

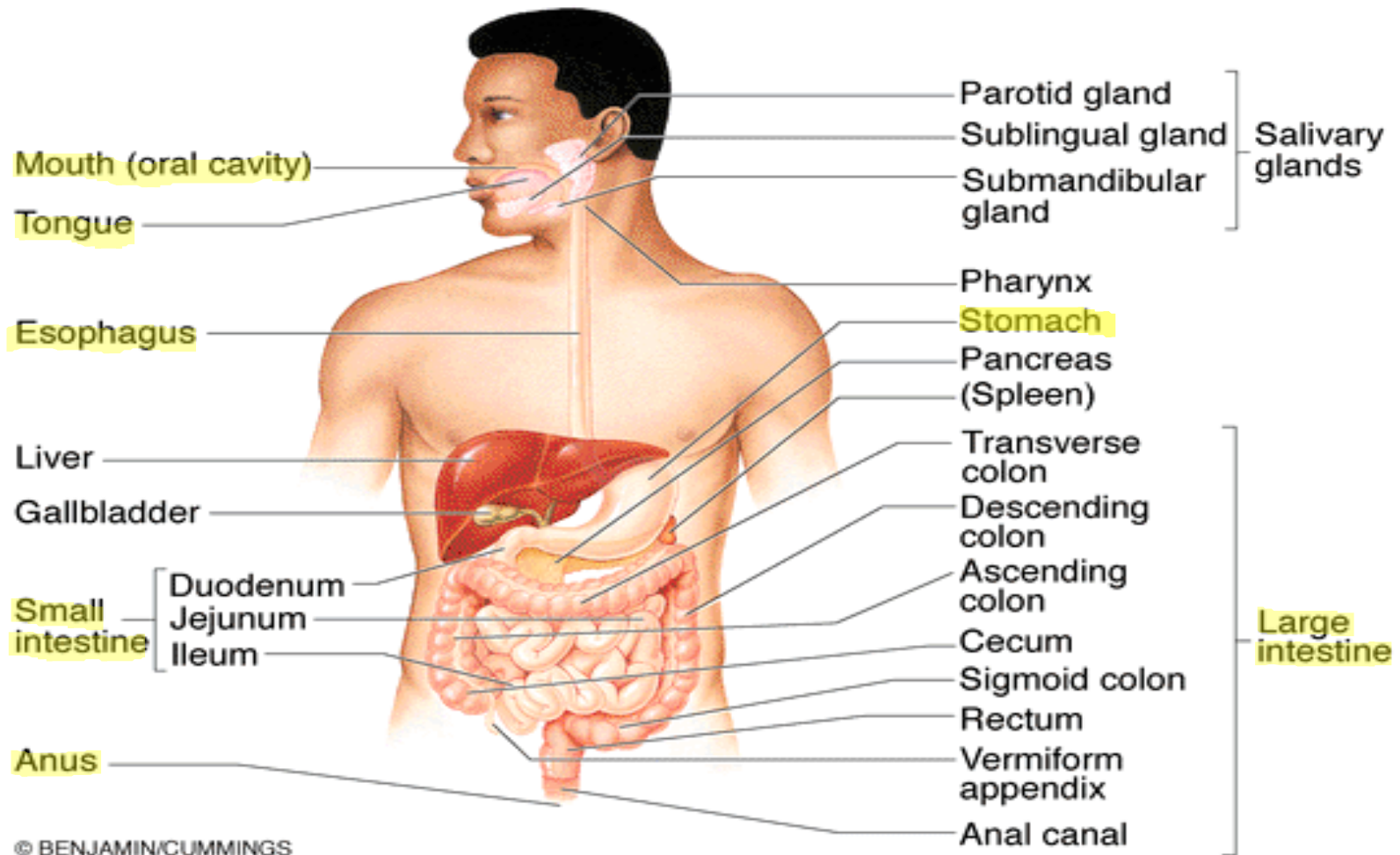
FISIOLOGI SISTEM PENCERNAAN



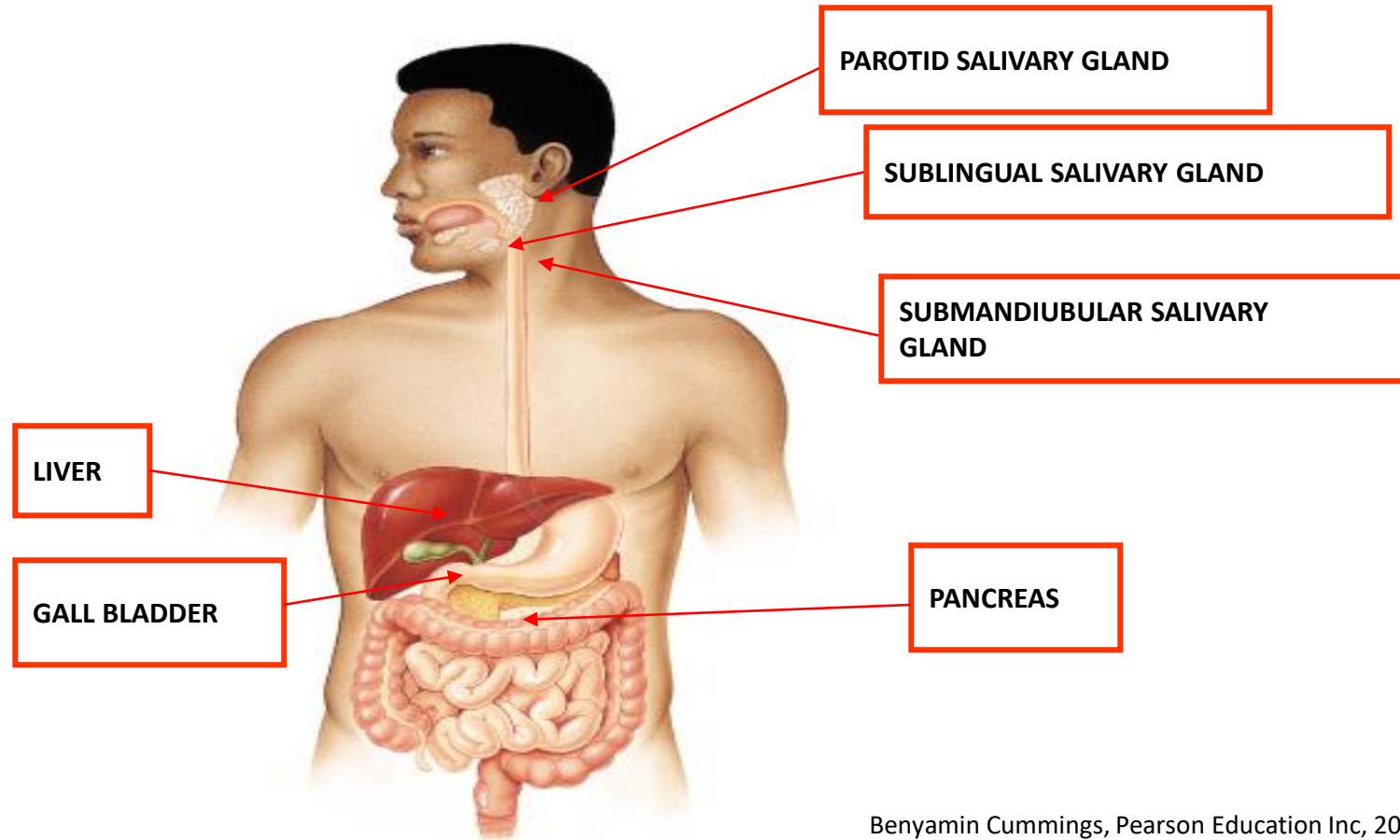
Oleh :

dr. Mustika Anggiane Putri, M.Biomed

Organ Primer



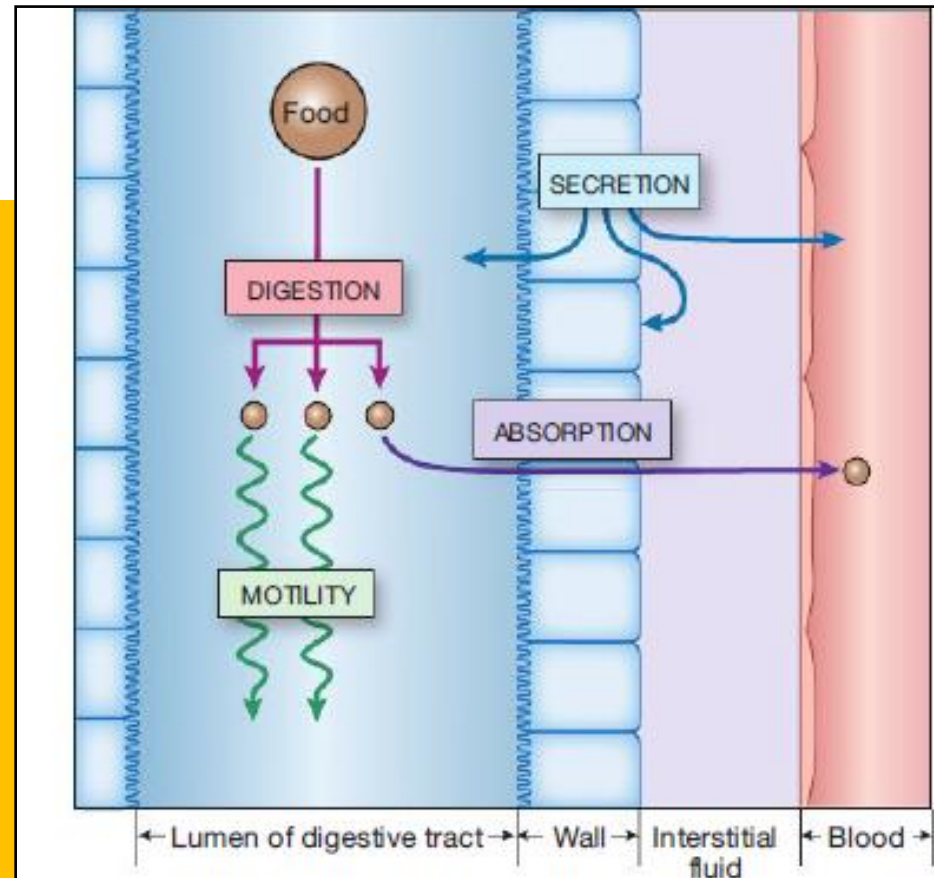
Organ Aksesoris (sekunder) cerna



Benyamin Cummings, Pearson Education Inc, 2004

4 Proses Dasar Pencernaan

1. Motilitas
2. Sekresi
3. Pencernaan
4. Penyerapan



● **FIGURE 21-2** *Four basic processes of the digestive system.* The four digestive system processes are *digestion* of food into smaller units; *absorption* of substances from the lumen into the ECF; *motility*, the movement of materials through the GI tract; and *secretion* of substances from epithelial cells into the lumen or ECF.

1. MOTILITAS

- Kontraksi otot yang mencampur dan mendorong maju isi saluran cerna
- Tonus → mempertahankan tekanan & mencegah dinding saluran cerna teregang permanen setelah mengalami distensi oleh adanya kimus.
- kontraksi otot polos dinding organ pencernaan → **di bawah kontrol involunter**
- Motilitas yg terjadi di ujung saluran cerna (mulut di bagian pangkal esofagus dan sfingter ani eksterus) :
 - ✓ melibatkan otot rangka
 - ✓ di bawah kontrol sadar (**volunter**)
 - ✓ menguyah, menelan, dan defekasi

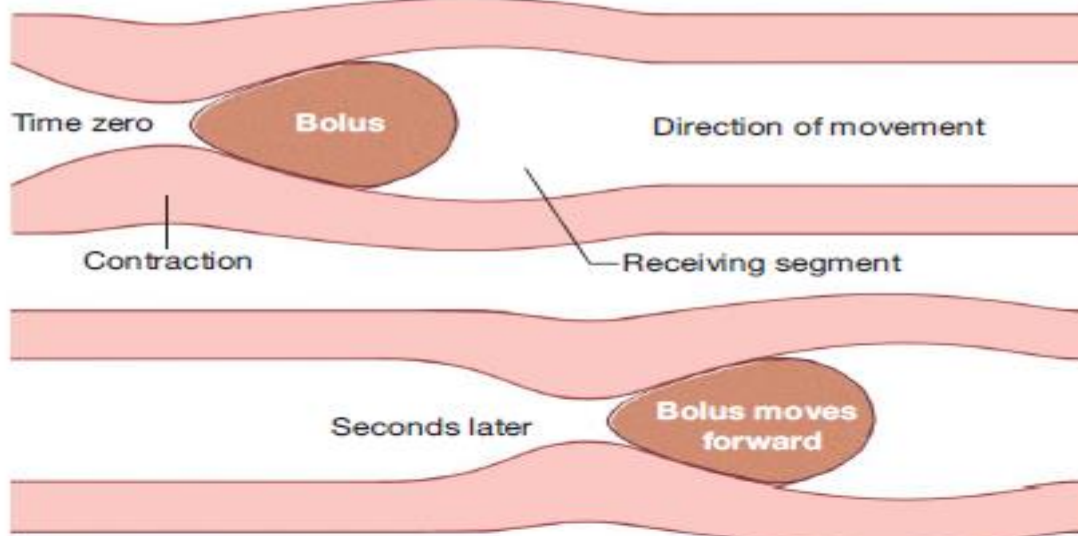
Dua tipe dasar motilitas saluran cerna:

1. Gerakan mendorong (propulsif):

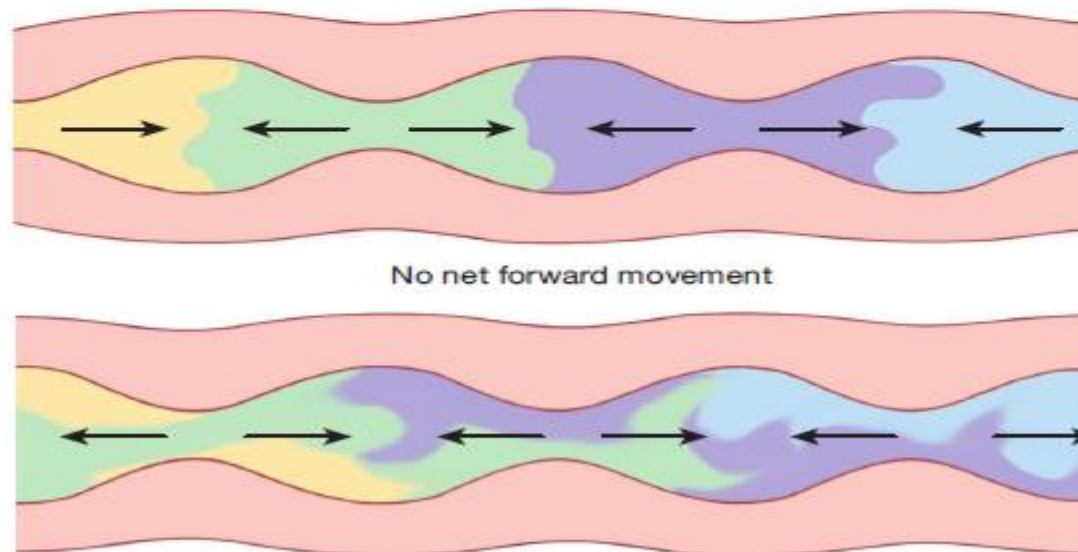
- mendorong maju isi saluran cerna
- Kecepatannya sesuai daerah segmen tertentu shg segmen tersebut dapat melaksanakan tugasnya.
- Contoh:
 - transit makanan di esofagus lebih cepat → fungsinya sbg saluran dari mulut ke lambung.
 - Di usus halus lebih lambat → butuh waktu untuk penguraian & penyerapan makanan.

2. Gerakan mencampur, Mempunyai fungsi ganda:

1. mencampur makanan dengan getah pencernaan → meningkatkan proses pencernaan
2. Memajukan semua bagian isi saluran cerna ke permukaan serap saluran cerna



(a) Peristaltic contractions are responsible for forward movement.



(b) Segmental contractions are responsible for mixing.

● **FIGURE 21-5** *Peristaltic and segmental contractions in the GI tract*

2. SEKRESI

- Proses yang dilakukan kelenjar-kelenjar yang terkait dengan saluran pencernaan
- Sekresi air, elektrolit, enzim, garam empedu atau mukus.
- Sel-sel eksokrin:
 - sekresi getah pencernaan → mempermudah motilitas, melindungi saluran cerna, proses pencernaan
- Sel endokrin
 - terletak pada dinding saluran cerna
 - sekresi hormon pencernaan ke dalam darah → kontrol motilitas pencernaan dan sekresi kelenjar eksokrin

3. PENCERNAAN

- Penguraian biokimiawi
- struktur kompleks makanan menjadi satuan-satuan yang lebih kecil
- oleh enzim-enzim yang diproduksi di dalam saluran pencernaan

→ sehingga dapat diserap oleh sel-sel tubuh.

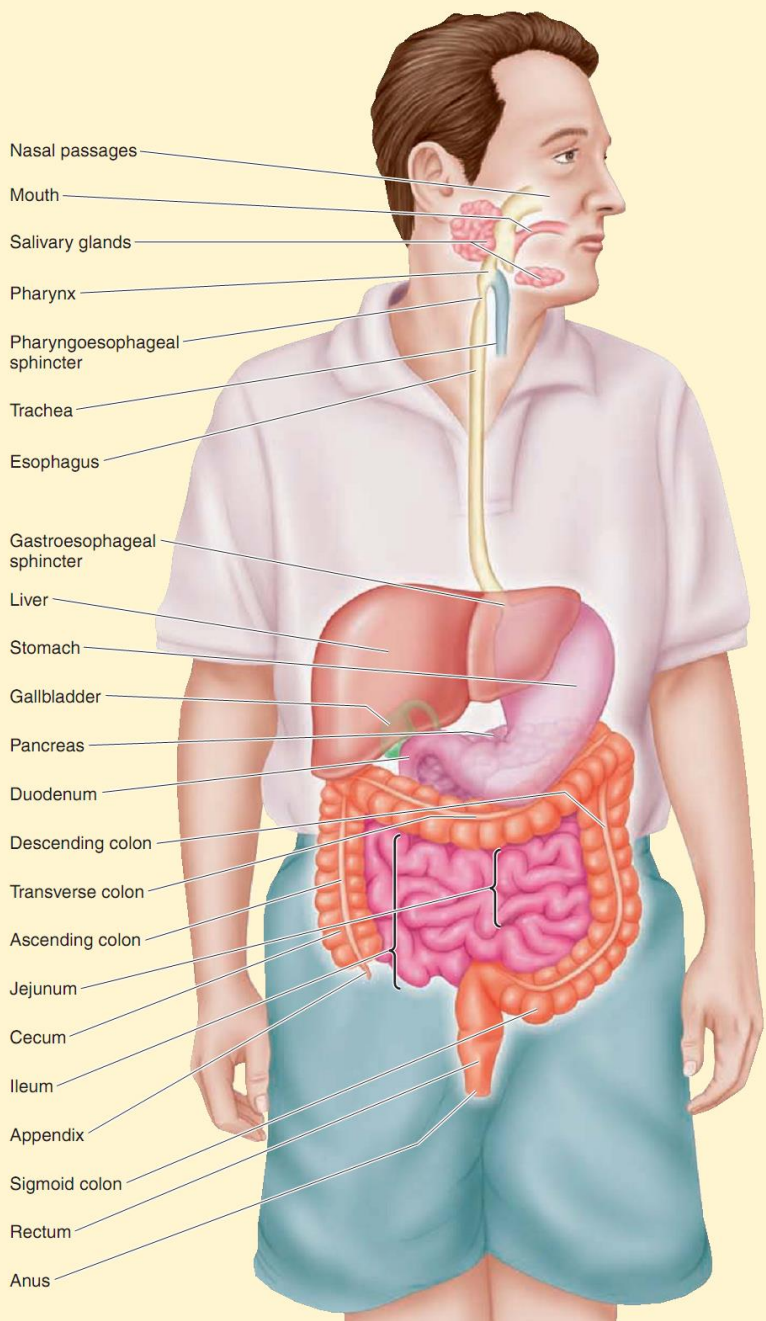
- Bahan makanan yang dikonsumsi manusia (*karbohidrat, protein, lemak*) : molekul besar → tidak dapat diserap dari lumen sal. cerna ke dalam darah atau limfe.

PENYERAPAN

- Di usus halus, pencernaan telah tuntas dan **terjadi sebagian besar penyerapan**
- Unit-unit kecil makanan hasil pencernaan bersama dengan air, vitamin, dan elektrolit dipindahkan dari lumen saluran cerna ke dalam darah atau limfe.

MEKANISME DASAR PENCERNAAN

TABLE 16-1 **Anatomy and Functions of Components of the Digestive System**



Digestive Organ	Motility	Secretion	Digestion	Absorption
Mouth and Salivary Glands	Chewing	Saliva ■ Amylase ■ Mucus ■ Lysozyme	Carbohydrate digestion begins	No foodstuffs; a few medications—for example, nitroglycerin
Pharynx and Esophagus	Swallowing	Mucus	None	None
Stomach	Receptive relaxation; peristalsis	Gastric juice ■ HCl ■ Pepsin ■ Mucus ■ Intrinsic factor	Carbohydrate digestion continues in body of stomach; protein digestion begins in antrum of stomach	No foodstuffs; a few lipid-soluble substances, such as alcohol and aspirin
Exocrine Pancreas	Not applicable	Pancreatic digestive enzymes ■ Trypsin, chymotrypsin, carboxypeptidase ■ Amylase ■ Lipase Pancreatic aqueous NaHCO_3 secretion	These pancreatic enzymes accomplish digestion in duodenal lumen	Not applicable
Liver	Not applicable	Bile ■ Bile salts ■ Alkaline secretion ■ Bilirubin	Bile does not digest anything, but bile salts facilitate fat digestion and absorption in duodenal lumen	Not applicable
Small Intestine	Segmentation; migrating motility complex	Succus entericus ■ Mucus ■ Salt (Small intestine enzymes—disaccharidases and aminopeptidases—are not secreted but function within the brush-border membrane)	In lumen, under influence of pancreatic enzymes and bile, carbohydrate and protein digestion continues and fat digestion is completely accomplished; in brush border, carbohydrate and protein digestion completed	All nutrients, most electrolytes, and water
Large Intestine	Haustral contractions, mass movements	Mucus	None	Salt and water, converting contents to feces

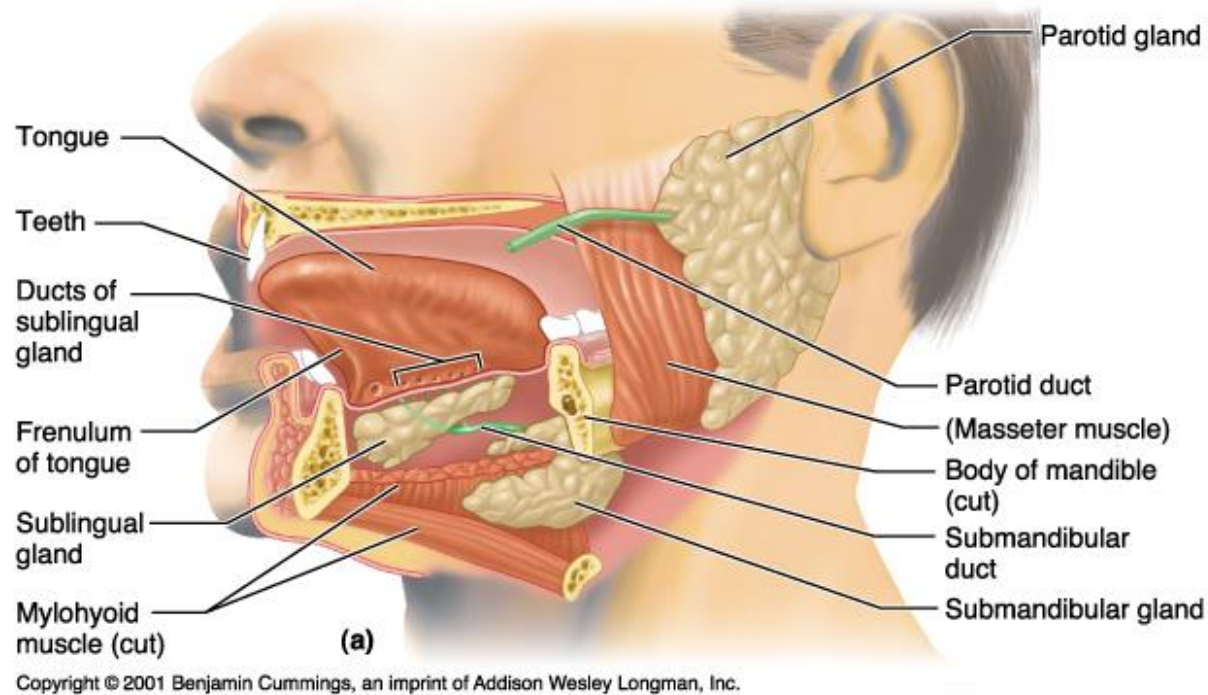
(Sherwood, 2009)

MULUT

1. Motilitas pada mulut

- Bibir → membantu mengambil, menuntun, dan menampung makanan di mulut
- Langit-langit (palatum) → memisahkan mulut dari saluran hidung → memungkinkan bernapas dan mengunyah atau menghisap berlangsung secara bersamaan
- Lidah → membentuk dasar rongga mulut → menuntun makanan di dalam mulut sewaktu mengunyah dan menelan
- Gigi → mengunyah (pengirisan, perobekan, penggilingan dan pencampuran makanan)

2. Sekresi pada mulut



- Kelenjar parotid → sekresi serosa (mengandung ptialin)
- kelenjar submandibular dan sublingual → sekresi mukus (mengandung mucin) dan sekresi serosa
- Kelenjar bukalis → sekresi mukus

Liur mengandung 99,5% air dan 0,5% elektrolit serta protein (terutama amilase, mukus, dan lisozim)

- Fungsi liur:
 - Amilase mengurai polisakarida menjadi disakarida (maltosa)
 - Mempermudah proses menelan
 - Lisozim sebagai antibakteri
 - Bahan pelarut yang merangsang kuncup kecap
 - Mempermudah gerakan bibir dan lidah
 - Membantu membilas residu makanan, partikel asing, dan sel epitel tua
 - Dapar bikarbonat → menetralkan asam dalam makanan serta asam yang dihasilkan bakteri

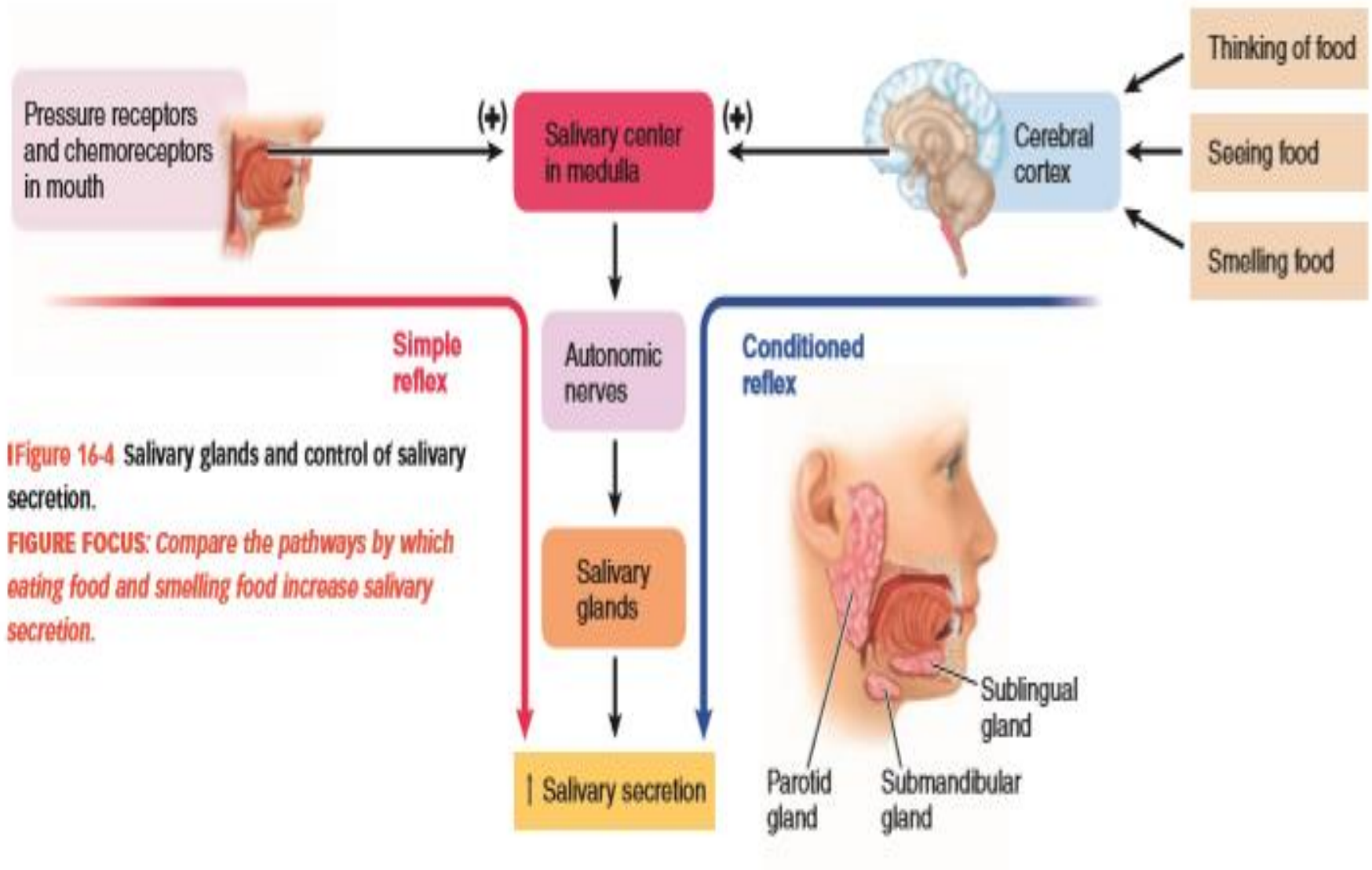
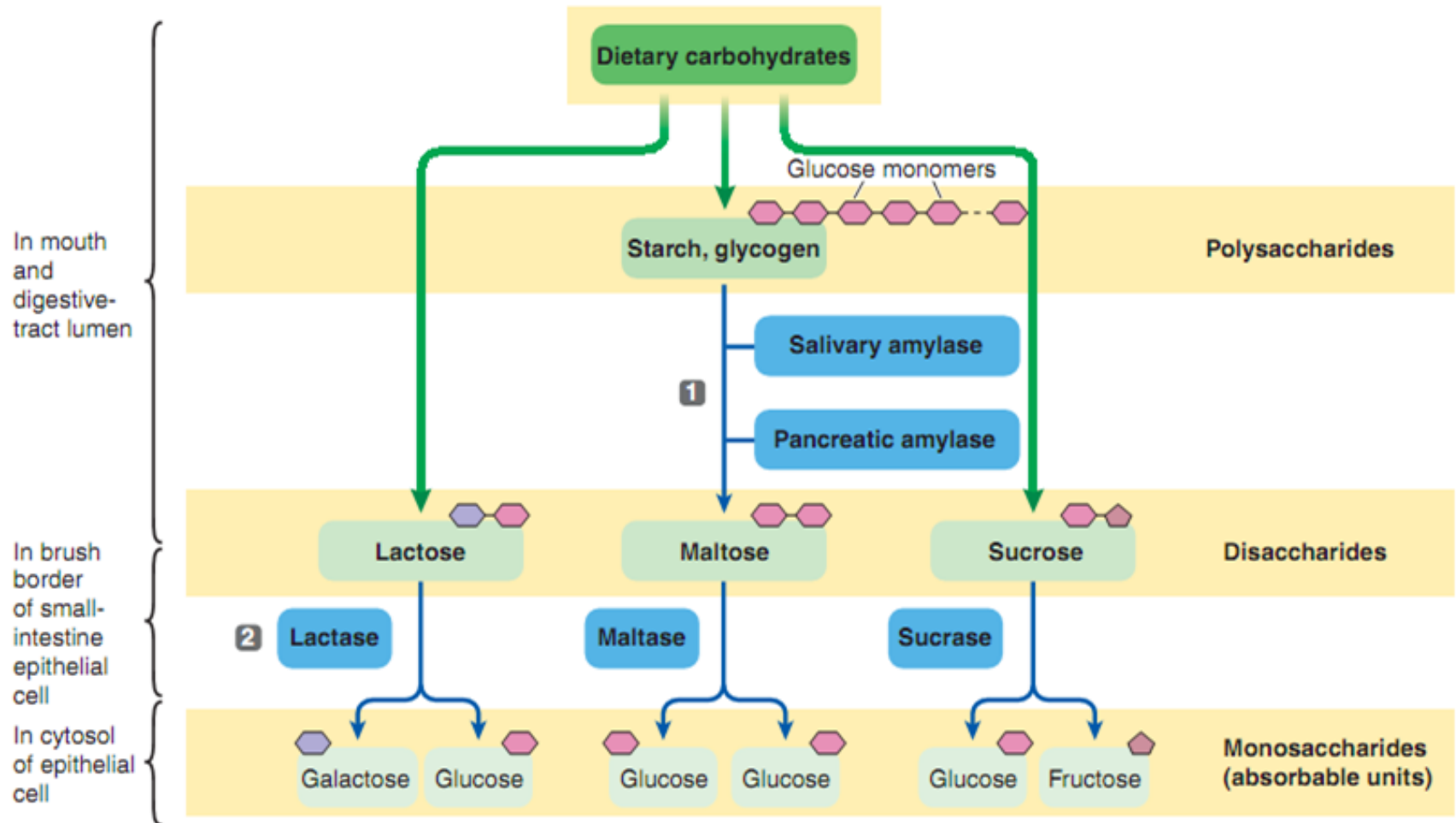


Figure 16-4 Salivary glands and control of salivary secretion.

FIGURE FOCUS: Compare the pathways by which eating food and smelling food increase salivary secretion.

3. Pencernaan pada mulut

Hidrolisis polisakarida menjadi disakarida oleh amilase



(a) Carbohydrate digestion

(Sherwood, 2009)

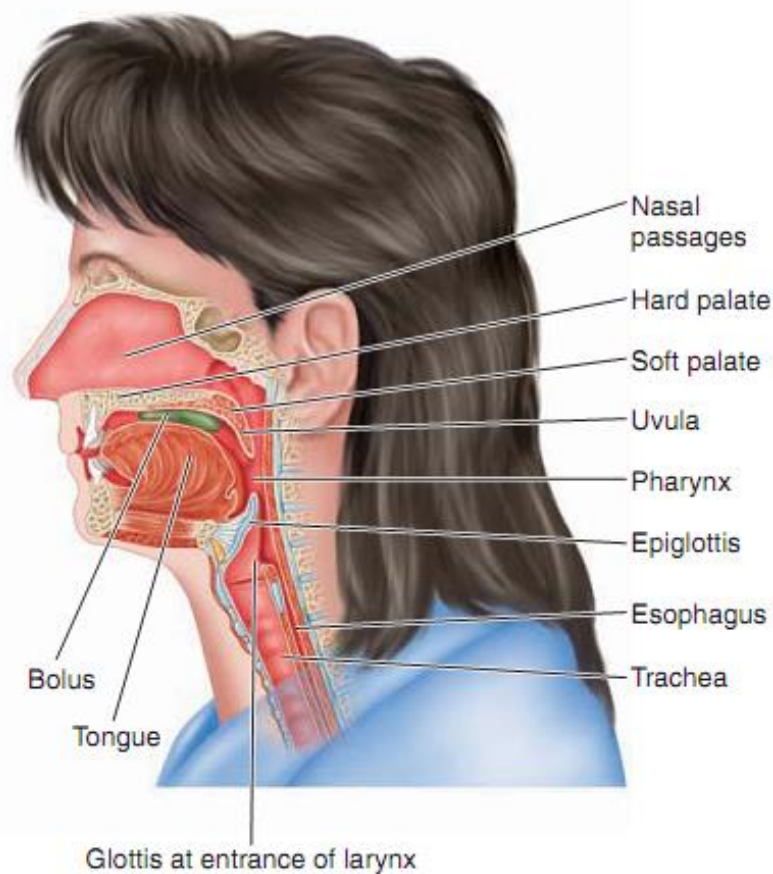
4. Penyerapan pada mulut

- Tidak terjadi penyerapan makanan di mulut
- Beberapa obat dapat diserap oleh mukosa oral, contoh nitrogliserin

FARING DAN ESOFAGUS

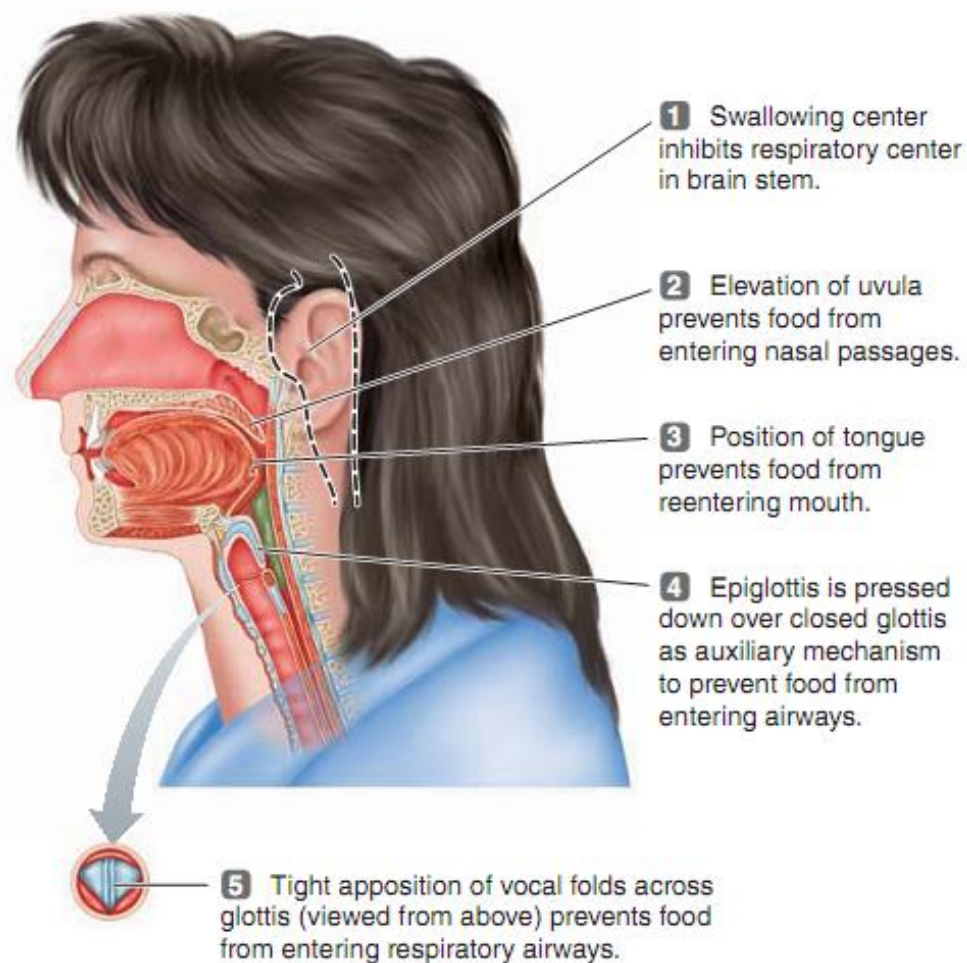
1. Motilitas pada faring dan esofagus

- Motilitas yang berkaitan dengan faring dan esofagus → menelan
- Menelan → keseluruhan proses memindahkan makanan dari mulut melalui esofagus hingga ke lambung
- Menelan dibagi menjadi 2 tahap:
 - tahap orofaring
 - tahap esofagus



(a) Position of the oropharyngeal structures at rest

● **FIGURE 16-5 Oropharyngeal stage of swallowing.**



(b) Changes during the oropharyngeal stage of swallowing to prevent food from entering the wrong passageways

2. Sekresi pada faring dan esofagus

Terdiri dari mukus, berfungsi untuk:

- pelumas mengurangi kemungkinan kerusakan esofagus oleh tepi-tepi tajam makanan yang baru masuk
- melindungi dinding esofagus dari asam dan enzim di getah lambung jika terjadi refluks lambung

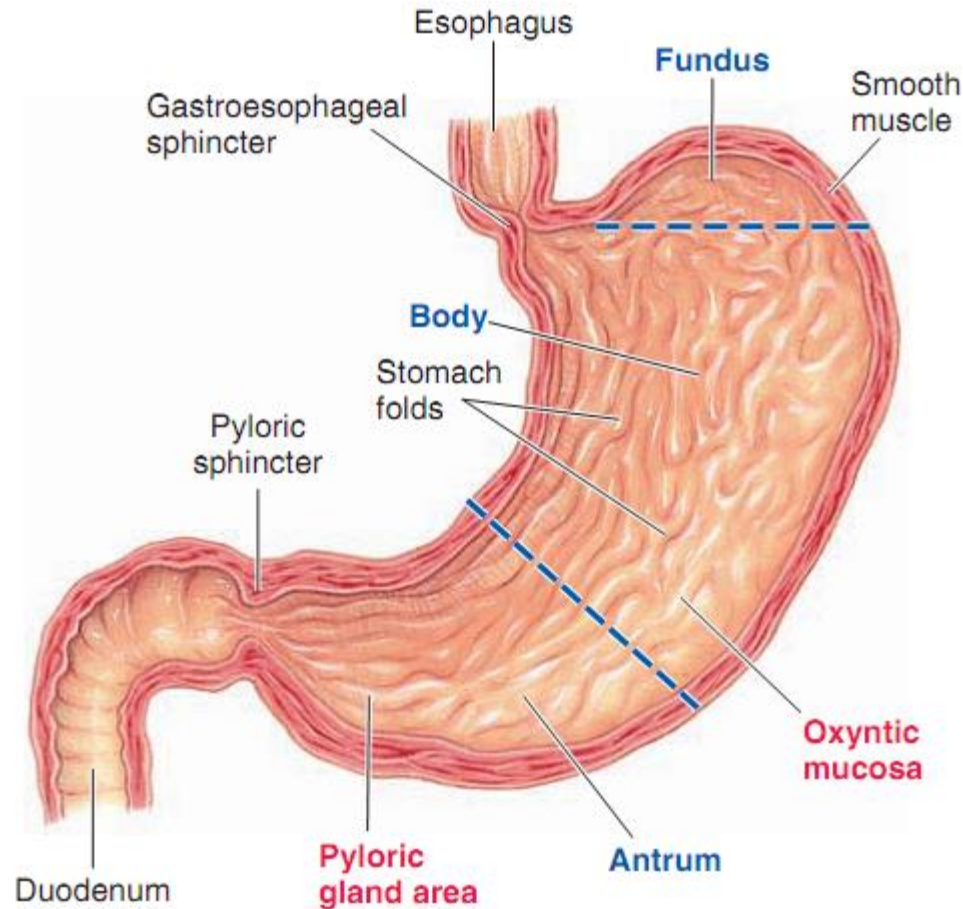
Keseluruhan waktu transit di faring dan esofagus hanya sekitar 6-10 detik terlalu singkat untuk terjadi pencernaan atau penyerapan

LAMBUNG

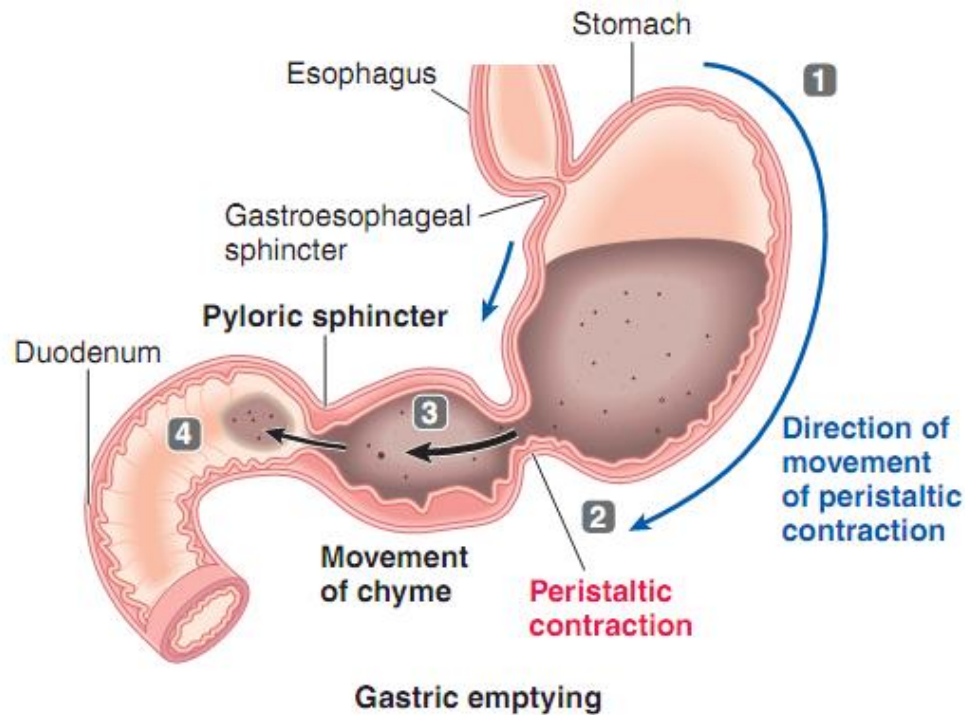
1. Motilitas pada lambung

4 motilitas lambung:

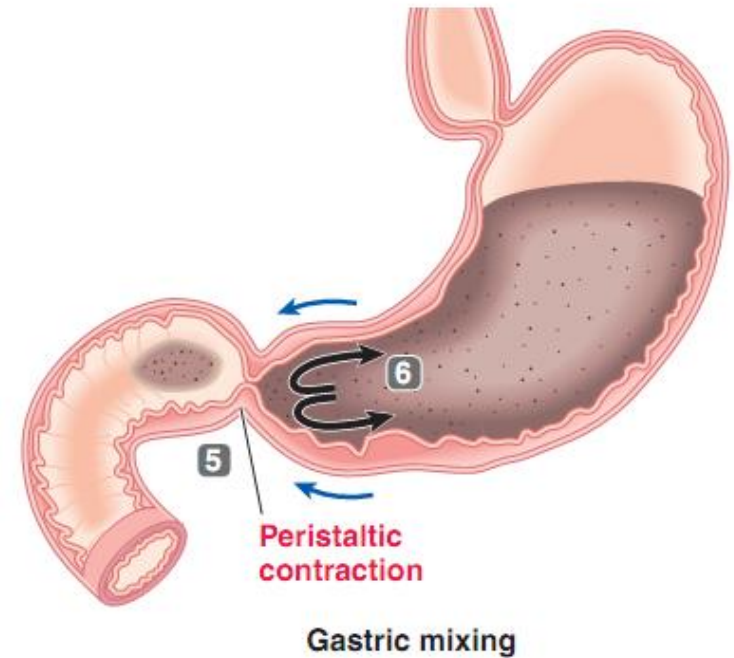
- **Pengisian** → relaksasi reseptif lipatan2 interior lambung mjd lebih kecil/datar → kapasitas lambung meningkat.
- **Penyimpanan** → makanan disimpan di korpus yang relatif tenang, tanpa mengalami pencampuran.
- **Pencampuran** → kontraksi peristaltik antrum mencampur makanan + sekresi lambung → **kimus**
- **Pengosongan** → kontraksi peristaltik antrum, gaya pendorong utk mengosongkan lambung



- **FIGURE 16-7 Anatomy of the stomach.** The stomach is divided into three sections based on structural and functional distinctions—the fundus, body, and antrum. The mucosal lining of the stomach is divided into the oxyntic mucosa and the pyloric gland area based on differences in glandular secretion.



- 1** A peristaltic contraction originates in the upper fundus and sweeps down toward the pyloric sphincter.
- 2** The contraction becomes more vigorous as it reaches the thick-musled antrum.
- 3** The strong antral peristaltic contraction propels the chyme forward.
- 4** A small portion of chyme is pushed through the partially open sphincter into the duodenum. The stronger the antral contraction, the more chyme is emptied with each contractile wave.



- 5** When the peristaltic contraction reaches the pyloric sphincter, the sphincter is tightly closed and no further emptying takes place.
- 6** When chyme that was being propelled forward hits the closed sphincter, it is tossed back into the antrum. Mixing of chyme is accomplished as chyme is propelled forward and tossed back into the antrum with each peristaltic contraction.

● **FIGURE 16-8** Gastric emptying and mixing as a result of antral peristaltic contractions.

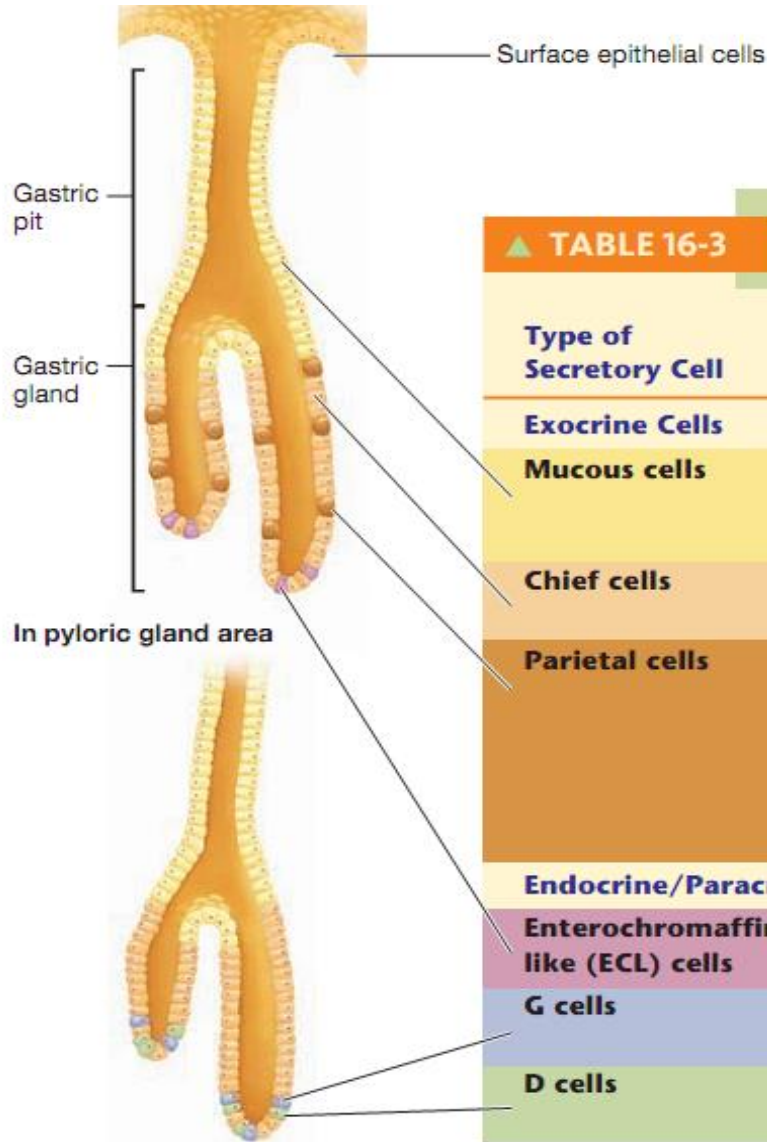
▲ TABLE 16-2

Factors Regulating Gastric Motility and Emptying

Factors	Mode of Regulation	Effects on Gastric Motility and Emptying
Within the Stomach		
Volume of chyme	Distension has a direct effect on gastric smooth muscle excitability, as well as acting through the intrinsic plexuses, the vagus nerve, and gastrin	Increased volume stimulates motility and emptying
Degree of fluidity	Direct effect; contents must be in a fluid form to be evacuated	Increased fluidity allows more rapid emptying
Within the Duodenum		
Presence of fat, acid, hypertonicity, or distension	Initiates the enterogastric reflex or triggers the release of enterogastrones (secretin, cholecystikinin)	These factors in the duodenum inhibit further gastric motility and emptying until the duodenum has coped with factors already present
Outside the Digestive System		
Emotion	Alters autonomic balance	Stimulates or inhibits motility and emptying
Intense pain	Increases sympathetic activity	Inhibits motility and emptying

(Sherwood, 2009)

2. Sekresi pada lambung



▲ TABLE 16-3

The Stomach Mucosa and the Gastric Glands

Type of Secretory Cell	Product Secreted	Stimuli for Secretion	Function(s) of Secretory Product
Exocrine Cells			
Mucous cells	Alkaline mucus	Mechanical stimulation by contents	Protects mucosa against mechanical, pepsin, and acid injury
Chief cells	Pepsinogen	ACh, gastrin	When activated, begins protein digestion
Parietal cells	Hydrochloric acid	ACh, gastrin, histamine	Activates pepsinogen, breaks down connective tissue, denatures proteins, kills microorganisms
	Intrinsic factor		Facilitates absorption of vitamin B ₁₂
Endocrine/Paracrine Cells			
Enterochromaffin-like (ECL) cells	Histamine	ACh, gastrin	Stimulates parietal cells
G cells	Gastrin	Protein products, ACh	Stimulates parietal, chief, and ECL cells
D cells	Somatostatin	Acid	Inhibits parietal, G, and ECL cells

(Sherwood)

Kontrol sekresi lambung

(Guyton)

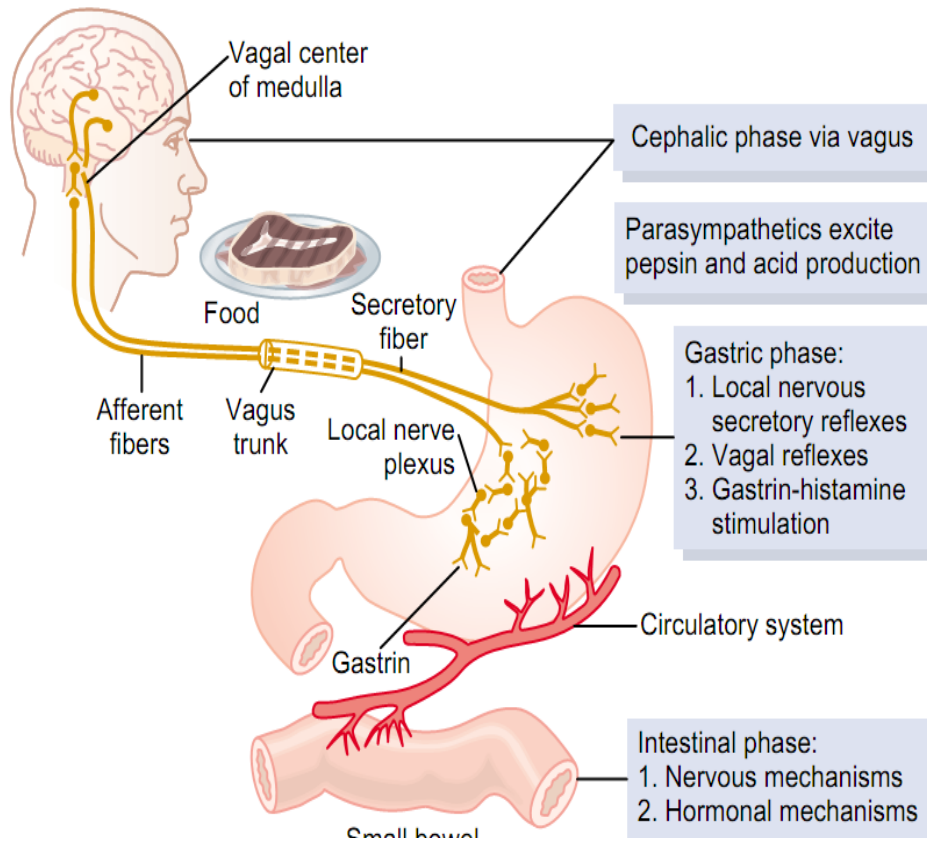


Figure 64-7

(Sherwood)

▲ TABLE 16-4

Stimulation of Gastric Secretion

Phase	Stimuli	Excitatory Mechanism for Enhancing Gastric Secretion				
Cephalic Phase of Gastric Secretion	Stimuli in the head—seeing, smelling, tasting, chewing, swallowing food	+ Vagus	+ Intrinsic nerves	→ ↑ACh	+ Chief and parietal cells	→ ↑Gastric secretion
			+ G cells	→ ↑Gastrin	+ ECL cells → ↑Histamine	
Gastric Phase of Gastric Secretion	Stimuli in the stomach—protein, (peptide fragments), distension, caffeine, alcohol	+ Vagus	+ Intrinsic nerves	→ ↑ACh	+ Chief and parietal cells	→ ↑Gastric secretion
			+ G cells	→ ↑Gastrin	+ ECL cells → ↑Histamine	

▲ TABLE 16-5

Inhibition of Gastric Secretion

Region	Stimuli	Inhibitory Mechanism for Gastric Secretion
Body and Antrum	Removal of protein and distension as the stomach empties	<pre> graph LR S[Removal of protein and distension as the stomach empties] --> I1[Intrinsic nerves] S --> I2[Vagus] S --> I3[G cells] I3 --> G[↓Gastrin] G --> H[↓Histamine] I1 --> GS[↓Gastric secretion] I2 --> GS I3 --> GS G --> GS H --> GS </pre>
Antrum and Duodenum	Accumulation of acid	<pre> graph LR S[Accumulation of acid] --> D[D cells] D --> Soma[↑Somatostatin] Soma --> P[Parietal cells] Soma --> G[G cells] Soma --> E[ECL cells] P --> GS[↓Gastric secretion] G --> GS E --> GS </pre>
Duodenum (Intestinal Phase of Gastric Secretion)	Fat Acid Hypertonicity Distension	<pre> graph LR S[Fat, Acid, Hypertonicity, Distension] --> ER[Enterogastric reflex] S --> EG[↑Enterogastrones (cholecystikinin and secretin)] EG --> P[Parietal cells] EG --> C[Chief cells] EG --> SM[Smooth muscle cells] ER --> GS[↓Gastric secretion and motility] P --> GS C --> GS SM --> GS </pre>

(Sherwood,)

3. Pencernaan pada lambung

- Korpus lambung → makanan belum tercampur dgn sekresi lambung
- pencernaan karbohidrat berlanjut di bawah pengaruh amilase liur
- Antrum lambung → makanan dicampur merata dengan HCl dan pepsin (**awal pencernaan protein**)

(Sherwood)

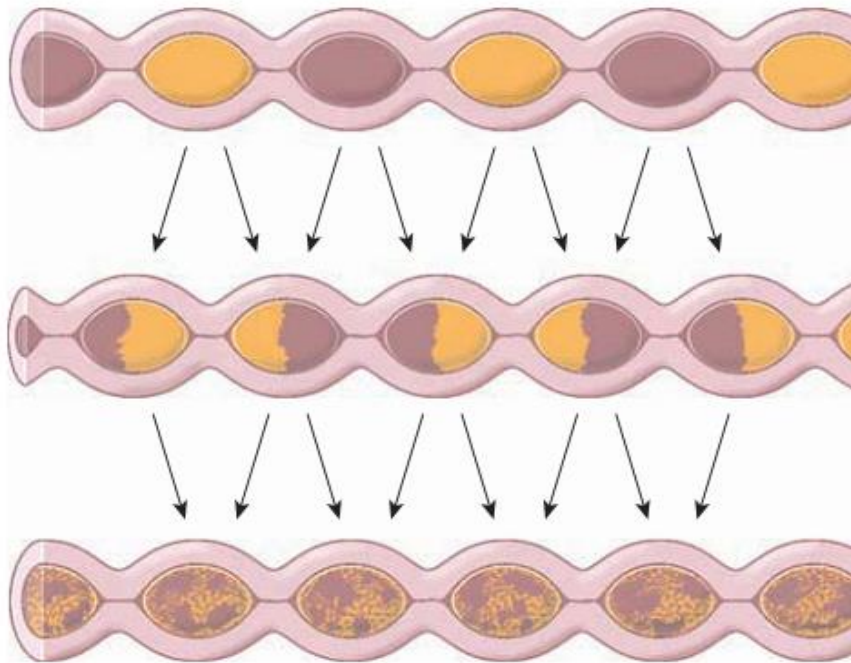
4. Penyerapan pada lambung

- Tidak ada makanan atau air yang diserap ke dalam darah melalui mukosa lambung
- Etil alkohol (bersifat agak larut lemak) dan aspirin (asam lemah) dapat diserap langsung oleh lambung

USUS HALUS

1. Motilitas pada usus halus

Motilitas usus halus mencakup: segmentasi dan *migrating motility complex*



● **FIGURE 16-19 Segmentation.** Segmentation consists of ring-like contractions along the length of the small intestine. Within a matter of seconds, the contracted segments relax and the previously relaxed areas contract. These oscillating contractions thoroughly mix the chyme within the small-intestine lumen.
(Sherwood, 2009)

Fungsi segmentasi:

- Mencampur kimus dengan getah pencernaan
- Memajukan semua kimus ke permukaan absorptif mukosa usus halus

migrating motility complex menyapu usus dengan gerakan peristaltik lemah berulang

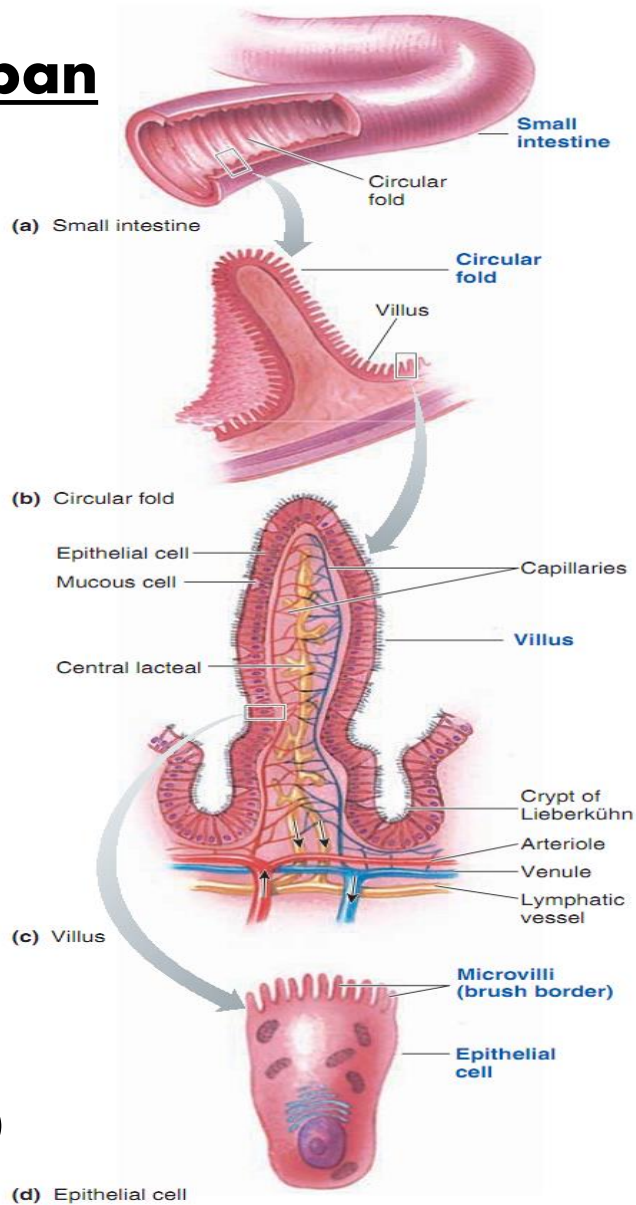
2. Sekresi pada usus halus

- sekresi mukus enterikus → melindungi dan melumasi dinding usus halus
- sintesis enzim pencernaan (enterokinase, disakaridase, aminopeptidase) berfungsi di dalam membran *brush border* sel epitel.

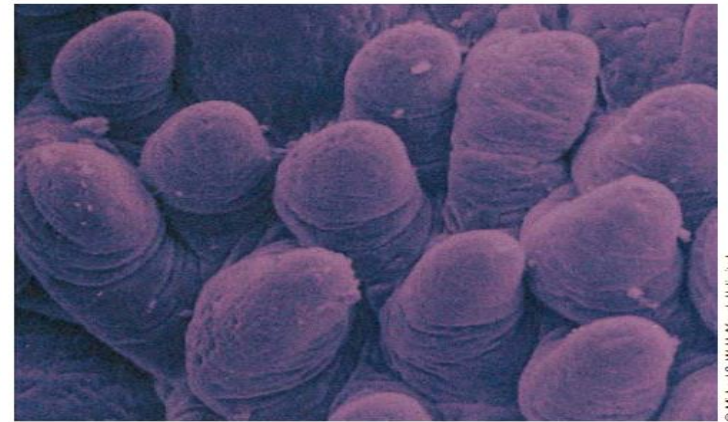
3. Pencernaan pada usus halus

- dilakukan oleh **enzim pankreas** dan **sekresi empedu**
- Pencernaan karbohidrat dan protein dituntaskan di *brush border*

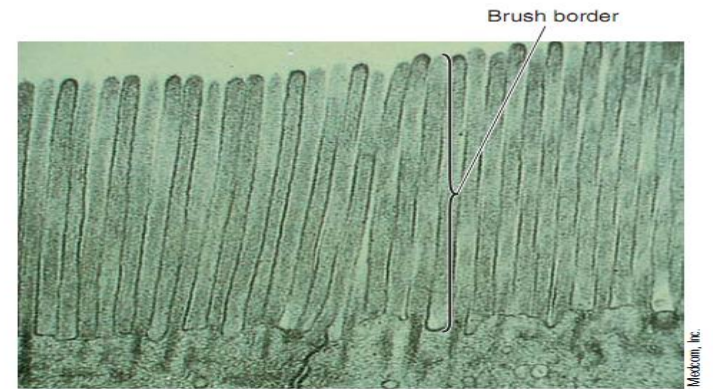
4. Penyerapan pada usus halus



- **FIGURE 16-21 Small-intestine absorptive surface.** (a) Gross structure of the small intestine. (b) The circular folds of the small-intestine mucosa collectively increase the absorptive surface area threefold. (c) Microscopic fingerlike projections known as villi collectively increase the surface area another 10-fold. (d) Each epithelial cell on a villus has microvilli on its luminal border; the microvilli increase the surface area another 20-fold. Together, these surface modifications increase the small intestine's absorptive surface area 600-fold.



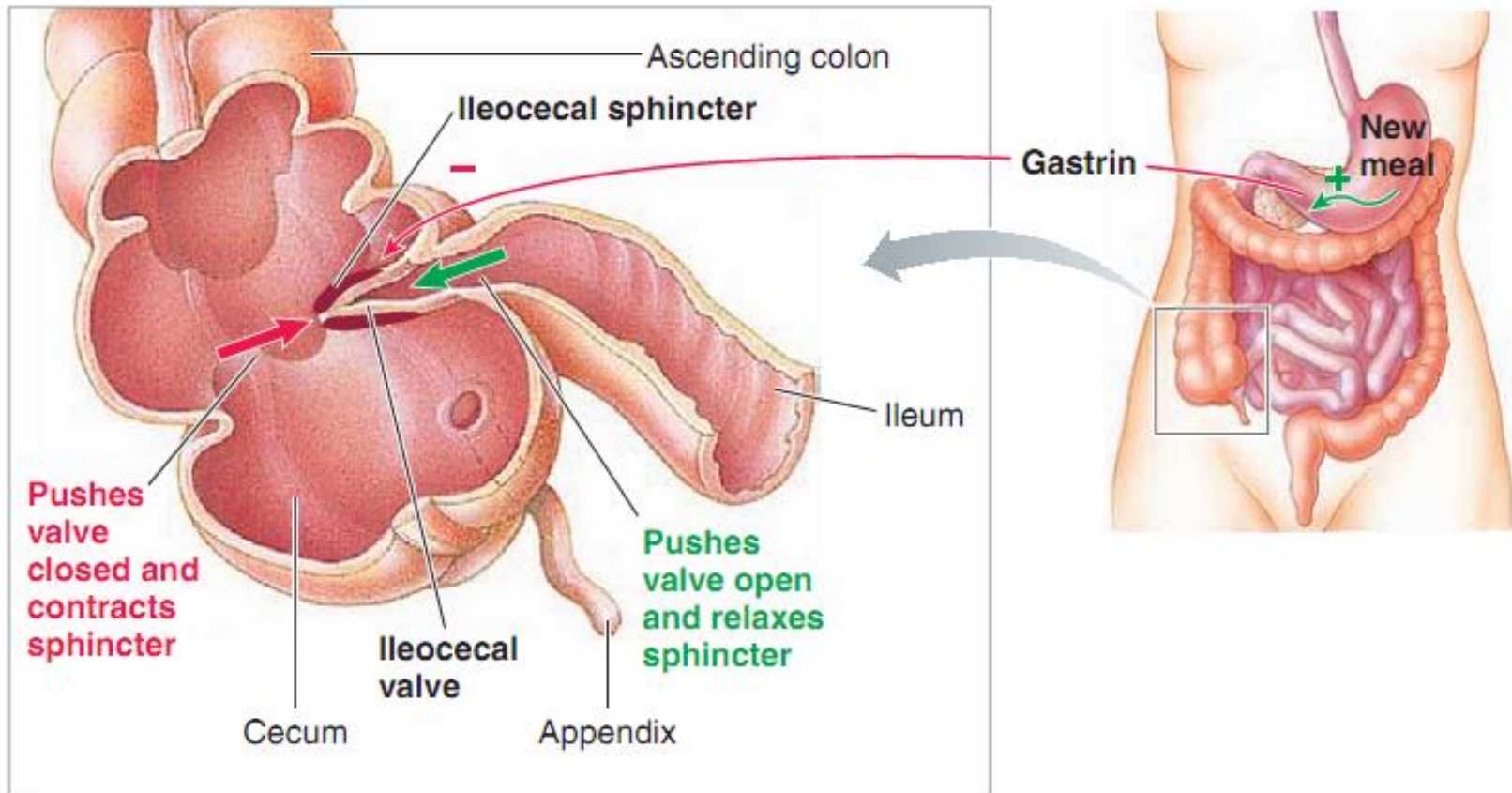
- **FIGURE 16-22 Scanning electron micrograph of villi projecting from the small-intestine mucosa.**



- **FIGURE 16-23 Reduction in the brush border with gluten enteropathy.** (a) Electron micrograph of the brush border of a small-intestine epithelial cell in a normal individual. (b) Electron micrograph of the short, stubby brush border of a small-intestine epithelial cell in a patient with gluten enteropathy.

(Source: Thomas W. Sheehy, M.D.; Robert L. Slaughter, M.D.: *The Malabsorption Syndrome* by Medcom, Inc. Reproduced by permission of Medcom, Inc.)

(Sherwood, 2009)



- FIGURE 16-20 Control of the ileocecal valve/sphincter.** The juncture between the ileum and large intestine is the ileocecal valve, which is surrounded by thickened smooth muscle, the ileocecal sphincter. Pressure on the cecal side pushes the valve closed and contracts the sphincter, preventing the bacteria-laden colonic contents from contaminating the nutrient-rich small intestine. The valve/sphincter opens and allows ileal contents to enter the large intestine in response to pressure on the ileal side of the valve and to the hormone gastrin secreted as a new meal enters the stomach.

(Sherwood)

USUS BESAR

1. Motilitas pada usus besar

1. Motilitas utama kolon adalah **kontraksi haustra**

Kontraksi haustra mengaduk sehingga isi kolon terpajan ke mukosa penyerapan.

2. **Gerakan massa** (kontraksi masif segmen besar kolon ascendens & transversum) → mendorong isi kolon ke distal usus besar → disimpan sampai terjadi defekasi.

2. Sekresi pada usus besar

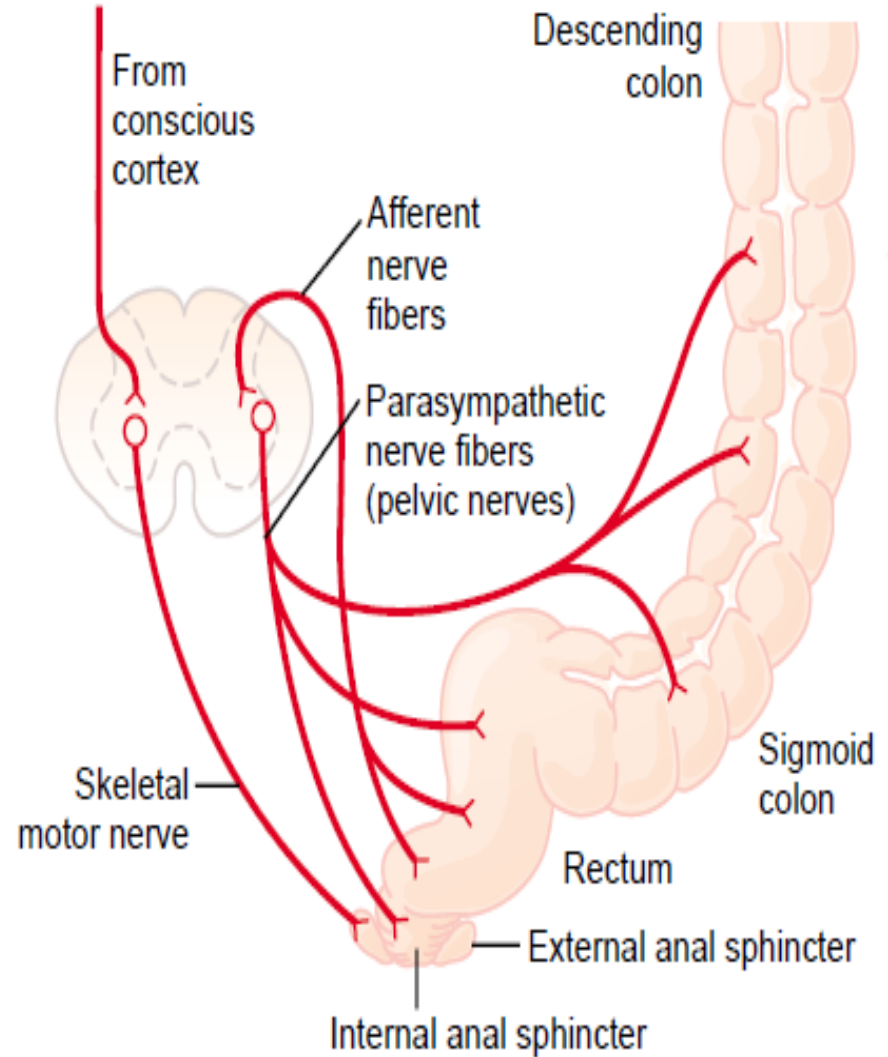
- Tidak mensekresi enzim pencernaan
- Mukus basa (NaHCO_3) :
 - melindungi mukosa usus besar dari cedera mekanis dan kimiawi
 - pelumas untuk mempermudah pergerakan feses
 - menetralkan asam-asam iritan yang diproduksi oleh fermentasi bakteri lokal

3. Pencernaan dan penyerapan pada usus besar

- Tidak terjadi pencernaan di usus besar
- Usus besar menyerap garam dan air → **feses**

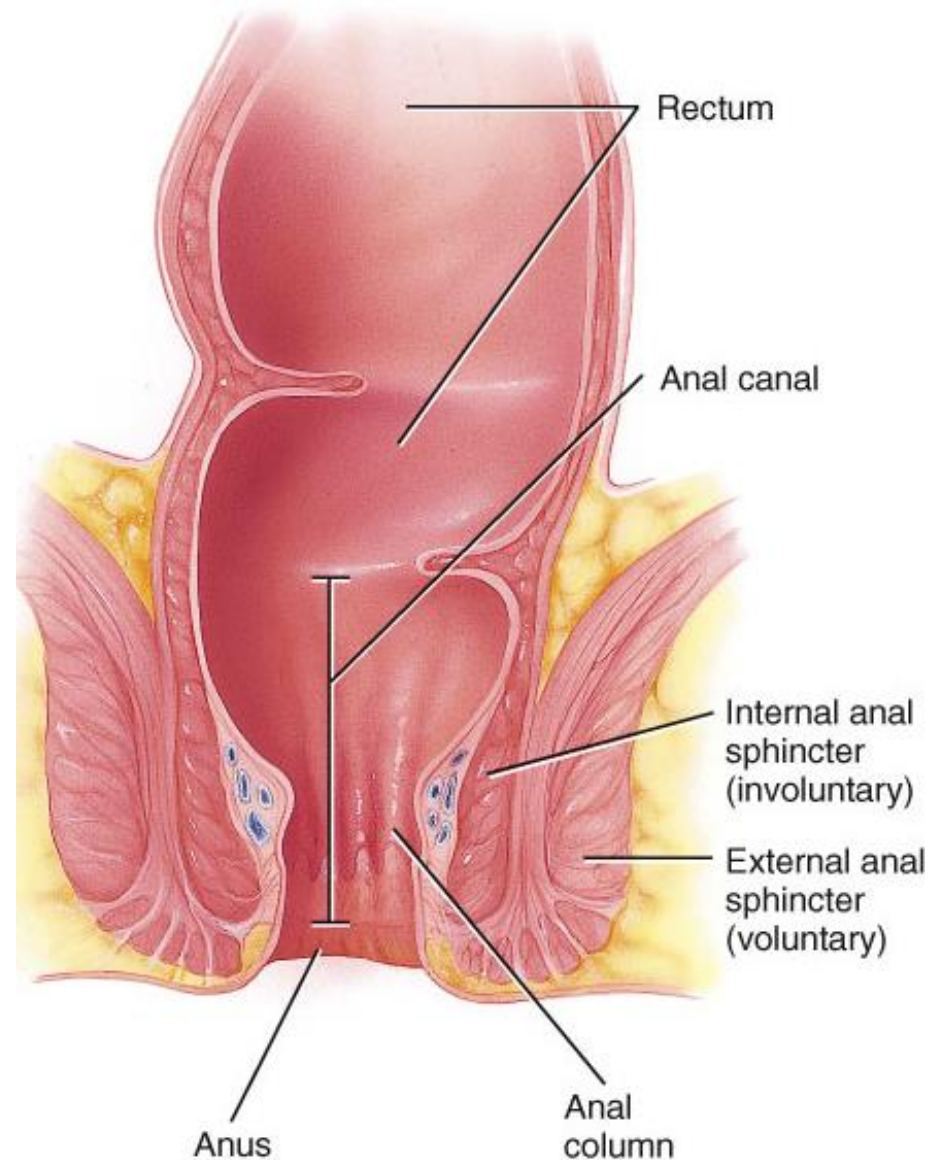
DEFEKASI

- Gerakan massa di kolon mendorong tinja ke dalam rektum.
- Peregangan di rektum merangsang **reseptor regang** di dinding rektum memicu **refleks defekasi**
- Refleks ini menyebabkan **sfingter ani internus** melemas dan **rektum & kolon sigmoid** berkontraksi lebih kuat
- Jika **sfingter ani eksternus** juga melemas maka terjadilah **defekasi**.
- Gerakan mengejan volunter → melibatkan kontraksi otot abdomen dan ekspirasi paksa → me↑ TIA agar membantu mendorong tinja.



Defecation

- Tonic constriction by:
 - **Internal anal sphincter (circular smooth muscle)**
 - **External anal sphincter (striated voluntary muscle)**
 - Controlled by pudendal nerve (somatic NS)
 - Voluntary control



Defekasi

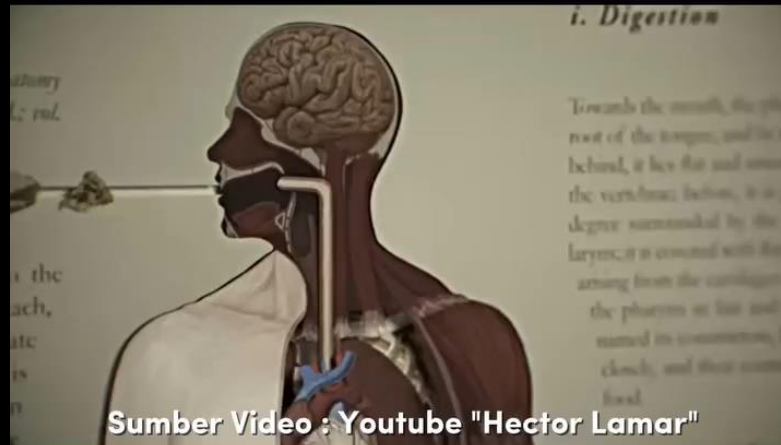
- Ketika gerakan massa di kolon mendorong tinja ke dalam rektum
- Peregangan yang tjd di rektum merangsang **reseptor regang** di dinding rektum memicu **refleks defekasi**
- Refleks ini menyebabkan **sfingter ani internus** melemas dan **rektum dan kolon sigmoid** berkontraksi lebih kuat
- Jika **sfingter ani eksternus** juga melemas maka terjadilah **defekasi**

Konstipasi

- Jika defekasi ditunda, dinding rektum yg semula teregang, sec perlahan melemas dan keinginan utk BAB mereda (smp tjd **pergerakan massa** brktnya)
- Jika ditunda terlalu lama dpt tjd **konstipasi** (sembelit)
- Maka H₂O yang diserap dari tinja meningkatkan sehingga tinja menjadi kering dan keras.

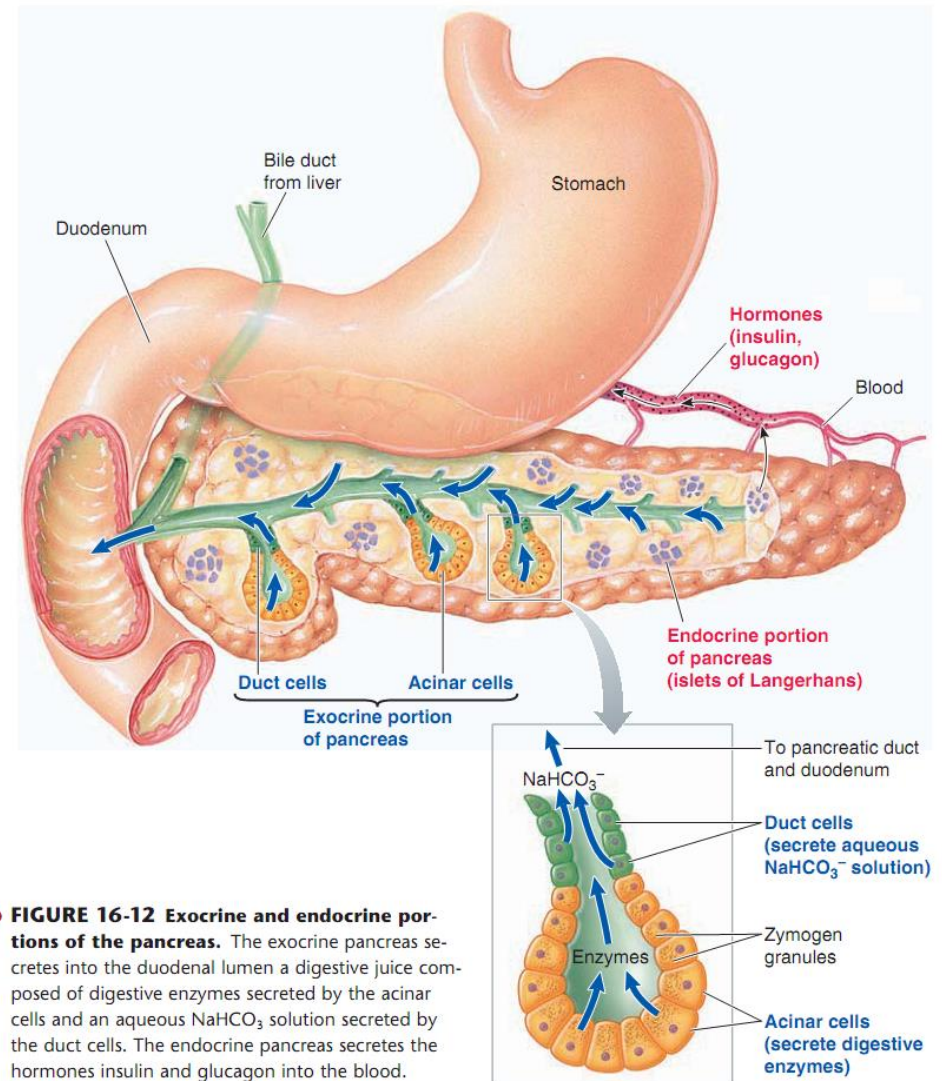
INI YANG TERJADI KETIKA TUBUH SEDANG PUASA!

SHARE KE BANYAK ORANG!



PANKREAS

- Kelenjar memanjang terletak di belakang dan di bawah lambung
- Mengandung jaringan eksokrin (asinus) dan endokrin (pulau-pulau Langerhans)



(Sherwood, 2009)

Getah pankreas terdiri dari 2 komponen:

1. Enzim pankreas yang disekresi kan oleh sel asinus
2. Larutan cair basa (banyak mengandung NaHCO_3) disekresikan oleh sel duktus → menetralkan keasaman kimus

Sel asinus mengeluarkan 3 jenis enzim:

1. Enzim proteolitik → pencernaan protein

— Tripsinogen $\xrightarrow{\text{enterokinase}}$ tripsin

— Kimotripsinogen $\xrightarrow{\text{tripsin}}$ kimotripsin

— Prokarboksipeptidase $\xrightarrow{\text{tripsin}}$ karboksipeptidase

disekresi dalam
bentuk inaktif dan
aktivasi di duodenum

2. Amilase pankreas → pencernaan karbohidrat

3. Lipase pankreas → pencernaan lemak

disekresi dalam bentuk
aktif

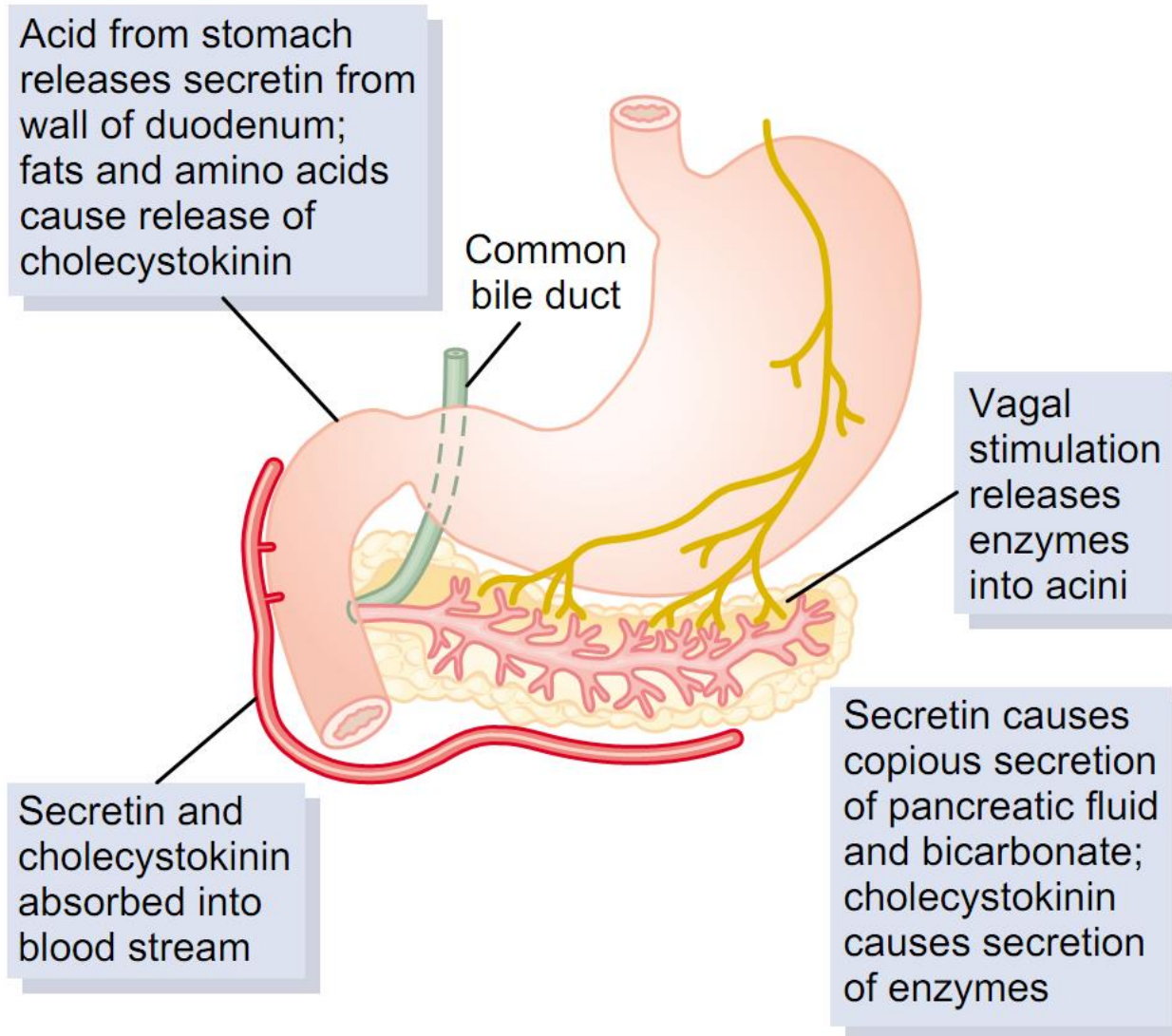
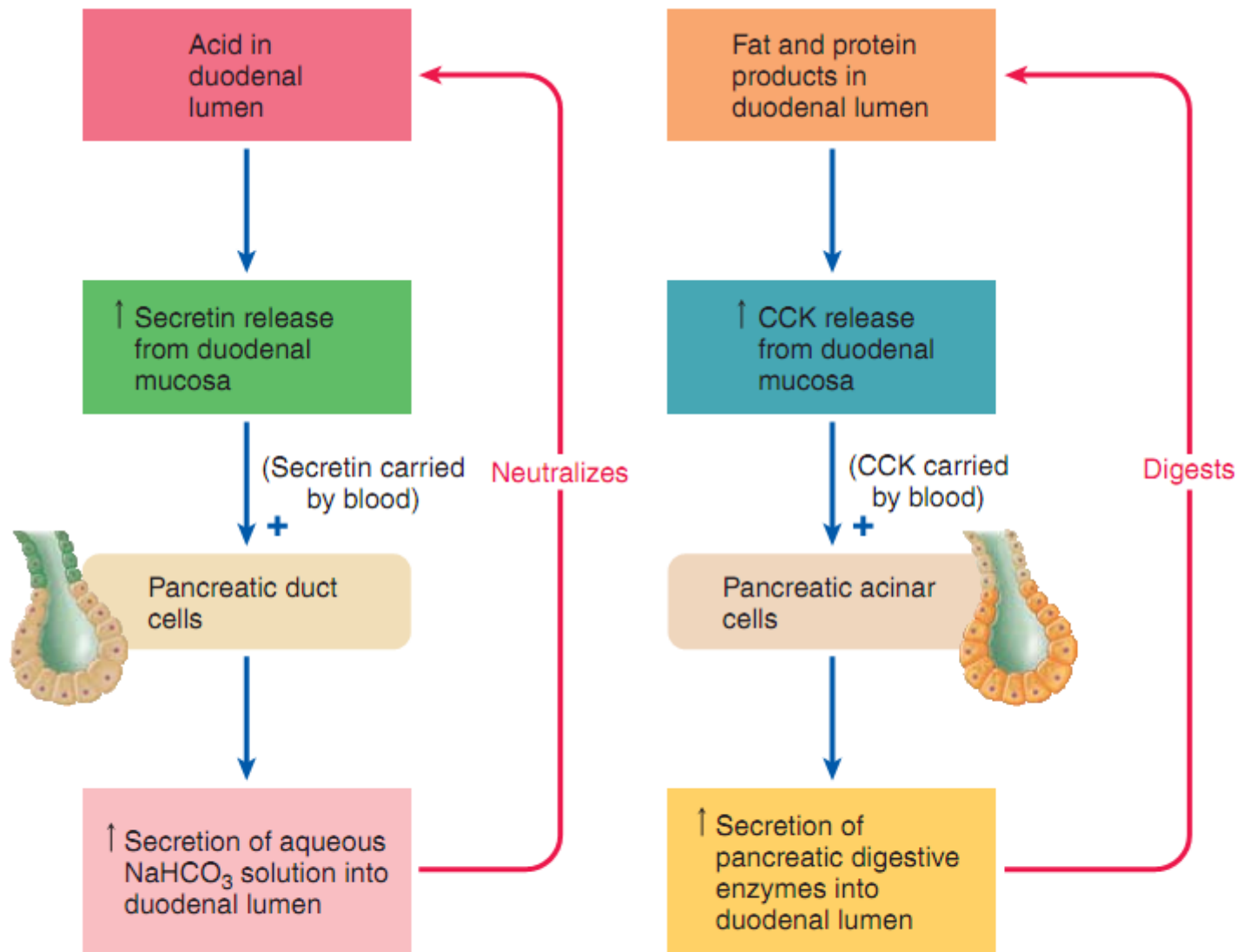


Figure 64–10

Regulation of pancreatic secretion.

(Guyton, 2006)



(a) Control of pancreatic aqueous NaHCO_3^- secretion

(b) Control of pancreatic digestive enzyme secretion

(Sherwood, 2009)

● **FIGURE 16-13 Hormonal control of pancreatic exocrine secretion.**

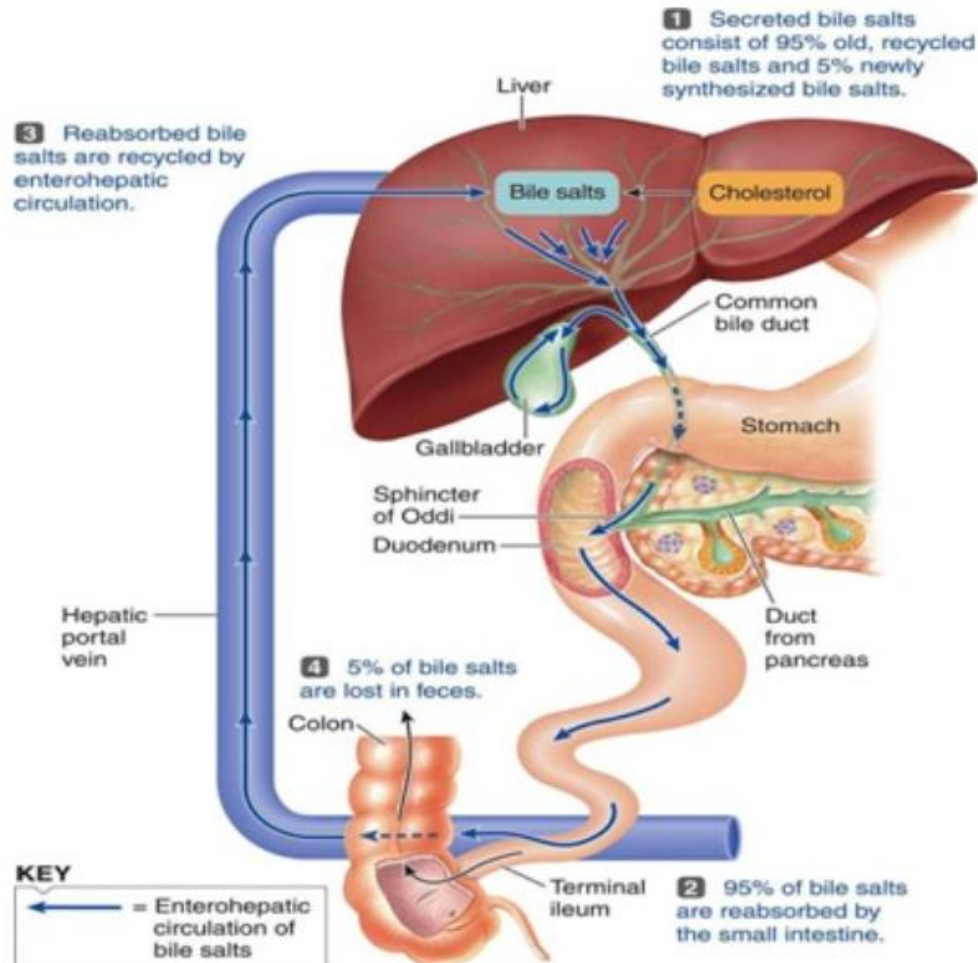
SISTEM HEPATOBILIER

- Peran hati dalam system pencernaan adalah ***sekresi garam empedu.***
- ***Garam empedu*** membantu pencernaan dan penyerapan lemak.
- Empedu disekresikan oleh Hati dan dialihkan ke kandung empedu di antara waktu makan
- Garam empedu didaur ulang melalui sirkulasi enterohepatik

Fungsi Hepar lainnya

- Metabolisme KH, protein, lemak sesudah diabsorpsi
- Detoksifikasi (zat kimia, obat)
- Sintesa protein plasma, vitamin K, faktor pembekuan
- Menyimpan glikogen, Fe, zinc, vitamin
- Mengaktifkan vitamin D
- Menyingkirkan bakteri & eritrosit yg rusak
- Ekskresi kolesterol & bilirubin

Sirkulasi Enterohepatik Garam Empedu



Rangsangan peningkatan sekresi empedu :

Stimuli

- garam empedu
- Mekanisme hormonal
Secretin
- Mekanisme neural
N. Vagus (Cephalic phase)

effect

Sekresi garam empedu ↑

Sekresi garam empedu ↑

Sekresi garam empedu ↑

CCK (cholecystokinin) mendorong pengosongan kandung empedu ke duodenum

Bila ada lemak di duodenum, maka
sekresi CCK ↑



kontraksi kandung empedu
relaksasi sfingter oddi

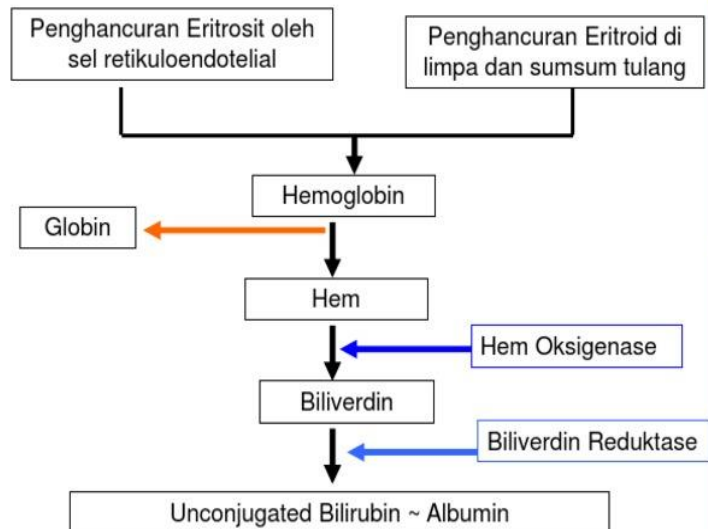


empedu keluar ke lumen duodenum

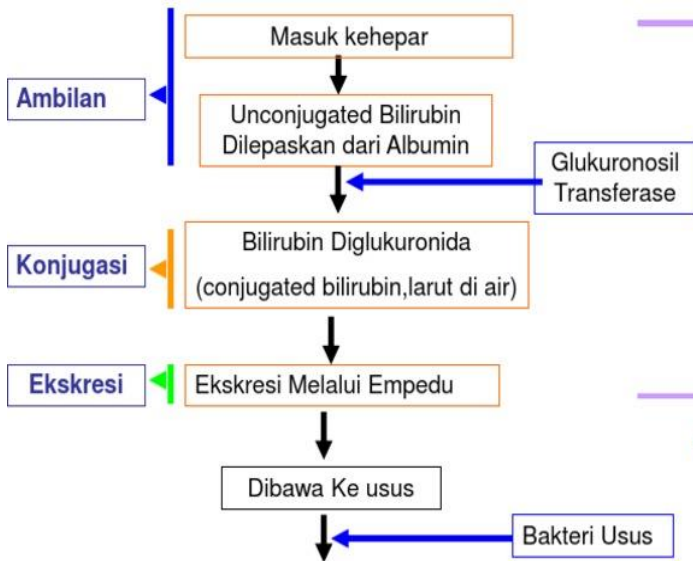
Bilirubin

- Bilirubin merupakan produk sisa yang diekskresikan ke dalam empedu
- pigmen empedu utama → empedu berwarna kuning
- Berasal dari penguraian sel darah merah usang
- Di saluran cerna pigmen ini dimodifikasi oleh enzim bakteri → warna tinja

Metabolisme Bilirubin

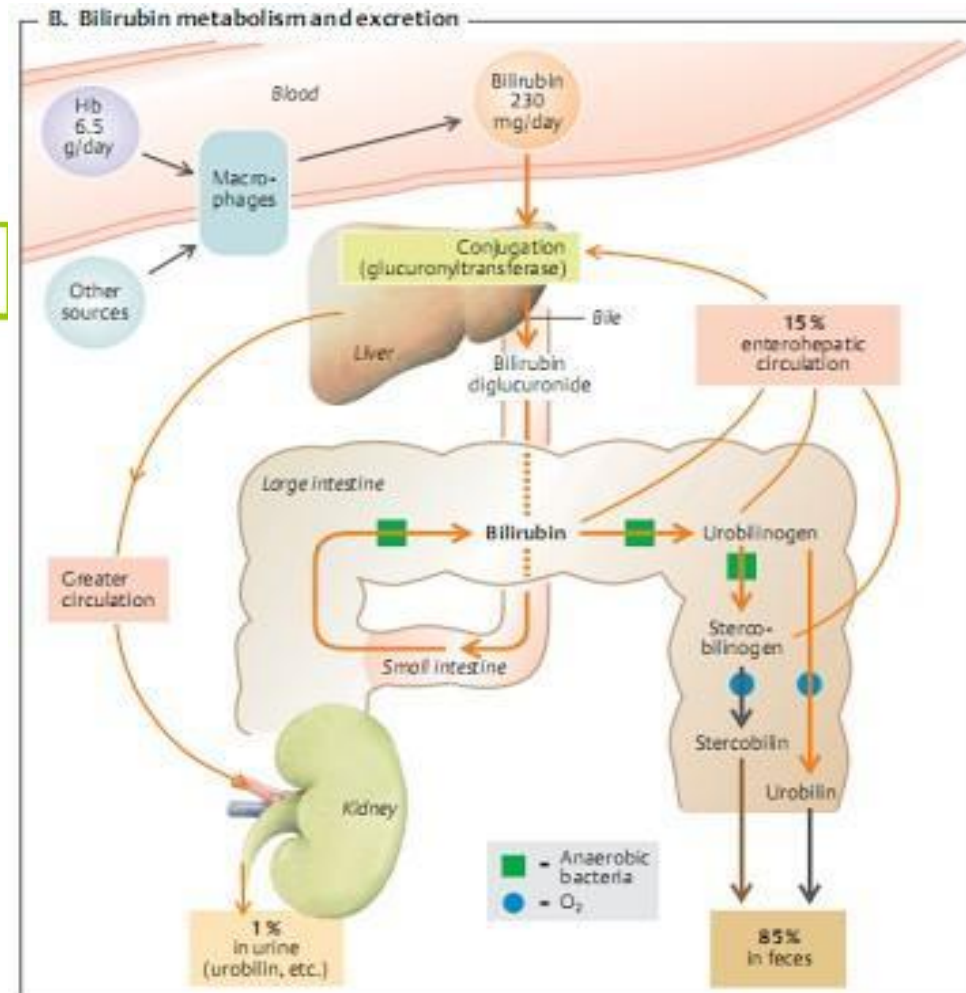


PRA HEPATIK

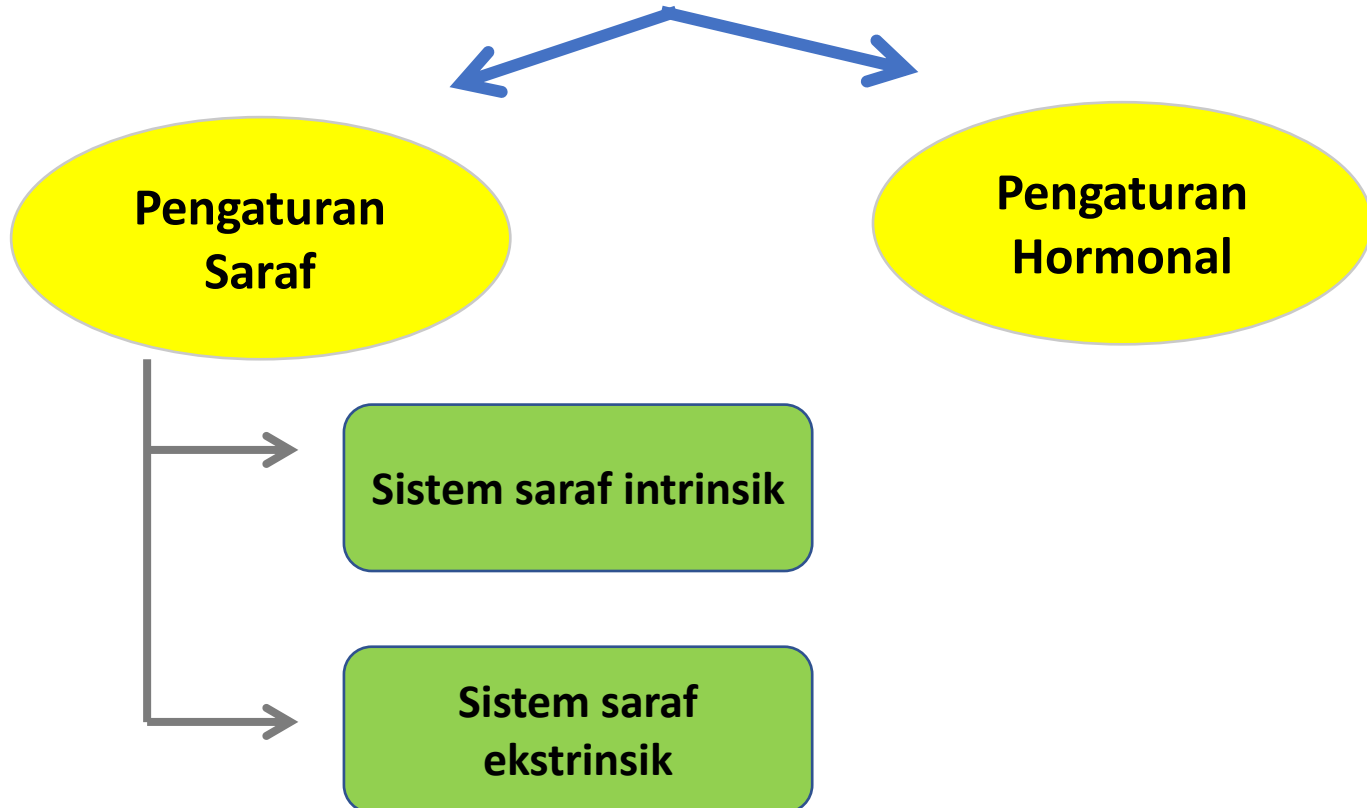


INTRA HEPATIK

POST HEPATIC



Pengaturan Fungsi Sistem Pencernaan

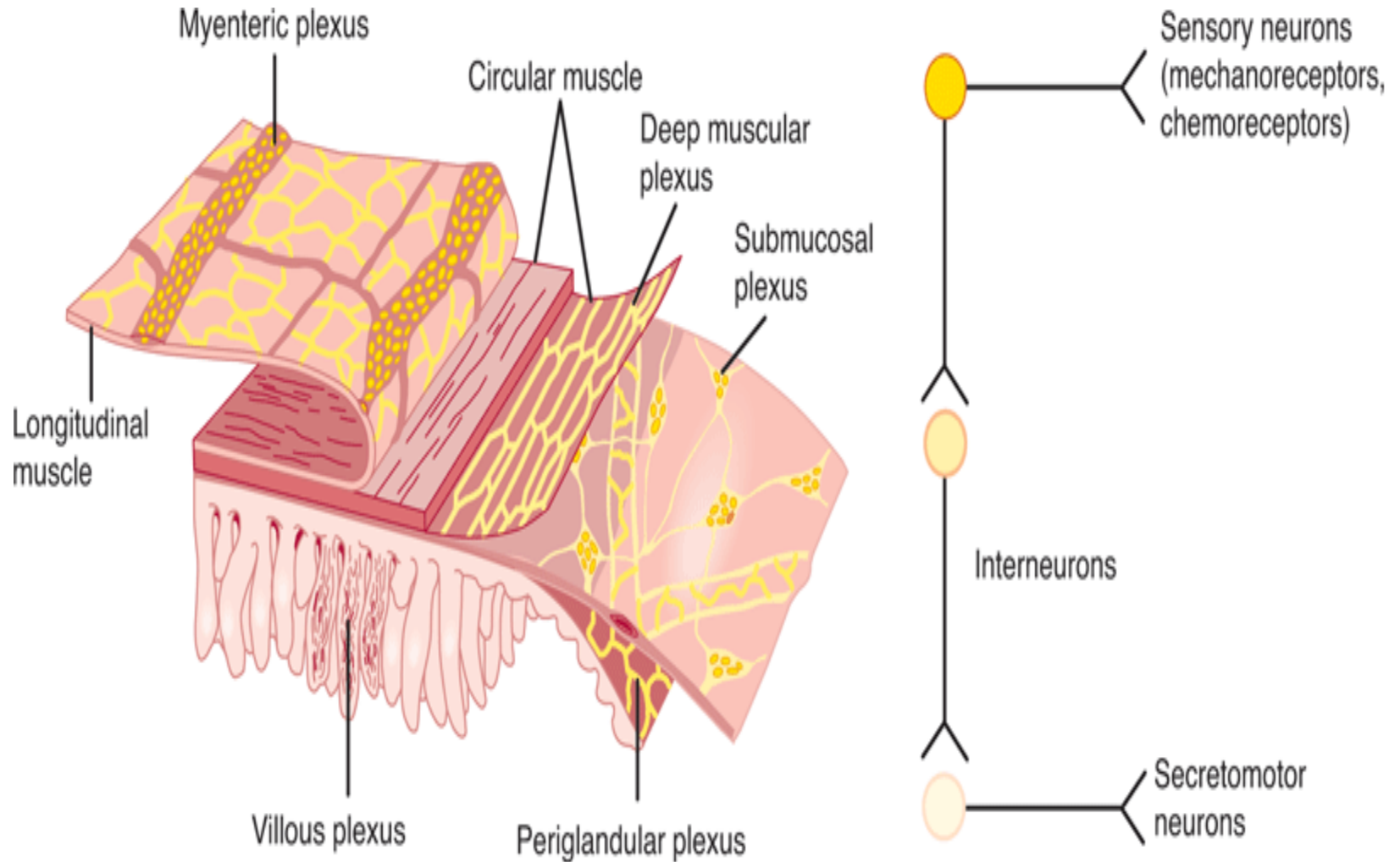


Neural Control

There are two components of GI innervation.

1. **Intrinsic innervation by the enteric nervous**
2. **Extrinsic innervation by parasympathetic and sympathetic nerves**—This can regulate the function of the enteric nervous system or directly control the activity of other cell types.

Intrinsic innervation by the enteric nervous



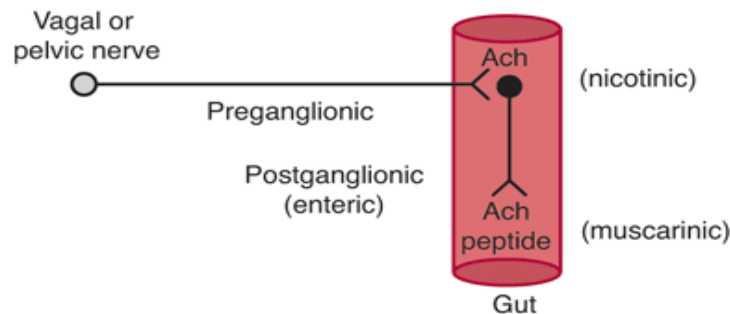
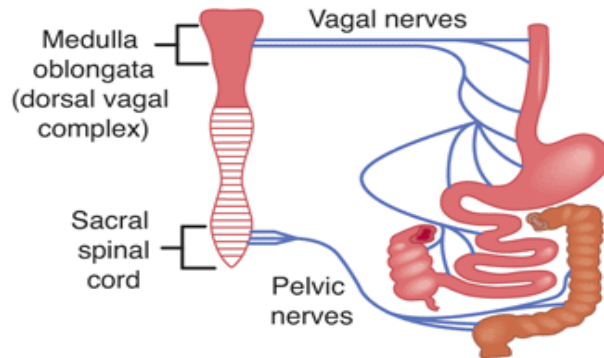
Source: McPhee SJ, Hammer GD: *Pathophysiology of Disease: An Introduction to Clinical Medicine*, 6th Edition: <http://www.accessmedicine.com>

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Extrinsic innervation by parasympathetic and sympathetic nerves

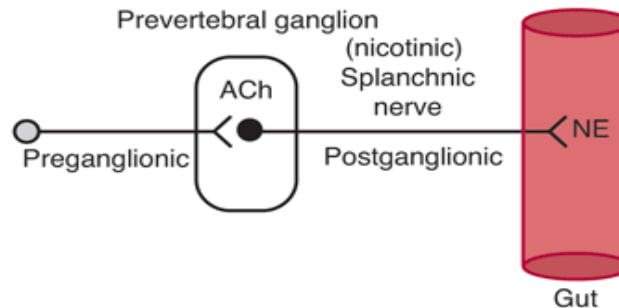
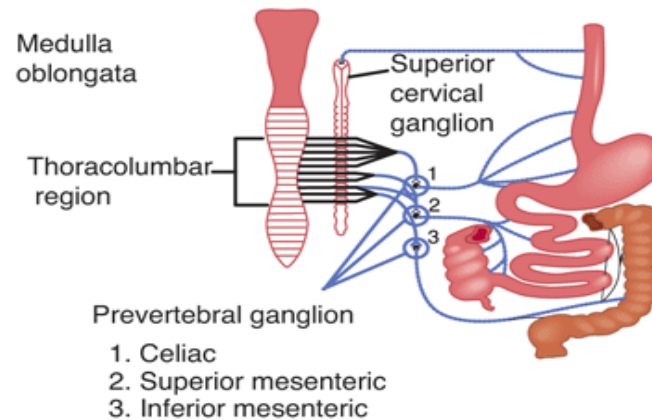
Parasympathetic

Neurons of the autonomic parasympathetic division project from the medulla oblongata and sacral regions of the spinal cord



Sympathetic

Neurons of the autonomic sympathetic division project to the gut from thoracic and first lumbar segments of the spinal cord



Source: McPhee SJ, Hammer GD: *Pathophysiology of Disease: An Introduction to Clinical Medicine*, 6th Edition: <http://www.accessmedicine.com>

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Parasimpatis divisi kranial → di dl nervus vagus → esofagus, lambung, pankreas, usus sampai 1/2 atas usus besar.
Parasimpatis sakral : S2-4 → melalui saraf pelvis → 1/2 bawah usus besar dan sepanjang anus. .
Simpatis → berasal dari T5 dan L2.

Neurotransmitter yang disekresi oleh Neuron-neuron enterik :

- Dua neurotransmitter yang penting :
 1. Asetilkolin → merangsang aktivitas gastrointestinal.
 2. Norepinefrin → menghambat aktivitas gastrointestinal.

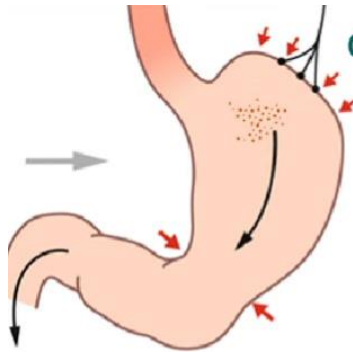
Pengaturan Hormon Terhadap Motilitas GI

- Di dalam mukosa bagian2 tertentu sal.cerna terdapat **sel-sel kelenjar endokrin**.
- Sekresi hormon ke dalam darah.
- Hormon-hormon GI dilepaskan ke dalam sirkulasi portal → reseptor → terjadi aktivitas fisiologis pada sel-sel target.
- Efek hormon tetap berlangsung bahkan setelah semua hub. Saraf antara tempat pelepasan & kerja hormon telah diputus.

Hormon

	STIMULUS FOR RELEASE	PRIMARY TARGET(S)	PRIMARY EFFECT(S)	OTHER INFORMATION
TOMACH				
gastrin	Peptides and amino acids; neural reflexes	ECL cells and parietal cells	Stimulates gastric acid secretion and mucosal growth.	Somatostatin inhibits release.
INTESTINE				
cholecystokinin (CCK)	Fatty acids and some amino acids	Gallbladder, pancreas, stomach	Stimulates gallbladder contraction and pancreatic enzyme secretion. Inhibits gastric emptying and acid secretion.	Promotes satiety. Some effects may be due to CCK as a neurotransmitter.
secretin	Acid in small intestine	Pancreas, stomach	Stimulates bicarbonate secretion. Inhibits gastric emptying and acid secretion.	
Motilin	Fasting: periodic release every 1.5–2 hours	Gastric and intestinal smooth muscle	Stimulates migrating motor complex.	Inhibited by eating a meal.
gastric inhibitory peptide (GIP)	Glucose, fatty acids, and amino acids in small intestine	Beta cells of pancreas	Stimulates insulin release (feedforward mechanism). Inhibits gastric emptying and acid secretion.	
glucagon-like peptide 1 (GLP-1)	Mixed meal that includes carbohydrates or fats in the lumen	Endocrine pancreas	Stimulates insulin release. Inhibits glucagon release and gastric function.	Promotes satiety.

GASTRIN



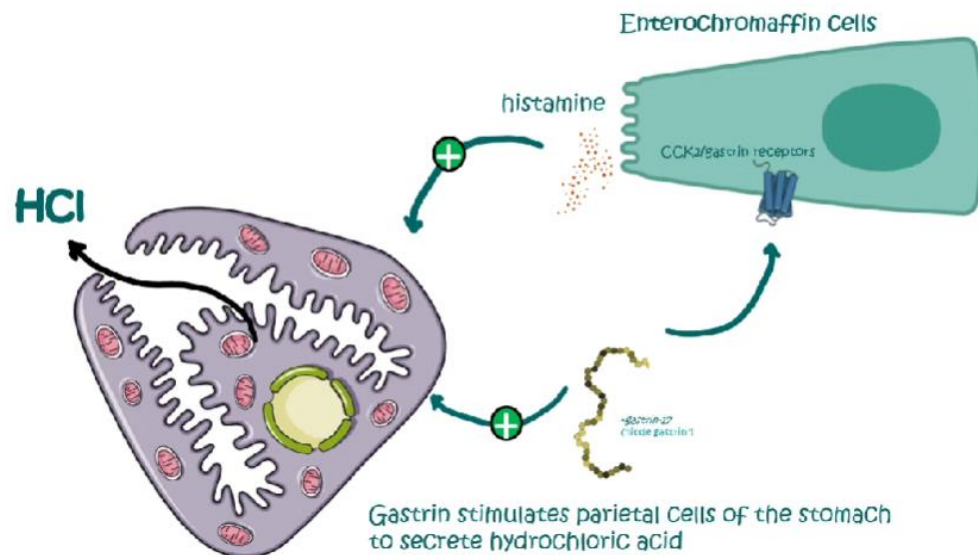
Gastrin is released in response to certain stimuli. These include:

- (1) The presence of partially digested proteins, especially amino acids, in the stomach. Aromatic amino acids are particularly powerful stimuli for gastrin release
- (2) hyperCalcemia (via Calcium-sensing receptors)
- (3) stomach antrum distension
- (4) vagal stimulation (mediated by the neurocrine bombesin, or GRP in humans)

Gastrin release is inhibited by:

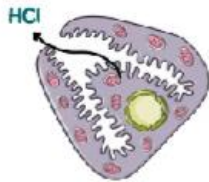
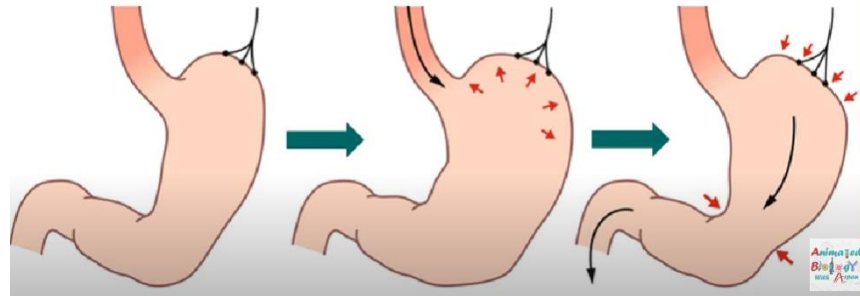
- the presence of too much acid (primarily the secreted HCl) in the stomach (a case of negative feedback)
- somatostatin also inhibits the release of gastrin, along with secretin, GIP (gastrin-inhibitory peptide), VIP (vasoactive intestinal peptide), glucagon and Calcitonin.

Animated
Biology
with Anissa



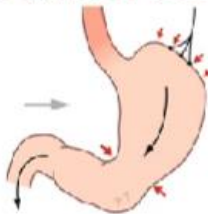
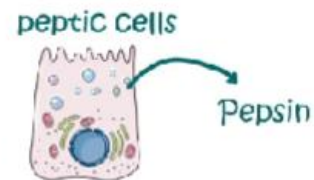
GASTRIN

Gastrin also aids in gastric motility.



• Stimulates parietal cell maturation and fundal growth.

• Causes chief cells to secrete pepsinogen, the zymogen (inactive) form of the digestive enzyme pepsin.



• Increases antral muscle mobility and promotes stomach contractions.

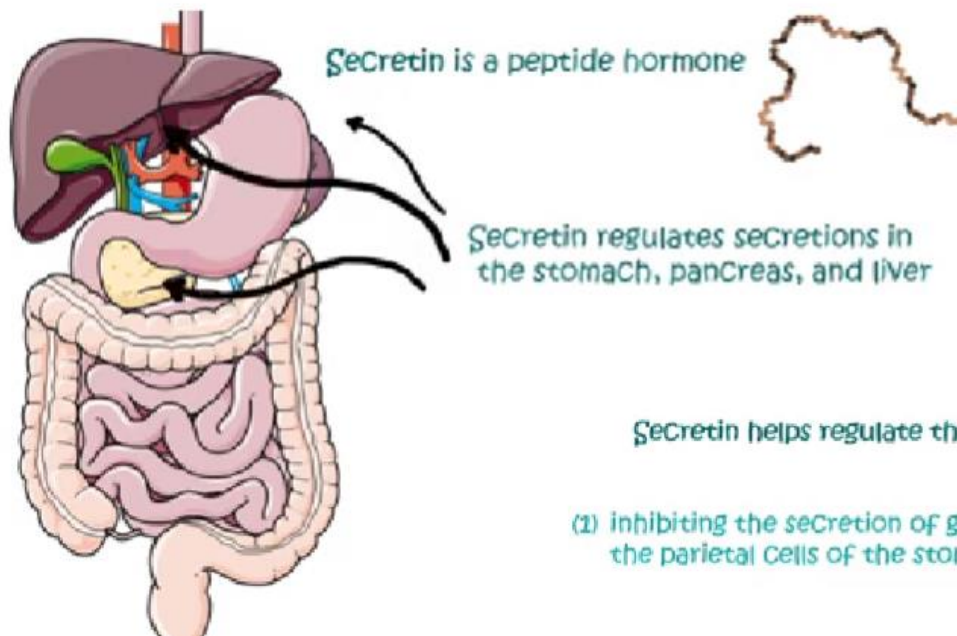
• Strengthens antral contractions against the pylorus, and relaxes the pyloric sphincter, which increases the rate of gastric emptying.

• Induces pancreatic secretions and gallbladder emptying.



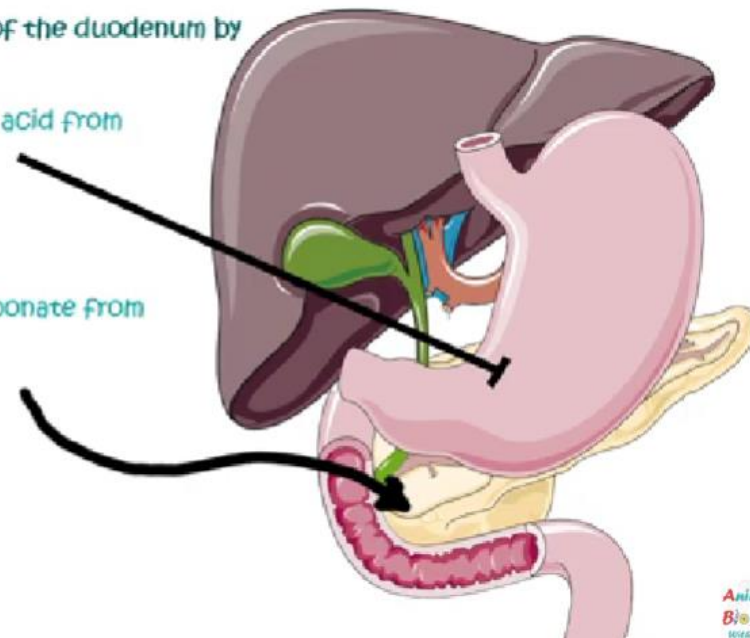
SECRETIN

Secretin is a hormone that regulates water homeostasis throughout the body and influences the **Alkaline** environment of the duodenum



Secretin helps regulate the pH of the duodenum by

- (1) inhibiting the secretion of gastric acid from the parietal cells of the stomach
- (2) stimulating the production of bicarbonate from the ductal cells of the pancreas.



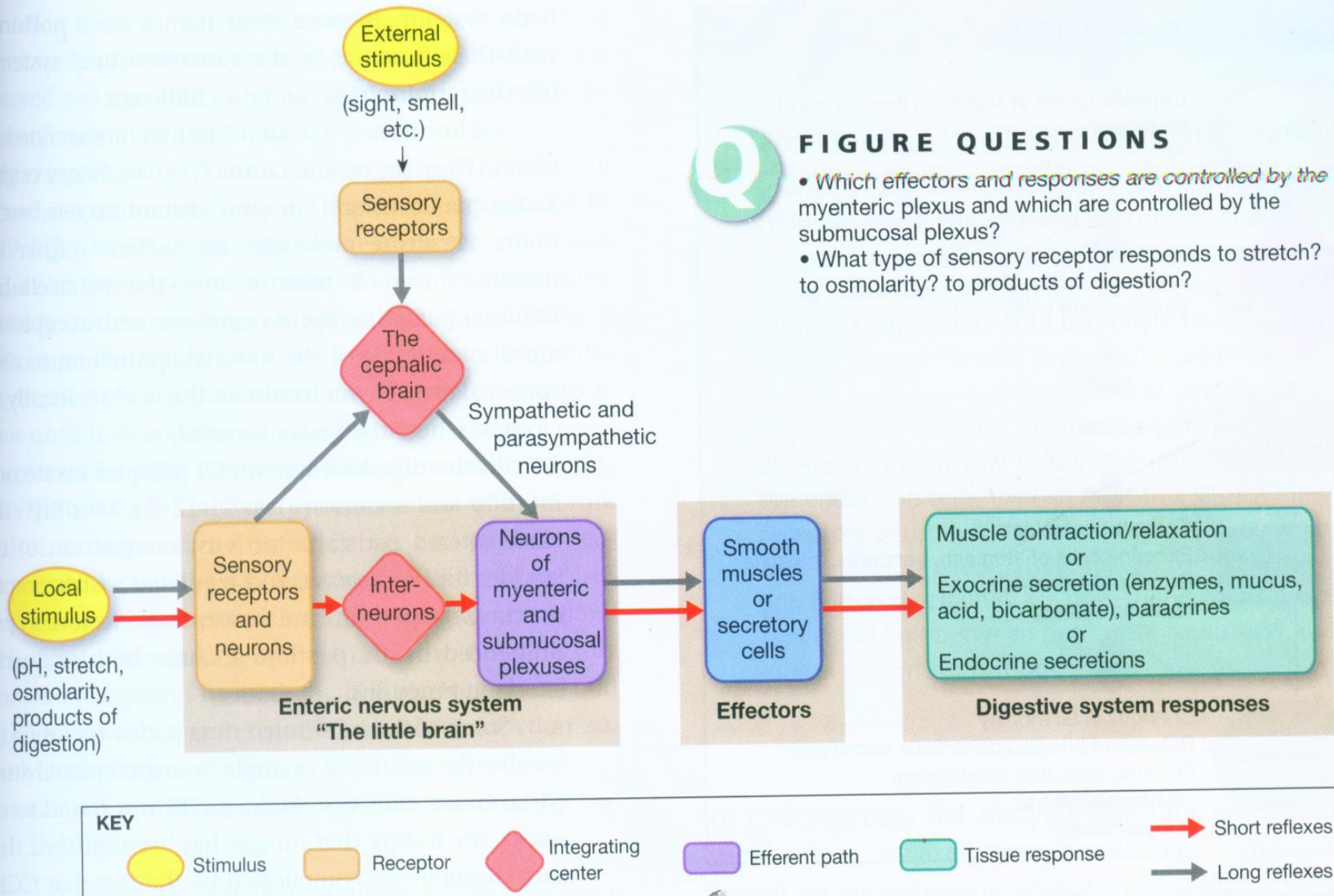
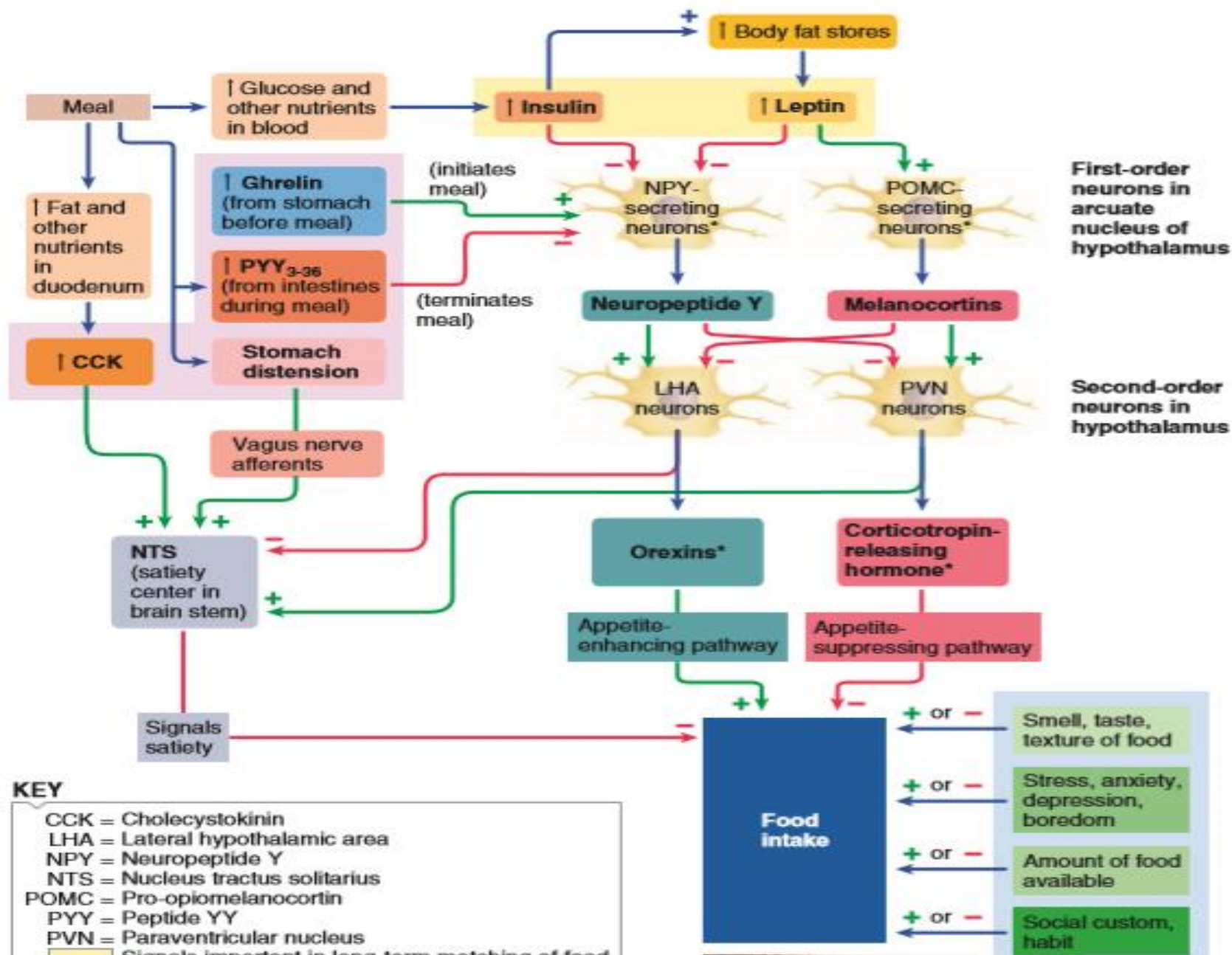
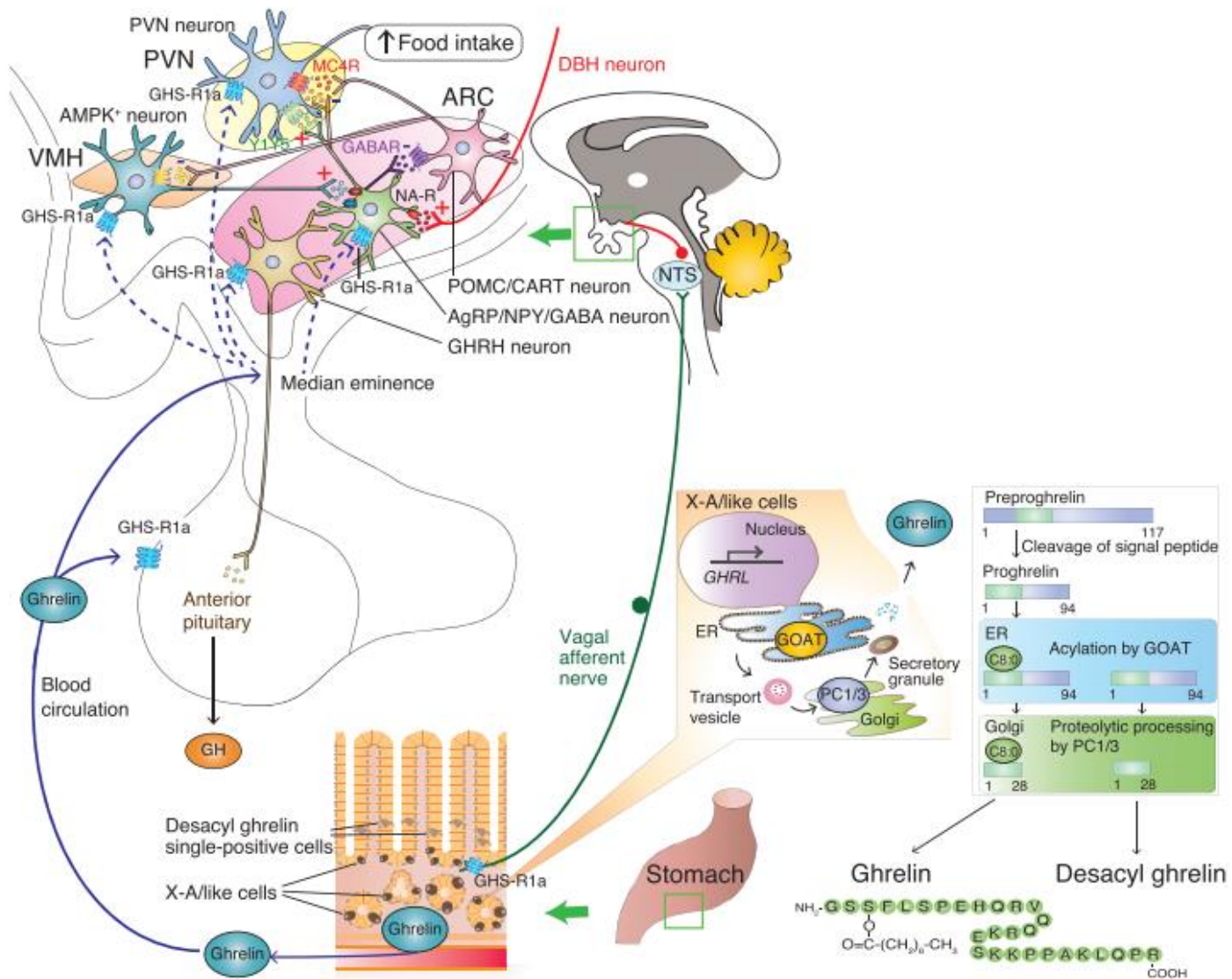


FIGURE QUESTIONS

- Which effectors and responses are controlled by the myenteric plexus and which are controlled by the submucosal plexus?
- What type of sensory receptor responds to stretch? to osmolarity? to products of digestion?

FIGURE 21-11 Integration of long and short reflexes in the digestive sys-





TERIMA KASIH