TABLE 7.5 Biomasses of populations of selected herbivores living in mixed communities in African national parks

Species are listed in order of increasing individual size. These species were chosen for listing because they are statistically about average in population biomass for their body sizes.

Species	Average biomass of entire population per square kilometer (kg/km^2)	Average individual body weight (kg)
Oribi (*Ourebia ourebi*)	44	13
Gray duiker (*Sylvicapra grimmia*)	62	16
Gray rhebok (*Pelea capreolus*)	105	25
Warthog (*Phacochoerus aethiopicus*)	95	69
Waterbuck (*Kobus ellipsiprymnus*)	155	210
Greater kudu (*Tragelaphus strepsiceros*)	200	215
Plains zebra (*Equus burchelli*)	460	275
White rhino (*Ceratotherium simum*)	2400	1900
African elephant (*Loxodonta africana*)	1250	3900

Source: After Owen-Smith 1988.

of weight than do related larger animals; a practical consequence is that small-bodied species may require relatively high doses of a veterinary drug per unit of weight to achieve and sustain the drug's intended effect. Overall, the dynamics of accumulation and dissipation of foreign chemicals often differ between related large- and small-bodied species.

The explanation for allometric metabolism–size relations remains unknown

The fact that b, the allometric exponent, tends to be near 0.7 in widely diverse groups of animals is profoundly intriguing. For a century, some of the greatest minds in biology have grappled with the questions of *why* metabolic rate and body size are related allometrically and *why* the allometric exponent is sometimes impressively consistent. Great minds have been drawn to these questions because of a conviction that the allometries are manifestations of fundamental organizing principles of life. As yet, however, no consensus exists about how to explain the allometries.

Ninety years ago, the problem seemed solved! Physiologists thought then that they understood the reasons for not only the allometric metabolism–size relation, but also the particular value of b. The theory offered at that time has been reinvented by every generation of biologists because it seems so "obvious." Thus an understanding of the theory's flaws remains important even today. At the time the theory first appeared in the early twentieth century, all the data on metabolism–size relations were data gathered on mammals, and mammals therefore dominated thinking about the subject. During that period, Max Rubner articulated an explanatory theory that is still known as *Rubner's surface "law."*

Euclidean geometry provides the starting point for understanding this "law" that is not a law. Recall from your study of geometry that the surface area s of a sphere is proportional to the square of r, the sphere's radius: $s \propto r^2$. The volume v of a sphere, however, is proportional to the cube of the radius: $v \propto r^3$. From the rules of exponents, we can write $r^2 = (r^3)^{2/3}$. Thus $s \propto (r^3)^{2/3}$; and substituting v for r^3, we get

$$s \propto v^{2/3} \qquad (7.6)$$

In words, as spheres increase in size, their surface area increases only as the two-thirds power of their volume. Big spheres, therefore, have less surface area per unit of volume (or of weight) than little spheres. Similar relationships hold true for all sets of geometrically similar objects. Whether you consider cubes, cylinders, hearts, or whole animals, as the objects within a geometrically similar set become larger, the area of the outside surface is expected to increase approximately as the two-thirds power of volume, and the ratio of outside-surface area to volume declines.

Rubner's surface "law" stated that the BMR of a mammal is proportional to its body-surface area[14] and that the allometric relation between BMR and body weight is a corollary of this proportionality. Rubner's explanation of the allometric relation rested on four logical steps:

1. Placental mammals maintain high, relatively constant body temperatures (near 37°C) and thus tend to lose heat to the environment when studied at thermoneutral environmental temperatures.
2. Because heat is lost across an animal's outer body surfaces, the rate of heat loss from a mammal is approximately proportional to the animal's body-surface area.[15]
3. Small mammals have more surface area per unit of weight than do large mammals and thus lose heat more rapidly per unit of weight.
4. Heat lost must be replaced metabolically for a mammal to stay warm. Accordingly, small mammals must produce heat at a greater rate per unit of weight than large ones.

The surface "law" as just outlined can hardly be faulted as a thought exercise. Why, then, do most physiologists today believe that it is not the correct mechanistic explanation of the allometric relation between BMR and body weight? The answer is that data contradict the "law" in two respects. First, although the surface "law" predicts an exponent b equal to about 0.67 (2/3), most physiologists who have estimated values of b for mammals have concluded that b is statistically higher than 0.67 to a significant degree. Second, by now we realize, as emphasized already, that poikilothermic animals—such as fish, frogs, and crabs—display allometric metabolism–size relations (see Figures 7.9 and 7.10). Rubner's "law" cannot possibly explain these relations in

[14] A modern holdover of the early emphasis on body-surface area is that surface areas are employed to calculate certain sorts of critical variables in the contemporary practice of medicine. In breast cancer chemotherapy, for example, the doses of chemotherapeutic agents administered to a woman are calculated from her body-surface area.

[15] During the era when Rubner's surface law was accepted, this concept seemed too obvious to be questioned. In fact, it is not exactly true because of details in the physics of heat transfer.

poikilotherms because the reasoning behind the "law" applies only to animals that warm their bodies to elevated, regulated temperatures using metabolic heat production. The consistency of the metabolism–size relation across many animal groups suggests a single mechanistic explanation. Because Rubner's "law" is irrelevant for most types of animals (most are poikilotherems), it cannot be that explanation.

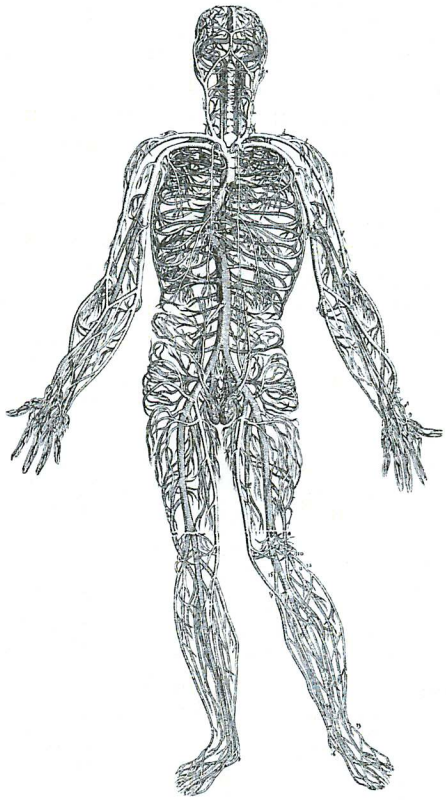
Since the time in the mid-twentieth century when the surface "law" started to be rejected by most physiologists, several alternative hypotheses have been put forward to explain allometric metabolism–size relations. Physiologists, however, have not reached a consensus in supporting any of the hypotheses. Until recently, debate tended to center on whether the true value of *b* is 2/3 or 3/4. Physiologists assumed that a single universal exponent existed and that, if it could be nailed down, the underlying mechanistic basis for allometry would be revealed. Why did difficulty exist in nailing down the exponent? For a long time, the amount of relevant data was not great, and everyone could assume, therefore, that when abundant data became available, the correct value for *b* would become obvious. What has actually happened is quite different. Now that great quantities of carefully scrutinized data are available, many specialists have concluded that in fact there is no universal exponent. With lots of high-quality data available, if researchers calculate two different values of *b* for two different animal groups, the difference cannot readily be dismissed as being merely a product of inadequate information.

Several research reports published in the past decade have concluded that although the exponent *b* generally tends to be *similar* in many animal groups, it is not *identical. The exponent* b *is not a constant,* according to these reports. Among placental mammals, for example, several meticulous efforts have concluded that *b* is different in some mammalian orders than in others. Also, as already noted, *b* is greater when mammals are exercising than when they are at rest. Moreover, *b* is higher when only large-bodied species are analyzed than when only small-bodied species are.[16]

As physiologists have searched for the mechanistic basis of metabolism–size relations, a key question has been, what attributes of animals are so *common* and so *fundamental* that they could explain the way in which metabolism varies with size? One attribute in particular has attracted a great deal of attention: internal transport. For metabolism to occur, internal transport of metabolic resources—notably O_2 and metabolic fuels—is critical. In mammals and many other types of animals, this transport is carried out by the circulatory system. Physiologists therefore realized that they had to understand how the circulatory system—first accurately described by Andreas Vesalius (1514–1564) in 1543 (**Figure 7.13*a***)—changes in its inherent capabilities for transport

[16] Accordingly, the log–log plot of metabolism–size data exhibits a bit of curvature and requires a more complex equation than Equation 7.3 to be described in detail.

(*a*) Vesalius 1543: One of the first anatomically accurate images of the human circulatory system



(*b*) Mandelbrot 1983: A fractal model of a branching system such as the circulatory system

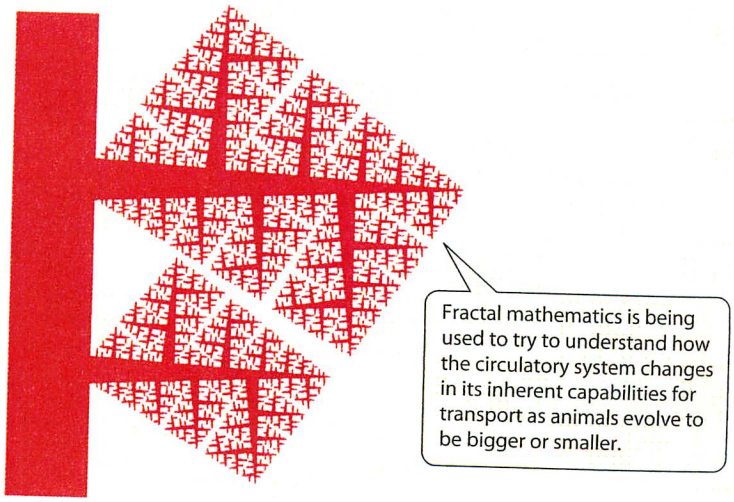


FIGURE 7.13 As the circulatory system is scaled up and down in size and extent, constraints predicated on fractal geometry may help give rise to allometric metabolic scaling A mammal's metabolism is dependent on distribution of required resources to tissues throughout the body. When Andreas Vesalius first described the circulatory system (*a*), its function was a mystery. Oxygen had not yet been discovered, and the fact that the circulatory system delivers O_2 to all tissues was inconceivable. In the years since, as many old questions were answered, new questions came to the fore. One modern question is this: Given that mammals all have a circulatory system built on similar principles—and yet the dimensions of that system have had to be scaled dramatically up and down as big and small species have evolved—what geometric and functional constraints might have been encountered? A fractal geometric approach to this question had to await Benoit Mandelbrot's invention of fractal mathematics around 1980. Fractal systems, as seen in (*b*), are "self-similar" at multiple scales, meaning that the patterns of branching of fine elements are miniatures of the patterns of branching of large elements. (*a* from *De Humani Corporis Fabrica*, produced by Andreas Vesalius in 1543, as reproduced in Saunders and O'Malley 1950; *b* after Mandelbrot 1983.)

as animals evolve to be bigger or smaller. The new mathematics of *fractal geometry*—invented more than 400 years later to describe the properties of branching systems (Figure 7.13*b*)—was marshaled to analyze this question. From this fractal research, a hypothesis was propounded, that allometric metabolism–size relations occur in part because of geometrically imposed constraints. This hypothesis stresses that in fractally structured transport systems, *rates* of transport—and thus rates of supply of resources required for metabolism—are geometrically constrained in distinctive ways as body size is scaled up or scaled down over the course of evolution. Computer models have been used to examine how the constraints of fractal geometry interact with evolutionary selection pressures to maintain optimized transport capabilities as animals evolve different body sizes. These models initially predicted a universal allometric exponent of 3/4—a discovery followed by an almost giddy application of that exponent to new branches of biology even though it is a product of theory and often not supported by empirical data. Now most physiologists acknowledge that b probably does not exhibit a single fixed value. Nonetheless, one of the major themes in ongoing research is to revisit the analysis of circulatory systems and other transport systems responsible for distributing metabolic resources in the body. Those systems may well play roles in constraining how metabolic rate can vary with body size. Several other fascinating hypotheses are also being investigated at present.

SUMMARY Metabolic Scaling: The Relation between Metabolic Rate and Body Size

- BMR, SMR, and other measures of resting metabolic rate are allometric functions of body weight within phylogenetically related groups of animals ($M = aW^b$, where b is usually in the vicinity of 0.7). Small-bodied species tend to have higher weight-specific metabolic rates than do related large-bodied species, an effect so great that the weight-specific BMR is 20 times higher in mice than in elephants.
- Maximal aerobic metabolic rate also tends to be an allometric function of body weight in sets of related species. In many cases studied thus far, the allometric exponent for maximal metabolic rate differs from that for resting metabolic rate.
- The allometric relation between metabolic rate and weight exerts important effects on the organization and structure of both individual animals and ecosystems. Heart rates, breathing rates, mitochondrial densities, and dozens of other features of individual animals are allometric functions of body weight within sets of phylogenetically related species. In ecosystems, population biomasses and other features of community organization may vary allometrically with individual body size.
- Physiologists are not agreed on the explanation for the allometric relations between metabolic rate and body weight. Rubner's surface "law," based on heat loss from homeothermic animals, does not provide a satisfactory explanation. Many of the newest hypotheses recognize that the allometric exponent varies in systematic ways and seek to explain that variation, as by examining evolutionary constraints in resource distribution systems such as the circulatory system.

Energetics of Food and Growth

Food and growth are important topics in animal energetics, aptly discussed together because one animal's growth is another's food. A consequential attribute of foods as energy sources is that lipids are at least twice as high in energy density—energy value per unit of weight (see Table 6.3)—as proteins and carbohydrates are. We asked at the start of this chapter why polar explorers carry lipid-rich foods, such as meat mixed with pure lard. If they are going to pull, push, and lift their food for many miles before they eat it, the explorers should choose food that provides a lot of energy per kilogram transported. Similarly, migrating animals often capitalize on the high energy density of lipids by carrying their fuel as body fat.

A key question about any food in relation to an animal's physiology is how efficiently the animal can digest (or ferment) the food and absorb the products of digestion. The **energy absorption efficiency** is defined to be the fraction of ingested energy that is absorbed for use:[17]

$$\text{Energy absorption efficiency} = \frac{\text{absorbed energy}}{\text{ingested energy}} \quad (7.7)$$

This efficiency matters because the absorbed energy is the energy actually available to an animal for use in its metabolism. To illustrate the importance of absorption efficiency, consider the processing of ingested cellulose by humans and ruminants. Because people cannot digest cellulose, they cannot absorb it, and their absorption efficiency for cellulose is essentially 0%; if they eat only cellulose, they starve. Ruminants such as cows, in contrast, commonly achieve about 50% absorption efficiency for cellulose because their rumen microbes ferment cellulose into compounds that the animals can absorb; thus ruminants are able to use about half of the energy available from cellulose in their metabolism. This example illustrates how the physiology of digestion and absorption, discussed in Chapter 6, bears on the physiology of energy.

Growing animals accumulate chemical-bond energy in their bodies by adding tissue consisting of organic molecules. An important question in many contexts is how efficiently they are able to use their available food energy to build tissue. Two types of growth efficiency, termed **gross growth efficiency** and **net growth efficiency**, are defined on the basis of whether the food energy is expressed as the ingested energy or the absorbed energy:[18]

$$\text{Gross growth efficiency} = \frac{\text{chemical-bond energy of tissue added in net fashion by growth}}{\text{ingested energy}} \quad (7.8)$$

$$\text{Net growth efficiency} = \frac{\text{chemical-bond energy of tissue added in net fashion by growth}}{\text{absorbed energy}} \quad (7.9)$$

[17] See Figure 7.2. Recall that the energetic efficiency of a process is the output of high-grade energy expressed as a ratio of input (see Equation 7.1). When digestion, fermentation, and absorption are the functions of interest, the output of high-grade energy is the absorbed energy, whereas the input is the ingested energy.

[18] Referring to Equation 7.1, when growth is the energy input–output process of interest, the output of high-grade energy is the chemical-bond energy of added tissue, whereas the input is food energy.