

process, and it is the unusual loads that are most significant, but this important question has not been subjected to much research.

Organisms that live in water inhabit a mechanical environment which is seemingly more predictable than the terrestrial one, and so should be able to mechanically 'fine-tune' (a teleological term!) their skeletons better than land dwellers. But the buoyancy of water works against the adaptive advantage of a lighter skeleton; that a heavy skeleton balances low-density fat, resulting in an organism with an average density close to that of water, thereby facilitating aquatic locomotion. Conversely, birds (that is, flying birds, not terrestrial birds like chickens and ostriches) find exceptional adaptive value in a minimal-weight skeleton, which improves the efficiency of flight. Other factors contribute to the skeleton's mechanical milieu, as well, including pregnancy and other seasonal fluctuations in body weight, antler growth, hibernation, migration, and fighting among males. These factors must complicate the optimization of skeletal structure.

This chapter is devoted to discussing the proposition that the skeleton contains adaptive mechanisms that optimize its structure in some sense. It would be a mistake, however, to hypothesize that evolution is directed toward maximizing efficiency of structures. Evolution is directed toward maximizing genetic representation in subsequent generations, which in turn is associated with efficiency, but only rather loosely. Indeed, the physiques of many ancient relict species contain features that are hardly paragons of efficiency. Consider the antlers of a moose. They are heavy and cumbersome, and get caught in branches, and require great amounts of calcium and phosphorus to construct. With respect to locomotion or food gathering, their contribution to efficiency is negative; yet they are favored by evolution, apparently because the moose with biggest antlers is the most likely to be genetically represented in the next generation.

## 6.7 Mechanical Adaptability and Evolutionary Adaptability

There has been considerable debate among engineers, biologists, and clinicians concerning the balance between genetics and mechanical adaptability in deciding the structures of bones. Some have argued that the anatomy and histology of a bone such as the human femur are determined primarily by genetic information expressed by various cells as they form the skeleton and maintain it in the adult. Others have come to believe that mechanical factors strongly influence the expression of the genetic plan carried by the cells. In closing this chapter, it is appropriate to consider the relationships between mechanical adaptability and evolutionary change as determinants of skeletal structure. This discussion is based on the ideas of Bateson (1963, reprinted in Bateson, 1987).

### *Somatic Vs. Evolutionary Adaptation*

The adaptation of bone to alterations in mechanical loading is an example of somatic change, that is, a change in body form or function that does not

entail a genetic change and is therefore **not passed on** to succeeding generations. Somatic change is distinct from **evolutionary change**, which does involve a modification of the genome of the organism. A simple example of somatic change is obtaining a sun tan during a beach vacation; obviously, this does not alter the genetic code for skin color, and the tan is not passed on to the vacationer's children. Nevertheless, somatic change plays an important role in evolution.

Bateson pointed out that the ability to adjust to environmental changes by somatic modification of body systems is essential to survival because evolutionary change operates relatively slowly, through chance mutations. In addition, somatic adjustments allow the organism to survive when a genetic mutation, which might ultimately have survival value, disrupts one or more body systems. Conversely, genetic adjustments to the somatic system itself allow the homeostatic range to be restored when a mutation has driven it to an extreme. Thus, evolution involves constant interaction between slow, haphazard genetic changes and rapid, predictable somatic changes. In short, somatic flexibility allows time for adaptive modifications of the genome to occur, and also helps the organism tolerate their adverse side effects.

### *A Nonskeletal Example*

Consider, for example, what happens when the vacationer mentioned above goes instead to visit Machu Picchu, the ancient Incan ruins in the Peruvian Andes. After flying to Lima, the tourist's next stop is the city of Cuzco with an elevation of 3700 m (12,000 feet). Picking up her backpack and heading for her hotel, she soon finds her heart pounding and her breath coming in gasps because of the thin air. Clearly, these are responses to the trouble her cells are having in getting enough oxygen. Her increased volume and rate of breathing and pumping blood are somatic changes allowing her to cope with the environmental changes. By the time she leaves Cuzco 2 days later to continue her journey to Machu Picchu itself, carrying her backpack no longer requires her heart and respiration rates to be adjusted upward so much because her hematopoietic system has produced more red blood cells. This second level of somatic adaptability requires more time to put in place, but much less metabolic energy than pumping blood and air faster.

However, more red blood cells per unit volume of blood (called *hematocrit*) is not the best physiologic adjustment for living permanently at high altitude, either. "Lowlanders" who go to high altitudes may suffer severe cardiovascular problems caused by the effects of increased hematocrit on the physical properties of their blood. Some individuals can adjust to living at high altitudes, but many cannot. However, groups of people have lived at high altitudes (principally in the Andes and Himalayan mountains) for many generations and appear

to be free of altitude sickness. It is possible that these populations have experienced genetic changes, through differential reproductive success, which have adapted them to high altitudes in ways that avoid the problems associated with increased hematocrit (Cruz-Coke, 1978).

### *Somatic Change in the Skeleton*

Comparing bone's mechanical adaptation to Bateson's altitude adaptation example, the correlate to increased heart and respiration rates is an increased remodeling rate to repair load-induced microdamage. The correlate to increased hematocrit is periosteal and endosteal modeling to decrease the stresses in the bone. These skeletal responses can have problematic sequelae, just as the respiratory ones can. The increased rate of remodeling requires metabolic energy, and it introduces remodeling space porosity, which can increase strains and the damage formation rate. The formation of additional bone mass also requires metabolic resources, and it increases the bone mass that must be carried about each day, at a very significant energy cost.

### *Somatic Vs. Evolutionary Effects*

How does one know when skeletal variations are of somatic or genetic (evolutionary) origin? The influence of skeletal somatic change is clear in the experiment of Woo and co workers, in which the exercise level of pigs was increased, or that of Utthoff and Jaworski, in which the forelimbs of dogs were immobilized. It is not at all clear, however, when one walks among the skeletons of existing and extinct animals in a museum. For example, the long bones and crania of Neandertals were much more robust than those of modern *H. sapiens*. Trinkaus (1983) has explained this as a somatic change: Neandertals lived during a very cold period in Europe and may have had to forage long distances for food. Trinkaus suggests that the bones adapted to this elevated level of exercise by becoming more robust. Data showing that bone turnover rates were lower in Neandertals than in modern populations (Abbott et al., 1996) support this hypothesis if the mechanostat theory is correct and if addition of bone to periosteal and endosteal surfaces prevented microdamage from accumulating as a result of the higher activity levels. It is also possible, however, that the robustness of the Neandertal skeleton represents a genetic difference from modern *H. sapiens*. For example, if the limits of the equilibrium strain range were genetically shifted to lower levels, ordinary loads would stimulate more bone formation through modeling in growing Neandertals, and suppress remodeling in adults, fitting the observations equally well. Alternatively, some aspect of just the periosteal tissue could have become genetically altered in Neandertals, so ordinary signals to periosteal cells resulted in enhanced bone formation and larger skeletons. The observation of a heav-

ier cranium, hardly useful for foraging long distances for food, seems to fit with this possibility and further supports the hypothesis that the skeletal robustness of these creatures was not somatic. The likelihood of this seems increased now that the analysis of mitochondrial DNA (Krings et al., 1997) makes it unlikely that Neandertals are a part of our own genetic heritage (to the relief of some!). In any case, this example illustrates the difficulty in distinguishing between somatic and evolutionary skeletal changes on the basis of skeletal appearance alone.

In fact, Bateson's point was that all evolutionary change involves endless somatic and genetic adjustments, which together achieve adaptations that would be impossible if only one or the other existed. Thus, it is shortsighted to debate which caused what, exactly. It is more important to learn how somatic and genetic change interact in the skeleton, as they do in other organ systems. This kind of knowledge should ultimately be very useful in treating many bone injuries and diseases.

## 6.8 Summary and Further Reading

Wolff's law and the mechanical adaptability of bones to mechanical loading are not only of great interest to basic scientists interested in the skeleton; they are of great clinical utility as well. Orthopedists see the effects of these phenomena in their daily practices. We have reviewed the history of these concepts in some detail to gain a better understanding of how the current ideas evolved. We saw that there are three key concepts, including optimization of strength with respect to weight, alignment of trabeculae with principal stress directions, and regulation by systems of cells able to respond to a controlling stimulus. We then reviewed some of the more important experiments in this area, and saw that they have emphasized cortical rather than trabecular bone. Although disuse and overload effects are clear, it may be not be appropriate to relate the latter to normal development because woven rather than lamellar bone formation occurred.

We also reviewed some of the theories regarding mechanical adaptability of cortical and trabecular bone, and found that the putative ability of osteocytes to sense strain or strain energy is emerging as an important element of such theories. The discovery that control of strain energy within a loaded structure leads to 0-1 architectures with load-aligned trabeculae has been a key development in recent work. Finally, we asserted that damage repair is an important adjunct to mechanical adaptability because it allows higher strains to be tolerated, and that damage repair and mechanical adaptability are somatic changes which extend the organism's ability to cope with environmental changes without committing to an irreversible genetic change.

Much of the historical material and interpretation at the beginning of this chapter came from two reviews by Roesler (1981, 1987); these are essen-