

# Thermal Relations

As this bumblebee flies from one flower cluster to another to collect nectar and pollen, temperature matters for the bee in two crucial ways. First, the temperature of the bumblebee's flight muscles determines how much power they can generate. The flight muscles must be at least as warm as about 35°C to produce enough power to keep the bee airborne; if the muscles are cooler, the bee cannot fly. The second principal way in which temperature matters is that for a bumblebee to maintain its flight muscles at a high enough temperature to fly, the bee must expend food energy to generate heat to warm the muscles. In a warm environment, all the heat required may be produced simply as a by-product of flight. In a cool environment, however, as a bumblebee moves from flower cluster to flower cluster—stopping at each to feed—it must expend energy at an elevated rate even during the intervals when it is not flying, either to keep its flight muscles continually at a high enough temperature to fly or to rewarm the flight muscles to flight temperature if they cool while feeding. Assuming that the flight muscles must be at 35°C for flight, they must be warmed to 10°C above air temperature if the air is at 25°C, but to 30°C above air temperature if the air is at 5°C. Thus, as the air becomes cooler, a bee must expend food energy at a higher and higher rate to generate heat to warm its flight muscles to flight temperature, meaning it must collect food at a higher and higher rate.

Overall, tissue temperatures have a two-fold significance in many animals, including ourselves. The temperatures of tissues help determine how the tissues perform. Tissue temperatures also help determine an animal's rate of energy expenditure. Bumblebees illustrate both of these points. The temperature of a bumblebee's flight muscles determines how intensely the muscles are able to perform their function of generating lift, and it determines how much food energy the bee must employ for heat production each day.

Physiologists now realize that animals are diverse in the types of thermal relations they maintain with their environments. To categorize the thermal relations of animals, one key concept is **endothermy**; if an animal's tissues are *warmed by its metabolic production of heat*, the animal is said to exhibit endothermy. A second key concept is **thermoregulation**, which refers to the maintenance of a relatively constant tissue temperature.<sup>1</sup> Suppose we classify animals according to whether or not they exhibit endothermy and whether or not they display thermoregulation. Doing so results in the matrix in **Figure 10.1**, which identifies the four most fundamental types of thermal relations that animals have with their environments.

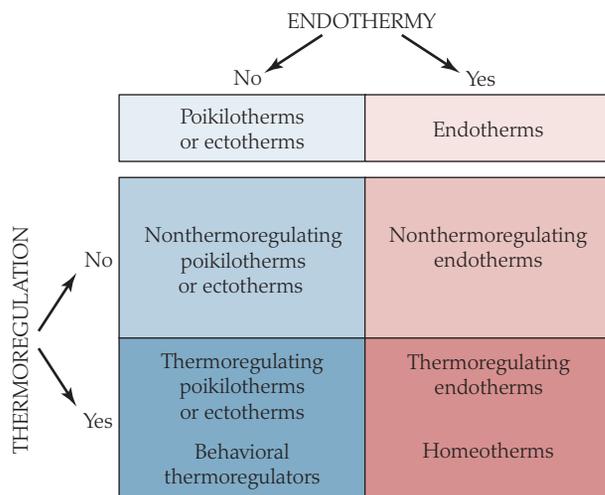
Most animals are incapable of endothermy and thus fall on the left side of the matrix in **Figure 10.1**.<sup>2</sup> Animals of this sort are termed **ectotherms** because the thermal conditions outside their bodies determine their body temperatures (*ecto*, "outside"). They are also

<sup>1</sup>Thermoregulation is a specific type of *regulation* as defined in Chapter 1 (see Figure 1.6).

<sup>2</sup>As stressed in Chapter 7, metabolic heat production is a universal feature of living organisms. When we say "most animals are incapable of endothermy," we do not mean they fail to produce heat metabolically. Remember, instead, that endothermy is *warming of the tissues* by metabolic heat production. Most animals are incapable of endothermy because, although they produce heat, they do not make heat fast enough or retain heat well enough for their tissues to be warmed by their metabolic heat production.

**For a foraging bumblebee, warming the thorax to a high temperature is a critical requirement** The process adds to the bee's energy costs and food needs on cool days. However, the flight muscles in the thorax require high temperatures to produce sufficient power for flight.





**FIGURE 10.1** Animals fall into four categories of thermal relations based on whether they display endothermy and whether they display thermoregulation

called **poikilotherms** because they have variable body temperatures (*poikilo*, “variable”); their body temperatures are high in warm environments but low in cool ones. Most fish are excellent examples of ectotherms or poikilotherms; their tissues are not warmed metabolically and therefore are at essentially the same temperature as the environmental water in which the fish swim.

A poikilotherm or ectotherm may or may not exhibit thermoregulation (see the vertical dimension of Figure 10.1). When a poikilotherm displays thermoregulation and thus falls into the lower left category of our matrix, it does so by *behavior*: It keeps its tissues at a certain temperature by behaviorally choosing to occupy environments that produce that temperature in its body.

Animals that exhibit endothermy—that is, animals that warm their tissues by their production of metabolic heat—are termed **endotherms** and fall on the right side of the matrix in Figure 10.1. Although endotherms may or may not be thermoregulators, most in fact exhibit thermoregulation (placing them in the lower right category of the matrix). Mammals and birds are outstanding examples of animals that exhibit both endothermy and thermoregulation. Many medium-sized and large insects, such as the bumblebees we have already discussed, also exhibit both endothermy and thermoregulation (in their flight muscles when they are flying). A **homeotherm** is an animal that thermoregulates by *physiological* means (rather than just by behavior). Mammals, birds, and insects such as bumblebees are homeotherms. Under many circumstances, the principal way they thermoregulate is by adjusting how rapidly they produce and retain metabolic heat: They thermoregulate by modulating their endothermy!

In addition to the categories identified by our matrix, further complexity arises in categorizing animal thermal relations because of the frequent occurrence of temporal and spatial variation. Speaking first of *temporal variation*, individuals may adopt different thermal relations to their environments at different times. In species of mammals that hibernate, for example, individuals are homeotherms during the seasons of the year when they are not hibernating, but often they exhibit neither endothermy nor thermoregulation when hibernating. Thermal relations may also exhibit *spatial variation*, differing from one region of an animal’s body to another. The abdomens of

bumblebees and other active insects, for example, are typically neither endothermic nor thermoregulated, even in individuals that exhibit endothermy and thermoregulation in their thoracic flight muscles.

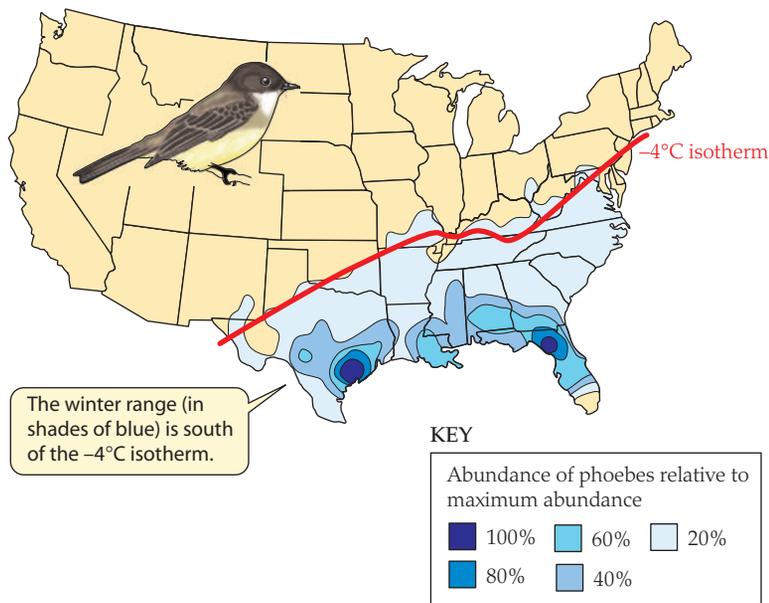
**Heterothermy** refers to a difference in thermal relations from one time to another, or one body region to another, within a single individual. Hibernating species of mammals exemplify **temporal heterothermy**. Flying bumblebees illustrate **regional** (i.e., spatial) **heterothermy**.

Temperature is *always* a major factor in the lives of individual animals, regardless of the particular thermal relations the animals exhibit. Whether animals are poikilotherms or homeotherms, for example, temperature is universally important in at least two ways:

- The environmental temperature—also known as *ambient temperature*—is a principal determinant of an animal’s metabolic rate and therefore the rate at which the animal must acquire food.
- The temperature of an animal’s tissues plays a principal role in determining the functional properties of the tissues and tissue constituents. For example, tissue temperature affects whether protein molecules are in high-performance or low-performance molecular conformations. Tissue temperature also affects the rates of biophysical processes (e.g., diffusion and osmosis), the rates of biochemical reactions, and the viscous physical states of cellular materials such as cell-membrane phospholipids.

The effects of temperature on the properties of entire ecosystems are just as notable as the effects on individual animals. Walking through a temperate woodland during the various seasons of the year provides a striking illustration. On a walk in summer, one is aware of vigorous photosynthesis by plants and sustained activity by mammals, birds, insects, turtles, snakes, amphibians, and other animals. In the winter, however, plants and most animals become cold and quiescent; activity in the woodland becomes restricted largely to the mammals and birds that keep their tissues warm. We cannot always say with certainty whether temperature is the primary determinant of the seasonal changes that we observe in a particular species, because in an entire community of this sort, the responses of any one species may be ripple effects of impacts on others. The evolution of winter quiescence in many animals, for example, has surely been influenced by the effects of cold on plant growth, because the animals face restricted food resources in winter. One cannot doubt, nonetheless, that much of the change in the animal life of a temperate woodland from summer to winter is a direct consequence of the seasonal change of temperature.

Biogeographers have built a strong case that temperature often plays one of the single greatest roles of all environmental factors in determining the geographical ranges of animal species. In North America, for example, the northern limits of the winter ranges of birds often correlate well with particular winter temperatures. Eastern phoebes illustrate this pattern. The northern limit of their geographical range in winter corresponds closely with a line that connects all the places where the average minimum air temperature is  $-4^{\circ}\text{C}$  (Figure 10.2). Eastern phoebes in winter do not extend northward to a fixed latitude, mountain range, river, or other geographical limit. Instead, they extend northward to a relatively fixed severity of winter cold stress. Where winter nights average warmer than about  $-4^{\circ}\text{C}$ , these birds are to be found. Where winter nights average colder than  $-4^{\circ}\text{C}$ , they do not occur.



**FIGURE 10.2** Eastern phoebes (*Sayornis phoebe*) overwinter where the average minimum air temperature in January is  $-4^{\circ}\text{C}$  or warmer. Blue areas show where eastern phoebes overwintered when studied in the 1980s (relative abundance is coded by the shade of blue). The average minimum air temperature in January was  $-4^{\circ}\text{C}$  or warmer below the red line (the “ $-4^{\circ}\text{C}$  isotherm”) and colder than  $-4^{\circ}\text{C}$  above the line. (After Root 1988.)

One reason that biologists today are acutely interested in the influences of temperature is the threat of global warming (Box 10.1). The need for accurate predictions of global warming effects has become a major new reason for the study of animal thermal relations.

## Temperature and Heat

The distinction between *temperature* and *heat* is tricky, and it is important for understanding the thermal relations of animals. To elucidate the distinction, consider a simple inanimate system: two blocks of copper—one of which is ten times more massive than the other, and both of which have been sitting in a room at  $20^{\circ}\text{C}$  long enough that they are at temperature equilibrium with the room. If you measure the *temperature* of each block, you will find that it is  $20^{\circ}\text{C}$ , even though one block is small and the other is large. Suppose, however, that you remove and measure the *heat* from each block; suppose, for instance, that you place each block at absolute zero and measure the amount of heat liberated as the block temperature falls from  $20^{\circ}\text{C}$  to absolute zero. You will find that the large block yields ten times more heat than the small one. Thus, as the two blocks sit in the room at  $20^{\circ}\text{C}$ , their *temperatures* are the same and independent of the amount of matter in each block, but their contents of *heat* are different and directly proportional to the amount of matter in each block.

To understand in greater depth these contrasting attributes of temperature and heat, recall from Chapter 5 (see page 101) that the atoms and molecules within any substance undergo constant random motions on an atomic-molecular scale. The **temperature** of a substance is a measure of the *speed*—or *intensity*—of these

incessant random motions.<sup>3</sup> In the two copper blocks sitting in the room at  $20^{\circ}\text{C}$ , the average speed of atoms during the random atomic-molecular motions is identical; thus, even though the blocks differ in size, they are the same in temperature. **Heat**, unlike temperature, is a form of energy; it is the energy that a substance possesses by virtue of the random motions of its atomic-molecular constituents (see page 163). The amount of heat in a piece of matter thus depends on the *number* of atoms and molecules in the piece, as well as the *speed* of each atom and molecule. A copper block with many copper atoms moving at a given average speed contains proportionally more heat energy than one with fewer atoms moving at the same speed.

A key property of temperature is that it dictates the *direction of heat transfer*. Heat always moves by conduction or convection from a region of high temperature to one of low temperature. To refine this concept, suppose you have a large copper block at  $20^{\circ}\text{C}$  in contact with a tiny copper block at  $30^{\circ}\text{C}$ ; although the large block contains more heat than the small one, heat will move from the small block into the large one because temperature, not energy content, dictates the direction of energy transfer. The net addition of heat to any object causes an increase in the temperature of the object. All in all, therefore, *temperature* and *heat* have intimate interactions:

- Heat moves by conduction or convection from high temperature to low.
- The transfer of heat raises the temperature of the object receiving heat and lowers the temperature of the object losing heat.
- In a simple physical system such as two solid objects in contact with each other, objects are at thermal equilibrium when their temperatures are the same because then heat does not tend to move in net fashion between them.

## Heat Transfer between Animals and Their Environments

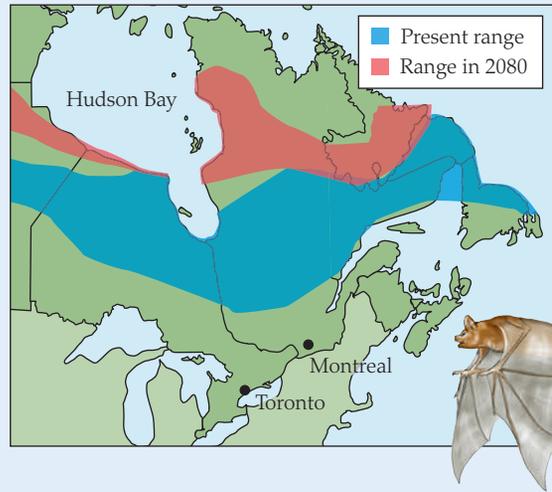
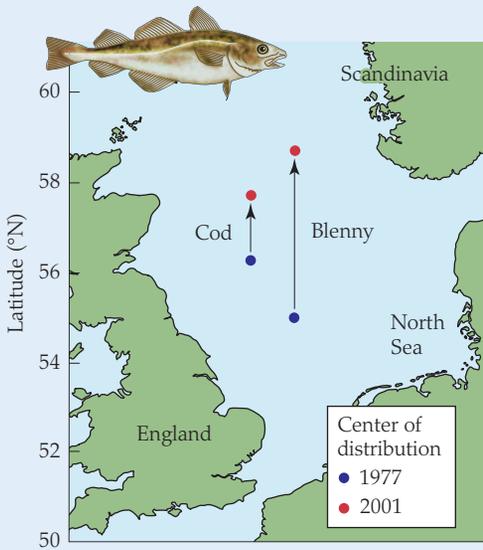
A living animal positioned in an environment, besides making heat internally because of its metabolism, exchanges heat with its surroundings by four distinct heat-transfer mechanisms: *conduction*, *convection*, *evaporation*, and *thermal radiation* (Figure 10.3). The animal may well gain heat by one mechanism of heat transfer while it simultaneously loses heat by another. A familiar illustration of this important point is that on a hot day in summer, people may simultaneously gain heat from the sun by *thermal radiation* while they lose heat by the *evaporation* of sweat. Because the four mechanisms of heat transfer follow distinct laws and can operate simultaneously in opposite directions, they cannot simply be lumped together. Instead, each mechanism needs to be analyzed in its own right, and then the effects of all four can be summed to determine an animal’s overall heat exchange with its environment.

<sup>3</sup>Temperature, more specifically, is proportional to the product of molecular mass and the mean square speed of random molecular motions. The speeds of the motions are astounding. In a gas, molecules collide with each other, bounce apart, and then fly through free space until they collide with other molecules. At  $20^{\circ}\text{C}$ , the average speed during each period of free flight is about 500 m/s! The speed is lower at lower temperatures, and higher at higher temperatures.

### BOX 10.1 GLOBAL WARMING

The great majority of scientists who have assessed the evidence on global climate change agree that effects of global warming are already right before our eyes or can be predicted with confidence.

**Species are tending to shift their ranges poleward.** Surveys of large sets of animal species find that there is a strong statistical bias for species in both hemispheres to be shifting their ranges toward the poles. For example, of 36 fish species studied in the North Sea over a recent 25-year period (1977–2001), 15 species changed their latitudinal center of distribution, and of those, 13 (87%) shifted northward (Figure A). This is the pattern



**FIGURE B** To survive, little brown bats (*Myotis lucifugus*) will need to find new hibernation caves in a new range by 2080. The map shows the ranges in eastern Canada where the bats can find suitable hibernation temperatures, based on a bioenergetic model. Blue shows the model prediction of the present range, whereas red shows the model prediction of the new range that will be required in 2080. The prediction of the present range is compatible with actual known hibernation sites, bolstering confidence in the prediction for the future. (After Humphries et al. 2002.)

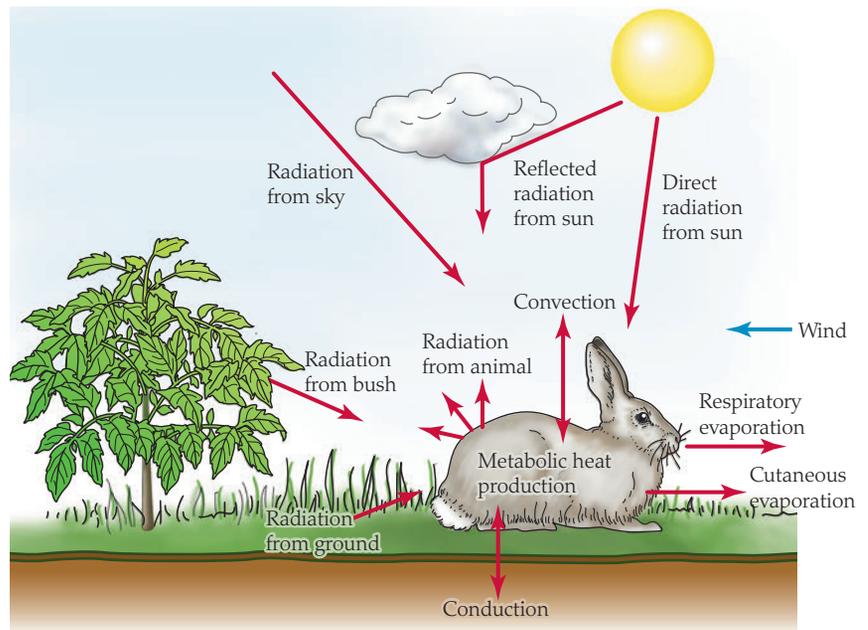
expected as a response to global warming: Faced with a warming environment, many species will shift to more-polar latitudes. A synthetic study of almost 900 animal and plant species that were monitored over a median observation period of 66 years found that 434 of the species shifted their ranges, and of

**FIGURE A** Shifts in the centers of distribution of two fish species in the North Sea from 1977 to 2001. Arrows symbolize the latitudinal shifts of cod (*Gadus morhua*) and blenny (*Lumpenus lampretaeformis*). Average sea temperature increased 1.1°C over the period. Both shifts of species distribution have been monitored annually and have strong statistical support. (After Perry et al. 2005.)

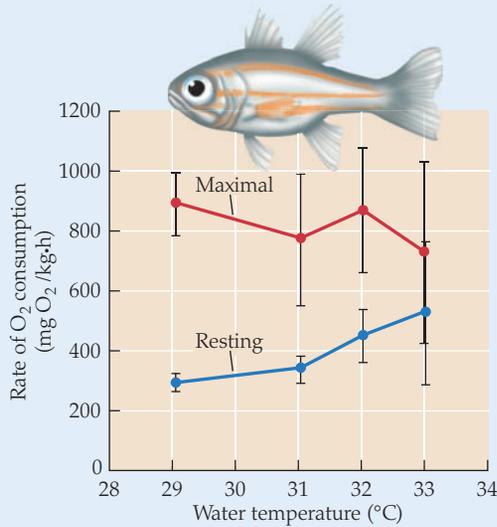
those, 80% shifted as expected in response to a warming world.

**Physiological principles enable researchers to predict with confidence that some species will need to shift their ranges poleward to survive.** Little brown bats provide an example. When small mammals hibernate, they allow their body temperature to fall to environmental temperature. The fall of body temperature is critical because it helps inhibit metabolism and thereby save energy. However, temperature cannot safely decline without limit. When a hibernator's body temperature reaches the lowest tolerable level, the hibernating animal increases its metabolic rate to keep the temperature from falling further. Consequently, energy costs in hibernation are el-

**FIGURE 10.3** An animal exchanges heat with its environment by conduction, convection, evaporation, and thermal radiation. The animal exchanges heat conductively with the ground and convectively with the wind. It loses heat by both respiratory and cutaneous evaporation. It receives thermal radiation from all objects in its surroundings and also emits thermal radiation toward all objects. Finally, it gains heat from its own metabolism. (The cutaneous evaporation from a rabbit is entirely of a nonsweating sort because rabbits lack sweat glands.)



## BOX 10.1 (continued)



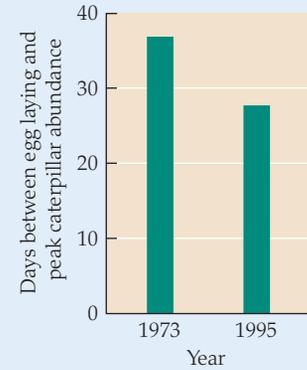
**FIGURE C** Rising water temperatures on the Great Barrier Reef could impair the ability of cardinalfish (*Ostorhinchus doederleini*) to engage in aerobic exercise. When water temperature is raised from 29°C to 31°, 32°, or 33°C, the difference between maximal and resting O<sub>2</sub> consumption becomes significantly smaller with each step. Symbols are means; error bars show  $\pm 1$  standard deviation. (After Nilsson et al. 2009.)

evated both if the temperature is too high and if it is too low. Many hibernators live entirely on fat stores. To survive, they must not exhaust their fat stores before winter's end. Temperatures determine their metabolic rates and therefore

the rates at which they use up their fat stores. Accordingly, a certain temperature range is required for overwinter survival. The latitudes at which hibernating little brown bats are likely to find suitable temperatures are predicted to shift as global warming proceeds, forcing the bats into a new winter range (Figure B).

**Experiments on physiological effects of anticipated temperatures sometimes point to severe future challenges.** Some species of fish on the Great Barrier Reef, for example, are in danger of losing much of their capability to be active, as exemplified by cardinalfish (Figure C). Experiments show that the difference between their maximal rate of O<sub>2</sub> consumption and their resting rate of O<sub>2</sub> consumption (i.e., their aerobic scope) becomes dramatically smaller if the water in which they live is warmed from its current temperature of 29°C to temperatures 2–4°C higher. A rise in water temperature on the reef could reduce the ability of the fish to increase their O<sub>2</sub> consumption, limiting their ability to engage in aerobic exercise (see Chapter 9).

**Effects of environmental warming on some animals can pose ecological challenges for others.** Because animals live in interconnected ecological communities, effects on one species affect others. At a study site in Holland, for example, peak caterpillar abundance in the spring has been occurring progressively earlier from year to year because spring temperatures have been rising, speeding caterpillar development and causing trees (food for the caterpillars) to leaf out earlier. For great tits (chickadee-like

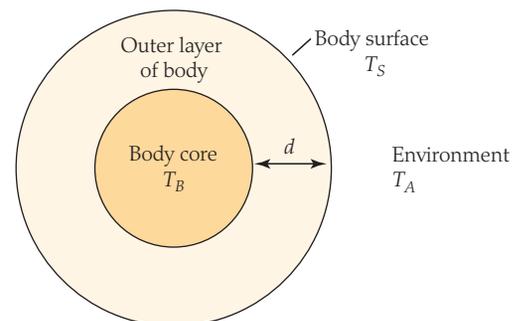


**FIGURE D** Days between egg laying by great tits (*Parus major*) and peak caterpillar abundance in a Holland woodland. When young birds hatch out of the eggs and then undergo their nestling development, the abundance of caterpillars in the woodland affects how well parents can feed them. (After Visser et al. 1998.)

birds), caterpillars for feeding their nestlings are a key to reproductive success. However, the dates when the tits lay their eggs have hardly changed at all. Accordingly, although the time between egg laying and peak caterpillar abundance was about ideal in 1973, it had shortened—and become too short to be ideal—by 1995 and remains so today (Figure D). Food for nestlings has become detectably inadequate because of the mismatch of ever-earlier caterpillar abundance—caused by warming temperatures—while bird reproduction has not shifted to be equally earlier.

For the body temperature of an animal to be constant, the sum total of its heat gains by all mechanisms taken together must equal the sum total of all its heat losses. For instance, suppose that an animal is gaining heat from its environment by conduction and thermal radiation, as well as from metabolism, while losing heat by convection and evaporation. Its body temperature will be constant if and only if the sum of its heat gains by conduction, radiation, and metabolism per unit of time is exactly matched by the sum of its heat losses through convection and evaporation per unit of time.

Figure 10.4 presents a simple model of an animal that will be useful as we discuss the individual mechanisms of heat exchange. The core of an animal's body is considered to be at a uniform **body temperature**, symbolized  $T_B$ . The temperature of the environment is called **ambient temperature**,  $T_A$ . The temperature of the body surface often differs from  $T_B$  and  $T_A$  and thus is distinguished as **surface temperature**,  $T_S$ . Separating the body core from the body surface is the outer layer of the body, where temperature gradually changes from  $T_B$  on the inside to  $T_S$  on the outside.



**FIGURE 10.4** A model of an animal's body showing key temperatures. The body core is at body temperature  $T_B$ , while the surrounding environment is at ambient temperature  $T_A$ . The temperature of the body surface is  $T_S$ . The outer layer of the body, separating the body core from the surface, has thickness  $d$ .

### Conduction and convection: Convection is intrinsically faster

Conduction and convection are usefully discussed together because, in a sense, these two mechanisms of heat transfer define each other. What they have in common is that when heat moves through a material substance by either mechanism, the atoms and molecules of the substance *participate* in the transfer of heat. **Conduction** is the transfer of heat through a material substance that is macroscopically motionless. A familiar example of conduction is the transfer of heat through a block of copper. We know that if the temperature of one side of a copper block is raised, heat will move through the block and appear on the other side even though the copper undergoes no macroscopic motion. The way heat makes its way through such a macroscopically motionless substance is strictly by *atomic-molecular interactions*; if atoms (or molecules) on one side are especially agitated, they increase the agitation of atoms farther into the substance by interatomic collisions, and by repetition of this process, successive layers of atoms relay the increased agitation through the entire thickness of the substance. Conduction mechanistically has much in common with simple solute diffusion (the movement of solute through a macroscopically motionless solution; see page 101), and conduction in fact is sometimes called *heat diffusion*.

**Convection**, in sharp contrast, is transfer of heat through a material substance *by means of macroscopic motion of the substance*. Fluid flow is required for convection. If a wind or water current is present, the macroscopic motion of matter carries heat from place to place. This transfer of heat is convection.

A critical difference between conduction and convection is that, for a given difference of temperature, heat transfer by convection is *much* faster than that by conduction. Consider, for example, a horizontal surface that is 10°C warmer than the surrounding air. If the air is moving at just 10 miles/hour (4.5 m/s), convection will carry heat away from the surface about 70 times faster than if the air is perfectly still! The acceleration of heat transfer by fluid movement is familiar from everyday experience. We all know, for instance, that a wind greatly increases the thermal stress of a cold day.

**THE LAWS OF CONDUCTION** We can better understand conduction if we focus on a specific object, such as a sheet of material of thickness  $d$ . If the temperature on one side of the sheet is  $T_1$ , that on the other is  $T_2$ , and heat is moving through the sheet by conduction, then the rate of heat transfer  $H$  from one side to the other per unit of cross-sectional area is

$$H_{\text{conduction}} = k \frac{T_1 - T_2}{d} \quad (10.1)$$

where  $k$  is a constant. The ratio  $(T_1 - T_2)/d$  is called the **thermal gradient**.<sup>4</sup> You can see from the equation that the rate of heat transfer by conduction through a sheet of material increases as the temperature difference between the two sides increases. In addition, the rate at which heat moves from one side of the sheet to the other decreases as the thickness of the sheet ( $d$ ) increases. The coefficient  $k$  depends in part on the type of material through which conduction is occurring. Some biologically important materials, such as air, conduct heat poorly; they are said to exhibit low *thermal conductivity* and have low

<sup>4</sup>Although the *thermal gradient* is technically defined to be  $(T_1 - T_2)/d$  (i.e., temperature difference per unit of distance), the expression *thermal gradient* is sometimes used to refer simply to a temperature difference,  $(T_1 - T_2)$ .

values of  $k$ . Other materials, such as water, exhibit higher thermal conductivity and higher  $k$  values (water's conductivity is about 20 times that of air).

Heat transfer through the fur of a furred mammal, or through a winter jacket worn by a person, is typically analyzed as a case of conduction because fur traps a layer of relatively motionless air around the body of a furred mammal, and a winter jacket envelops a person's body in a shell of relatively still air. The stillness of the air layer trapped by fur or a jacket is the key to the insulative value of the fur or jacket. To the extent that the air is motionless, heat must move through it by conduction; thus heat moves much more slowly than if convection were at work. Indeed, from the viewpoint of physics, the benefit of fur or a jacket in a cold environment is that it favors an intrinsically slow mechanism of heat loss from the body, conduction, over an intrinsically faster mechanism, convection. In Figure 10.4, the "outer layer" of the body might be taken to represent the fur or jacket. Equation 10.1 shows that increasing the thickness ( $d$ ) of the motionless air layer trapped by the fur or jacket will tend to slow heat loss from an animal or person to a cold environment.

**THE LAWS OF CONVECTION** When air or water flows over an object, the rate of heat transfer by convection between the object and the moving fluid depends directly on the difference in temperature between the *surface* of the object and the fluid. Suppose, for instance, that the model animal in Figure 10.4 is exposed to a wind. Then the rate of convective heat transfer between the animal and the air per unit of surface area is calculated as follows:

$$H_{\text{convection}} = h_c(T_S - T_A) \quad (10.2)$$

The animal will lose heat by convection if its surface temperature ( $T_S$ ) exceeds the ambient air temperature ( $T_A$ ); however, it will gain heat by convection if  $T_A$  is higher than  $T_S$ .

The coefficient  $h_c$ , called the **convection coefficient**, depends on many factors, including the wind speed, the shapes of the body parts of the animal, and orientation to the wind. If the shape of a body part is approximately cylindrical (as is often true of the limbs or torso) and the wind is blowing perpendicularly to the cylinder's long axis, then

$$h_c \propto \frac{\sqrt{V}}{\sqrt{D}} \quad (10.3)$$

where  $V$  is the wind speed and  $D$  is the diameter of the cylinder. This equation shows that the rate of heat transfer per unit of surface area by convection tends to increase with the square root of the wind speed. The rate of heat transfer per unit of surface area also tends to increase as the square root of the diameter of a cylindrically shaped body part is decreased; this physical law helps explain why body parts of small diameter (e.g., fingers) are particularly susceptible to being cooled in cold environments.

### Evaporation: The change of water from liquid to gas carries much heat away

Evaporation of body water from the respiratory passages or skin of an animal takes heat away from the animal's body because water absorbs a substantial amount of heat whenever its physical state changes from a liquid to a gas. The amount of heat required to vaporize water, called the **latent heat of vaporization**, depends on the prevailing temperature. It is 2385–2490 J (570–595 cal) per gram of  $\text{H}_2\text{O}$  at physiological temperatures. These are *large* values.



**FIGURE 10.5** An antelope jackrabbit (*Lepus alleni*) This species of jackrabbit is found principally in the low-altitude desert plains of southern Arizona and northern Mexico.

Whereas heating a gram of liquid water from 0°C to 100°C requires 100 cal, changing a gram from a liquid to a gas requires 570–595 cal—almost six times as much. The enormous heat absorption that occurs when water vaporizes means that evaporation can be a highly effective cooling mechanism for an animal. The heat is absorbed from the body surface where the vaporization occurs, and it is carried away with the water vapor.<sup>5</sup>

### Thermal radiation permits widely spaced objects to exchange heat at the speed of light

For terrestrial animals, including people, thermal-radiation heat transfer often ranks as one of the quantitatively dominant mecha-

<sup>5</sup>The opposite of evaporation, *condensation*, usually does not occur in biological systems; when it does, however, heat is added to the site where water changes to its liquid form from its gaseous form. See Chapter 27 (page 706) for a detailed discussion of the physical laws of evaporation and condensation.

nisms of heat exchange with the environment, yet it tends to be the least understood of all the mechanisms. Although we are all familiar with radiant heating by the sun, such heating is only a special case of a sort of heat transfer that is in fact ubiquitous.

The first fact to recognize in the study of thermal-radiation heat transfer is that all objects *emit* electromagnetic radiation. That is, all objects are *original sources* of electromagnetic radiation. If you look at a wall, your eyes see electromagnetic radiation (light) coming from the wall, but that radiation is merely reflected; it originated from a lamp or the sun and reflected off the wall to enter your eyes. As a completely separate matter, the wall also is the original source of additional electromagnetic radiation. The radiation *emitted* by the wall is at infrared wavelengths and thus invisible. It travels at the speed of light, essentially unimpeded by the intervening air, until it strikes a solid surface (such as your body), where it is absorbed. Simultaneously, your body emits electromagnetic radiation, some of which strikes the wall. In this way the wall and your body can exchange heat even though they are not touching and in fact may be far apart. Any two objects that are separated only by air undergo exchange of heat at the speed of light by thermal-radiation heat transfer.<sup>6</sup>

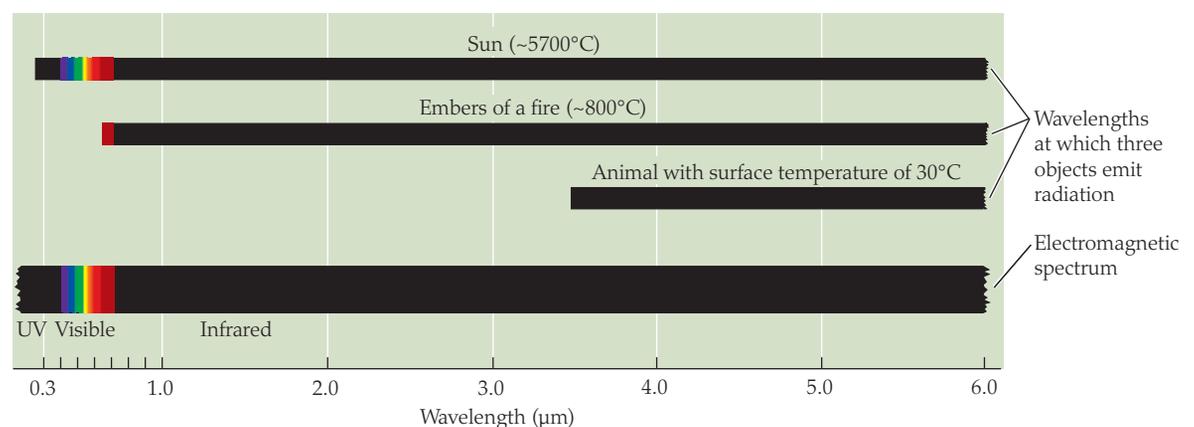
An interesting application of the principles of thermal-radiation heat transfer is to the huge ear pinnae of jackrabbits (**Figure 10.5**). In some species, such as the one pictured, the ear pinnae constitute 25% of the total body surface area. Despite decades of interest, physiologists still do not definitely know the function of these pinnae. The most likely function is that they act as radiators. Jackrabbits modulate blood flow to the pinnae. When blood flow is brisk and the pinna blood vessels are engorged (as in Figure 10.5), the pinnae are warmed, and they thereby increase the intensity at which they emit electromagnetic radiation. When heat is lost in this way, it need not be lost by panting or other forms of evaporation—a water-saving benefit for animals that live in deserts or semideserts.<sup>7</sup>

When objects emit electromagnetic radiation, they do so over a *range* of wavelengths. A key principle of thermal-radiation physics, illustrated in **Figure 10.6**, is that the range of wavelengths emitted by an object depends on the *surface* temperature of the object ( $T_s$ ) and shifts toward shorter wavelengths as the surface temperature

<sup>6</sup>Water, being far more opaque to infrared radiation than air, largely blocks this sort of heat transfer in aquatic environments.

<sup>7</sup>When the pinnae are warmer than the air, heat will also be carried away from them by convection if a breeze or wind is present. Like heat loss by thermal-radiation heat transfer, loss by convection also occurs without making demands on body water.

**FIGURE 10.6** As objects reach higher surface temperatures, the ranges of wavelengths at which they emit thermal radiation extend to shorter wavelengths. Temperatures specified are surface temperatures. All three of the objects shown also emit energy at wavelengths longer than 6  $\mu\text{m}$  (not shown).



increases. The lowest thin black bar in Figure 10.6 shows the wavelengths emitted by an animal or other object with a surface temperature of about 30°C. Note that the shortest wavelengths emitted by a surface at this temperature are between 3 and 4 μm; energy is also emitted over a broad range of longer wavelengths. All the emitted wavelengths are in the infrared range and thus invisible. The embers of a fire (middle thin black bar in the figure) emit at shorter wavelengths because they are hotter. They are in fact hot enough that the shortest wavelengths they emit are within the visible range. Because we *see* those wavelengths, we see the coals *glow*. The visible wavelengths emitted by the coals are limited to the red-orange end of the visible spectrum; thus the glow of the coals is red-orange. The sun is so hot that it emits electromagnetic energy (upper thin black bar in the figure) at all wavelengths of the visible spectrum and therefore glows with a nearly white light. The most important concept illustrated by Figure 10.6 is that the radiative emissions from organisms are of the same basic nature as those from a fire or the sun. The only reason we do not see organisms glow is that the wavelengths they emit are out of our visible range.

An important principle of thermal-radiation physics is that the *total intensity* of radiation emitted by an object—summing the radiation emitted at all wavelengths—increases as surface temperature increases:

$$H_{\text{radiative emission}} = \epsilon\sigma T_s^4 \quad (10.4)$$

In this equation, which is known as the **Stefan-Boltzmann equation**,  $H$  is the rate of emission per unit of surface area at all wavelengths combined,  $\epsilon$  is a surface property called *emissivity (emittance)*,  $\sigma$  is a constant called the Stefan-Boltzmann constant, and the surface temperature  $T_s$  must be expressed in absolute degrees (K).

Another important principle of thermal-radiation physics is that when electromagnetic radiation strikes an object, the radiant energy may be *absorbed* or *reflected*, or it may *pass through*. The fractions of the energy absorbed, reflected, and transmitted depend on the surface properties of the object and are wavelength-specific. Energy that is absorbed is converted into heat at the surface of the absorbing object, as illustrated in everyday experience by the fact that our skin is warmed by radiant energy from the sun or from the embers of a fire.

**RADIANT EXCHANGES IN THE BIOSPHERE THAT DO NOT INVOLVE THE SUN** In natural biological communities, the sun is usually the only object that is hot enough to emit energy at wavelengths shorter than 3–4 μm. The surface temperatures of animals, plants, rocks, and all other objects besides the sun are typically between –50°C and 50°C, and surfaces at such temperatures emit only wavelengths of 3–4 μm or longer (see Figure 10.6). Thus, if we exclude the sun from consideration, all radiant exchanges among objects in the biosphere are at such wavelengths: Various organisms and objects emit at 3–4 μm and longer, and the emitted radiation that they receive from other organisms and objects is at 3–4 μm and longer. This fact massively simplifies the analysis of radiant exchanges because although organisms and objects in the biosphere commonly differ from one another in surface temperature, all are essentially identical in their other radiative properties at wavelengths of 3–4 μm and longer. Specifically, all exhibit about the same value for  $\epsilon$  in the Stefan-Boltzmann equation (Equation 10.4) at these wavelengths; and all are highly absorptive at these

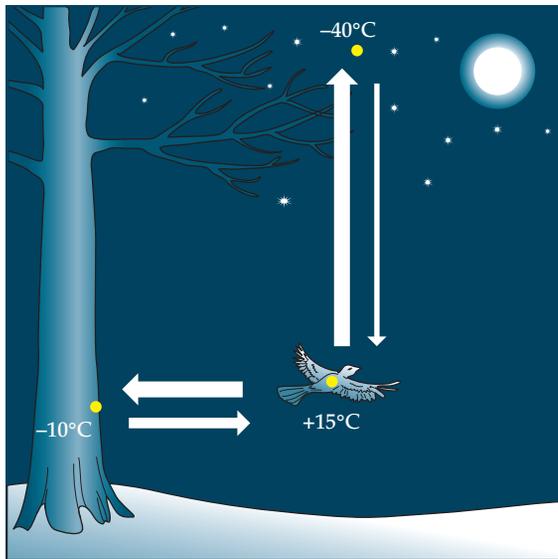
wavelengths, meaning that they absorb (rather than reflect or transmit) most energy that strikes them. Put loosely, organisms and objects in the biosphere do not differ in color at these wavelengths. If this idea sounds strange, recognize that the color you see with your eyes is a property at *visible* wavelengths of 0.4–0.72 μm. Whether the visible color of an organism or object is brown, green, or even white, the color at wavelengths of 3–4 μm and longer is, in all cases, nearly black.

Because all organisms and objects in the biosphere are virtually identical in  $\epsilon$  and in their absorptive properties at wavelengths of 3–4 μm and longer, surface temperature ( $T_s$ ) is the sole major determinant of radiative heat exchange when the sun is excluded from consideration. If two organisms or objects are exchanging heat radiatively, each can be considered to emit a beam of energy toward the other. Whereas the warmer of the two emits a relatively strong beam (see Equation 10.4), the cooler emits a relatively weak beam. Each absorbs most of the energy that it receives from the other. For these reasons, energy is passed in *net* fashion from the warmer object to the cooler one. Quantitatively, if the surface temperatures of the two objects (on the Kelvin scale) are  $T_1$  and  $T_2$ , the net rate of heat transfer between them is proportional to  $(T_1^4 - T_2^4)$ , and the direction of net heat transfer is from the one with the higher  $T_s$  to the one with the lower  $T_s$ .

As examples, consider first a relatively cool lizard standing in the early nighttime hours near a rock that remains hot from the preceding day. The rock emits a relatively strong beam of radiant energy toward the lizard, and the lizard absorbs most of this radiant energy; simultaneously, the lizard emits a weaker beam of energy toward the rock, and the rock absorbs that energy. The net effect is that the lizard is warmed by standing near the rock. A less familiar example is provided by a bird flying past cold trees on a frigid winter night (Figure 10.7). The surface temperature of the bird (+15°C in Figure 10.7) is higher than that of the tree trunks (–10°C). In this case the beam of energy carrying heat away from the bird is more intense than the beam striking the bird from each tree, and the net effect of thermal-radiation heat transfer is to cause a loss of heat from the bird to the trees.

**THE NIGHT SKY AS A RADIANT OBJECT** The sky is one of the objects in the biosphere that deserves special note. Here we consider just the night sky; because the sun is absent at night, the discussion in this section is a special case of the last section's discussion. In the atmosphere above us at night, each gas molecule—whether positioned just above Earth's surface or at the limits of outer space—emits radiation as a function of its temperature. In this way, the surface of Earth steadily receives a beam of radiation emitted from the sky above. One way to express the intensity of this radiation is to pretend that the sky is a solid surface and ask what the temperature of that surface would have to be for it to emit at the intensity observed (assuming  $\epsilon = 1.0$ ). This temperature is called the **radiant temperature of the sky** (or the *black-body sky temperature*). A characteristic of the radiant temperature of the clear night sky is that it is far lower than the simultaneous air temperature at ground level. For example, during a particular summer night in the Arizona desert when the air temperature near the ground was +30°C, the radiant temperature of the clear sky was simultaneously –3°C; that is, the sky on that warm night behaved like a subfreezing object! The low radiant temperature characteristic of the clear night sky

The widths of the arrows symbolize the relative intensities of the beams of thermal radiation.



**FIGURE 10.7** A bird loses heat in net fashion to tree trunks by thermal radiation as it flies past them on a cold winter night. The bird also loses heat in net fashion to the night sky. More than half of a bird's total heat loss may be by thermal-radiation heat transfer. The temperatures shown for the tree and bird are their surface temperatures; that shown for the sky is the radiant sky temperature expected on a night when the air temperature near the ground is  $-10^{\circ}\text{C}$ . Quantitatively, thermal-radiation heat transfer depends on temperature on the absolute (Kelvin) scale. On that scale,  $+15^{\circ}\text{C} = 288\text{ K}$ ;  $-10^{\circ}\text{C} = 263\text{ K}$ ; and  $-40^{\circ}\text{C} = 233\text{ K}$ .

explains how frosts can form on nights when the air temperature at ground level stays above freezing.

When animals are exposed to the clear night sky, they emit a beam of radiation toward the sky. In return, they receive only a relatively weak beam of radiation from the sky (see Figure 10.7). Accordingly, animals tend to lose energy in net fashion to the clear night sky, which is often, therefore, said to act as a “radiant heat sink.” The radiative loss of heat to the clear sky is often of substantial importance. Because of this, animals confronted with cold stress may benefit considerably by avoiding exposure to the clear sky. When small mammals burrow under the snow in winter, for example, one of the advantages they obtain is that they reduce their radiant heat losses at night by interposing the snow as a barrier to radiant exchange between themselves and the sky; however cold the snow may be, it is warmer than the clear night sky.

**SOLAR RADIATION** The sun is the one object in the biosphere that routinely emits radiation at wavelengths shorter than  $3\text{--}4\ \mu\text{m}$  (see Figure 10.6). Much of the solar radiant energy is at visible or near-visible wavelengths. Accordingly, when we consider objects exposed to solar radiation, the visible colors of the objects matter; visible color affects the fraction of the energy that is absorbed. If an animal's body surfaces are opaque (nontransparent),<sup>8</sup> the analysis

<sup>8</sup>If the body surfaces of an animal are significantly transparent, additional complexities are introduced. For example, the pelages of mammals and plumages of birds are partly transparent (radiant energy can pass through them to some extent) and therefore do not necessarily follow the simple rule that black means high absorption and white means low absorption of solar visible energy.

of the effects of the animal's visible color on the absorption of the visible and near-visible solar radiation is straightforward: Dark surfaces absorb more of this solar radiation—and are heated more by it—than light ones. Black beetles, for instance, absorb the visible and near-visible wavelengths relatively well, whereas light-colored beetles tend more to reflect these wavelengths and absorb them relatively poorly. Animals that can change their skin color, such as many species of lizards, can increase and decrease the solar heating of their bodies by darkening and lightening, respectively.

### SUMMARY Heat Transfer between Animals and Their Environments

- In addition to making heat metabolically, animals exchange heat with their environments by conduction, convection, evaporation, and thermal radiation. An animal's body temperature depends on heat gains and losses; it is constant only if the sum total of gains equals the sum total of losses.
- Conduction and convection have in common the property that when heat moves through a material substance by either mechanism, the atoms and molecules of the substance participate in the transfer of heat. Conduction, also called thermal diffusion, occurs when a material substance is macroscopically motionless. Convection is heat transfer brought about by flow of a material substance (e.g., by wind). Convection is much faster than conduction.
- Evaporation is a potentially potent mechanism for heat transfer because the change of state of water from a liquid to a gas absorbs a great deal of heat per gram of water. The heat is absorbed from the surface where evaporation occurs and is carried away with the water vapor.
- Thermal-radiation heat transfer occurs by means of beams of radiant energy that all objects emit and that travel between objects at the speed of light. Because of thermal-radiation heat transfer, objects can exchange heat at a distance. In most instances of thermal-radiation heat transfer in the biosphere, the heat transfer occurs at invisible infrared wavelengths; because all objects are nearly black at such wavelengths, visible color plays little role, and the net transfer of heat is from the object with higher surface temperature to the one with lower surface temperature. Visible color, however, is a major factor in how well objects absorb the visible and near-visible wavelengths of solar radiation.

### Poikilothermy (Ectothermy)

Poikilothermy is by far the most common type of thermal relation exhibited by animals. Amphibians, most fish, most nonavian reptiles, all aquatic invertebrates, and most terrestrial invertebrates are poikilotherms. The defining characteristic of poikilothermy is that the animal's body temperature is determined by equilibration with the thermal conditions of the environment and varies as environmental conditions vary. *Poikilothermy* and *ectothermy* are the same thing. The two terms simply emphasize different aspects of one phenomenon; whereas *poikilothermy* emphasizes the variability of body temperature, *ectothermy* emphasizes that outside conditions determine the body temperature (see page 226).

Poikilothermy manifests itself differently depending on whether an animal is aquatic or terrestrial. Aquatic poikilotherms typically have body temperatures that are essentially the same as water temperature. Terrestrial poikilotherms, however, do not necessarily have body temperatures that equal “air” temperature, because thermal-radiation heat transfer or evaporation on land can tend to draw the body temperature away from air temperature. For instance, if a frog or snail on land basks in the sun, its body temperature may be much higher than the air temperature. Such animals nonetheless still meet the definition of poikilothermy or ectothermy, because their body temperatures are determined simply by equilibration with the sum total of thermal conditions in their environments.

Poikilothermic or ectothermic animals are often called *cold-blooded* in nonscientific writing, in reference to their coolness to the touch under certain conditions. Many species, however, may have high body temperatures when in warm environments. For example, desert lizards and insects that are perfectly fine poikilotherms often have body temperatures that substantially exceed human body temperature! *Cold-blooded* is therefore not a suitable general term to describe poikilotherms or ectotherms.

### Poikilotherms often exert behavioral control over their body temperatures

The natural environments of poikilotherms typically vary from place to place in thermal conditions. In a forest, for example, the temperature on the exposed forest floor might be higher than that under a log, and the temperature in a spot of sunlight might be higher yet. Poikilotherms in the wild can behaviorally choose where they position themselves and, in this way, control their body temperatures. One reason to stress this point near the start of our discussion of poikilothermy is that the situation of captive poikilotherms is so different and potentially misleading. In an aquarium or cage, environmental thermal conditions are usually uniform, meaning that the body temperatures of captive poikilotherms are deterministically set by the environmental conditions. In dramatic contrast, when the thermal environment is heterogeneous—as it nearly always is in nature—poikilotherms typically can and do exert some control over their body temperatures.

If a poikilotherm behaviorally maintains a *relatively constant* body temperature, it is said to exhibit **behavioral thermoregulation**. Sometimes behavioral thermoregulation is rather simple. In a lake or the ocean, for instance, various large water masses (such as those at the surface and at greater depth) often differ in temperature (see Figure 1.14). Fish that elect to stay in one water mass, rather than another, take on the temperature of the water they occupy and remain at that temperature for extended periods. The behavior of the fish is accordingly a simple form of behavioral thermoregulation.

In other cases, behavioral thermoregulation is far more complex and dynamic. Many lizards, for example, maintain relatively stable body temperatures during daylight hours, and they do so by complex, moment-to-moment behavioral exploitation of environmental opportunities for heating and cooling. A desert lizard, for instance, ordinarily emerges in the morning and basks in the sun until its body temperature rises to be within a “preferred” range that it maintains during its daily activity. Thereafter, the lizard keeps its body temperature within that range until nightfall by a variety of

mechanisms. One common strategy is to shuttle back and forth between sun and shade; when its body temperature starts to drop too low, the lizard moves into sunlight, and then later, when its body temperature starts to rise too high, it enters shade. The lizard might also modify the amount of its body surface exposed to the direct rays of the sun by changing its posture and orientation to the sun. It might flatten itself against the substrate to lose or gain heat (depending on substrate temperature), and when the substrate has become very hot during midday, the lizard might minimize contact by elevating its body off the ground or even climbing on bushes. By thus exploiting the numerous opportunities for heating and cooling in its thermally heterogeneous environment, a lizard may well maintain a body temperature that varies only modestly for long periods. The desert iguana illustrated in Figure 1.12, for instance, typically maintains an average abdominal temperature of 38–42°C during daylight hours, and it often keeps its temperature within 2–3°C of the mean for hours on end.

Investigators have worried a lot about the question of documenting true behavioral thermoregulation. They thus have compared living animals with inanimate model animals. In one study, living lizards in a natural setting on a Mediterranean island were found to exhibit far less variable body temperatures than lizard models placed widely in the same environment (Figure 10.8). Such evidence documents that real lizards do not simply position themselves at random, but *behave* in ways that keep their body temperatures within a relatively narrow preferred range.

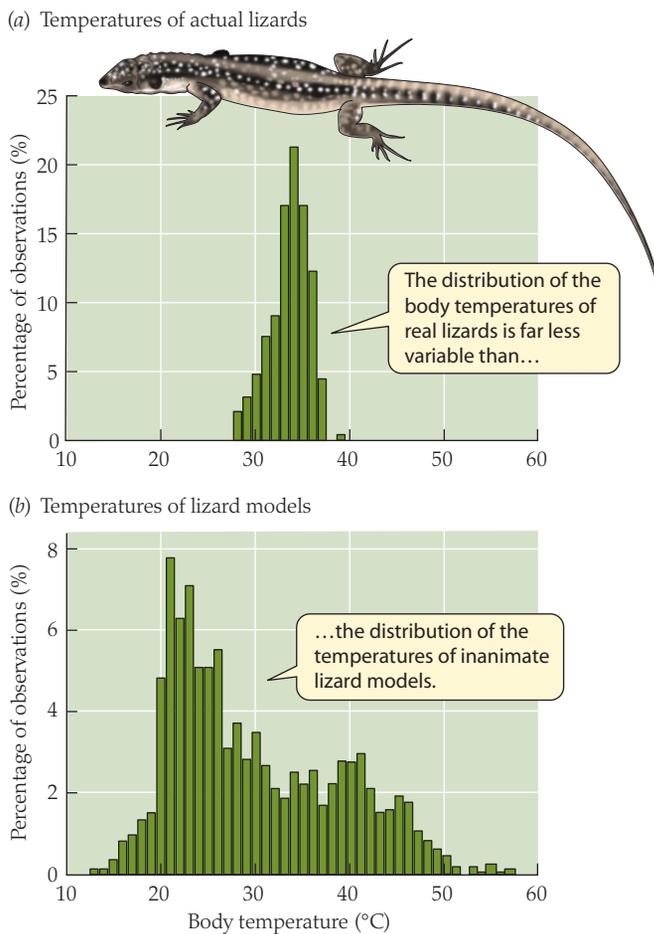
### Poikilotherms must be able to function over a range of body temperatures

A limitation of behavioral thermoregulation is that it is dependent on the thermal opportunities available in the environment, and thus it may be thwarted by changes of weather or other conditions outside an animal’s control. A desert iguana, for example, may never reach a body temperature that is even close to its “preferred” level of 38–42°C on a day that happens to be cloudy and cool. Similarly, a fish that would select a cool water mass if it could, cannot do so if all the water in its lake or pond is warm.

For these and other reasons, poikilotherms must typically be thermal generalists: They must be capable of functioning at a variety of different body temperatures. Species differ in how wide a range of body temperatures is acceptable. Some species, termed **eurythermal**, can function over wide ranges of body temperature; goldfish, for instance, maintain normal body orientation, feed, and swim at body temperatures of 5–30°C. Other poikilotherms, termed **stenothermal**, have comparatively narrow ranges of body temperature over which they can function.

### Poikilotherms respond physiologically to their environments in all three major time frames

The three major time frames of physiological response to the environment identified in Chapter 1 (see Table 1.2) provide a useful way to organize knowledge of the relations of poikilotherms to their thermal environments. In three of the next four sections, we discuss poikilotherms in each of the three time frames. First, in the next section, we address the *acute responses* of poikilotherms to changes in their body temperatures. The acute responses are those that individual animals exhibit *promptly* after their body temperatures are altered. After that we address the *chronic responses*



**FIGURE 10.8 Behavioral thermoregulation documented by comparison of real lizards with inanimate lizard models**

Multiple daytime measurements of the body temperatures of real lizards (a) and inanimate lizard models (b) were made. The lizards (*Podarcis hispanica*) were living freely on a Mediterranean island. The lizard models were placed as comprehensively as possible in all the various microhabitats available to real lizards during their daytime activities on the same island. Data on the y axes are the percentages of all observations in various 1°C intervals of temperature. (After Bauwens et al. 1996.)

of poikilotherms, termed *acclimation* and *acclimatization*.<sup>9</sup> What changes do individual animals undergo when they live in an altered thermal environment (and have altered body temperatures) for a

<sup>9</sup>The distinction between *acclimation* and *acclimatization* is discussed on page 15. As also discussed there, they are forms of *phenotypic plasticity*.

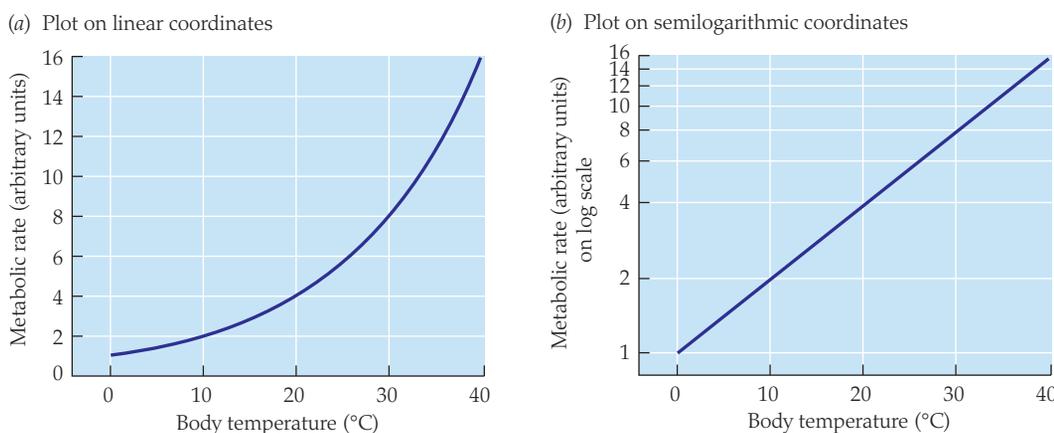
prolonged period? Finally, after discussing temperature limits, we discuss *evolutionary changes*—the ways in which the physiology of poikilotherms may be modified by changes in the frequencies of genes when populations live in different environments over many generations.

### Acute responses: Metabolic rate is an approximately exponential function of body temperature

When the body temperature of an individual poikilotherm is raised in a series of steps and its metabolic rate is measured promptly after each upward step, the usual pattern is that the resting metabolic rate increases approximately exponentially with the animal's body temperature (Figure 10.9a).<sup>10</sup> An *exponential* relation signifies that the metabolic rate increases by a particular *multiplicative factor* each time the body temperature is stepped up by a particular *additive increment* (see Appendix F). For example, the metabolic rate might increase by a factor of 2 for each increment of 10°C in body temperature. Then, if the metabolic rate were 1 joule per minute (J/min) at 0°C, it would be 2 J/min at 10°C, 4 J/min at 20°C, and 8 J/min at 30°C (see Figure 10.9a). The acute relation between metabolic rate and body temperature is usually, in fact, only *approximately* exponential. That is, the factor by which the metabolic rate increases for a given increment in temperature is usually not precisely constant from one temperature range to the next but might, for example, be 2.5 between 0°C and 10°C but only 1.8 between 20°C and 30°C.

The reason that the metabolic rate of a poikilotherm increases as its body temperature goes up relates back to the concept of activation energy discussed in Chapter 2. Each biochemical reaction involved in metabolism is characterized by a particular activation energy, a certain minimum energy level that a reacting molecule must attain in order to undergo the reaction (see Figure 2.13). As the temperature of a cell increases, all molecules in the cell tend to become more agitated and have higher energy levels. Svante Arrhenius (1859–1927) demonstrated in the late nineteenth century that if one specifies any particular activation energy, the fraction of molecules that have that level of energy—or more—at any moment increases approximately exponentially as temperature increases. Reactions tend, therefore, to speed up approximately exponentially as cellular temperature rises. In this context, it is vital to recall that

<sup>10</sup>There are limits to this process: An exponential increase is seen only within a particular range of body temperatures, a range that depends on the species and individual. We discuss the limits later in the chapter.



**FIGURE 10.9 An exponential relation between metabolic rate and body temperature plotted in two ways**

(a) A plot employing linear scales for both variables. (b) A semilogarithmic plot of the same data as in part (a); metabolic rate is plotted on a logarithmic scale, whereas body temperature is plotted on a linear scale. Metabolic rate is expressed in the same arbitrary units in both parts. See Appendix E for background on logarithmic scales.

most metabolic reactions are enzyme catalyzed, and the enzymes determine the activation energies. Thus the detailed, quantitative relations between biochemical reaction rates and cellular temperature depend on the particular enzyme proteins that cells synthesize.

If the resting metabolic rate of a poikilotherm, symbolized  $M$ , were a true exponential function of its body temperature ( $T_B$ ), the relation would be described by an exponential equation (see Appendix F):

$$M = a \cdot 10^{n \cdot T_B} \quad (10.5)$$

where  $a$  and  $n$  are constants. If one takes the common logarithm of both sides of Equation 10.5, one gets

$$\log M = \log a + n \cdot T_B \quad (10.6)$$

According to this second equation,  $\log M$  is a *linear* function of  $T_B$  ( $\log a$  and  $n$  are constants).

Thus, if  $M$  is an exponential function of  $T_B$  as in Equation 10.5,  $\log M$  is a linear function of  $T_B$  (Equation 10.6). This result represents the basic reason why physiologists usually plot metabolism–temperature data for poikilotherms on semilogarithmic coordinates. The logarithm of the animal’s metabolic rate is plotted on the  $y$  axis, and the animal’s body temperature itself is plotted on the  $x$  axis. The curve of Figure 10.9a is replotted on semilogarithmic coordinates in Figure 10.9b, illustrating the “linearizing” effect of semilogarithmic coordinates. A similar comparison is seen in Figure 10.10 using data on actual animals. As we have emphasized, metabolic rate in fact is usually an *approximately* exponential function of body temperature, not a truly exponential one. Thus the semilogarithmic plot for actual animals is typically not precisely linear, as exemplified in Figure 10.10b.

One simple way to describe an exponential relation between metabolic rate (or any other physiological rate) and temperature is to specify the multiplicative factor by which the rate increases when the body temperature is increased by a standardized increment of 10°C. This factor is called the **temperature coefficient,  $Q_{10}$** :

$$Q_{10} = \frac{R_T}{R_{(T-10)}} \quad (10.7)$$

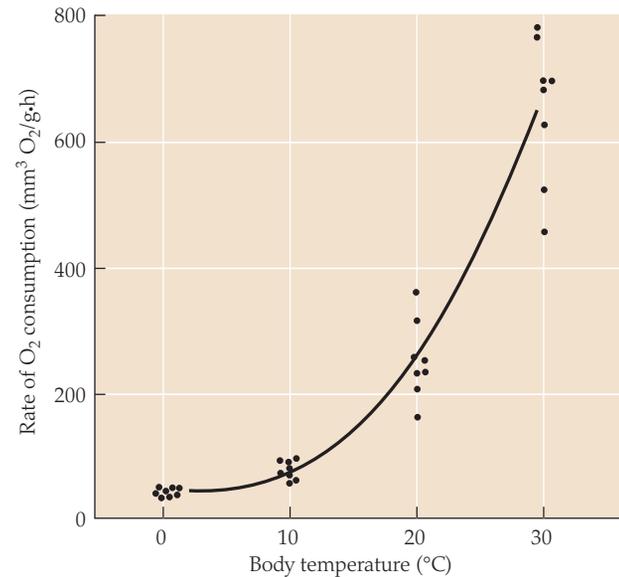
where  $R_T$  is the rate at any given body temperature  $T$ , and  $R_{(T-10)}$  is the rate at a body temperature 10°C lower than  $T$ . To illustrate, if the resting metabolic rate of an animal is 2.2 J/min at a body temperature of 25°C and 1.0 J/min at 15°C, the  $Q_{10}$  is 2.2. As a rough rule of thumb, the  $Q_{10}$  for the metabolic rates of poikilotherms is usually between 2 and 3. If metabolic rate were a truly exponential function of body temperature, you could calculate the  $Q_{10}$  of an animal from data for *any* two body temperatures that are 10°C apart and always get the same value. Because metabolic rate is not a truly exponential function of temperature, however, the  $Q_{10}$  of an animal in fact varies with the particular range of body temperatures considered.<sup>11</sup>

### Chronic responses: Acclimation often blunts metabolic responses to temperature

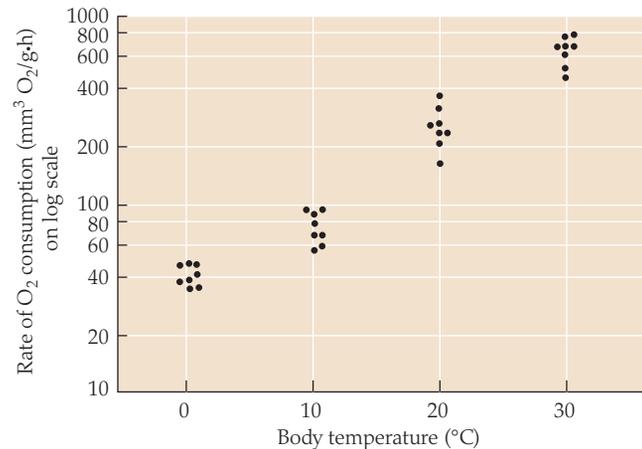
When an individual poikilotherm is kept chronically at one body temperature for a number of weeks and then is kept chronically at

<sup>11</sup> An equation called the *van’t Hoff equation* permits  $Q_{10}$  to be calculated for ranges of temperature that are less than 10°C wide. Thus, for detailed analysis, the temperature scale need not be artificially divided into 10°C segments.

(a) Plot on linear coordinates



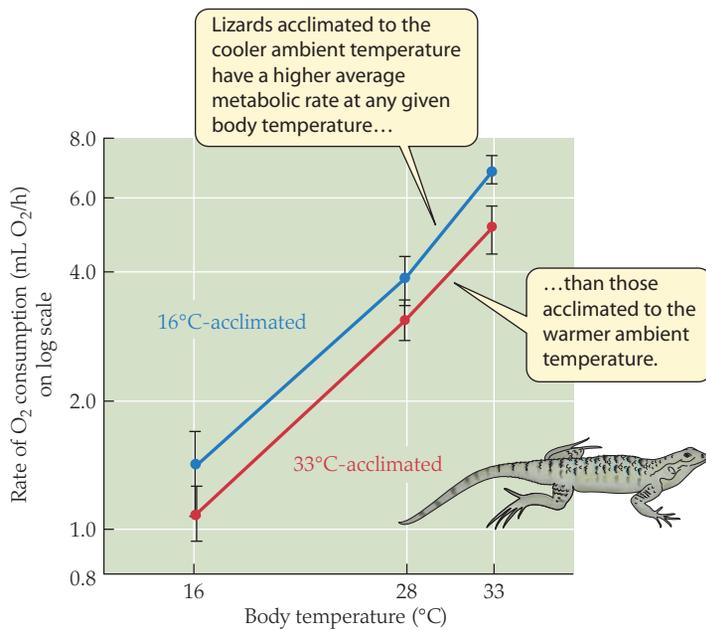
(b) Plot on semilogarithmic coordinates



**FIGURE 10.10** The relation between metabolic rate and body temperature in tiger moth caterpillars (family Arctiidae), plotted in two ways The metabolic rate was measured as the rate of  $O_2$  consumption. (a) A plot employing linear scales for both variables. (b) A semilogarithmic plot. Note that the data do not plot exactly as a straight line in (b). This fact signifies that the relation is not truly exponential, although it is approximately exponential. (After Scholander et al. 1953.)

a different body temperature for a number of weeks, the details of its acute metabolism–temperature relation usually change. Such a change is an example of *acclimation* (see page 15). Understanding this sort of acclimation and its implications can be tricky. The best way to gain clear insight is to start with the actual procedures that are followed to study acclimation. To this end, let’s discuss the acclimation study in Figure 10.11.

In the experiment represented by Figure 10.11, a group of lizards, named the “33°C-acclimated” group, was maintained for 5 weeks at 33°C. At the end of this chronic exposure to 33°C, the lizards were exposed acutely (i.e., briefly) to three different body temperatures—16°C, 28°C, and 33°C—and their resting metabolic rates were measured at each of the three. The line labeled “33°C-acclimated” shows the results. It represents the *acute* relation between resting metabolic rate and body temperature for lizards



**FIGURE 10.11** Acclimation of the metabolism–temperature relation to a change in chronic temperature in a poikilotherm One group of fence lizards (*Sceloporus occidentalis*) was acclimated for 5 weeks to 33°C prior to testing. A second, otherwise identical group was acclimated to 16°C for 5 weeks prior to testing. After the 5 weeks, the animals in each group were placed briefly at body temperatures of 16°C, 28°C, and 33°C, and their standard metabolic rates were measured at all three temperatures as rates of O<sub>2</sub> consumption. The circles show the average metabolic rates; error bars indicate  $\pm 2$  standard deviations of the mean. (After Dawson and Bartholomew 1956.)

that were living *chronically* at 33°C during the weeks before the measurements were made.

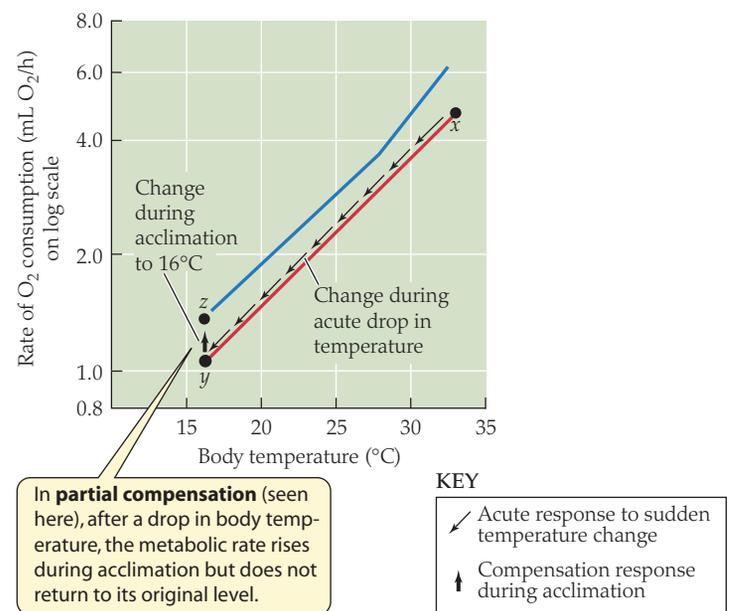
Another group of lizards, called the “16°C-acclimated” group, was maintained for 5 weeks at 16°C. These 16°C-acclimated lizards were a closely matched but different set of individuals from the 33°C-acclimated group; however, physiologists know from other research that if the individuals that had been acclimated to 33°C were themselves later acclimated to 16°C, the results for the 16°C-acclimated group would be the same as shown. After 5 weeks at 16°C, the 16°C-acclimated lizards were exposed acutely to the same three study temperatures employed for the 33°C-acclimated group, and their metabolic rates were measured. The line labeled “16°C-acclimated” in Figure 10.11 shows the results and thus represents the *acute* relation between resting metabolic rate and body temperature for lizards that were living *chronically* at 16°C.

As Figure 10.11 shows, the acute metabolism–temperature relation is altered when lizards have been living chronically at 16°C rather than 33°C. Lizards acclimated to the cooler ambient temperature, 16°C, have a higher average metabolic rate at any given body temperature than those acclimated to the warmer ambient temperature, 33°C. Although this specific sort of change during temperature acclimation is not universal, it is the most common type of acclimation response in poikilotherms and has been observed in well over half the species studied.

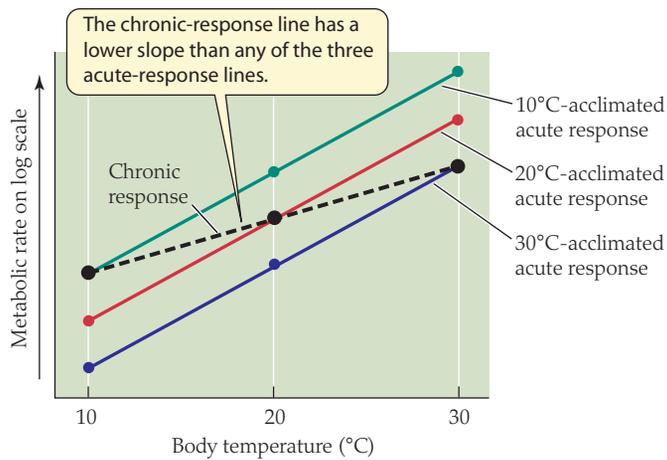
What is the *significance* of this acclimation response? One way to understand the significance is provided by Figure 10.12. As a thought exercise, imagine that we have some lizards that have been living at 33°C for 5 weeks. The average metabolic rate of these lizards—that is, the metabolic rate of 33°C-acclimated lizards at

33°C—is marked *x* in Figure 10.12. Imagine now that we suddenly lower the temperature of these lizards to 16°C and leave the lizards at 16°C for 5 weeks. The key question we need to address is: How will their average metabolic rate change from the moment their temperature is lowered? Let’s begin by considering the *first hour*. In other words, what is the *acute* (prompt) response of the lizards to the change of their temperature? As the animals cool from a body temperature of 33°C to 16°C during the first hour, their average metabolic rate will decline along the acute-response line for 33°C-acclimated animals, following the thin arrows from *x* to *y*. Immediately after the lizards have cooled fully to 16°C, their average metabolic rate will be *y*, the metabolic rate of 33°C-acclimated lizards at 16°C. Note that the drop of body temperature causes a profound fall in metabolic rate. Now we come to the most critical question to answer for understanding acclimation: *What will happen to the average metabolic rate of the lizards during the following 5 weeks at 16°C?* The answer is that the metabolic rate will *rise* from *y* to *z* because during those 5 weeks the lizards will become 16°C-acclimated animals! At the end of the 5 weeks, they will have the metabolic rate of 16°C-acclimated animals at 16°C (*z*). Acclimation in these lizards thus reduces—blunts—the effect of the change of their body temperature. Although cooling to 16°C initially lowers the lizards’ average metabolic rate by a profound amount, the metabolic rate is lowered to a lesser extent after acclimation has occurred. Put another way, acclimation tends to return the metabolic rate toward its level prior to the drop in body temperature (see Figure 10.12).

After a physiological rate has been raised or lowered by an abrupt change in body temperature, any subsequent, long-term tendency



**FIGURE 10.12** Compensation through acclimation This diagram shows one way to interpret the data on acclimation by fence lizards in Figure 10.11 (the blue and red lines in the diagram are carried over from Figure 10.11). If lizards that initially are 33°C-acclimated and living at 33°C are switched abruptly to 16°C and left at 16°C, their metabolic rate changes from *x* to *y* during the first hour as they cool acutely from 33°C to 16°C. Then their metabolic rate rises from *y* to *z* over the next 5 weeks as they become acclimated to 16°C: an example of partial compensation.



**FIGURE 10.13** Because of acclimation, the chronic metabolism–temperature curve is relatively flat compared with the acute metabolism–temperature curves. The three solid lines show the acute relations between metabolic rate and body temperature for hypothetical fish when 10°C-, 20°C-, and 30°C-acclimated; symbols on the lines highlight metabolic rates measured during the acute exposure of each acclimation group to body temperatures of 10, 20, and 30°C. The dashed line shows the relation between metabolic rate and body temperature when the fish live *chronically* at each temperature.

for the rate to return toward its original level even though the new temperature continues is called **compensation**. The rise from  $y$  to  $z$  in Figure 10.12 illustrates compensation. Compensation is **partial** if the rate returns only partially to its original level, as in Figure 10.12. When compensation occurs, it is nearly always partial.

An alternative way to understand the *significance* of the sort of acclimation response we have been discussing is presented in **Figure 10.13**. Fish of a particular species were acclimated to 10°C, 20°C, and 30°C by being kept at the three temperatures for several weeks. The 30°C-acclimated fish were then tested acutely at all three temperatures, resulting in the lowermost solid line in Figure 10.13. Similarly, the 20°C- and 10°C-acclimated fish were tested acutely at the three temperatures. Note that each of the three solid lines is an *acute-response line*: Each shows how the metabolic rate of fish varies when it is measured promptly after changes in their body temperature. Now let's construct the *chronic-response line* for these fish: The chronic-response line will show how metabolic rate varies with temperature when the fish are permitted to live at each temperature for several weeks before their metabolic rate is measured. The three bold, black circles are the metabolic rates of the fish when living chronically at the three temperatures. For instance, the black circle at the left is the metabolic rate at 10°C of fish that have been living at 10°C for several weeks (10°C-acclimated fish), and the black circle at the right is the metabolic rate at 30°C of fish that have been living at 30°C. We obtain the chronic-response line by connecting the three black circles. The chronic-response line has a shallower slope than *any* of the acute-response lines. This means that *if the fish are allowed to acclimate to each temperature before their metabolic rate is measured, their metabolic rate is less affected by changes of body temperature than if they are shifted rapidly from one temperature to another*. Acclimation blunts the response to changes of temperature.

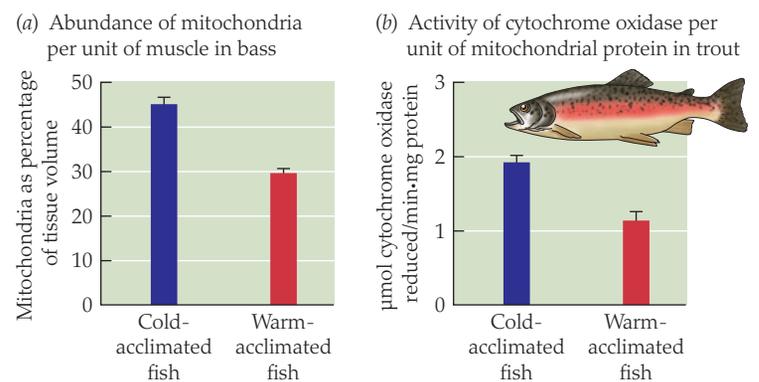
What are the mechanisms of metabolic acclimation? During acclimation, what responses occur in the biochemistry and molecular biology of metabolism? The best understood response

is that cells modify their *amounts* of key, rate-limiting enzymes, notably enzymes of the Krebs cycle and the electron-transport chain. During acclimation to cold temperatures, greater amounts of these enzymes are synthesized.<sup>12</sup> For example, in the red swimming muscles of fish, the number of mitochondria per unit of tissue increases dramatically during cold acclimation in some species (**Figure 10.14a**); the mitochondria, of course, are the sites where the enzymes of the Krebs cycle and electron-transport chain reside and operate. In other species of fish, although the numbers of mitochondria change little, if at all, the amounts of key enzymes per mitochondrion are increased during cold acclimation (**Figure 10.14b**). Responses of these sorts require time; this is one reason why the acclimation response is not observed immediately after a drop in temperature but requires a more extended length of time to be expressed. As the amounts of key, rate-limiting enzymes increase in cells, the presence of the increased enzymes tends to speed metabolic reactions, helping to account for the compensation observed (see Figure 10.12). During acclimation to warm temperatures, enzyme amounts are reduced. Thus, although a shift to a higher body temperature initially speeds an animal's metabolism dramatically, the metabolic rate tends to slow as acclimation occurs (another manifestation of compensation).

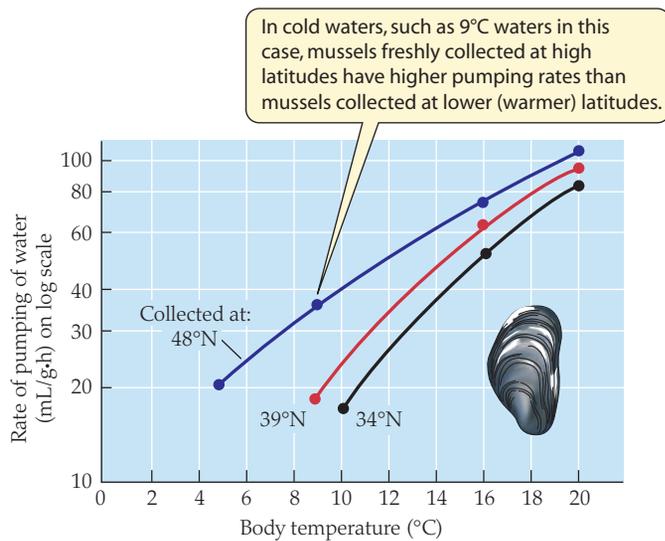
These acclimation responses illustrate in an outstanding way that animals can modify their own cell composition and biochemistry in potentially adaptive ways. In studying biochemistry, it is sometimes easy to get the impression that cells are simply like miniature reaction vessels in which test-tube reactions take place. However, because most reactions must be catalyzed by enzymes to occur and the cells make the enzymes, cells in fact orchestrate their own biochemistry.

A classic study of *acclimatization* by poikilotherms living in their natural habitats (see page 15) was conducted on three groups of

<sup>12</sup> Chapter 2 reviews the effects of enzyme concentration and the processes by which cells modify it.



**FIGURE 10.14** Mitochondrial and biochemical mechanisms of cold acclimation in the red swimming muscles of fish. (a) Striped bass (*Morone saxatilis*) increase the abundance of mitochondria per unit of muscle tissue when acclimated to 5°C (cold-acclimated) rather than 25°C (warm-acclimated). (b) Rainbow trout (*Oncorhynchus mykiss*) increase the activity per unit of mitochondrial protein of the key electron-transport enzyme cytochrome oxidase when acclimated to 5°C (cold-acclimated) rather than 15°C (warm-acclimated). Error bars show  $\pm 1$  standard error. (a after Egginton and Sidell 1989; b after Kraffe et al. 2007.)



**FIGURE 10.15 Acclimatization in mussels** Mussels (bivalve molluscs) are extremely important members of intertidal and subtidal marine communities. They pump water through their bodies by ciliary action. Being suspension feeders (see Chapter 6, page 139), they acquire food as well as  $O_2$  from the water they pump. Mussels of the most abundant West Coast species (*Mytilus californianus*) were collected from nature at the three latitudes indicated. The three lines are acute-response lines for the three groups of mussels, which were acclimatized to different latitudes; symbols along the lines represent actual data, to which the lines were fitted. (After Bullock 1955.)

mussels (believed to be genetically similar) of a single species that were collected at three latitudes along the West Coast of the United States. Each group was acutely exposed to several test temperatures, and the rate at which the animals pumped water across their gills was measured. Because of acclimatization, as seen in Figure 10.15, the populations of mussels living in relatively cold, high-latitude waters and warm, low-latitude waters were more similar to each other in pumping rates than they otherwise would have been.

As a consequence of acclimation and acclimatization, the physiology of an individual animal often depends significantly on its recent individual *history*. This point is important in many ways. For example, when doing research on poikilotherms, investigators need to recognize that the recent histories of the individuals studied may affect the results obtained.

At the present time in the history of biology, a question given emphasis is whether the acclimation and acclimatization responses are always beneficial for animals. Evolutionary physiologists interested in this question stress that a response cannot be assumed to be beneficial just because it occurs (see page 25). The weight of evidence at present is that the responses observed are usually advantageous to the animals, but ever-more-sophisticated insight can be expected in the decades ahead.

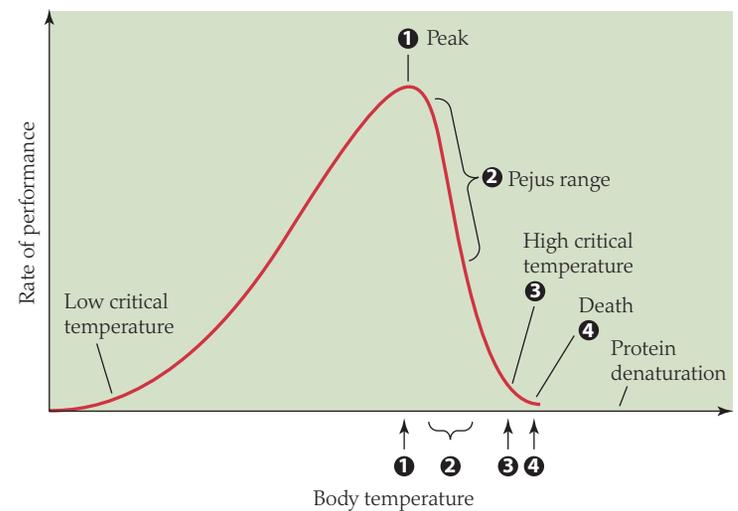
### The rate–temperature relations and thermal limits of individuals: Ecological decline occurs at milder temperatures than acute stress

Animals need to *perform* in a variety of ways to succeed. They need to move, grow, raise their rate of  $O_2$  delivery so they can be active, and so forth. With these points in mind, we can ask how the performance of an individual animal varies with its body temperature. From research on this question, the concept of a generalized, asymmetrical *performance curve*, seen in Figure 10.16a, has emerged.

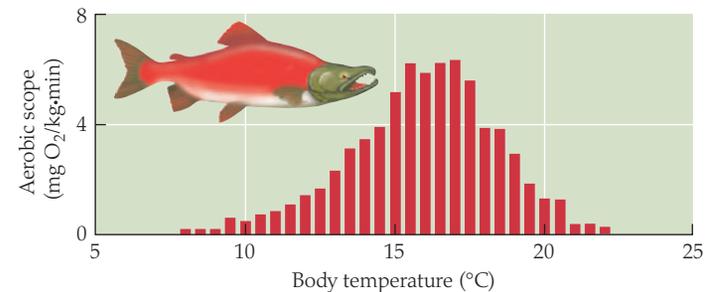
Many types of performance roughly follow a curve of this shape. The rate of performance is low at low body temperature. It increases gradually as body temperature rises, over a relatively wide range of temperatures, up to a certain body temperature where the rate of performance peaks. Then, however, if body temperature goes still higher, the rate of performance declines relatively rapidly, over a relatively narrow range of temperatures, to a low level. In discussing the performance curve, we will focus here mostly on the high-temperature end, because doing so simplifies discussion while still illuminating the most important basic concepts. It is also the end most relevant to understanding the effects of global warming.

If an animal dies because of too-high a body temperature, people often say “it died because of protein denaturation.” Actually, as shown in Figure 10.16a, irreversible protein denaturation

(a) Generalized performance curve



(b) Actual performance curve for aerobic scope in sockeye salmon



**FIGURE 10.16 Performance curves** (a) A generalized performance curve, showing key benchmarks discussed in the text. Numbers along the x axis show body temperatures that cause performance to be at the numbered spots on the performance curve. The term “critical temperature” can mean entirely different things in different contexts; in the study of performance curves, it is the temperature at which the rate of  $O_2$  consumption can barely be raised above the resting rate (but this meaning cannot be extrapolated to other contexts). (b) An example of an actual performance curve: maximal ability to increase rate of  $O_2$  consumption above the resting rate (i.e., aerobic scope) in a population of sockeye salmon (*Oncorhynchus nerka*) during migration (see Figure 17.3). The ability to increase  $O_2$  consumption is highly relevant to the fish in this population because they must generate swimming power to swim up the Fraser River (British Columbia) to reach spawning areas that are more than 1000 km from the sea. (b after Eliason et al. 2011.)

typically occurs only at body temperatures significantly higher than the temperature that kills. When animals die because their body temperature becomes a bit higher than the tolerable temperature, they typically die, with their proteins intact, of causes other than irreversible protein denaturation. What kills them, then? A crisp, general answer is not yet available. Often, however, *performance limitations* are thought to be the answer.

To clarify the significance of performance limitations, we can consider points ❶ to ❹ on the generalized performance curve (Figure 10.16a). When body temperature is at the level associated with ❶, the rate of performance is at its peak. This means that if the type of performance we are studying is elevation of O<sub>2</sub> delivery, the rate of O<sub>2</sub> delivery is highest at ❶; if the performance we are studying is growth, growth is fastest at ❶. If body temperature rises above ❶, performance will shift to the range labeled ❷. European researchers have created a new term—**pejus temperatures**—to refer to the range of body temperatures at ❷. *Pejus* is from Latin and means “turning worse.” If we assume that the highest possible rate of performance is best—that is, if we assume that an animal’s fitness is highest when its capacity to perform is highest<sup>13</sup>—then a rise in body temperature from ❶ to ❷ will place the animal in a weakened (“turning worse”) condition. If we are interested in O<sub>2</sub> delivery, the animal will not be able to deliver O<sub>2</sub> at the rate that is best for its fitness; if we are interested in growth, it will not be able to grow at the rate that is best. If body temperature rises still further to ❸, the animal is still alive, but it is unable to *do* much. Point ❸ marks the body temperature at which an animal’s maximal rate of O<sub>2</sub> consumption is little higher than its resting rate of O<sub>2</sub> consumption. At ❸ the animal is passive, and its survival—if it cannot lower its body temperature—is time-limited. Point ❹ is the temperature at which elevated body temperature is itself directly lethal.

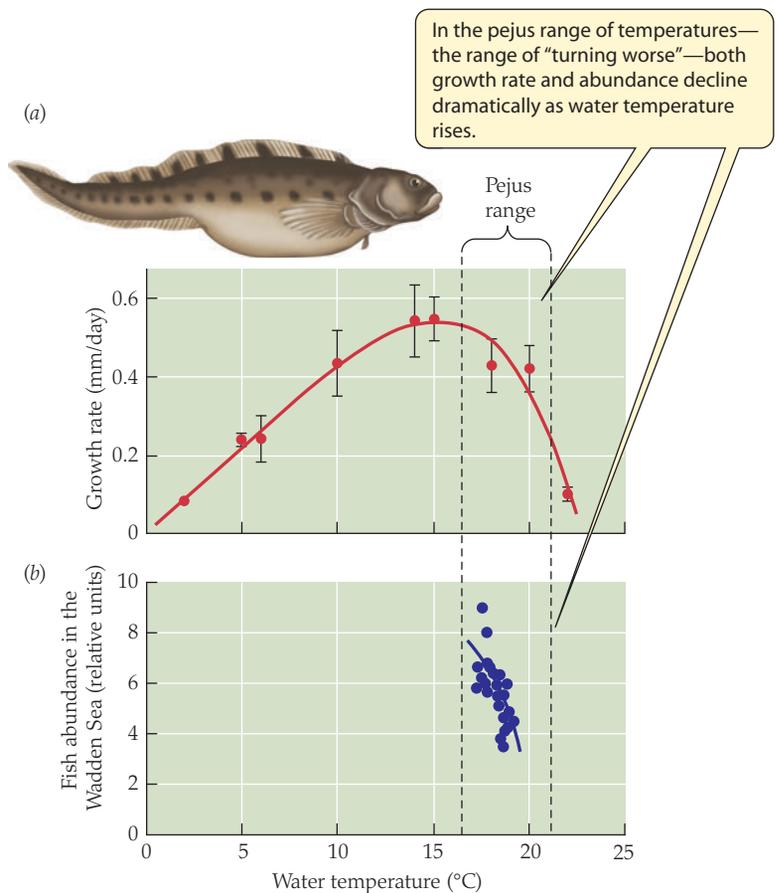
The most important message of this analysis is that, as body temperature rises beyond the point of peak performance, an animal’s circumstances probably usually “turn worse” in subtle ways before the body temperature becomes high enough to render the animal passive or kill it outright. This distinction is believed to explain why animals *living in natural ecological communities* can be weakened—and their populations may even go extinct—at temperatures distinctly lower than lethal temperatures measured *in laboratories*. In a natural ecological community, a capacity for mere survival is often far from adequate. “Turning worse” may reduce an animal’s competitive ability so that it is eliminated by superior competitors, or “turning worse” may impair the animal’s ability to swim or run so that it cannot catch sufficient prey.

Where is the performance curve positioned on the scale of body temperature? Various species differ greatly in this regard. For a terrestrial species that evolved at temperate latitudes, the low and high critical temperatures<sup>14</sup> might be  $-10^{\circ}\text{C}$  and  $+33^{\circ}\text{C}$ . For a terrestrial species that evolved in tropical rainforests, they might be  $+10^{\circ}\text{C}$

and  $+35^{\circ}\text{C}$ . As a specific example, Figure 10.16b shows the range for an aquatic species, the sockeye salmon. The performance curve is a general concept of how the rate of performance varies within each species’ range of body temperatures compatible with life.

Is the performance curve determined acutely or chronically? It can be determined either way, depending on research objectives. A curve based on chronic responses is usually most informative for analyzing animals living in natural ecological communities, because body temperatures usually change slowly in natural settings (in part because of behavioral thermoregulation), permitting animals to express their chronic responses.

A recent study of the common eelpout—a nonmigratory fish—in the Wadden Sea in northern Europe illustrates the sorts of insight that can be gained by interpretation of performance curves. Figure 10.17a shows the eelpouts’ performance curve for growth. One can see that as water temperature rises, the pejus range of “turning worse” starts at  $17^{\circ}\text{C}$ , a temperature  $6^{\circ}\text{C}$  lower than temperatures the fish can tolerate in a laboratory setting! Eelpouts are abundant at water temperatures cooler than  $17^{\circ}\text{C}$ . However, as shown in Figure 10.17b, their abundance declines sharply as the water temperature increases within the pejus range—indicating that “turning worse” has severe



**FIGURE 10.17** Nonlethal water temperatures that suppress growth are also associated with ecological decline in populations of common eelpouts (*Zoarces viviparus*). The fish were studied in the Wadden Sea, where water temperature has risen  $1.1^{\circ}\text{C}$  over the past 40 years. The upper plot is the performance curve for growth: rate of growth as a function of water (= body) temperature. The lower curve shows fish abundance. As water temperature becomes higher in the pejus range for growth, fish abundance plummets. (After Pörtner and Knust 2007.)

<sup>13</sup> Although a peak capacity for performance probably often confers greatest fitness, this is not necessarily true. Some authors call the body temperature at ❶ the *optimal* temperature—an unfortunate practice. The concept that the *highest* rate of performance represents *optimal* performance must be tested empirically in the analysis of each particular case.

<sup>14</sup> Later, when we discuss homeotherms, we will again encounter critical temperatures. Because the term “critical” is used in many different contexts and its meaning sometimes varies, the “critical” temperatures of ectotherms are entirely unrelated to those of homeotherms. Be sure, therefore, to apply the analysis here only to ectotherms.

consequences. Temperatures in the pejus range—although not high enough to kill the fish outright—are associated with *ecological* demise of the fish.

Why are animals impaired at temperatures in the pejus range? In aquatic poikilotherms, O<sub>2</sub> limitation seems to be the most likely general answer. As the ambient water warms, its ability to dissolve O<sub>2</sub> declines (see page 579 yet the metabolic needs of animals for O<sub>2</sub> tend to rise (see Figure 10.9). These clashing trends evidently impair function in subtle ways at temperatures (the pejus temperatures) that are distinctly lower than those that bring about an all-out O<sub>2</sub> crisis. The concept that rising temperatures cause O<sub>2</sub> limitation (see Figure 10.16b), which in turn limits other critical functions, is termed the *theory of oxygen- and capacity-limited thermal tolerance*.

### Evolutionary changes: Species are often specialized to live at their respective body temperatures

Related species of poikilotherms often spend much of their time at different body temperatures. Dramatic examples are provided by animals that live in different geographical regions. For example, species of fish, sponges, and sea urchins living on coral reefs in the tropical oceans (see Figure 1.15) live at tissue temperatures that are 25–30°C higher than those of related species of fish, sponges, and urchins that live in polar seas (see Figure 1.10). As another example, among species of lizards that live in the American West, some differ substantially from others in the behaviorally regulated “preferred” body temperatures they maintain during the daylight hours of each day. Whereas one species might employ behavior to thermoregulate at an average body temperature of 34°C, another coexisting species might thermoregulate at a body temperature of 40°C. A key question for physiologists is whether related species that live and reproduce at different body temperatures have evolved adaptations to their respective temperatures.

Some physiological differences among species living at different body temperatures are so dramatic that there can be no doubt about the existence of evolved, adaptive specializations. For example, certain Antarctic species of molluscs promptly die if their body temperature rises above +2°C, even though other species of molluscs live with great success in tropical oceans. Many Antarctic species of fish thrive at temperatures near freezing and die of *heat* stress when warmed to 4–6°C. Meanwhile, many tropical species of fish thrive at tropical temperatures and die of *cold* stress if cooled to 4–6°C. One can hardly doubt that these Antarctic and tropical species have evolved adaptive specializations to their respective body temperatures. Most differences among species, however, are not so categorical and are more challenging to interpret.

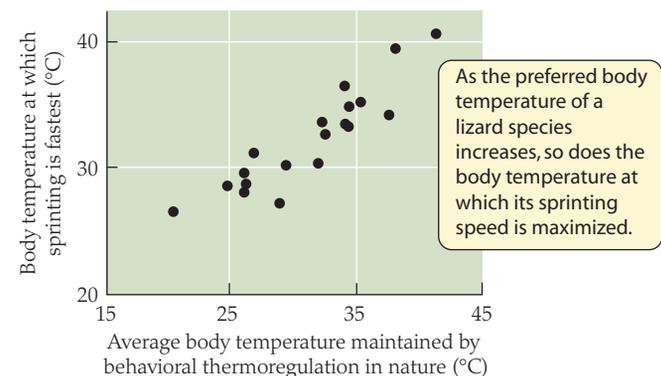
One problem for the study of evolutionary adaptation to temperature is that most animal species cannot be bred in captivity, and therefore individuals must be collected from nature for study. When biologists try to interpret data gathered on wild-caught adults, they must always worry that differences may exist between sets of animals of different species—not because the species differ genetically, but because the study animals underwent their early development under different conditions in their respective natural habitats. Another problem for the study of evolutionary adaptation is that species from thermally different environments are often unable to live successfully at a single temperature; in such cases, biologists cannot do the “obvious” experiment of comparing species in a single laboratory environment. These concerns and

others pose methodological challenges for physiologists working on evolutionary differences among species.

**LIZARD SPECIES WITH DIFFERENT PREFERRED BODY TEMPERATURES** Of what advantage is thermoregulation? A plausible hypothesis is that when a species thermoregulates, its tissues and cells can improve their performance by becoming thermally specialized to function at the body temperatures maintained. In the complete absence of thermoregulation, tissues are equally likely to be at almost any temperature; accordingly, specialization to function at particular temperatures might be disadvantageous. However, if thermoregulation occurs and tissue temperatures are thereby maintained for substantial periods of time in a narrow range, a tissue might profit by becoming specialized (over evolutionary time) to function at temperatures in that range.

Species of lizards with different preferred body temperatures provide excellent models for testing the hypothesis that tissues become specialized to function at the body temperatures maintained by thermoregulation. If the hypothesis is correct, species with relatively high preferred body temperatures should have tissues specialized to function at relatively high temperatures, whereas species with lower preferred temperatures should exhibit tissue specializations to lower temperatures.

Many tissue functions of lizards, when tested, seem in fact to be carried out best in various species when the species are at their respective preferred body temperatures. For example, in species that have preferred body temperatures near 40°C, testicular development at the onset of the breeding season is often most rapid and complete at such high temperatures; in other species that prefer body temperatures near 30°C, the testicles develop optimally near 30°C and are damaged by 40°C. For another example, consider the optimum body temperature for sprint running by various species of lizards. This temperature is well correlated in certain groups of related lizards (but not in all groups) with the respective preferred body temperatures of the species (Figure 10.18). Hearing, digestion,



**FIGURE 10.18** The body temperatures at which 19 species of iguanid lizards are able to sprint fastest correlate well with the behaviorally regulated preferred body temperatures of the species. In each species of lizard, as the body temperature is raised, sprinting speed increases to a certain point, but then if the body temperature is raised further, sprinting speed starts to decline (a typical performance curve). The body temperature at which the sprinting speed is maximized is plotted on the y axis for each of the 19 species. Preferred body temperatures maintained by behavioral thermoregulation in nature are on the x axis. (After Huey and Kingsolver 1993.)

and the response of the immune system to bacterial invasion are just some of the other processes known to take place optimally, in at least certain sets of related species, when body temperatures are at preferred levels. There are exceptions to these patterns, and there are traits that seem in general not to be optimized at preferred temperatures. Nonetheless, the data on lizards indicate that thermoregulation and tissue thermal specialization have often evolved in tandem.

**FISH AND INVERTEBRATES OF POLAR SEAS** Many decades ago, investigators hypothesized that the species of fish and invertebrates in polar seas maintain higher resting and average metabolic rates in cold waters than related temperate-zone or tropical species could maintain in the same waters. Today, most specialists conclude that the hypothesis is correct, at least for certain groups of polar poikilotherms. This conclusion, however, follows 60 years of contentious debate, which continues.

Studies of isolated tissues provide more-certain evidence for evolutionary specialization in polar poikilotherms. For instance, investigators have studied the rate of protein synthesis in isolated fish livers. At near-freezing tissue temperatures, protein synthesis is much more rapid in livers taken from polar species than in those taken from temperate-zone species. Similarly, the skeletal muscles of polar fish are able to generate more mechanical power at polar temperatures than are the muscles of temperate-zone fish; one reason, in at least some cases, is that the polar muscles are richer in mitochondria (and mitochondrial enzymes). These sorts of evidence point to the evolution in polar fish of distinctive physiological properties that permit them to function more vigorously at low body temperatures than unspecialized fish can.

### Temperature and heat matter to animals because they affect the rates of processes and the functional states of molecules

One of the most important reasons to study poikilotherms is that they clarify the fundamental ways in which temperature and heat are significant factors for the tissues of animals. Recall from Chapter 7 that heat energy cannot be used to do work by organisms. If heat cannot do work, why does it even matter?

Temperature and heat are important for animal tissues for two reasons. The first we have already discussed: The temperatures of tissues (which are determined by heat inputs and outputs) *affect the rates of tissue processes*.<sup>15</sup> The second is that the temperatures of tissues *affect the molecular conformations and therefore the functional states of molecules*.

The exact three-dimensional conformation of a protein molecule depends on prevailing temperature because three-dimensional conformation is stabilized by weak, noncovalent bonds—not strong, covalent bonds (see Box 2.1). When the temperature is modified, the various weak bonds in a molecule change in their relative strengths, and thus the molecule assumes a different conformation at each temperature.<sup>16</sup> The *functional properties* of a protein molecule

<sup>15</sup>These rates include metabolic rates, rates of particular biochemical reactions, and rates of biophysical processes such as diffusion and osmosis.

<sup>16</sup>To be more exact, one current view is that a given molecule has multiple microconformations, which it “flickers” among from instant to instant. In this view, changes of temperature alter the probabilities that a molecule will be in its various microconformations. Thus changes of temperature alter the time-averaged conformation of the molecule. From this viewpoint, when we speak of *conformation*, we mean *time-averaged conformation*.

depend on its molecular conformation.<sup>17</sup> With this background in mind, we can understand in principle why the functional properties of protein molecules often vary with the prevailing temperature.

One of the most significant discoveries of the last few decades in the study of comparative physiology is the realization that animals living in different temperature regimes often have evolved *different molecular forms* of proteins: forms that are differentially suited to function in the divergent temperature regimes. **Figure 10.19** provides a dramatic visual illustration of this point. At the left in Figure 10.19b are the freshly dissected eye lenses of three vertebrates that live in different temperature regimes. The eye lens of the cow normally functions at 37°C. The two fish are from coral-reef ecosystems (the soldierfish) and the Antarctic Ocean (the toothfish), and their lenses normally function at 25°C and –2°C, respectively. Ostensibly the three lenses at the left are all the same: All are composed of a type of protein—called crystallin protein—that is perfectly clear. Testing the lenses revealed, however, that they are not the same. When the cow lens and tropical-fish lens were placed at 0°C, they underwent denaturation: a type of protein-conformation change that disrupts normal protein function. As a consequence of the denaturation, instead of being clear, they became opaque (a phenomenon called cold cataract). This sort of change would have blinded the animals! The lens of the Antarctic toothfish, however, exists for a lifetime (up to 30 years) at –2°C without undergoing denaturation; and tests showed that it could be cooled to –12°C without denaturing. In brief, all these vertebrates have crystallin proteins, but they have *different molecular forms* of the proteins: forms differentially suited to the distinct temperatures at which their eye lenses function. This is a theme that is repeated throughout the study of proteins and other macromolecules.

The enzyme–substrate affinity of an enzyme molecule is one of the molecule’s most important functional properties because it determines how readily the molecule is able to form an enzyme–substrate complex (see page 43). The enzyme–substrate affinity, however, is not a fixed property of an enzyme molecule. Instead, it changes as the prevailing temperature is raised and lowered. Biochemists believe that a certain intermediate level of enzyme–substrate affinity is ordinarily ideal. Whereas too low an affinity can render an enzyme molecule incapable of forming complexes with substrate molecules, too high an affinity can make the enzyme molecule so prone to forming complexes with substrate that it becomes uncontrollable by regulatory processes. **Figure 10.20a** illustrates how the enzyme–substrate affinity of one particular enzyme molecule—lactate dehydrogenase (LDH) isolated from the muscles of a goby fish—varies with the prevailing temperature because of reversible, temperature-induced conformational changes in the protein.

Because the functional properties of enzymes depend on the prevailing temperature, any particular enzyme protein can be highly functional at certain tissue temperatures while being only marginally functional (or even nonfunctional) at other tissue temperatures. How, then, can animals living in different thermal regimes all have suitably functional enzymes?

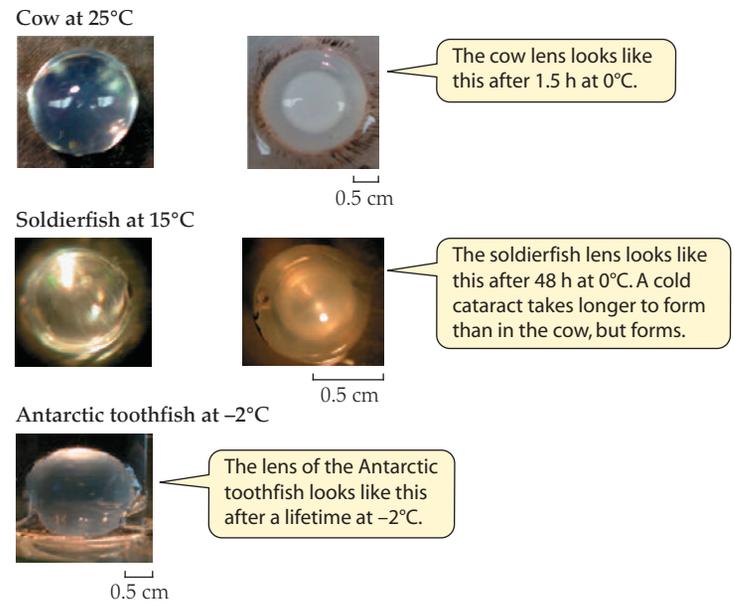
An important part of the answer is that during evolution, species that have different body temperatures have often evolved

<sup>17</sup>This is probably true for several reasons. One important reason is that (as discussed in Chapter 2) a protein molecule often must *flex* (change shape) to carry out its functions, and conformation affects how readily various molecular subregions are able to flex.

(a) An Antarctic toothfish



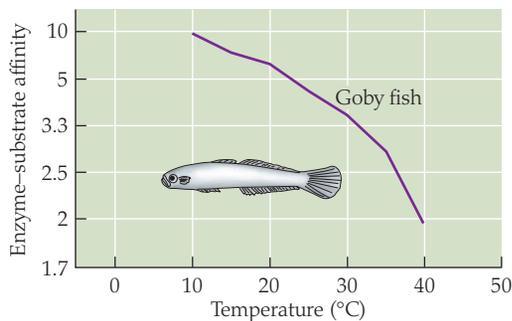
(b) Eye lenses of a cow, a coral-reef soldierfish, and an Antarctic toothfish



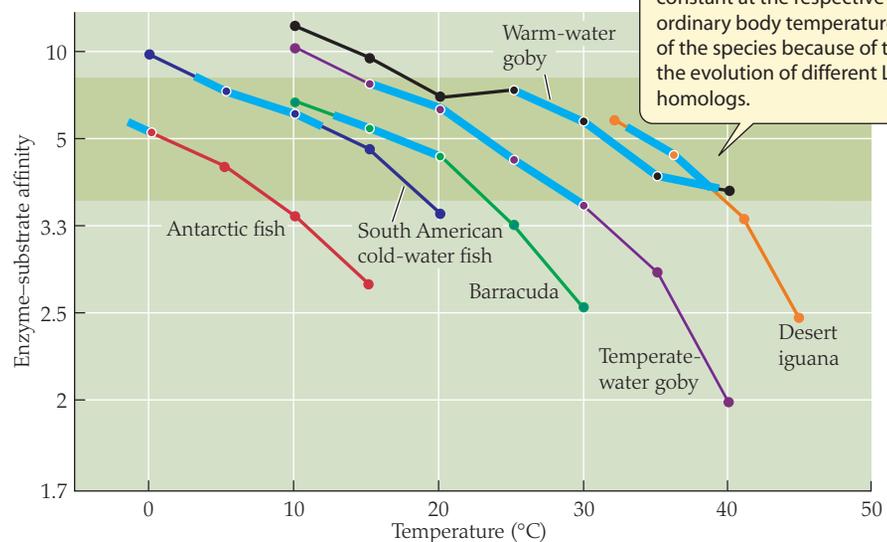
**FIGURE 10.19 Seeing at  $-2^{\circ}\text{C}$  requires specialized eye-lens crystallin proteins** (a) An Antarctic toothfish (*Dissostichus mawsoni*) living at  $-2^{\circ}\text{C}$  in the ocean near Antarctica. Toothfish sometimes live for 30 years, and their eye lenses remain crystal clear throughout. (b) At the left are normal eye lenses taken from three species: a cow, a coral-reef fish called the blackbar soldierfish (*Myripristis jacobus*), and the Antarctic

toothfish. In life, the lenses of these three species function at about  $37^{\circ}\text{C}$ ,  $25^{\circ}\text{C}$ , and  $-2^{\circ}\text{C}$ , respectively. Note at the right in (b) that the lenses of the cow and soldierfish develop cold cataracts—which would blind the animals—with only short-term exposure to  $0^{\circ}\text{C}$ . (Photographs in b courtesy of Andor Kiss and C.-H. Christina Cheng; photographs from Kiss et al. 2004.)

(a) Enzyme–substrate affinity as a function of temperature in a goby



(b) Enzyme–substrate affinity as a function of temperature in six species of poikilotherms



All the blue line segments, which identify the ordinary body temperatures of the species, fall within the narrow vertical distance marked by the shaded band. Thus, affinity for substrate is kept relatively constant at the respective ordinary body temperatures of the species because of the evolution of different LDH homologs.

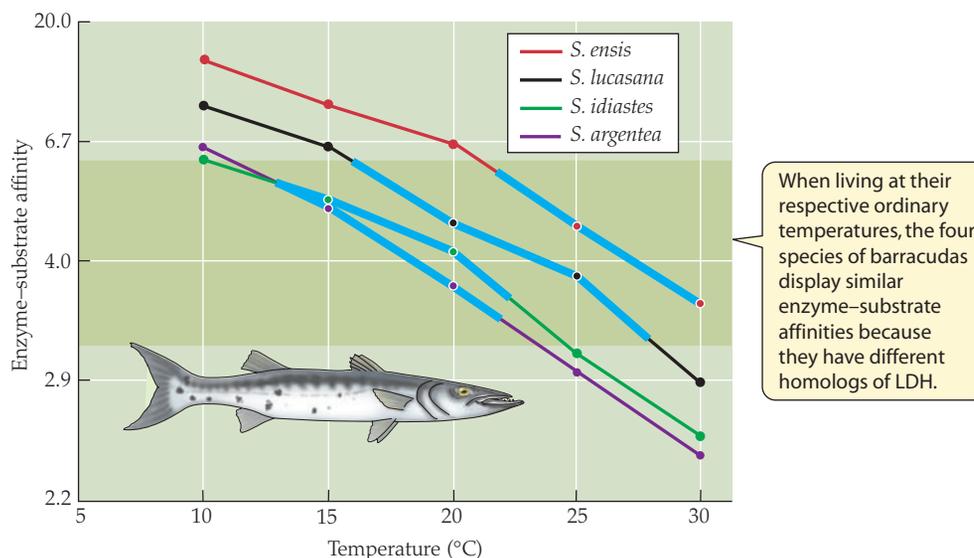
**FIGURE 10.20 The affinity of the enzyme lactate dehydrogenase (LDH) for substrate as a function of temperature** This relation is shown in (a) for a goby fish (*Gillichthys mirabilis*) and in (b) for six species of poikilotherms—five fish and a desert lizard—that ordinarily live at different body temperatures. The blue, thickened portion of each line identifies the range of body temperatures ordinarily experienced by the species. The enzyme–substrate affinity shown in both plots is the affinity

of muscle LDH ( $\text{LDH-A}_4$ ) for pyruvic acid. Affinity is expressed as the inverse of the apparent Michaelis constant ( $mM$  pyruvate); see Chapter 2 (page 43) for background. The Antarctic fish is an Antarctic notothenioid; the South American fish is also a notothenioid; the barracuda is *Sphyaena idiaestes*; the temperate-zone goby is *Gillichthys mirabilis*; the warm-water goby is *Gillichthys seta*; and the desert iguana is *Dipsosaurus dorsalis* (see Figure 1.12). (After Hochachka and Somero 2002.)

different molecular forms of enzyme proteins. Not all species of vertebrates, for instance, have the same molecular form of LDH that the goby fish in Figure 10.20a has. If they did, species that ordinarily have low body temperatures would routinely have far higher enzyme–substrate affinities than species that have high body temperatures. Instead, as Figure 10.20b shows, different species have evolved different molecular forms of LDH. The six species of poikilotherms shown in Figure 10.20b, some of which ordinarily live at very different body temperatures than others, have six different (although homologous) LDH proteins.<sup>18</sup> Although all six LDH proteins catalyze the same reaction, they differ in their detailed structures and functional properties, so the six exhibit different relations between enzyme–substrate affinity and temperature. The line for each species in Figure 10.20b is thickened and colored blue at the temperatures that correspond to the usual body temperatures of the species. For example, the line for the warm-water goby is thickened and colored blue at temperatures between 25°C and 40°C because that species of fish ordinarily has body temperatures of 25–40°C, and the line for the Antarctic fish is thickened and colored blue at temperatures near –1°C because that species ordinarily has a body temperature near –1°C. All the blue, thickened segments are at about the same height on the y axis. Specifically, all fall within the vertical distance marked by the shaded band. What this shows is that *all six species have about the same enzyme–substrate affinity when they are at their respective body temperatures*. The way they have achieved this remarkable condition, even though they live at body temperatures as much as 40°C apart, is by having evolved different molecular forms (homologs) of the enzyme.

The conservation of enzyme–substrate affinity by the evolution of enzyme homologs that are adapted to function best at different temperatures is very common. One of the most striking and instructive examples is provided by the four species of barracudas in Figure 10.21. These four species, all closely related evolutionarily, behaviorally elect to live in waters that are just modestly different in temperature. For example, the waters occupied by *Sphyrna lucasana* average just 3–4°C warmer than those occupied by *S. argentea*, and those occupied by *S. ensis* average just 3–4°C warmer yet. Even these relatively small differences in habitat temperature (and body temperature) have led to the evolution of different molecular forms of the LDH protein. Consequently, the four species all have similar enzyme–substrate affinities when living at their respective (and different) temperatures.

Earlier we noted that there are two major ways in which temperature and heat matter for animals. The second of those ways should now be clear enough that we can state it succinctly: *Particular enzyme molecules (and other sorts of protein molecules) are typically specialized to function best within certain temperature ranges. The protein molecules therefore require certain temperatures to function optimally.* With few



**FIGURE 10.21 Enzyme adaptation in four species of barracudas** The four species, all in the genus *Sphyrna*, live at somewhat different temperatures. The enzyme studied is LDH. All details are the same as in Figure 10.20b. (After Somero 1997.)

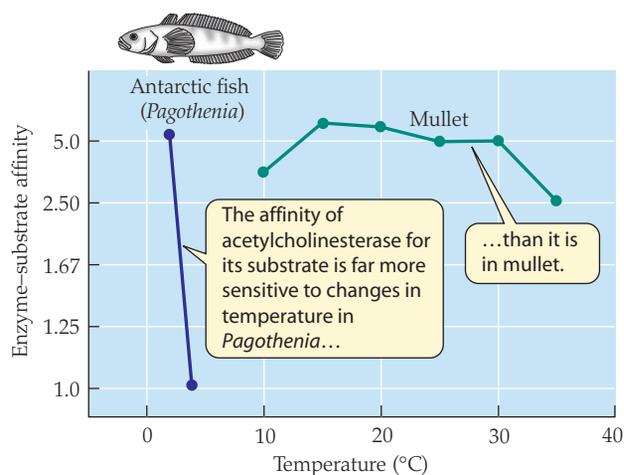
known exceptions, the tissues of the adults of any particular species are fixed in the homolog of each enzyme they synthesize; although a tissue may change the *amount* of the enzyme it synthesizes (as often occurs during acclimation or acclimatization), it cannot change the *type* of enzyme.<sup>19</sup> Thus individuals of a species of fish (or other aquatic poikilotherm) ordinarily found in warm waters typically *require* warm tissue temperatures for their enzyme molecules to have ideal functional forms. Conversely, individuals of a cold-water species of fish typically *require* cold tissue temperatures for their particular types of enzyme molecules to have ideal functional forms. The same principles apply to homeotherms. For instance, the LDH of cows needs to be at about 37°C to have an appropriate enzyme–substrate affinity, just as the crystallin proteins of cows need to be warm to be clear. Certain tissue temperatures, in brief, are crucial because the conformations and functional properties of proteins are not deterministically set by the chemical compositions of the proteins but depend as well on the prevailing temperature.

**IMPLICATIONS FOR GLOBAL WARMING** A key question in the study of global warming is *how much* the tissue temperature of animals must change for the change to have significant consequences. Data such as those on the barracudas (see Figure 10.21) suggest that the answer is sometimes “not very much.” The species of barracudas, which live in waters differing by 3–4°C and have evolved different enzyme homologs, appear to be telling us that a 3–4°C change in body temperature is sufficiently consequential that natural selection favors the evolution of new molecular variants of key enzymes. A worry about human-induced global warming is that it may occur so rapidly that evolution will not immediately “keep up,” and many poikilotherms may be forced to function for years with nonoptimized molecular systems.

In situations in which tissue temperatures are different from ideal, an important question is the steepness of the relation between molecular functional properties and temper-

<sup>18</sup>These are *interspecific enzyme homologs*. See page 46 in Chapter 2 for an extensive discussion of both LDH and the concepts of protein homology.

<sup>19</sup>As discussed later in the chapter, this statement does not necessarily apply to other proteins.



**FIGURE 10.22 An enzyme that shows extreme sensitivity to temperature change** The plot shows the affinity of brain acetylcholinesterase for acetylcholine in a stenothermal, polar fish (*Pagothenia borchgrevinki*, pictured) and a eurythermal, warm-water species of fish, a mullet. Because acetylcholinesterase is a lipoprotein enzyme, lipid moieties may be involved in interspecific differences. Affinity is expressed as the inverse of the apparent Michaelis constant ( $mM$  acetylcholine). (After Somero 1997.)

ature. A classic example of a very steep relation is provided by the acetylcholinesterase homolog found in the brains of *Pagothenia borchgrevinki*, a red-blooded Antarctic fish (Figure 10.22). Acetylcholinesterase is essential for brain function because it keeps the neurotransmitter acetylcholine from building up excessively at synapses (see page 343). The enzyme–substrate affinity of *Pagothenia*'s acetylcholinesterase is exceptionally sensitive to changes of temperature, so much so that the enzyme undergoes functional collapse—it loses almost all affinity for its substrate—when warmed to 5–10°C. An enzyme form of this sort would cause any species possessing it to be unusually vulnerable to climate change. It also probably helps explain why *Pagothenia* is one of the most stenothermal fish known; it ordinarily lives its entire life at temperatures near –2°C and dies of heat stress at +4–6°C.

**MORE ON ENZYME ADAPTATION TO TEMPERATURE** Besides enzyme–substrate affinity, another critical functional property of enzymes is the *catalytic rate constant*,  $k_{cat}$ , which measures the number of substrate molecules that an enzyme molecule is capable of converting to product per unit of time. If the  $k_{cat}$  of a particular type of enzyme, such as LDH, is measured under fixed conditions, the usual pattern is that homologs of the enzyme from cold-water species tend to exhibit higher  $k_{cat}$  values than homologs from related warm-water species. Thus the enzyme homologs of the species living in cold waters have a greater intrinsic ability to speed reactions, an attribute that in nature helps offset the reaction-slowing effects of low temperatures. An example is provided by the four barracuda species shown in Figure 10.21. The  $k_{cat}$  of their LDH enzymes, measured at a fixed study temperature, increases as the temperature of their habitat decreases.<sup>20</sup> The  $k_{cat}$ s of LDH forms in Antarctic fish are four to five times higher than the  $k_{cat}$ s of LDH forms in mammals.

<sup>20</sup> Enzyme–substrate affinity and  $k_{cat}$  tend to coevolve because of molecular structural reasons that are only starting to become clear. Thus the evolution of particular interspecific patterns in  $k_{cat}$  is not entirely independent of the evolution of particular patterns in enzyme–substrate affinity.

An important question from the viewpoint of evolutionary biochemistry is how much the amino acid composition of an enzyme must change for the enzyme to take on new functional properties. One of the most interesting studies on this question also involved the barracudas. Using modern sequencing techniques, researchers found that in the LDH protein—which consists of about 330 amino acid units—four amino acids at most are changed from one barracuda species to another; only one amino acid is different between some of the species. Of equally great interest, none of the changes in amino acid composition in these LDH homologs is at the substrate-binding site; the changes, therefore, affect the function of the enzyme by altering properties such as molecular flexibility, not the properties of the catalytic site per se. These characteristics, exemplified by the barracudas, are emerging as important generalizations: (1) Homologous enzyme molecules often differ in only a relatively few amino acid positions—helping explain how species can readily evolve adaptively different enzyme homologs; and (2) the altered amino acid positions are located outside the substrate-binding site, so the substrate-binding site is constant or near-constant, explaining why all homologs catalyze the same reaction.

#### MYOSIN ISOFORMS EXEMPLIFY THAT PROTEIN ISOFORMS SOMETIMES CHANGE DURING ACCLIMATION AND ACCLIMATIZATION

Muscle contractile function depends on a variety of proteins, such as myosin, troponin, and actin (see Chapter 20). An individual of any particular species is typically capable of synthesizing multiple molecular forms of each of these proteins, providing the basis for a wide range of phenotypic plasticity in muscle function. The various molecular forms of a particular protein that can be synthesized by a species are termed *isoforms* of the protein—a term with similar meaning to *isozymes* (see page 46) but preferred in this context because not all muscle proteins have enzymatic activity. How is it possible for an individual to synthesize multiple forms of one protein? One reason is that multiple genes in the gene family coding for the protein are present in one individual.

The isoforms of the *myosin heavy-chain* protein in fish fast muscle provide an elegant and instructive example of the importance of the properties just outlined. This protein plays a central role in muscle contraction (see Chapter 20) and thus is critical for swimming—one of the most important of all activities of a fish. After individual carp (*Cyprinus carpio*) or goldfish (*Carassius auratus*) that have been living in warm water are switched to cold water, they initially cannot swim particularly fast, but over several weeks, they exhibit increasing swimming performance in the cold water. A change in isoforms of the myosin heavy-chain protein is a key part of this acclimation. That is, according to available evidence, the muscle cells of the fish synthesize isoforms in altered proportions, and they switch out old isoforms for new ones in the contractile apparatus! Although the proteins in thoroughly cold-acclimated individuals are relatively unstable if subjected to warm temperatures, at cold temperatures they have molecular properties that enhance contractile performance. The change in isoforms is thus a key reason that the swimming performance of the fish increases as cold acclimation takes place. Whereas the enzymes of glycolysis and the Krebs cycle that have been so thoroughly studied (see the preceding sections of this chapter) typically are adjusted only in *amount*, not in *type*, during acclimation, the myosin isoforms illustrate that some proteins undergo isoform changes.

**LIPIDS AND HOMEOVISCIOUS ADAPTATION** As is true of proteins, the functional properties of lipids depend on the prevailing temperature as well as the chemical compositions of the molecules. One of the most important functional properties of lipids is the fluidity of the phospholipids in cell membranes and intracellular membranes. As stressed in Chapter 2 (see page 33), individual phospholipid molecules—and protein molecules embedded in the phospholipid matrix—diffuse from place to place within the leaflets of cell membranes and intracellular membranes, and this mobility is exceedingly important for membrane function. **Membrane fluidity** is a measure of how readily the phospholipid molecules in a membrane move.

**Figure 10.23** depicts membrane fluidity as a function of temperature for membrane lipids extracted from the brains of nine vertebrate species—seven fish from a broad range of habitats, a mammal, and a bird. If you focus on any *particular* species, you will note that fluidity is a regular function of the prevailing temperature. Fluidity increases as temperature increases, much as any particular household lipid, such as butter, becomes more fluid as it is warmed.

When different species of animals are taken from their natural habitats and analyzed, they typically differ in the compositions of their membrane phospholipids. Consequently, as can be seen when all nine species in Figure 10.23 are compared, species differ in the details of their relations between membrane fluidity and temperature. The line for each species is thickened and colored blue at temperatures that correspond to the usual body temperatures of the species. As in Figures 10.20*b* and 10.21, the blue, thickened line segments all fall within a narrow range on the *y* axis, marked by the shaded band. In this case, the meaning of this result is that *all nine species have about the same membrane fluidity when they are living at their respective, normal body temperatures*. Such maintenance of a

relatively constant membrane fluidity regardless of tissue temperature is called **homeoviscous adaptation** (*homeoviscous*, “steady viscosity”).

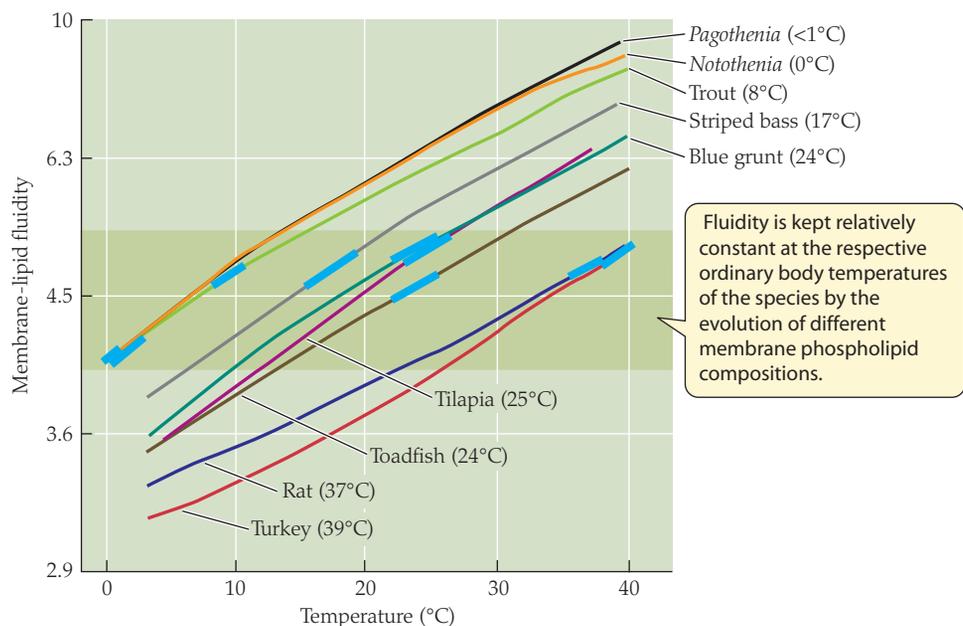
Homeoviscous adaptation is possible because the chemical composition of membrane phospholipids is not fixed but instead can differ among species. If all animal species had the same membrane phospholipid composition, the species with high body temperatures would have very fluid membranes, whereas those with low body temperatures would have stiff membranes. In reality, all have about the same membrane fluidity because species that have evolved to operate at different body temperatures have also evolved systematically different phospholipid compositions. As we saw in studying proteins, again this means that tissue temperature is critical because it must be “matched” to the particular molecules present: *A tissue in which cell membranes are built of particular phospholipids will have the “correct” membrane fluidity only if its temperature is correct.*

The best-understood chemical basis for homeoviscous adaptation is modification of the number of double bonds in the fatty acid tails of the membrane phospholipids. Double bonds create bends in the fatty acid tails (see Figure 2.2*a*), and these bends interfere with close packing of the tails in a membrane. Thus membrane fluidity tends to increase as the number of double bonds increases—that is, as the lipids become more chemically unsaturated. We saw in Chapter 2 (see Figure 2.3) that among fish species native to different thermal environments, the degree of unsaturation of brain phospholipids increases as habitat temperature decreases: Whereas polar species have highly unsaturated lipids that, because of their chemical structure, remain reasonably fluid at polar temperatures, tropical species have much more saturated lipids that, because of their chemical structure, resist becoming too fluid at tropical temperatures.

Individual animals are able to alter the membrane phospholipids that they synthesize: Phospholipid composition is phenotypically plastic (often greatly so). The phospholipid composition of cell membranes and intracellular membranes is commonly restructured during acclimation and acclimatization in ways that promote homeoviscous adaptation. The restructuring of membrane phospholipids by an individual exposed to a chronically changed temperature typically requires many days or more. However, some fish in desert ponds undergo substantial phospholipid restructuring on a day–night cycle, thereby keeping membrane fluidity relatively constant even as the ponds heat up during the day and cool at night.

### Poikilotherms threatened with freezing: They may survive by preventing freezing or by tolerating it

If poikilotherms are exposed to temperatures even slightly colder than those necessary to freeze water, they face a threat of freezing. A classic example of this threat is provided by barnacles, mussels, and other animals attached to rocks along the seacoast. In places such as Labrador in the winter, when the tide is out, such animals may be exposed to extremely cold air and become visibly encased in ice (Figure 10.24). In this way, they themselves may freeze. Animal body fluids



**FIGURE 10.23** The fluidity of lipid-bilayer membranes from brain tissue as a function of temperature This relation is shown for nine species of vertebrates—seven fish that ordinarily live at different body temperatures (see temperatures listed after each species name), a mammal, and a bird. The blue, thickened portion of each line marks the body temperatures ordinarily experienced by the species. Fluidity is measured in terms of the mobility of a molecular probe, to which the units of measure refer. (After Hochachka and Somero 2002.)



**FIGURE 10.24** Barnacles encased in ice during low tide along a northern seacoast. The animals—glued to rocks and unable to flee when exposed to frigid air at low tide—face a threat of freezing. They have met the threat, not by preventing freezing, but by evolving an ability to tolerate—and thereby survive—extensive freezing of their body fluids, as discussed later in the text.

have lower freezing points than pure water because the freezing point is ordinarily a colligative property and becomes lower as the total concentration of dissolved matter increases.<sup>21</sup> Because of their solute content, animal body fluids have lowered freezing points. Nonetheless, they typically freeze at  $-0.1^{\circ}\text{C}$  to  $-1.9^{\circ}\text{C}$  (depending on the animal group) unless they are specially protected.

**THE FREEZING PROCESS IN SOLUTIONS AND TISSUES** To understand the threat of freezing and the possible strategies that animals might use to avoid freezing damage, the first step is to examine the freezing process. An important and seemingly strange point to mention at the outset is that when aqueous solutions are progressively cooled, they commonly remain unfrozen even when their temperatures have fallen below their freezing points, a phenomenon called **supercooling**. Supercooling is an intrinsically unstable state, and a supercooled solution can spontaneously freeze at any moment. Nonetheless, solutions in the supercooled state sometimes remain supercooled for great lengths of time.

An important determinant of a supercooled solution's likelihood of freezing is its *extent* of supercooling; freezing becomes more likely as the solution's temperature drops further below its freezing point. If the temperature of a supercooled solution is gradually lowered while the solution is not otherwise perturbed, a temperature is reached at which the likelihood of freezing becomes so great that the solution spontaneously freezes within a short time. This temperature is called the **supercooling point** of the solution.

Exposure to ice *induces* freezing in a supercooled solution. This has two important implications. First, an unfrozen but supercooled solution immediately freezes if it is seeded with even just a tiny ice crystal, regardless of its extent of supercooling. Second, if a solution,

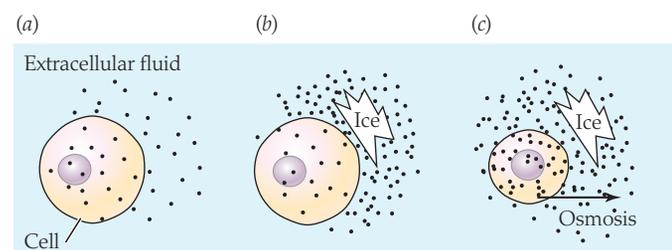
initially at  $0^{\circ}\text{C}$ , is gradually cooled in the presence of an ice crystal, the crystal will prevent supercooling.

An important application of this second point is that the cooling of a solution in the presence of an ice crystal permits determination of the solution's **freezing point**. The freezing point is the temperature above which a solution cannot freeze and below which it deterministically freezes in the presence of preexisting ice. A solution's freezing point is typically a colligative property, depending on the concentration of dissolved entities (see page 117). If a frozen solution is gradually warmed, its **melting point** is the lowest temperature at which melting occurs. The freezing point and the melting point are usually equal.

In tissues, additional complexity is involved in understanding freezing because the *location* of freezing is an important factor. Under natural conditions, freezing *within cells* (*intracellular freezing*) almost always kills the cells in which it occurs. Intracellular freezing is thus fatal for animals unless they can survive without the cells that are frozen. However, many animals are remarkably tolerant of widespread ice formation in their *extracellular* body fluids. This tolerance of extracellular freezing is significant because, for reasons only partly understood, when freezing occurs in an animal, the formation of ice often begins in the extracellular fluids and thereafter tends to remain limited to the extracellular fluids.

To understand the implications and dangers of extracellular freezing more thoroughly, we need to look at the *process* of extracellular ice formation (Figure 10.25). An important attribute of the slow freezing of a solution is that water tends to freeze out of the solution in relatively pure form. Thus, when ice crystals form in extracellular fluid, solutes (excluded from the ice crystals) tend to accumulate in the portion of the extracellular fluid that remains unfrozen, raising the total solute concentration of the unfrozen fluid (see Figure 10.25*b*). The freezing point of the unfrozen fluid is lowered by the increase in its solute concentration. Thus, at a fixed temperature, the formation of ice in extracellular fluid is a *self-limiting* process: Water freezes out of the extracellular fluid only until the freezing point of the unfrozen fluid becomes low enough to equal the prevailing temperature.

The intracellular and extracellular fluids have similar osmotic pressures in an unfrozen animal, meaning that water has little or no tendency to enter or leave cells by osmosis. This benign state is



**FIGURE 10.25** The process of extracellular freezing in a tissue. Dots represent solute molecules. (a) The unfrozen condition. The intracellular fluid inside the cell and the surrounding extracellular fluid have the same osmotic pressure. (b) Slow extracellular freezing produces ice that consists of virtually pure water. Solutes excluded from the ice elevate the osmotic pressure of the unfrozen extracellular fluid. (c) Because of the difference in osmotic pressure created in part (b), water leaves the cell by osmosis, shrinking the cell and raising the osmotic pressure of the intracellular fluid.

<sup>21</sup> See Chapter 5 (see page 117) for a discussion of the colligative properties of solutions, including the freezing point.

disrupted by freezing in an animal's extracellular fluids. Immediately after extracellular ice forms, the unfrozen extracellular fluids are osmotically more concentrated than the intracellular fluids (see Figure 10.25*b*). Thus the ice formation leads to the osmotic loss of water from cells (see Figure 10.25*c*). This loss of intracellular water is itself self-limiting; it stops after the intracellular osmotic pressure has risen to equal the extracellular osmotic pressure. Within limits, the osmotic loss of water from cells is protective: By concentrating the intracellular fluids and thus lowering the intracellular freezing point, the loss of water from cells helps prevent intracellular freezing, which usually is fatal.

**THE ADAPTIVE RESPONSES OF ANIMALS TO FREEZING CONDITIONS: INTRODUCTION** Many poikilotherms behaviorally avoid environments where freezing conditions prevail. For example, many species of frogs, turtles, and crayfish move to the bottoms of lakes and ponds during winter. This location is a safe microhabitat because lakes and ponds do not normally freeze to the bottom.

Many poikilotherms, in contrast, are actually exposed to freezing conditions and must cope physiologically. The mechanisms by which they do so are classified into three types: (1) production of antifreeze compounds, (2) supercooling, and (3) tolerance of freezing. Antifreeze production and supercooling are mechanisms of *preventing* freezing. Usually, species that employ antifreezes and supercooling are **freezing-intolerant**; they die if they freeze and thus are absolutely dependent on successful prevention. By contrast, some species are **freezing-tolerant**; they have evolved the ability to survive extensive freezing of extracellular body water and typically respond to freezing conditions by freezing. It remains largely a mystery why some species have evolved along lines of freezing intolerance, whereas others, sometimes closely related, have evolved toward freezing tolerance.

**PRODUCTION OF ANTIFREEZE COMPOUNDS** Many animals gain protection against freezing by synthesizing **antifreeze compounds**, defined to be dissolved substances that are added to the body fluids specifically to lower the freezing point of the body fluids. Two types of physiologically produced antifreezes are recognized.

1. *Colligative antifreezes*. Some antifreezes lower the freezing point of the body fluids strictly by colligative principles: They affect the freezing point by increasing the total concentration of solutes in the body fluids, not by virtue of their particular chemical properties. The most common of these colligative antifreezes are polyhydric alcohols, especially glycerol, sorbitol, and mannitol.
2. *Noncolligative antifreezes*. Some antifreezes lower the freezing point of the body fluids because of specialized chemical properties. Certain proteins and glycoproteins produced by a variety of insects and marine fish are the best-understood antifreezes of this sort. They are believed to act by binding (through weak bonds such as hydrogen bonds) to nascent ice crystals in geometrically specific ways, thereby suppressing growth of ice by preventing water molecules from freely joining any crystals that start to form. The noncolligative antifreezes can be quite dilute and yet highly effective because they depress the freezing point hundreds of times more than can be accounted

for by simple colligative principles. The noncolligative antifreezes, however, do not depress the melting point any more than colligative principles explain. Thus solutions containing these antifreezes exhibit the unusual property—termed **thermal hysteresis**—that their freezing points are substantially lower than their melting points. The noncolligative antifreezes are usually called **thermal hysteresis proteins (THPs)** or **antifreeze proteins**.

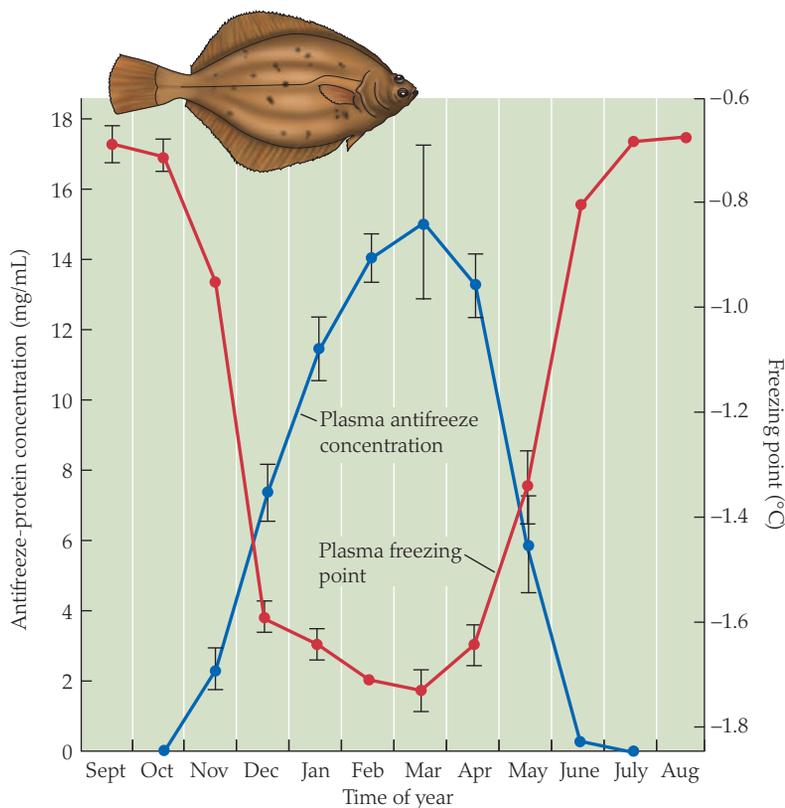
Antifreezes are synthesized principally by certain species in two sets of animals: the marine teleost fish (bony fish) and the insects. The marine teleost fish, in comparison with most other aquatic animals, face unique problems of freezing because their body fluids are osmotically more dilute than seawater (see page 725).<sup>22</sup> Specifically, marine teleosts have blood and other body fluids that—without special protection—freeze at temperatures of  $-0.6^{\circ}\text{C}$  to  $-1.1^{\circ}\text{C}$ . Seawater, being more concentrated, has a lower freezing point:  $-1.9^{\circ}\text{C}$ . Marine teleost fish therefore can potentially freeze even when they are swimming about in unfrozen seas!

A great many of the marine teleost species that live at polar and subpolar latitudes prevent freezing by synthesizing antifreeze proteins. These proteins are found in the blood and most other extracellular fluids of the fish. Although some polar species maintain high antifreeze-protein concentrations in all months, most species synthesize antifreeze proteins just in the cold seasons. The winter flounder (*Pleuronectes americanus*) is one of the best-known antifreeze-producing species. It has an unusually large number of copies of the gene for antifreeze-protein synthesis (about 100), and these gene copies are transcribed and translated in an anticipatory way as winter approaches (Figure 10.26), under photoperiodic control.

Antifreezes are also found in the body fluids of many species of insects during winter. Colligative antifreezes such as glycerol, sorbitol, and mannitol are common and sometimes accumulate to impressive levels; in extreme cases, 15–25% of an insect's overwintering body weight consists of glycerol and other polyhydric alcohols. Antifreeze proteins—noncolligative antifreezes—are also known in many insects, probably having evolved independently several times. When insects are devoid of antifreezes, they have freezing points higher than  $-1^{\circ}\text{C}$ . With high levels of antifreeze solutes, however, insects may exhibit freezing points as low as  $-10^{\circ}\text{C}$  or, in one known case,  $-19^{\circ}\text{C}$ . Even freezing points this low, however, may be well above temperatures that insects encounter in many terrestrial environments. Prevention of freezing in freezing-intolerant insects is thus often a result of both antifreezes and supercooling working in concert. Both polyhydric alcohols and antifreeze proteins promote supercooling and may have other favorable effects in addition to their antifreeze effects.

**SUPERCOOLING** Supercooling is a perfectly ordinary, commonplace phenomenon in both the inanimate and animate worlds; animals do not *cause* themselves to supercool. However, animals

<sup>22</sup> Marine invertebrates generally have body fluids that are as concentrated as seawater. Their freezing points thus match the freezing point of seawater, and—when they are immersed in seawater—they are not threatened with freezing unless the seawater itself freezes. Freshwater animals of all kinds have body fluids that are more concentrated than freshwater. Thus their freezing points are below the freezing point of freshwater, and—when they are immersed in freshwater—they also do not freeze unless the water in which they are living freezes.



**FIGURE 10.26** Seasonal changes in antifreeze protection in winter flounder (*Pleuronectes americanus*) The concentration of antifreeze protein in the blood plasma (blue line) rises as winter approaches, because of increased expression of the genes coding for antifreeze protein. The freezing point of the plasma is synchronously lowered (red line) and in winter is below the lowest winter temperatures the fish experience, ensuring protection against freezing. The winter flounder—an important commercial species—is named for the fact that it spawns in frigid waters in late winter or early spring. (After Fletcher et al. 1998.)

can modify their probabilities of spontaneous freezing during supercooling. Many animals, in fact, undergo adjustments whereby they exhibit low probabilities of spontaneous freezing even when they are supercooled to temperatures far below their freezing points. At one level of understanding, animals achieve this result by altering the quality or quantity of **ice-nucleating agents** in their bodies. Ice-nucleating agents are dissolved or undissolved substances that act as foci for the initiation of freezing. An animal containing an abundance of ice-nucleating agents may freeze when its body temperature is barely below the freezing point of its body fluids. In contrast, an animal that has substantially cleansed its body of ice-nucleating agents may have a supercooling point that is far below its freezing point.

Extensive supercooling is the principal means by which the overwintering life stages of many freezing-intolerant species of insects survive, and it is among these insects that the greatest known capacities to supercool are found. An ability to supercool to 20–25°C below the freezing point of the body fluids—and remain unfrozen because of supercooling for prolonged periods of time—is about average for such insects, and prolonged supercooling to 30–35°C below the freezing point is not uncommon. At the extreme, there are now several known examples of insects that remain unfrozen at –50°C to –65°C by virtue of extensive supercooling, combined with antifreeze depression of their freezing points. These species

can overwinter, unfrozen, in exposed microhabitats, such as plant stems, in some of the most severe climates on Earth.<sup>23</sup>

Less-extreme supercooling is employed by a variety of other types of animals to avoid freezing. For example, some species of *deep-water* marine teleost fish found in polar seas have been shown to have freezing points of about –1.0°C, yet they swim about unfrozen in waters that have a temperature of about –1.9°C. Supercooled fish in deep waters are unlikely to encounter floating ice crystals that might induce them to freeze.

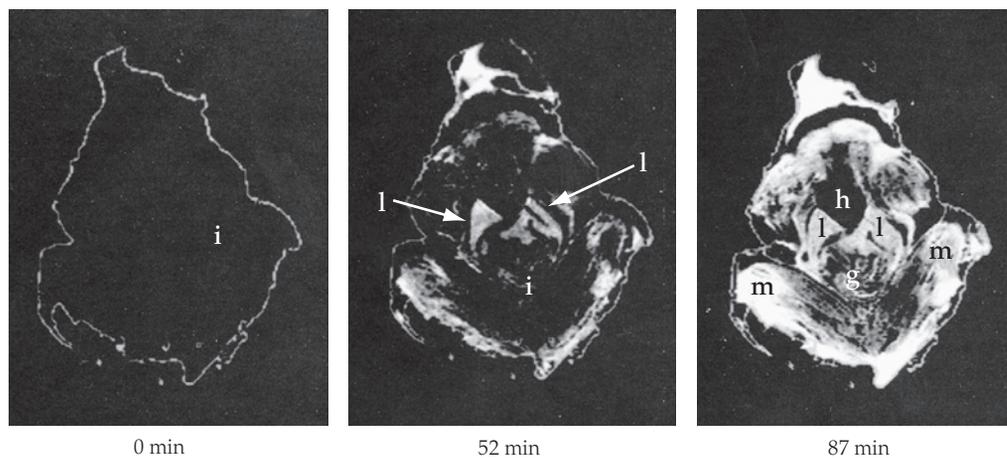
**TOLERANCE OF FREEZING** An ability to survive extracellular freezing is far more widespread than was appreciated even 30 years ago. In the intertidal zone along ocean shores at high latitudes, sessile or slow-moving invertebrates clinging to rocks frequently experience freezing conditions when exposed to the air during winter low tides (see Figure 10.24). Many of these animals—including certain barnacles, mussels, and snails—actually freeze and survive; some tolerate solidification of 60–80% of their body water as ice. Increasing numbers of insect species are also known to tolerate freezing of their blood; tolerance of freezing is probably the most common overwintering strategy of Arctic insects, and some survive temperatures lower than –50°C in their frozen state. One of the extreme examples is a larval insect (*Gynaephora*)—one of the type called woolly bears—that lives in places such as Ellesmere Island in the Arctic. These woolly bears live for many years as larvae and thus must survive many winters before they can metamorphose into adults. They overwinter, frozen, in relatively exposed sites, tolerating body temperatures as low as –70°C! Certain amphibians that overwinter on land, such as spring peepers (*Hyla crucifer*) and wood frogs (*Rana sylvatica*), survive freezing at body temperatures of –2°C to –9°C (Figure 10.27).

For freezing-tolerant animals, whereas intracellular freezing is destructive, extracellular freezing is safe and helps prevent intracellular freezing (see Figure 10.25). These animals commonly undergo physiological changes in winter that limit *the degree of supercooling* that is possible in their extracellular fluids—thereby promoting freezing in the extracellular fluids, where the freezing is safe. Some synthesize ice-nucleating agents (e.g., proteins or lipoproteins) and add them to their extracellular fluids. In some cases, the animals expose themselves to environmental ice and have body surfaces that permit external ice to induce freezing (inoculative freezing) of their extracellular fluids.

The ability of animals to *tolerate* freezing depends in part on the addition of certain organic solutes to their body fluids. Polyhydric alcohols (principally glycerol) are the primary organic solutes promoting tolerance of freezing in insects. Glucose and glycerol are the solutes of primary importance in most freezing-tolerant amphibians. These organic solutes enter both the intracellular and the extracellular fluids of the animals, thereby increasing the amount of solute in both places. The increased solute in the extracellular fluids limits the amount of extracellular ice formation that occurs before the concentration of the unfrozen extracellular fluids rises high enough to prevent further freezing (see Figure 10.25b). The increased solute in the intracellular fluids limits the amount of

<sup>23</sup> Disruption of supercooling is a potential tool for insect control. Some bacteria and other microbes are known to act as highly effective ice-nucleating agents. Such microbes are being investigated as biological control agents against insect pests that depend on supercooling for winter survival.

**FIGURE 10.27** Frozen wood frogs (*Rana sylvatica*) thaw approximately synchronously throughout the body. Magnetic resonance imaging (MRI) was used to detect the state of water in the body of a thawing wood frog. In the images, ice is dark; liquid water is light. The images were taken at specified times after the frozen frog was placed at +4°C. Contrary to what might be expected, wood frogs do not thaw from outside to inside. Instead, deep and superficial regions of a frog's body thaw approximately simultaneously, probably because deep regions have lower melting points than superficial ones have. Synchronous thawing may ensure that blood flow to thawed tissues can start promptly. g = gut; h = heart; i = ice; l = liver; m = leg muscle. (Photographs used with gratitude; from Rubinsky et al. 1994.)



water that must be lost from the cells for cells to come to osmotic equilibrium with freeze-concentrated extracellular fluids (see Figure 10.25c), thus limiting cell shrinkage. These are thought to be some of the principal ways by which the organic solutes aid the tolerance of freezing by poikilotherms.

### SUMMARY Poikilothermy (Ectothermy)

- Poikilotherms, also called ectotherms, are animals in which body temperature ( $T_B$ ) is determined by equilibration with external thermal conditions. They often thermoregulate. Their mechanism of thermoregulation is behavioral; a poikilotherm controls its  $T_B$  by positioning its body in environments that will bring its  $T_B$  to the set-point (“preferred”) level.
- The resting metabolic rate of a poikilotherm is usually an approximately exponential function of its  $T_B$ . The  $Q_{10}$  is typically 2 to 3. The metabolism–temperature curves of poikilotherms are often plotted on semilogarithmic coordinates because exponential functions are straight on such coordinates.
- From the viewpoint of metabolic rate, when poikilotherms acclimate to cold or acclimatize to low-temperature environments in nature, their most common response is partial compensation. Partial compensation returns an animal's metabolic rate toward the level that prevailed prior to the change in environment, and thus it blunts the effect of environmental change. The most common known mechanism of partial compensation is for cells to change their concentrations of key, rate-limiting enzymes.
- Different species of poikilotherms that have long evolutionary histories of living at different body temperatures frequently display evolved physiological differences that suit them to function best at their respective body temperatures. Species of lizards sprint fastest at their respective preferred body temperatures, and polar species of fish function at higher rates in frigid waters than temperate-zone species can. The important mechanisms of evolutionary adaptation to different body temperatures include molecular specialization: Species with evolutionary histories in different environments often synthesize different molecular forms of protein molecules and different suites of cell-membrane phospholipids. The evolution of *structurally* distinct protein forms and phospholipids conserves *functional* properties of the molecules; because of evolved molecular specialization, species living in different thermal environments exhibit similar enzyme–substrate affinities and similar membrane-lipid fluidities.

- When exposed to threat of freezing, some poikilotherms actually freeze and are freezing-tolerant; freezing must be limited to the extracellular body fluids, however. Other poikilotherms are freezing-intolerant and exploit one of three strategies—behavioral avoidance, antifreeze production, or supercooling—to avoid freezing. Antifreezes lower the freezing point. Stabilization of supercooling permits animals to remain unfrozen while at temperatures below their freezing points.

### Homeothermy in Mammals and Birds

**Homeothermy**, the regulation of body temperature by physiological means, gives mammals and birds a great deal more independence from external thermal conditions than is observed in lizards, frogs, or other poikilotherms. On a cool, cloudy day, a lizard or other behaviorally thermoregulating poikilotherm may be unable to reach its preferred body temperature, because warming in such animals depends on a source of outside heat. A mammal or bird, however, produces its own heat for thermoregulation and thus can maintain its usual body temperature whether the environment is warm, moderately cold, or subfreezing.

Mammals and birds *independently* evolved the full-fledged forms of homeothermy they exhibit today. Although the extent of convergence in their physiology of homeothermy is remarkable, they also exhibit consistent differences, one being in their average body temperatures.

Placental mammals typically maintain deep-body temperatures averaging about 37°C when they are at rest and not under heat or cold stress.<sup>24</sup> Birds maintain higher temperatures under similar conditions: about 39°C. One of the most remarkable attributes of mammals and birds is that in both groups, the average body temperatures of thermally unstressed animals do not vary much with climate. One might expect, for instance, that species of mammals living in the Arctic would have lower average body temperatures than related species living in the tropics. Actually, however, differences of this sort are slight, if present at all.

<sup>24</sup>Marsupials, some of the primitive placental mammals, and especially monotremes have lower body temperatures; the platypus, for example, exhibits a deep-body temperature of 30–33°C.

Deep-body temperature is not absolutely constant. Daily cycles occur; the body temperatures of mammals and birds are typically 1–2°C higher during their active phases each day than during their resting phases. Moreover, in some species the body temperature is permitted to rise when individuals are under heat stress, or it is permitted to fall in winter.

Regardless of the variations that occur, the body temperatures of mammals and birds are among the most stable in the animal kingdom. Thus one consequence of homeothermy is that cellular functions are able to be specialized to take place *especially reliably* at certain temperatures. However, as we will see, homeothermy has a very high energy cost and greatly increases the food requirements of mammals and birds in comparison with like-sized nonavian reptiles or fish.

Thermoregulation by a mammal or bird requires neurons (nerve cells) that sense the current body temperature and also requires thermoregulatory control centers in the brain that, by processing thermal sensory information, properly orchestrate the use of heat-producing and heat-voiding mechanisms in ways that stabilize the body temperature (Box 10.2). The detection of body temperature in a mammal or bird occurs in multiple parts of the body; thermosensitive neurons of importance are found in the skin, spinal cord, and brain, and sometimes also in specialized locations such as the scrotum. The principal control centers—which process the multiple sensory inputs and command the thermoregulatory mechanisms—are located in the hypothalamus and the associated preoptic regions of the brain.

A behavioral thermoregulator like a fish or a lizard must also have thermoregulatory control centers that receive and process thermosensory information and that orchestrate the processes of thermoregulation (see Box 10.2). Physiologists generally hypothesize that during the course of vertebrate evolution, there has been a thread of continuity in the control centers. According to this hypothesis, the control centers were already present in a rudimentary way when the only vertebrates were fish and the only thermoregulatory mechanisms to be controlled were behaviors. Recognizing that some modern lizards pant, change color to aid their thermoregulation, or employ other physiological mechanisms, physiologists usually conclude that the early reptilian ancestors of mammals and birds probably had some physiological thermoregulatory mechanisms that supplemented their dominant behavioral mechanisms. The control centers of those early reptiles would therefore have had both physiological and behavioral mechanisms to control. Then, as mammals and birds appeared, the control centers assumed control of predominantly physiological mechanisms. There is some evidence for this sort of scenario—with the evidence from the study of fever being particularly intriguing (see Box 10.2).

A comparison of modern nonavian reptiles with mammals and birds suggests that the single most revolutionary step that occurred in the evolution of mammalian and avian homeothermy was the evolution of endothermy. Modern lizards, turtles, crocodylians, and snakes (with isolated exceptions) cannot warm their bodies by metabolic heat production. Mammals and birds, in dramatic contrast, have an endogenous ability to stay warm in cold environments because of endothermy. With endothermy plus their physiological mechanisms of keeping cool in hot environments, mammals and birds are able to maintain relatively constant tissue temperatures over exceedingly wide ranges of environmental conditions.

### Metabolic rate rises in cold and hot environments because of the costs of homeothermy

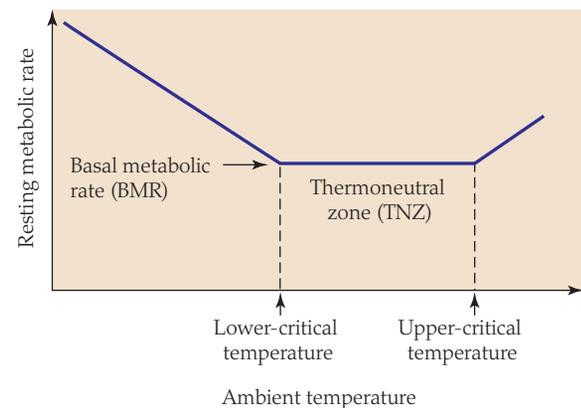
The resting metabolic rate of a mammal or bird typically varies with ambient temperature, as shown in Figure 10.28. Within a certain range of ambient temperatures known as the **thermoneutral zone (TNZ)**, an animal's resting metabolic rate is independent of ambient temperature and constant. The lowest ambient temperature in the TNZ is termed the **lower-critical temperature**; the highest is the **upper-critical temperature**.<sup>25</sup> The lower-critical and upper-critical temperatures depend on the species, and they can also be affected by acclimation or acclimatization. An animal's **basal metabolic rate (BMR)** is its metabolic rate when resting and fasting<sup>26</sup> in its thermoneutral zone.

The resting metabolic rate of a mammal or bird increases as the ambient temperature falls below the animal's lower-critical

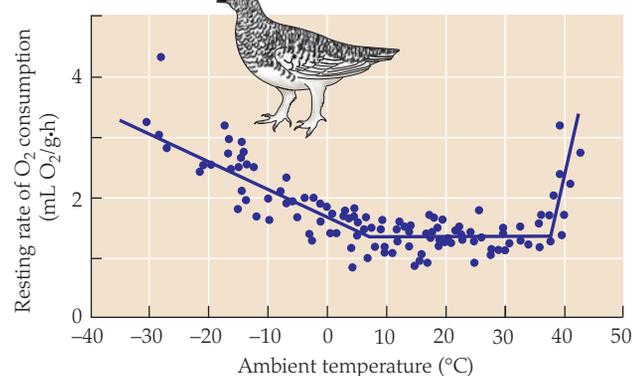
<sup>25</sup>The meaning of *critical temperature* in the study of homeotherms is very different from the meaning of the same term in the study of poikilotherms (see Figure 10.16). The critical temperatures of a homeotherm are simply benchmarks within the spectrum of ordinary, nonstressful ambient temperatures. The critical temperatures of a poikilotherm are temperatures at which the animal is in danger of becoming nonfunctional and dying.

<sup>26</sup>In this context, *fasting* means that the animal has not eaten for long enough that the specific dynamic action of its last meal has ended. *Postabsorptive* is a synonym.

(a) The general relation



(b) An example



**FIGURE 10.28** The relation between resting metabolic rate and ambient temperature in mammals and birds (a) The general relation and the terminology used to describe it. (b) An example, the metabolism–temperature relation of the white-tailed ptarmigan (*Lagopus leucurus*). (b after Johnson 1968.)

## BOX 10.2 THERMOREGULATORY CONTROL, FEVER, AND BEHAVIORAL FEVER

Of all the physiological control systems, the system for thermoregulation is the one that usually seems the most straightforward conceptually. Virtually every introductory treatment of control theory in physiology uses the thermoregulatory control system as its central example. This is undoubtedly true because analogies can so readily be drawn with engineered thermal control systems, which are common in our everyday lives.

In a house with a furnace and air conditioner, the thermostat controls heat production by the furnace and heat removal by the air conditioner to maintain a stable air temperature. Using the terminology of control theory to describe this system, the air temperature is the **controlled variable** (see Box 1.1), and the furnace and air conditioner are **effectors**, instruments that are capable of changing the controlled variable. The thermostat itself actually includes three separate elements that are essential for a control system:

1. A **sensor**, a device that can measure the controlled variable so that the control system knows its current level (the current air temperature).
2. A **set point** or **reference signal**. The set point is a type of information that remains constant in a control system even as the controlled variable goes up and down, and that tells the system the desired level

of the controlled variable. We usually call the set point of a home thermostat its “setting.” If, for example, we “set” the thermostat to 20°C, the device is able to retain that set-point information in an invariant form, so that the air temperature detected by the sensor can be compared with it. An important point to recognize is that a thermostat does not remember its set point by having inside it an object that is kept literally at the set-point temperature. Instead, the set-point temperature is represented in the thermostat by a physical system that is not a temperature, but corresponds to a temperature.

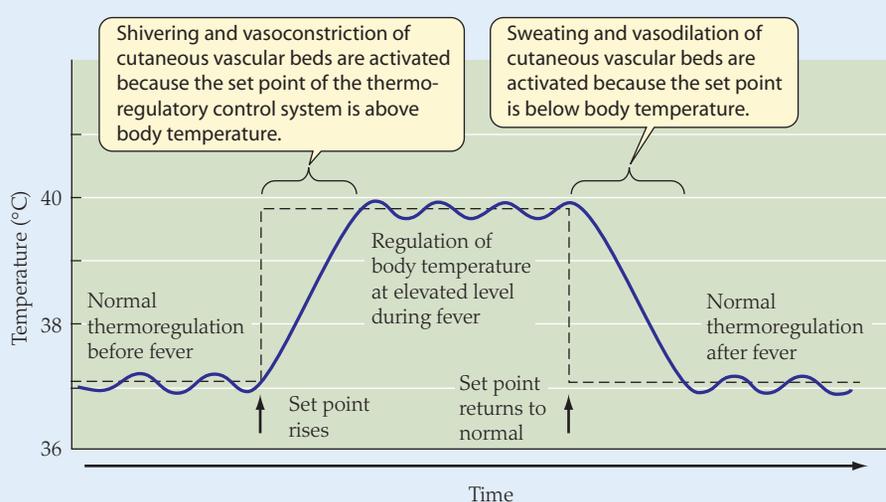
3. A **controller**, a mechanism that compares the set point with the current level of the controlled variable to decide whether the controlled variable is too high or low.

The control system in a house, considered as a whole, operates as a **negative feedback** system (see Box 1.1). It controls the effectors to bring the controlled variable back toward the set point. For example, if the air temperature goes below the set point, the furnace is commanded to add heat to the house.

By analogy, it is easy to describe the thermoregulatory control system of a lizard or mammal (or any other thermoregulator) in terms of the same basic concepts. The principal *effectors* in a lizard (a behavioral thermoregulator) are the skeletal muscles that

move the limbs and control posture. Effectors in a mammal include muscle cells that can produce heat by shivering, sweat glands that can promote evaporative cooling, hair-erector muscles that determine how fluffed the pelage is, and so forth. A lizard or mammal has multiple *sensors*: temperature-sensitive neurons that measure the current temperatures of the skin, spinal cord, and brain. These sensors send their temperature data to a *controller* in the brain that compares the current temperatures with a *set point* to decide what to do. The exact nature of the controller and set point remain far from fully understood because they consist of many tiny neurons in the depths of the brain. As in the case of the home thermostat, however, we recognize that the set point is not literally a temperature in the brain but is *represented* in some way by neurons.

If we disregard the uncertainties that exist about the nature of the set point and simply use the terminology of control theory, the set point of a lizard or mammal can be adjusted to different “settings” at different times, just as the setting of a home thermostat can be adjusted. **Fever** in a mammal provides an elegant example of resetting of the thermostat (see the figure). Fish, amphibians, and nonavian reptiles sometimes develop fevers—called **behavioral fevers** because the effectors are skeletal muscles that modify behaviors. **Box Extension 10.2** discusses thermoregulatory control and especially fever in more detail.



### KEY

- Set point of the thermoregulatory control system
- Deep-body temperature

### A bout of fever in a placental mammal: The relation between the set point of the thermoregulatory control system and body temperature

When the set point jumps up at the start of a bout of fever and falls back down at the end, the thermoregulatory control system detects the mismatch between the set point and the body temperature and commands vigorous effector responses to correct the mismatches. These responses include shivering at the start and sweating at the end.

temperature or rises above its upper-critical temperature (see Figure 10.28a). These increases in metabolic rate in both cold and warm environments arise from the animal’s need to perform physiological work to keep its deep-body temperature constant regardless of whether the ambient temperature is low or high.

### The shape of the metabolism–temperature curve depends on fundamental heat-exchange principles

Before we study the physiological mechanisms used by mammals and birds to thermoregulate, it is important to analyze why the metabolism–temperature curve has the specific shape it does. A

useful first step for this analysis is to recognize the concept of **dry heat transfer**, defined to be heat transfer that does not involve the evaporation (or condensation) of water. Dry heat transfer occurs by conduction, convection, and thermal radiation. As stressed earlier, these three mechanisms of heat transfer must be analyzed separately in heterogeneous thermal environments. However, in a *uniform thermal environment* such as a laboratory cage or test chamber, where the radiant temperatures of all objects are typically similar to air temperature, these three mechanisms of heat transfer can be meaningfully lumped together. They can be lumped in this circumstance because, in all three cases, the rate of heat transfer between an animal and its environment tends to increase approximately in proportion to the difference in temperature between the animal's body and the environment ( $T_B - T_A$ ) (see Figure 10.4). By lumping the three together, we can study dry heat transfer as a whole (because dry heat transfer = conduction + convection + thermal-radiation heat transfer).

In a uniform thermal environment, if factors other than temperature are held constant,

$$\text{Rate of dry heat transfer} \propto T_B - T_A \quad (10.8)$$

Heat moves out of an animal's body by dry heat transfer when  $T_B$  exceeds  $T_A$ ; conversely, heat moves into the body when  $T_B$  is less than  $T_A$ . The rate of dry heat transfer is proportional to  $(T_B - T_A)$  in either case, and thus  $(T_B - T_A)$  can be thought of as being the "driving force" for dry heat transfer.

To analyze the shape of the metabolism–temperature curve, Equation 10.8 *taken by itself* can be used at ambient temperatures that are *within* and *below* the thermoneutral zone (TNZ). At temperatures above the TNZ, evaporative heat transfer is too important to be ignored. At ambient temperatures within and below the TNZ, however, evaporative heat transfer is in general sufficiently minor that it can be disregarded for purposes of a conceptual analysis, explaining why the equation for dry heat transfer (Equation 10.8) can be used. The body temperature of a mammal or bird is typically higher than the animal's upper-critical temperature. Thus, when the ambient temperature is within or below the TNZ,  $(T_B - T_A)$  is always positive, and dry heat transfer carries heat out of the body.

Under such conditions, in which an animal is losing heat to its environment, the only way the animal can maintain a constant body temperature is to make heat *metabolically* at a rate that matches its rate of heat loss. Accordingly, if  $M$  is the animal's metabolic rate,  $M$  must equal the animal's rate of heat loss. Thus, if we assume that Equation 10.8 describes the rate of heat loss at ambient temperatures within and below the TNZ, then (at those ambient temperatures)  $M \propto (T_B - T_A)$ . We can rewrite this expression as an equation by introducing a proportionality coefficient ( $C$ ):

$$M = C (T_B - T_A) \quad (10.9)$$

This equation, which is a famous equation for analyzing a mammal's or bird's thermal relations, is called the **linear heat-transfer equation**, also described sometimes as **Newton's law of cooling** or **Fourier's law of heat flow**. The coefficient  $C$ , which is termed the animal's **thermal conductance**, is a measure of *how readily* heat can move by dry heat transfer from an animal's body into its environment.

To see the significance of  $C$ , suppose that two placental mammals are in the same environment and therefore have the same driving force for dry heat loss ( $T_B - T_A$ ), but one has a higher thermal

conductance than the other. The one with the higher  $C$  will lose heat faster because heat can move out of its body more readily than heat can move out of the body of the other. Therefore, the one with the higher  $C$  will require a higher metabolic rate to stay warm.

An animal with a *high*  $C$  can be thought of as having a *low resistance* to dry heat loss. Conversely, an animal with a *low*  $C$  can be thought of as having a *high resistance* to dry heat loss. Physiologists, accordingly, define an animal's **resistance to dry heat loss** to be the inverse of  $C$ :  $1/C$ . The resistance to dry heat loss is often called **insulation** ( $I$ ). Thus  $I = 1/C$ . The linear heat-transfer equation can therefore also be written as:<sup>27</sup>

$$M = \frac{1}{I} (T_B - T_A) \quad (10.10)$$

An important point to note about the concept of insulation ( $I$ ) introduced here is that it is not simply a measure of the heat-retaining properties of the fur or feathers. Instead, insulation ( $I$ ) is a measure of an animal's *overall* resistance to dry heat loss. For instance, because both posture and fur affect a mammal's resistance to dry heat loss, the value of  $I$  for a mammal depends on its posture as well as its fur (and also on additional factors).

#### THE THERMONEUTRAL ZONE: INSULATION IS MODULATED TO KEEP THE RATE OF HEAT LOSS CONSTANT

Let's now use the concepts we have developed to understand why the metabolism–temperature curve of a mammal or bird has the shape it does in the thermoneutral zone. The defining property of the TNZ is that an animal's metabolic rate ( $M$ ) remains constant at all the different ambient temperatures in the TNZ. This property probably seems impossible or paradoxical at first. After all, if  $T_A$  changes, then  $(T_B - T_A)$  changes, and Equation 10.10 suggests that  $M$  would have to change. The answer to this paradox is that in its TNZ, a mammal or bird *varies* its insulation. *Modulation of insulation against a background of constant metabolic heat production is the principal means by which a mammal or bird thermoregulates in its thermoneutral zone.*

Let's discuss this key concept in more detail. As the ambient temperature is lowered in the TNZ and  $(T_B - T_A)$  accordingly becomes greater, a mammal or bird responds by increasing its insulation,  $I$ .<sup>28</sup> This increase in the animal's resistance to heat loss *counterbalances* the increase in the *driving force* for heat loss,  $(T_B - T_A)$ , so that the animal's *actual rate of heat loss* remains constant (or nearly so). The animal's rate of metabolic heat production, therefore, can also remain constant. These points are mathematically apparent in Equation 10.10. In the TNZ, as  $T_A$  decreases and  $(T_B - T_A)$  therefore increases,  $I$  is increased in a precisely counterbalancing way so that the ratio  $(T_B - T_A)/I$  remains constant. The metabolic rate of the animal,  $M$ , can therefore be constant.

The *width* of the TNZ varies enormously from species to species, depending in part on the extent to which various species are able to modulate their insulation. Small-bodied species tend to have narrower TNZs than large-bodied species do. Species of mice, for instance, often have TNZs extending only from about 30°C

<sup>27</sup>In this form, the linear heat-transfer equation bears a close similarity to Ohm's Law.  $(T_B - T_A)$ , the driving force, is analogous to potential difference (voltage);  $I$  is analogous to electrical resistance; and  $M$  is analogous to current flow. Current = voltage/resistance.

<sup>28</sup>Starting on page 256, we discuss the actual mechanisms of increasing insulation.

to 35°C. At another extreme, Eskimo dogs have a TNZ extending from -25°C to +30°C—a range of 55°C!

**TEMPERATURES BELOW THERMONEUTRALITY** Unlike the case within the thermoneutral zone, the principal means by which a mammal or bird thermoregulates at ambient temperatures below thermoneutrality is modulation of its rate of metabolic heat production. Specifically, below the TNZ, as the environment becomes colder, a mammal or bird must raise its rate of metabolic heat production to higher and higher levels if it is to stay warm. In this way, mammals and birds resemble a furnace-heated house in which the furnace must increase the rate at which it produces heat as the air outside becomes colder.

How exactly is the lower-critical temperature determined? To see the answer, consider an animal, initially in its TNZ, that is subjected to a steadily declining ambient temperature. As the  $T_A$  declines while remaining in the TNZ, the rate at which the animal loses heat to its environment stays constant because the animal increases its insulation,  $I$ . Insulation cannot be increased without limit, however. An animal's lower-critical temperature represents the  $T_A$  below which its insulatory adjustments become inadequate to counterbalance fully the increase in the driving force favoring heat loss. As the  $T_A$  falls below the lower-critical temperature, the rate at which an animal loses heat increases, and the animal must therefore increase its rate of heat production to match the increased rate of heat loss.

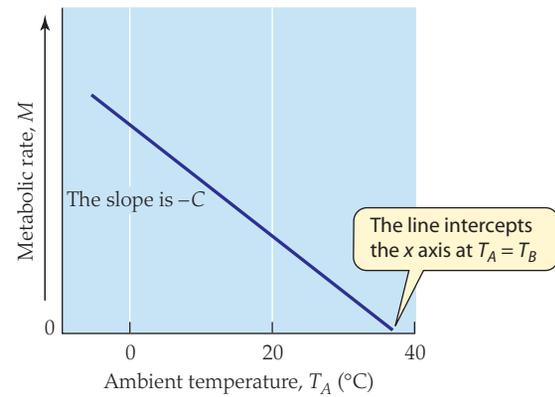
The insulation of a mammal or bird sometimes becomes maximized at the lower-critical temperature. Cases like this are particularly straightforward to understand in terms of the linear heat-transfer equation (Equation 10.9 or 10.10).

If an animal maximizes its insulation at the lower-critical temperature, then its value of  $I$  at ambient temperatures below the TNZ is a *constant* (equaling its maximum value of  $I$ ). In addition, because  $C = 1/I$ , the animal's value of  $C$  below the TNZ is a *constant* (equaling its minimum value of  $C$ ). For such an animal, therefore,  $T_B$ ,  $I$ , and  $C$  in the linear heat-transfer equation are all constants below the TNZ. Accordingly, the linear heat-transfer equation—whether written as Equation 10.9 or 10.10—is a simple linear equation (accounting for its name) that has two variables:  $M$  and  $T_A$ . If we plot  $M$  as a function of  $T_A$  for this linear equation—using Equation 10.9—we obtain a straight line having two particular properties, illustrated in **Figure 10.29a**: First, the slope of the line is  $-C$ . Second, the line intersects the  $x$  axis at the ambient temperature that is equal to  $T_B$ .

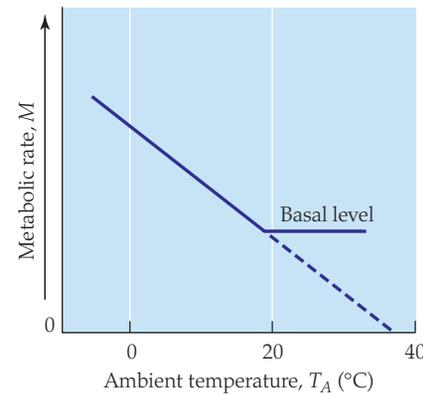
As a model of an animal's metabolism–temperature curve, the plot in Figure 10.29a is flawed because it ignores the fact that an animal's metabolic rate ( $M$ ) does not truly fall below the basal level. **Figure 10.29b** is thus more realistic. By comparing Figures 10.29a and b, you can see that *the portion of an animal's metabolism–temperature curve below the TNZ is simply a plot of the linear heat-transfer equation*.

The fact that the slope of the metabolism–temperature curve below the TNZ is equal to  $-C$  (for animals that maintain a constant  $C$ ) provides a useful tool for the visual interpretation of metabolism–temperature curves. As shown in **Figure 10.29c**, if two otherwise similar animals differ in thermal conductance ( $C$ ) below the TNZ, the relative slopes of their metabolism–temperature curves mirror their differences in  $C$ : The animal with a high value of  $C$  (low insulation) has a steeper slope than the animal with low  $C$  (high insulation). Using this principle, one can look at Figure 10.40b

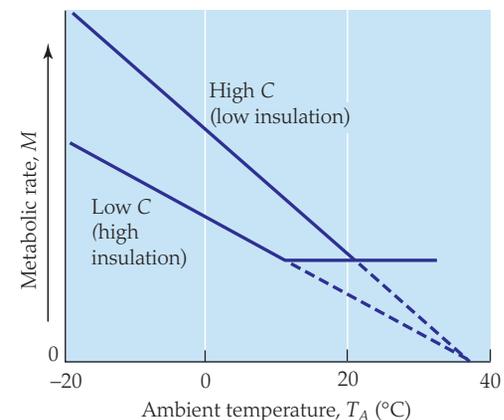
(a)  $M = C(T_B - T_A)$  with  $C$  and  $T_B$  constant ( $T_B = 37^\circ\text{C}$ )



(b) The plot from (a), recognizing that  $M$  actually falls only to the basal level



(c) Comparison of two animals that differ in  $C$  below thermoneutrality



**FIGURE 10.29** A model of the relation between metabolic rate and ambient temperature in and below the thermoneutral zone

(see page 264), for example, and tell at a glance that the winter fox has lower conductance and higher insulation than the summer fox. Figure 10.29c also highlights the energy advantages of high insulation. Note that the animal with relatively high insulation (low  $C$ )—analogous to a well-insulated house—has a relatively low requirement for metabolic heat production and a low metabolic rate at any given ambient temperature below the TNZ.<sup>29</sup>

<sup>29</sup> Although the slopes of metabolism–temperature curves were used to calculate  $C$  quantitatively some years ago, better approaches for the calculation of  $C$  have been developed. Thus the use of slopes today should be reserved for just qualitative, visual interpretation.

**TEMPERATURES ABOVE THERMONEUTRALITY** Mammals and birds employ two principal processes to respond to ambient temperatures above their thermoneutral zones:

- Most mammals and birds actively increase the rate at which water evaporates from certain of their body surfaces, a process called **active evaporative cooling**. Sweating, panting, and gular fluttering (Figure 10.30) are the most common mechanisms of active evaporative cooling.
- Some mammals and most birds allow their body temperatures to rise to unusually high levels, a phenomenon called **hyperthermia**.

Both active evaporative cooling and hyperthermia can cause an animal's metabolic rate to rise at temperatures above thermoneutrality. Active evaporative cooling causes a rise in metabolic rate because physiological work must be done to enhance water evaporation (panting, for example, requires an increase in the rate of breathing). Hyperthermia can also cause a rise in metabolic rate because tissues tend to accelerate their metabolism when they are warmed; according to recent research, hyperthermia does not always cause metabolic acceleration in mammals and birds, but in some cases it does.

To appreciate more fully the processes at work above the TNZ, it is informative to take a dynamic approach by considering an animal that is initially within its TNZ and subjected to a steadily increasing ambient temperature. As the  $T_A$  rises, the driving force for dry heat loss ( $T_B - T_A$ ) decreases, meaning that the animal faces a greater and greater challenge to get rid of its *basal* metabolic heat production. While the  $T_A$  remains in the TNZ, the animal responds to the rising  $T_A$  by decreasing its resistance to dry heat loss, its insulation. Consequently, even high in the TNZ, metabolic heat is carried away as fast as it is produced by a combination of dry heat transfer and *passive* evaporation. This handy state of affairs comes to an end when the  $T_A$  reaches the upper-critical temperature and goes higher. Near the upper-critical temperature, insulation either reaches its minimum or at least becomes incapable of sufficient further reduction to offset additional decreases in ( $T_B - T_A$ ). Thus, as the  $T_A$  rises above the upper-critical temperature, the rate of dry heat loss tends to fall too low for the combination of dry heat loss and passive evaporation to void metabolic heat. Both of the principal responses of mammals and birds—active evaporative cooling and hyperthermia—serve to promote heat loss so that animals are not overheated by their metabolic heat production. Hyperthermia does this because a rise in  $T_B$  increases the driving force for dry heat loss ( $T_B - T_A$ ).

If the  $T_A$  keeps rising and becomes so high that it exceeds  $T_B$ , heat stress becomes extraordinary because—when the  $T_A$  is above the  $T_B$ —dry heat transfer carries environmental heat *into* the body!<sup>30</sup> Then active evaporative cooling must assume the entire burden of removing heat from the body.

From a quick glance at the metabolism–temperature curve above the TNZ (see Figure 10.28), it may seem extremely paradoxical that a mammal or bird increases its metabolic rate—its rate of internal heat production—when it is under heat stress. To understand this paradox, it is important to recall the very large amount of heat

<sup>30</sup>For the  $T_A$  to exceed  $T_B$ , it must rise substantially above the upper-critical temperature. When the  $T_A$  is just moderately above the upper-critical temperature, it is typically below the  $T_B$ .



**FIGURE 10.30** Gular fluttering is one means of actively increasing the rate of evaporative cooling. During gular fluttering, which occurs in birds but not mammals, an animal holds its mouth open and vibrates the floor of the mouth, termed the gular area (arrow). In this way, airflow is increased across the moist, vascular mouth membranes, promoting a high rate of evaporation. The bird seen here is a young great egret (*Casmerodius albus*) on a hot day in Florida. In some species the gular area vibrates up and down at 800–1000 cycles/minute during gular fluttering.

carried away by the evaporation of each gram of water (see page 230). Although an animal must increase its metabolic rate to pant, gular flutter, or otherwise actively increase its rate of evaporation, the amount of heat carried away by the evaporation of each gram of water far exceeds the heat produced per gram by the physiological processes that accelerate evaporation.

### Homeothermy is metabolically expensive

One of the most important attributes of homeothermy in mammals and birds is that it is metabolically expensive in comparison with vertebrate poikilothermy. Homeothermy in mammals and birds in fact provides an outstanding example of a point stressed in Chapter 1: When physiological *regulation* and *conformity* are compared, the greatest downside of regulation is that its energy costs are high.

To quantify the cost of homeothermy, physiologists have compared the metabolic rates of vertebrate homeotherms and poikilotherms at similar tissue temperatures. Specifically, they have compared the basal metabolic rates of mammals and birds with the resting metabolic rates of like-sized poikilotherms held at the same body temperatures as the mammals and birds. A typical experiment would be to obtain a 100-g placental mammal and place it in its thermoneutral zone, and simultaneously obtain

a 100-g lizard and place it in a chamber at 37°C so that its body temperature matches that of the mammal. If both animals are at rest and fasting and you measure their metabolic rates, you will obtain (1) the basal metabolic rate (BMR) of the mammal, and (2) the standard metabolic rate (SMR) of the lizard *at mammalian body temperature*. Typically what you will find is that the metabolic rate of the mammal is four to ten times higher than that of the lizard, even though the cells of the two animals are at one temperature and the mammal's metabolic rate under these conditions is its *minimal* rate! Many studies of this sort have been carried out on a variety of species, and they have confirmed repeatedly that the BMRs of mammals and birds are four to ten times the SMRs of poikilothermic vertebrates at mammalian or avian body temperatures (see Figure 7.9). *Metabolic intensity stepped up dramatically when vertebrates evolved homeothermy.*

If mammals, birds, and poikilothermic vertebrates studied as we have just described are transferred to cold ambient temperatures, the metabolic rates of the mammals and birds *rise* (see Figure 10.28), whereas the metabolic rates of the poikilotherms *fall* (see Figure 10.9). At cold ambient temperatures, therefore, the difference in metabolic intensity between homeotherms and poikilotherms is far greater than just four- to tenfold.

Animals living in the wild experience both high and low ambient temperatures at various times. Their *average* metabolic rates thereby integrate the effects of different temperatures. As discussed in Chapter 9, field metabolic rates have now been measured in many free-living terrestrial vertebrates by use of the doubly labeled water method. Those measures reveal that the average field metabolic rate is typically 12–20 times higher in mammals and birds than in lizards or other nonavian reptiles of the same body size! The mammals and birds must therefore acquire food at a much higher rate.

### Insulation is modulated by adjustments of the pelage or plumage, blood flow, and posture

Now we turn (in this section and several that follow) to the *mechanisms* that mammals and birds employ to thermoregulate physiologically. First we discuss the mechanisms by which mammals and birds modulate their resistance to dry heat transfer, their insulation. As we have seen, these are the predominant mechanisms of thermoregulation within the thermoneutral zone.

One means of varying insulation is erection or compression of the hairs or feathers. Each hair or feather can be held upright or allowed to lie flat against the skin by the contraction or relaxation of a tiny muscle at its base, under control of the sympathetic nervous system. These responses are termed **pilomotor responses** in mammals and **ptilomotor responses** in birds. If the ambient temperature declines within the TNZ, the hairs or feathers are erected to an increased degree. In this way the pelage or plumage is fluffed out and traps a thicker layer of relatively motionless air around the animal, thereby increasing the resistance to heat transfer through the pelage or plumage (see Equation 10.1).

Another mechanism of modulating insulation is the use of **vasomotor responses** in blood vessels (see page 659)—responses that alter the rate of blood flow to the skin surface and other superficial parts of the body. Arterioles supplying superficial vascular beds are constricted at cool ambient temperatures because of stimulation by the sympathetic nervous system. This response retards transport of heat to the body surfaces by blood flow. Conversely, vasodilation

at warm ambient temperatures enhances blood transport of heat to body surfaces where the heat is readily lost.

Insulation may also be modified by **postural responses** that alter the amount of body surface area directly exposed to ambient conditions. At low ambient temperatures, for example, mammals often curl up, and some birds tuck their heads under their body feathers or squat so as to enclose their legs in their ventral plumage. Many birds hold their wings away from their bodies when ambient temperatures are high.

In addition to the insulative properties that can be modulated by an individual animal, there are also properties that affect insulation but are more or less fixed for any given individual. Outstanding among these is body size. At temperatures *below thermoneutrality*, small size tends to increase the weight-specific rate at which animals lose heat—and thus the weight-specific cost of thermoregulation—because relatively small animals have more body surface per unit of weight than large ones have.<sup>31</sup> Another reason that small size tends to enhance heat loss is that small animals cannot have as thick pelage or plumage as large ones. Whereas large mammals commonly have pelage that is at least 5–6 cm thick, mice could not conceivably have such thick pelage; a mouse with 5–6 cm of pelage would be trapped inside its own hair!

### Heat production is increased below thermoneutrality by shivering and nonshivering thermogenesis

When a mammal or bird is below its lower-critical temperature, it must increase its rate of heat production as the ambient temperature declines. Although all metabolic processes produce heat as a by-product, mammals and birds have evolved mechanisms, termed **thermogenic mechanisms**, that are specialized to generate heat for thermoregulation. One of these, shivering, is universal in adult mammals and birds.

**SHIVERING** **Shivering** is unsynchronized contraction and relaxation of skeletal-muscle motor units in high-frequency rhythms, mediated by motor neurons (nerve cells) of the somatic nervous system. Skeletal muscles can basically contract in two patterns. When muscles are being employed in locomotion to move a limb, all the motor units in each muscle contract synchronously, and antagonistic muscles contract in ways that they do not work against each other. When the same muscles are employed in shivering, various motor units within each muscle contract more or less at random relative to each other, antagonistic muscles are activated simultaneously, and the muscles quiver. Either mode of contraction uses ATP and liberates heat. When a muscle shivers, the conversion of ATP-bond energy to heat becomes the primary function of contraction because no useful mechanical work is accomplished.

**NONSHIVERING THERMOGENESIS** The concept of **nonshivering thermogenesis (NST)** is most readily understood by taking a look at the classic studies on laboratory rats that originally led to the discovery of NST. If lab rats that have been living at warm temperatures are transferred to a room at 6°C, they shiver violently during their first days there. If one observes them visually over the next few weeks as they acclimate to 6°C, however, they appear gradually

<sup>31</sup> Be certain that you do not extend this argument to the thermoneutral zone. The argument is valid below thermoneutrality but probably does not apply in the thermoneutral zone (see page 178).

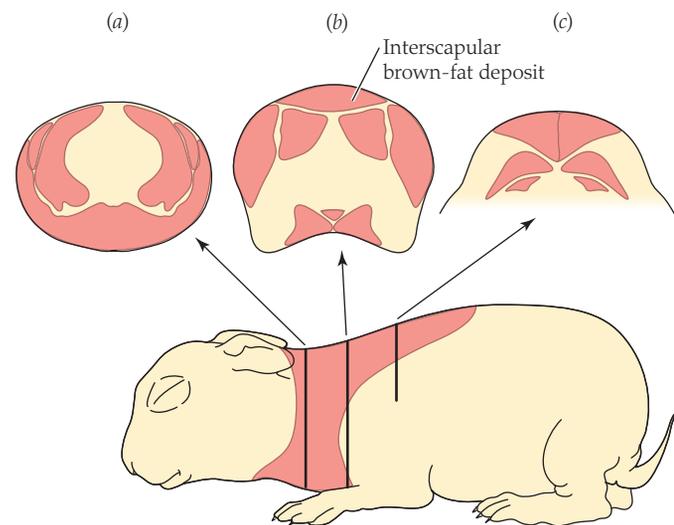
to stop shivering even though they continue to maintain elevated metabolic rates. This visual observation suggests that during acclimation to cold, the rats develop mechanisms of thermogenesis that do not involve shivering. To test if this is in fact the case, cold-acclimated rats can be injected with curare, a plant extract that blocks the contraction of skeletal muscle and therefore prevents shivering. Curare-injected, cold-acclimated rats continue to have elevated metabolic rates and thermoregulate, confirming that they have well-developed nonshivering thermogenic mechanisms.

Whereas shivering is universal in mammals and birds, NST is not. NST is best known, and very common, in placental mammals. It has been reported in the young of a few species of birds (e.g., ducklings), but its occurrence in adult birds remains controversial.

Of all the possible sites of NST in placental mammals, the one that is best understood and dominant is **brown adipose tissue (BAT)**, also called **brown fat**.<sup>32</sup> This is a specialized type of adipose tissue—often reddish brown—that differs greatly in both structure and function from the commonplace white fat that is more familiar.<sup>33</sup> Brown fat tends to occur in discrete masses, located in such parts of the body as the interscapular region, neck, axillae, and abdomen (Figure 10.31). Deposits of BAT receive a rich supply of blood vessels and are well innervated by the sympathetic nervous system. The cells of BAT are distinguished by great numbers of relatively large mitochondria. The rich, red blood supply of BAT and the abundant, yellow cytochrome pigments in its mitochondria impart to the tissue its distinctive color.

When the sympathetic nervous system releases norepinephrine in BAT, the BAT responds by greatly increasing its rate of oxidation of its stored lipids, resulting in a high rate of heat production. BAT is biochemically specialized to undergo uncoupling of oxidative phosphorylation from electron transport (see Figure 8.4c) and uses this mechanism to produce heat rapidly. Uncoupling does two things that result in rapid heat production: (1) It suspends the ordinary controls on the rate of aerobic catabolism, permitting unbridled rates of lipid oxidation; and (2) it causes the chemical-bond energy of oxidized lipid molecules to be released immediately as heat (rather than being stored in ATP). The property that gives BAT its specialized ability to undergo uncoupling is that BAT expresses a distinctive proton-transport protein, **uncoupling protein 1 (UCP1; thermogenin)**, in the inner membranes of its mitochondria (see Figure 8.4c).

Norepinephrine released in BAT binds to  $\beta$ -adrenergic receptors (and other receptors) in the cell membranes of the BAT cells. These receptors are G protein-coupled receptors; as discussed in Chapter 2 (see Figure 2.27), the binding of norepinephrine to the receptors activates G proteins in the cell membranes and leads to the intracellular production of the second messenger cyclic AMP. Cyclic AMP then activates (by phosphorylation) an intracellular lipase enzyme that rapidly hydrolyzes triacylglycerols stored in the cells to release free-fatty-acid fuels for mitochondrial oxidation. Simultaneously, by a mechanism that remains ambiguous, existing



**FIGURE 10.31** The deposits of brown adipose tissue in a newborn rabbit (a), (b), and (c) are cross sections of the body at the positions indicated on the side view. Brown adipose tissue also occurs typically in discrete deposits in adult mammals that have the tissue. (After Dawkins and Hull 1964.)

molecules of the uncoupling protein UCP1 are activated, and thus the mitochondria carry out the lipid oxidation in an uncoupled state. In addition, if norepinephrine stimulation continues for tens of minutes or longer, increased amounts of UCP1 are synthesized because  $\beta$ -adrenergic activation stimulates increased transcription of the gene that encodes UCP1. Still another effect of stimulation of BAT is that a fatty acid transport protein (FATP) in cell membranes is upregulated, permitting the cells to carry out rapid uptake of fatty acids brought from elsewhere in the body.

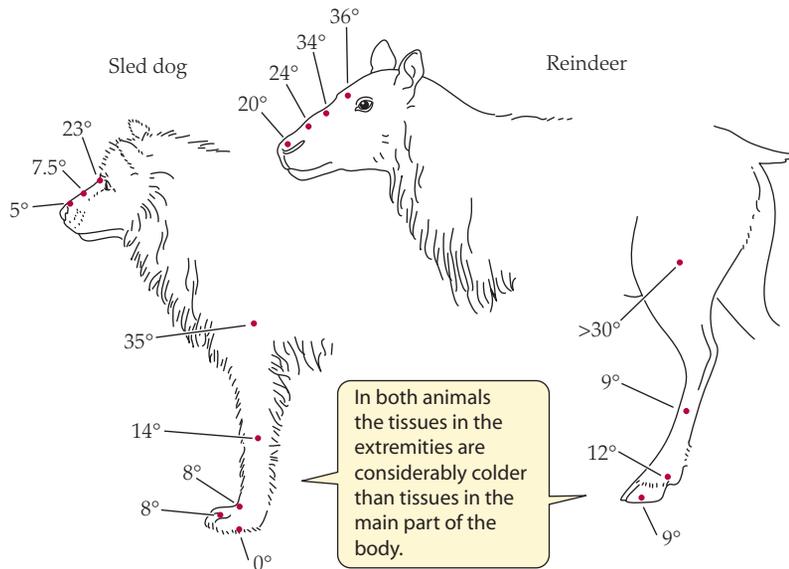
Brown fat—like NST in general—is particularly prominent in three types of placental mammals: (1) cold-acclimated or winter-acclimatized adults (particularly in species of small to moderate body size), (2) hibernators, and (3) newborn individuals. During acclimation to cold or acclimatization to winter, BAT often markedly increases its potential to produce heat; deposits of the tissue grow, BAT cells increase their numbers of mitochondria, and the mitochondria become richer in uncoupling protein. In part, this development of BAT probably serves to free the skeletal muscles to perform exercise. A muscle cannot shiver and exercise at the same time; as BAT develops, muscles are less likely to be employed in shivering, leaving them free to be used in exercise. BAT in newborns (including human babies; see Figure 11.7), hibernators, and human adults is discussed in detail in Chapter 11.

### Regional heterothermy: In cold environments, allowing some tissues to cool can have advantages

Appendages such as legs, tails, and ear pinnae present particular thermal challenges when mammals and birds are below thermoneutrality. The appendages are potentially major sites of heat loss because they have a great deal of surface area relative to their sizes, are often thinly covered with fur or feathers, and exhibit (because of their dimensions) intrinsically high rates of convective heat exchange (see Equation 10.3). If a mammal or bird in a cold environment were to keep its appendages at the same temperature as its

<sup>32</sup>Brown adipose tissue does not occur in birds. Nor does uncoupling protein 1, which we soon discuss. In young birds that show NST, skeletal muscles are apparently the NST site.

<sup>33</sup>Although brown fat is a type of adipose tissue like white fat, recent evidence indicates that the two types of fat do not develop from a single type of precursor cell during the development of mammals. Brown fat develops from a type of precursor cell that also gives rise to skeletal muscle!



**FIGURE 10.32** Regional heterothermy in Alaskan mammals

The air temperature was  $-30^{\circ}\text{C}$  when these data were gathered. The temperatures shown are subcutaneous temperatures ( $^{\circ}\text{C}$ ) at various locations on the body. Note that the footpad tissues of the sled dog were at  $0^{\circ}\text{C}$ . Both animals had deep-body temperatures in the typical mammalian range:  $37\text{--}38^{\circ}\text{C}$ . (After Irving and Krog 1955.)

body core, the appendages would contribute disproportionately to the animal's overall weight-specific metabolic cost of homeothermy.

A mammal or bird can limit heat losses across its appendages in cool environments by allowing the appendage tissues to cool. The difference between the temperature of an appendage and the ambient temperature is the driving force for heat loss from the appendage. Allowing the appendage to cool toward ambient temperature reduces this driving force, in effect compensating for the appendage's relatively low resistance to heat loss. Cooling of the appendages, a type of *regional heterothermy*, is in fact very common. When the ambient temperature is low, the tissues of appendages—especially their distal parts—are often  $10\text{--}35^{\circ}\text{C}$  cooler than tissues in the core parts of an animal's thorax, abdomen, and head (Figures 10.32 and 10.33).<sup>34</sup>

The usual mechanism by which appendages are allowed to cool is by curtailing circulatory delivery of heat to them. Appendages (or parts of appendages) often consist in large part of bone, tendon, cartilage, skin, and other tissues that metabolically are relatively inactive. Such appendages typically do not have sufficient endogenous heat production to keep themselves warm in cold environments. Their temperatures depend, therefore, on how rapidly heat is brought to them from the thorax, abdomen, or head by the circulating blood. Accordingly, curtailing circulatory heat delivery to the appendages lets them cool. Heat delivery to an appendage may be curtailed simply by the restriction of blood flow to the appendage, but as we will see in the next section, more-elaborate mechanisms of restricting heat delivery are usually employed.

Species that have long evolutionary histories in frigid climates often display exquisite control over the extent of appendage cooling. For example, in a variety of Arctic canids—including foxes

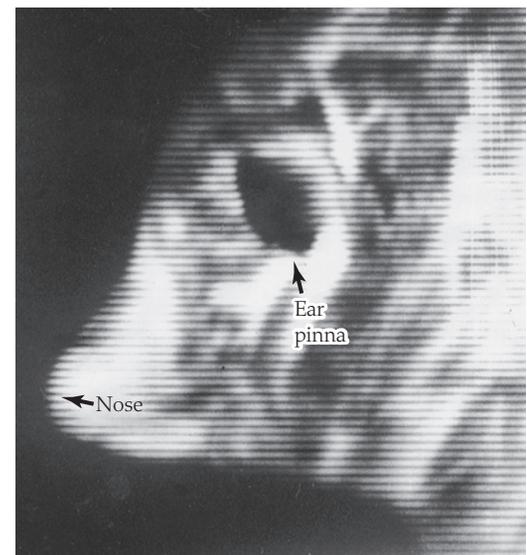
<sup>34</sup>Because regional heterothermy reduces the total metabolic cost of maintaining a given core body temperature, it effectively increases the animal's overall insulation ( $I$ ) in the linear heat-transfer equation (Equation 10.10).

and wolves, as well as sled dogs—the tissues of the footpads are routinely allowed to cool to near  $0^{\circ}\text{C}$  in winter (see Figure 10.32), but even when the feet are in contact with much colder substrates (e.g.,  $-30^{\circ}\text{C}$  to  $-50^{\circ}\text{C}$ ), the footpads are not allowed to cool further. The footpads, therefore, are thermoregulated at the lowest temperature that does not subject them to a risk of frostbite!

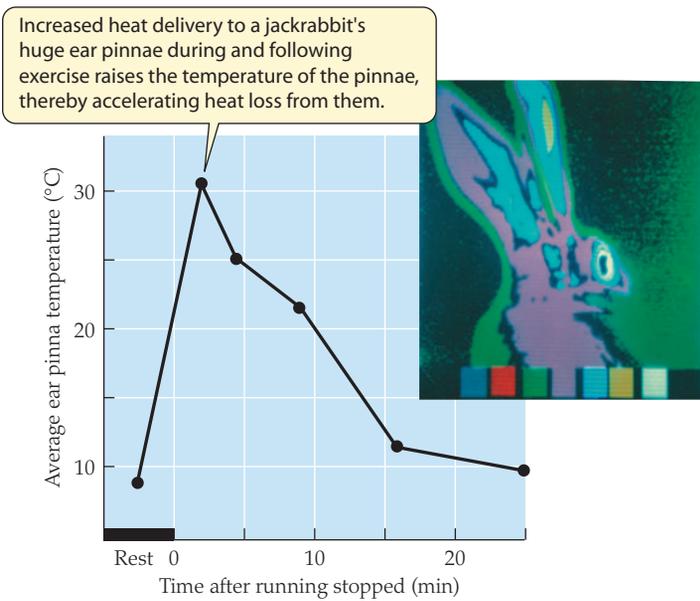
Appendages also often play special roles in the dissipation of excess metabolic heat. If a high rate of circulatory heat delivery is provided to an appendage, the heat is lost readily to the environment because of the ease of heat loss from appendages. Accordingly, whereas animals curtail circulatory heat delivery to their appendages when heat conservation is advantageous, they often augment heat delivery to their appendages when they need to get rid of heat. In a cool environment, for example, when jackrabbits are at rest, they limit blood flow to their huge ear pinnae (see Figure 10.5)—so much so that the pinnae become as cool as the air. However, when the jackrabbits run, they increase blood flow and pinna temperature considerably (Figure 10.34). Running evidently produces an excess of metabolic heat, and the pinnae are used to void the excess heat. Opossums, rats, and muskrats sometimes warm their tails when they exercise; seals heat up their flippers; and goats warm their horns.

### Countercurrent heat exchange permits selective restriction of heat flow to appendages

Although circulatory heat flow into an appendage can be curtailed simply by reduction of the rate of blood flow to the appendage, this mechanism has the disadvantage of being highly nonspecific. Reducing the rate of blood flow not only limits heat flow into an appendage but also subjects the appendage to a reduced rate of  $\text{O}_2$



**FIGURE 10.33** A thermal map of an opossum showing regional heterothermy in the pinna of the ear In this image, which was produced by infrared radiography, shades of gray represent the temperatures on the animal's body surface. Surfaces that were at or close to ambient temperature are black; those that were much warmer than ambient are white; intermediate temperatures are represented by intermediate gray tones. The animal, a Virginia opossum (*Didelphis marsupialis*), was resting at an ambient temperature of  $10^{\circ}\text{C}$ . Note that the surface temperature of its ear pinna, a naked flap of skin, was virtually the same as ambient temperature.



**FIGURE 10.34** Heat loss across appendages is sometimes modulated in ways that aid thermoregulation. The average surface temperature of the ear pinnae of this black-tailed jackrabbit (*Lepus californicus*) was near ambient temperature (8°C) when the rabbit was resting but increased to more than 30°C following running. The inset is an infrared radiograph of the jackrabbit when it had an elevated ear-pinna temperature after exercise. In this presentation, temperature is color-coded. The color blocks at the bottom symbolize increasing temperatures from left to right. The environment fell into the range of temperatures coded by green. Part of the right ear pinna was warm enough to fall within the much higher temperature range coded by yellow. (After Hill et al. 1980.)

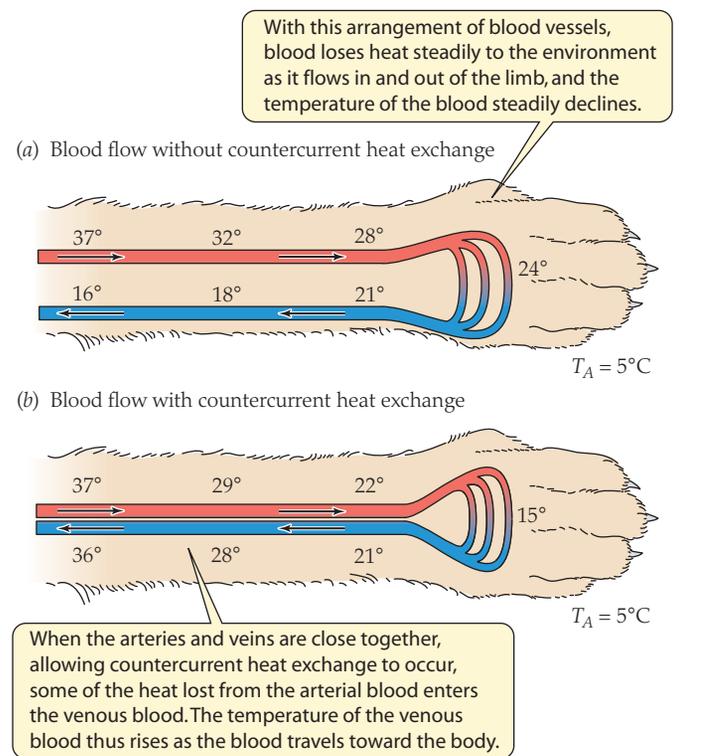
supply, for example. Heat flow to an appendage can be *selectively* curtailed by *countercurrent heat exchange*, a process that depends on a specialized morphological arrangement of the blood vessels that carry blood to and from the appendage.

To understand countercurrent heat exchange, let's examine the two different arrangements of the arteries and veins in a limb diagrammed in **Figure 10.35**. The arteries (red) are located deep within the appendage. In **Figure 10.35a** the veins (blue) are superficial, but in **Figure 10.35b** the veins are closely juxtaposed to the arteries. The vascular arrangement in **Figure 10.35a** does nothing to conserve heat; as blood flows into the appendage through the arteries and then flows back through the veins, it loses heat all along the way, without any opportunity to regain it. In contrast, the vascular arrangement in **Figure 10.35b** promotes heat conservation because it encourages a transfer of heat from the arterial blood to the venous blood; the venous blood then can carry that heat back to the body core, thereby helping to keep heat in the body. If the area of contact between the veins and arteries in **Figure 10.35b** is sufficiently extensive, blood may be little cooler when it reenters the body core in veins than it was when it flowed out into the appendage in arteries. The heat exchange in **Figure 10.35b** is **countercurrent heat exchange**. By definition, such heat exchange depends on the transfer of heat between two closely juxtaposed fluid streams flowing in opposite directions (*counter* = "opposite"; *current* = "flow").

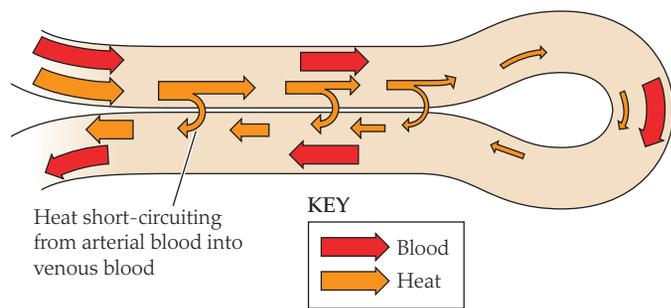
A particularly useful way to conceive of the effect of countercurrent heat exchange in an appendage is to think of it as *short-circuiting*

the flow of heat into the appendage. **Figure 10.36** illustrates that in the presence of a suitable vascular arrangement, although blood flows all the way to the end of an appendage before returning to the body core, heat tends to flow only part of the length of the appendage before it short-circuits from the arteries to the veins and starts its return to the body core. This short-circuiting impedes the access of heat to the outer extremities of the appendage. The outer extremities are therefore cooler than they otherwise would be, limiting heat loss to the environment.

A *vascular countercurrent exchange system* short-circuits the flow of only those commodities that are able to pass through the walls of the blood vessels involved. Heat is short-circuited by the vascular systems we have been discussing precisely because heat can pass through the walls of arteries and veins. If O<sub>2</sub>, nutrients, or wastes could pass through the walls of arteries and veins, they too would be short-circuited. However, they cannot pass through the walls of such thick-walled vessels, and thus they travel with the blood all the way to the outer limits of an appendage and back. This is how selectivity is achieved: This is how a vascular system can conserve heat while not affecting the flow of other commodities in and out of an appendage.



**FIGURE 10.35** Blood flow with and without countercurrent heat exchange. Arrows show blood flow in both parts. All temperatures are in degrees Celsius (°C). (a) In this arrangement, which does not permit countercurrent heat exchange, the veins (blue) returning blood from the limb are just under the skin and separate from the arteries (red) that carry blood into the limb. (b) In this case, countercurrent heat exchange can occur because the veins returning blood from the limb are closely juxtaposed to the arteries carrying blood into the limb. In part (b) the arterial blood is cooled more than in part (a) because of the close proximity of cool venous blood. Furthermore, in (b), more heat is returned to the body than in (a) because heat that enters the venous blood is carried back to the body rather than being lost to the environment.



**FIGURE 10.36** Countercurrent heat exchange short-circuits the flow of heat in an appendage. In a vascular countercurrent exchanger, commodities that can pass through the walls of the blood vessels short-circuit from one fluid stream to the other. Other commodities travel with the blood. This illustration shows heat being short-circuited. The widths of the arrows symbolize the relative magnitudes of heat flow and blood flow from place to place.

Vascular arrangements that meet the prerequisites for countercurrent heat exchange (close juxtaposition of arteries and veins) are commonly found in appendages that display regional heterothermy. Such vascular arrangements are known, for example, in the arms of humans, the legs of many mammals and birds, the flippers and flukes (tail fins) of whales, the tails of numerous rodents, and the ear pinnae of rabbits and hares. Anatomically the vascular arrangements vary from relatively simple to highly complex. The vessels in some cases are simply ordinary veins and arteries touching each other; this is the case in the human arm. Greater complexity is found in the flippers and flukes of whales, where the major arteries are almost *completely surrounded* by venous channels; with this arrangement, heat leaving the arteries virtually has to enter venous blood. Another type of complex arrangement found in some animals (e.g., armadillos and some storks) is that the main arteries and veins in a limb split up to form a great many fine vessels that intermingle. A complex network of tiny vessels like this is termed a **rete mirabile** (“wonderful net”) or simply a **rete**.<sup>35</sup>

A common way for countercurrent heat exchange to be controlled is for an appendage to have *two* sets of veins, only one of which is juxtaposed to the arteries. Countercurrent exchange can then be activated or deactivated by control of the set of veins in use. In the arm of a person, for example, one set of veins is deep in the arm and closely juxtaposed to the arteries, whereas a second set is just under the skin. Under control of the autonomic nervous system, the deep set of veins is used when there is a premium on heat conservation, but the superficial set is used when heat loss is advantageous. These controls explain why the superficial veins of our arms seem to disappear on cold days while bulging with blood on warm days.

### Mammals and birds in hot environments: Their first lines of defense are often not evaporative

Sweating, panting, and other modes of actively increasing the rate of evaporative cooling are so easy to observe when they occur that they are often thought to be the principal or only means by which

mammals and birds cope with high environmental or metabolic heat loads. Evaporation, however, has a potentially lethal price: It carries body water away. Although evaporative cooling may *solve* problems of temperature regulation, it may *create* problems of water regulation. For many mammals and birds, especially species that have long evolutionary histories in hot, arid climates, active evaporative cooling is in fact a *last* line of defense against heat loading. Other defenses are marshaled preferentially, and only when these other defenses have done as much as they can is body water used actively to void heat. In this section we discuss the nonevaporative defenses. When these defenses are employed as the preferential or first-line defenses, they act as water-conservation mechanisms.

*Behavioral defenses* are one set of commonly employed nonevaporative defenses. Desert rodents, for instance, construct burrows, which they occupy during the day (see Figure 1.16), and most emerge on the desert surface only at night. They thus evade the extremes of heat loading that could occur in deserts. Mammals and birds that are active during daylight hours often rest during the heat of the day, thereby minimizing their metabolic heat loads. Resting camels shift the positions of their bodies to present a minimum of surface area to the sun throughout hot days.

*Insulatory defenses* are also important nonevaporative defenses in some cases. For example, some species of large, diurnal mammals and birds native to hot, arid regions have evolved strikingly thick pelages and plumages. The dorsal pelage of dromedary camels in summer can be at least 5–6 cm thick, and when ostriches erect their plumage, it can be 10 cm thick. Such thick pelages and plumages probably evolved because in very hot environments they can act as heat shields, increasing body insulation and thereby acting as barriers to heat influx from the environment. The outer surface of the dorsal pelage of camels and sheep has been measured to get as hot as 50–80°C when exposed to solar radiation on hot days! The pelage shields the living tissues of the animals from these enormous heat loads.

*Body temperature* is a third nonevaporative attribute of mammals and birds that can be used in the first line of defense against the challenges of hot environments. Both high-amplitude cycling of body temperature and profound hyperthermia can act as defenses and, in fact, are commonly employed as water-conservation mechanisms by species adapted to hot environments.

**CYCLING OF BODY TEMPERATURE** Dromedary camels provide a classic and instructive example of how animals can employ high-amplitude cycling of body temperature as a nonevaporative defense and water-conservation mechanism in hot environments (see also Figure 30.11). A dehydrated dromedary in summer permits its deep-body temperature to fall to 34–35°C overnight and then increase to more than 40°C during each day. Its body temperature therefore cycles up and down by about 6°C. The advantage of such cycling is that it permits some of the heat that enters the body during the intensely hot part of each day to be temporarily *stored in the body* and later voided by nonevaporative rather than evaporative means. When dawn breaks on a given day, a camel’s body temperature is at its lowest level. As the day warms and the sun beats down on the camel, the animal simply lets heat accumulate in its body, rather than sweating to void the heat, until its body temperature has risen by 6°C. Physiologists have measured that about 3.3 J (0.8 cal) is required to warm 1 g of camel flesh by 1°C. From this figure,

<sup>35</sup>The word *rete* is pronounced with both syllables rhyming with sea: “ree-tee.” *Rete mirabile* and *rete* are general terms used to refer to intricately complex systems of small-diameter arterial and venous vessels wherever they occur. We will encounter many additional examples in this book.

one can calculate that a 400-kg camel will accumulate about 7920 kJ (1900 kcal) of heat in its body by allowing its body temperature to rise 6°C; to remove this amount of heat by evaporation would require more than 3 L of water, but the camel simply stores the heat in its body. Later, after night falls and the environment becomes cooler, conditions become favorable for convection and radiation to carry heat out of the camel's body. At that point the camel is able to get rid of the heat stored during the day by nonevaporative means. Its body temperature falls overnight to its minimum, poising the animal to take full advantage of heat storage during the following day, thereby again saving several liters of water.

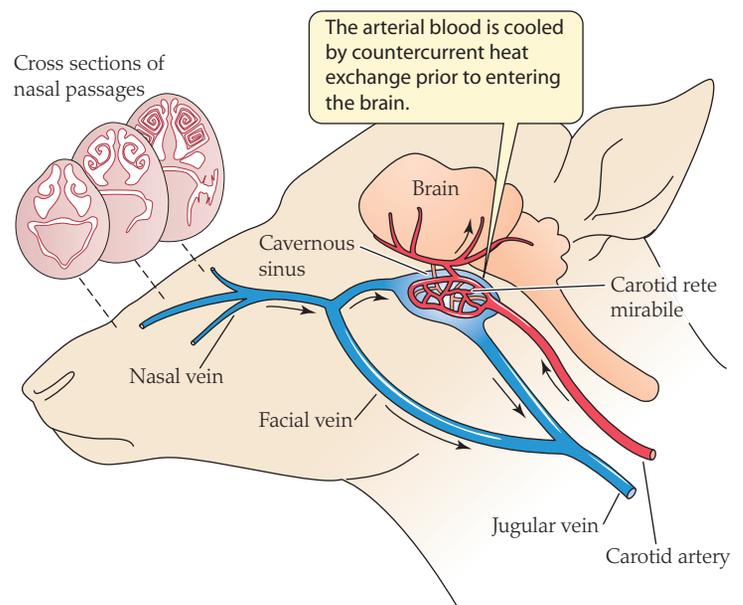
**HYPERTHERMIA** Many mammals and birds employ controlled, profound hyperthermia as a principal nonevaporative, water-conserving mechanism of coping with hot environments. Because a rise in body temperature entails heat storage, the benefits of hyperthermia are to some extent the very ones we have just noted in discussing cycling. In addition, however, a high body temperature *in and of itself* holds advantages for water conservation. As mentioned already, under conditions when dry heat *loss* occurs, a high  $T_B$  promotes such nonevaporative heat loss by elevating the driving force ( $T_B - T_A$ ) that favors it. A high  $T_B$  also aids water conservation under conditions when an animal has stored as much heat as it can and yet the environment is so hot that dry heat *gain* occurs. Under such conditions, evaporation of water must be used to get rid of all the heat that enters an animal's body. A high  $T_B$  impedes heat gain from the environment by decreasing the driving force ( $T_A - T_B$ ) that favors heat influx, and thus the high  $T_B$  reduces the rate at which body water must be evaporated to void the incoming heat.

Birds commonly permit their body temperatures to rise to profoundly high levels when in hot environments; whereas resting birds typically have body temperatures near 39°C in the absence of heat stress, they commonly have body temperatures as high as 43–46°C in hot environments. Among mammals, profound hyperthermia typically occurs only in species with long evolutionary histories in hot, arid climates, but among such species it is common. Certain antelopes native to the deserts and dry savannas of Africa provide the extreme examples. Two such species, the beisa oryx (*Oryx beisa*) and Grant's gazelle (*Gazella granti*), sometimes permit their rectal temperatures to reach 45.5–47°C (114–116°F) without ill effect!

**KEEPING A COOL BRAIN** A large body of evidence indicates that the brain is kept cooler than the thorax and abdomen in many species of mammals and birds when the animals are in warm or hot environments, especially during exercise. To cite an extreme example, when a Thomson's gazelle (*Gazella thomsonii*) runs vigorously in a warm environment, its brain is kept as much as 2.7°C cooler than its thorax. Camels, dogs, pronghorns, and sheep are other animals that exhibit brain cooling.

The advantage of brain cooling is believed to be that it permits an animal to take enhanced advantage of the benefits of high-amplitude body-temperature cycling and hyperthermia. The brain tolerates less elevation of temperature than most organs. Thus the bulk of an animal's body can cycle to a higher temperature, and become more hyperthermic, if the brain can be kept from becoming as hot as most of the body.

What is the mechanism of brain cooling? In many cases, the key process is cooling of the arterial blood supplying the brain by



**FIGURE 10.37** Structures hypothesized to be responsible for cooling the brain in sheep and other artiodactyls The carotid artery branches and anastomoses in the cavernous sinus, forming the carotid rete mirabile. Venous blood from the upper respiratory passages (e.g., nasal passages) flows around the vessels of the carotid rete. The insets above the snout show representative cross sections of the nasal passages of an artiodactyl (specifically, reindeer), illustrating that surface area in many species is greatly elaborated by folds and scrolls of tissue termed the *nasal turbinates*. The high surface area facilitates evaporation—and thus evaporative cooling of blood—in the nasal passages, as well as having other effects. (Principal drawing after Maloney and Mitchell 1997; turbinates after Johnsen 1988.)

countercurrent heat exchange (Figure 10.37). The arteries carrying blood toward the brain from the heart come into intimate contact with veins or venous blood draining the nasal passages and other upper respiratory passages. The site of this contact in many of the mammals involved is the cavernous sinus located at the base of the skull; there the arteries divide into a plexus of small vessels (the *carotid rete mirabile*) that is immersed in a lake of venous blood. As noted, the venous blood juxtaposed to the arteries is traveling back toward the heart from the upper respiratory passages. Blood in the upper respiratory passages is cooled by the inevitable evaporation of water from the walls of the respiratory passages into breathed air. As the cooled venous blood traveling back to the heart flows by the arteries, it cools the arterial blood traveling toward the brain.

### Active evaporative cooling is the ultimate line of defense against overheating

Active facilitation of evaporation is the ultimate line of defense for mammals and birds faced with high environmental or metabolic (e.g., exercise-induced) heat loads. If heat is accumulating in the body to excessive levels and all the other means we have already discussed fail to stop the accumulation, active evaporative cooling becomes the only mechanism available to reestablish a balance between heat gain and heat loss. As stressed earlier, the loss of water during evaporative cooling can dehydrate an animal if replacement water is not readily available; this probably explains why species native to arid habitats employ other defenses against overheating

before turning to evaporative cooling. Three major mechanisms of active evaporative cooling are known: sweating, panting, and gular fluttering.<sup>36</sup>

**SWEATING** During **sweating**, a fluid called *sweat* is secreted, by way of the ducts of sweat glands, through the epidermis of the skin onto the skin surface. Even when an animal is not sweating, water loss occurs through the substance of the skin—but at a low rate.<sup>37</sup> Sweating increases the rate of cutaneous evaporation by a factor of 50 or more by wetting the outer surface of the skin. Sweat is not pure water but instead is a saline solution. Concentrations of Na<sup>+</sup> and Cl<sup>-</sup> in sweat are lower than in the blood plasma, and during acclimation to hot conditions the salinity of sweat becomes reduced. Nonetheless, prolonged sweating can cause a significant depletion of the body's pool of Na<sup>+</sup> and Cl<sup>-</sup>. Secretion by the sweat glands is activated by the sympathetic nervous system.

A capability to sweat vigorously is found in a variety of mammals, including humans, horses, camels, and some kangaroos. Sweat production can be profuse. Humans working strenuously in the desert, for example, can attain sweating rates of 2 L/h! Many types of mammals, however, do not sweat. Rodents, rabbits, and hares lack integumentary sweat glands. Although dogs and pigs have sweat glands, the secretion rates of the glands are so low that sweating appears to play little or no role in thermoregulation. Birds do not sweat.

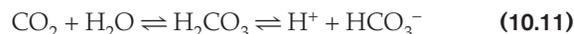
**PANTING** **Panting** is an increase in the rate of breathing in response to heat stress. It is common in both birds and mammals. Panting increases the rate of evaporative cooling because water evaporates from the warm, moist membranes lining the respiratory tract into the air that is breathed in and out.

In some species, the respiratory frequency (number of breaths per minute) during panting increases progressively as the extent of heat stress increases. In others, the respiratory frequency changes abruptly at the onset of panting, and within a wide range of thermal stress, the rate of breathing during panting is independent of the degree of heat stress. Dogs exemplify this second pattern; whereas in cool environments they breathe 10–40 times per minute, their respiratory frequency jumps abruptly to 200 or more breaths per minute when panting begins. Analysis indicates that animals with such a stepwise change in respiratory frequency often pant at the *resonant frequency* of their thoracic respiratory structures. At the resonant frequency, the thorax has an intrinsic tendency to “vibrate” between its inspiratory and expiratory positions. Thus less muscular work needs to be done—and less heat is produced by the muscular work—than at other frequencies.

By comparison with sweating, panting holds certain advantages. One is that no salts are lost during panting because evaporation occurs within the body and only pure water vapor leaves the body

in the exhalant air. A second advantage of panting is that it *forcibly* drives air saturated with water vapor away from the evaporative surfaces.

Panting also has liabilities in comparison with sweating. Because of the muscular effort required for panting, evaporation of a given quantity of water is likely to require more energy—and entail more heat production—when panting is employed than when sweating is. Another potential liability of panting is that it can induce *respiratory alkalosis*, an elevation of the pH of the body fluids caused by excessive removal of carbon dioxide (see page 645). Ordinarily, when animals are not panting, ventilation of the respiratory-exchange membranes deep in the lungs (e.g., the alveolar membranes of mammals) is closely regulated so that the rate at which CO<sub>2</sub> is voided is equal to the rate of metabolic production of CO<sub>2</sub>. During panting, the potential exists for breathing to carry CO<sub>2</sub> away faster than it is produced, because the rate of breathing is increased for thermoregulation rather than being governed only by metabolic needs. If CO<sub>2</sub> is carried away by breathing faster than it is produced by metabolism, the concentration of CO<sub>2</sub> in the blood will fall, causing the following reactions in the blood to shift to the left:



Consequently, the concentration of H<sup>+</sup> in the blood will fall, and the pH of the blood will rise. Such excessive alkalinity—*alkalosis*—can have major deleterious effects because many enzymes and cellular processes are acutely sensitive to pH. (In middle school, we probably all witnessed friends make themselves dizzy by deliberately breathing too rapidly.)

From extensive research, physiologists now know that little or no alkalosis develops during panting in many species of mammals and birds when the heat stress to which they are exposed is light to moderate. These animals avoid alkalosis by restricting the increased air movement during panting to just their *upper* airways,<sup>38</sup> where no exchange of CO<sub>2</sub> occurs between the air and blood (**Figure 10.38**); the respiratory-exchange membranes *deep* in the lungs receive about the same rate of airflow during panting as they usually do. By contrast, when heat stress becomes extreme, resting but panting animals often develop severe alkalosis. Some panting species have evolved superior tolerance to alkalosis.

**GULAR FLUTTERING** Many birds (but not mammals) augment evaporative cooling by rapidly vibrating their gular area (the floor of the buccal cavity) while holding their mouth open, a process termed **gular fluttering** (see Figure 10.30). The process is driven by flexing of the hyoid apparatus and promotes evaporation by increasing the flow of air over the moist, highly vascular oral membranes. Gular fluttering usually occurs at a consistent frequency, which apparently matches the resonant frequency of the structures involved. Birds commonly use gular fluttering simultaneously with panting.

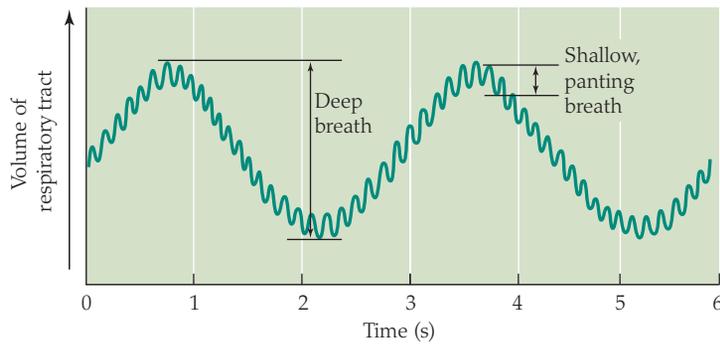
Gular fluttering shares certain positive attributes with panting: It creates a vigorous, forced flow of air across evaporative surfaces and does not entail salt losses. Unlike panting, gular fluttering cannot induce severe alkalosis, because it enhances only *oral* airflow, and CO<sub>2</sub> is not exchanged between air and blood across oral membranes.

<sup>36</sup>A fourth mechanism is **saliva spreading**, seen in many rodents and marsupials, which spread saliva on their limbs, tail, chest, or other body surfaces when under heat stress. Spreading of saliva on furred regions of the body is a relatively inefficient use of body water for cooling because the evaporative surface created—on the outer surface of the fur—is insulated from the living tissues of the animal's body by the pelage. For many rodents, however, saliva spreading is the only means available to increase evaporative cooling, and the animals use it in heat-stress emergencies.

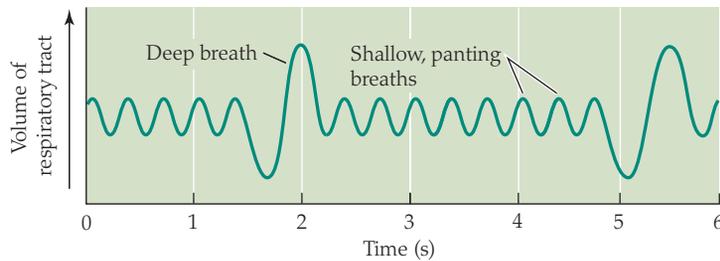
<sup>37</sup>Water lost through the skin in the absence of sweating is termed *transpirational water loss* or *insensible* (“unperceived”) *water loss*.

<sup>38</sup>In birds, both the upper airways and air sacs may be involved.

(a) Shallow breaths superimposed on deep breaths (compound breathing)



(b) Shallow breaths occasionally interrupted by deep breaths



**FIGURE 10.38 Two breathing patterns that limit hyperventilation of the respiratory-exchange membranes during panting** Both of these breathing patterns permit the upper airways to receive a greatly increased flow of air during panting while simultaneously the respiratory-exchange membranes deep in the lungs are ventilated about as much as usual. In (a), shallow breaths are superimposed on deep breaths. In (b), periods of shallow breathing alternate with periods of deep breathing.

Gular fluttering involves the movement of structures that are less massive than those that must be moved in panting; thus it entails less muscular work—and less heat production—to achieve a given increment in evaporation.

### Mammals and birds acclimatize to winter and summer

When individual mammals and birds live chronically in cold or warm environments, they usually undergo long-term alterations in their thermoregulatory physiology. During acclimatization to winter, for example, a mammal or bird typically exhibits one or more of three sorts of chronic responses, discussed in this section.<sup>39</sup> Because the change of seasons is complex, these responses are not necessarily triggered solely (or even primarily) by the drop in temperature as winter approaches, but may be triggered by photoperiod (shortening day length) or other seasonal cues.

One possible chronic response to the approach of winter is **acclimatization of peak metabolic rate**. When a mammal or bird exhibits this response, it increases the maximal rate at which it can produce heat by sustained, aerobic catabolism. If an animal displays *only* this sort of acclimatization, the metabolic rate it requires to thermoregulate at any given ambient temperature remains un-

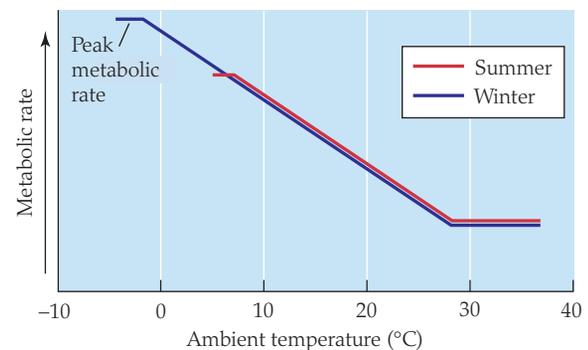
<sup>39</sup> Acclimatization to summer, to some extent, involves simply the same chronic responses in reverse. *Acclimation* of mammals or birds to cold in a laboratory sometimes has dramatically different effects than *acclimatization* to winter has (see page 15 for the distinction). Results of laboratory acclimation studies on mammals and birds, therefore, cannot confidently be extrapolated to acclimatization in nature. One reason for the difference between acclimation to cold and acclimatization to winter is that some of the winter responses occur only when there are changes in photoperiod, and usually just temperature is altered in laboratory studies.

changed, but it can thermoregulate in colder environments than it could before, as shown by **Figure 10.39a**. The development in winter of enlarged brown adipose tissues in which cells are biochemically especially poised for heat production is a common mechanism by which small and medium-sized mammals increase the rate at which they can produce heat and thus undergo acclimatization of peak metabolic rate.

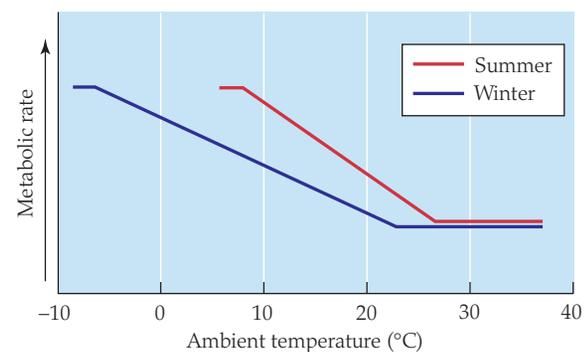
A second possible chronic response to the approach of winter is **acclimatization of metabolic endurance**, meaning an increase in the length of time that a high rate of metabolic heat production can be maintained. Although current evidence indicates that this sort of acclimatization is common, little is known about its mechanisms.

The third major sort of chronic response that a mammal or bird might exhibit in winter is **insulatory acclimatization**, an increase in the animal's maximal resistance to dry heat loss (maximal insulation). If this sort of acclimatization occurs, the metabolic rate required to thermoregulate at any particular ambient temperature below thermoneutrality is reduced. Accordingly, even if an animal's peak metabolic rate remains unchanged, the animal is able to thermoregulate in colder environments than it could before (**Figure 10.39b**). The most obvious way for insulatory acclimatization to occur is for an animal to molt into a more protective pelage or plumage in winter, but other determinants of insulation (such as peripheral blood flow) can also change. Of the three chronic responses to winter we have described, two—or all three—can occur together.

(a) Acclimatization of peak metabolic rate

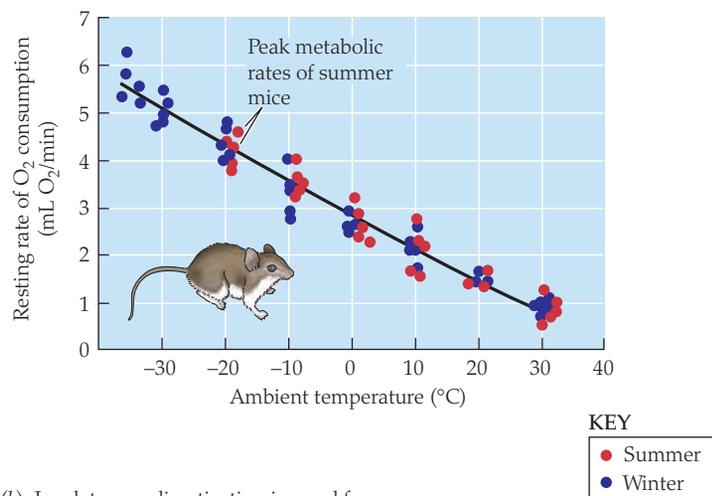


(b) Insulatory acclimatization

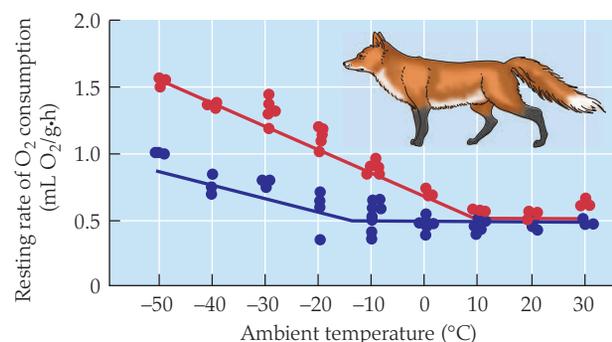
**FIGURE 10.39 Two types of seasonal acclimatization**

(a) Acclimatization of peak metabolic rate without insulatory acclimatization. (b) Insulatory acclimatization without acclimatization of peak metabolic rate. The plateau at the left of each curve indicates where metabolic rate has peaked.

(a) Acclimatization of peak metabolic rate without insulatory acclimatization in deer mice



(b) Insulatory acclimatization in a red fox



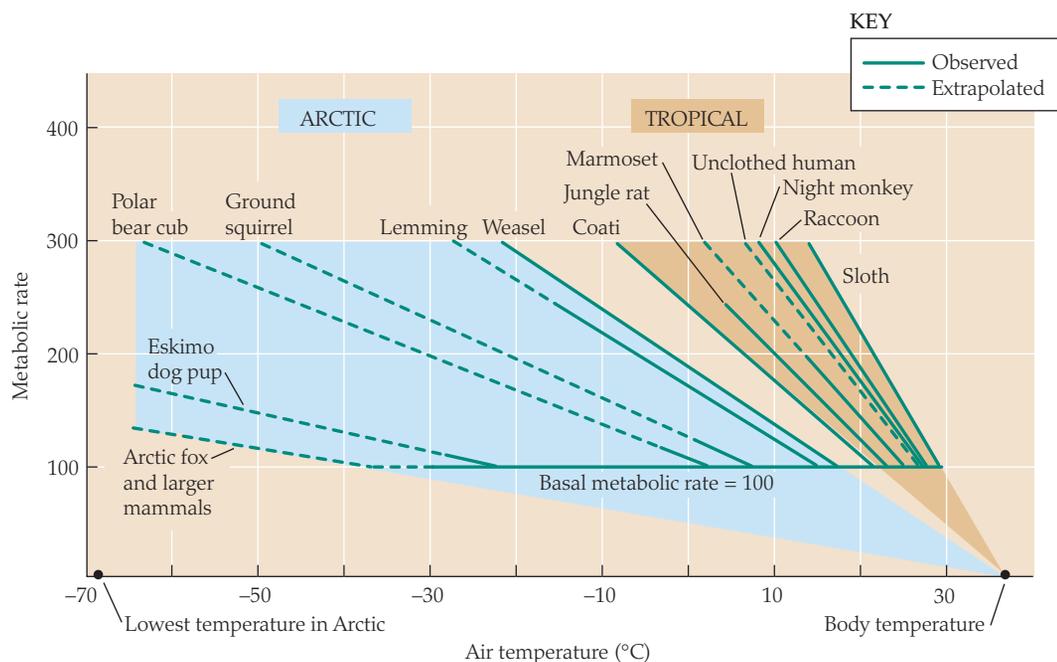
**FIGURE 10.40** Seasonal acclimatization in two species of mammals (a) The deer mice (*Peromyscus maniculatus*) studied had the same insulation in winter and summer, but their peak metabolic rates rose in winter, meaning they could thermoregulate at lower ambient temperatures. (b) A single red fox (*Vulpes vulpes*) individual, studied in both seasons, had far greater insulation in winter than in summer. (After Hart 1957.)

Acclimatization of peak metabolic rate occurs in perhaps half of small birds and is the norm in small and medium-sized mammals. As for insulatory acclimatization, some small-bodied species of mammals and birds fail to exhibit it and thus undergo only metabolic forms of acclimatization (Figure 10.40a). Among the mammals that undergo insulatory acclimatization, medium-sized and large species tend to exhibit greater changes in insulation between summer and winter than do small species. Red foxes (Figure 10.40b), collared lemmings, and varying hares in northern Alaska all exhibit substantial increases in insulation in winter. The air temperature in northern Alaska averages  $-30^{\circ}\text{C}$  in winter and  $+5^{\circ}\text{C}$  in summer. For the foxes, lemmings, and hares, the metabolic cost of thermoregulating at  $-30^{\circ}\text{C}$  in winter is little higher than the cost of thermoregulating at  $+5^{\circ}\text{C}$  in summer, because of their winter increase in insulation (see Figure 10.40b).

Mammals and birds commonly acclimatize to heat stress as well as cold stress, as seen in Chapter 1 (see Figure 1.8). Among humans, acclimatization to heat stress occurs much more rapidly than that to cold stress. Partly for that reason, we tend to notice our own acclimatization to heat more than our acclimatization to cold.

### Evolutionary changes: Species are often specialized to live in their respective climates

Abundant evidence indicates that the thermoregulatory physiology of mammals and birds has undergone evolutionary adaptation to different climates. One sort of evidence is shown in Figure 10.41, which is one of the classic sets of data in animal physiology. As the figure shows, species of mammals native to the Arctic and the tropics differ dramatically in their thermal relations; Arctic species—compared with tropical species—have lower-critical temperatures that are lower (i.e., they have broader TNZs), and they increase their metabolic rates proportionally less above basal levels at ambient temperatures below thermoneutrality. Direct studies of pelage insulation demonstrate that the Arctic species typically have thicker and better insulating pelages than do similarly sized tropical species. As a consequence of all these differences, Arctic



**FIGURE 10.41** Mammalian physiological specialization to different climates

Species found in the Arctic (Alaska) expend less energy to thermoregulate at cold ambient temperatures, and they can thermoregulate at lower temperatures, than species found in the tropics (Panama) can. In this presentation, each species' basal metabolic rate is set equal to 100, and metabolic rates outside the thermoneutral zone are expressed relative to basal; this convention facilitates comparison in certain ways but means that the slopes of the metabolism-temperature curves below thermoneutrality can be used in only a qualitative way to compare insulation. (After Scholander et al. 1950.)

species are in a far better position to thermoregulate under Arctic conditions than tropical species are. Arctic foxes (*Alopex*) and reindeer (*Rangifer*) have lower-critical temperatures in winter that are below  $-30^{\circ}\text{C}$ ! This means that they can maintain homeothermy with only their basal rates of heat production until the air becomes exceedingly cold.

In hot climates, a major pattern that has emerged with ever-increasing clarity in recent decades is that species of both mammals and birds native to such climates often have lower basal metabolic rates than are observed in related species native to temperate or cold climates. The evolution of an exceptionally low BMR has probably been favored in hot climates because, with a low BMR, an animal has a particularly low internal heat load.

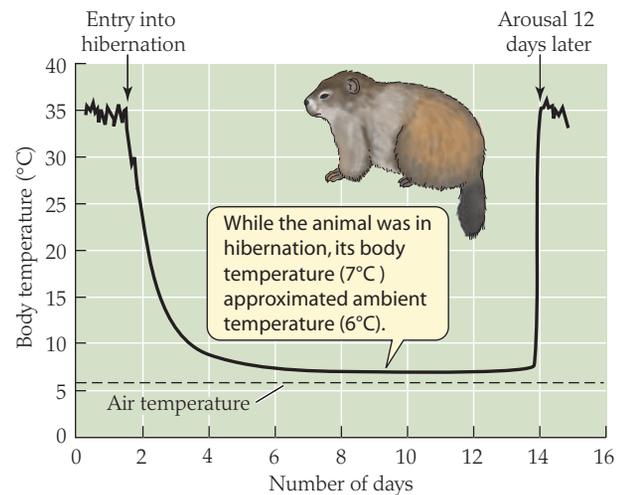
As mentioned earlier, body temperature is basically a *conserved* character; within any taxonomic group of mammals or birds, the core body temperature maintained in the absence of heat or cold stress tends to be the same in species from various climates. Adaptation of body temperature to climate is clearly evident, however, in one specific respect among mammals exposed to heat stress: Mammal species native to hot climates typically tolerate greater degrees of hyperthermia than species native to temperate or cold climates do.

### Mammals and birds sometimes escape the demands of homeothermy by hibernation, torpor, or related processes

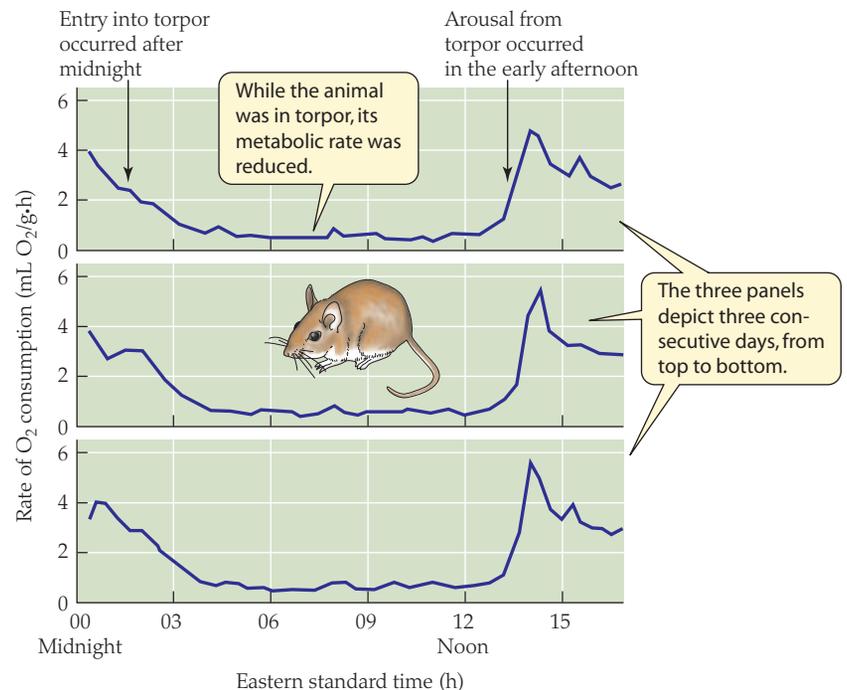
Many species of mammals and birds allow their body temperatures to fall in a controlled manner under certain circumstances. **Controlled hypothermia** is a general term for this sort of phenomenon; **hypothermia** is the state of having an unusually low body temperature, and in the cases we are discussing, it is “controlled” because the animals orchestrate their entry into and exit from hypothermia rather than being forced.

The most well known and profound forms of controlled hypothermia are **hibernation**, **estivation**, and **daily torpor**. According to definitions that have been in place for several decades, these are all states in which an animal *allows its body temperature to approximate ambient temperature within a species-specific range of ambient temperatures*.<sup>40</sup> Hibernation, estivation, and daily torpor are generally viewed as being different manifestations of a *single* physiological process. They are distinguished by differences in their durations and seasons of occurrence. When an animal allows its body temperature to fall close to ambient temperature for periods of several days or longer during winter, the process is termed *hibernation*. When this occurs during summer, it is called *estivation*. When an animal permits its body temperature to fall close to ambient temperature for only part of each day (generally on many consecutive days), the process is termed *daily torpor* in any season. **Figures 10.42** and **10.43** illustrate the sorts of changes in body temperature and metabolic rate that occur in episodes of controlled hypothermia.

Hibernation, estivation, and daily torpor permit mammals and birds to escape the energy demands of homeothermy. As stressed earlier, homeothermy is energetically costly. A hamster, for example, needs to acquire and consume a great deal of food energy to keep its body temperature at



**FIGURE 10.42** Changes in body temperature during hibernation A woodchuck (groundhog) (*Marmota monax*) was implanted with a small temperature transmitter that broadcast its body temperature continuously, and after it healed from the surgery, it was studied at an air temperature of  $6^{\circ}\text{C}$ . The record shows its body temperature during a 12-day episode of hibernation. (After Armitage et al. 2000.)

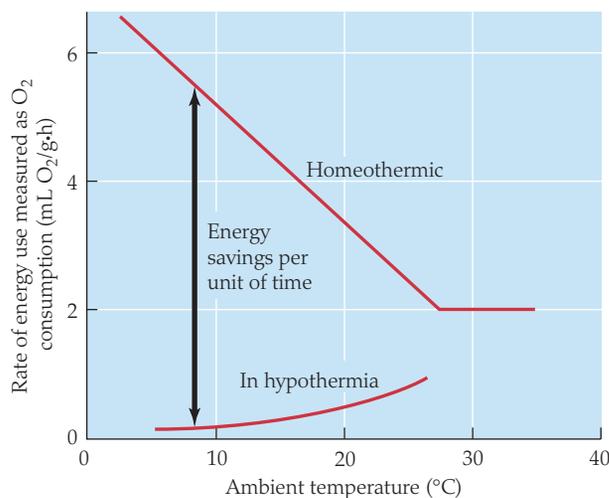


**FIGURE 10.43** Changes in metabolic rate during daily torpor The rate of  $\text{O}_2$  consumption of a white-footed mouse (*Peromyscus leucopus*) studied at an air temperature of about  $14^{\circ}\text{C}$  is shown for three consecutive days. The x axis shows time of day on a 24-h scale (e.g., 15 = 3:00 pm). The animal required a resting metabolic rate of about  $3.0 \text{ mL O}_2/\text{g}\cdot\text{h}$  to be able to maintain high body temperatures. It underwent a prolonged episode of daily torpor on each day, as indicated by the drop in its metabolic rate. Its body temperature measured during an episode of torpor was  $17^{\circ}\text{C}$ . (After Hill 1975.)

<sup>40</sup>We return to definitions at the end of this section.

37°C when the temperature of its environment is near freezing. If the hamster abandons homeothermy and temporarily allows its body temperature to fall close to ambient temperature, it is temporarily freed of homeothermy's energy costs. Animals capable of hibernation, estivation, or daily torpor are in essence able to switch back and forth between two very different thermal worlds. They are *temporal heterotherms*. When they function as ordinary homeotherms do, they reap the benefits of homeothermy, such as physiological independence of external thermal conditions; but they pay the high energy cost. When they suspend homeothermy, they take on many of the attributes of poikilotherms: Their tissues are subjected to varying tissue temperatures, but they have low energy needs.

Quantitatively, the amount of energy saved by controlled hypothermia depends on the ambient temperature at which hypothermia occurs and the duration of the hypothermia. To elucidate the importance of ambient temperature, **Figure 10.44** shows the different metabolism–temperature relations that exist in a single species when the animals are homeothermic and when they are in controlled hypothermia. At any given ambient temperature, the difference between the two curves shows the degree to which animals can reduce their energy costs per unit of time by entering hypothermia; the amount of energy saved per unit of time becomes greater as the ambient temperature falls. If a hibernating animal remains in hibernation at low ambient temperatures for long periods of time, its total energy savings can be enormous. For example, free-living ground squirrels of at least two species, living in cold climates, have been measured to expend only 10–20% as much energy per month by hibernating as they would if they failed to



**FIGURE 10.44 Energy savings depend on temperature** In common with other species that undergo hibernation, estivation, or daily torpor, kangaroo mice (*Microdipodops pallidus*) alternate between two metabolism–temperature relations, shown here. The double-headed arrow shows how much a kangaroo mouse's rate of energy use is reduced when the animal is in hypothermia instead of being homeothermic. The amount of energy saved by being in hypothermia is greater at low ambient temperatures than at higher ones because the metabolic cost of homeothermy is particularly high at low ambient temperatures, whereas the cost of hypothermia is particularly low at low ambient temperatures. At ambient temperatures between 5°C and 25°C, the body temperatures of these mice when they are in hypothermia are just 1.0–3.5°C higher than ambient. (After Brown and Bartholomew 1969.)

hibernate, and they reap these monthly savings throughout their 7- to 8-month hibernating seasons.

Controlled hypothermia also permits mammals and birds to escape the high water demands of homeothermy. This point is not as widely significant as the escape from energy demands, because the escape from water demands matters only for animals that face water shortages. Sometimes, nonetheless, the escape from water demands can be the most important consequence of entering controlled hypothermia; this is especially true for animals that enter estivation or daily torpor in hot, dry environments. As we will discuss in detail in Chapter 28, homeotherms have relatively high rates of water loss. One reason is simply that they must breathe rapidly to acquire the amounts of O<sub>2</sub> they need for their high metabolic rates. Another is that the air they exhale tends to be relatively warm, and warm air holds more water vapor (which is exhaled with the air) than cooler air. Entry into controlled hypothermia reduces an animal's rate of water loss by reducing both (1) its breathing rate and (2) the temperature, and therefore the water vapor content, of its exhaled air.

#### WHAT ARE THE MECHANISMS BY WHICH METABOLIC RATE IS LOWERED DURING CONTROLLED HYPOTHERMIA?

Recent research has established that—in at least some mammalian hibernators—*biochemical downregulation of metabolism* takes place during hibernation. Until about 20 years ago, the almost-universal view was that animals initiate their entry into controlled hypothermia simply by turning off thermoregulation. According to this view, the sequence of events during entry into hypothermia is that thermoregulation is deactivated, body temperature falls because of cooling by the environment in the absence of thermoregulatory responses, and tissue metabolic rates then decline because the tissues cool. This sort of lowering of metabolic rate—driven by tissue cooling and therefore following the  $Q_{10}$  principle (see Equation 10.7)—is often described as a “ $Q_{10}$  effect.” The newer view is that the first step in the sequence of events during entry into hypothermia is biochemical downregulation of tissue metabolism, and body temperature then falls as a consequence of the reduced metabolic rate. In this sequence of events, after biochemical downregulation initiates the fall of body temperature, the declining body temperature can potentially exert a  $Q_{10}$  effect that reinforces the biochemical downregulation in depressing metabolism.

The evidence currently available indicates that both of the sequences of events discussed are observed during controlled hypothermia in mammals. One recent analysis identifies a divergence between species that undergo only daily torpor and those that hibernate. According to this analysis, the former tend to employ just a  $Q_{10}$  effect (a drop in tissue temperature) to suppress metabolism, whereas the latter tend to employ biochemical downregulation to suppress metabolism, plus potentially a  $Q_{10}$  effect. In some hibernators, the metabolic rate during hibernation is determined by biochemical controls in a way that body temperature, over wide ranges, does not matter.

#### IN WHAT RESPECTS IS “CONTROLLED” HYPOTHERMIA CONTROLLED?

Mammals and birds that display controlled hypothermia orchestrate their entry into and exit from hypothermia, and they exhibit control over their situation in other respects as well. The most dramatic evidence of the controlled nature of hibernation, estivation,

and daily torpor is the fact that animals are able to arouse from these conditions. **Arousal** is the process of rewarming the body by metabolic heat production. The animals do not require outside warming to return to homeothermy. Instead, they are in control: They return to homeothermy on their own by employing intense shivering and, in mammals, intense nonshivering thermogenesis to warm their tissues. All episodes of controlled hypothermia end with arousal. In addition, hibernating animals universally undergo periodic, short arousals during the period of time they are hibernating; for instance, an animal that hibernates for 6 months might arouse for a few hours every 14 days or so. The possible functions of periodic arousals are discussed in Chapter 11.

A second, particularly fascinating sort of control exhibited by animals in controlled hypothermia is the control they display when their body temperatures start to fall too low. Each species that undergoes hibernation, estivation, or daily torpor has a species-specific range of body temperatures that it can tolerate, and for an animal to survive hypothermia, it must respond if its body temperature starts to go below the tolerable range. *Within* the tolerable range, animals typically let their body temperatures drift up and down as the ambient temperature rises and falls. For instance, if an animal can tolerate a  $T_B$  as low as  $3^\circ\text{C}$  and the  $T_A$  varies between  $5^\circ\text{C}$  and  $15^\circ\text{C}$ , the animal typically allows its  $T_B$  to vary as the  $T_A$  varies (always being a bit higher than the  $T_A$ ). What happens, however, if the ambient temperature falls below  $3^\circ\text{C}$ ? Frequently (although not always), the animal exerts control in one of two life-preserving ways. It may arouse. Alternatively and more remarkably, it may start to *thermoregulate* at a reduced body temperature, its thermoregulatory control system functioning with a lowered set point. For example, an animal that must stay at a body temperature of at least  $3^\circ\text{C}$  to survive may keep its body temperature at  $3^\circ\text{C}$  even if the ambient temperature drops to  $-10^\circ\text{C}$  or  $-20^\circ\text{C}$ , increasing its metabolic rate as the ambient temperature falls so as to offset the increasing cooling effect of the air (see Figure 11.11).<sup>41</sup>

**DISTRIBUTION AND NATURAL HISTORY** Hibernation is known to occur in at least six different orders of mammals. Species that hibernate include certain hamsters, ground squirrels, dormice, jumping mice, marmots, woodchucks, bats, marsupials, and monotremes. Because of its seasonal nature, hibernation is often preceded by long-term preparation. Hibernating mammals, for instance, typically store considerable quantities of body fat during the months before their entry into hibernation (see Figure 6.25). Hibernation is rare in birds; it may occur in only a single species, the poorwill (*Phalaenoptilus nuttallii*). We discuss mammalian hibernation at considerably more length in Chapter 11.

Estivation is not nearly as well understood as hibernation, partly because it is not as easy to detect. It has been reported mostly in species of desert ground squirrels.

Daily torpor is widespread among both mammals and birds, and it occurs not only in species facing cold stress but also in species occupying tropical or subtropical climates. It occurs in numerous species of bats and rodents and in certain hummingbirds, swallows, swifts, and caprimulgid birds (e.g., nightjars and poorwills). Animals undergoing daily torpor are homeothermic for part of

each day, and they feed during their homeothermic periods. When bats are undergoing daily torpor, they become hypothermic during daylight hours and forage at night; hummingbirds, in contrast, become torpid at night and feed in daylight. In some species, the proclivity to enter daily torpor is seasonally programmed. However, daily torpor seems to be employed most commonly, regardless of season, as an immediate response to hardship. Many species, for example, undergo daily torpor only when they are suffering food shortage; in some cases they increase the length of time they spend in torpor each day as food shortage becomes more severe.

**CONTROLLED HYPOTHERMIA IN WHICH THE BODY TEMPERATURE REMAINS WELL ABOVE AMBIENT TEMPERATURE**

Over the last 30 years, there has been an escalating realization that many species of small birds undergo hypothermia without ever allowing their body temperatures to approximate ambient temperature. Black-capped chickadees (*Poecile atricapillus*) provide an excellent example. They sometimes allow their core body temperature to fall by roughly  $7^\circ\text{C}$  while sleeping overnight in freezing-cold winter weather. They then have body temperatures ( $31\text{--}34^\circ\text{C}$ ) that are distinctly hypothermic but nonetheless far above ambient temperature. This hypothermia does not eliminate their need to expend metabolic energy to stay warm. However, because the hypothermia reduces the difference between body temperature and ambient temperature, the birds lose heat more slowly—and have lower costs for thermoregulation—than if they maintained higher body temperatures. Chickadees do not feed at night and are so small (11 g) that they may virtually exhaust their body fat in a single night of frigid weather; their hypothermia helps them survive until they can feed the next day.

A variety of mammals also exhibit subtle forms of hypothermia in which core body temperature falls to only a relatively small extent, or it may even happen that the core temperature remains unaltered, but the extent of peripheral regional hypothermia increases. Bears of some species are the most famous examples of mammals exhibiting moderate core hypothermia.

**HIBERNATING BLACK BEARS** Until recently, many physiologists doubted that black bears (*Ursus americanus*) should be considered hibernators because, although they allow their core body temperature to fall for 5–7 months in winter, their body temperature stays at  $30^\circ\text{C}$  or higher and therefore is far above ambient temperature, violating the traditional criterion for hibernation. Bears stand out because of their size. Nearly all hibernating species of mammals weigh 5 kg or less, whereas black bears weigh 30–150 kg. Recent research has revealed that black bears, during their winter hypothermia, exhibit a dramatic degree of biochemical downregulation of metabolism, so much so that their metabolic rates are only one-quarter as high as their basal rates; metabolic downregulation is the principal control of their metabolic rates, with body temperature ( $Q_{10}$  effect) playing little role. Under these circumstances, despite their body temperatures being only mildly hypothermic, the bears have weight-specific metabolic rates similar to those of most hibernators. Hibernation physiologists now, therefore, rank the black bear as a specialized hibernator. Its size may preclude it from reaching a body temperature as low as ambient temperature even though it metabolically resembles other hibernators.

<sup>41</sup>This phenomenon features prominently in the prediction of bat hibernation ranges discussed in Box 10.1.

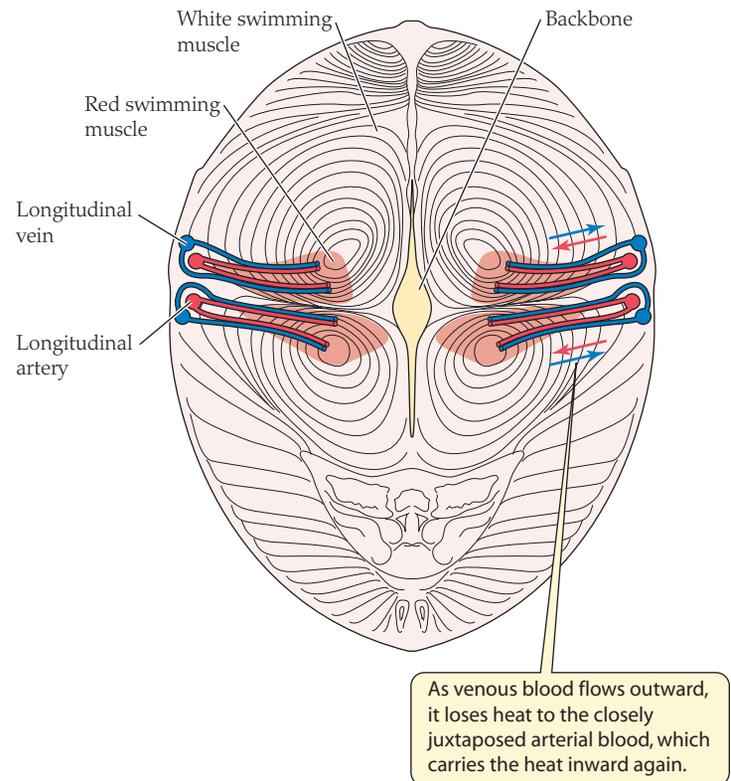
## SUMMARY Homeothermy in Mammals and Birds

- Homeothermy—thermoregulation by physiological means—is energetically expensive.
- The principal way that a mammal or bird thermoregulates in its thermoneutral zone is that it varies its body insulation to offset changes in the driving force for dry heat loss ( $T_B - T_A$ ). Insulation can be modulated by changes in posture, cutaneous blood flow, the thickness of the relatively motionless air layer trapped by the pelage or plumage, and regional heterothermy.
- Below thermoneutrality, variation in the rate of metabolic heat production (thermogenesis) is the principal mechanism of thermoregulation. The two most prominent mechanisms of increasing heat production are shivering—found in both mammals and birds—and nonshivering thermogenesis (NST)—found mainly in placental mammals. The principal site of NST in mammals is brown adipose tissue, which, by expressing uncoupling protein 1, is able to employ uncoupling of oxidative phosphorylation to achieve very high rates of lipid oxidation with immediate heat release.
- Regional heterothermy, which is often exhibited when animals are at ambient temperatures below thermoneutrality, usually depends on countercurrent heat exchange. Close juxtaposition of arteries and veins short-circuits the flow of heat into appendages.
- Above thermoneutrality, species with long evolutionary histories in hot, dry environments typically use nonevaporative mechanisms—notably hyperthermia and cycling of body temperature—as first lines of defense. When active evaporative cooling occurs, the specific mechanisms usually employed to increase the rate of evaporation are sweating (only in certain mammals), panting (mammals and birds), and gular fluttering (only birds). Both hyperthermia and the effort involved in active evaporative cooling can cause metabolic rate to rise at ambient temperatures above thermoneutrality.
- Acclimatization to changing seasons is the norm and may involve one or more of three mechanisms: acclimatization of peak metabolic rate, acclimatization of metabolic endurance, and insulatory acclimatization.
- Controlled hypothermia permits animals to evade temporarily the high energy costs and water costs of homeothermy. During hibernation, estivation, and daily torpor,  $T_B$  is generally allowed to fall close to  $T_A$  within a species-specific range of  $T_A$ . Forms of shallow hypothermia also occur.

## Warm-Bodied Fish

The body temperatures of 99% of all species of fish closely approximate water temperature. However, in tunas, lamnid sharks, and billfishes, temperatures within *certain body regions* exceed water temperature, sometimes substantially. All the warm-bodied fish are large, streamlined, fast-swimming predators that lead wide-ranging lives and feed on such speedy prey as squid and herring. The lamnid sharks include the great white shark, mako, porbeagle, and salmon shark. The billfishes include the marlins and swordfish.

In tunas and lamnid sharks, the red (dark) swimming muscles are warmed above water temperature.<sup>42</sup> These muscles provide the

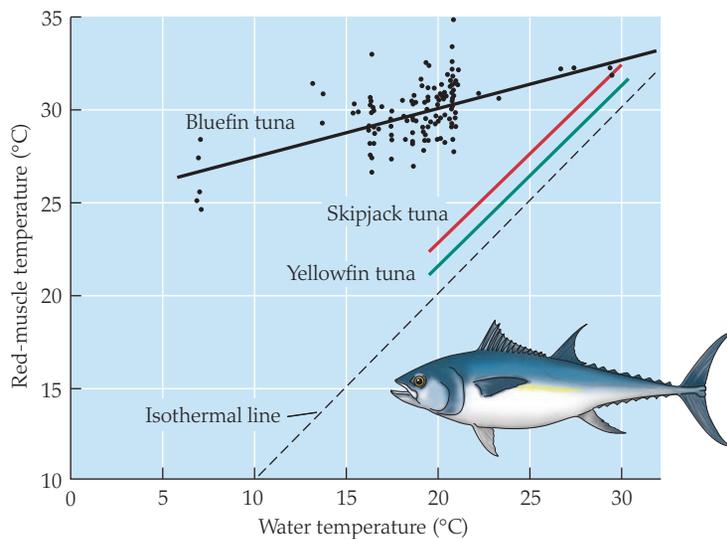


**FIGURE 10.45** A cross section of a tuna showing the nature of the blood supply to the red swimming muscles. The longitudinal arteries, which carry blood along the length of the body, give off small arteries that penetrate (toward the backbone) into the muscles. Small veins running in close juxtaposition to the small arteries return blood peripherally to the longitudinal veins, which lead back to the heart. Red vessels and arrows refer to arterial blood flow; blue vessels and arrows refer to venous flow.

power for steady swimming in these vigorously active animals, and the contractile activity of the muscles produces the heat that warms the muscles. A critical principle to recognize, however, is that *a high rate of heat production is never in itself adequate to elevate tissue temperature in water-breathing animals*. If metabolic heat is carried freely to the gills by the circulation of the blood, the heat is lost so readily to the surrounding water across the gills that no significant elevation of body temperature can occur. Thus, for a region of the body to be warmed, transport of heat away from that body region by the circulation must be impeded. Not only in the red swimming muscles of tunas and lamnid sharks, but universally in warm-bodied fish, the mechanism of impeding heat loss is countercurrent heat exchange.

The vasculature of the red swimming muscles in tunas and lamnid sharks is diagrammed in **Figure 10.45**. Note that the red muscles are found deep in the body near the spinal column in these fish (an unusual placement compared with that in most other fish). The major longitudinal arteries and veins that carry blood along the length of the body, to and from the swimming muscles, run just under the skin on each side of the body (again, an unusual placement). Small arteries branch off from the longitudinal arteries and penetrate inward to the red muscles. In turn, blood is brought outward from the muscles in veins that discharge into the longitudinal veins leading back to the heart. The arteries carrying blood inward to the red swimming muscles and the veins carrying blood outward from those muscles are closely juxtaposed, forming

<sup>42</sup>The roles of the red and white muscles in powering swimming in fish are discussed in Chapter 8 (see page 196).



**FIGURE 10.46 Red-muscle temperatures of tunas at various ambient water temperatures** The upper line and data points are for wild bluefin tunas (*Thunnus thynnus*) captured in waters of various temperatures. The other two solid lines show the average relation between red-muscle temperature and water temperature in small, captive skipjack tunas (*Katsuwonus pelamis*) and yellowfin tunas (*Thunnus albacares*) swimming in an aquarium; larger, wild fish of these species are sometimes observed to exhibit greater temperature differentials between muscles and water (e.g., 5–10°C in skipjacks). The isothermal line shows how tissue temperature would vary with water temperature if there were no endothermy and tissue temperature simply equaled water temperature. The fish shown is a bluefin tuna. (After Carey and Teal 1969; Dizon and Brill 1979.)

countercurrent exchange networks. Figure 10.45 is highly simplified in the way it presents these networks. In actuality, the arteries and veins going to and from the red muscles branch profusely, forming thick layers of vascular tissue in which huge numbers of minute arterial and venous vessels, each only about 0.1 mm in diameter, closely intermingle—a true rete mirabile (see page 260). Because of the countercurrent-exchange arrangement, much of the heat picked up by the venous blood in the red muscles is transferred to the ingoing arterial blood rather than being carried by the venous flow to the periphery of the body and the gills, where it would readily be lost to the water. Thus heat produced by the red swimming muscles tends to be retained within them.

Bluefin tunas, which reach body weights of 700 kg and are the largest of all tunas, maintain fairly constant red-muscle temperatures over a wide range of water temperatures (Figure 10.46). In most other tunas, such as the yellowfin and skipjack tunas, red-muscle temperature is elevated over water temperature by a relatively constant amount regardless of the water temperature. Referring back to our scheme for classifying animal thermal relations (see Figure 10.1), all the tunas are endotherms, but species differ in whether they also thermoregulate. Whereas yellowfin and skipjack tunas are endotherms without being thermoregulators,<sup>43</sup> bluefin tunas are endothermic thermoregulators (homeotherms).

The warming of the red swimming muscles in tunas and lamnid sharks is generally thought to aid power development and locomotory performance, although exactly how is debated. Any aid to the

performance of the swimming muscles would be significant for fish that are so dependent on high-intensity exertion for their livelihood.

The swimming muscles are not the only tissues kept warm in tunas and lamnid sharks. In certain species, the stomach and other viscera are warmed when food is being digested. The brain and eyes are also warmed in some species. Each warmed organ is served by arteries and veins that form a rete mirabile, which short-circuits the outflow of heat produced in the organ, thereby favoring heat accumulation in the organ.

Now let's turn to the billfishes. They differ in two ways from the tunas and lamnid sharks. First, in the billfishes, only the brain and the retinas of the eyes are warmed. Second, the billfishes possess "heater tissues" specialized for exceptional heat output.<sup>44</sup>

The heater tissues of billfishes are derived from portions of the extraocular eye muscles (the muscles on the outside of each eyeball that serve to turn the eyeball to look in various directions). These portions of the muscles have lost most of their contractile apparatus and are very rich in mitochondria. Current evidence suggests that they produce heat at a high rate by a "futile cycle" of  $\text{Ca}^{2+}$  pumping: ATP is used to transport  $\text{Ca}^{2+}$  actively from one intracellular compartment to another, and then the  $\text{Ca}^{2+}$  leaks back to where it started, where once again ATP is used to pump it; the principal net result is breakdown of ATP at a high rate to release heat. The heat produced by the heater tissues is retained in the head by countercurrent vasculature and in that way warms the brain and retinas. If warming of the brain by specialized eye muscles sounds impossible, remember that in a fish, the eyes and eye muscles are far larger than the brain! Warming of the brain and the retinas is hypothesized to aid marlins, swordfish, and other billfishes in their pursuit of prey because the tissues are kept from becoming cold when the fish swim through cold water.

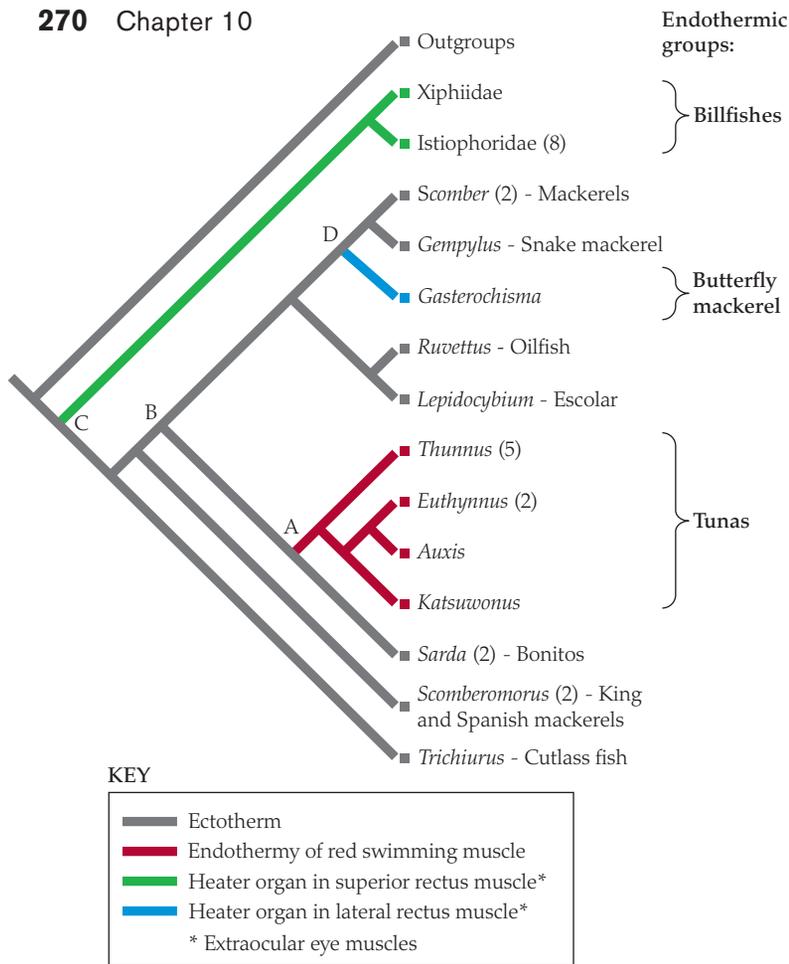
A family tree (phylogeny) has been developed for the warm-bodied teleost fish and their close relatives (Figure 10.47), to provide a basis for better understanding the evolution of the warm-bodied condition.<sup>45</sup> In a manner similar to the family trees discussed in Chapter 3, this family tree is based entirely on information other than physiology and thus is independent of physiological knowledge of the fish.

One of the physiological features mapped onto the tree is endothermy in the red swimming muscles. Specifically, all the little boxes that represent fish with such endothermy are colored red. When endothermy is mapped in this way onto the independently derived family tree, a coherent pattern emerges, which strongly suggests that red-muscle endothermy appeared in the evolutionary history of these fish at a single time, at the spot marked A. Not just tunas, but also bonitos, exhibit some degree of morphological shift of their red swimming muscles toward the unusual location near the spinal column, and they are the only teleost fish that do. Thus spot B is apparently where the shift in red-muscle position started. Evidently, the new red-muscle position evolved first, and then the tunas (but not the bonitos) capitalized on it to evolve red-muscle endothermy. The concept that the new red-muscle position set the stage for the evolution of endothermy in the red swimming muscles is bolstered by information on the lamnid sharks; they and the tunas

<sup>43</sup>There is some evidence for active thermoregulation in these fish. For example, they decrease heat retention when they are highly active in warm water, thus preventing their activity from driving their muscle temperature too high.

<sup>44</sup>In tunas and lamnids, the rate of heat production in each warmed organ or tissue is believed to be simply the ordinary rate, based on available evidence.

<sup>45</sup>The sharks, which are not teleosts, were not included in the study to produce the family tree.



**FIGURE 10.47** A family tree (phylogeny) of teleost fishes belonging to the suborder Scrombroidei. Physiological features (see key) are mapped onto the family tree. The tree, however, was derived entirely independently of physiological information, being based on an analysis of DNA nucleotide sequences in the gene for mitochondrial cytochrome *b* in the species included. The colors in the little boxes on the right side of the tree indicate the actual, known physiological nature of the various modern-day fish specified. The coloring of the lines of the family tree represents an interpretation of past history. Endothermy occurs only in the groups so identified at the right side of the diagram. Numbers are numbers of species studied if greater than one. “Outgroups” are other species of fish used to establish a base of comparison. Refer to the text for an explanation of the letters at branch points of the tree. (After Block and Finnerty 1994.)

## Endothermy and Homeothermy in Insects

A solitary insect at rest metabolizes at a sufficiently low rate that no part of its body is warmed by its metabolic heat production. Insects in flight, however, often exhibit very high metabolic rates; species that are strong fliers in fact release more heat per gram than active mammals or birds. This high heat production is localized in the flight muscles in the thorax. Given that insects do not have the profound problems of retaining heat that characterize water-breathers, it is quite possible for the thorax to be warmed by the high metabolism of the flight muscles during flight, and thus, as we saw at the beginning of this chapter, the thorax may be endothermic.

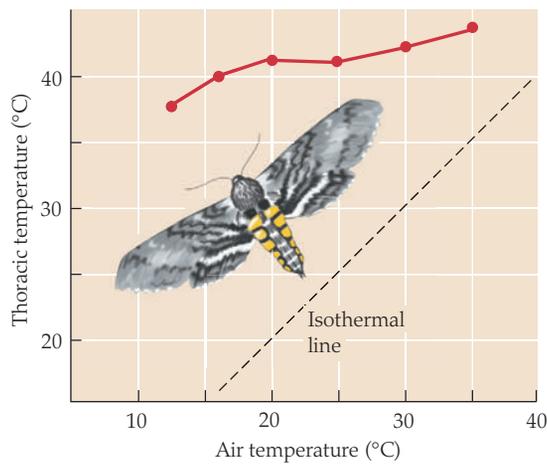
Some insects that display thoracic endothermy during flight do not thermoregulate; examples are provided by certain species of small geometrid moths, which maintain a thoracic temperature that is about 6°C above air temperature regardless of what the air temperature is. Other sorts of insects physiologically thermoregulate during flight and thus exhibit *thoracic homeothermy*. The thermal relations of endothermic insects are particularly complex because they exhibit both *temporal* and *spatial* heterothermy. The insects exhibit endothermy only when they are active, not when they are resting. Moreover, even when they exhibit endothermy, they usually do so just in their thorax, not their abdomen.

Historically, sphinx moths were the first group of insects discovered to display thoracic physiological thermoregulation during flight, and to this day they are model examples of the phenomenon. Sphinx moths are stronger fliers and often (for insects) are particularly large; some species weigh as much as several grams and thus are similar in weight to some of the smallest mammals and birds. Flying sphinx moths closely regulate their thoracic temperatures. Those in [Figure 10.48](#), for example, maintained thoracic temperatures within a narrow range, 38–43°C, over a wide range of air temperatures. Thermoregulation is not limited just to insects of such large body size. Worker bumblebees (*Bombus vagans*), averaging 0.12 g in body weight, for instance, maintain thoracic temperatures near 32–33°C whether the air temperature is 9°C or 24°C when they are foraging. Honeybees, averaging 0.09 g in body weight, exhibit impressive thoracic thermoregulation over a somewhat narrower range of air temperature, and also illustrate the usual insect pattern that—at moderate to cool air temperatures—the abdominal temperature tends approximately to match air temperature ([Figure 10.49](#)). The list of insects known today to exhibit thoracic homeothermy during flight also includes many other lepidopterans and bees, some dragonflies, and some beetles.

exhibit a remarkable convergence in the mechanics of how they swim, and part of that convergence is that the lamnids as well as the tunas have red swimming muscles positioned in an unusual position near the spinal column. Another discovery from the family tree in [Figure 10.47](#) is that the three teleost groups with some sort of endothermy—the billfishes, tunas, and butterfly mackerel—do not form a coherent group; they are separated by lines of the family tree in which there is no endothermy. The family tree thus indicates that endothermy evolved separately three times in teleost fish (spots A, C, and D)—plus at least one additional time in the lamnid sharks.

### SUMMARY Warm-Bodied Fish

- Tunas, lamnid sharks, and billfishes are distinguished from other fish by exhibiting endothermy in certain body regions. The tissues that are endothermic in tunas and lamnids are (1) the red swimming muscles and (2) sometimes the stomach, other viscera, brain, and retinas. In billfishes, only the brain and retinas are endothermic.
- A countercurrent vascular array that short-circuits outflow of heat from a tissue is required for the tissue to be endothermic in water-breathing animals such as these fish.
- Ordinary metabolic heat production is the source of heat for endothermy in all cases except the billfishes, which have specialized “heater” tissues that are derived from extraocular eye muscles.



**FIGURE 10.48** The average thoracic temperature of freely flying sphinx moths (*Manduca sexta*) as a function of air temperature. The isothermal line shows how thoracic temperature would vary with air temperature if there were no endothermy or thermoregulation and the temperature of the thorax simply equaled air temperature. (After Heinrich 1971.)

Although endothermy and physiological thermoregulation occur principally during flight in insects, a few types of insects display the phenomena during solitary terrestrial activities. In nearly all such cases, the primary source of heat is the flight muscles, which instead of being used to fly, are activated to “shiver” (as discussed shortly). Dung beetles—which transport energy-rich elephant dung or other dung to preferred locations by forming the dung into balls—sometimes become markedly endothermic while working in dung piles and rolling their dung balls. Some crickets and katydids thermoregulate while they sing.

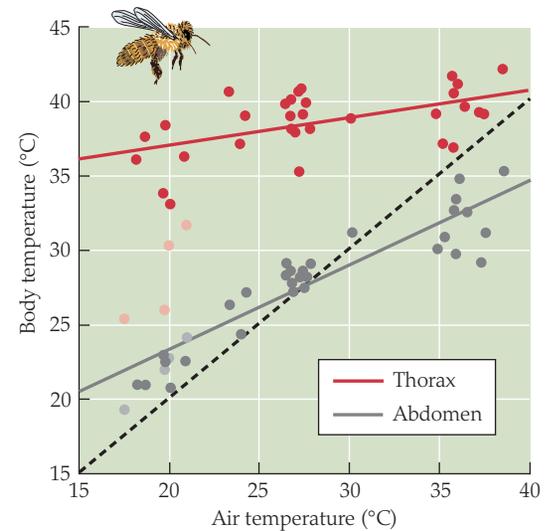
### The insects that thermoregulate during flight require certain flight-muscle temperatures to fly

The flight muscles of an insect must be able to generate mechanical power at a certain minimal rate (which is species-specific) for the insect to be able to fly. Within a broad range of temperatures, the power output that flight muscles can attain increases as their temperature increases. Thus the temperature of an insect’s flight muscles is potentially an important determinant of whether the insect can fly.

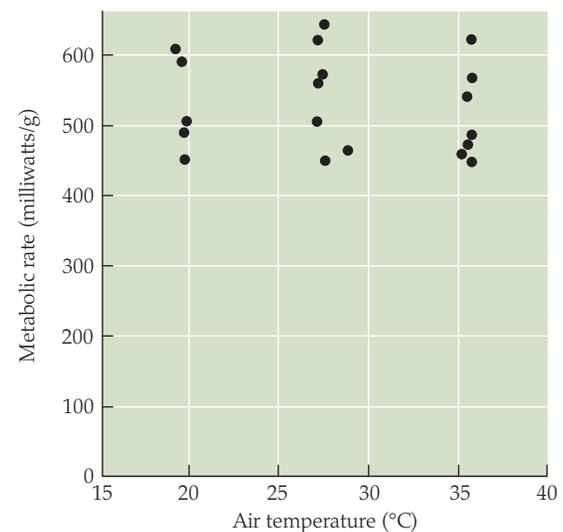
Tiny insects such as fruit flies, mosquitoes, and midges have such high surface-to-volume ratios that the activity of their flight muscles cannot warm the thorax significantly. Correlated with their inability to be endothermic, the tiny insects commonly can fly with very broad ranges of thoracic temperatures, including, in some species, thoracic temperatures as low as 0–5°C. An important property of the flight physiology of these tiny, poikilothermic fliers is that they apparently require only a modest fraction of their maximal power output to stay aloft; thus they can fly at relatively low thoracic temperatures, at which their power output is substantially submaximal.

In sharp contrast, many medium-sized and large insects, including the species known to thermoregulate, require a near-maximal power output from their flight muscles to take off and remain airborne. They therefore require that their flight muscles be at high temperatures to fly. The sphinx moth *Manduca sexta*, for example,

(a) Temperatures of thorax and abdomen



(b) Metabolic rates



**FIGURE 10.49** Temperature and metabolism in steadily flying honeybees (*Apis mellifera*). Honeybees vary considerably in how well they fly under controlled circumstances, and the data presented here are specifically for individuals that flew without prodding. (a) Temperatures in the thorax and abdomen at the end of flight; lighter-colored symbols are for four bees that showed particularly great reductions in wing-beat frequency while flying. (b) Metabolic rates during flight. (After Woods et al. 2005.)

cannot fly unless its thorax is at least as warm as 35–38°C, and worker bumblebees (*Bombus vagans*) require about 29°C.

The need for high flight-muscle temperatures for flight raises the question of how resting insects are able to get warm enough to take off. Because insects typically cool to environmental temperature when they are fully at rest, an insect that requires a high flight-muscle temperature to fly will often be too cold to take off after it has been resting for a while. Diurnal species may be able to warm their flight muscles to flight temperature by basking in the sun. Most species, however, have an endogenous ability to warm their flight muscles to flight temperature, a phenomenon known as **physiological preflight warm-up**.

Physiological preflight warm-up is accomplished by contraction of the flight muscles in a nonflying mode, a process often called **shivering** (not homologous to vertebrate shivering). Several forms of shivering are known. In many types of insects, including moths and butterflies, what happens during shivering is that the muscles responsible for the upstroke and downstroke of the wings contract simultaneously (rather than alternately as they do in flight), thus working against each other. The wings merely vibrate during shivering, rather than flapping, but heat is evolved by the muscular contraction, warming the flight muscles. When a sphinx moth warms from a low temperature, its flight muscles shiver in this manner at an ever-higher intensity as its thoracic temperature increases to the flight level. Then suddenly the pattern of muscular contraction changes, the wings are driven through the flapping motions of flight, and the moth takes to the air.

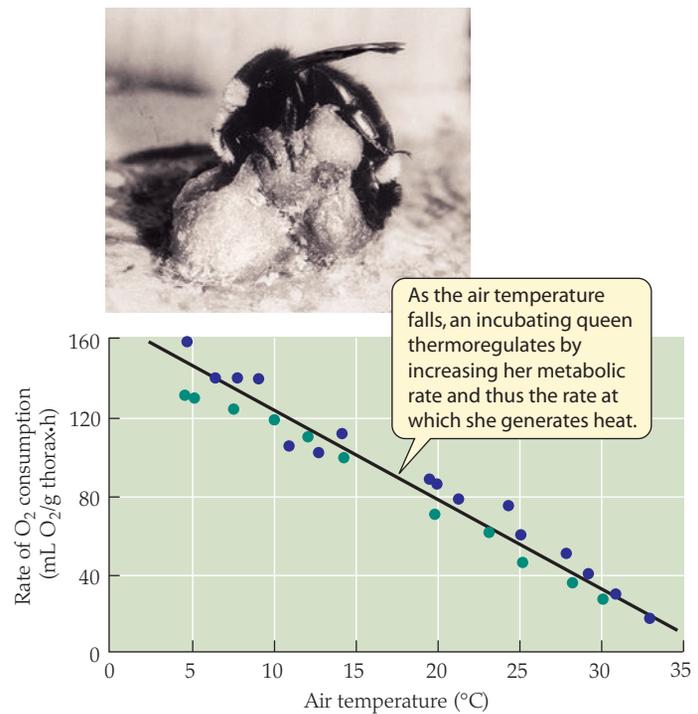
### Solitary insects employ diverse mechanisms of thermoregulation

Innovative investigators continue to progress in understanding the mechanisms that insects employ to thermoregulate, despite the obstacles of working on such small animals.

As the ambient temperature drops, one mechanism of maintaining a constant thoracic temperature is for an insect to increase its rate of heat production, much as mammals and birds do below thermoneutrality. Many insects do this when they are *not flying*. Heat is generated in these circumstances by shivering of the flight muscles, and because the muscles can engage in various intensities of shivering, they can modulate their rate of heat production to serve thermoregulatory needs. Honeybees and bumblebees working in the hive, for example, often maintain high and stable body temperatures for long periods by increasing and decreasing their rates of shivering heat production as the air temperature falls and rises. An intriguing example is also provided by the brood incubation of queen bumblebees (Figure 10.50). A queen, which overwinters alone and thus is solitary when she rears her first brood in the spring, incubates her brood by keeping her abdomen at an elevated temperature and pressing it against the brood. Heat is brought to her abdomen from her thorax, where it is produced by her flight muscles. As the ambient air temperature falls, the queen thermoregulates by increasing her rate of heat production (see Figure 10.50).

Modulation of shivering can also be used to thermoregulate during *intermittent flight*. Bumblebees are known to do this, for instance. As a bumblebee, such as that pictured at the start of this chapter, flies from flower to flower during foraging, it can shiver or not shiver while it is clinging to each flower. More shivering of this sort occurs as the air temperature falls, and thus the bumblebee's overall, time-averaged metabolic rate increases as air temperature decreases.

When insects *fly continuously*, their flight muscles are employed in flight movements all the time and cannot shiver. Investigators hypothesized years ago that under these circumstances, the rate of heat production by the flight muscles would be determined by the requirements of flight and not modulated to serve thermoregulation. Early, seminal experiments on sphinx moths supported the truth of this hypothesis, because when the moths flew at a certain speed, their metabolic rates were essentially constant whether the air temperature was 15°C or 30°C. If insects in continuous flight do not modulate their rates of heat production as a means of thermoregulating, how do they thermoregulate?



**FIGURE 10.50** The rate of O<sub>2</sub> consumption by queen bumblebees as a function of air temperature when they are incubating their broods. In the species studied, *Bombus vosnesenskii*, a queen incubates her brood by pressing her abdomen against it as seen in the photograph. In the plot, the two colors of symbols refer to two different individuals. (After Heinrich 1974; photograph courtesy of Bernd Heinrich.)

Studies of sphinx moths, bumblebees, and some other insects reveal that their primary mechanism of thermoregulation during continuous flight is much akin to that used by mammals and birds in the thermoneutral zone; namely, they vary their insulation—in this case their *thoracic insulation*. A flying insect keeps its thorax at a steady temperature by modulating how readily heat can exit the thorax. This modulation is accomplished in some moths, dragonflies, and bumblebees by control of the rate of blood flow between the thorax and abdomen. In a continuously flying sphinx moth, for example, when the air temperature is low, the heart beats weakly and blood circulates slowly between the thorax and abdomen; thus heat produced by the flight muscles tends to remain in the thorax, which retains the heat effectively because it is densely covered with furlike scales. As the air temperature is raised, the heart beats more vigorously and circulates blood to the abdomen more rapidly; in this way, heat is transported at an increased rate out of the thorax into the abdomen, where it can be relatively easily lost to the environment because the abdomen has a large and thinly “furred” surface. Honeybees sometimes carry out an analogous process in which they modulate blood transport of thoracic heat to the head; at elevated air temperatures, heat is transported at an increased rate into the head, where it is lost in part by evaporation of fluid regurgitated out of the mouth.

Over the last 15 years, the old paradigm of thermoregulation during continuous flight—which held that all flying insects thermoregulate by modulating thoracic heat loss but not heat production—has been challenged by studies on certain species. Some investigators, for example, have observed at times that continuously flying honeybees increase their rates of metabolic heat production as the air temperature falls, at least in part by raising their wing-beat

frequency. Other investigators, however, find that the metabolic rate during honeybee flight is the same at all air temperatures (see Figure 10.49b): the result expected if the demands of flight rather than those of thermoregulation exclusively govern the activity of the flight muscles.

### Colonies of social bees and wasps often display sophisticated thermoregulation

Physiological regulation of colony temperature is widespread within colonies of social bees and wasps. Honeybee hives (*Apis mellifera*) provide the best-studied example. Thermoregulation by honeybee hives is so dramatic that it was recognized for almost two centuries before thermoregulation by solitary insects was first demonstrated.

A honeybee hive that is rearing a brood maintains the temperature of its brood combs within a narrow range, about 32–36°C, even if the air temperature outside the hive falls to –30°C or rises to +50°C. When the air outside the hive is cold, worker bees cluster together within the hive and shiver. When the air outside becomes warm enough that the hive is threatened with metabolic overheating, workers disperse within the hive and fan with their wings in a cooperative pattern that moves fresh air from outside the hive across the brood combs. At very high outside air temperatures, workers also collect water and spread it within the nest, where it evaporates into the airstream produced by fanning. Honeybees provide an outstanding example of coevolution between thermal requirements and thermoregulation. Their broods of young must have temperatures of about 32–36°C for proper development. Thus the sophisticated thermoregulation of the hive by the workers is essential for a hive’s reproductive success.

#### SUMMARY Endothermy and Homeothermy in Insects

- Many solitary insects, especially those of medium to large size, display thoracic endothermy or homeothermy during flight or certain other sorts of activity. Warming of the flight muscles increases their power output. Often in these insects, a certain minimum flight-muscle temperature is required for flight.
- When insects are not flying, activation of the flight muscles in a nonflight mode—termed *shivering*—is the mechanism they employ to warm the thorax. Shivering is used for preflight warm-up. Nonflying insects also sometimes thermoregulate by modulation of shivering, as observed in bees working in their hives.
- When insects are flying, the best-known mechanism of thermoregulation is modulation of thoracic insulation, brought about by raising and lowering circulatory transport of heat out of the thorax.
- Colonies of social bees and wasps sometimes employ group efforts to maintain exquisitely stable hive temperatures.

### Coda

Endothermy can provide organisms with distinct advantages. Accordingly, despite the fact that endothermy usually has a high energy cost, it has evolved independently in animals multiple times. It even occurs—as still another independently evolved example—in plants (Box 10.3).

### BOX 10.3 WARM FLOWERS

In the early spring when snow is still on the ground, the flower structures of the arum lily called eastern skunk cabbage (*Symplocarpus foetidus*) melt their way to the snow surface by being as much as 30°C warmer than the ambient temperature. In this way, this species dramatically announces that plants have evolved endothermy! The eastern skunk cabbage in fact displays thermoregulatory properties, in that its flower structures increase their rate of metabolic thermogenesis—responsible for endothermy—as the ambient temperature becomes colder. The function of endothermy in this case is believed to be to enhance the volatilization of odor compounds that attract pollinators. **Box Extension 10.3** discusses this fascinating topic further.



### Study Questions

1. As discussed in Chapter 1 (see page 13), Claude Bernard, a nineteenth-century French physiologist often considered the father of modern animal physiology, is still remembered today for his famous dictum: “Constancy of the internal environment is the condition for free life.” Does the study of thermal relations lend support to his dictum? Explain.
2. There is currently a worldwide movement to create protected marine parks. If the parks have an Achilles heel, it is that they have fixed geographical positions, just in the way that Yellowstone National Park is at a fixed geographical location. Suppose that a certain endangered species of fish exists only in a marine park. If the ocean temperature rises in the park because of global warming, explain what physiological problems the species of fish might confront. How might the species face a brighter future if parks could have moveable boundaries rather than fixed ones?
3. Referring to Figure 10.11, suppose you have some lizards that are at 16°C and have been living at that temperature for 5 weeks. What is their resting metabolic rate? If the lizards are suddenly shifted to a room at 33°C, trace on the graph how their metabolic rate will change from the moment they are placed in the new room until 5 weeks have passed. According to the graph, will they exhibit compensation?
4. Discuss ways that the cryobiology (“freezing biology”) of insects could be manipulated to control insect pests. One factor to consider is that certain bacteria and fungi act as highly effective ice nucleators.
5. In the animal kingdom today, poikilotherms outnumber homeotherms by a great margin. Why is poikilothermy a successful way of life even though poikilotherms sometimes must compete successfully with homeotherms to survive?

6. Suppose you travel to a tropical place such as the Bahamas and watch the coastal poikilotherms, such as fish, crabs, and starfish, swim and crawl about in the warm waters. Suppose then that you travel to northern Maine and watch the related species of poikilotherms in the cold waters there. In the abstract, it would not be unreasonable to expect to see the animals in Maine moving about in slow motion compared with those in the Bahamas. In fact, however, rates of locomotion are likely to look to your eye to be more similar than different in the two places. Design experiments to assess whether the Maine animals are especially able to be active in cold waters. If you find that they are, how might their high ability for activity in cold waters be explained? For each hypothesis you present, design an experiment to test the hypothesis.
7. During winter, when people are in a well-insulated house, they usually feel comfortable if the air temperature is near 22°C (72°F). If you have ever spent a night in a poorly insulated cabin in winter, however, you will recognize that paradoxically, when people are in poorly insulated buildings, they often feel chilly even when the air inside is heated to 22°C or higher. One important reason for the difference in how warm people feel in the two sorts of buildings is that even if a well-insulated and poorly insulated building are identical in the air temperature inside, they differ in thermal-radiation heat transfer. Specifically, a person standing in the two types of buildings experiences different heat exchange by thermal radiation in the two. Explain how thermal-radiation heat transfer accounts for the sense of chill in the poorly insulated building. (Hint: Think of the outer walls of the two types of buildings, and think specifically of the temperatures of the interior surfaces of those walls.)
8. What is homeoviscous adaptation? Although we discussed it in our study of poikilotherms, the phenomenon was actually first discovered about a century ago in studies of pigs in Sweden. Some pigs were dressed in blankets during winter while others were allowed to roam about stark naked. When their subcutaneous fat was analyzed, the two sets of pigs turned out to have laid down lipids of differing chemical composition. How could different lipids give the two groups similar lipid fluidities?<sup>46</sup>
9. Suppose you are trying to choose between two winter jackets. Suppose also that you have a heat-producing mannequin available for your use and you are able to adjust the mannequin's rate of heat production. According to Equation 10.10, insulation is equal to  $(T_B - T_A)/M$  (this is in fact a general equation for insulation). How would you make a quantitative comparison of the insulation provided by the two jackets?
10. In the rete mirabile serving the red swimming muscles of tunas, some key enzymes of catabolism show gradients of concentration: They are more concentrated at the cold end of the rete, and less concentrated at the warm end. These variations parallel variations that are often seen in the thermal acclimation of poikilotherms, when enzyme concentrations rise during acclimation to cold and fall during acclimation to heat. What do you think could be some of the reasons for these spatial and temporal variations in enzyme concentration? Why not have the highest observed enzyme concentrations everywhere at all times?
11. Humphries, Thomas, and Speakman presented a bioenergetic model to predict how global warming might force insectivorous bats to alter the latitudes at which they seek hibernation sites. The investigators stress that not only is the model fairly simple, but also it allows the *existing* distribution of hibernation sites to be predicted reasonably well. Study their model, and assess its pros and cons. Why is the little brown bat a particularly suitable species for the application of this method? See M. M. Humphries, D. W.

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Go to [sites.sinauer.com/animalphys3e](http://sites.sinauer.com/animalphys3e) for box extensions, quizzes, flashcards, and other resources.

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<sup>46</sup>To avoid introducing any confusion, it may be important to mention that the subcutaneous lipid deposits of mammals (which are known as *depot fats*) consist of ordinary triacylglycerols, not phospholipids like membrane lipids, but the basic concepts of homeoviscous adaptation remain the same.

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*See also **Additional References**, Figure and Table Citations, and References in Chapter 11.*