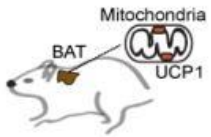
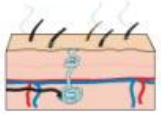
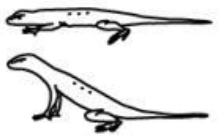
Thermoregulation- Contd.

Lecture 7

			Cold-defense	Heat-defense
Physiological	Non-shivering (BAT) thermogenesis		Activated	Inhibited
	Skin blood flow		Vasoconstriction	Vasodilation
	Water evaporation		Inhibited	Sweating Panting Saliva spreading
	Shivering thermogenesis		Activated	Inhibited
Behaviorial	Postural changes		Reduce exposed surface	Increased exposed surface
	Temperature choice		Heat-seeking	Cool-seeking
	Altering microenvironment		Nesting Social huddling	Air conditioning

A. Thermogenesis

Endotherms have various ways of increasing metabolic heat production, or **thermogenesis**, in response to cold environments.

One way to produce metabolic heat is through muscle contraction—for example, if you shiver uncontrollably when you're very cold. Both deliberate movements—such as rubbing your hands together or going for a brisk walk—and shivering increase muscle activity and thus boost heat production.

Nonshivering thermogenesis provides another mechanism for heat production. This mechanism depends on specialized fat tissue known as **brown fat**, or brown adipose tissue. Some mammals, especially hibernators and baby animals, have lots of brown fat. Brown fat contains many mitochondria with special proteins that let them release energy from fuel molecules directly as heat instead of channeling it into formation of the energy carrier ATP.

To learn more about how energy is released as heat in brown fat cells, have a look at the section on uncoupling proteins in the oxidative phosphorylation.

Non shivering thermogenesis

- Nonshivering thermogenesis, produce heat in response to cold stress. This type of thermogenesis is stimulated by sympathetic nervous system activation, which releases norepinephrine and epinephrine, which in turn increase metabolic activity and heat generation.

- Brown fat, sympathetic nervous stimulation causes liberation of large amounts of heat. This type of fat contains large numbers of mitochondria and many small globules of fat instead of one large fat globule. In these cells, the process of oxidative phosphorylation in the mitochondria is mainly "uncoupled."

- When the cells are stimulated by the sympathetic nerves, the mitochondria produce a large amount of heat but almost no ATP, so almost all the released oxidative energy immediately becomes heat.

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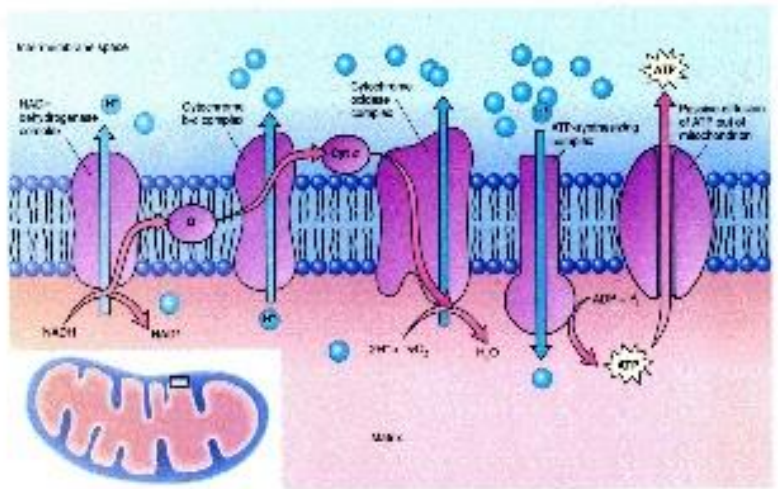
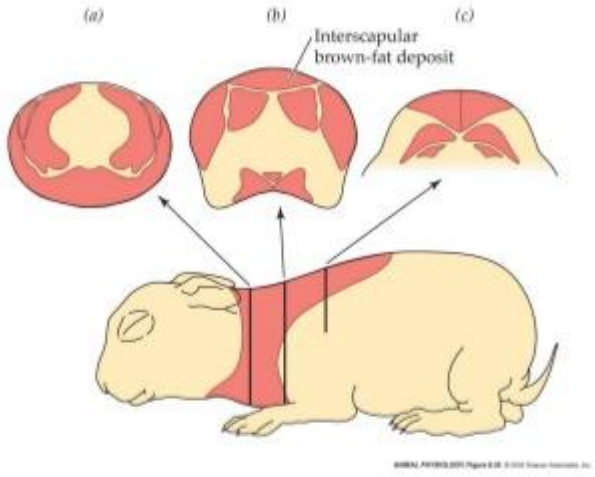


Mitochondrial ET Chain

Typically, the Electron transport chain and oxidative phosphorylation (ATP production) is coupled.

When they are not, the energy released by electron transport is released as heat, rather than used to make ATP

Nonshivering thermogenesis



Increase rate of oxidation of stored lipids

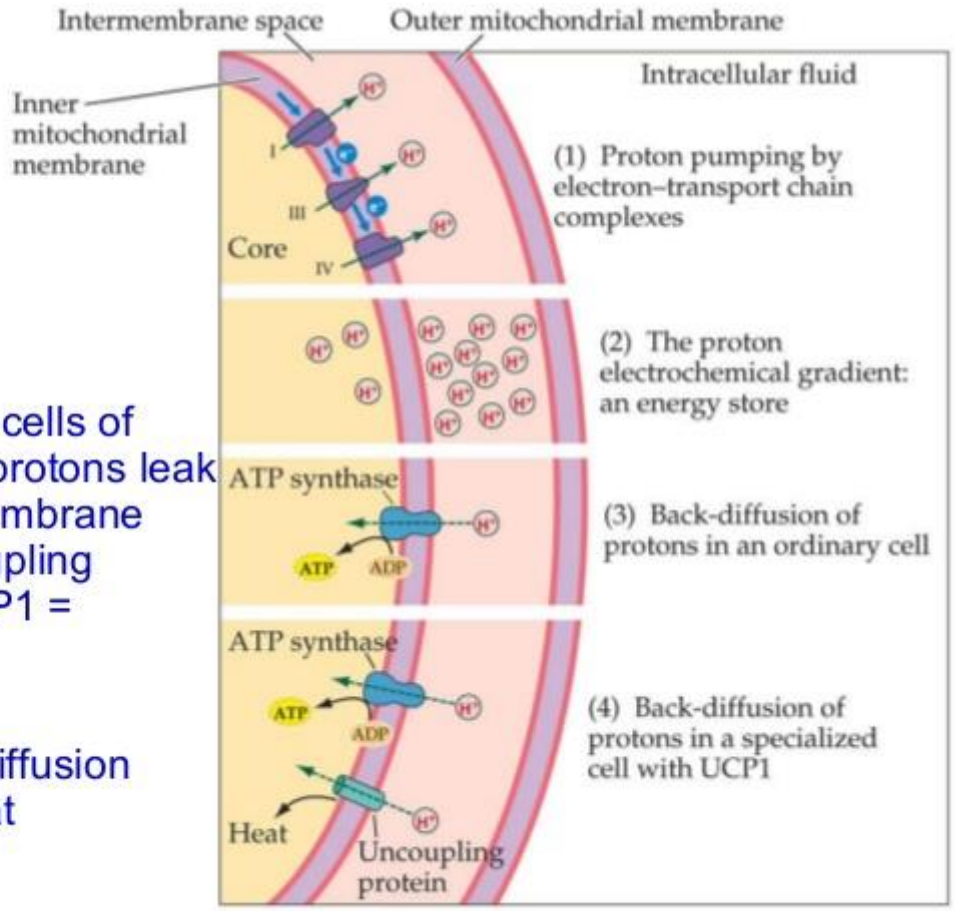
Uncoupling of oxidative phosphorylation from electron transport in the mitochondria

Allows energy to be released as heat rather than stored as ATP

More prominent in cold-adapted mammals, hibernators, newborns

In specialized cells of Endotherms, protons leak across the membrane through uncoupling protein 1 (UCP1 = thermogenin)

Such proton diffusion generates heat



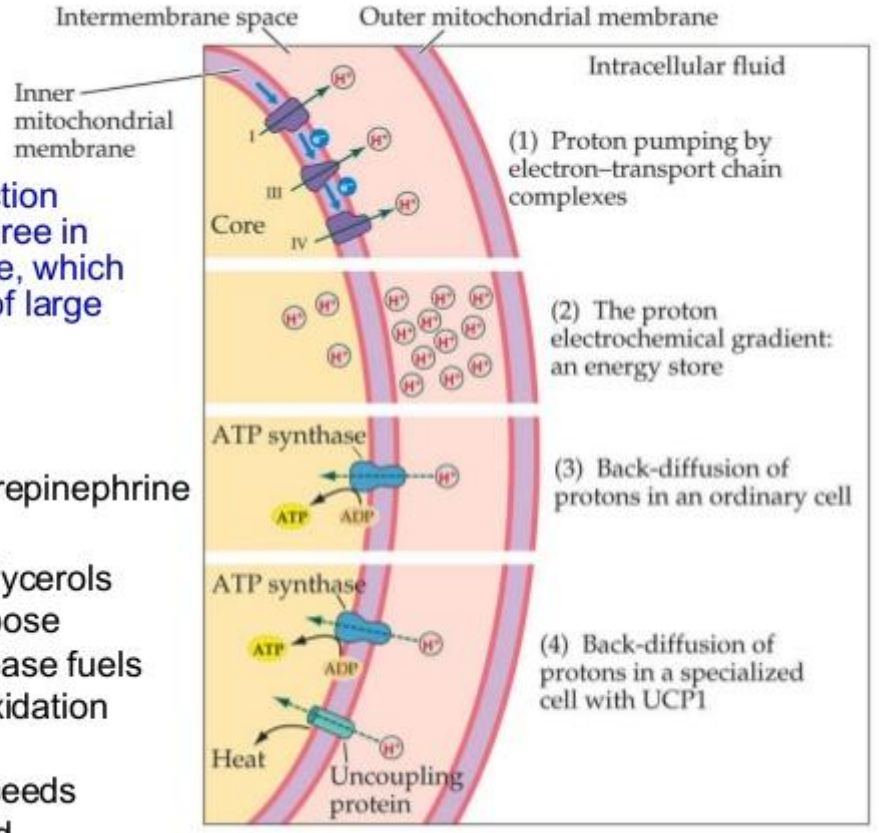
Box 6.1, p. 220

This uncoupled reaction occurs to a high degree in brown adipose tissue, which has large numbers of large mitochondria

Cold --> release Norepinephrine

Hydrolyzes triacylglycerols in BAT (Brown Adipose Tissue) cells to release fuels for mitochondrial oxidation

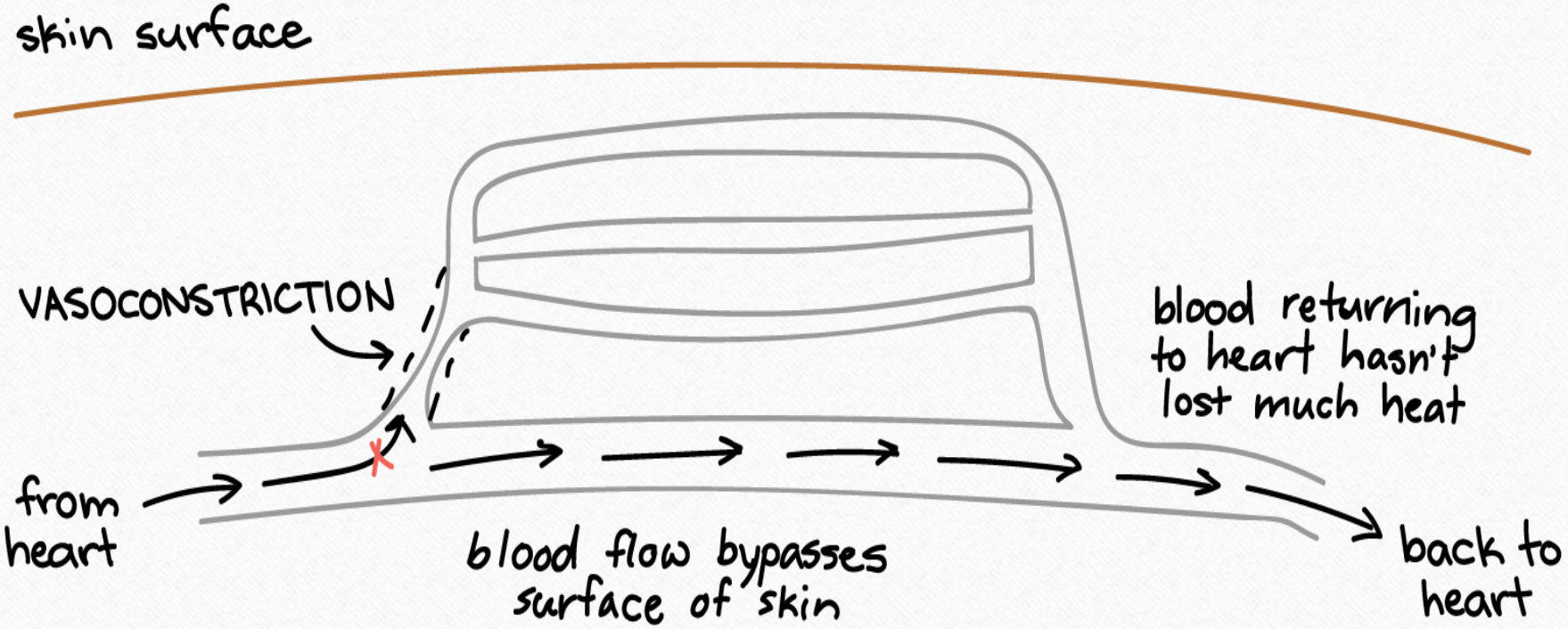
Lipid oxidation proceeds with UCP1 activated

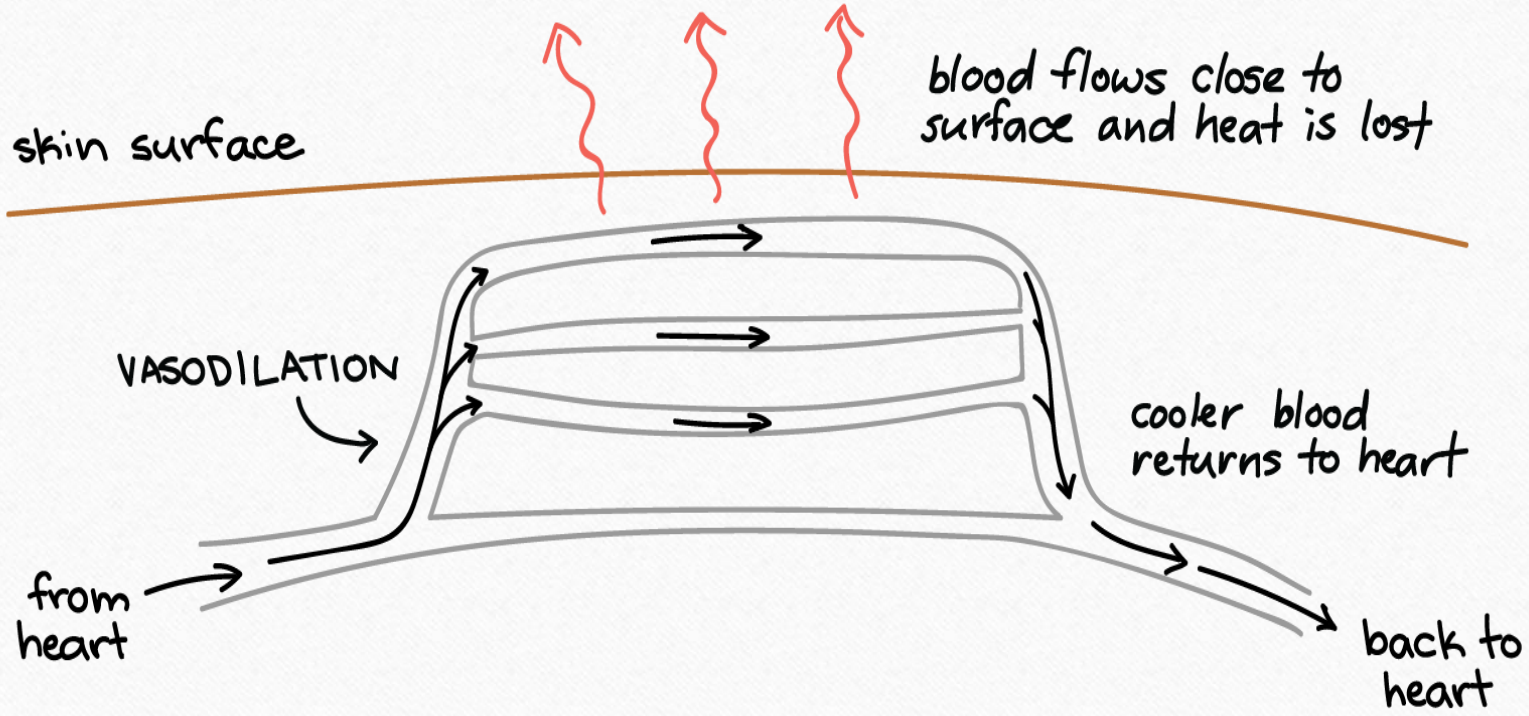


B. Vasoconstriction and vasodilation

In endotherms, warm blood from the body's core typically loses heat to the environment as it passes near the skin. Shrinking the diameter of blood vessels that supply the skin, a process known as **vasoconstriction**, reduces blood flow and helps retain heat.

Vasodilation is the widening of blood vessels. It results from relaxation of smooth muscle cells within the vessel walls, in particular in the large veins, large arteries, and smaller arterioles. This causes more heat to be carried by the blood to the skin, where it can be lost to the air.The process is the opposite of vasoconstriction, which is the narrowing of blood vessels.

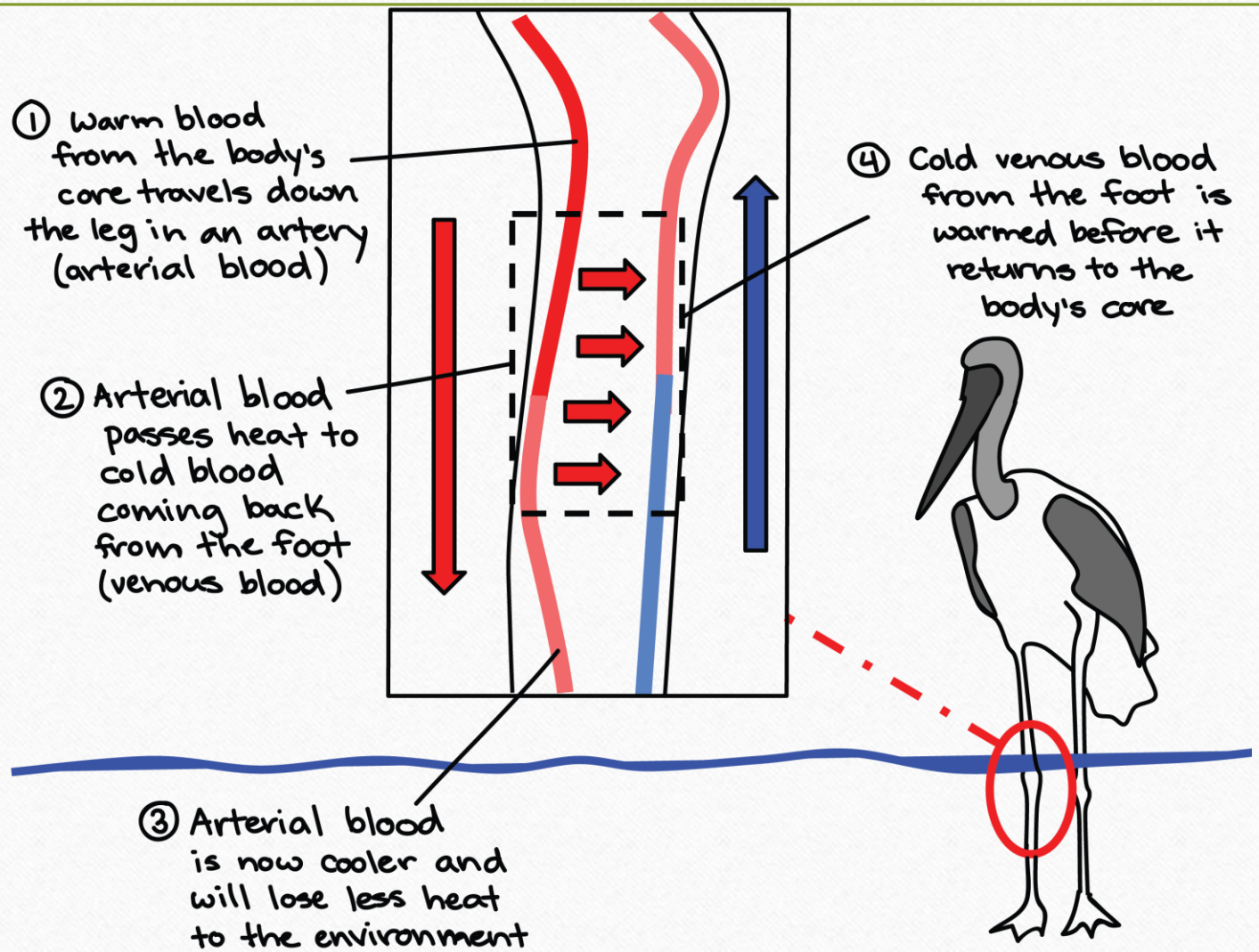

skin surface
VASOCONSTRICTION
blood returning to heart hasn't lost much heat
from heart
blood flow bypasses surface of skin
back to heart


skin surface
blood flows close to surface and heat is lost
VASODILATION
cooler blood returns to heart
from heart
back to heart

C.Countercurrent heat exchange

Many birds and mammals have **counter current heat exchangers**, circulatory adaptations that allow heat to be transferred from blood vessels containing warmer blood to those containing cooler blood. To see how this works, let's look at an example.

In the leg of a wading bird, the artery that runs down the leg carries warm blood from the body. The artery is positioned right alongside a vein that carries cold blood up from the foot. The descending, warm blood passes much of its heat to the ascending, cold blood by conduction. This means that less heat will be lost in the foot due to the reduced temperature difference between the cooled blood and the surroundings and that the blood moving back into the body's core will be relatively warm, keeping the core from getting cold.


① Warm blood from the body's core travels down the leg in an artery (arterial blood)
② Arterial blood passes heat to cold blood coming back from the foot (venous blood)
③ Arterial blood is now cooler and will lose less heat to the environment as it travels through the foot
④ Cold venous blood from the foot is warmed before it returns to the body's core

Countercurrent heat exchangers

- Many marine mammals and birds
  - Have arrangements of blood vessels called countercurrent heat exchangers that are important for reducing heat loss

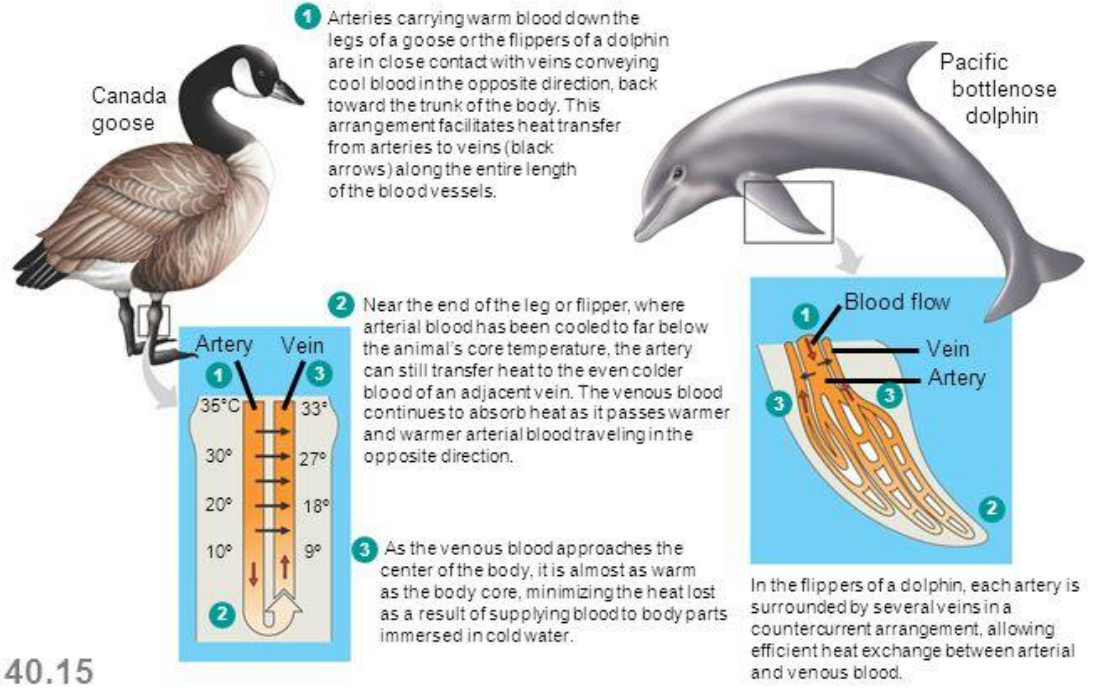
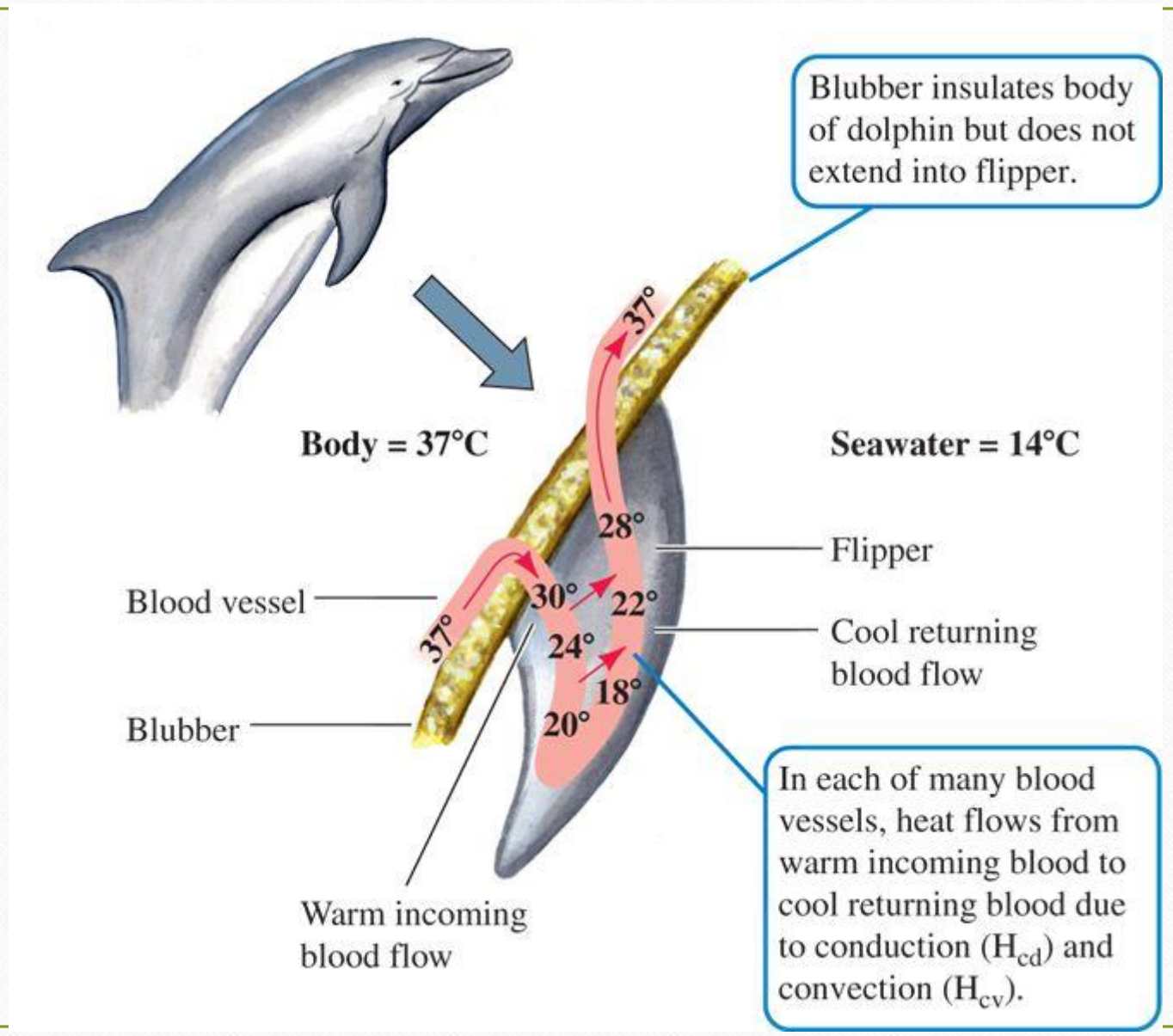


Figure 40.15


Blubber insulates body of dolphin but does not extend into flipper.
Body = 37°C
Seawater = 14°C
37°
28°
30°
22°
24°
18°
20°
37°
Blood vessel
Blubber
Flipper
Cool returning blood flow
Warm incoming blood flow
In each of many blood vessels, heat flows from warm incoming blood to cool returning blood due to conduction (H_{cd}) and convection (H_{cv}).

HORMONES involved in THERMOREGULATION

Reproductive hormones have important influences on mechanisms of thermoregulation.

Estrogen promotes heat dissipation via peripheral vascular effects favoring vasodilation, as well as central neural thermoregulatory effects promoting more efficient cutaneous vasodilator and sweating responses.

Progestogens may have the opposite effect: the combination of progesterone and estrogen favors heat conservation and/or increased body temperature (such as that seen in the mid-luteal phase of the menstrual cycle). Like estrogen, the influences of progestogens may also include both CNS and peripheral influences.

Testosterone has influences on central neural control of thermoregulation, and may interact with estrogen in overall thermoregulation; however, both central and peripheral thermoregulatory influences of this hormone await further clarification. From a practical perspective, it is relevant that the reproductive hormone influences discussed here are unlikely to result in substantial sex differences in core temperature during many commonly performed activities (mild to moderate intensity exercise) in healthy people.

In Birds:

The thermoregulatory system that enables maintenance of a relatively constant Tb (body temperature)over a wide range of environmental temperatures in birds consists of

(1) a sensory part, which detects changes in the environment (thermo-, osmo-, and baroreceptors),

(2) an integrating part, the thermoregulatory center at preoptic anterior hypothalamus (PO/AH), in which temperature-sensitive neurons monitor local temperature changes and temperature information received from peripheral thermoreceptors. The Tb set point is then defended by mechanisms for heat production or heat loss depending on the thermal status of the bird; and

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- The role of the hypothalamus-pituitary-thyroid (HPT) axis in avian thermoregulation Thyroid hormones (THs, triiodothyronine, T3 and thyroxine, T4) are the most important hormones controlling thermogenesis; a link that was established already in the late.

- **PO/AH** is activated by input from thermoreceptors, which stimulate the hypothalamic paraventricular nucleus (PVN) and leads to an increase in thyrotropin (TRH) synthesis and secretion.

- **TRH** stimulates the thyrotrophs in the anterior pituitary to secrete thyroid stimulating hormone (TSH), which interacts with the follicular cell membrane receptors in the thyroid gland. This results in the activation of adenylate cyclase and cAMP production ultimately leading to increased thyroid hormone (mainly T4) synthesis and release.

- Alternatively, cold exposure can lead to increased conversion of T4 to T3 (and less to inactive rT3) by deiodinase enzymes (DIO1-3) in tissues, mostly in the liver), thus leading to higher circulating T3 levels.