CHAPTER

15 Thermal Physiology

FIGURE 15.1 **Opah (*Lampris guttatus*)**

Photo source: Southwest Fisheries Science Center, NOAA Fisheries Service.

Learning Objectives

After reading this chapter, you should be able to:

1. Explain how the environment impinges on heat exchange in animals.
2. Utilize the precise terminology required to distinguish between thermal strategies.
3. Explain the factors that affect thermal tolerance of animals.
4. Explain how temperature affects macromolecular structure and function.
5. Discuss the ways animals alter these molecular structures and functions as part of compensatory strategies.
6. Explain the factors that influence metabolic heat production in animals.
7. Discuss how the central thermostat works, and how it alters anatomy and physiology to influence heat exchange.

A high metabolic rate leads to heat production and an increase in body temperature permits a higher metabolic rate. The connection between metabolism and heat production is rooted in chemistry and physics, but that relationship has profound consequences for how thermal physiology evolved.

Before about 200 million years ago (mya), most of the animals on the planet had a body temperature (T_B) determined by the environment, primarily ambient temperature (T_A), a pattern known as *ectothermy*. However, there were a few species, such as large dinosaurs, that were able to retain metabolic heat to elevate T_B above T_A, which is known as *endothermy*. As first discussed in Chapter 1, large animals benefit from a low surface area-to-volume ratio because it reduces heat loss. Though not as large as dinosaurs, several modern large animals, such as leatherback turtles and whale sharks, have some degree of endothermy, unlike their smaller relatives living in the same environment.

The first truly endothermic lineages were mammals and later birds. Though they arose from different reptile ancestors (see Chapter 2), their independent routes to endothermy included convergent evolution of insulation: fur in mammals, feathers in birds. In the mammalian lineage, the ability to retain metabolic heat in small-bodied animals allowed them a nocturnal life, where they avoided predation by larger reptiles. The birds arose from one of several feathered reptilian lineages, but in the bird lineage the feathers were asymmetrical. While serving perfectly well for insulation, these asymmetrical feathers also altered airflows in a way that predisposed them for use in flight. In each of these endothermic lineages, subsequent evolutionary events led to spatial and temporal variations in thermal physiology. With both birds and mammals, select species permit anatomical regions to get cold, and others allow their whole body to cool, decreasing metabolic rate dramatically.

A completely different form of endothermy is seen in select fish, specifically those large enough to attain favorable surface area:volume ratios. This is all the more difficult for fish for two reasons: they live in water, which is more effective at conducting heat from the body, and their circulatory system is configured in a way that brings warm blood from the core to the gills, where it reaches thermal equilibrium with the cooler water. Still, some fish lineages have evolved an ability to maintain parts of the body warm through the use of countercurrent heat exchangers called **retia** (singular, *rete*). Retia are found in the locomotor muscles of large tuna and lamnid sharks and in the GI tract of large billfish. Some fish, such as blue marlin (*Makaira nigricans*) and opah (Figure 15.1), use these circulatory adaptations, in combination with modified muscles called heater organs, to warm regions of the central nervous system.

In this chapter, we explore the diversity in thermal strategies employed by animals. Throughout, we emphasize the relationship between the underlying physical laws of physics and chemistry and the biological adaptations that govern the relationship between an animal and its thermal environment. ■

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LOOKING BACK

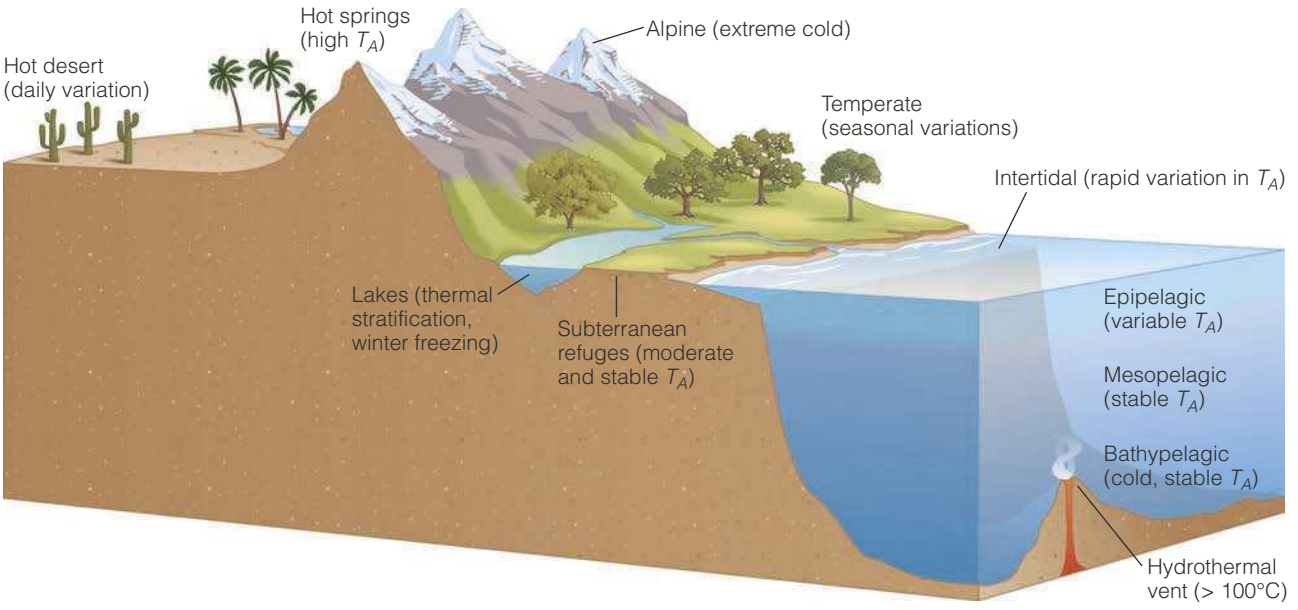
You may find it helpful to review Chapter 3, where we described the nature of energy, the fundamentals of energy metabolism, and the thermal sensitivity of macromolecular structures. In Chapter 2 we discussed the evolutionary history of the reptiles, which helps you understand the independent origins of thermal physiology of birds and mammals. Chapter 9 described the organization of the circulatory system, and its role in transport of heat between tissues and mediating the exchange of heat between the animal and its environment. In Chapter 12 we discussed the role of muscle in production of metabolic heat, and the nature of neural control of muscle activity. Finally, in Chapter 14 we discussed the relationship between the digestive system and metabolic rate.

OVERVIEW

Thermal energy influences chemical interactions in ways that affect macromolecular structure and biochemical reactions. Consequently, temperature has pervasive effects on all physiological processes. As a result of these temperature effects, every animal displays a thermal strategy: a combination of behavioral, biochemical, and physiological responses that ensures body temperature (T_B) is within an acceptable limit. The most important environmental influence on the thermal strategy (though not the only one) is ambient temperature (T_A). Animals must survive the highest and lowest T_A in their niche (thermal extremes), as well as the changes in T_A (thermal change).

Animals inhabit most thermal niches on the planet (Figure 15.2). The hottest environments exploited by animals are the regions near thermal vents, such as the hydrothermal vents of the deep sea, volcanoes, and geysers. The coldest

FIGURE 15.2 Thermal niches



places inhabited by animals are alpine and polar regions. The animals that survive in the extremes of heat and cold are impressive, but the ability to tolerate changing temperature is every bit as challenging physiologically. Environmental temperatures are most variable in terrestrial ecosystems; air temperatures change more rapidly and reach greater extremes than do water temperatures.

Many ecosystems exhibit spatial variation in temperature. Underground refuges are buffered from thermal extremes on the surface. The T_A in alpine regions varies as a result of altitudinal gradients arising over only a few kilometers. Large bodies of water, such as lakes and oceans, can vary in T_A with depth. Deep-ocean (bathypelagic) temperatures are often close to 4°C, whereas midwater (mesopelagic) and surface water (epipelagic) temperatures can be much warmer and more variable. Large temperate lakes may be nearly uniform in temperature, or have sharp demarcations (thermoclines) between top and bottom water, sometimes differing more than 10°C in less than a meter of depth.

Ecosystems can also change in temperature temporally. Terrestrial and aquatic ecosystems in the tropics tend to have a relatively constant T_A, but polar and temperate zones experience seasonal and daily cycles of cold and heat. Air temperatures can change more rapidly than water temperatures, sometimes more than 20°C in a single day. Intertidal animals may experience the heat of a summer day mere seconds before the cold ocean washes over them. Many animals incorporate behavior into their thermal strategy, but animals must also cope with the effects of temperature on biochemistry and physiology.

HEAT EXCHANGE AND THERMAL STRATEGIES

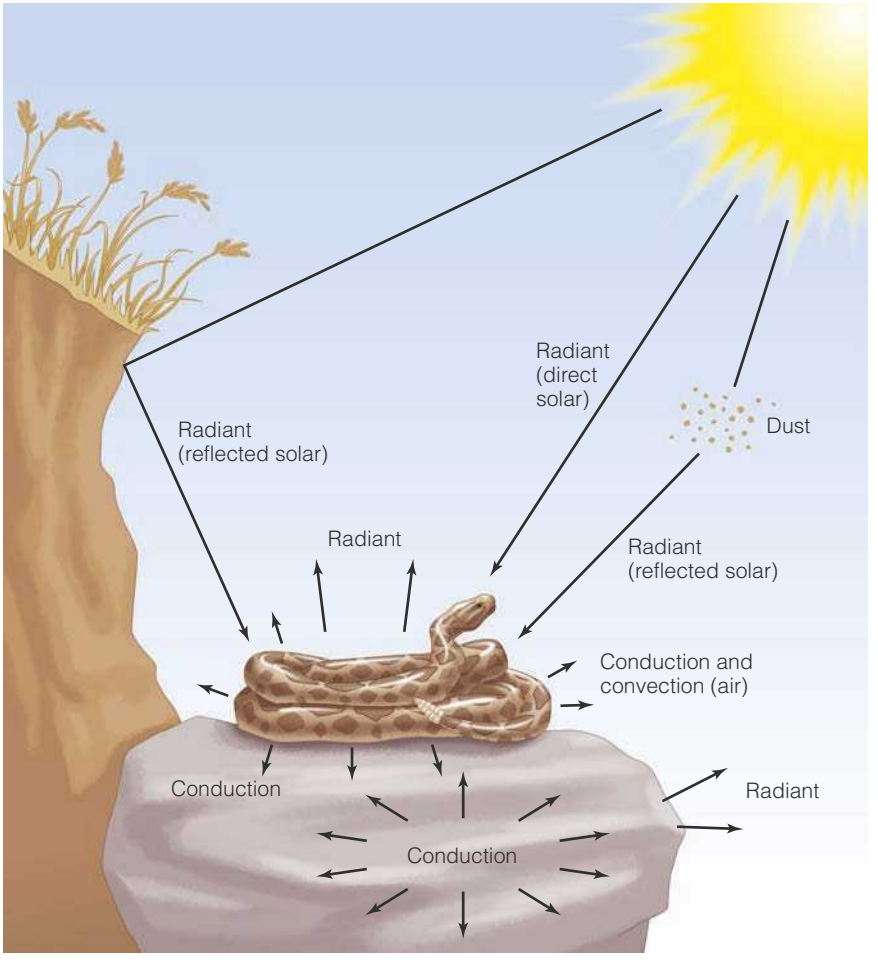
The most important physiological parameter in an animal's thermal physiology is body temperature (T_B). An animal's thermal strategy serves to control the transfer of energy between the animal and the environment. Some animals tolerate wide changes in T_B and the effects of these changes on many physiological processes. Others must use a combination of physiological and behavioral means to ensure that T_B remains nearly constant. As in other physiological systems, both strategies—conforming and regulation—have costs and benefits. The physiological mechanisms that impart a constant T_B use energy. When T_B is allowed to vary, important physiological processes such as development become sensitive to environmental changes. Although T_A has the most obvious impact on animal thermal biology, other routes of heat exchange are also important in many contexts.

Controlling Heat Fluxes

An animal's T_B is a reflection of the thermal energy held within the molecules of the body. Thermal energy can move from the animal to the environment, or from the environment to the animal, depending on temperature gradients. Metabolism—the sum of all biochemical reactions occurring within the body—is the main source of thermal energy in the heat balance equation of most animals. However, other important sources and sinks for thermal energy also affect an animal's thermal budget (Figure 15.3). The thermal

FIGURE 15.3 Sources and sinks for thermal energy

The body temperature of an animal is influenced by heat exchange with the environment. This snake is warmed by radiant energy from the sun, as well as thermal energy radiated from its surroundings. The animal exchanges thermal energy through objects and fluids in contact with its external surface (conduction). Movement of the air enhances the efficiency of thermal exchange by convection. The animal itself radiates thermal energy to the surrounding air.



balance equation takes into consideration all of the routes through which thermal energy, abbreviated as H, can enter or exit the body:

$$H_{total} = \Delta H_{metabolism} + \Delta H_{conduction} + \Delta H_{convection} + \Delta H_{radiation} + \Delta H_{evaporation}$$

If the equation above sums to zero ($H_{total} = 0$), there will be no net change in the thermal energy of the animal and T_B will remain constant. If the flow of thermal energy into the animal exceeds the heat loss, T_B will increase. Each of these routes of thermal energy exchange depends on the thermal properties of the environment as well as the physical properties and physiology of the animal.

- **Conduction** is the transfer of thermal energy from one region of an object or fluid to another. Animals can be cooled when thermal energy is conducted away from the body, or can be warmed as they absorb heat from conductive objects.
- **Convection** is the transfer of thermal energy between an object (the animal in this case) and an external fluid that is moving. For example, warm air feels cooler when it flows over your skin than when the air is still. Most often, convection causes a loss of thermal energy from animals.
- **Radiation** is a general term that refers to the emission of electromagnetic energy from an object. An animal can absorb radiant heat emitted from the surroundings, but can also emit radiant heat from its own surface, a major form of heat loss. The infrared radiation emitted from an object indicates its surface temperature.
- **Evaporation** of water molecules from the surface of an object absorbs thermal energy from the object. Thus, evaporative heat exchange is almost always a heat loss from the animal.

The relative importance and even the direction of heat transfer from each of these parameters differ among animals and conditions. The properties of the animal, including physical composition and color, have a profound influence on the relative importance of these exchanges.

Water has a higher thermal conductivity than air

Conduction is difficult to quantify because of the many factors that affect heat exchange. Let's begin our discussion by considering how conduction is involved in the transfer of thermal energy through a single material, such as a thin metal bar heated at one end. The rate of heat transfer from the warm end to the cool end (heat flux) is described by Fourier's law and the following equation:

$$Q = \frac{\lambda \ T}{L}$$

where heat flux (Q) depends upon the temperature gradient (ΔT), the distance over which the gradient extends (L), and the thermal conductivity (λ) measured in watts per meter per kelvin (W/m per K). Thermal conductivity is a specific

property of a material. Those objects we think of as *heat sinks* have high thermal conductivity. For example, an aluminum pot feels cold to the touch because it has a high thermal conductivity (210 W/m per K) and readily draws heat from your hand. Similarly, 5°C water feels cooler than 5°C air because water has a thermal conductivity that is 25-fold higher than air (0.58 versus 0.024 W/m per K). Because water has more molecules per unit volume, there is a greater likelihood of a molecular collision that results in a transfer of energy.

The Fourier equation describes how thermal energy moves in a very simple system: heat transfer in a single dimension (from the heat source to heat sink) in a single uniform material. These same parameters (λ, ΔT, and L) apply in thermal biology, but animals are much more complex systems. Consider the influence of **thermal conductance**. Heat is conducted from the internal tissues, through other tissues and fluids, and to the external surroundings, each with a characteristic thermal conductivity (Table 15.1). The body surface layers may possess insulation that reduces conductive heat transfer. Insulation, such as fur and feathers, also increases the distance between the hottest point near the skin and the coldest point in the bulk phase.

Calculations of heat flux are complicated by the geometry of the environment and the animal. Heat does not move from your body through a one-dimensional cylinder of air extending from your skin, but rather is conducted in multiple dimensions from the source. Animal geometry also plays a role. A long, thin animal produces as much heat as a short, round animal of the same mass, but the differences in surface area affect heat exchange. Because conductive heat losses occur across the external surfaces, an animal can alter conductive heat exchange by engaging in activities that alter its effective surface area. For example, a penguin reduces heat loss from the foot by rolling back on its heels, using its tail feathers for balance. Because its tail feathers are less conductive than its feet, less heat is lost. Figure 15.3 shows a snake simultaneously exchanging heat with multiple surfaces. It loses heat via conduction across its upper surface while also exchanging heat through its lower surface in contact with the rock.

Table 15.1 Thermal conductivity of materials

Material	Thermal Conductivity (W/m per K)
Air	0.02
Snow	0.10
Water	0.59
Rock	1–3
Ice	2.1
Muscle	0.5
Fat	0.2

Convective heat exchange depends on fluid movements

Imagine yourself immersed in a pool of water that is 10°C colder than your body. Almost immediately, your body begins to lose thermal energy as it warms the water in the boundary layer by almost 10°C. Once the boundary layer is warmed, thermal energy is slowly conducted to the bulk phase of the water. When the heat exchanges reach steady state, the body loses thermal energy at the rate required to rewarm this boundary layer that slowly cools as it dissipates its thermal energy outward to the bulk phase. Much less energy is required to rewarm the boundary layer under these steady-state conditions than was required to heat the boundary layer in the first place. Now consider how the gradients change when fluid is flowing over the body. The body rapidly loses thermal energy warming a boundary layer that is immediately replaced by another, colder boundary layer. Heat lost to a moving fluid, either air or water, is *convective heat loss*. The rate of convective heat loss depends on the thermal gradient between the surface and the fluid, the rate of flow of the fluid over the surface, and its conductivity.

Radiant energy warms some animals

In the natural world, the most important source of radiant heat is the sun. Photons from the sun excite the molecules in the atmosphere, the soil, and the water, warming them by radiant heat. Thus, when animals are warmed by conduction from air, water, or soil, the ultimate source of the heat is radiant energy. But animals can also be warmed directly by solar radiation, which many species accentuate by the behavior known as basking. White body coloration reflects photons in the visible range, and dark coloration absorbs the photons within this range of wavelengths. Animals that bask to warm themselves often possess high levels of black or brown pigments to help absorb thermal energy. As a result of diversity in color, animals in the same area can have markedly different temperatures (Figure 15.4).

In terrestrial systems, the ground warms during the day and then becomes an important source of thermal energy in the form of conduction and radiant heat when the sun sets. Animals also lose thermal energy when they emit radiant heat. Thus, radiant heat may be a net gain or net loss from animals. The relationship that describes radiation from a warm animal is described by the Stefan-Boltzmann equation:

$$P = Ae\delta(T_B^{\,4}\, T_A^{\,4})$$

where P is the radiating power, A is its surface area, e is the ability of the object to emit radiation, δ is the Stefan constant, and T the temperature of the body (T_B) or surroundings (T_A)

FIGURE 15.4 Heterogeneity of T_B in the intertidal zone

Infrared photography can be used to compare the body temperature (T_B) of animals. In this image, the mussels are warmer than the starfish because they are better at absorbing radiant energy. The starfish, with its greater surface area, may also be affected more by evaporative cooling.

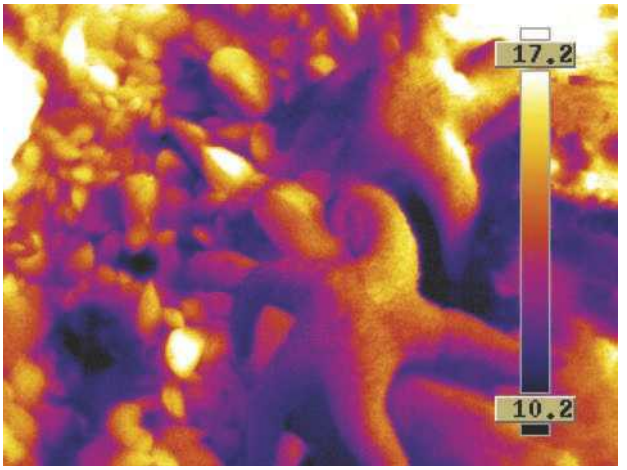


Photo source: Photo by Brian Helmuth, Northeastern University.

in kelvins. Animals can influence their radiant heat loss through changing the nature of the surface (e) and the surface area (A).

Evaporation induces heat losses

Evaporative cooling arises when fluids draw thermal energy from the body surface as the water molecules make the transition from liquid to vapor. The magnitude of the heat loss depends on the volume of water and its heat of vaporization. It requires more energy to evaporate water from salty sweat than from pure water because the solutes increase the heat of vaporization of water. The efficiency of evaporative cooling also depends on the partial pressure of water vapor in the air. If the air has high humidity, then the water is less likely to evaporate.

Sweating is only one of the ways that animals employ evaporative cooling. When a hippopotamus rolls in the mud of a wet riverbank, the cool mud draws heat from the body (conduction). This is an effective cooling strategy even if the mud is warm: thermal energy is absorbed from the body as the mud dries. Other animals cover their body surfaces with water, such as an elephant that sprays water onto its back or birds that splash in a pool of water. Wet feathers also have a diminished insulatory capacity, allowing more metabolic heat to be lost. Birds that live in hot environments may soak the belly before returning to the nest, allowing the eggs to benefit from evaporative cooling. Kangaroos, which do not produce sweat, lick well-vascularized skin surfaces, which then cool as the saliva evaporates.

Not all evaporative cooling is positive. As discussed in Chapter 13, desert animals must balance the challenges of water balance with the benefits that would accrue from evaporative cooling. Reliance on sweating would enhance dehydration. When semiaquatic animals leave the water, they are typically left with wet body surfaces, causing body temperature to decrease due to evaporative cooling.

Ratio of surface area to volume affects heat flux

The ratio of surface area to volume (see Figure 11.5) can influence all aspects of the heat exchange equation: conduction, convection, radiation, and evaporation. Variation in the ratio is important in several contexts. A given animal may alter its exposed surface area to change heat flux. Dogs stretch out when hot to maximize conductive heat loss to the ground, but roll up when cold to minimize conductive heat loss to the air. Ratios of surface area to volume also come into play when comparing animals of different body dimensions or body mass.

The significance of body size, or more precisely, the ratio of surface area to mass, is apparent in many comparisons. An arctic wolf is about one-tenth the mass of a grizzly bear, but it has twice the ratio of surface area to volume. Although they live in similar niches, the arctic wolf incurs greater thermoregulatory costs because of its size. Similarly, a growing animal increases its body mass faster than its surface area. In general, larger animals lose heat more slowly and retain heat better than do small animals. The effects of body size and shape also manifest themselves in animal evolution. *Bergmann's rule* states that animals living in cold environments tend to be larger than animals in warmer environments. *Allen's rule* states that animals in colder climates tend to have shorter extremities than animals in warmer climates. Thus, mammals or birds living in polar regions or high altitudes tend to be larger and shorter legged than individuals of the same species from more temperate regions. These rules of ecogeography apply to most of the mammals and birds studied to date, but have little relevance to animals that allow T_B to change.

An animal regulates heat exchange by altering the posture of the body to minimize or maximize the exposed surface area. Pythons will roll into a ball to conserve metabolic heat during digestion. When the python, approximately cylindrical in shape, rolls into a ball, its externally exposed surface area decreases by about 85 percent, greatly reducing heat loss.

Animals can also reduce effective surface area by huddling with other animals. Naked mole rats (Figure 15.5) live in burrows at relatively constant temperatures and have a very limited ability to use metabolism to control their body temperature. If housed in groups, they huddle when

FIGURE 15.5 Naked mole rats

Photo source: Science Source.

temperatures drop below about 22°C. This allows them to maintain a relatively constant T_B near 22°C. However, a solitary naked mole rat is unable to defend its T_B at low T_A. When prevented from huddling, its T_B closely reflects T_A, decreasing to as low as 12°C. From the perspective of the individual animal, huddling reduces heat by increasing T_A, replacing cold air with a warm neighbor. From the perspective of the colony, huddling works as a thermoregulatory strategy by reducing ratios of surface area to volume.

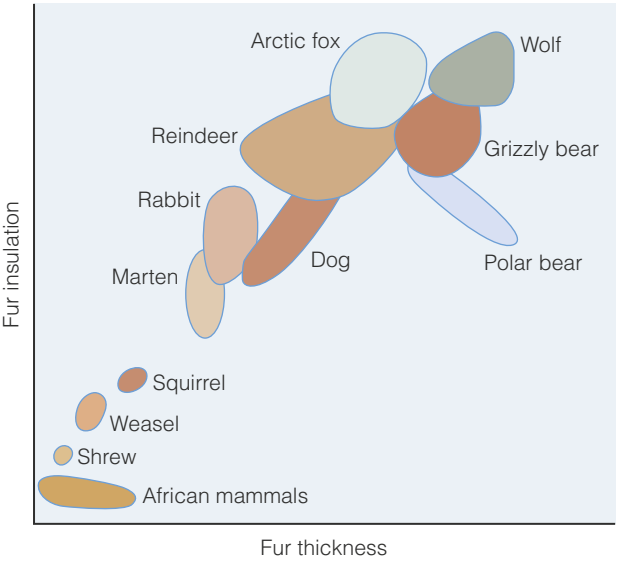
Insulation reduces thermal exchange

Internal and external insulation also reduce heat losses by increasing the distance over which a thermal gradient extends (the L in the Fourier equation). Marine mammals have a thick layer of adipose tissue under the skin in the form of blubber. This lipid layer disrupts the flow of thermal energy from the core to the external surface of the animal. More commonly, animals use external insulation to reduce heat loss. Fur and feathers restrict the movement of molecules between the surface of the animal and the bulk phase of the environment. Heat is lost from the animal in proportion to the thermal gradient (T) at the surface of the animal. Molecules of air or water in the insulation layer are warmed by the animal and then trapped within the insulation. The overall temperature gradient from the skin to the bulk phase is the same, but the distance is greater and the animal loses less heat to conduction. The fur also impedes the flow of fluids over the surface of the skin, so there is less convective heat loss.

The effectiveness of insulation depends on its thickness. When faced with cold temperatures, birds (or mammals) can change the orientation of the feathers (or fur) to alter the volume of air trapped within the coat. Similarly, animals that live in colder environments have thicker coats with greater insulating capacity (Figure 15.6). Some species change the

FIGURE 15.6 Insulation

There is a direct relationship between the thickness of fur and its ability to act as insulation.



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thickness of the external insulation seasonally. Thick coats are a thermoregulatory burden in the warm season, so it is beneficial to shed fur in spring. Because much of hair is composed of dead cells, the cost of rebuilding the coat when temperatures cool is minor in comparison with the metabolic costs the animal would incur trying to cool itself using physiological mechanisms. Mammals alter the nature of their fur coat seasonally, producing a greater density of hairs. Some birds, such as the ptarmigan, produce specialized feathers with an additional shaft to increase the feather density.

CONCEPT CHECK

1. What are the sources and sinks in an equation describing thermal balance?
2. How does insulation reduce heat loss?
3. Why might intertidal animals in the same microenvironment differ in T_B?

Thermal Strategies

There are many remarkable stories in the realm of thermal physiology, with species that are capable of tolerating extreme and rapidly changing temperatures. Consider the deep-sea vent animals discussed in the Chapter 14 opening essay. Some vent dwellers live on the edge of undersea volcanoes, and experience near-instantaneous changes in water temperatures, ranging from superheated vent water to frigid deep-sea water. More familiar may be the temperate zone animals that cope

with seasonal changes, and desert animals that survive daily fluctuations. Invertebrates are the most thermotolerant animals in each thermal niche. The hottest deserts are populated by myriads of insects, but only a few vertebrates. Invertebrates can also tolerate the coldest temperatures, often by entering an inactive, dormant state. Once stabilized in this state of "suspended animation," they can survive temperatures far colder than even the coldest natural environments. In contrast, only a few vertebrates, such as the wood frog, can survive subzero body temperatures, frozen in underground refuges.

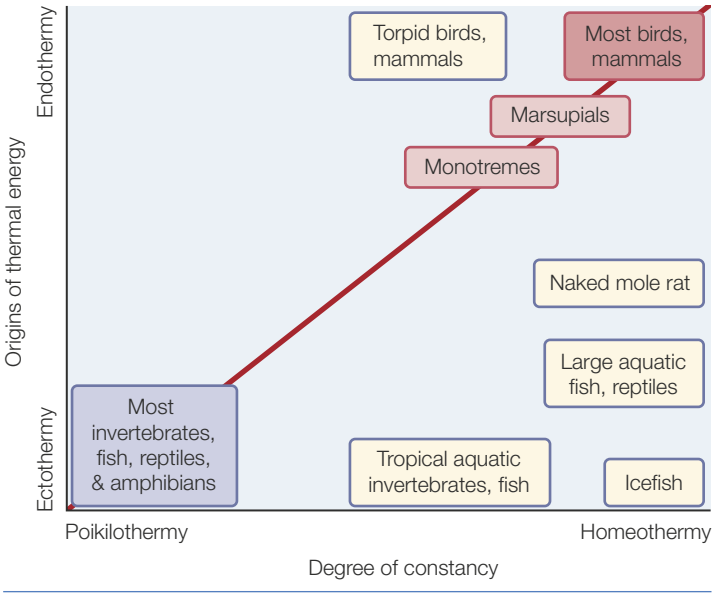
To understand the distinctions between the various thermal strategies, we begin by distinguishing them using two complementary criteria: the degree of T_B stability and the source of heat (Figure 15.7). The terms *poikilothermy* and *homeothermy* distinguish animals on the stability of their body temperature. *Endothermy* and *ectothermy* distinguish animals on the source of the heat that determines T_B. In the following sections, we explain these terms in more detail, and discuss the many animals that make even these simple distinctions a challenge.

Poikilotherms and homeotherms differ in the stability of T_B

A **poikilotherm** is an animal with a variable T_B—one that varies in response to environmental conditions. A **homeotherm**, in contrast, is an animal with a relatively constant T_B. The distinction between poikilotherm and homeotherm depends on both the properties of the animal and the nature of the environment.

FIGURE 15.7 Thermal strategies

Most animals can be classified as homeothermic endotherms (red) or poikilothermic ectotherms (blue), but there are many exceptions discussed in more detail in the accompanying text.



A homeothermic species has a near constant T_B, but the category does not distinguish between a species that achieves constancy through physiological processes versus other means. For example, many Antarctic fish live in waters that are invariably cold and die if subjected to even slightly warmer temperatures. Though fish in general are poikilotherms, these species are fairly defined as homeotherms. In contrast, most eutherian mammals are homeotherms, yet the naked mole rat (see Figure 15.5) allows T_B to change with T_A, and could be considered poikilothermic; it maintains T_B relatively constant by living in underground colonies where T_A changes little. Marsupials and monotremes are considered more poikilothermic than eutherians because they allow T_B to vary over a wider range.

Ectotherms and endotherms differ in the source of body thermal energy

The terms *ectotherm* and *endotherm* distinguish animals by the physiological mechanisms that determine T_B. The environment determines the T_B of an **ectotherm**. An **endotherm** is an animal that generates internal heat to maintain a high T_B. Figure 15.7 provides some examples of animals categorized on the basis of the mechanism that determines T_B.

Both this and the preceding approach to classifying thermal strategies works effectively for most animals. Most birds and mammals can be classified as homeotherms, because T_B is stable, and also as endotherms, because metabolic heat elevates T_B. Most reptiles, amphibians, fish, and invertebrates are poikilotherms, because T_B is variable, and also ectotherms, because the external conditions determine T_B. However, each group of organisms has exceptions. Polar icefish are homeothermic ectotherms; T_B is constant but determined by T_A. Large aquatic ectotherms, such as sea turtles and basking sharks, lack specific metabolic adaptations for heating, but remain warmer than the water by using a favorable surface area-to-volume ratio to retain metabolic heat. As discussed in the next section, there are also exceptions that arise when animals periodically deviate from their normal conditions (*temporal heterotherms*) or keep different parts of the body warmer than T_A (*regional heterotherms*).

Heterotherms exhibit temporal or regional endothermy

Just how constant does T_B have to be for an animal to be considered a homeotherm? In actuality, most animals experience some variation in temperature, either spatially or temporally. Many endothermic animals place greater priority on maintaining certain anatomical regions within very narrow thermal ranges. Typically, homeotherms maintain the central nervous system and

internal organs at a more constant temperature, while allowing the periphery to vary. The temperature of these deep, internal regions is often called the *core temperature*. Humans, for example, maintain a near-constant core temperature. However, regions of the human body can experience temperatures much lower than the core T_B. In the cold, humans change blood flow to allow hands and feet to cool to conserve internal heat. Males alter the position of the scrotum to keep spermatogenic tissue from overheating. However, human core T_B can also change under some circumstances. T_B can change in females during the reproductive cycle. It can rise several degrees as a result of a fever. In comparison with other animals, these are relatively minor regional and temporal differences in T_B, and a human is considered an endothermic homeotherm.

In contrast to humans, many other mammals and some birds can undergo dramatic, prolonged changes in T_B. When exposed to cold nighttime temperatures, T_B may decrease by several degrees (Figure 15.8). Hibernating mammals, such as ground squirrels and bats, allow T_B to drop for the winter months. Although these animals allow their bodies to cool, they are still considered endotherms because they produce and retain metabolic heat to maintain T_B above T_A. However, these endothermic animals are more precisely described as **temporal heterotherms**, to reflect the variability in T_B over time. Some ectothermic animals also fit the description of temporal heterotherms. Many large snakes, such as pythons, wind their bodies into a ball after they have ingested their prey. This helps the snake retain the metabolic heat produced by digestion. Temporal **heterothermy** is a strategy that has different benefits for endotherms and ectotherms. It allows an endotherm to conserve energy in cold temperatures by reducing the costs of thermoregulation. It provides an ectotherm with a period of accelerated metabolism to speed digestion, nutrient assimilation, and biosynthesis.

FIGURE 15.8 Short-term cooling in birds

Many temperate birds, such as the willow tit (*Parus montanus*), allow T_B to decrease when nighttime temperatures decrease. In this experiment, the birds were held at one of three temperatures, each maintained constantly throughout the night. This strategy of temporal heterothermy saves metabolic energy.

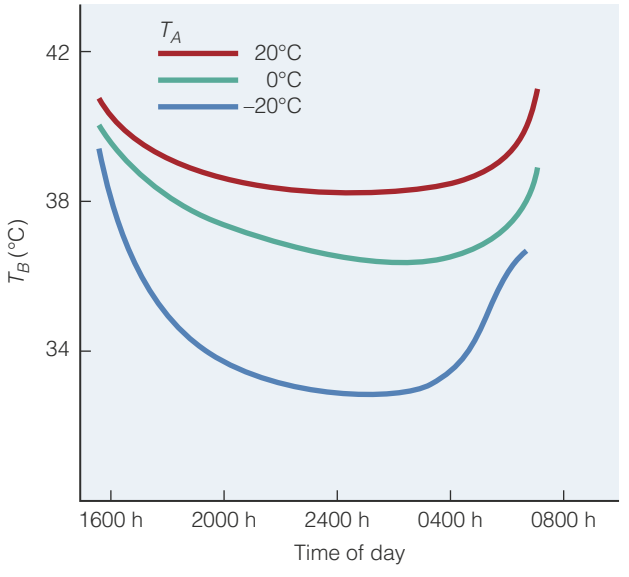


Figure source: Adapted from Reinertsen, R. E., & Haftorn, S. (1986). Figure 1a from Different metabolic strategies of northern birds for nocturnal survival. Journal of Comparative Physiology, Part B: Biochemical, Systemic, and Environmental Physiology. *Springer Science + Business Media,* 156: 655–663. Reprinted with permission.

Most ectotherms rapidly lose their metabolic heat to the environment, and consequently cannot elevate T_B much above T_A. However, a **regional heterotherm** can retain heat in certain regions of the body. Billfish, such as marlin and swordfish, are ectotherms but are also able to warm specific regions of the body. Their heater organs produce enough heat near the eye and optic nerves to improve visual clarity when they swim deep into cold waters (see Chapter 6). Large pelagic fish possess countercurrent heat exchangers to conserve the heat of digestion within the body core (see Chapter 14). Tuna and lamnid sharks are able to retain myogenic heat within the muscle. Warming of the red muscle increases metabolic capacity and may improve contractile performance during swimming (see Chapter 12). Thermal gradients occur within the bodies of many animals, but these regional heterotherms have specific physiological mechanisms to produce and retain heat regionally.

Although most insects are ectotherms, some species are regional heterotherms, others temporal heterotherms, and some species are both, depending on the time of year. The largest of flying insects, such as bumblebees, large moths, and cicadas, have a very high metabolic rate in the flight muscles. Thoracic temperature in a large flying insect can increase by more than 10°C, even while other regions of the body remain near T_A. Interestingly, these animals are also able to modulate heat production. Prior to flight they initiate thermogenic pathways to warm the thorax. When flight commences, they can alter heat exchange to maintain near-constant thoracic temperatures during flight, even when T_A is variable (Figure 15.9). Social insects use huddling as a means of controlling the temperature of the colony. Honeybees survive the cold winters by forming tightly crowded clusters. An individual bee in the colony is uniformly warm or uniformly cold, depending upon its position in the cluster. The clusters act like the body of a regional heterothermic animal. The "core" body heat of the colony is generated by the bees that are located near the center of the cluster. The outermost bees (mantle bees) act as insulation.

Animals have a characteristic degree of thermotolerance

Animals differ in their ability to tolerate changing ambient temperature. A **eurytherm** can tolerate a wide range of T_A, whereas a **stenotherm** can tolerate a narrow range of T_A.

FIGURE 15.9 Insect heterotherms

Many large insects are able to conserve metabolic heat that arises when their flight muscles are activated during flight. This warms the thorax while the rest of the body remains near ambient temperature, an example of regional heterothermy.

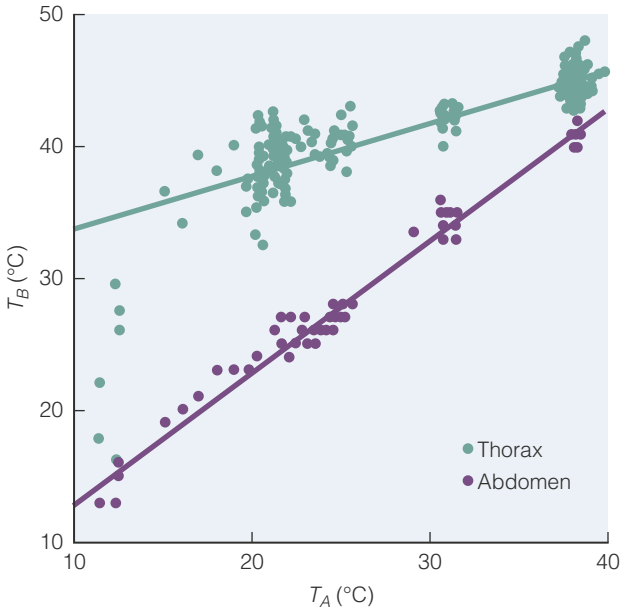
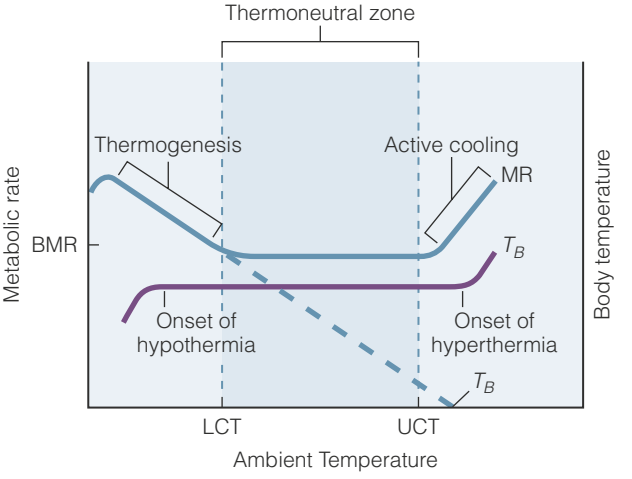


Figure source: From Harrison, J. F., Fewell, J. H., Roberts, S. P., & Hall, H. G. (1996). Figure 2 (p. 89) from Achievement of thermal stability by varying metabolic heat production in flying honeybees. *Science*, *274*, 88–90. Reprinted with permission from AAAS.

FIGURE 15.10 Zones of thermal effects of a resting homeotherm

Homeothermic endotherms maintain near-constant body temperature over a wide range of ambient temperatures (purple line). Once ambient temperatures decrease below the lower critical temperature (LCT), the animal must increase its metabolic rate (MR) to generate heat to help maintain a constant T_B. By extending the line explaining the metabolic rate below LCT to the x-axis, the body temperature (T_B) can be obtained as the intercept. Below a certain point, the animal can no longer maintain a constant core temperature and hypothermia results. When ambient temperatures increase past the upper critical temperature (UCT), the animal increases metabolic rate to shed heat. At still higher temperatures, the animal can no longer defend its body temperature and hyperthermia results.



These categories apply to animals regardless of whether they are ectotherms or endotherms.

Physiological strategies for coping with temperature differ in ectotherms and endotherms. For ectotherms, a change in T_A alters T_B and directly changes the rates of many biological processes. In contrast, an endotherm responds to a change in T_A by inducing a compensatory regulatory response. Despite the differences, both endotherms and ectotherms incur physiological costs and consequences when environmental conditions change.

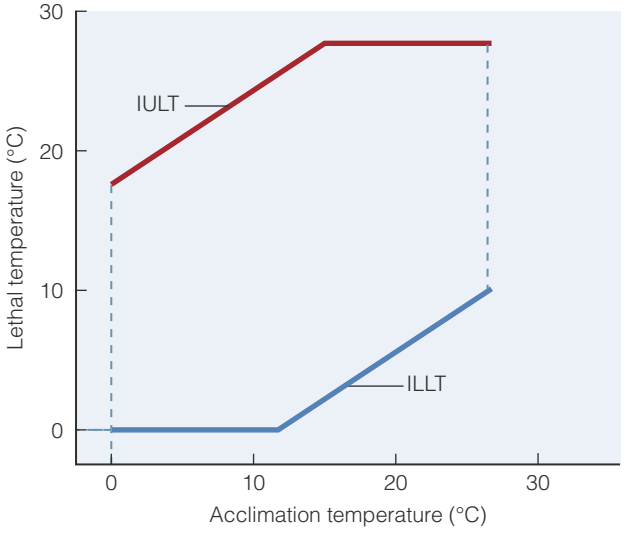
The effects of temperature can be defined in terms of its impact on animal function. An animal typically spends most of its life in a range of temperatures that is optimal for physiological processes. The **thermoneutral zone** of a resting homeothermic endotherm is the range of ambient temperatures where metabolic rate is minimal, which is considered the basal metabolic rate, or BMR (Figure 15.10). If temperatures rise to a point called the **upper critical temperature** (UCT), the metabolic rate rises as the animal induces a physiological response to prevent overheating. If the temperature falls below a **lower critical temperature** (LCT), the metabolic rate rises to increase heat production. For many animals, the T_B can be predicted from the extrapolation of the line that describes the metabolic rate at temperatures below LCT. When faced with a hypothermic challenge, animals may reduce T_B to maintain homeostasis at metabolic rate. In general, these compensatory responses at high T_A or low T_A allow the animal to maintain a constant T_B, but beyond a point, the animal cannot sustain a constant T_B.

The concept of a thermoneutral zone does not apply to animals that alter T_B, but ectotherms also have ranges of T_A (and T_B) within which growth and reproduction are optimal. Animals actively seek out their *preferred temperature*, a T_A that is within its range for optimal function. At low temperatures, all developmental processes slow because the lower T_A reduces the rate of metabolic reactions. Higher temperatures damage molecules, cells, and tissues, jeopardizing an animal's health. Researchers can assess the thermal tolerance of an ectotherm or a poikilotherm by transferring an animal from its acclimation temperature to a new temperature and assessing survival. The *incipient lethal temperature* is the temperature that has a 50 percent probability of killing the animal within an identified period. The range of tolerance is the difference between the **incipient upper lethal temperature** (IULT) and the **incipient lower lethal temperature** (ILLT) (Figure 15.11).

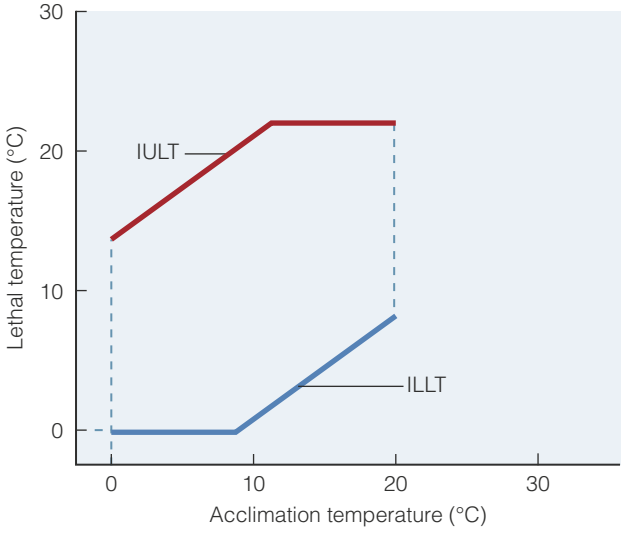
For ectotherms and poikilotherms, the ability to tolerate temperature changes with thermal history. Many temperate

FIGURE 15.11 Temperature polygon

Acclimation affects the incipient upper lethal temperature (IULT) and incipient lower lethal temperature (ILLT) for ectotherms and poikilotherms. The tolerance of an animal is reflected in the area of the polygon created by joining the upper line (IULT, in red) and the lower line (ILLT, in blue). Analysis of a eurythermal fish **(a)** yields a larger polygon than that of a stenothermal fish **(b)**.



(a) Eurythermal fish



(b) Stenothermal fish

zone animals remodel their cells, tissues, and systems to alter their sensitivity to temperature. Whether responding to temperature (thermal acclimation) or the more complex seasonal changes (seasonal acclimatization), the remodeling response makes the animal better able to cope with the effects of temperature. The impact of thermal history can also be seen in Figure 15.11, where acclimation to a high temperature tends to increase both the ULLT and ILLT. Likewise, acclimation to a low temperature reduces the upper and lower lethal temperature points. The physiological remodeling in response to temperature may include modifications of cellular features, such as the nature of membranes and levels of critical enzymes, or tissue properties, such as gill surface area.

Eurythermal endotherms/homeotherms possess a wide thermoneutral zone, maintaining a constant T_B over a wide range of T_A; eurythermal ectotherms/poikilotherms display a large thermal tolerance polygon area, with well-separated incipient lethal temperatures.

Temperature affects metabolic scope

The intrinsic links between metabolism, metabolic rate, and temperature mean that effects on one process inevitably influence the others. In many cases, animals survive stress by producing enough energy to overcome the negative consequences of the stress. For a poikilotherm, an increase in T_A increases T_B, which increases the standard metabolic rate (SMR) of the animal, requiring more energy to be consumed and expended, even at rest. However, the animal does not have the physiological capacity to increase its metabolic rate indefinitely in response to an increase in temperature. At some point, temperature causes the SMR to increase to the maximal metabolic rate (MMR), and such an animal would not be able to generate enough energy to do anything more than just survive.

This relationship is articulated in the **oxygen- and capacity-limitation of thermal tolerance** hypothesis (OCLTT). Consider the plight of a poikilothermic animal that experiences an increase in environmental warming (Figure 15.12). Its SMR increases with environmental temperature. The maximal metabolic rate increases likewise, but at some point, called the **pejus temperature** (T_p) the maximal physiological capacity declines as systems deteriorate or fail to meet metabolic demands. At the critical temperature (T_c), the rise in SMR equals the MMR, and the animal has no scope for aerobic activity.

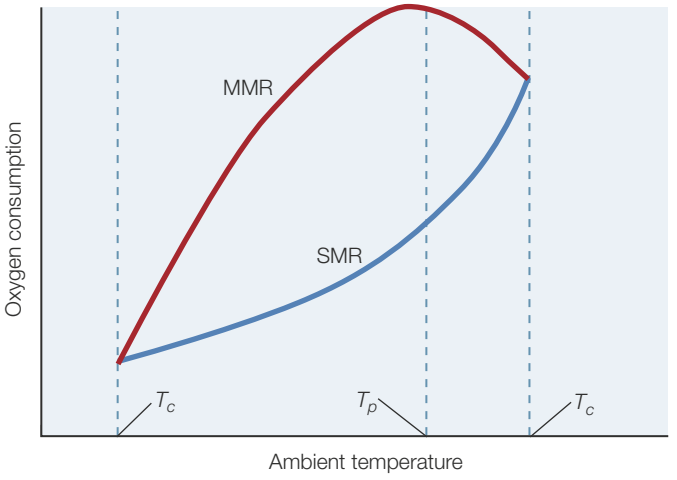
These threshold points are distinct from the incipient lethal temperatures shown in Figure 15.11; the animal dies when it exceeds the IULT or ILLT. In contrast, the OCLTT hypothesis deals with long-term survival of organisms. Short-term deficits in aerobic demand can be met by anaerobic pathways; however, the animal is unable to grow or survive over the long term.

The evolution of thermal tolerance has complex origins

Differences in thermotolerance can be observed in comparisons of populations or species that have evolved in regions separated by latitude, altitude, or other forms of thermal gradients. The ability of an animal to tolerate a lower T_A than its competitor allows the tolerant animal to expand into a

FIGURE 15.12 Oxygen- and capacity-limitation of thermal tolerance

As ambient temperature increases, a poikilotherm experiences increases in standard metabolic rate (SMR) and maximal metabolic rate (MMR). At the pejus temperature (T_p), the MMR begins to decline, eventually reaching the SMR. The upper and lower temperatures beyond which the animal has no aerobic scope are identified as the critical temperatures (T_c).



colder environmental niche. Many closely related animals have distinct differences in thermal preferences that contribute to their geographical distributions. Latitudinal patterns are common in both marine and freshwater fish species. Closely related species of barracuda, for example, live at specific latitudes along the Pacific coast with a characteristic average T_A. From north to south, one species gradually replaces another once the average water temperature changes by only 3–8°C. There are also altitudinal patterns seen with terrestrial animals. Many bird species exist in high-altitude and low-altitude populations, each with physiological specializations and morphological differences. The thermal environment resulting from the combination of altitude and latitude also determines the range of many amphibians. Andean tree frogs (*Hyla andina*) can be found at low elevation far from the equator, but closer to the equator they can live at higher altitudes.

The genetic basis of a difference in thermotolerance is not always clear. We can often determine why levels or properties of a single protein differ in two animals in relation to temperature. However, the underlying basis for intricate differences in thermal physiology is more complex. For example, two species of Siberian hamsters, *Phodopus campbelli* and *P. sungorus*, differ in thermal biology in terms of morphology, insulation, behavior, and physiology. Although these are very closely related species, they last shared a common ancestor more than 2 million years ago. A complex trait such as fur density depends on multiple genes, many cell types, and networks of genetic regulators. Furthermore, the two species may have many genetic differences, but only some of these may influence their thermal biology.

An understanding of the physiological basis of thermotolerance is vital to predicting the effects of global environmental change. In Box 15.1: Applications: Thermal Tolerance and Conservation Biology of Atlantic Cod, we discuss how recent changes in water temperature have influenced populations of Atlantic cod.

CONCEPT CHECK

4. What is the difference between an endotherm and an ectotherm?
5. What is the difference between a homeotherm and a poikilotherm?
6. What is the difference between a regional and a temporal heterotherm?

COPING WITH A CHANGING BODY TEMPERATURE

Although many ectotherms and poikilotherms live in thermally stable environments—underground burrows, tropical rainforests, the deep sea, or a homeotherm's intestine—others must cope with frequent and dramatic changes in T_B. Because of the effects of temperature on macromolecular function and metabolism, ectotherms and poikilotherms must either tolerate or compensate for the complex, often deleterious, effects of changing temperature.

Macromolecular Structure and Metabolism

Of the four classes of macromolecules, only proteins and lipids are substantially affected by temperature over the normal range encountered by animals. Weak bonds (van der Waals forces, hydrogen bonds, and hydrophobic interactions) govern the interactions within and between these macromolecules. Each type of bond has a characteristic response to temperature. Whereas hydrogen bonds and van der Waals forces are disrupted at high temperature, hydrophobic interactions are stabilized at high temperature. Thus, the effects of temperature on macromolecular structures depend on the relative importance of each type of bond.

Animals can remodel membrane fluidity

In Chapter 3, we discussed the structure of cellular membranes and the importance of membrane fluidity. Van der Waals forces hold membrane lipids together. Although the interactions between phospholipids are strong, the membrane must also remain fluid enough to allow proteins to rotate and diffuse laterally within the membrane. Low temperatures cause membrane lipids to solidify, which impairs

APPLICATIONS 15.1

THERMAL TOLERANCE AND CONSERVATION BIOLOGY OF ATLANTIC COD

Fish have proven to be a useful model for exploring the potential impact of global environmental change on organisms. Animals in a constant or predictable environment often evolve physiological traits that make them well suited to that environment. As the environment changes, animals are faced with a physiological challenge. As individuals they can survive using physiological responses, and within a population there will be individuals that are better able to tolerate the environmental stressor. If the environmental change is sustained, it is likely that the nature of the population changes. Though dramatic environmental change is possible on a local scale, in most cases the rate of change is sufficiently slow to permit evolution of populations. When the rate is too rapid, populations can become extinct. Global warming is an unusual environmental stressor because it is happening relatively quickly and on a massive scale. In many cases the success or failure of populations can be traced back to physiological properties. In the case of thermal stress, there is growing evidence that survival at high temperatures depends on cardiovascular (supply) limitations. These physiological traits, in turn, influence the natural ranges of animals.

The Atlantic cod (*Gadus morhua*) has a thermal optimum (T_o) of about 5°C, with pejus temperatures (T_p) of 2°C and 7°C. Recall that the T_p is the temperature beyond which the physiology of the animal deteriorates. The North Sea population of this species is able to tolerate temperatures as high as 16°C. Beyond this critical temperature (T_c), the oxygen tension of the venous blood drops dramatically, and heart rate becomes irregular.

The optimum temperature for growth in cod also depends on body mass (Figure 15.13a). The smallest fish have a higher thermal optimum for growth. As the cod grows, its optimal temperature (for growth) decreases. However, the lower thermal tolerance changes little, which means that the range for optimal growth is much narrower in large fish. In recent years, the North Sea has grown warmer, and the cod have moved northward. This shift is thought to be due to the thermal sensitivity of the large spawners, which have a lower T_o. It is not yet known if this local environmental change has translated into microevolutionary changes in thermal tolerance. However, Atlantic cod display population-level adaptation for thermal environments. For example, cod from cooler Icelandic waters have a lower T_o, T_p, and T_c. As discussed in a previous feature on sockeye salmon (Box 12.2), the underlying constraint that limits thermal tolerance may be related to the cardiorespiratory system.

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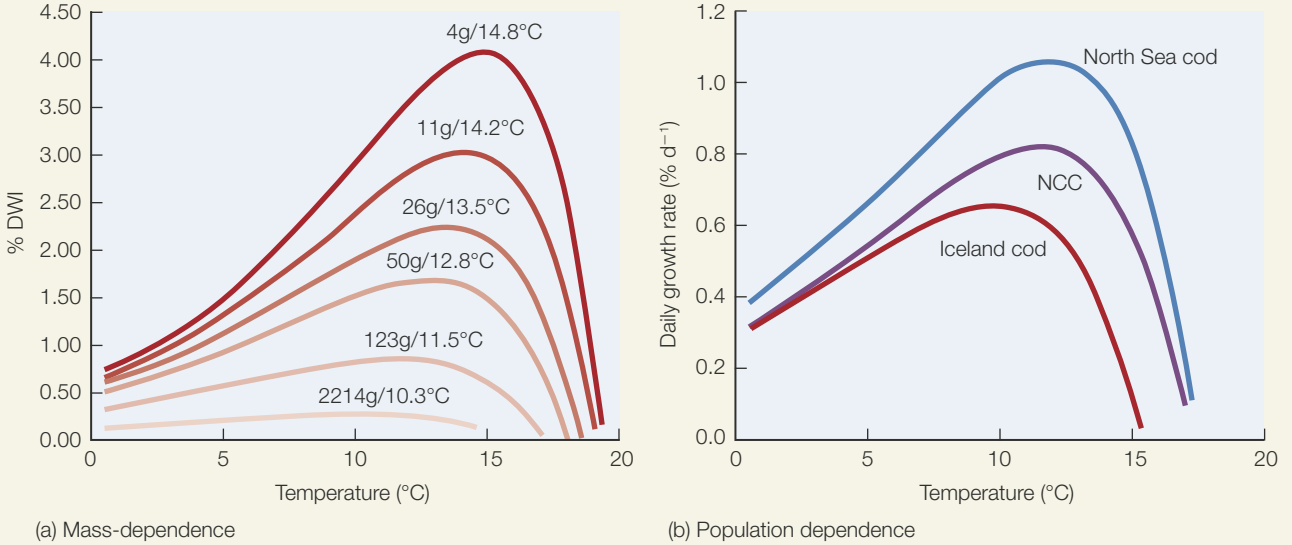
FIGURE 15.13 Thermal optima for cod growth



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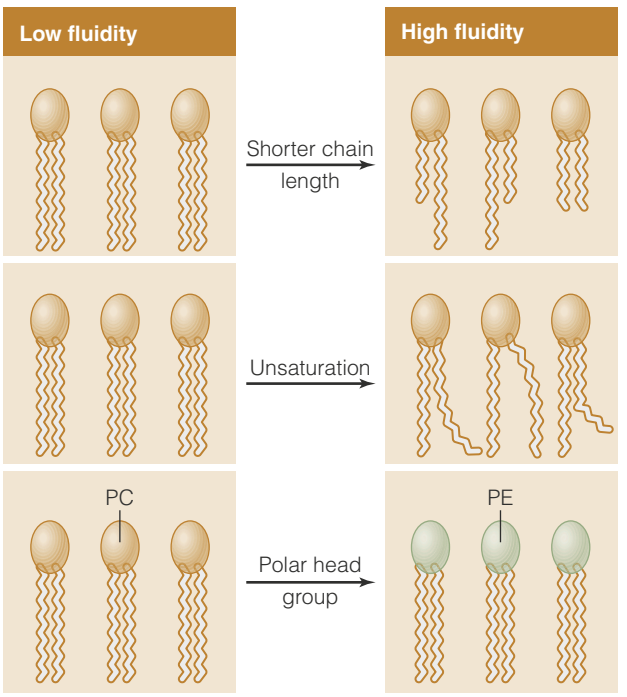
protein movement. Conversely, high temperatures liquefy the membrane, which can compromise its integrity and reduce its effectiveness as a permeability barrier. Cells regulate the balance between the solid *gel* state and the liquid *sol* state.

Ectothermic animals reduce the deleterious effects of temperature by changing the composition of their membranes. In this process, called **homeoviscous adaptation**, cells remodel membranes to preserve fluidity. Three mechanisms target phospholipids (Figure 15.14), and a fourth mechanism alters cholesterol content.

1. **Fatty acid chain length.** Phospholipids with short-chain fatty acids cannot form as many interactions with adjacent fatty acids and therefore are highly mobile. The effectiveness of chain shortening depends upon the fatty acid position on the phospholipid. Due to the three-dimensional structure of a phosphoglyceride, a short-chain fatty acid in position 1 makes a greater contribution to enhancing fluidity than does the same fatty acid in position 2.
2. **Saturation.** Double bonds create a kink in the fatty acid chain that prevents effective bond formation with other fatty acids. With fewer bonds between fatty acid chains, the membrane is more fluid. For example, pure stearic acid (C18:0) becomes liquid only at temperatures above 69°C, whereas oleic acid (C18:1) is liquid at 12°C. The position of the double bond is also critical. A double bond near the midpoint of the fatty acid chain (as with oleic acid) is more effective than a double bond near the end of the fatty acid chain.
3. **Phospholipid classes.** The difference in the shape of the polar head groups alters the ability of the phospholipids to interact at the surface of the membrane. Phosphatidylcholine (PC) is more common in membranes of warm-acclimated cells, whereas phosphatidylethanolamine (PE) is more common in cold-acclimated cells. The ratio of PC to PE decreases in cold acclimation and adaptation.
4. **Cholesterol content.** A pure phospholipid bilayer is mostly fluid at high temperature and mostly solid at low temperature. Cholesterol has a dual effect that makes it useful in buffering thermal effects on membrane fluidity. Cholesterol in a fluid membrane tends to make it more stable by plugging spaces in between phospholipids, buffering against a loss of integrity at high temperatures. When cholesterol is in a fluid membrane that is being cooled, it disrupts the structure in a way that prevents the membrane from solidifying.

FIGURE 15.14 Phospholipid properties and membrane fluidity

Cells change the fluidity of membranes by altering the composition of membrane phospholipids.



Cells use two general pathways to modify membrane composition in response to temperature: **in situ** modification and *de novo* synthesis. Both pathways require cells to modify the properties of the fatty acids within the fatty acid pool using suites of enzymes that elongate, shorten, saturate, and desaturate fatty acids. Because these enzymes begin with fatty acids derived from the diet, the nature of the diet also affects the profile of fatty acids within the membrane.

Enzymes alter the structure of individual phospholipids directly within the membrane (Figure 15.15). First, phospholipase A removes an acyl chain from membrane phospholipids to form a lysophospholipid. Next, lysophospholipid acyltransferase uses a more appropriate fatty acid (in the form of fatty acyl CoA) to rebuild the phospholipid.

More commonly, membranes are remodeled by endocytosis and exocytosis (see Figure 15.16). The old membrane is removed using endocytosis. Phospholipids are synthesized *de novo* within the endoplasmic reticulum, then packaged into vesicles that fuse with cellular membranes.

Animals remodel membranes to maintain near-constant fluidity

Membrane fluidity is measured in biological membranes using a dye (diphenyl hexatriene) that changes in optical properties in relation to its freedom to move within the membrane (Figure 15.17). When membranes from different species are compared, each exhibits a decrease in fluidity (measured as a change in optical properties) when the membrane is cooled. Taking into consideration the differences in thermal niche, this analysis shows that animals produce membranes that exhibit the same fluidity at the natural temperature. This

FIGURE 15.15 Phospholipid remodeling

Cells can remodel the phospholipids directly within membranes by removing a fatty acid. A phospholipid is rebuilt by lysophospholipid acyltransferase, which attaches another fatty acid produced by the cell. The fatty acid must first be activated by the esterification of coenzyme A.

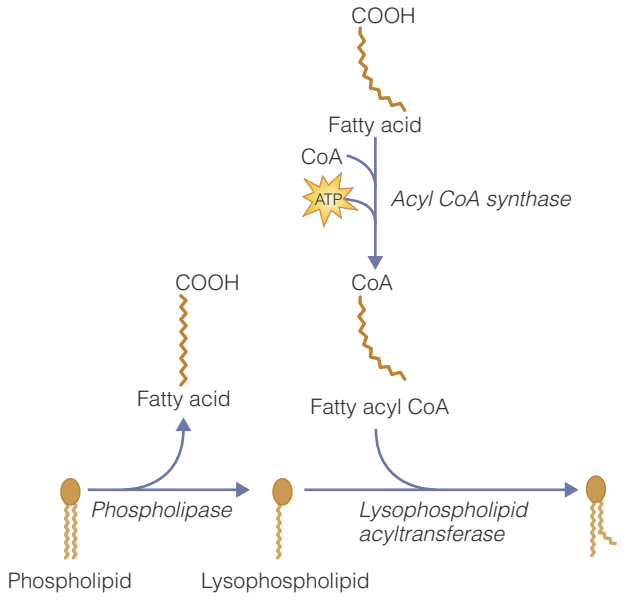


FIGURE 15.17 Conservation of membrane fluidity

Membranes are treated with a dye (diphenyl hexatriene) with optical properties that change in relation to membrane fluidity. Anisotropy is an optical property that reflects the ability of a dye to alter the behavior of plane polarized light. Anisotropy is inversely related to fluidity; at warmer temperatures, a decrease in anisotropy reflects an increase in fluidity. Animals that live in different environments produce membranes that possess a similar fluidity at their normal range of temperatures (indicated by the thickened portion of the lines).

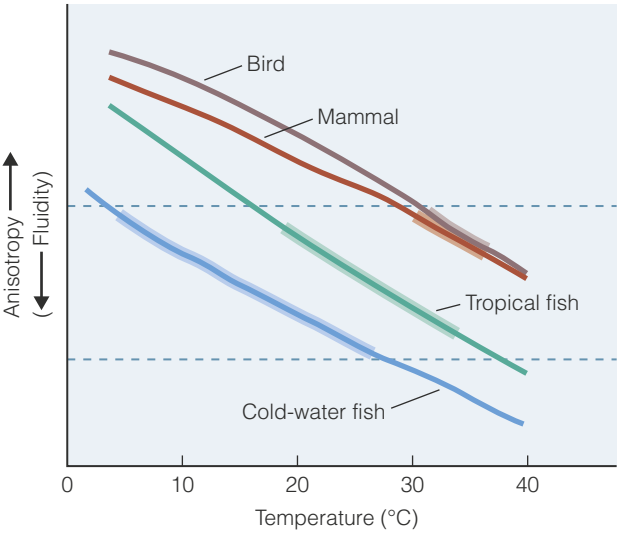


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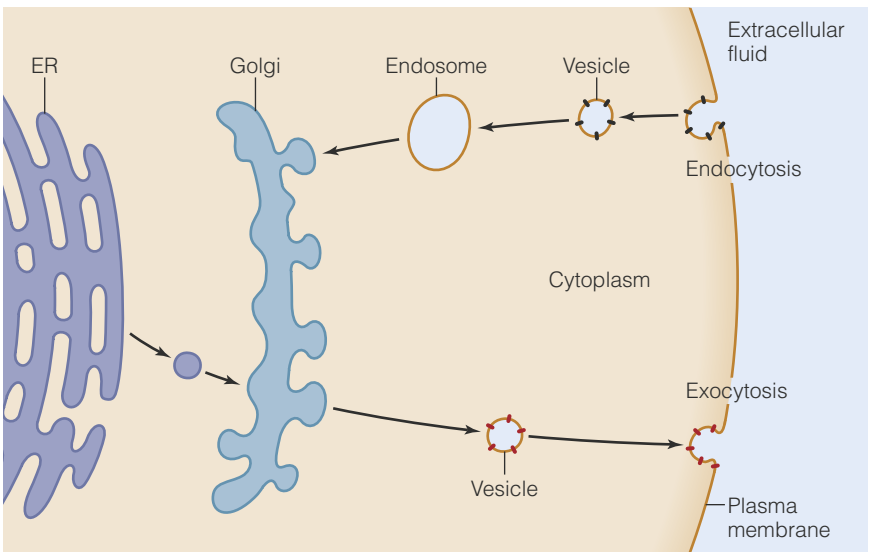
FIGURE 15.16 Membrane remodeling

Cell membranes are constantly remodeled by endocytosis and exocytosis. When temperature decreases, the cell produces vesicles possessing phospholipids with fatty acids that are shorter and more unsaturated than those in the cell membrane. Over time, the cycles of endocytosis and exocytosis remove undesirable phospholipids, replacing them with more desirable phospholipids.



observation is analogous to the conservation of K_m seen in enzymes from animals in different niches (see Chapter 3). The same pattern is seen when an animal is acclimated to different temperatures.

Temperature changes enzyme kinetics

Temperature affects protein structure and function in complicated ways. Changes in temperature alter the number of bonds that form within and between molecules. Even minor changes in protein structure can have noticeable effects on protein function. In enzymes, for example, these structural effects manifest as changes in catalytic properties. First, changes in weak bonds can alter the three-dimensional structure of the enzyme. For instance, warm temperatures could break bonds that are necessary to fold the protein in a way that forms the active site. Second, temperature can alter

the ionization state of critical amino acids within the active site. For instance, the amino acid histidine is important in many active sites, and changes in histidine protonation state can alter enzyme substrate affinity. Any increase or decrease in K_m could be disruptive. Third, temperature can alter the ability of the enzyme to undergo the structural changes necessary for catalysis. Enzymes must be rigid enough to maintain the proper conformation, but flexible enough to undertake conformational changes during catalysis. Thus, temperature can affect enzyme kinetics through effects on maximal velocity (V_{max}) or affinities for substrates (K_m), allosteric activators (K_a), and inhibitors (K_i). When animals experience a change in T_B, they may either tolerate the effects on enzyme kinetics or alter metabolic regulation to compensate.

Biochemical reactions are accelerated by higher temperature and reduced at lower temperature. The rate of a chemical reaction depends on the proportion of molecules within the system that possess energy equal to or greater than the activation energy (E_A). As temperature increases, the average kinetic energy of the substrates increases and a greater proportion of molecules has sufficient energy to be converted to products, causing the enzyme velocity to increase (see Figure 3.14). For most enzymes working over a biologically relevant range of temperatures, an increase of 10°C results in a two- to threefold increase in reaction velocity. Researchers express the effects of temperature on rates using an *Arrhenius plot* (see Box 15.2: Math in Physiology: Evaluating Thermal Effects on Physiological Processes Using Q_{10} and Arrhenius Plots).

Evolution may lead to changes in enzyme kinetics

When animals are exposed to suboptimal temperatures for generations, there is the possibility of evolutionary changes in the genes encoding enzymes. We draw again on research with LDH for examples of evolutionary changes that cause differences in enzyme kinetics as well as enzyme synthesis.

Mutations may lead to structural changes in the enzyme that impart a favorable difference in enzyme kinetics. As we learned in Chapter 3, lowering temperature increases the affinity of LDH for its substrate pyruvate (see Figure 3.43). Evolution has led to a fine-tuning of enzyme properties such that subtle structural differences allow each species to possess a similar K_m at its respective normal T_A. This strategy, called *conservation of* K_m, is commonly seen when we compare the effects of temperature on the enzyme kinetics of different animals.

Alternately, mutations in the promoter for an enzyme cause a change in the level of gene expression of an otherwise unchanged enzyme. If such changes are beneficial, they may evolve to become fixed in the population. Killifish live along the eastern coast of North America from Newfoundland to Florida. Within the population as a whole, there are different alleles of the LDH-B gene. One allele predominates in northern populations, while another allele predominates in southern populations. Intermediate populations have both alleles. These alleles have differences in enzyme properties and differ in the level of gene expression. The northern allele is expressed at twofold higher levels than the southern allele, due to mutations in the promoter. The northern fish produce more LDH enzyme molecules, which compensates for the debilitating effects of temperature on enzymatic activity that would occur as a result of living in the colder waters.

Ectotherms can remodel tissues in response to long-term changes in temperature

Many ectothermic animals remodel their cellular machinery to mitigate the effects of variation in T_B. In the laboratory, where the researcher changes only T_A, this remodeling process is called *thermal acclimation*. In the natural world, seasonal transitions in temperature are accompanied by other environmental changes and the response of the animal to complex seasonal changes is called *acclimatization*. In winter, photoperiods get shorter, food may be less abundant, and oxygen levels may change. The complexity of these seasonal environmental changes makes it difficult to link remodeling with the temperature. On one hand, there is uncertainty about the trigger for the remodeling process: Is the change initiated by changes in temperature or by some other factor, such as photoperiod? On the other hand, it is not always clear that the remodeling itself serves to compensate specifically for temperature.

Temperature-dependent remodeling involves combinations of quantitative and qualitative strategies. In Chapter 13, we discussed how ectotherms remodel their muscles in response to temperature. Low temperature may increase the number of mitochondria in muscle, or trigger the hypertrophic growth of the heart. This is an example of a *quantitative* strategy; there is simply more of the same machinery. Muscles can also alter the types of proteins they use to build the contractile machinery. For instance, animals express different myosin isoforms in winter and summer—an example of a *qualitative* strategy.

Surprisingly little is known about the hormones and signaling pathways that cause an ectotherm to remodel its tissues during acclimation and acclimatization. Cold-sensing and warm-sensing neurons are important for detecting temperature, but the links to gene expression are not well known. In some cases, seasonal changes in physiology that mitigate the effects of temperature are triggered by changes in the photoperiod. In Chapter 8, we discussed the importance of the various photoperiod signaling pathways that act through the hypothalamus and pineal glands.

MATH IN PHYSIOLOGY 15.2

EVALUATING THERMAL EFFECTS ON PHYSIOLOGICAL PROCESSES USING Q_{10} AND ARRHENIUS PLOTS

For many physiological processes, an increase in temperature typically increases the rate of the process. The sensitivity of a reaction to temperature is expressed as the Q_{10} value, which is essentially the ratio between reaction rates at two temperatures, adjusted for a 10°C temperature difference. It is calculated as

$$Q_{10} = \left[\frac{K_2}{K_1}\right]^{[10/(T_2 - T_1)]}$$

where the rates of a reaction (K) are compared at two temperatures (1 and 2). Thus, if a rate of 10 units/min (K_1) was observed at 15°C (T_1), and a rate of 20 units/min (K_2) at 25°C (T_2), then

$$Q_{10} = \left[\frac{20}{10}\right]^{[10/(25 - 15)]} = 2^1 = 2$$

A Q_{10} value for a reaction allows you to make predictions about the potential impact of temperature on reaction rates. A desert lizard may experience a change in temperature of as much as 30°C over a single day, from the midday heat to the cool morning. Consider how temperature affects the maximal activity (V_{max}) of lactate dehydrogenase (LDH) in the lizard muscle as it experiences changes in T_B. Over this time frame, the total number of LDH enzyme molecules does not change appreciably, but the catalytic activity changes with temperature. Assume that LDH displays a $Q_{10} = 2$, and that the lizard tissue at 35°C has an LDH $V_{max} = 400$ U/g tissue (1 unit of enzyme converts 1 µmol of substrate to product each minute). Using the Q_{10} equation, calculate the effects on V_{max} when T_B decreases from 35°C to 25°C, 15°C, and 5°C. Over the course of a single day, changes in T_B mean the desert lizard experiences an eightfold change in its LDH V_{max}. To put this into context, if you were to undergo extensive weight training, your muscle LDH V_{max} might change only twofold.

How does a lizard cope with an eightfold decrease in enzyme activity? Superimposed on the Q_{10} effects are numerous layers of metabolic regulation that ensure that energy metabolism remains in homeostasis. If an individual enzyme is more sensitive to temperature than are other enzymes in the pathway, the cell has several options to increase flux through that step. Most enzymes do not operate near their V_{max}, so changes in flux can arise through changes in substrate or product concentrations. Enzymes can overcome debilitating effects of temperature by changes in kinetic regulation through allosteric effectors or covalent modification.

Another factor to consider is that a reduction in temperature reduces both anabolic and catabolic reactions. It is easy to imagine how an eightfold reduction in LDH capacity might severely impair the capacity to produce ATP by glycolysis. However, rates of ATP synthesis decline in parallel with the rates of ATP utilization, with each step exhibiting a Q_{10} ranging from 2 to 3. Put another way, the animal can tolerate lower rates of muscle ATP production because it slows down and needs less ATP for muscle activity. However, it is important to recognize that $Q_{10} = 2$ is quite different from $Q_{10} = 3$. The levels of ATP in a tissue reflect a balance between rates of synthesis and degradation. If a 10°C decrease in temperature caused ATP synthesis to decrease threefold when ATP demands decreased only twofold, the tissue would be depleted of ATP within seconds or minutes.

The Q_{10} for a process is the best way to express the influence of temperature on reaction rates, but a better approach to exploring the mechanism of action is through an **Arrhenius plot**. In the late 1800s, the chemist Svante Arrhenius described a mathematical approach to exploring the impact of temperature on macromolecular processes. We now use his approach to study processes such as enzymatic reactions, diffusion of molecules, and lipid membrane phase transitions. The sensitivity of a reaction to temperature reflects the activation energy (E_A) of the process. The Arrhenius equation describes the relationship between the

CONCEPT CHECK

7. What is homeoviscous adaptation?
8. What is conservation of K_m in relation to temperature effects on enzymes?
9. How can an animal alter membrane fluidity?

Life at High and Low Body Temperatures

Animals that can tolerate extreme temperatures can invade and colonize niches that are underexploited by their competitors. Ectothermic animals exposed to thermal challenges must possess mechanisms to mitigate the effects of temperature on macromolecular structure and metabolism. In

activation energy, temperature, and the rate of the process under study:

$$k = Ae^{[-E_a/RT]}$$

More often, the Arrhenius equation is shown as

$$\ln[k] = \ln[A] - E_a/[RT]$$

where k is a rate coefficient, R is the gas constant (8.31447 $\times 10^{-3}$ kJ/K per mol), T is temperature (in degrees kelvin), A is called the pre-exponential factor, and E_a is the activation energy (kJ/mol).

The versatility of the Arrhenius plot allows researchers to describe the thermal behavior of any simple or complex process. Figure 15.18 displays the results of an experiment comparing the effects of temperature on two enzymes.

The researcher varied temperature over a range of interest and measured enzymatic rates. The data she collected could be plotted on a graph with axes chosen from a rearrangement of the Arrhenius equation that generates a linear equation ($y = mx + b$):

$$\ln[k] = -E_a/R \times [1/T] + \ln[A]$$

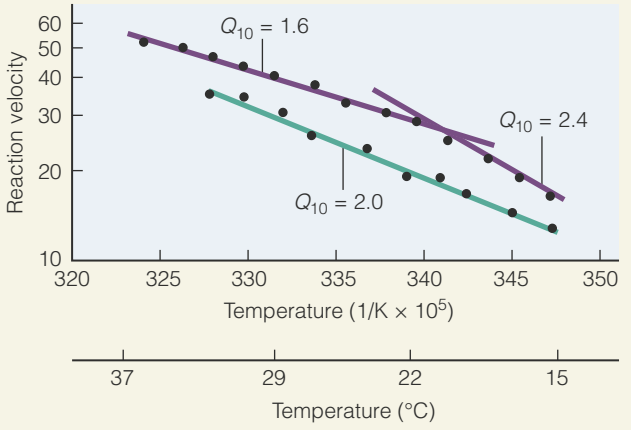
Plotting $\ln(k)$ versus $1/T$ gives a slope of E_a/R and a y intercept of $\ln(A)$.

This Arrhenius plot illustrates two potential outcomes for an enzyme. For the green line, the data fall along a straight line. The slope of the line reflects the activation energy of the reaction. This is the type of data that would be expected from analysis of a cytosolic (soluble) enzyme such as LDH.

The purple lines show data where one line fits the data at low temperatures, but a different line fits the relationship at high temperatures. The point where the two lines cross is called the **breakpoint**. Because the slope differs between the two lines, we can infer that different activation energies govern the reaction over each temperature range. In many cases, this is due to a mechanistic transition from one state to another state. If the enzyme under consideration is a membrane enzyme, for instance, the breakpoint might reflect the transition from a liquid to a solid phase. If the process is an enzymatic reaction, the breakpoint might occur at a temperature where a critical bond is broken, converting the enzyme from an efficient catalyst to a less-efficient catalyst or a partially denatured enzyme.

This type of analysis can be used to explore complex processes, such as whole metabolic pathways. However, the results reflect a complex summation of the thermal sensitivities of the various individual steps. Though useful for characterizing the effects of temperature on such pathways, it is much more difficult to assess underlying mechanisms that explain the thermal sensitivity.

FIGURE 15.18 Arrhenius plot



Reference

- Metz, J. R., van den Burg, E. H., Bonga, S. E., & Flik, G. (2003). Regulation of branchial Na^+/K^+ ATPase in common carp *Cyprinus carpio* L. acclimated to different temperatures. *Journal of Experimental Biology, 206*, 2273–2280.

contrast, endothermic animals survive thermal extremes using complex regulatory pathways to maintain a constant T_B. Their existence at extremes is a testament to their physiological capacity to resist the effects of T_A.

Some enzymes display cold adaptation

Earlier in this chapter we discussed how relatively subtle differences in T_A can lead to evolutionary changes in enzyme structure and gene expression. However, the need for enzymatic structural modification is much more pronounced at thermal extremes, particularly at the subzero temperatures encountered in polar seas. Psychrotrophs are organisms that thrive in the extreme cold, in contrast to mesotrophs that live at more moderate temperatures. Animal psychrotrophs, including polar invertebrates and fish, remain active at body temperatures near the point of freezing. Many psychrotrophic organisms possess cold-adapted proteins that function optimally at very

low temperatures. Although these enzymes are more stable in the cold, they are rapidly inactivated at slightly higher temperatures.

The catalytic and structural differences between enzymes of psychrotrophs and mesotrophs can be traced to the weak bonds that stabilize enzyme structure. Enzymes undergo pronounced changes in three-dimensional shape during the catalytic cycle, known as protein breathing. During these transitions in folding, weak bonds break and form. When temperatures decrease, most of these weak bonds are strengthened, stabilizing the protein in a form that occupies a smaller volume. In this conformation, it is much harder for the protein to breathe, and consequently enzymes in the cold are less efficient. The psychrotroph enzyme has fewer weak bonds stabilizing its structure; it occupies a larger volume and has an easier time breathing during catalysis. The reduced stability allows it to function better in the cold, but makes it vulnerable to temperature-dependent unfolding. In comparison to mesotroph enzymes, cold-adapted enzymes are more efficient enzymes at low temperatures, but inferior enzymes at high temperatures.

Unique loss-of-function mutations also occur in polar animals. Many Antarctic fish have lost the ability to express functional oxygen-binding proteins, such as hemoglobin and myoglobin. These fish can survive without these oxygen carriers because they have low metabolic rates and the surrounding polar waters are rich in oxygen.

There are many such examples of thermal adaptations of individual selected genes in polar animals. However, more controversial is the question of whether or not polar animals have a fundamentally different organization of metabolism as a result of evolution in the extreme cold. Early studies suggested that polar animals had metabolic rates that were much higher than the metabolic rates of temperate animals measured near 0°C. These observations were used to support a theory that became known as metabolic cold adaptation. It was proposed that thousands of years in the extreme cold led to evolutionary changes that provided these polar animals with an ability to elevate their metabolic rate. Even with years of study it remains unclear whether metabolic cold adaptation is a real phenomenon. The earliest studies were based on comparisons of goldfish and arctic cod. Now that more species have been analyzed using more sophisticated technologies, it seems less likely that metabolic cold adaptation occurs as a general phenomenon. Nonetheless, many studies have identified evolutionary differences and physiological peculiarities in some polar animals.

Stress proteins are induced at thermal extremes

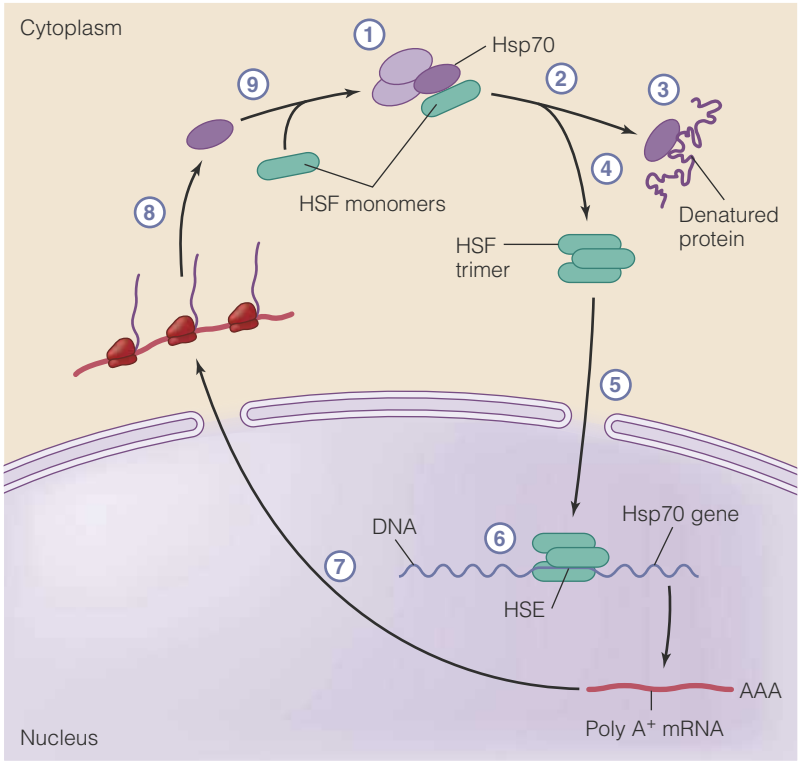
Many proteins are best suited to function over narrow ranges of temperature that span the biological range of the animal. During the normal structural change that occurs when a protein breathes, the protein is vulnerable to further changes in structure. Occasionally, the protein can unfold or misfold into a nonfunctional conformation. This denatured protein must be repaired or cleared from the cell before it disrupts other cellular functions. Denaturation is a normal process, and cells are able to detect and remove denatured proteins using pathways of protein quality control. These pathways function throughout the lifetime of a cell, but become even more important during times of thermal stress, when denatured proteins can accumulate and kill the cell.

Recall from Chapter 3 that a heat shock protein (Hsp) is a molecular chaperone, which uses the energy of ATP to catalyze protein folding after translation. Chaperones can also help refold proteins that have become denatured as a result of thermal stress. Many cells exposed to extreme temperatures undergo a *heat shock response,* which leads to a dramatic increase in the levels of specific proteins that help repair damaged proteins. During a heat shock, the cell undertakes a rapid increase in the synthesis of several critical heat shock proteins. The cell can halt the transcription and translation of other genes, sparing biosynthetic resources for Hsp synthesis. It stimulates the expression of the Hsp genes by activating a heat shock factor (HSF), a transcription factor that binds to the heat shock elements in the promoters of genes for heat shock proteins. Although there is still some uncertainty about the exact mechanism of activation of HSF, the trigger for the process is thought to involve damaged protein (Figure 15.19). In the absence of thermal stress, most of the cellular HSF is bound to Hsp70 as inactive monomers. When the cell is stressed, the chaperones are lured away from HSF by damaged proteins. The released HSF can then form trimers, which in turn bind the heat shock element on the Hsp genes, activating them. Once the damaged proteins are repaired, Hsp70 is free to bind HSF monomers and reverse the transcriptional activation.

The Hsp response is central to the ability of ectothermic animals to survive brief periods of extreme temperature that often occur within their natural environments. For most species, the Hsp response is induced at temperatures only a few degrees above the typical thermal range. This powerful protective process may be central to the evolution of thermal sensitivities and thermal ranges. If heat shock proteins are protective, why haven't animals evolved greater expression to expand thermal niches? Experiments in fruit flies (*Drosophila*) have shown that elevated Hsp expression comes at a cost. Flies that have evolved extra copies of Hsp70 genes survive better at high temperatures, but at lower temperatures they experience a decrease in fecundity.

Some species have lost their ability to mount a heat shock response. Antarctic fish have lived for thousands of years at 1.96°C. At some point, the species experienced

FIGURE 15.19 **Heat shock response**



1. Complex of HSF and Hsps under unstressed conditions.
2. Heat stress causes complex to dissociate.
3. Hsp70 binds to denatured proteins.
4. HSF monomers associate into trimers.
5. Trimers move into the nucleus and bind to the promoter of genes with heat shock element (HSE).
6. Hsp70 gene transcription increases.
7. Poly A^+ mRNA is exported to the cytoplasm.
8. Poly A^+ mRNA is translated to form more Hsp70.
9. The increase in Hsp70 levels allows the complex to form again, stopping transcriptional activation.

genetic changes that disrupted the capacity to invoke a heat shock response. Because the Antarctic waters remain very constant in temperature, these mutations have no deleterious consequences to the animals. However, when taken out of their natural environment, these fish rapidly succumb to temperatures only a few degrees above 0°C.

Ice nucleators control ice crystal growth in freeze-tolerant animals

Ectotherms that live at freezing temperatures use two strategies to survive the cold: freeze tolerance and freeze avoidance. Freeze-tolerant animals allow their tissues to freeze and even encourage ice to form in the body. Animals that avoid freezing use behavioral and physiological mechanisms to prevent ice crystal formation and growth. To understand why ice is so dangerous, let's consider what happens to water molecules as temperatures decrease.

The freezing point of pure water is 0°C. This is the temperature at which ice could form if enough water molecules cluster together to begin an ice crystal. Below the freezing point, water is on the verge of freezing, awaiting an event that triggers ice formation. When water is below its freezing point, but not yet frozen, it is considered **supercooled**. Pure water, left undisturbed, can be supercooled to almost −40°C before ice forms spontaneously. The trigger for ice formation is a cluster of water molecules that act as a seed for an ice crystal. Alternatively, a macromolecule in solution can act as a **nucleator**, seeding ice crystal formation. Once the ice formation begins, water molecules bind to each face of the growing crystal to create a complex three-dimensional structure.

Ice crystals forming within a tissue have two deleterious effects. First, because ice crystals have points and sharp edges, the growing ice crystal can pierce membranes, killing the cell. Second, ice crystal growth removes surrounding water, causing hyperosmotic stress. If ice forms outside cells, then water is drawn out of cells, causing a hypertonic stress that shrinks the cell, perhaps even killing it. Still, many ectotherms survive freezing (Figure 15.20). Insects such as the goldenrod gall fly (*Eurosta solidaginis*) overwinter in senescent galls on the stems of goldenrod. The stems reach above the snow, and expose the larva to temperatures as low as −55°C. Intertidal bivalves living in northern tidal flats can freeze when exposed to cold air temperatures, then thaw when the warmer water returns at high tide. Several terrestrial vertebrates can also survive freezing. A wood frog in the north temperate zone enters the leaf litter in late fall, in preparation for overwintering. When temperatures drop below freezing, the animal supercools but ice does not form. At still lower temperatures, the animal begins to freeze. First to freeze are the frog's digits. The body core begins to freeze shortly thereafter.

Freeze-tolerant animals usually produce ice nucleators to control the location and kinetics of ice crystal growth.

FIGURE 15.20 Freeze-tolerant animals

Eurosta in gall

Photo source: Valerie Giles/Science Source.

Ice is most damaging when it forms inside cells, so freeze-tolerant animals secrete nucleators out of the cell. This restricts ice formation to the extracellular fluids, such as hemolymph, and allows the intracellular space to remain liquid. Many different types of molecules can act as nucleators in animals: calcium salts, membrane phospholipids, and long-chain alcohols. However, it is not always clear that these ice nucleators are actually necessary or helpful to freeze-tolerance strategies. For example, the wood frog has an ice nucleator that triggers ice formation at about 7°C. The same ice nucleator is also found in the tissues of frogs that cannot survive freezing. It may induce the formation of ice, but it does not necessarily provide the wood frog with its freeze tolerance. Some nucleators may simply be present for other functions and have no adaptive role in freeze tolerance.

Because ice formation draws water from the cells, freeze-tolerant animals also produce intracellular solutes to counter the movement of water. Large glycogen reserves of the liver are broken down and converted to compatible solutes consisting of organic polyols, such as trehalose and glycerol. As we discussed in Chapter 13, compatible solutes have two main beneficial effects. First, by increasing the osmotic pressure within the cells, they reduce the movement of water and cell shrinkage. Second, the solutes help stabilize macromolecular structure.

Antifreeze proteins can prevent intracellular ice formation

Freeze avoidance is the second strategy animals use to survive extreme cold. In a car, antifreeze elevates the osmotic concentration of the radiator fluid. Solutes in general depress the freezing point of a solution, preventing ice formation at subzero temperatures. **Freezing point depression** is one of the colligative properties of solutes. The solutes in animal tissues reduce the freezing point of water, but generally not lower than about 2°C. Some animals possess antifreeze macromolecules—typically proteins or glycoproteins—that reduce the freezing point of body fluids by noncolligative actions. They disrupt ice crystal formation by binding to the surface of small ice crystals to prevent their growth (Figure 15.21).

The first **antifreeze protein**, or AFP, was discovered in an Antarctic fish in the 1970s by Dr. Art DeVries. Since then, AFPs have been found in many distantly related taxa of fish, as well as insects and plants. Four classes of AFPs are distinguished by their structure: types I, II, and III, as well as antifreeze glycoproteins, or AFGPs. Interestingly, each of

FIGURE 15.21 Antifreeze proteins

Antifreeze proteins bind to the surface of ice crystals to prevent their growth. They bind along the face of the ice crystal, where the protein forms weak bonds with water molecules immobilized in the ice crystal. Because ice growth is very orderly, the presence of the bound protein prevents ice crystal growth.

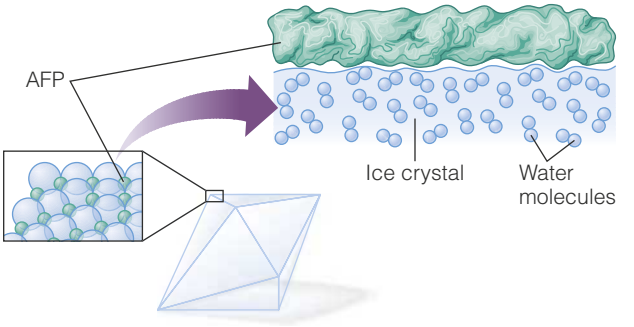


Figure source: Based on Davies, P. L., Baardsnes, J., Kuiper, M. J., & Walker, V. K. (2002). Figure 6 from Structure and function of antifreeze proteins. *Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences*, *357*, 927–935.

the classes of AFPs has arisen multiple times in evolution. In fish, AFPs arose less than 20 million years ago. This coincides with recent (in geological terms) sea level glaciation, which probably represented a strong selective pressure on the local marine species. The phylogenetic distribution of AFPs suggests an intriguing evolutionary history.

AFPs provide good examples of parallel evolution. For example, AFP II appears in herring, salmon, and sea ravens, fish from three separate orders. This suggests that AFPs arose multiple times in these lineages but well after the modern species diverged. These AFP II genes may have arisen from similar genes independently in each lineage. The structure of AFP II suggests that the ancestral gene was a Ca^{2+}-dependent lectin, a protein that binds sugars. In structural models, the interaction of a lectin with the hydroxyl groups of sugars is similar to the interaction of AFP with the hydroxyl group of a water molecule.

The evolutionary origins of AFGP are also unusual in terms of protein evolution. The ancestral gene was probably a gene for pancreatic trypsinogen, a digestive protease we introduced in Chapter 14. A region between the first intron and second exon was duplicated not just once but more than 40 times. The resulting gene possessed multiple, tandem sequences that resulted in a repeating Thr-Ala-Ala motif necessary to prevent ice crystal growth. In most cases of gene duplication and divergence, the resulting gene has properties similar to those of the ancestral gene, with relatively subtle differences in function. In the case of AFGP, the resultant gene has a totally distinct function. AFGPs have no protease activity, and trypsinogen has no antifreeze activity.

CONCEPT CHECK

10. Distinguish between freeze-tolerance and freeze-avoidance strategies.
11. Distinguish among nucleators, antifreeze proteins, and stress proteins.
12. What is meant by metabolic cold adaptation?

MAINTAINING A CONSTANT BODY TEMPERATURE

Endothermy is so inextricably intertwined with a high metabolic rate that it is not known which trait arose first. High T_B allows metabolic processes such as growth, development, digestion, and biosynthesis to operate at faster rates, and the higher metabolic rate in turn produces more heat. The ability to become warm bodied requires metabolic pathways to produce heat (**thermogenesis**) as well as physiological mechanisms to retain heat. Most endotherms are also homeotherms and committed to maintaining a constant T_B. To do so, they must control both thermogenesis and heat exchange. In cold environments, endotherms stimulate thermogenesis and reduce heat loss. In hot environments they increase heat loss, but may also reduce thermogenesis. To control T_B, animals must be able to sense both environmental temperature and body core temperature.

Endothermy, the ability to generate and maintain elevated body temperatures, has arisen several times in the evolutionary history of animals. It goes hand in hand with the capacity to produce heat through metabolism, and therefore activity levels. Most modern birds and mammals have high metabolic rates and are able to maintain their body temperatures well above ambient temperature, often within narrow thermal windows. While both are perceived as "higher vertebrates," birds and mammals arose from separate reptilian ancestors. Thus, endothermy arose independently at least twice. However, fossil evidence suggests that other extinct reptiles may also have been endotherms. The fossil record of the animals in the paleontological period from 200 to 65 million years ago is particularly clear, showing definitive examples of the transitions from reptiles to mammals and birds.

Recall from Chapter 2 that the first mammals appeared approximately 200 million years ago (mya), evolving from small, nocturnal reptiles that were only distantly related to the dinosaurs that would dominate Earth in later years. Fossils dating back to this period reveal the existence of several distinct mammalianlike reptilian lineages. These animals differed from other reptiles by the morphology of the skull and the organization of the teeth. Although most of these lineages disappeared, one group of reptiles called *cynodonts* gave rise to true mammals. The earliest mammals retained the reptilian trait of egg laying, like the modern monotremes, echidna and platypus. By the early Cretaceous period (144 mya), mammals had diversified into several lineages of marsupials and insectivores. When the dinosaurs disappeared about 65 mya, at the end of the Cretaceous period, there was an explosion of mammalian diversification. New species of mammals began to occupy the environmental niches vacated by the dinosaurs. It cannot be said for certain when endothermy arose in the transition from mammalianlike reptiles to true mammals. However, it is likely that the cynodont reptiles were already endothermic. Unlike most other reptiles of the day, cynodonts possessed a bony, secondary palate in the roof of the mouth that would have allowed them to breathe while chewing. This anatomical arrangement is a characteristic of endotherms because they must maintain uninterrupted respiration to sustain high metabolic rates. Cynodonts also appear to have possessed hair, which could have helped insulate their bodies.

Birds, the other group of modern endotherms, also arose from reptiles, although much later than mammals and from different reptilian ancestors. Around the time dinosaurs were declining, several reptilian lineages had already evolved featherlike body coverings. In one group, the theropod dinosaurs (see Figure 2.20) such as *Archaeopteryx*, the feathers were similar in structure to those of modern birds. Their feathers were asymmetrical, a trait that is necessary to be useful in feathered flight. In contrast, the other feathered reptiles of the era, such as *Protarchaeopteryx robusta* and *Caudipteryx zoui,* had symmetrical feathers (Figure 15.22). Because these symmetrical feathers would be useless in flight, they must have arisen in these dinosaurs for other benefits, likely as insulation. Although these other lineages of feathered reptiles became extinct, they were likely also endothermic animals.

Many researchers believe that endothermy arose in other, nonfeathered dinosaur lineages as well. The largest dinosaurs were simply too big to shed metabolic heat, and therefore remained warm-bodied. Many smaller dinosaurs may also have been endothermic. Multiple lines of evidence support the notion that these animals had the high metabolic rates necessary for an endothermic animal. Bone structure and posture suggest rapid rates of locomotion, which in modern animals require high metabolic rates that are possible only in warm-bodied animals. Just as in modern endotherms, many dinosaurs had relatively large brains associated with superior sensory processing. Because brain tissue has a high energy demand, a large brain can have an important influence on the whole-body metabolic rate. Other theories have been raised to support arguments that dinosaurs were endotherms. However, no argument is definitive because of the limitations in using the properties of modern animals as guidelines in predicting the physiological features of these long-extinct animals.

FIGURE 15.22 Asymmetrical fossilized feather

Photo source: O. Louis Mazzatenta/National Geographic/Getty Images.

Thermogenesis

Heat production is an inevitable consequence of being alive. An endotherm warms its body using heat that arises as a by-product of other metabolic processes, primarily energy metabolism, digestion, and muscle activity. All animals—endotherms and ectotherms—generate heat during these processes, but only the endotherms possess the physiological adaptations that enable them to retain enough metabolic heat to elevate T_B above T_A.

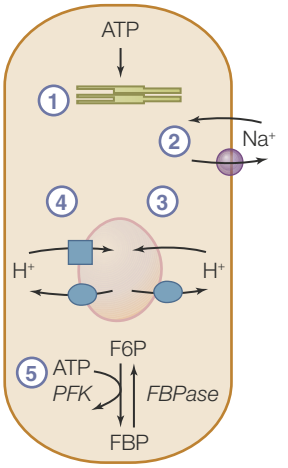
In addition to the pathways that produce heat as a by-product, endotherms possess specific thermogenic pathways with the main purpose of heat production. Thermogenic pathways rely on *futile cycling*, in which chemical potential energy is spent to generate heat. Most futile cycles involve cycling of ATP hydrolysis and ATP synthesis. Heat is released in ATP hydrolysis to ADP + phosphate, but a great deal more heat is produced when the cell uses intermediary metabolism to regenerate the ATP. Endotherms can enhance heat production either by increasing the rate of ATP turnover or by reducing the efficiency of ATP production. In both cases, most of the metabolic heat arises directly or indirectly from mitochondrial oxidative phosphorylation, discussed at length in Chapter 3. A summary of the main futile cycles we discuss in this section is presented in Figure 15.23.

Shivering thermogenesis results from unsynchronized muscle contractions

Muscle plays a critical role in the thermal budget of endotherms. Because muscle is the most abundant tissue in birds

FIGURE 15.23 Futile cycles and thermogenesis

(1) Myofibrillar ATPase, recruited during shivering thermogenesis.
(2) Plasma membrane ion (Na^+) leakage and pumping.
(3) Nonspecific mitochondrial proton leakage and pumping.
(4) Thermogenin-mediated proton leakage and pumping.
(5) Futile cycling in glycolysis.



and mammals, it produces considerable heat, even at rest. Locomotion enhances the rate of muscle heat production. However, many birds and mammals can also use skeletal muscle to generate heat by **shivering thermogenesis**.

The act of shivering is controlled by the motor neurons. Individual myofibers contract but the motor units are uncoordinated and the whole muscle undergoes no gross movement. During hypothermia, the shivering response is complex, manifesting a sustained low-intensity shivering with interspersed high-intensity bouts. The low-intensity shivering involves type I fibers, the same muscles that permit low-intensity sustained exercise. Like exercise, this activity is fueled by lipid oxidation. The high-intensity bouts are induced by activation of larger type II fibers, and fueled by glycogen, just as with burst exercise. The balance between low- and high-intensity shivering is regulated by the central nervous system, and is affected by the availability of fuel stores.

Shivering thermogenesis is a strategy that works for short periods of cold exposure, but it is not useful for prolonged cold stress. The mechanics of shivering prevent an animal from using its locomotor muscles to hunt prey or escape predators. Furthermore, if shivering persists, or repeats frequently, the muscles are rapidly depleted of nutrients and they become exhausted, just as they would after high-intensity exercise.

Some insects use metabolic futile cycles to warm flight muscle

Large flying insects, such as bumblebees and some moths, can generate enough heat to warm the thoracic flight muscles, which improves flight muscle performance in terms of energy production, excitation-contraction coupling, and cross-bridge cycling. The high metabolic rate during flight generates abundant heat, enough to warm the flight muscles by several degrees. Remarkably, these insects are even able to warm their flight musculature prior to takeoff.

Three distinct mechanisms allow insects to warm the thorax prior to flight. These same thermogenic pathways also allow social insects to work collectively to warm the hive. The first mechanism is a metabolic futile cycle in carbohydrate metabolism. Within the flight muscle, two opposing enzymes are activated simultaneously: the glycolytic enzyme phosphofructokinase and the gluconeogenic enzyme fructose-1,6-bisphosphatase. The metabolic cycle causes ATP hydrolysis and heat production, but without changes in the levels of the other substrates and products. A second warming mechanism relies on muscle contraction. Two sets of antagonistic flight muscles power wing movements during flight. Bumblebees can induce both sets of muscles to contract simultaneously prior to flight, so that energy is expended without productive movement. The third mechanism for heat generation is actual wing movement. The insect moves its wings fast enough to buzz, but controls the frequency and orientation of the wings to avoid generating lift. Collectively, these thermogenic pathways allow the flight muscle to warm up prior to takeoff. There appears to be a critical thoracic temperature that must be achieved before the insect will attempt to fly (Figure 15.24). At high T_A, less of a preflight warm-up is necessary to reach the threshold.

FIGURE 15.24 Thermogenesis in insect flight muscle

Many large flying insects can undertake a preflight warm-up, using metabolic futile cycles and muscle activity to elevate thoracic temperatures to a threshold temperature required for flight.

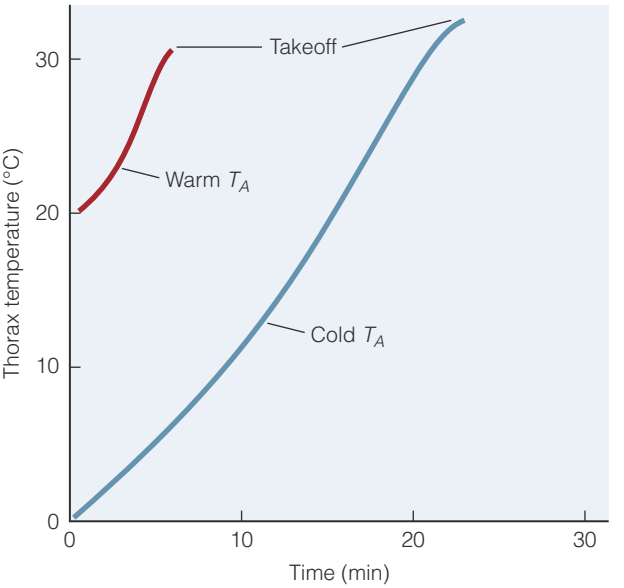


Figure source: Based on Heinrich, B. (1987). Thermoregulation in winter moths. *Scientific American, 256*, 104–111.

Membrane leakiness enhances thermogenesis

Most cellular membranes maintain an electrochemical gradient arising from differential distribution of ions across the membrane. Cells use chemical energy, usually in the form of ATP, to create these gradients. Consequently, any process that dissipates ion gradients will cause the cell to use chemical energy to reestablish the gradient.

Ion gradients collapse for two main reasons. First, many specific membrane proteins use electrochemical energy to drive other processes such as metabolite transport and biosynthesis. For example, many cells transport glucose and amino acids into the cell using Na^+-dependent cotransporters, causing the cell to use Na^+/K^+ ATPase to pump the Na^+ back out of the cell. The mitochondrial F_1F_o ATPase is another transporter that dissipates ion gradients, in this case

the proton motive force. Heat is produced when the mitochondrial electron transport system oxidizes reducing equivalents to regenerate the proton gradient.

The second pathway of ion gradient dissipation is ion leak, in which ion movements are not coupled to any other transport process. Because no biological membrane is completely impermeable, some ions leak across the bilayer or through gaps between proteins and phospholipids. Ion-pumping membrane proteins produce heat as a by-product, and a high proportion of the resting heat production, as much as 50 percent in some tissues, is due to the costs of maintaining ion gradients. Any process that increases the need for ion pumping will also increase thermogenesis. Typically, an endotherm has a resting metabolic rate that is as much as tenfold greater than that of an ectotherm of the same size and T_B. The higher metabolic rate is due in part to membrane leakiness; endotherm plasma membranes and mitochondrial membranes are inherently leakier than those of ectotherms. Endotherms generate more heat to maintain ion gradients across leakier membranes.

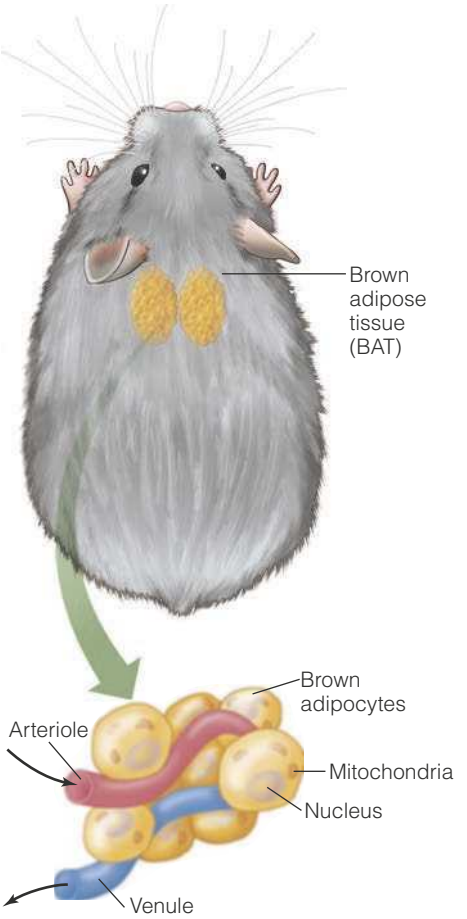
Thermogenin enhances mitochondrial proton leak

Mammals possess a unique way of generating heat in specialized deposits of **brown adipose tissue** (BAT), typically located near the back and shoulder region (Figure 15.25). BAT is most important for thermogenesis in small mammals and newborns of larger animals, particularly those that live in cold environments. Its growth and thermogenesis is under the control of the sympathetic nervous system. Norepinephrine released from these nerves causes BAT to grow in cell number (**hyperplasia**) and cell size (**hypertrophy**). Undifferentiated precursor cells are induced to proliferate and then later differentiate into BAT. Triglyceride is synthesized and mitochondria proliferate. BAT heat production is often called **nonshivering thermogenesis (NST)**; while the other pathways we have discussed also differ from shivering, NST is a term usually reserved for BAT-mediated thermogenesis.

The feature that makes BAT unique is the expression of a protein called **thermogenin**. When inserted into the inner mitochondrial membrane, this protein stimulates the rate of mitochondrial respiration and consequently heat production. It works by *uncoupling* mitochondria, dissipating the proton gradient. The oxidation of fuels continues in the electron transport chain, but the protons pumped out of the mitochondria are allowed to return to the mitochondria. Without a proton motive force, the mitochondria cannot produce ATP. However, the reduction of the proton gradient permits respiration to continue at high rates.

Thermogenin was first characterized in the early 1980s and much has been learned about the evolutionary origins and physiological functions of uncoupling proteins in vertebrates (see Box 15.3: Challenges to Homeostasis: Evolution and Development of Thermogenin). However, the molecular mechanism by which it induces uncoupling is still not certain. One theory suggests that thermogenin acts as a proton **ionophore**. It picks up protons from the cytoplasm and carries them into the mitochondria, dissipating the proton gradient. An alternative theory suggests that thermogenin dissipates the proton gradient by causing the futile cycling of fatty acids. Thermogenin carries an ionized fatty acid ($R\text{-}COO^-$) from the mitochondrial side of the inner membrane and flips it across the bilayer to face the cytoplasm. Because of the higher proton concentration (lower pH), the ionized fatty acid is rapidly protonated (R-COOH). In this neutral form it readily flops back into the inner leaflet of the bilayer, where it ionizes again. The complete "flip-flop" cycle causes a proton to be translocated across the inner mitochondrial membrane. Regardless of the molecular mechanism, thermogenin has the dual actions of dissipating the proton gradient and stimulating respiration, thereby generating heat.

FIGURE 15.25 Brown adipose tissue in hamsters
Hamsters possess thick pads of BAT behind the shoulders.



EVOLUTION AND DEVELOPMENT OF THERMOGENIN

The story of thermogenin is intriguing because it has a very specific function restricted to select mammals, yet has a deep evolutionary history extending to the origins of vertebrates.

Thermogenin was once thought to be a mammalian evolutionary invention, inextricably linked to the appearance of brown adipose tissue (BAT). It is now known to be one member of the **uncoupling protein** (UCP) gene family. In addition to thermogenin (also called UCP1), mammals express at least two other UCPs. These proteins, UCP2 and UCP3, can increase mitochondrial proton leak, but not enough to cause significant uncoupling or contribute to heat production. Instead, these proteins appear to reduce oxidative stress by preventing production of superoxide anions by mitochondria.

The UCP gene family is ancient, with members in ectothermic animals, such as fish, as well as plants, fungi, and protists. Thermogenin itself has homologs throughout vertebrates, but it is difficult to imagine circumstances that would have led the ectotherm UCP1 homologs to evolve thermogenic functions. Its role in thermogenesis appears to be limited to eutherian mammals.

The picture that has emerged in recent years suggests that the ancestral function of UCP1 was likely similar to that of other UCP paralogs, UCP2 and UCP3. High rates of proton conductance by UCP1 likely arose in mammals. Remarkably, the UCP1 gene appears to have undergone loss-of-function mutations in select mammals, such as the pig. BAT is most important in small animals with unfavorable surface area-to-volume ratios. It has been suggested that large-bodied mammals have little need for nonshivering thermogenesis (NST) and that loss-of-function mutations could be tolerated without negative consequences. Though its role may differ among vertebrates, there is a growing appreciation for the role that it plays in metabolic and thermal homeostasis of humans.

Small mammals, with an unfavorable surface area-to-volume ratio, have the greatest use for BAT. Not surprisingly, BAT is common in small mammals, such as rodents, typically found between the shoulder blades (interscapular). The same type of interscapular BAT is found in infant humans, though this deposit disappears with age. Recently, humans and mice have been shown to possess another type of thermogenic adipose tissue that is intermediate between brown and white, called brite (from *brown/white*) or beige adipose tissue. The brite adipose tissue deposits, identified by high-resolution imaging methods, are found on top of the collarbone (supraclavicular). The differences in the appearance of brown and brite AT reflect different cellular origins. Brown adipocytes arise from the same precursor cells that differentiate to produce skeletal muscle, whereas brite adipocytes are derived from vascular cells within the WAT. Both brown and brite adipocytes are mitochondria-rich cells, but only brown adipocytes express high levels of UCP1 under normal conditions. However, when brite adipocytes are stimulated by hormones that elevate cAMP, they increase the expression of UCP1 and stimulate mitochondrial respiration and heat production. A similar response occurs when mice are cold exposed (Figure 15.26). Within hours of cold exposure, the mRNA for UCP1 increases manyfold. However, it takes many days before a corresponding change in UCP1 protein is seen. This delay is due in part to the different kinetics of mRNA and protein synthesis.

FIGURE 15.26 Changes in UCP1 mRNA and protein with cold exposure in mice

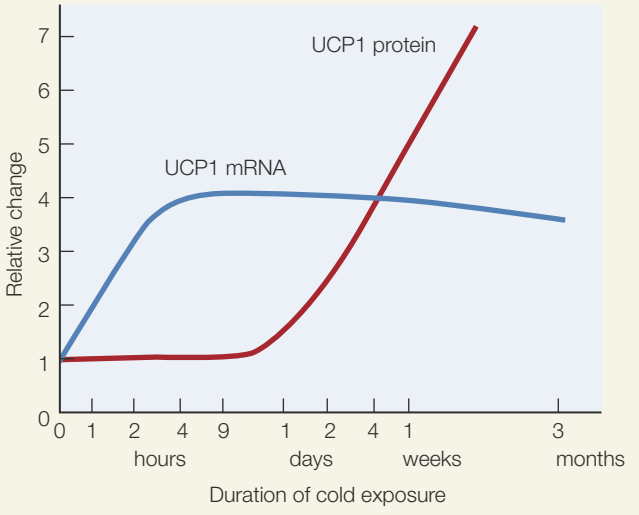


Figure source: Based on Nedergaard, J., & Cannon, B. (2013). UCP1 mRNA does not produce heat. *Biochimica et Biophysica Acta Molecular and Cell Biology of Lipids, 1831*, 943–949.

Given the role in energy dissipation and heat production, researchers explored the relationship between BAT activity and metabolic diseases, such as obesity and type II diabetes mellitus. In mice, the overexpression of the transcription factor PRDM16, known to increase production of BAT, is protective against diet-induced metabolic dysfunction, including obesity. In humans, the possible therapeutic approaches are being explored, and it is advantageous that there are at least two different types of thermogenic adipose tissue: brown and brite. Their different developmental origins may impart different sensitivities to pharmacological interventions intended to reduce obesity and related metabolic disorders.

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CONCEPT CHECK

13. What is shivering thermogenesis?
14. What are the futile cycles used by various animals for heat production?
15. What animals have thermogenin and what animals have brown adipose tissue?

Regulating Body Temperature

Control of body temperature in endothermic animals requires coordination of multiple physiological systems. Animals must be able to monitor T_B in critical anatomical regions. By monitoring internal core T_B, animals can assess their overall thermal balance. Peripheral thermoreceptors allow animals to detect T_A. The information from thermal sensing neurons is received and interpreted by a thermostat within the central nervous system. The central thermostat triggers the appropriate behavioral and physiological response.

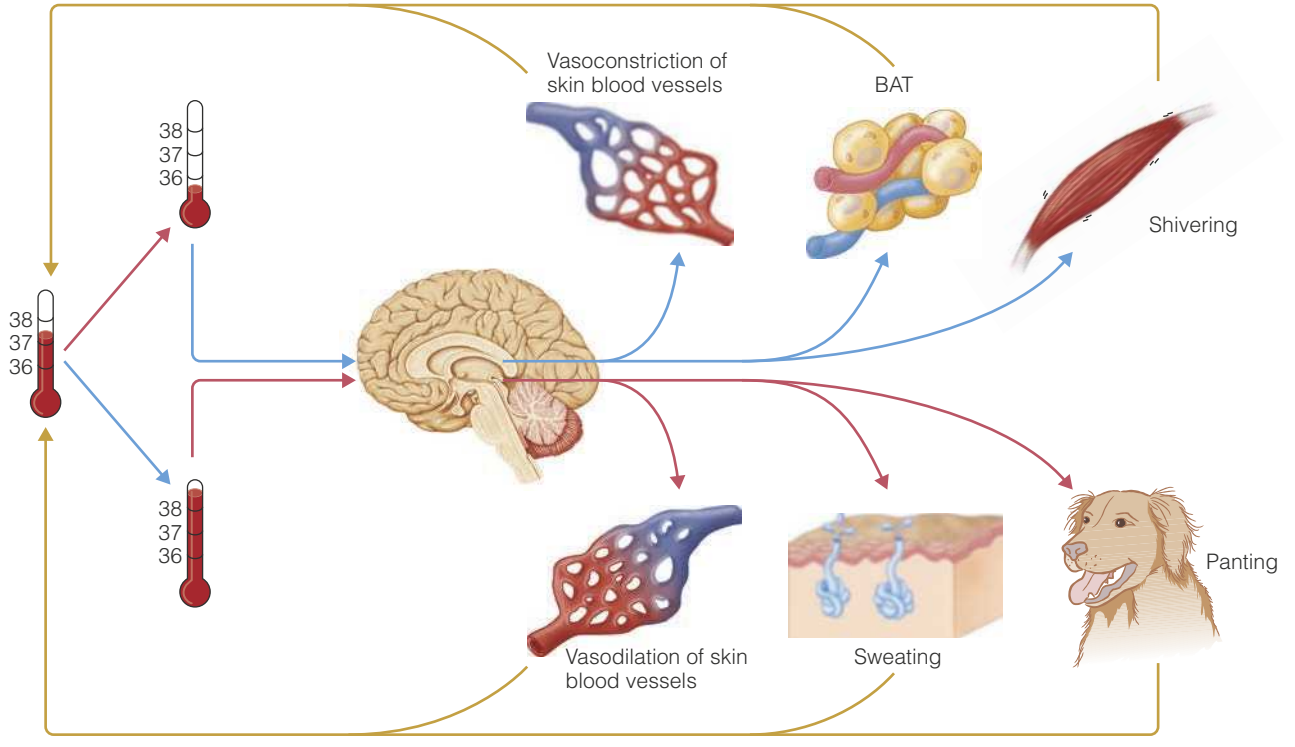
A central thermostat integrates central and peripheral thermosensory information

As we discussed in Chapter 7: Sensory Systems, animals possess different types of neurons to sense and respond to temperature. Temperatures are monitored peripherally and centrally by temperature-sensitive neurons, both cold sensing and warm sensing. Birds and mammals monitor temperature using similar neurons, although the location of the central thermostat differs in the two taxa.

Mammals monitor T_A by peripheral cold-sensitive neurons located in the skin and the viscera. When T_A decreases, peripheral neurons send signals to the hypothalamus (Figure 15.27). The preoptic area of the anterior hypothalamus has both cold-sensing and warm-sensing neurons that monitor core body temperature. Information from the peripheral and the central thermal sensors is integrated in the posterior hypothalamus, which sends signals to the body to alter the rates of heat production and dissipation. The hypothalamus is much more responsive to information from the central thermoreceptors than from the peripheral thermoreceptors. Changes of less than 1°C can excite central thermoreceptors, triggering a rapid hypothalamic response. Conversely, peripheral thermoreceptors may record and respond to a change of several degrees without invoking a hypothalamic response. Surface temperatures can change by several degrees without harming the animal, whereas the temperature of the central nervous system must be more stable.

FIGURE 15.27 Hypothalamus and thermoregulation

The hypothalamus is the thermal control center of mammals. It interprets signals from peripheral and central thermosensitive neurons and sends neuronal signals to other tissues, altering heat flux.



Bird T_B regulation is less understood but is clearly different from that of mammals. Heating or cooling the hypothalamus has little effect on the thermoregulatory response of birds. The central thermostat in birds appears to be the spinal cord, not the hypothalamus. However, the thermostat is still responsible for integrating information from central and peripheral thermosensors. When the central thermostat detects changes in temperature, it responds by firing neurons that lead to a compensatory response. Both birds and mammals alter T_B by changing rates of heat production and heat dissipation.

Piloerection reduces heat losses

Earlier in this chapter we discussed how body coverings, such as hair and feathers, act as insulation for endotherms. Because the efficiency of the insulatory layer depends on its thickness, animals can regulate heat loss by changing the orientation of the hair (in mammals) or feathers (in birds). Birds (and mammals) get fluffier in the cold by forcing their feathers (and hair) to orient perpendicular to the body surface. The mechanism by which this orientation is controlled is best understood with mammalian hair, but the position of bird feathers is controlled in a similar way.

Hair itself is a collection of cells that possess abundant keratin, an intermediate filament of the cytoskeleton. The distal end of a hair is primarily dead tissue, but the proximal end is composed of living cells embedded within the hair follicle. Depending on the species, a hair follicle can produce either a single hair shaft or complex combinations of hairs of various lengths and structures. Whereas human hair follicles produce single hairs, dog hair follicles produce a primary guard hair and multiple secondary hairs—soft, fine hairs that form the undercoat of the fur (Figure 15.28). The pit of the hair follicle is composed of epidermal cells. Intimately associated with each hair follicle is a sebaceous gland, which releases complex secretions of lipid (squalene, wax esters, triglyceride, and fatty acids) that form a protective coating on the hair and provide moisturization.

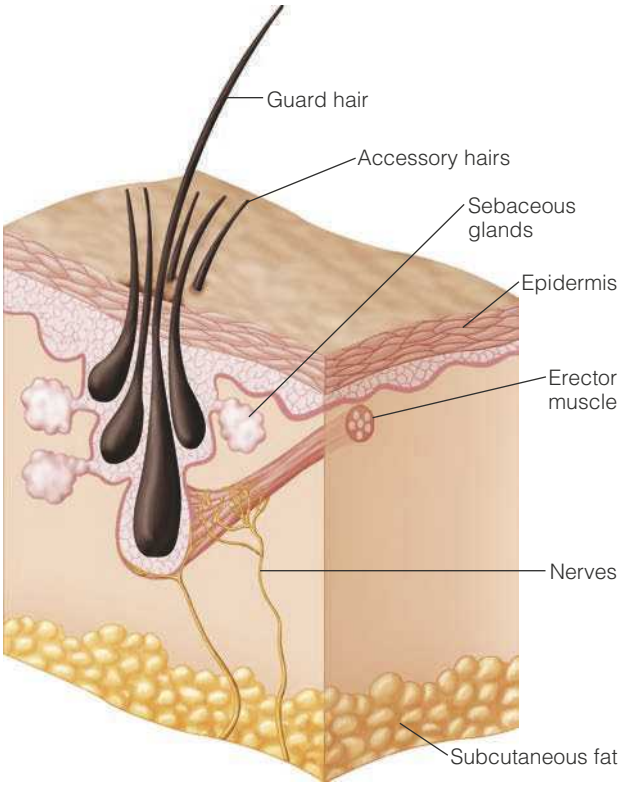
Tiny smooth muscles, called erector muscles, connect each hair follicle to the undersurface of the epidermis. When the erector muscle contracts, the hair is pulled perpendicular, a process termed **piloerection**, so that the fur offers better insulation. The erector muscle contractility is regulated by numerous factors, both blood-borne and neural in origin. The situation is similar in birds, where erector muscles also control the orientation of the feathers.

Changes in blood flow affect thermal exchange

All animals exchange heat at the external surfaces of the body, but they are able to alter the *effective ess* of surface heat exchange by changing the pattern of blood flow. Internal heat is equilibrated throughout the body by the blood. Where blood vessels approach the body surface, they will more readily lose heat. Similarly, increasing the flow of blood through the vessels increases the capacity for heat loss because it warms the surface of the skin, the site of heat loss by conduction, convection, and radiation.

FIGURE 15.28 Hair follicles of a dog

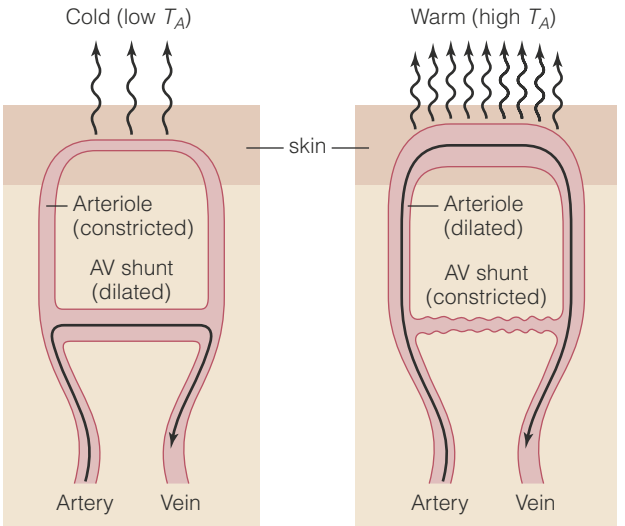
A hair is produced by cells in the hair follicle. Erector muscles attached to the base of the hair contract in response to neural stimulation, causing the hair to become upright. Sebaceous glands secrete lipids into the follicle ducts.



The regulation of the amount of blood flowing into the vasculature is known as the **vasomotor response** (Figure 15.29). Directly under the skin are capillary beds fed by subcutaneous arteries and drained by veins that empty into a network called the *ve ous plexus.* There is also direct exchange of some blood between the veins and arteries through connections called *arteriovenous anastomoses*, or metarterioles. At normal T_B, the sympathetic nervous system constricts the arterioles to reduce blood flow. This tonic constriction is mediated by vascular smooth muscle in response to adrenergic signals. When body temperature rises, there is a loss of tonic constriction and arterioles dilate to allow more blood into the skin vasculature. At the same time, the blood vessels of the anastomoses constrict, forcing more blood to move through the vessels near the skin. The large volume

FIGURE 15.29 Skin vasculature

When blood travels close to the surface of the animal, heat is lost across the skin. When temperatures are cold (left), blood is diverted from the skin through arteriovenous (AV) shunts, called arteriovenous anastomoses, reducing heat loss. When an animal is in a hot environment, shunts are constricted and blood moves through the vessels closer to the skin surface, enhancing heat loss.



and high compliance of the venous system allows the blood to readily exchange heat to the skin surface. The greater the temperature of the skin, the greater the rate of heat loss. The changes in vascular smooth muscle tone are controlled by the posterior hypothalamus.

Changes in blood flow through these capillary beds allow an endotherm to control heat exchange. The effects are perhaps most obvious in Caucasian humans, whose rapid changes in skin color reflect subdermal blood flow. Exercise increases the core body temperature and triggers an increase in blood flow to the skin, causing it to turn red. Similarly, cold temperatures cause peripheral vasoconstriction, reducing blood flow to the hands and feet, causing them to turn white. Prolonged restriction of blood flow can cause the extremities to turn purple, as the blood pooled in the venous system is slowly deoxygenated.

Countercurrent exchangers in the vasculature help retain heat

In addition to restricting blood flow to the periphery, some animals are able to extract heat from warmed blood and transfer it to cooler blood. This is accomplished by arranging the vasculature into *countercurrent heat exchangers*. The exact arrangement depends upon the animal and the tissue.

Because fish breathe water, any metabolic heat is rapidly lost across the gills. Some regionally heterothermic fish, discussed earlier in this chapter, are active swimmers that produce abundant heat in their red muscle. In tuna, veins leaving the red muscle are juxtaposed to the arteries that supply the red muscle, allowing the transfer of myogenic heat from the veins back to the arteries (Figure 15.30). This allows red muscle to reach temperatures more than

FIGURE 15.30 Countercurrent heat exchangers in tuna muscle

Each heterothermic scombrid species relies on different combinations and numbers of retes to retain heat. Red muscle of bluefin tuna is served by cutaneous arteries and veins that run beneath the skin. From these main vessels, smaller lateral vessels run over the surface of the red muscle, with branches penetrating the muscle. These lateral vessels are arranged in a countercurrent manner, with lateral venules transferring myogenic heat to lateral arterioles.

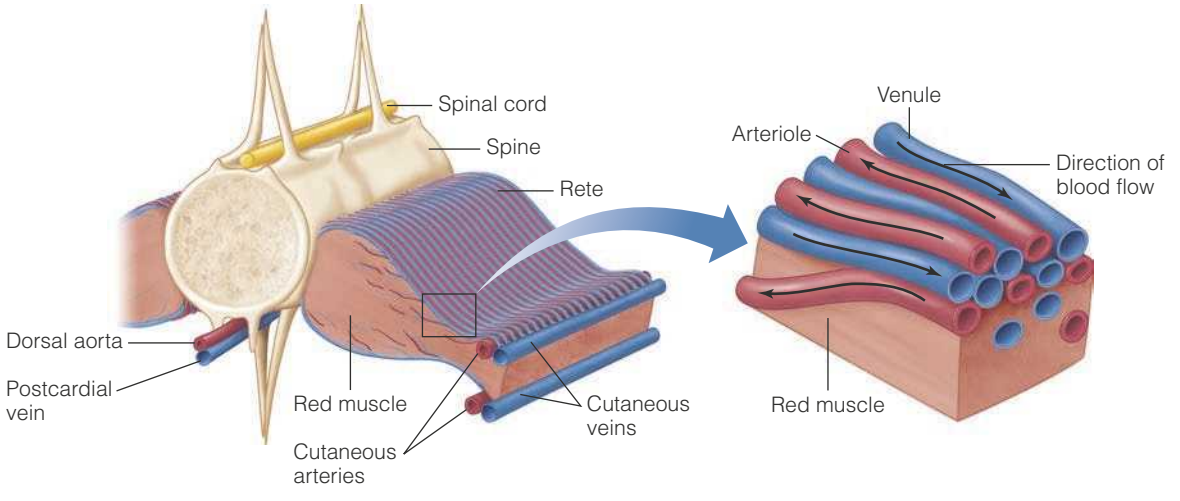


Figure source: Based on Carey, F. G. (1973). Fishes with warm bodies. *Scientific American*, *228*, 36–44.

10°C warmer than other tissues, including **white muscle**. Countercurrent heat exchangers are important in other regionally heterothermic fish. As we discussed in Chapter 6, billfish possess a modified eye muscle, called a heater organ, that warms the eye and optical nerves. Countercurrent heat exchangers help retain heat in the optical system. Many large fish, such as bluefin tuna, use countercurrent heat exchangers in the gastrointestinal tract to retain the heat of digestion.

Countercurrent heat exchangers are used by endotherms to reduce heat loss at the periphery. Birds standing on cold surfaces, such as ice, can lose a great deal of heat through the feet (Figure 15.31). They can reduce heat loss by restricting blood flow to the periphery, but over long periods this would cause the peripheral tissues to starve. Countercurrent heat exchangers transfer heat from arteries emerging from the body core to veins returning from the cold periphery. Warming of the venous blood lessens the impact of the peripheral cooling. Also, cooling the arterial blood decreases the thermal gradient across the skin and therefore reduces heat loss.

Sweating reduces body temperature by evaporative cooling

One mode of shedding excess heat is evaporative cooling. In mammals, many species sweat, releasing a mixture of water, salts, and some oils. The salt in sweat raises the boiling point of water, making evaporative cooling more efficient. Loss of water and salts can affect ion and osmoregulation, but animals exposed to hot weather for long periods can change the chemical composition of their sweat to minimize ionic and osmotic problems. They produce a larger volume of sweat with a lower NaCl content, preserving vital salts. Sweating is controlled by the anterior hypothalamus and triggered by activation of the sympathetic nerves that control the activity of sweat glands.

The evolution of sweat as an important route of heat loss was influenced by many factors. Small animals have a favorable ratio of surface area to volume for heat loss, so evaporative cooling is used primarily by larger mammals. In species with fur, sweat glands are present but sweating as a means of thermoregulation is less effective because the fluid simply mats the fur. In primates in general, and humans specifically, the increase in the importance of sweat glands for thermoregulation coincided with the evolution of a hairless skin and large body size.

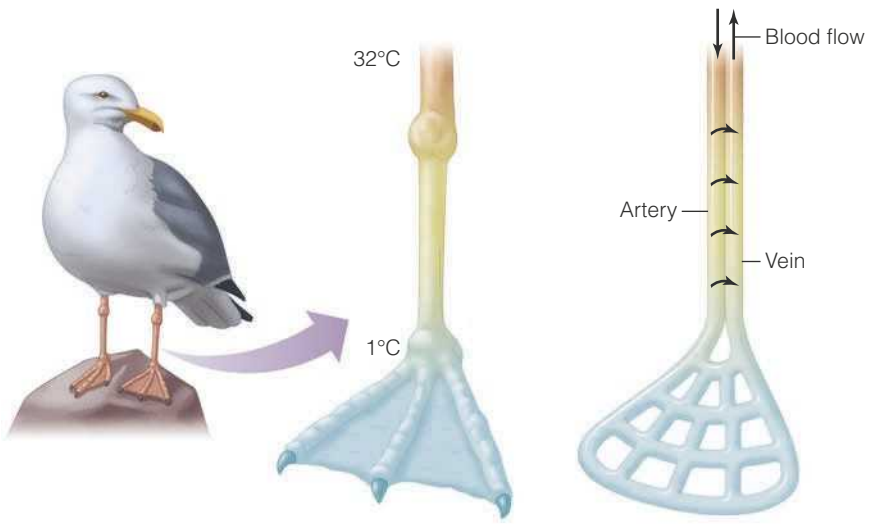
Panting increases heat loss across the respiratory surface

Another way animals lose heat is through ventilation. The properties that make a respiratory surface good at gas exchange—high vascularity, moist surfaces, and high airflow—also enhance heat loss. Whether respiratory heat loss is beneficial or detrimental depends on the situation. In the cold, birds and mammals minimize heat loss from respiration, but at high T_A, animals may alter their breathing pattern to accentuate heat loss.

Cooling through ventilation is a strategy that must balance respiratory demands with thermoregulation. Cooling is enhanced when animals increase ventilation frequency while reducing tidal volume. Shallow, rapid breathing is a sign that an animal may be overheated. Gular fluttering is a cooling behavior seen in birds, characterized by rapid contraction and relaxation of the throat muscles. Mammals pant. Each of these behaviors cools the animal in multiple ways. First, rapid ventilation increases the heat loss across the respiratory surface by convection. Second, and perhaps more important, the rapid ventilation causes water to evaporate from the surface of the airway, from the pulmonary surface to the tongue. Animals that rely on ventilatory cooling often possess well-vascularized respiratory surfaces that are kept wet through secretions. These ventilatory

FIGURE 15.31 Peripheral vasoconstriction in cold endotherms

Birds standing on cold surfaces can alter the flow of blood into the feet, reducing heat loss. The blood vessels of the leg and foot are arranged in parallel, allowing the formation of a countercurrent heat exchanger.



patterns could alter the nature of the blood gas profile, impinging on respiratory physiology. The increase in ventilation frequency is offset in part by a reduction in tidal volume.

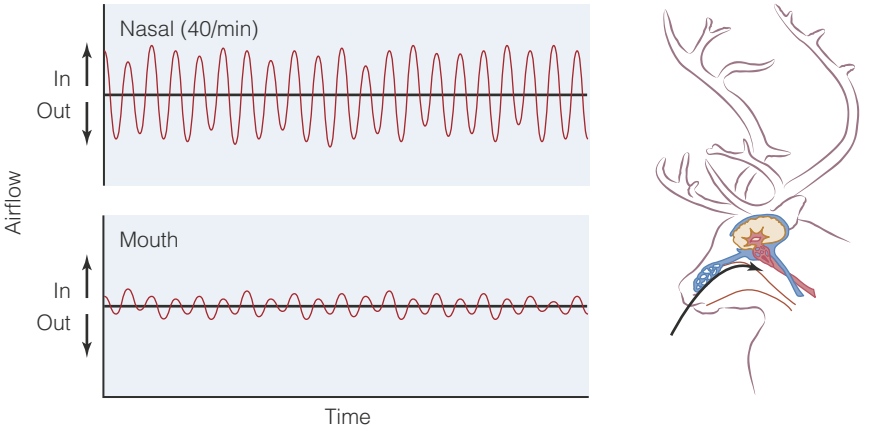
Reindeer provide a good example of the links between respiration and thermoregulation. Although they live in the cold, reindeer are at risk of heat stress because of their large size and thick layer of fur insulation. At normal cold temperatures (10°C), a reindeer breathes through its nose at low frequency. The upper part of the nasal cavity is rich in capillaries, and nasal respiration helps cool the nearby brain regions. When a reindeer becomes too warm, it shifts its respiratory pattern. Breathing frequency increases, and the animal begins to pant through the mouth (Figure 15.32). Although this change in breathing pattern may reduce direct cooling of the brain, it reduces body core temperature more efficiently.

Relaxed endothermy results in hypometabolic states

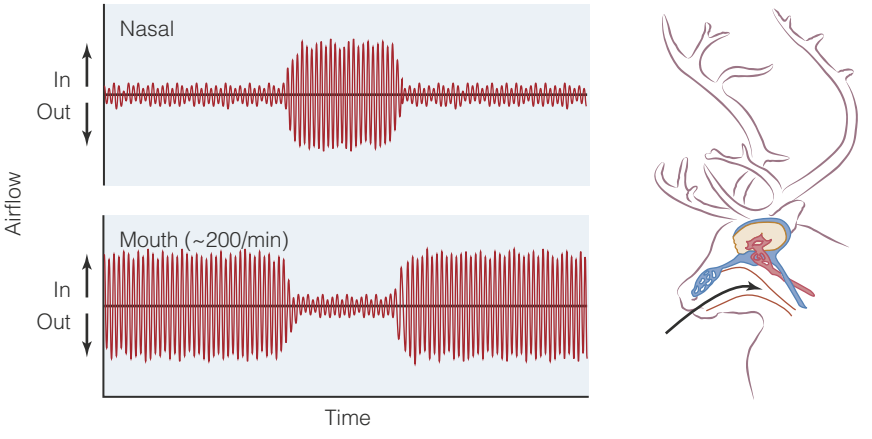
In previous chapters, we have encountered how endotherms use various forms of hypometabolism to survive adverse conditions. Hummingbirds, for example, undergo a nightly

FIGURE 15.32 Heat loss during panting

Like other mammals, reindeer alter breathing to increase heat loss. Reindeer breathe through the nose at low temperatures. The flow of air cools the blood circulating through the vessels that line the nasal cavity. When temperatures increase, reindeer breathe through the mouth and at a faster rate (200–300 breaths per minute).



(a) Low temperature

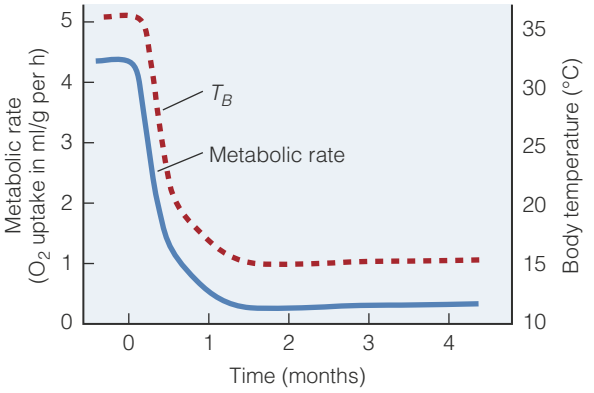


(b) High temperature

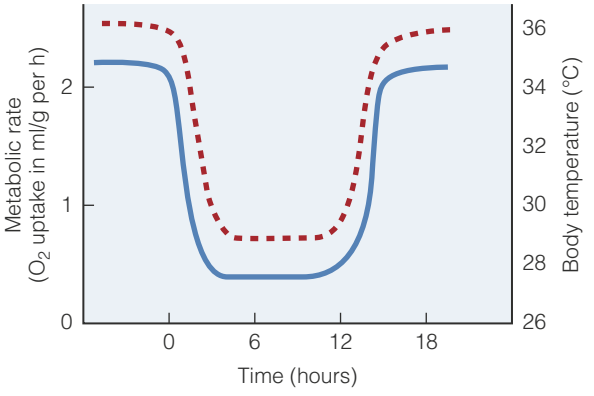
Figure source: Republished with permission of American Physiological Society, from Panel (b) graph: Adapted from Aas-Hansen, O., Folkow, L. P., & Blix, A. S. (2000). Figure 1 from Panting in reindeer (*Rangifer tarandus*). *American Journal of Physiology: Regulatory, Integrative, and Comparative Physiology*, *279*: R1190–R1195; © 2000. Permission conveyed through Copyright Clearance Center, Inc.

FIGURE 15.33 Hypometabolic states

Many endotherms respond to cold temperatures by entering some form of dormancy. Body temperature generally declines in parallel with metabolic rate. The dormancy is called **(a)** hibernation when the metabolic depression lasts for weeks to months or **(b)** torpor when the animal enters a hypometabolic state in daily cycles.



(a) Hibernation



(b) Torpor

reduction in metabolic rates. Hibernating mammals also undergo metabolic suppression during the long, cold winter months when food is scarce. Whether a daily dormancy (torpor) or a more prolonged seasonal dormancy (hibernation), the hypometabolic phase is accompanied by a decrease in T_B, a phenomenon called **relaxed endothermy**. The time course and magnitude of reduction in T_B differ among animals and types of dormancy (Figure 15.33). An Arctic squirrel, for example, can allow T_B to fall close to the freezing point. However, even minor reductions in T_B can offer important energetic savings for a dormant animal.

Under normal (euthermic) conditions, mammals and birds maintain T_B within a narrow range. A euthermic animal induces a compensatory response when its central thermostat—the hypothalamus in mammals—senses a decrease in T_B. During periods of relaxed endothermy, the animal recalibrates its central thermostat to recognize and defend a different T_B set point. The endothermic animal may allow T_B to fall close to T_A, well below the euthermic set point. In many species, long periods of dormancy are interrupted by brief periods of arousal. In these episodes, lasting minutes to hours, T_B rises and the animal elevates heart rates and respiration rates, before returning to the hypometabolic condition.

The links between metabolism and T_B regulation make it difficult to establish which parameter causes hypometabolic cooling. For most animals entering dormancy, T_B and metabolic rate decline in parallel, and it is not clear if the colder T_B slows metabolism, or alternately if the slower metabolic heat production causes cooling. In some studies, animals show a reduction in metabolic rate before T_B declines, suggesting that hypometabolism initiates the reduction in T_B. However, in larger animals a delay in cooling upon entering dormancy is due in part to thermal inertia; the large mass and low ratio of surface area to volume delay the impact of reduced thermogenesis, allowing the animal to remain much warmer than T_A even with a reduced metabolic rate.

CONCEPT CHECK

16. What regions of the body detect and respond to changes in temperature in mammals?
17. What are the various types of hypometabolism?
18. How do animals control heat flux across the external body surface?

SUMMARY

T_B of an organism depends on the environment (e.g., radiation), anatomy (e.g., shape, insulation), metabolic rate, and heat exchange (e.g., thermal conductance, fluid movement). Thermal strategies are categorized based on the source of heat and the degree of constancy, both temporally and spatially.

Thermal tolerance is influenced by many anatomical and physiological factors, and modified by thermal history. Changes in T_A have greater consequences for ectotherms than for endotherms, altering many aspects of macromolecular structure and metabolism. Temperature alters membrane fluidity and protein structure and function but animals have cellular pathways for minimizing perturbations and mitigating damage.

Poikilotherms that have lived for long periods in extreme cold often possess cold-adapted proteins. Some animals are able to survive freezing by ensuring that it happens in a controlled manner using ice nucleators. Others avoid freezing through antifreeze proteins.

Endothermic animals produce metabolic heat and retain it to elevate T_B above T_A. Their metabolic reactions produce more heat than in ectotherms and they retain more of it internally. Thermal balance depends on neural systems to detect external and internal temperatures, and a central thermostat, such as the mammalian hypothalamus, to integrate central and peripheral thermal sensory information, and adjust physiological systems to alter heat production and retention. Endotherms may combine a reduction in metabolic rate with decreased T_B to reduce metabolic demands.

REVIEW QUESTIONS

1. LO1 Water at 10°C feels colder than air at 10°C. Why?
2. LO1 What behaviors reduce heat losses due to (a) conduction; (b) convection?
3. LO2 Compare and contrast the following terms: homeothermy, poikilothermy, endothermy, and ectothermy.
4. LO2 Use examples to distinguish between regional heterothermy and temporal heterothermy.
5. LO3 Why does aerobic scope decline as temperature rises?
6. LO3 Discuss the different sources of energy an ectotherm can use to raise T_B.
7. LO4 Why are antifreeze proteins found in marine fish but not freshwater fish?
8. LO4 Why does a higher temperature generally increase enzymatic rates?
9. LO5 How do we know that antifreeze proteins arose several times in evolution?
10. LO5 How can cells alter the fluidity of cellular membranes?
11. LO6 Compare and contrast the mechanisms of shivering and nonshivering thermogenesis.
12. LO6 Which biochemical steps are responsible for heat production?
13. LO7 How do countercurrent heat exchangers work?
14. LO7 Discuss the mechanisms that permit an increase in T_A to trigger sweating.

SYNTHESIS QUESTIONS

1. Compare the effects of high and low temperature on molecules, cells, tissues, and organisms.
2. How could you convert a stenothermal animal to a eurythermal animal?
3. Summarize the physiological changes that accompany thermal acclimation.
4. Why do endothermic animals need both peripheral and central temperature-sensitive neurons?
5. Thermoregulation requires active control of blood flow through vessels. How do animals dilate some blood vessels while constricting others?
6. What would you expect to happen to blood pressure when a mammal is exposed to cold temperatures?
7. What gene regulatory changes must have accompanied the evolution of brown adipose tissue?
8. Animal color influences many aspects of physiology and ecology. Identify some examples of animals whose color patterns are consistent with a role in thermoregulation.
9. Many mammals grow coats that differ in winter and summer. What factors affect the costs and benefits of seasonal shedding?
10. Compare and contrast the structures of hair and feathers.

QUANTITATIVE QUESTIONS

1. The metabolic rate of a fish heart is studied at various temperatures. The metabolic rate is 20 mol ATP per min per g tissue at 25°C, 8 mol ATP per min per g tissue at 10°C, 4 mol ATP per min per g tissue at 5°C, and 1 mol ATP per min per g tissue at 2°C. Calculate the Q_{10} values over this range of temperatures and offer an explanation for the patterns.
2. The levels of ATP are maintained through a balance between the rates of ATP synthesis and ATP utilization. For a given tissue (e.g., heart) at a given T_B (e.g., 15°C), assume that (a) the rates of ATP synthesis and utilization are both 10 mol/min/g, (b) the rate of ATP synthesis exhibits a $Q_{10} = 2$, (c) the rate of ATP utilization has a $Q_{10} = 2.05$, and (d) the starting ATP level was 5 mol/g tissue. Calculate the change in ATP levels over time that would result if the animal were moved to an environment that caused a 10°C increase in T_B.
3. Recall the Stefan-Boltzmann equation, $P = Ae\delta(T_B^4\ T_A^4)$, where P is the radiating power, A is its surface area, e is the ability of the object to emit radiation, δ is the Stefan constant, and T is the temperature of the body (T_B) or surroundings (T_A) in kelvins. Consider an animal that uses a strategy of changing posture to alter the surface area as a way of controlling heat loss. It assumes a particular posture when it is in an environment that is 5°C below its body temperature. How does it need to change its surface area when it moves to a new environment that is 20°C cooler?