometry is trivial in the sense that a one-to-one proportionality maintained across any series of objects differing in size requires no special biophysical “rules” (or metabolic “effort”). Yet, the state of being a “trivial condition” in light of solid mechanics or physiology does not preclude the possibility that a phenomenon is biologically non-trivial. Indeed, “simple” often translates into “elegant.”

Likewise, can we say that a developmental system is “understood” once the genes that drive it are identified even when their gene products remain unknown? Indole-2-acetic acid (IAA) is known to alter gene expression in very rapid and selective ways. This explains why IAA signaling is linked to a large number of physiological and developmental responses, including gravitropism, cell wall extension, embryo axis polarity, and vascular tissue differentiation. But not one of the products of these genes has been identified and even a very general description of their downstream effects on plant development remains frustratingly elusive.

In many ways, we are at the same stage as classical genetics was before the Modern Synthesis when it was difficult to reconcile Mendelian quantitative genetics with the Darwinian supposition that evolution involves gradual phenotypic changes. The rapidly expanding information gained from biophysics, allometry, and genomics has not been integrated with the knowledge from more traditional physiological, developmental, or evolutionary disciplines. This situation will change, but only when we collectively consider all levels of biological organization simultaneously, from the molecular to the phenotype, as well as their evolutionary history. This challenge requires a new mindset. It will also require exploring taxa from deeper nodes in phylogenetic trees. The task is intimidating, but if left unaccomplished we run a risk described in T.S. Eliot’s *Four Quartets*:

*It seems, as one becomes older,
That the past has another pattern, and ceases to be a mere sequence—
Or even development: the latter a partial fallacy
Encouraged by superficial notions of evolution,
Which becomes, in the popular mind, a means of disowning the past.*

References

- Amundson, R. 1994. Two concepts of constraint: Adaptationism and the challenge from developmental biology. *Philosophy of Science* 61: 556–578.
- Banavar, J.R., A. Maritan, and A. Rinaldo. 1999. Size and form in efficient transportation networks. *Nature* 399: 130–132.
- Bhalla, U.S., P.T. Ram, and R. Iyengar. 2002. MAP kinase phosphatase as a locus of flexibility in a mitogen-activated protein kinase signaling network. *Science* 297: 1018–1023.
- Bierhorst, D.W. 1971. *Morphology of vascular plants*. New York: Macmillan.
- Bold, H.C. 1967. *Morphology of plants*. New York: Harper and Row.
- Bonner, J.T., and H.S. Horn. 1982. Selection for size, shape, and development timing. In *Evolution and development*, ed. J.T. Bonner, 259–277. Berlin: Springer.
- Blum, J.J. 1977. On the geometry of four-dimensions and the relationship between metabolism and body mass. *Journal of Theoretical Biology* 64: 599–601.
- Bremer, K., C.J. Humphries, B.D. Mishler, and S.P. Churchill. 1987. On cladistic relationships in green plants. *Taxon* 36: 339–349.
- Darveau, C.A., R. Suarez, R.D. Andrews, and P.W. Hochachka. 2002. Allometric cascade as a unifying principle of body mass effects on metabolism. *Nature* 417: 166–170.
- Dodds, P.S., D.H. Rothman, and J.S. Weitz. 2001. Re-examination of the “3/4-law” of metabolism. *Journal of Theoretical Biology* 209: 9–27.

- Gifford, E.M., and A.S. Foster. 1988. *Morphology and evolution of vascular plants*. New York: Freeman.
- Gödel, K. 1931. Über formal unentscheidbare Sätze der Principia Mathematica und verwandter Systeme. I. *Monatshefte Mathematik Physik* 38: 173–198.
- Gould, S.J. 1966. Allometry and size in ontogeny and phylogeny. *Biological Review* 41: 587–640.
- Gould, S.J. 1977. *Ontogeny and phylogeny*. Cambridge, MA: Harvard University Press.
- Gould, S.J. 1982. Change in developmental timing as a mechanism of macroevolution. In *Evolution and development*, ed. J.T. Bonner, 333–346. Berlin: Springer.
- Graham, L.E., and L.W. Wilcox. 2000. *Algae*. New Jersey: Prentice Hall.
- Gray, B.F. 1981. On the 'surface law' and basal metabolic rate. *Journal of Theoretical Biology* 93: 757–767.
- Halmos, P.R. 1963. *Lectures on Boolean algebras*. New York: Van Nostrand.
- Hammond, S.T., and K.J. Niklas. 2012. Computer simulations support a core prediction of a contentious plant model. *American Journal of Botany* 99: 508–516.
- Harrison, M.A. 1965. *Introduction to switching and automata theory*. New York: McGraw-Hill.
- Harvey, P.H. 1982. On rethinking allometry. *Journal of Theoretical Biology* 95: 37–41.
- Harvey, P.H., and M.D. Pagel. 1991. *The comparative method in evolutionary biology*. Oxford: Oxford University Press.
- Hill, F.J., and G.R. Peterson. 1968. *Introduction to switching theory and logical design*. New York: John Wiley.
- Horn, H.S., J.T. Bonner, W. Dohle, M.J. Katz, M.A.R. Koehl, H. Meinhardt, R.A. Raff, W.-E. Reif, S.C. Stearns, and R. Strathmann. 1982. Adaptive aspects of development, Group report. In *Evolution and development*, ed. J.T. Bonner, 215–235. Berlin: Springer.
- Huxley, J.S. 1932. *Problems of relative growth*. London: MacVeagh.
- Jolicoeur, P. 1990. Bivariate allometry: Interval estimation of the slope of the ordinary and standardized normal major axes and structural relationship. *Journal of Theoretical Biology* 144: 275–285.
- Kleiber, M. 1947. Body size and metabolic rate. *Physiological Reviews* 27: 511–541.
- Kolokotronis, T., E.J. Van Savage, and W. Fontana Deeds. 2010. Curvature in metabolic scaling. *Nature* 464: 753–756.
- Liepmann, A.H., R. Wightman, N. Geshi, S.R. Turner, and H.V. Scheller. 2010. Arabidopsis—a powerful model system for plant cell wall research. *The Plant Journal* 61: 1107–1121.
- Lewis, L.A., and R.M. McCourt. 2004. Green algae and the origin of land plants. *American Journal of Botany* 91: 1437–1445.
- McArdle, B.H. 1988. The structural relationship: Regression in biology. *Canadian Journal of Zoology* 66: 2329–2339.
- McGhee Jr., G.R. 1999. *Theoretical morphology*. New York: Columbia University Press.
- McMahon, T.A. 1973. Size and shape in biology. *Science* 179: 1201–1204.
- Niklas, K.J. 1992. *Plant biomechanics: An engineering approach to plant form and function*. Chicago: University of Chicago Press.
- Niklas, K.J. 1994. *Plant allometry: The scaling of form and process*. Chicago: University of Chicago Press.
- Niklas, K.J. 1997. *The evolutionary biology of plants*. Chicago: University of Chicago Press.
- Niklas, K.J. 2000. The evolution of plant body plans—a biomechanical perspective. *Annals of Botany* 85: 411–438.
- Niklas, K.J. 2003. The bio-logic and machinery of plant morphogenesis. *American Journal of Botany* 90: 515–525.
- Niklas, K.J. 2004. Plant allometry: Is there a grand unifying theory? *Biological Review* 79: 871–889.
- Niklas, K.J. 2005. Modelling below- and above-ground biomass for non-woody and woody plants. *Annals of Botany* 95: 315–321.

- Niklas, K.J., and B.J. Enquist. 2001. Invariant scaling relationships for interspecific plant biomass production rates and body size. *Proceedings of the National Academy of Sciences of the United States of America* 98: 2922–2927.
- Niklas, K.J., and B.J. Enquist. 2002. On the vegetative biomass partitioning of seed plant leaves, stems, and roots. *American Naturalist* 159: 482–497.
- Pantin, C.F.A. 1932. Form and size. *Nature* 129: 775–777.
- Prothero, J. 1986. Methodological aspects of scaling in biology. *Journal of Theoretical Biology* 118: 259–286.
- Ratner, J.M.V. 1985. Linear relations in biomechanics: the statistics of scaling functions. *Journal of Zoology* 206: 415–439.
- Raup, D.M. 1962. Computer as aid in describing form of gastropod shells. *Science* 138: 150–152.
- Riska, B. 1990. Regression models in evolutionary allometry. *American Naturalist* 138: 283–299.
- Savage, V.M., J.F. Gillooly, W.H. Woodruff, G.B. West, A.P. Allen, B.J. Enquist, and J.H. Brown. 2004. The predominance of quarter-power scaling in biology. *Functional Ecology* 18: 257–282.
- Seim, E. 1983. On rethinking allometry: Which regression model to use? *Journal of Theoretical Biology* 104: 161–168.
- Smith, R.J. 1980. Rethinking allometry. *Journal of Theoretical Biology* 87: 97–111.
- Solow, A.R. 2005. Power laws without complexity. *Ecology Letters* 8: 361–363.
- Stein, W.E. 1998. Developmental logic: Establishing a relationship between developmental process and phylogenetic pattern in primitive vascular plants. *Review of Palaeobotany and Palynology* 102: 15–42.
- Thompson, D'.A.W. 1942. *On growth and form*, 2nd ed. Cambridge: Cambridge University Press.
- Vogel, S. 1981. *Life in moving fluids*. Boston: Willard Grant Press.
- Weibel, E.R. 2002. Physiology—the pitfalls of power laws. *Nature* 417: 131–132.
- Wainwright, P.C., and S.M. Reilly (eds.). 1994. *Ecological morphology: Integrative organismal biology*. Chicago: University of Chicago Press.
- Wainwright, P.C., W.D. Biggs, J.D. Currey, and J.M. Gosline. 1976. *Mechanical design in organisms*. New York: John Wiley & Sons.
- West, G.B., J.H. Brown, and B.J. Enquist. 1997. A general model for the origin of allometric scaling laws in biology. *Science* 276: 122–126.
- West, G.B., J.H. Brown, and B.J. Enquist. 1999. The fourth dimension of life: Fractal geometry and allometric scaling of organisms. *Science* 284: 167–169.

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