Figure 22-2

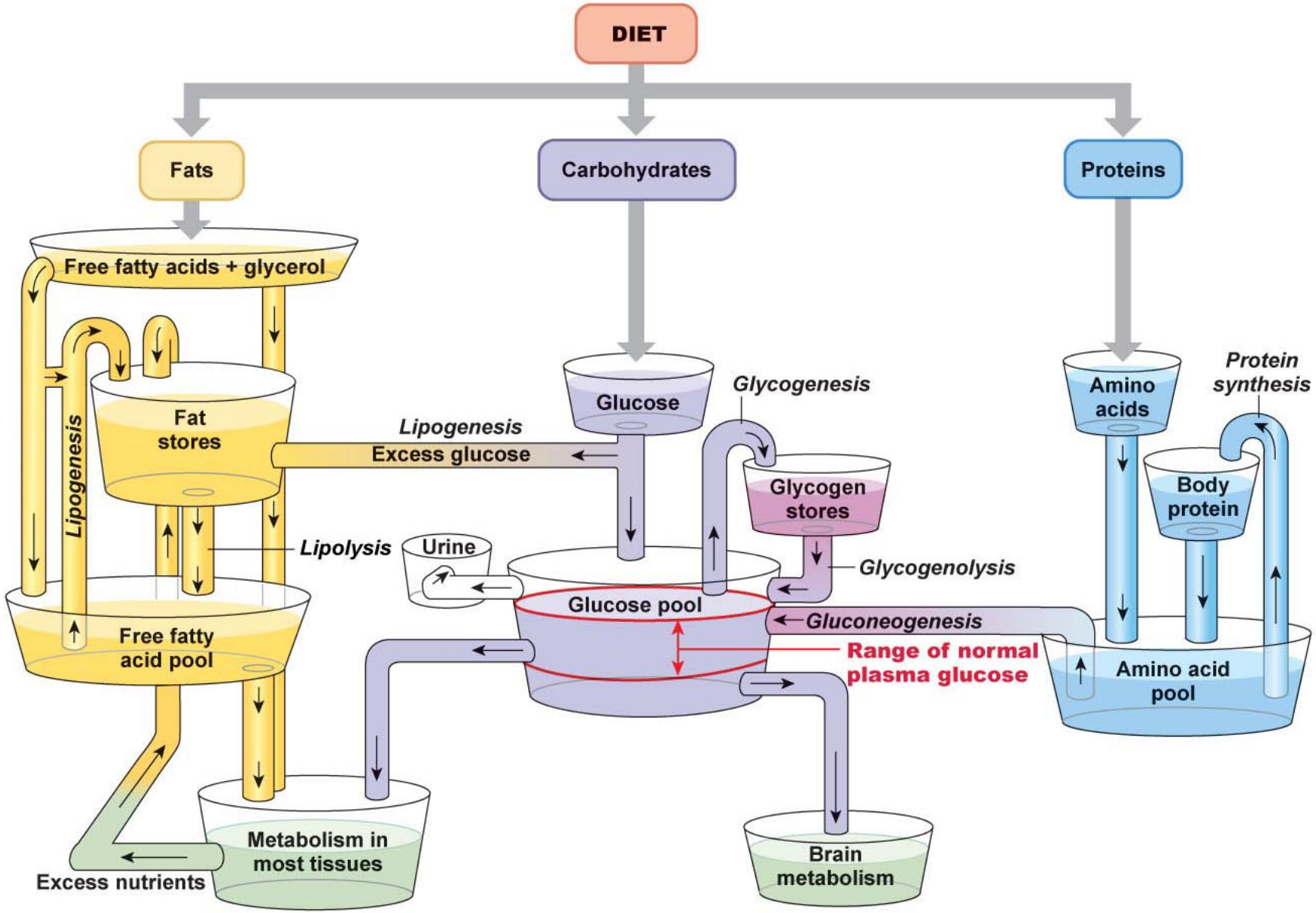


Figure 22-3, overview

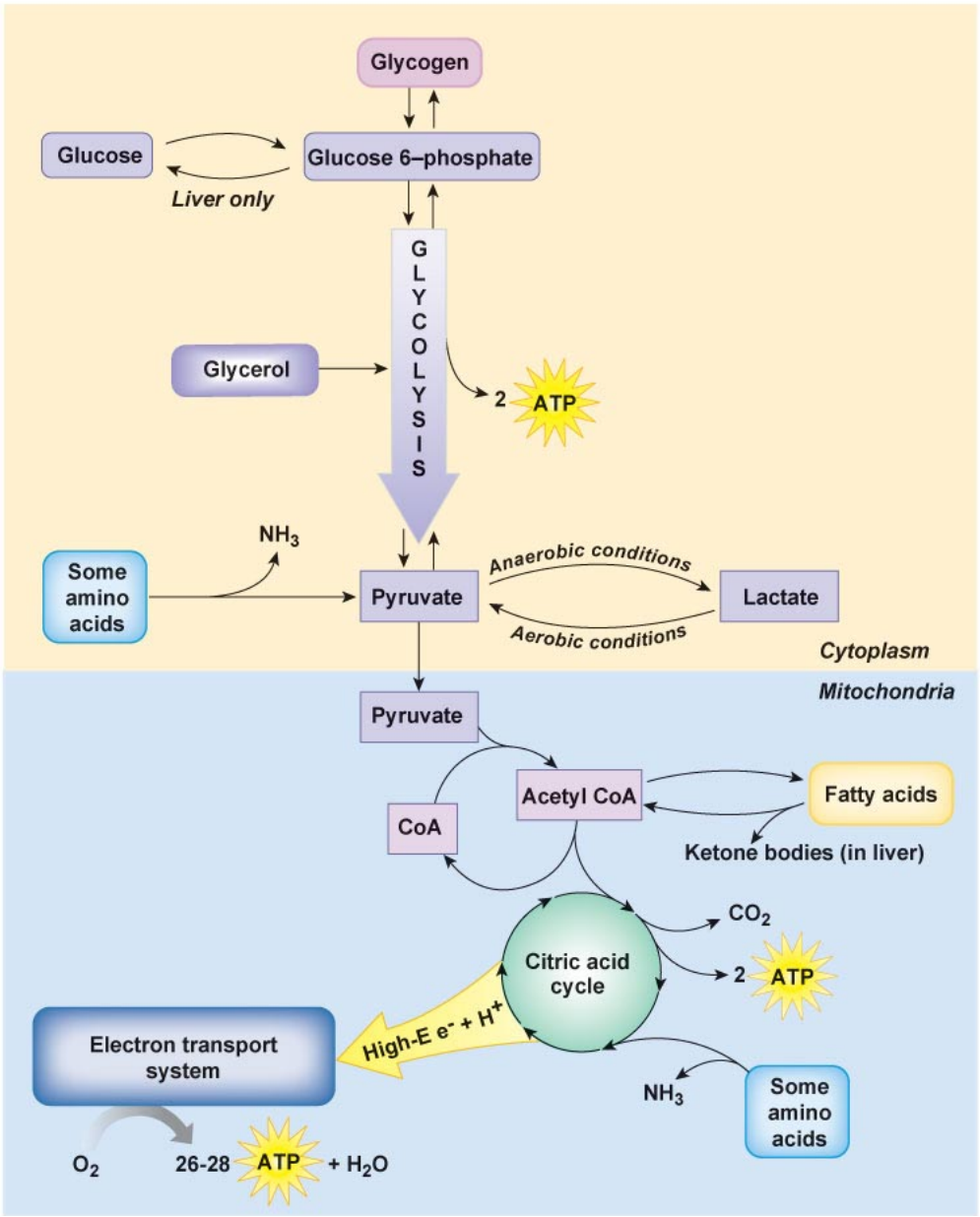


Figure 22-4

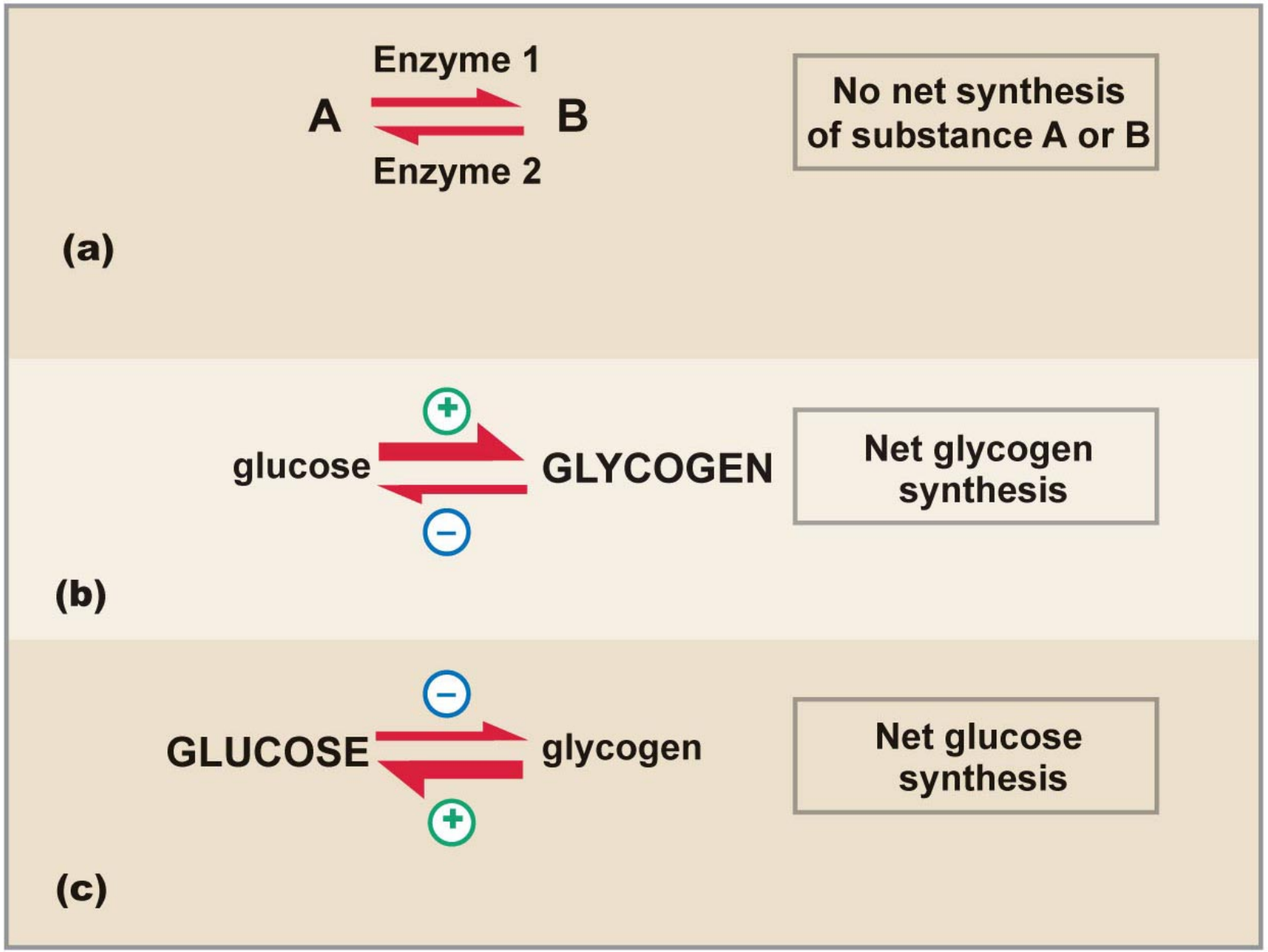


Figure 22-5

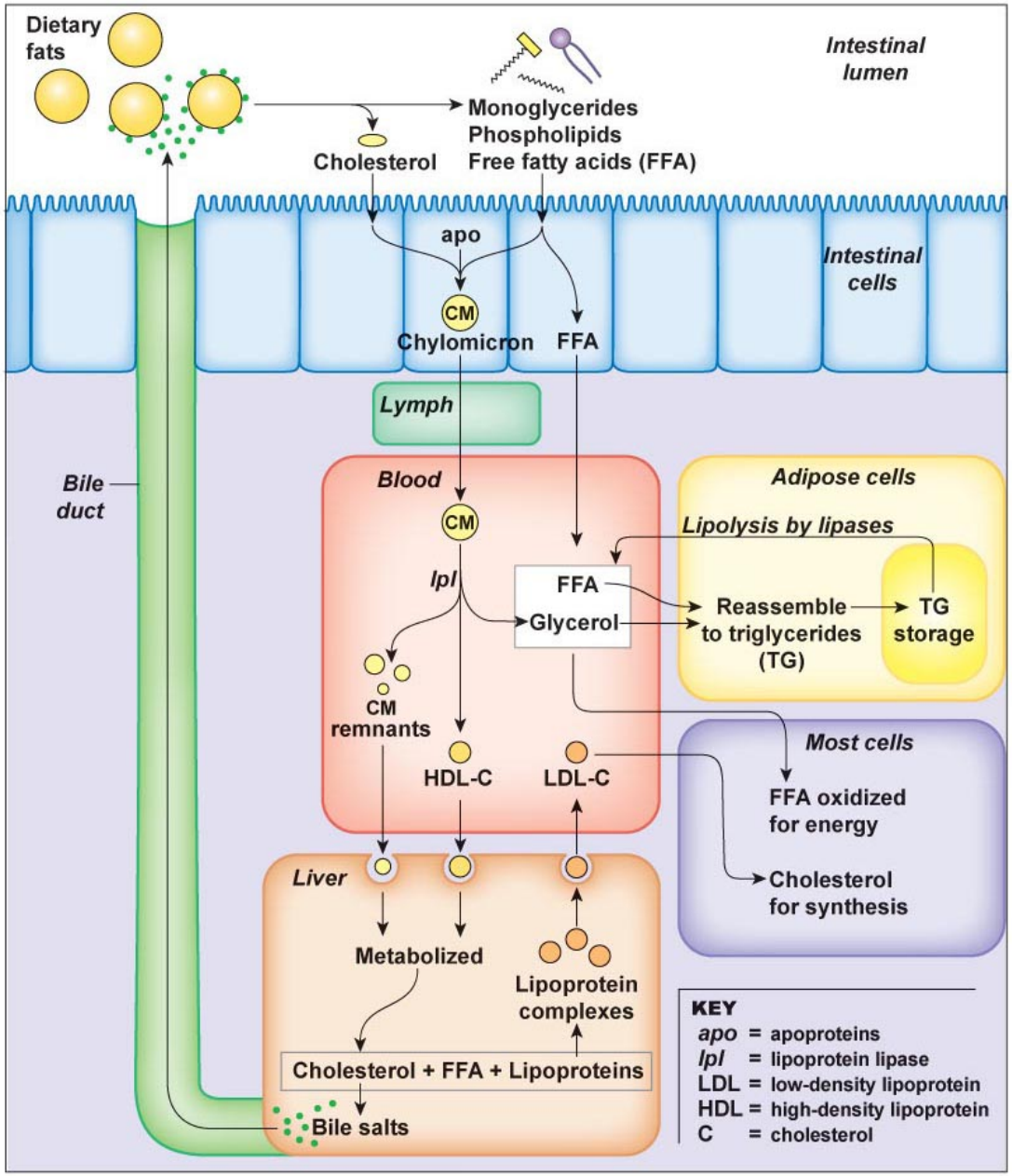


Figure 22-7, overview

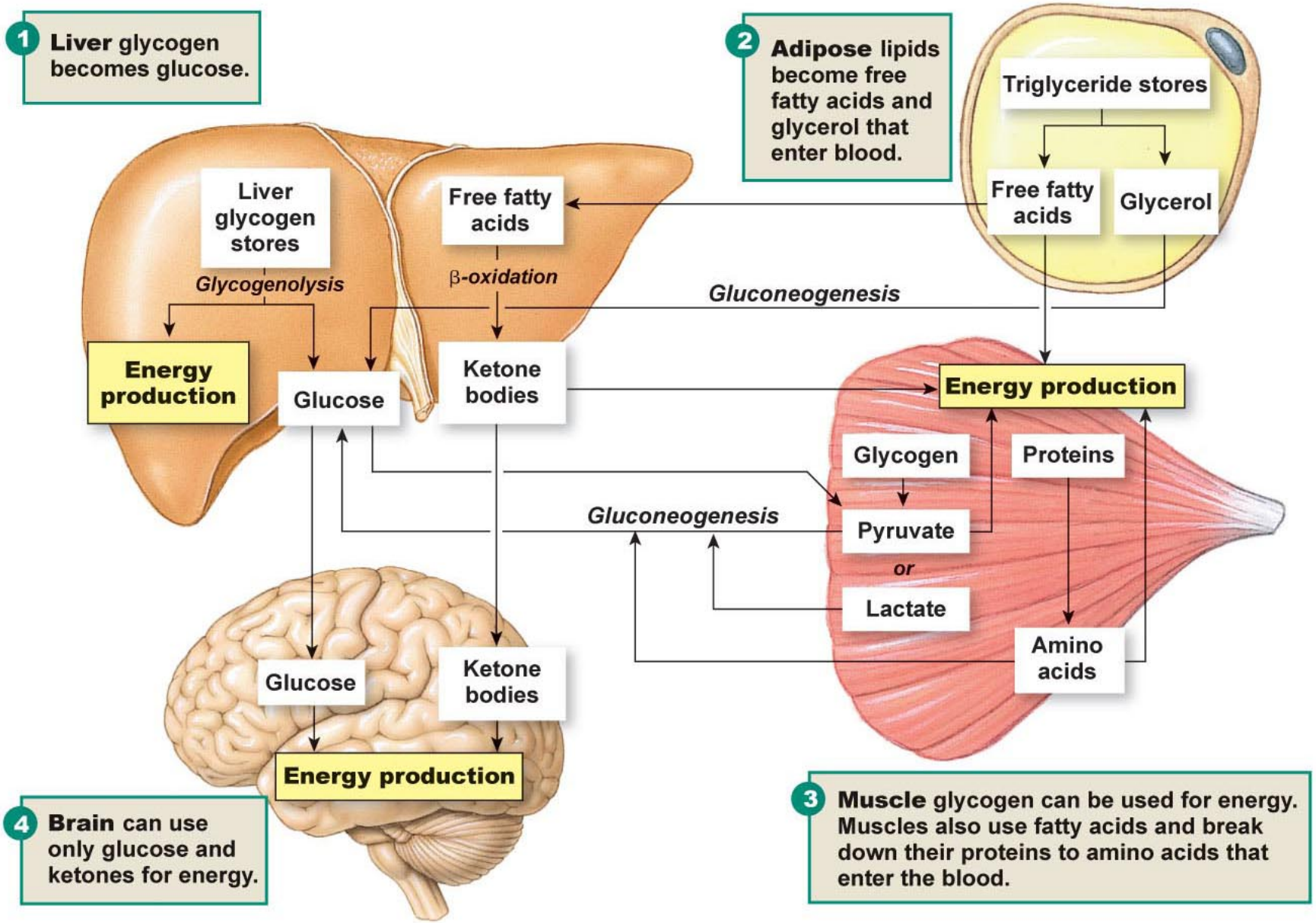


Figure 22-8

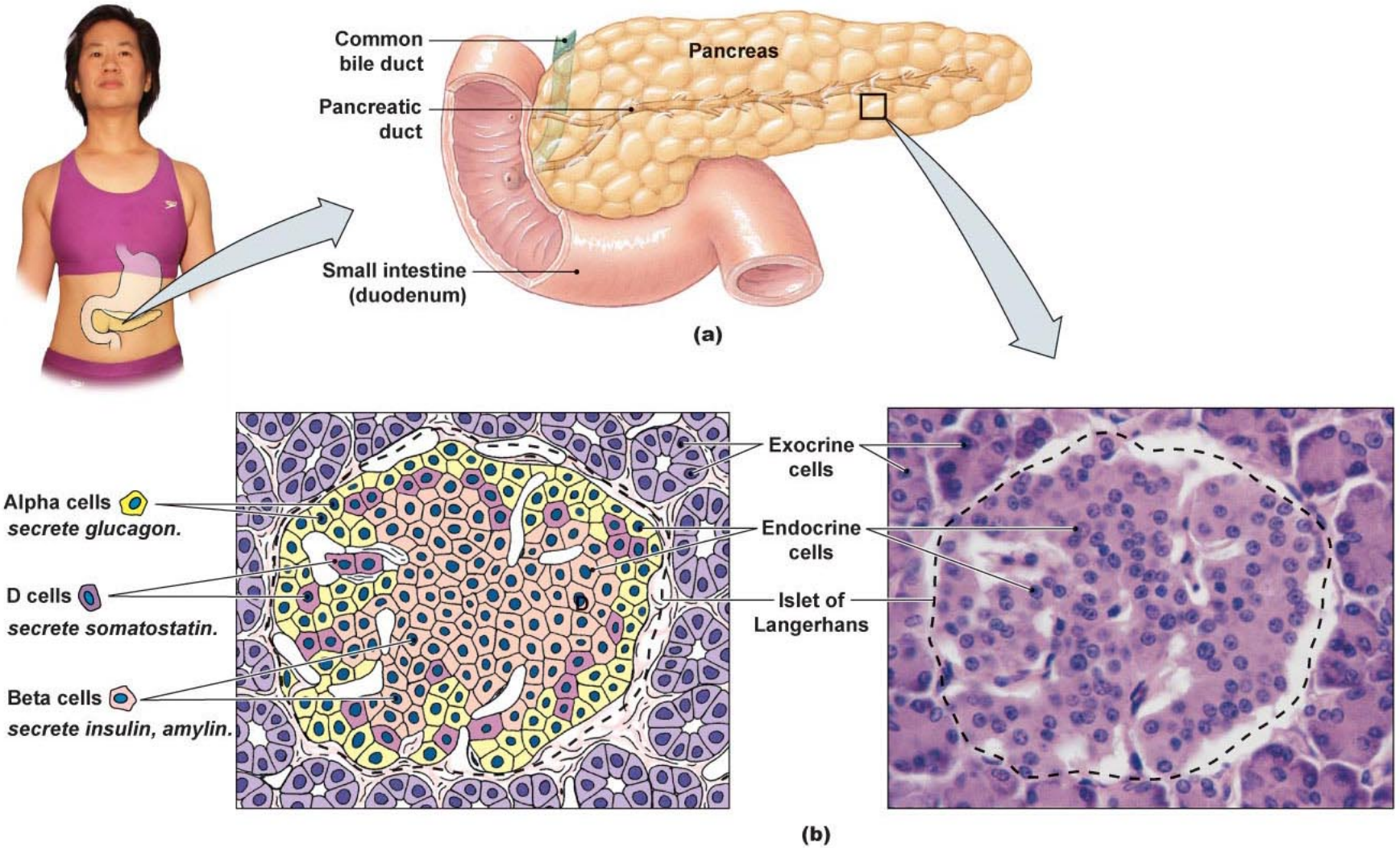
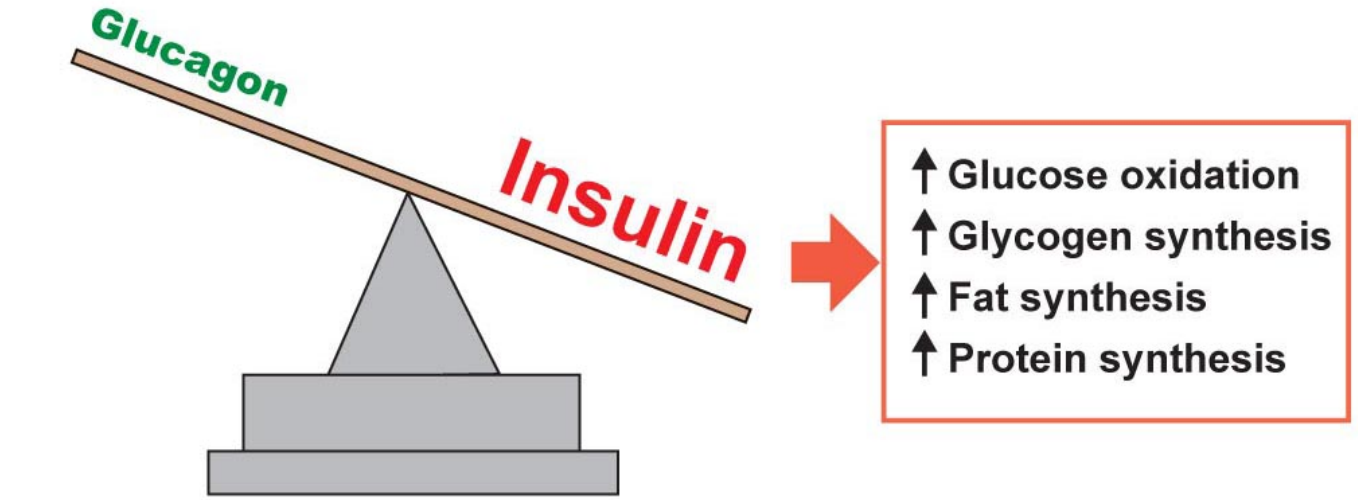
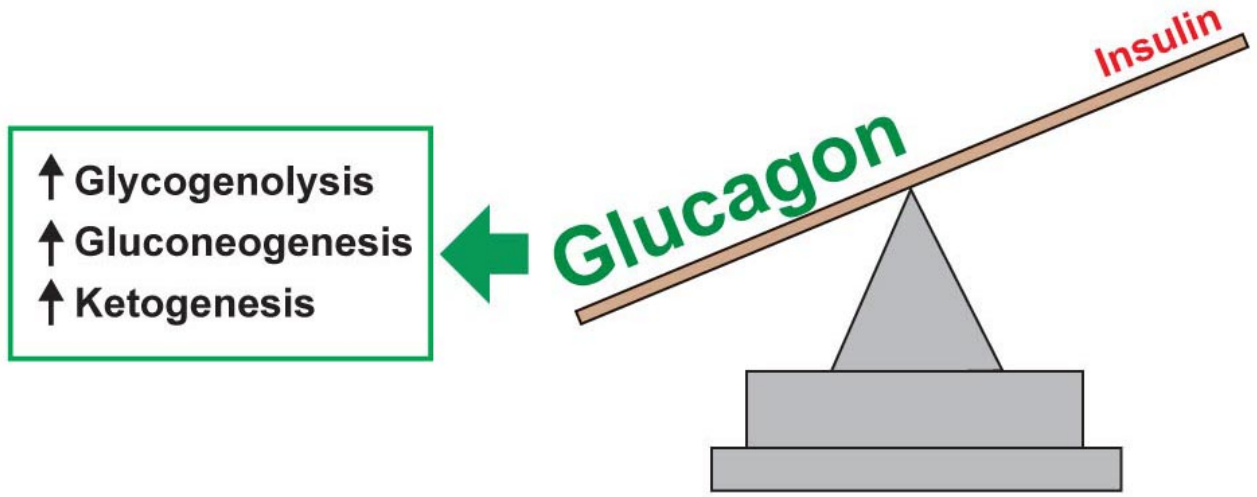


Figure 22-9



(a) Fed state: insulin dominates



(b) Fasted state: glucagon dominates

Table 22-2

TABLE 22-2 Fates of Nutrients in the Fed State

CARBOHYDRATES (absorbed primarily as glucose)
1. Used immediately for energy through aerobic pathways*
2. Used for lipoprotein synthesis in liver
3. Stored as glycogen in liver and muscle
4. Excess converted to fat and stored in adipose tissue (glucose → pyruvate → acetyl CoA → fatty acids)
PROTEINS (absorbed primarily as amino acids)
1. Most amino acids go to tissues for protein synthesis*
2. If needed for energy, amino acids converted in liver to intermediates for aerobic metabolism
3. Excess converted to fat and stored in adipose tissue (amino acids → acetyl CoA → fatty acids)
FATS (absorbed primarily as triglycerides)
1. Stored as fats primarily in liver and adipose tissue*

Table 22-3

TABLE 22-3	Insulin
Cell of origin	Beta cells of pancreas
Chemical nature	51-amino acid peptide
Biosynthesis	Typical peptide
Transport in the circulation	Dissolved in plasma
Half-life	5 minutes
Factors affecting release	Plasma [glucose] > 100 mg/dL; ↑ blood amino acids; GLP-1 (feedforward reflex). Parasympathetic activity amplifies. Sympathetic activity inhibits.
Target cells or tissues	Liver, muscle, and adipose tissue primarily; brain, kidney, and intestine not insulin dependent
Target receptor	Membrane receptor with tyrosine kinase activity; pathway with insulin-receptor substrates
Whole body or tissue action	↓ Plasma [glucose] by ↑ transport into cells or ↑ metabolic use of glucose
Action at cellular level	↑ Glycogen synthesis; ↑ aerobic metabolism of glucose; ↑ protein and triglyceride synthesis
Action at molecular level	Inserts GLUT transporters in muscle and adipose cells; alters enzyme activity. Complex signal transduction pathway involved.
Feedback regulation	↓ Plasma [glucose] shuts off insulin release.
Other information	Growth hormone and cortisol are antagonistic.

Figure 22-10

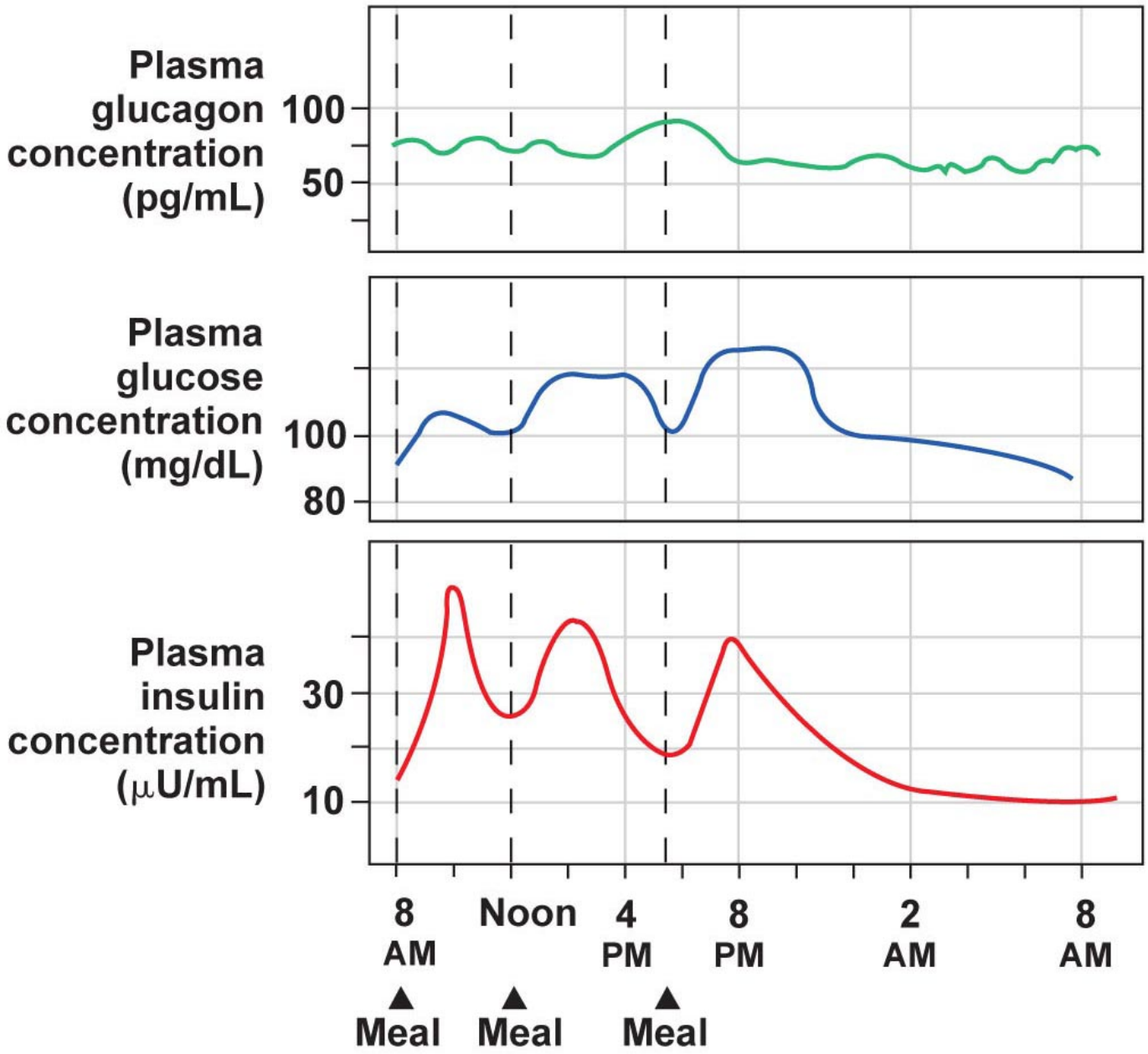
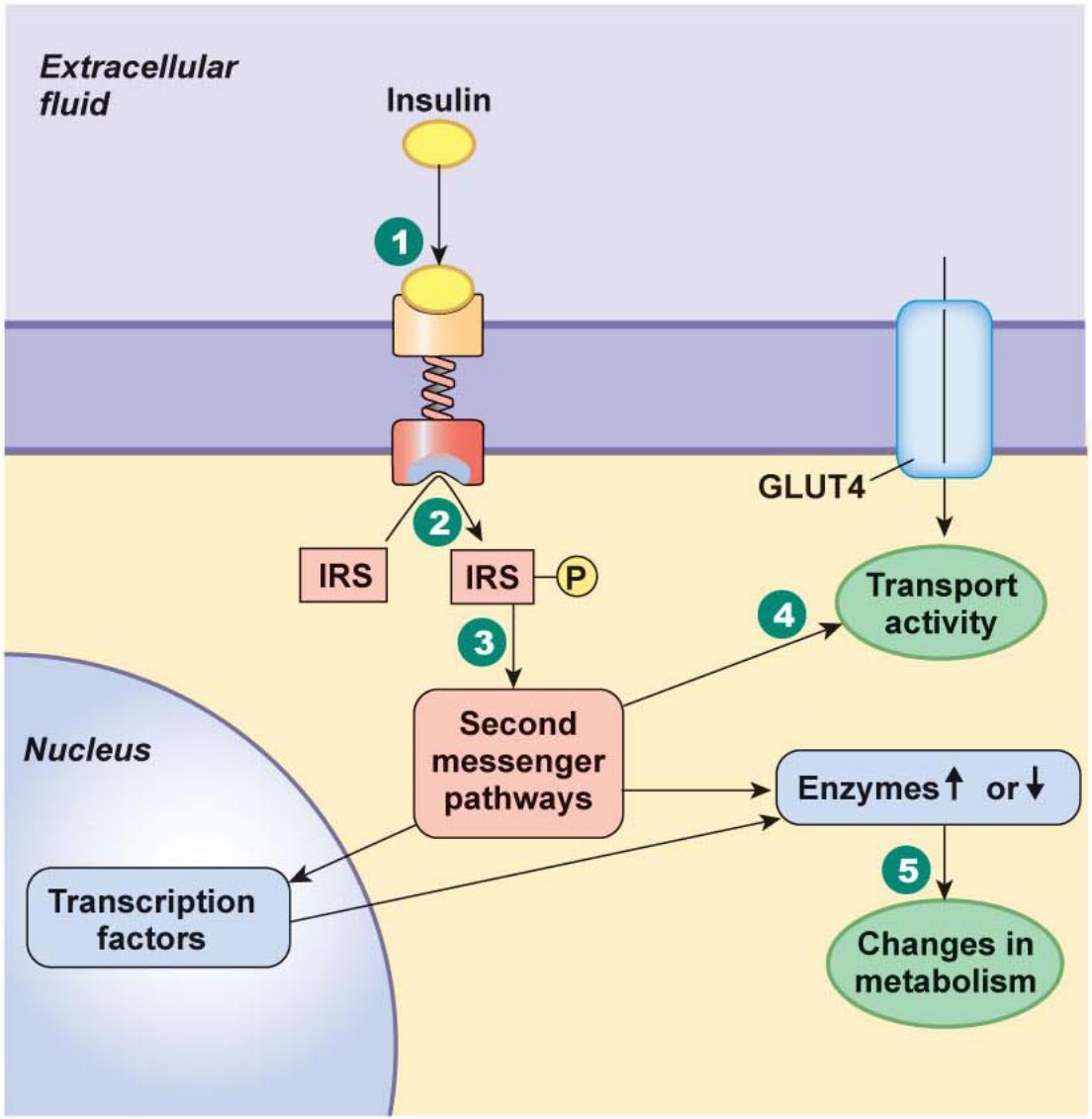


Figure 22-11, overview



1. Insulin binds to tyrosine kinase receptor.
2. Receptor phosphorylates insulin-receptor substrates (IRS).
3. Second messenger pathways alter protein synthesis and existing proteins.
4. Membrane transport is modified.
5. Cell metabolism is changed.

Figure 22-12

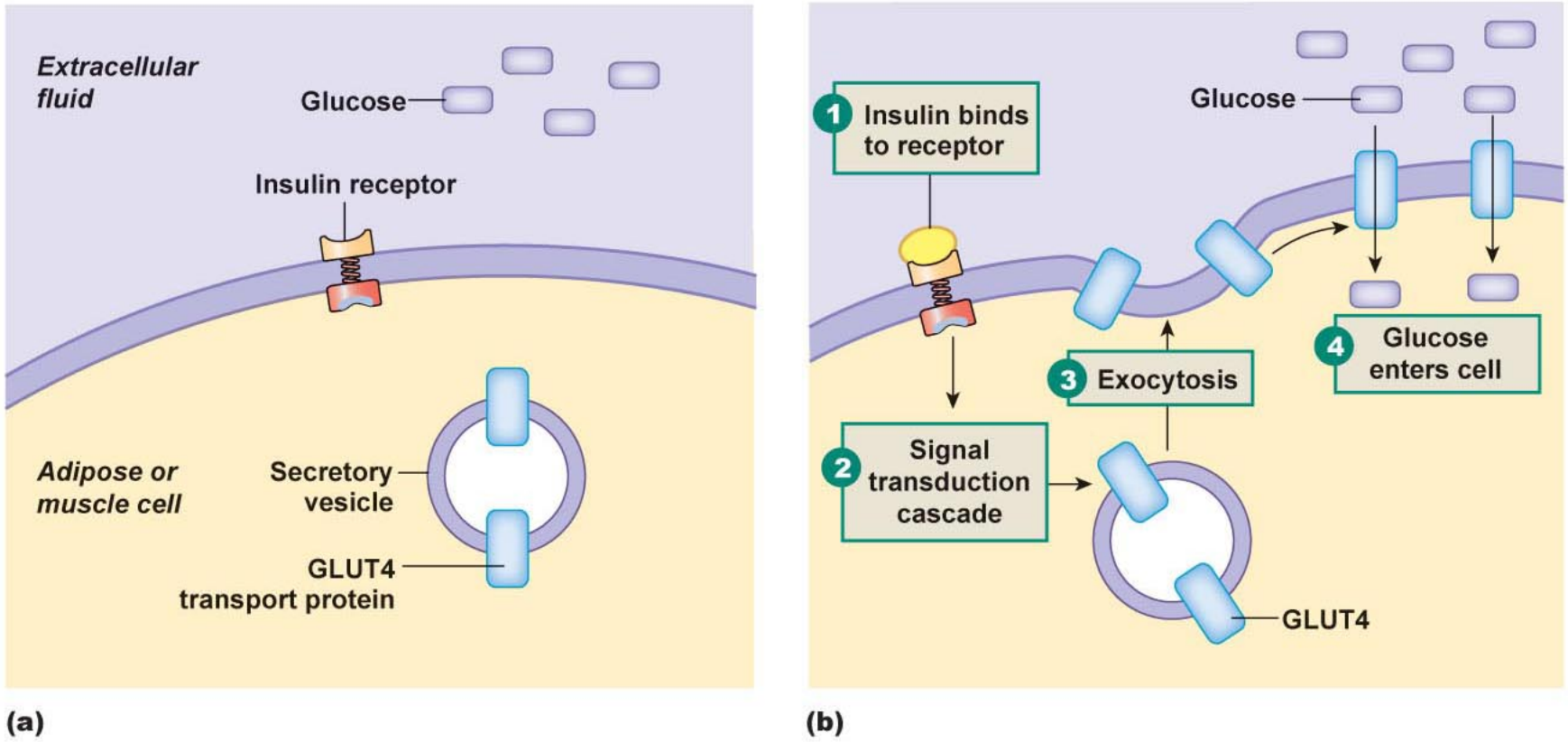


Figure 22-13

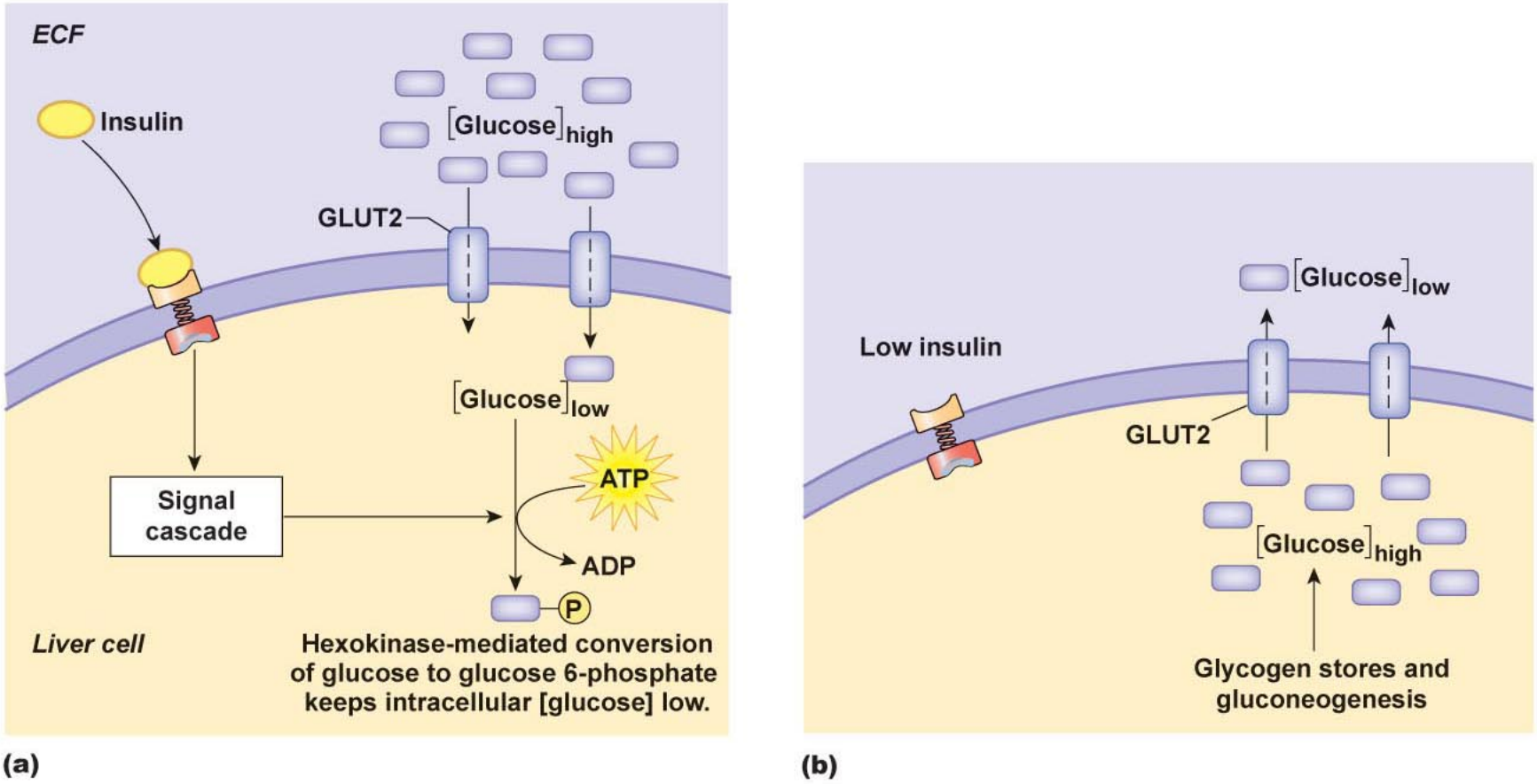


Figure 22-14

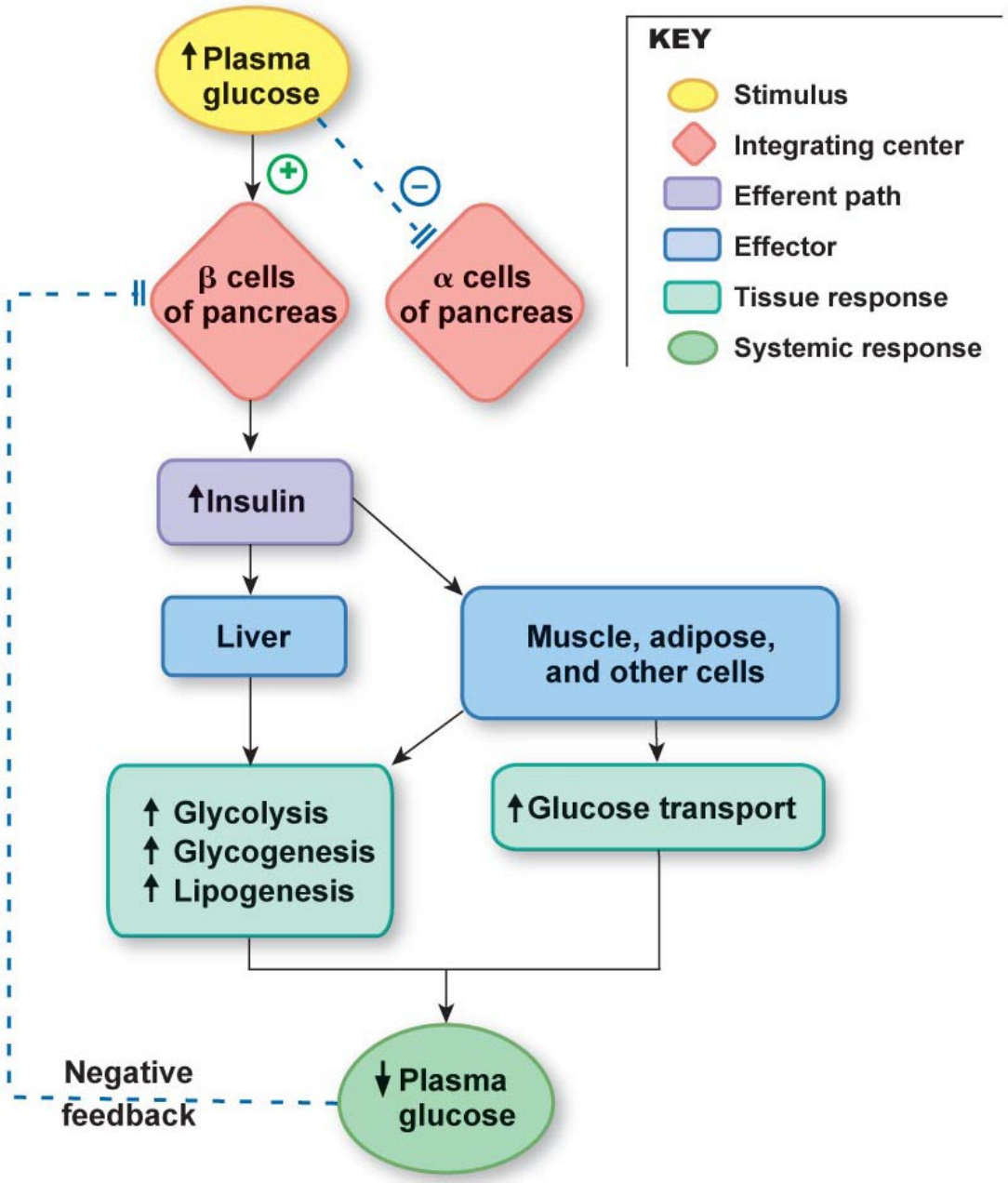


Table 22-5

TABLE 22-5	Glucagon
Cell of origin	Alpha cells of pancreas
Chemical nature	29-amino acid peptide
Biosynthesis	Typical peptide
Transport in the circulation	Dissolved in plasma
Half-life	4–6 minutes
Factors affecting release	Enhanced secretion when plasma [glucose] < 65-70 mg/dL; ↑ blood amino acids
Target cells or tissues	Liver primarily
Target receptor/ second messenger	G protein–coupled receptor linked to cAMP
Whole body or tissue action	↑ Plasma [glucose] by glycogenolysis and gluconeogenesis; ↑ lipolysis leads to ketogenesis in liver
Action at molecular level	Alters existing enzymes and stimulates synthesis of new enzymes
Feedback regulation	↑ Plasma [glucose] shuts off glucagon secretion
Other information	Member of secretin family (along with VIP, GIP, and GLP-1)

Figure 22-15

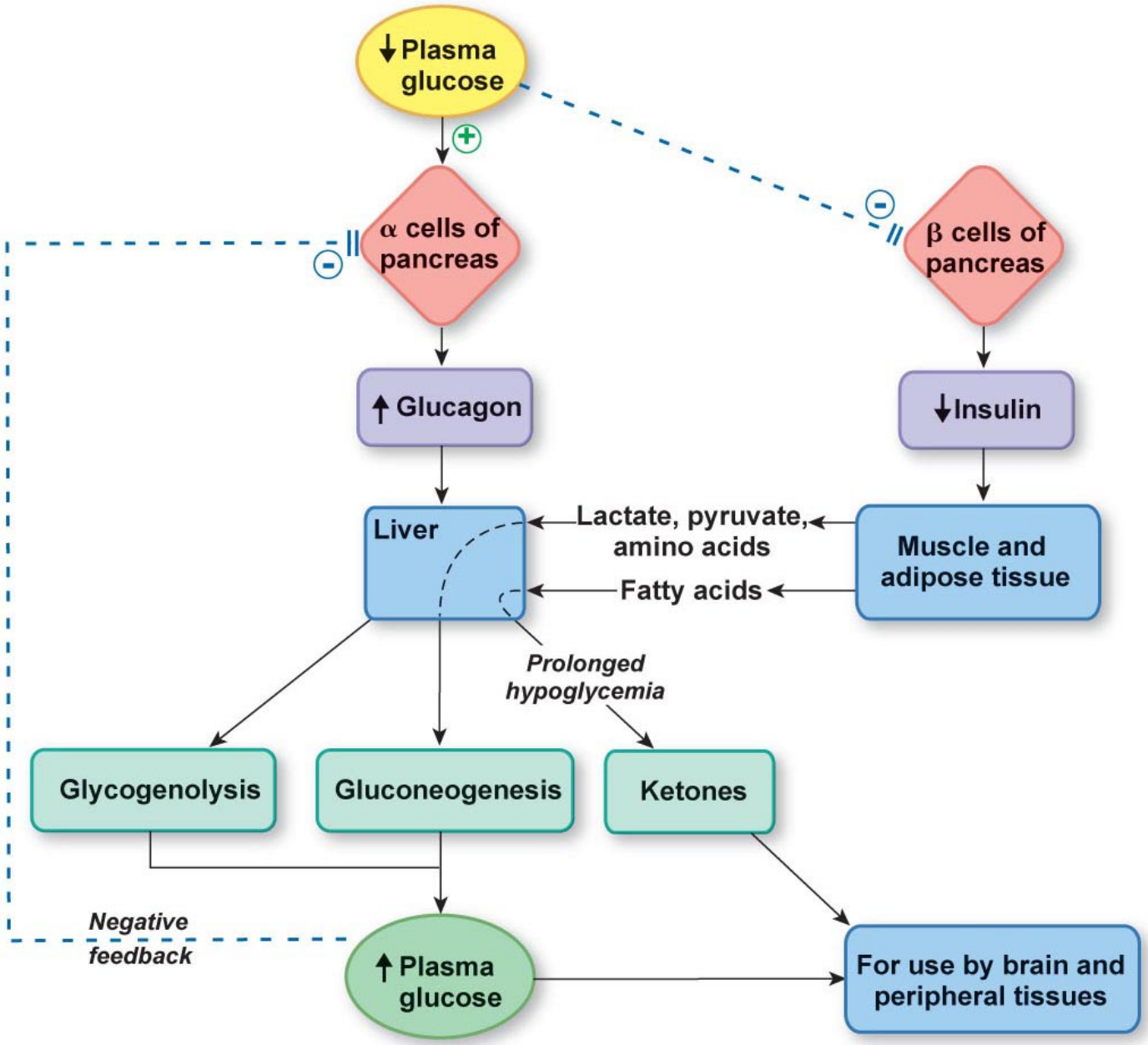


Figure 22-16

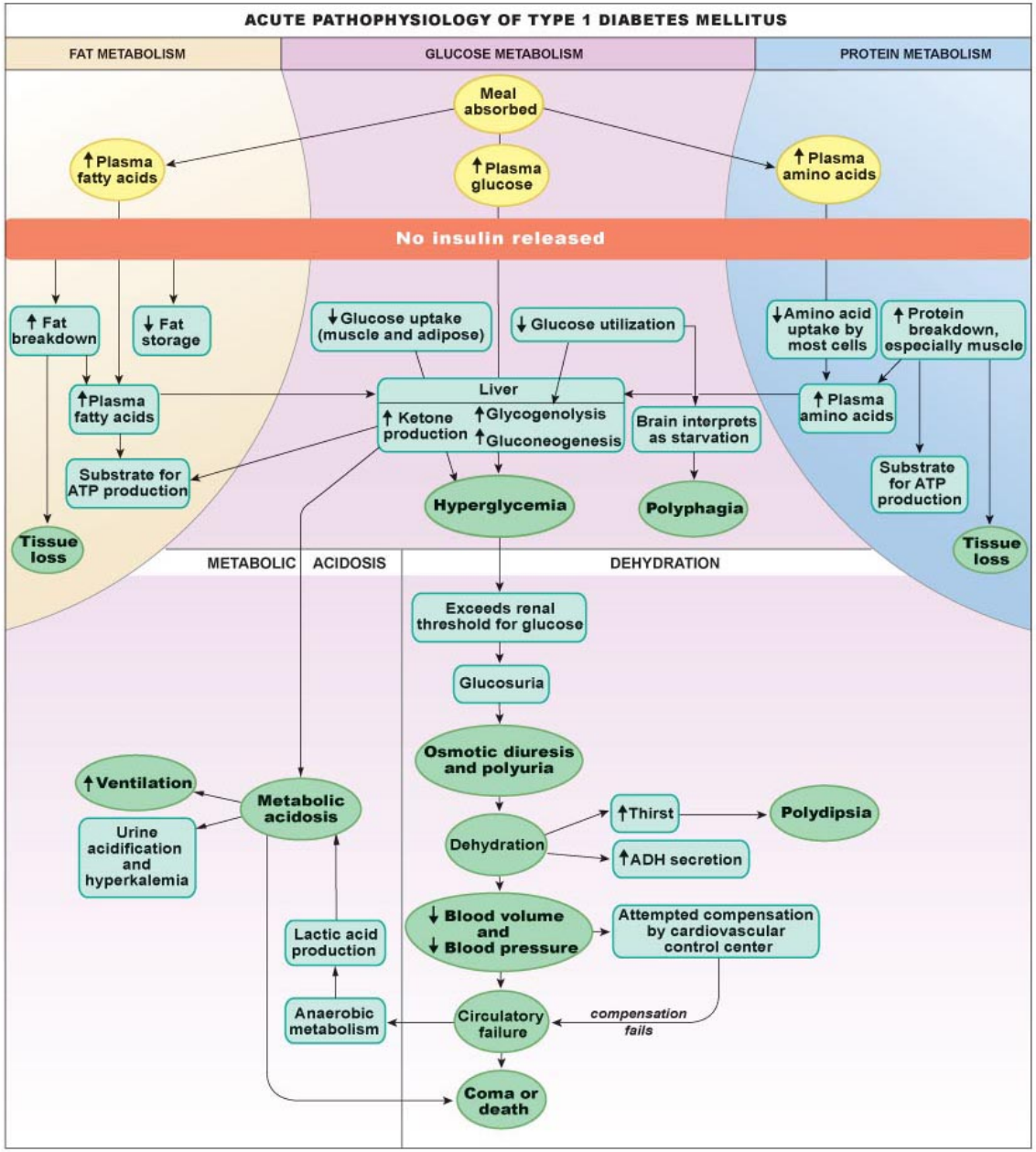


Figure 22-17

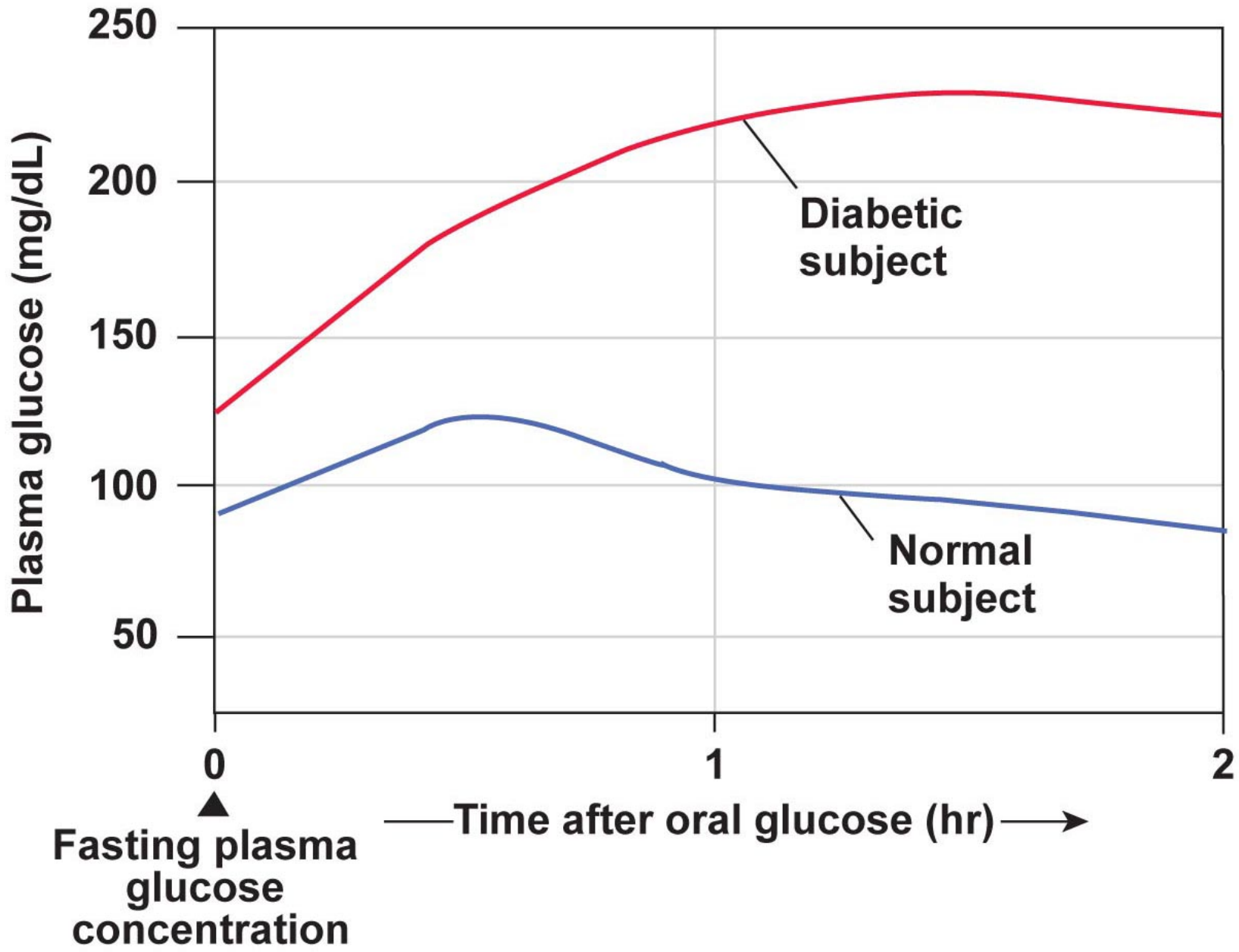


Figure 22-18

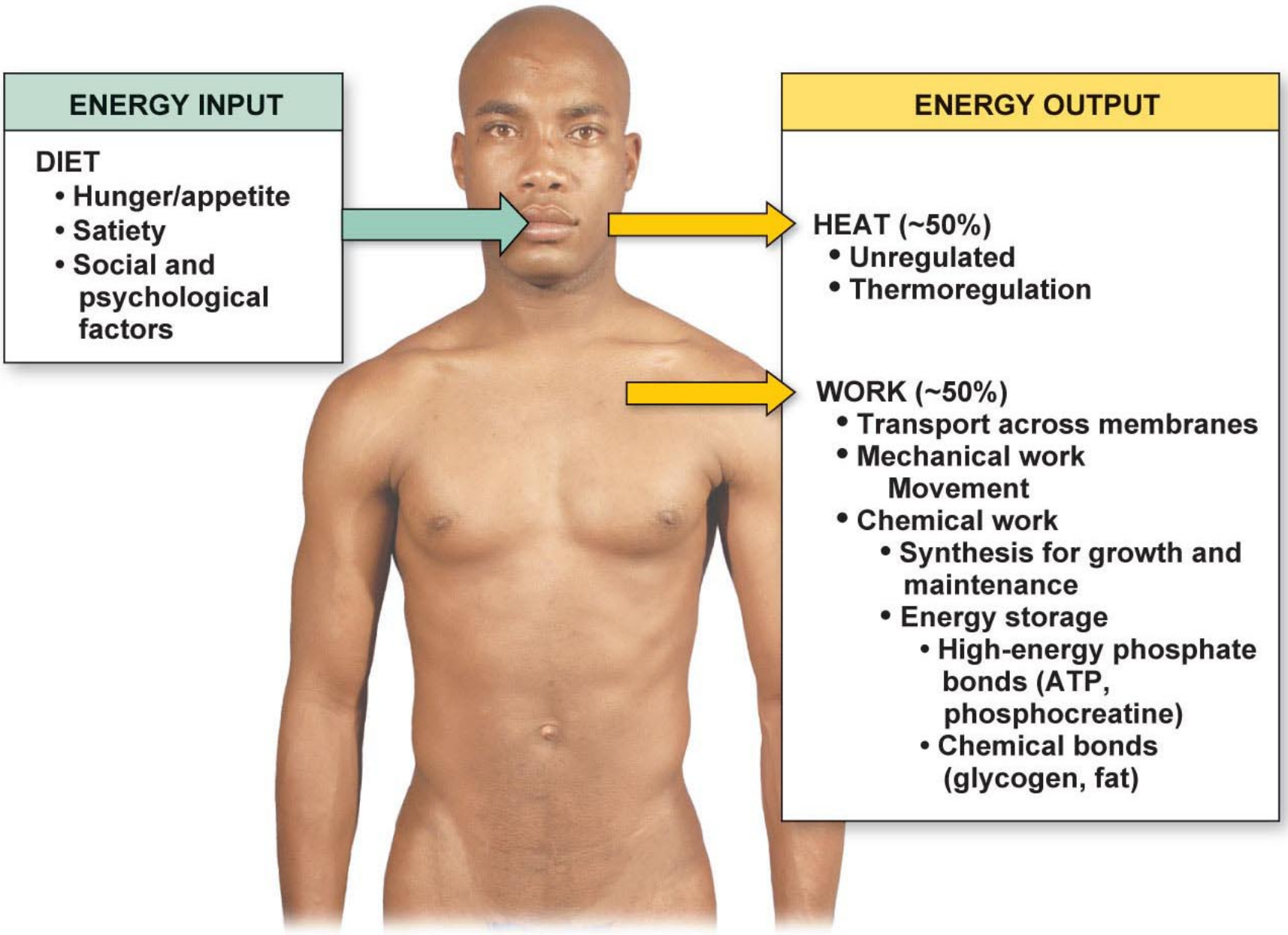


Figure 22-19

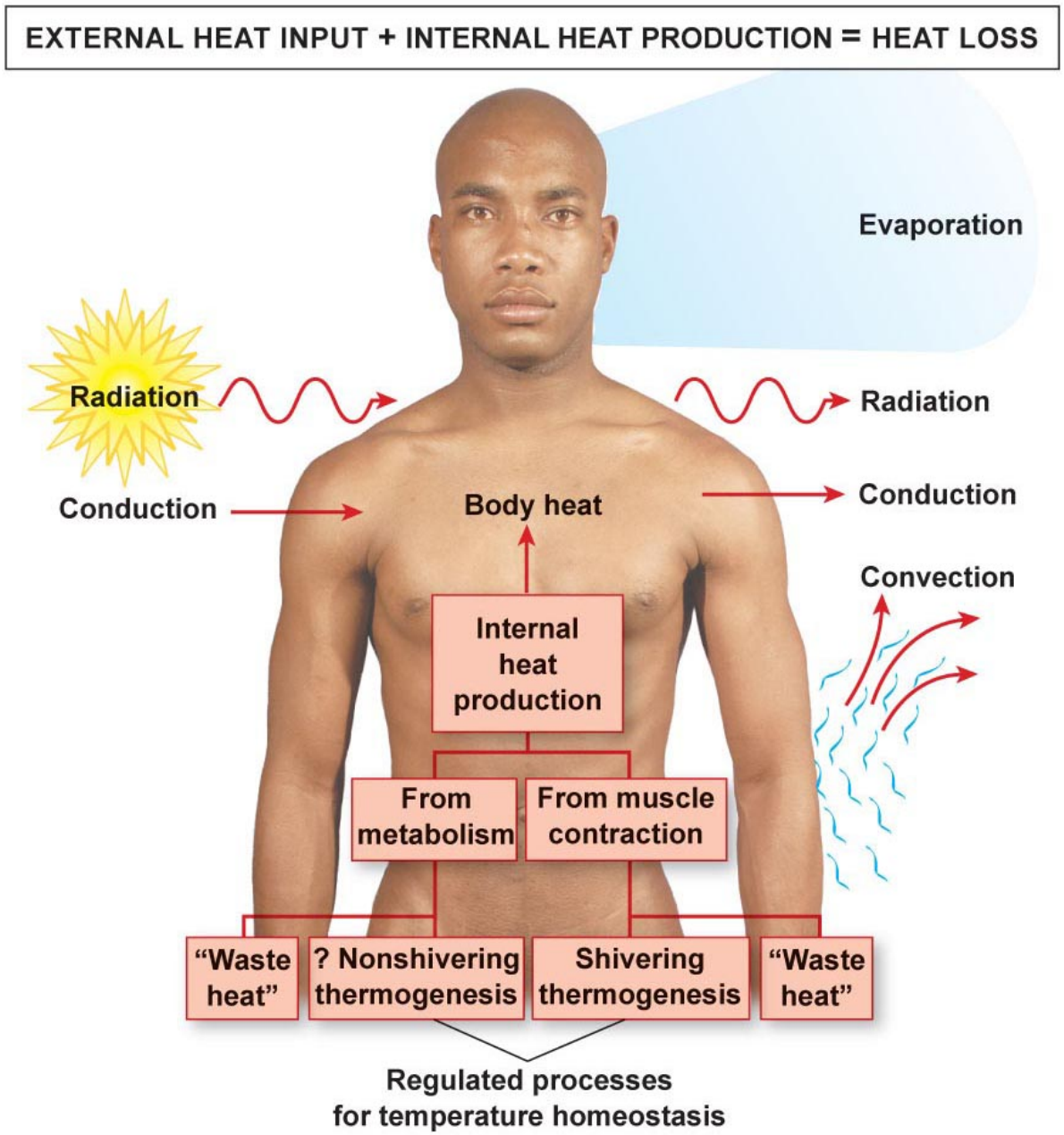


Figure 22-20, overview

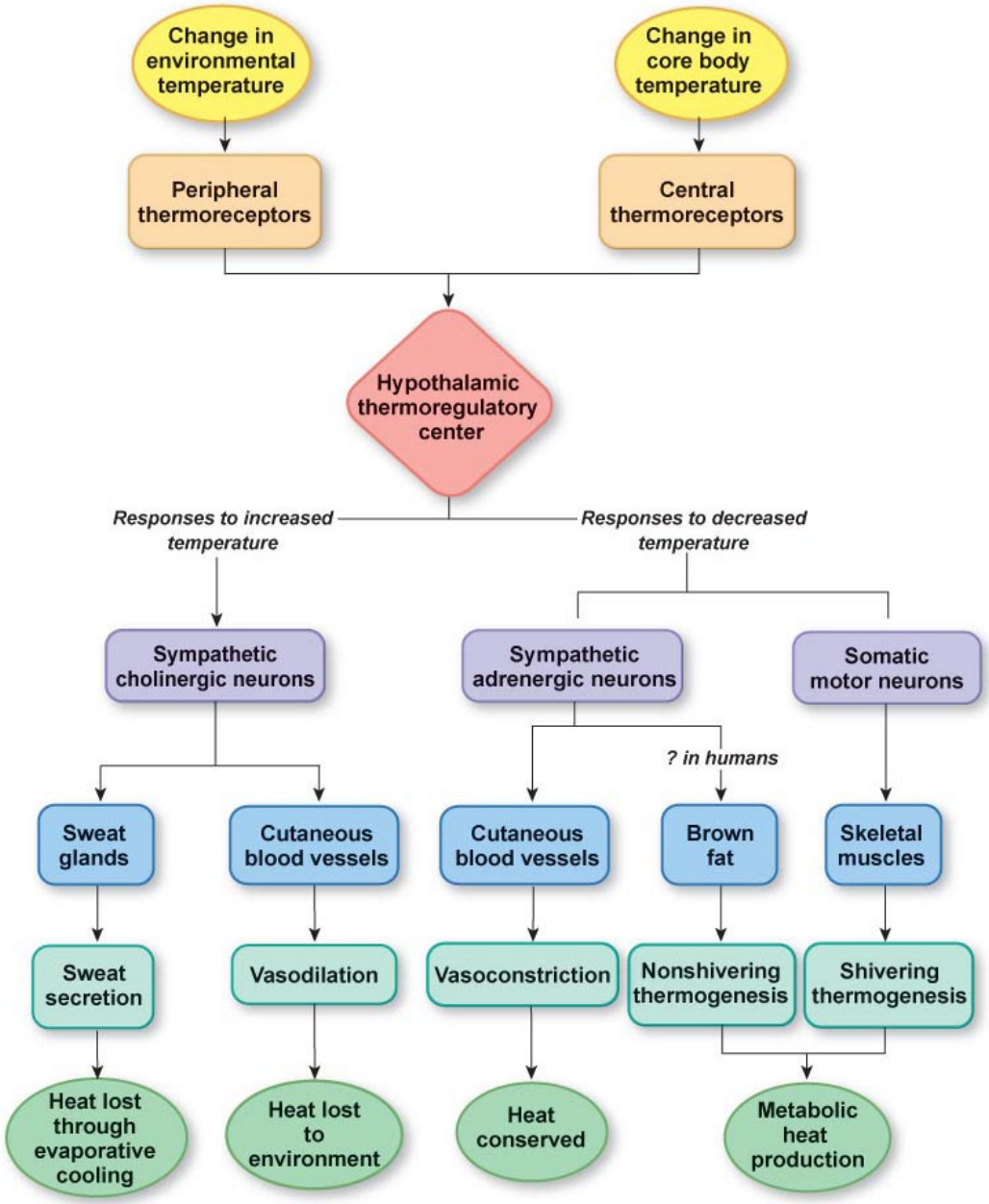


Figure 22-21, overview