

BIOL 103 - Biology of Organisms

- Basic themes and concepts of biology spanning organizational levels from organisms to ecosystems in an evolutionary context
- Builds on cell biology covered in BIOL 102*, as different cell types combine into complex tissues and organs
- Explores how multicellular organisms evolved to exploit different environments, and the diverse array of biochemical, physiological, and behavioural mechanisms promoting survival and reproduction
- Introduces basic concepts of the way organisms are grouped into populations and species, and how groups of species interact with the environment to form dynamic ecosystems

Biology 103* Lectures. Part 1, emphasizing Organismal Biology and Animal Physiology

The first half of the course, from now until reading week, will be taught by Dr. Virginia Walker.

Dr. Virginia Walker's contact details:

- email (walkervk@queensu.ca)
- telephone 613-533-6123; fax 613-533-6617
- Biosciences office 2522

Office hours (Mon & Wed with 5 sessions)

Biosci ones at the walrus skeleton

After lectures:

Mon (**West**)* 11:30 -> ; (Biosci) 2:30-> & 5:30->

Wed (Biosci) 1:30-> 4:30->

Right by the walrus, 2nd
floor mezzanine



*on table outside lecture theatre

Biology 103 Lectures. Part 2, emphasizing Population Biology, Evolution, and Ecology

The second half of the course (after Reading Week) will be taught by Dr. Adam Chippindale

Contact Dr. Chippindale with any of your questions by

- email (adam.chippindale@queensu.ca)



Dr. Chippindale's research is perhaps the most 'spicy' in our department. He studies "sexual selection and conflict, from the gamete to the grave" - but he will probably tell you more about this after reading week



Biology 103 Labs. They start *next week or the week after* - know your section and time!

You will have a TA for your labs, held on the 2nd floor north wing Biosciences. The lab program and general course logistics are run by Mr. Rob Snetsinger, Biol 103 Lab Instructor

Contact Mr. Snetsinger by:

- email (snetsing@queensu.ca)
- calling him at 613-533-6000, ext. 77439
- or dropping by his office (Biosciences Rm 2322)



NO LABS FIRST WEEK

BIOLOGY 103 LAB SCHEDULE

(check on Solus for your time and day)

Mr. Snetsinger says:

1. YOU ARE STRONGLY ADVISED TO READ THE LAB MANUAL BEFORE COMING TO THE LAB AND COMPLETE ANY WORK (where possible) AHEAD OF TIME. FIRST LABS ARE POSTED via OnQ.
2. REGISTER YOUR CLICKER – details to be announced (it may be a different system than Biol 102). You can use your clicker if it is not registered (since it still registers) but eventually you will need to get it registered so your name can be linked to the clicker number.



Lab Objectives and Schedule

- The lab manual in onQ will cover lab objectives, schedule and ancillary material (e.g. tips on report writing, seminar presentation, plagiarism, etc.)
- Similar to Biology 102, labs are designed to introduce basic communication skills in data collection and analysis, and to create an awareness of how biological variation affects experiments designed to test hypotheses generated from biological theory
- An important component of the lab program is learning that original research requires one to read scientific literature (e.g. know what others have done), and to communicate results to the public and colleagues

Lectures and Presentation Material

- Generally, try to attend lectures in your assigned time slot. Check the Bio 103 OnQ site regularly for changes or announcements! Do not miss classes- attending all classes is the easiest way to do well in this course.
- PowerPoint presentations will be available via onQ as PDF files
- Presentation files will usually be available before lectures. PDF files ***may be updated*** if corrections are made.
- The lectures may be recorded (this is a technology that is not strongly supported by the university and doesn't always work). If so, they will be available ~1-2 days after the lecture. Remember that one of the skills you should learn this year is to take good notes in class.

Announcements and Supporting Material

Announcements and supporting material can be posted on the Biol 103* OnQ page. There may also be some general information on the course homepage:

We want you to check the OnQ site regularly.

NOTE that for the first six weeks of classes, classes will be held for all sections (001, 002, and 003) at the scheduled time slot, unless there is an extreme condition/illness.

You will need to register your clicker- “clicks” will be recorded starting next week- clicker practice will be this week for those that are new to the system.

How to do well in Biol 103* (and get help when you need it)

- there is a lot of material in the lectures, readings, and labs. Attend lectures and labs....you MUST keep up - don't wait until April!
- focus on key concepts for each lecture or lab. Use the review tools available in each chapter, on the textbook website, and Bio forum (see OnQ site)
- if in doubt about expectations for labs, ask your TA or Mr. Snetsinger *before* submitting your work
- for problems, major or minor, the sooner you ask, the more likely we can do something to help you out

(continued.....)

How to do well (and get help when you need it), pt. 2

- for basic questions about labs or course logistics (schedules, due dates, marks, missed assignments, illness etc.) **Mr. Rob Snetsinger** first. He will refer you if he can't help
- for lecture questions, post them on Bio forum (OnQ) and if not satisfied, contact your professor, **Dr. Virginia Walker** or **Dr. Chippindale**
- for questions about labs, first ask your TA, and then speak to **Mr. Snetsinger** if required
- if you still aren't satisfied, contact **Dr. Chippindale**, then the Biology Chair of Undergraduate Studies (**Dr. W. Nelson**), and then the Head of Biology (**Dr. B. Cumming**) in that order

Biol 103* Tentative Assessment

Lab assignments (details in lab manual)	36%
3 on-line quizzes (2 lecture/1 lab)	12%
i-Clicker use	3%
'Mastering Biology' assignments (5)	8%
Final lecture exam	41%

Biology 103 lecture schedule, Virginia Walker

References (Campbell text)

1. Jan 9, Mon (10:30 west)
1. Jan 9, Mon (1:30 Biosci) Nutrition and digestion (Ch 41)
1. Jan 9, Mon (4:30 Humph)
2. Jan 11, Wed (9:30 west)
2. Jan 11, Wed (12:30 Biosci) Digestion and absorption (Ch 41)
2. Jan 11, Wed (3:30 Humph)
3. Jan 13, Fri (8:30 west)
3. Jan 13, Fri (11:30 Biosci) Excretion; review (Ch 44)
3. Jan 13, Fri (2:30 Humph)
4. Jan 16 Excretion and ion regulation (Ch 44)
5. Jan 18 Movement & muscles (Ch 50)
6. Jan 20 The nervous system; week review (Ch 48)

***Bring scrap paper with you on every Friday for our review sessions**

continued....

- 7. Jan 23 Neural transmission & nerves (Ch 48)
- 8. Jan 25 Neuroscience (Ch 48)
- 9. Jan 27 Circulatory systems; week review (Ch 42)
- 10. Jan 30 Respiration (Ch 42)
- 11. Feb 1 Immunity (Ch 43)
- 12. Feb 3 Immunogenetics; week review (Ch 43)
- 13. Feb 6 Immunity and cancer (Ch 43 & 19)
- 14. Feb 8 Evading the immune system; HIV (Ch 43; Ch 19)
- 15. Feb 10 Immune systems in other animals; review (Ch 43)
- 16. Feb 13 Endocrine system (Ch 45)
- 17. Feb 15 Hormones and development (Ch 45)
- 18. Feb 17 Wrapping up; review course to date

Reading Week

Feb 28 Tues (most-likely date), On-line (midterm) quiz (2% of grade)

- *Notes: 1. these lecture topics are subject to change- but every effort will be made to present the same material in replicated lectures
2. OnQ does not accept large files- in some cases, I will have to delete (unimportant, copyright OR text) pictures from the on-line PDFs



Welcome to Biology 103*

Dr. Virginia K. Walker
Room 2522, BioSciences
email: walkervk@queensu.ca

BSc: Acadia University
PhD: Medical Biochemistry, University
of Calgary
Postdoc: University of Cambridge

Sabbaticals at CSIRO, Australia, U.
New England, Australia, U. Calgary
and U. Alaska, Fairbanks

When I am not teaching, what
do I do?

Solomon/Berg/Martin, Biology, 6/e
Figure 1.4A



(a)

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***We will go from digestion to insect
development in 18 lectures**

Freeze and freeze-thaw resistance in plants, invertebrates and microbes



Searching for psychrophilic organisms and their secrets of survival Daring Lake, north of Yellowknife, NWT



Freeze resistance in organisms from brine lakes in BC



How do microbes from brine lakes survive? Are they preadapted' for freeze resistance?

With data thanks to many undergraduate honours students!

Towards a sustainable fishery for Nunavummiut

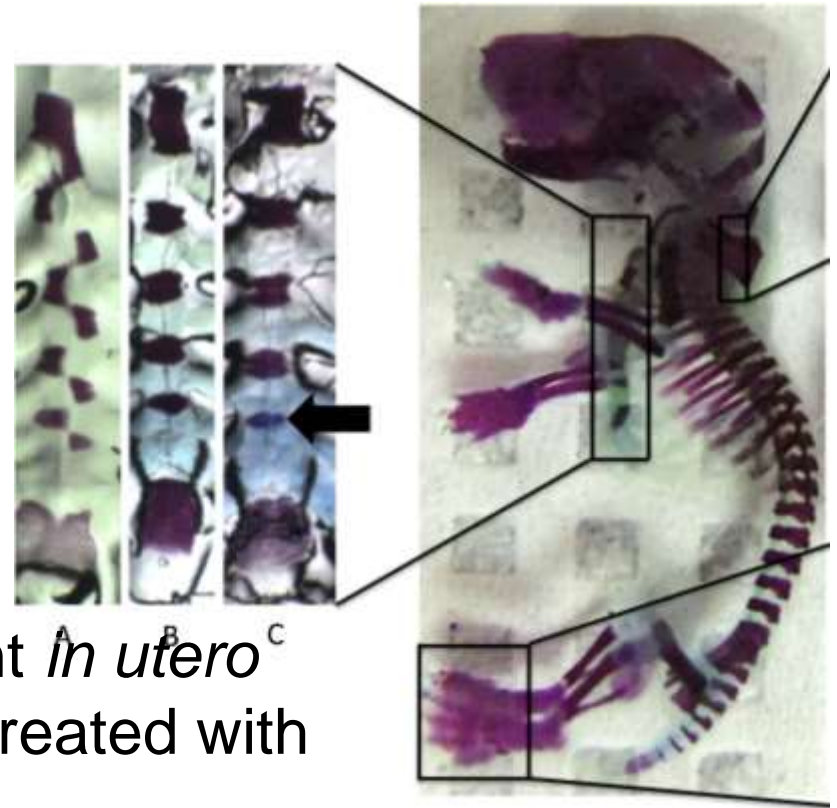


Through genomic analysis and in partnership with communities we will identify the fish stocks & examine their health

The effects of 'pollutants' on organisms

The impact of manufactured nanoparticles on microorganisms and animals

Manufactured nanoparticles first started to appear in numerous applications in 1990. Now they are 'everywhere' it seems.....



There is an impact on development *in utero*^C when pregnant mouse mums are treated with nanoparticles (C nanotubes)

I. Nutrition and Digestion

Lecture Reference: Campbell Chapter 41

Your text illustrates this chapter with a black bear foraging on berries since this is a Canadian edition- here is a picture of a fly being eaten by a plant since I want you to think broadly about digestion



(b)

Two major categories of organisms based on their mode of obtaining nutrients

1. **Autotrophs** (Gk = self nutrition)

These organisms harvest light or chemical energy and store it in carbon compounds. They can exist in an inorganic environment and they manufacture organic compounds. They are primary producers.

For instance: $\text{CO}_2 + \text{H}_2\text{O} \rightarrow$ organic compound such as $\text{C}_6\text{H}_{12}\text{O}_6$

2. **Heterotrophs** (Gk = other nutrition)

These organisms must get complex nutrients from the environment. They receive nutrition by eating other organisms. They are found at higher trophic levels.

Heterotrophs have evolved multiple strategies for obtaining food

(some of these are listed in your text, pg. 944)

Bulk Feeders



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However, there are more challenges....if you are a heterotroph, how do you get large organic compounds past the cell membranes?



What about fungi?



Starch
(polysaccharide)

Bread mold



Mmm...so fungi can get nutrients from dead organic matter. Can they get nutrients from living animals?



Athlete's foot fungus extends fungal branches (= hyphae) into the cells of the foot to absorb nutrients

Dactylella dreschleri is a fungus that has sticky knobs along the hyphae that can hold nematode worms



A



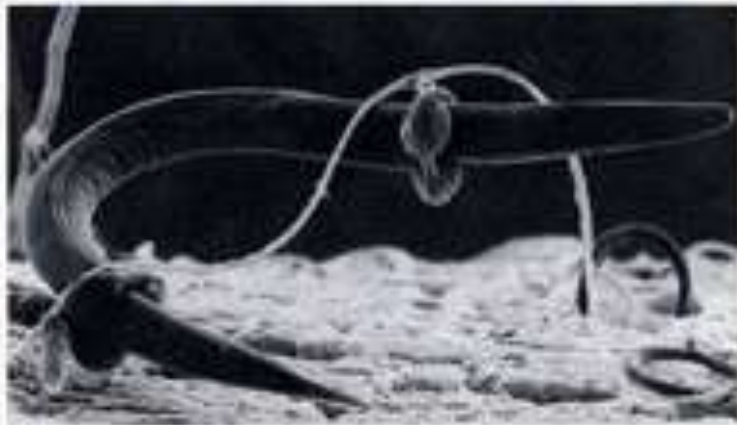
B

The hyphae then penetrate the worm's body, digestive enzymes are released and extracellular digestion takes place.

Arthrobotrys dactyloides is another example. It is a fungus that makes traps that are used to capture nematode worms



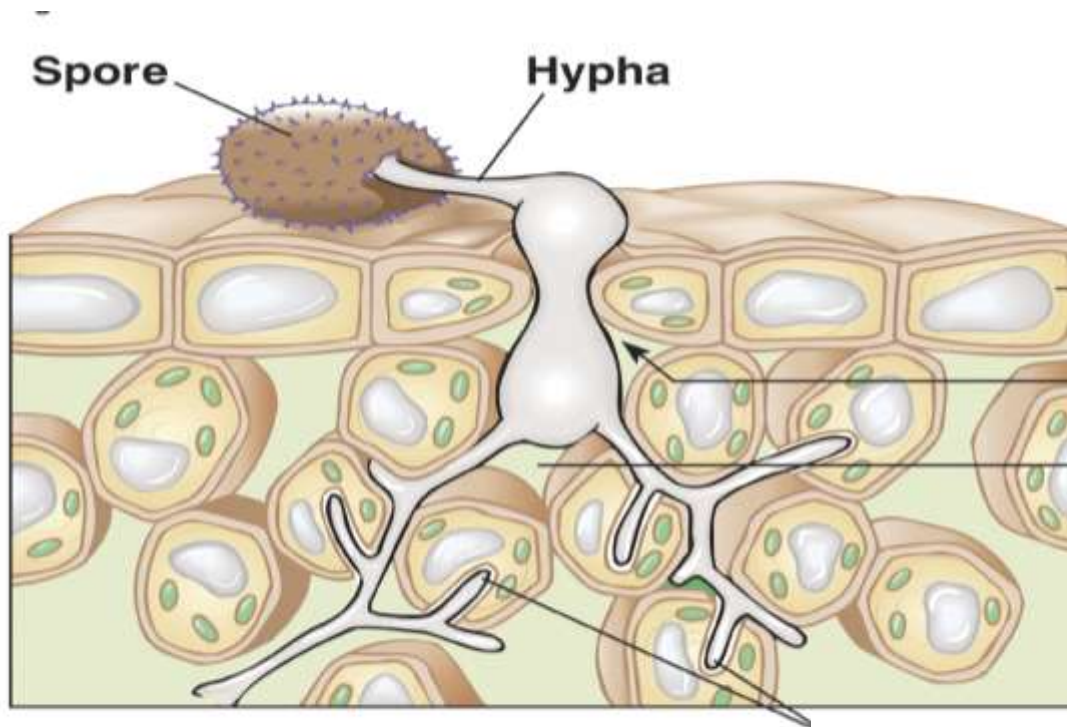
c



D

When nematodes enter the traps made by three cells, the cells swell and constrict essentially lassoing the worm. Then hyphae penetrate the worm, and extracellular digestion takes place.

So the take home message is that fungi can be parasitic or predatory (e.g. plant parasitic fungi, Athlete's foot, "cowboy fungi")



Intracellular Digestion

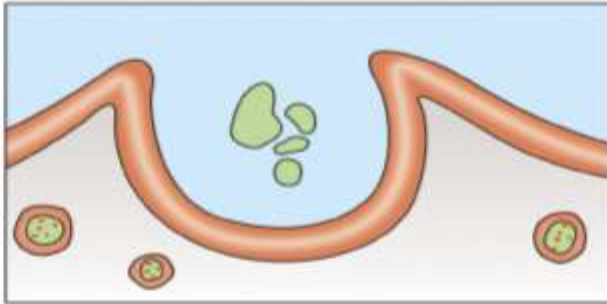
Reference: your text page 945



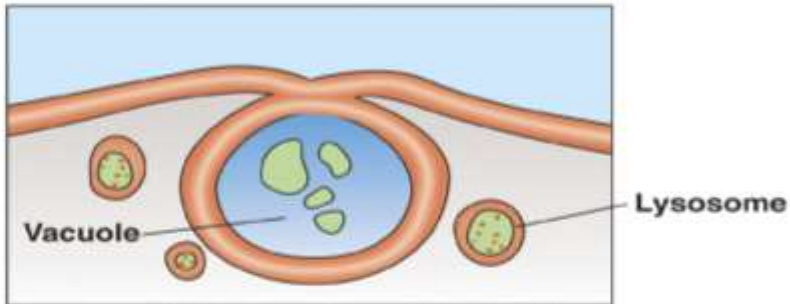
Protozoa: simple, single cell

Hmmm...how do membranes fuse?

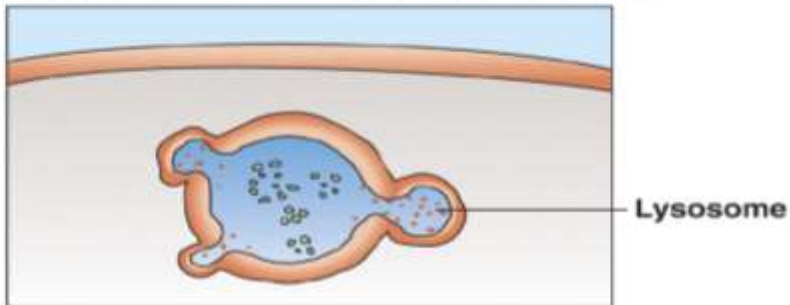
Phagocytosis



- 1 Folds of the plasma membrane surround the particle to be ingested, forming a small vacuole around it.



- 2 The vacuole then pinches off inside the cell.

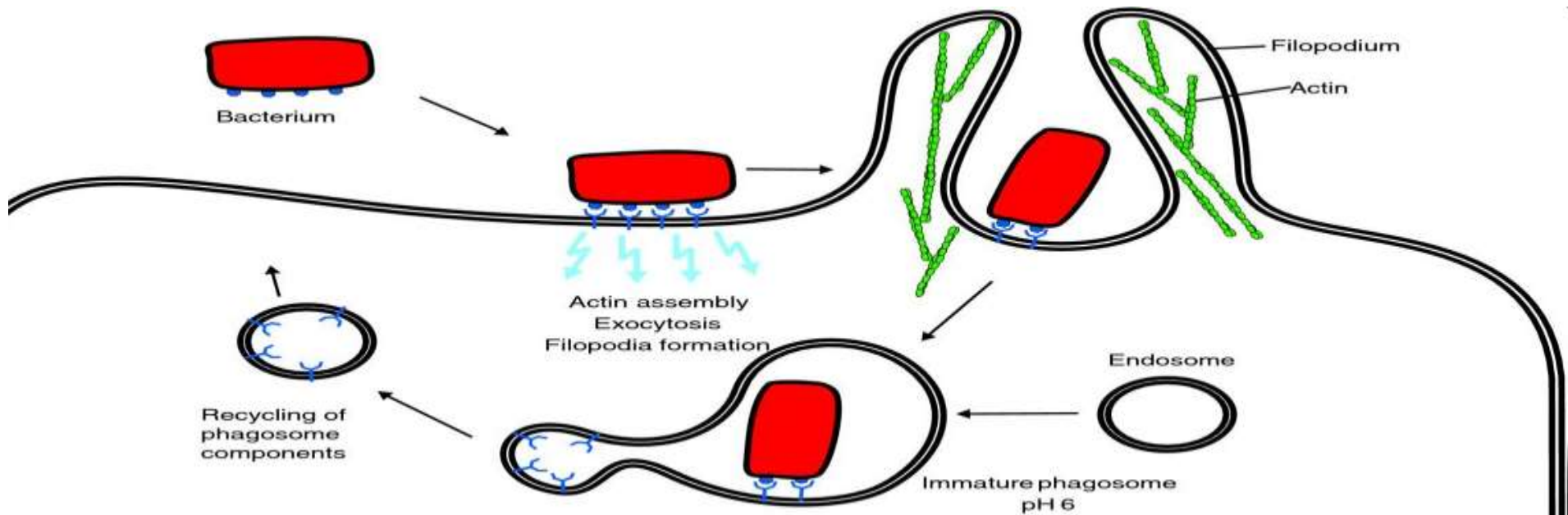


- 3 Lysosomes may fuse with the vacuole and pour their potent enzymes onto the ingested material.

(a)

The molecular biology of phagocytosis in protozoans can be complicated, for example Okada and colleagues showed that there were 85 different proteins involved in the process in a genus that infects up to 10% people.

Phagocytosis is also important in vertebrates



*Okada et al. Eukaryot Cell. 4: 827–831. (*no, you do not need to look this up*)

<http://images.google.com/imgres>

New approach for treating skin cancer

Study shows how dulling 'don't eat me' signal and targeting dangerous tumor cell subset can curb metastatic melanoma

Cancer cells interfere with phagocytosis by producing a 'don't eat me' signal. Here they block this protein

Date: August 1, 2016

Source: University of California - Irvine

Summary: Using new and innovative immune-therapeutic approaches to silence 'don't eat me' signaling proteins recognized by specialized cells of the immune system, molecular biologists have identified an effective way to combat metastatic melanoma.

Share:



In another cover story the green cancer cell gets 'eaten' by the phagocyte



FULL STORY

Using new and innovative immune-therapeutic approaches to silence "don't eat me" signaling proteins recognized by specialized cells of the immune system, University of California, Irvine molecular biologists and their colleagues have identified an effective way to combat metastatic melanoma.

Led by Alexander D. Boiko, UCI assistant professor of molecular biology & biochemistry at the Ayala School of Biological Sciences and the Sue and Bill Gross Stem Cell Center, the researchers discovered that blocking the cell surface protein, CD47 (known as a "don't eat me" signal), on melanoma cells, increased the degree by which these cells were phagocytosed, or "eaten," by macrophages. The team further discovered that blocking CD47 in combination with targeting a second cell surface protein, CD271,

Recall that you studied endocytosis in Biol 102, a similar process, with some of the same components. Indeed, Braun and Niedergang published a paper on “Linking exocytosis and endocytosis during phagocytosis” in *Biology of the Cell*, 98:195 [No- you don't need to look this up!](#)

Important bit: Various proteins (including SNARES) and Ca^{++} are involved

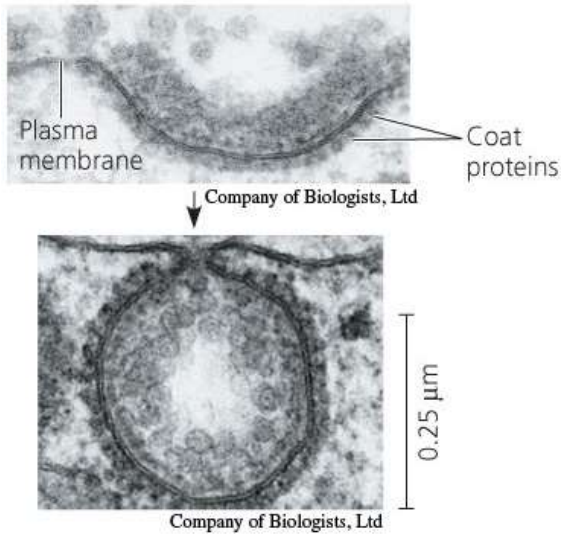
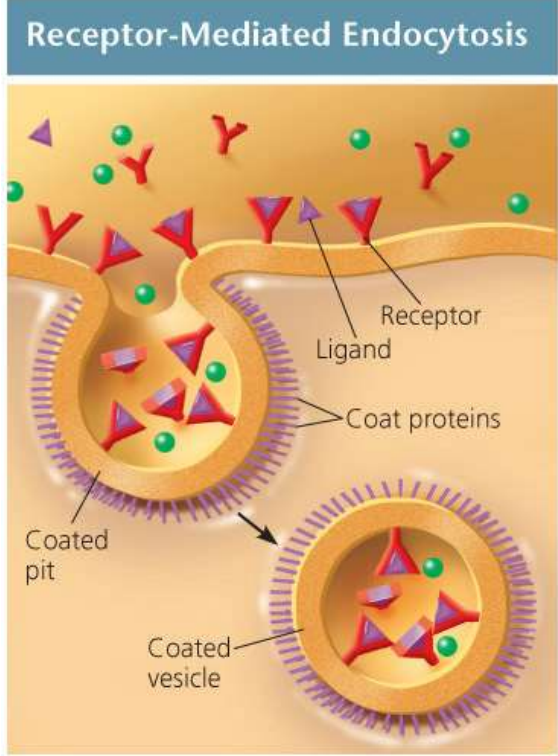
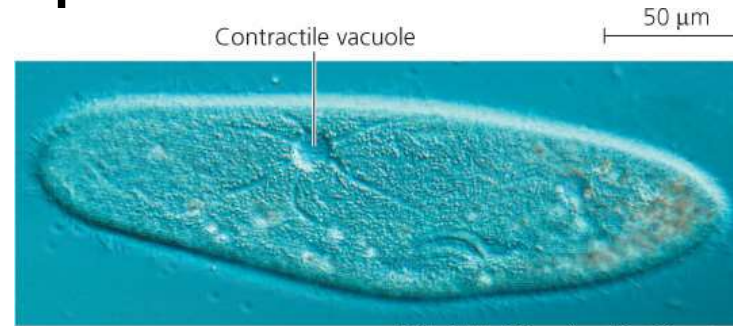
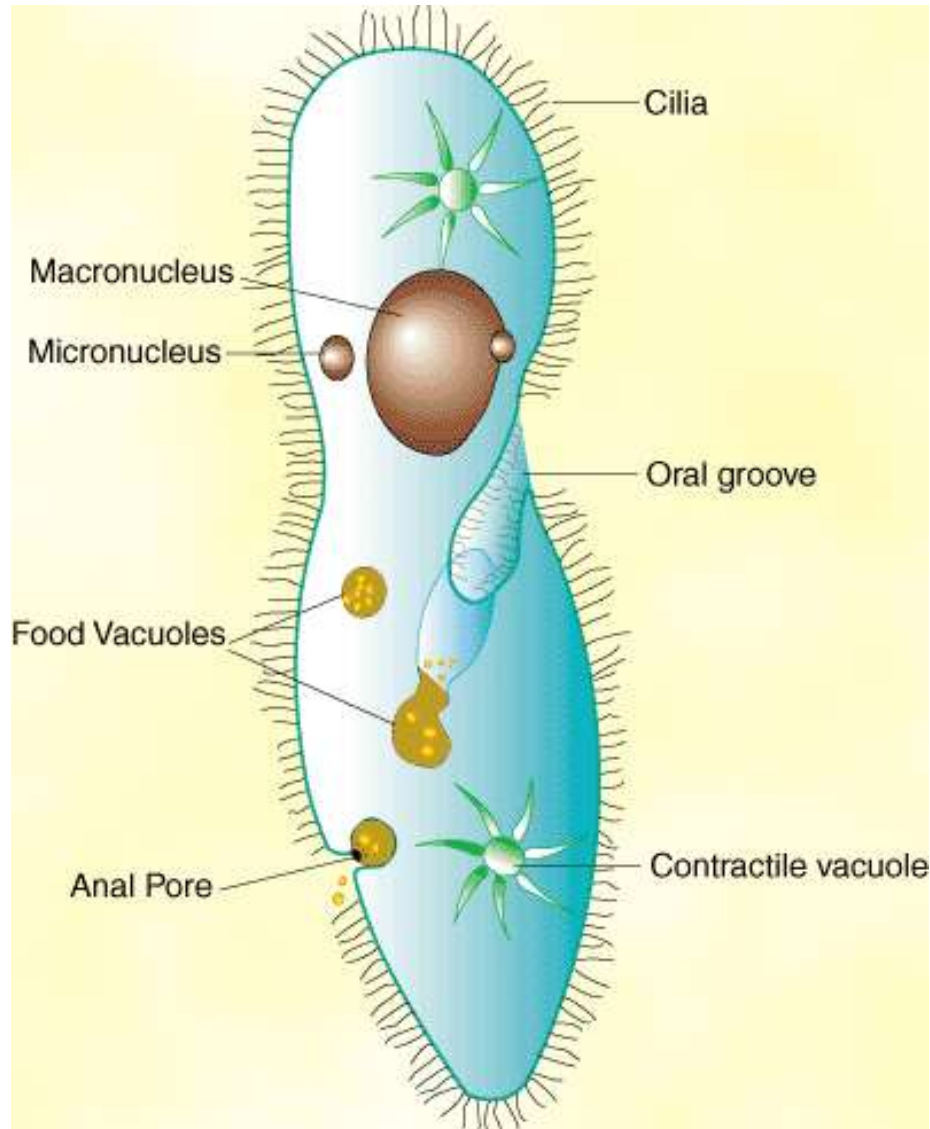


Fig. 7.20 from your text

So we protect ourselves from microbes using phagocytosis, but some microbes have evolved mechanisms to try to escape phagocytosis....

Food vacuoles in a paramecium

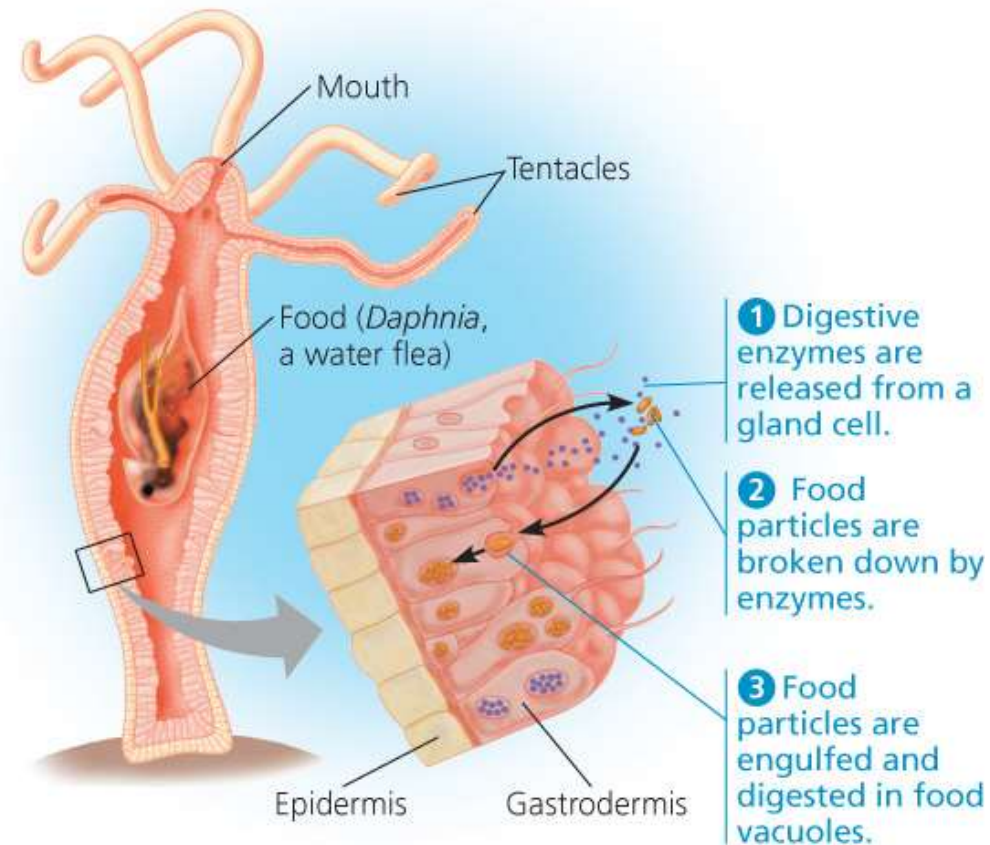


Michael Abbey/Science Source/Photo Researcher

▲ **Figure 7.14** The contractile vacuole of *Paramecium caudatum*. The vacuole collects fluid from a system of canals in the

Extracellular digestion: your text discusses digestion in the hydra (pg. 945)

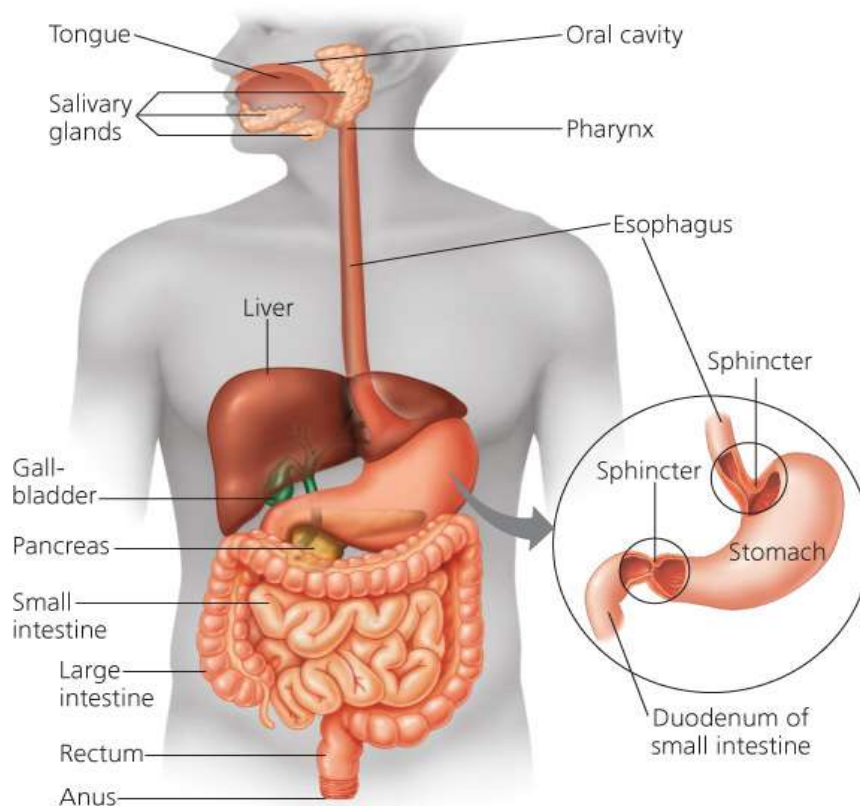
- Digested food products are phagocytosed directly into the cells that line the gastrovascular cavity- further digested intracellularly
- Wastes are excreted out of the mouth
- *Note*: there is one opening that serves as both entry and exit for food



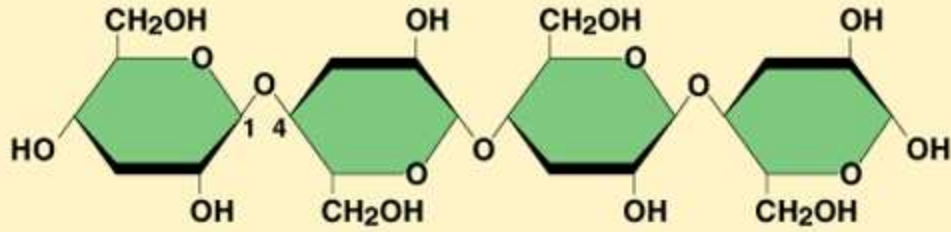
▲ **Figure 41.6 Digestion in a hydra.** Digestion begins in the gastrovascular cavity and is completed intracellularly after small food particles are engulfed by specialized cells of the gastrodermis.

In contrast, more complex organisms have an alimentary canal with openings at both ends.

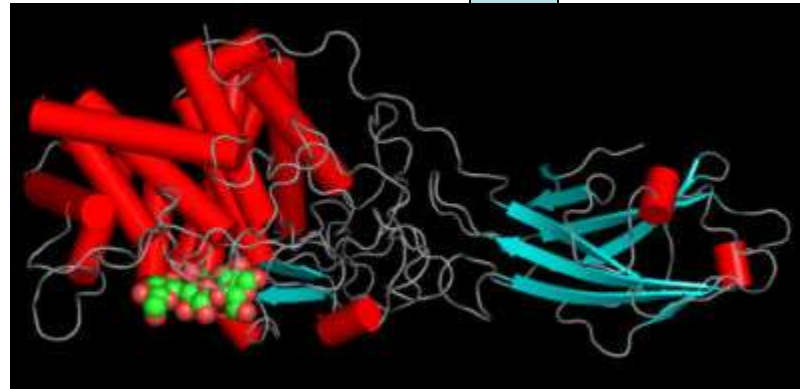
Homework: know the parts of our digestive system



◀ **Figure 41.8 The human digestive system.** After food is chewed and swallowed, it takes 5–10 seconds for it to pass down the esophagus and into the stomach, where it spends 2–6 hours being partially digested. Final digestion and nutrient absorption occur in the small intestine over a period of 5–6 hours. In 12–24 hours, any undigested material passes through the large intestine, and feces are expelled through the anus.



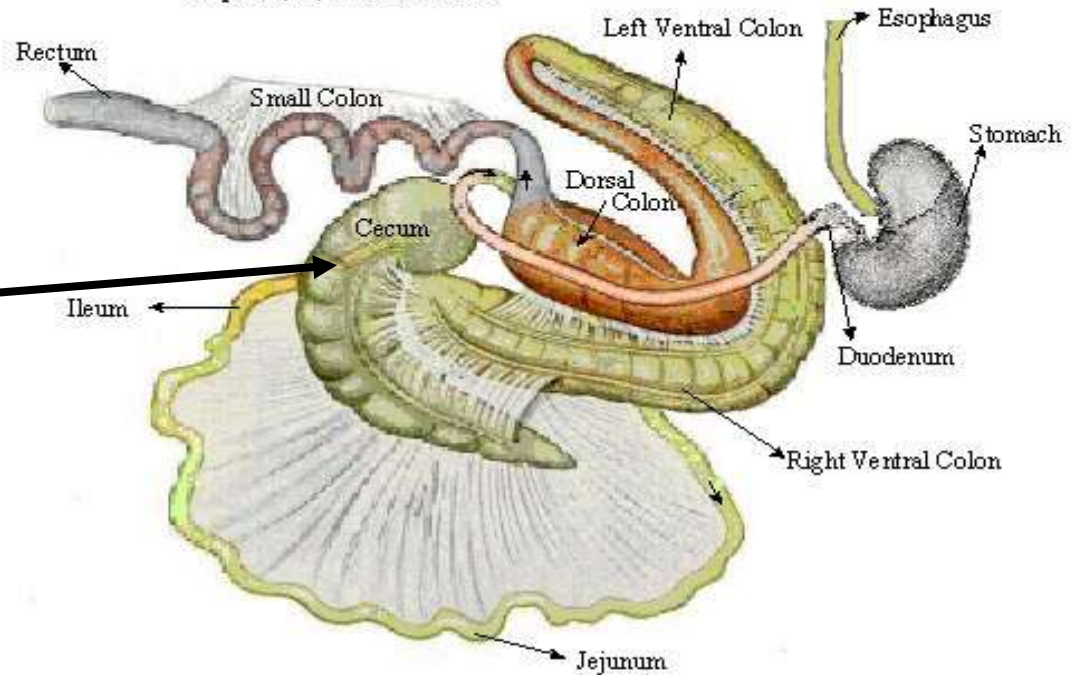
cellulose



cellulase
-produced by some
microorganisms
(bacteria and protozoa)

Equine Digestive Tract

Reference: Adapted from Atlas of Topographical Anatomy of the Domestic Animals, Popesko, P., W. B. Saunders



A caecum (or cecum) developed during the evolution of plant eating animals. In horses, it can have >30 litre capacity

Type	Fore Gut	Capacity	% of Gastrointestinal System
Enzymatic Digestion	Stomach	8 - 15 litres	8%
	Duodenum, Jejunum, Ileum (70 ft. or 21 meters)	68 litres	30%
Type	Hind Gut	Capacity	% of Gastrointestinal System
Microbial Digestion	Cecum (4 ft. or 1.2 meters)	28 - 36 litres	15%
	Large Colon (Right Ventral, Left Ventral and Dorsal Colons) (10 - 12 ft. or 3 - 3.6 meters)	86 litres	38%
	Small Colon (10 - 12 ft. or 3 - 3.6 meters)	16 litres	9%

Rabbits are well adapted



See Fig. 41.4 in your text

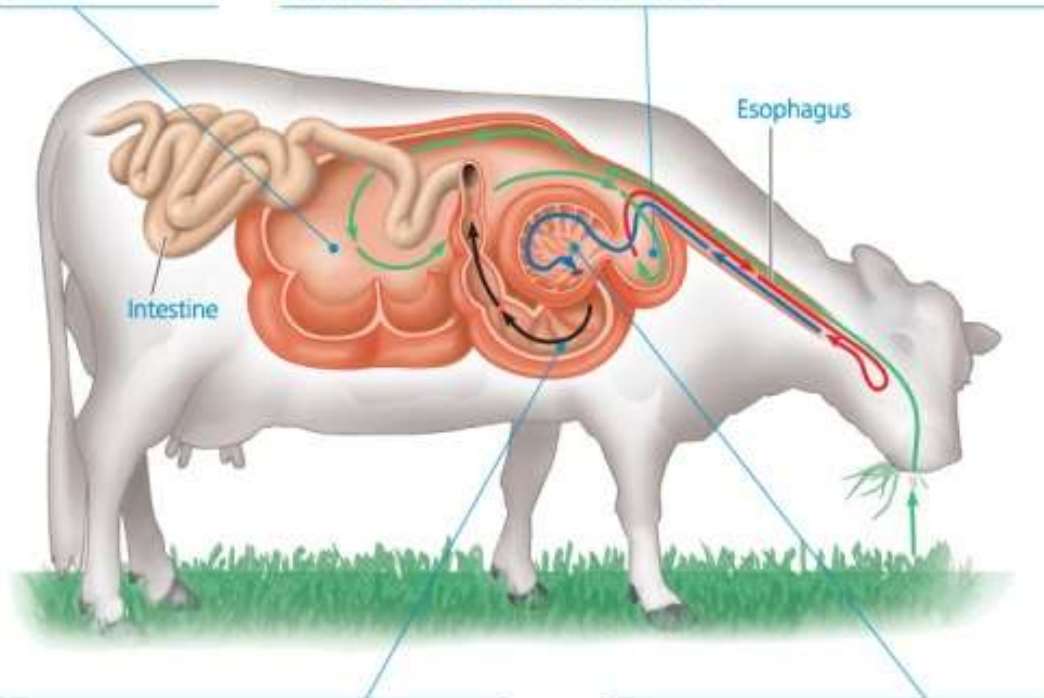
Homework: Please read about ruminants, pg. 954

1 **Rumen.** When the cow first chews and swallows a mouthful of grass, boluses (green arrows) enter the rumen.

2 **Reticulum.** Some boluses also enter the reticulum. In both the rumen and the reticulum, mutualistic prokaryotes and protists (mainly ciliates) go to work on the cellulose-rich meal. As by-products of their metabolism, the microorganisms secrete fatty acids. The cow periodically regurgitates and rechews the cud (red arrows), which further breaks down the fibres, making them more accessible to further microbial action.

◀ **Figure 41.17 Ruminant digestion.**

The stomach of a ruminant has four chambers. Because of the microbial action in the chambers, the diet from which a ruminant actually absorbs its nutrients is much richer than the grass the animal originally eats. In fact, a ruminant eating grass or hay obtains many of its nutrients by digesting the mutualistic microorganisms, which reproduce rapidly enough in the rumen to maintain a stable population.



4 **Abomasum.** The cud, containing great numbers of microorganisms, finally passes to the abomasum for digestion by the cow's own enzymes (black arrows).

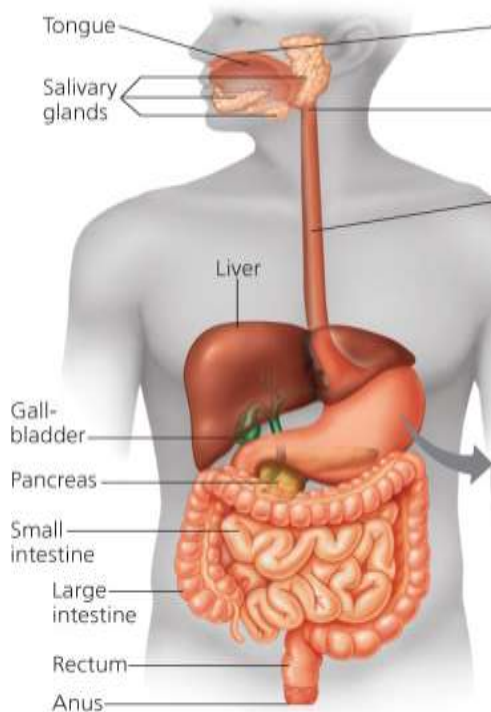
3 **Omasum.** The cow then reswallows the cud (blue arrows), which moves to the omasum, where water is removed.

Research news: Dr. Steven Chu (Nobel laureate in Physics) is currently studying termites since they are much more efficient at converting cellulose into ethanol than some of our current industrial processes. He suggests this may be a better and cheaper way of producing “green energy”.



Digestion

Complex organic molecules \longrightarrow simpler molecules
digestion

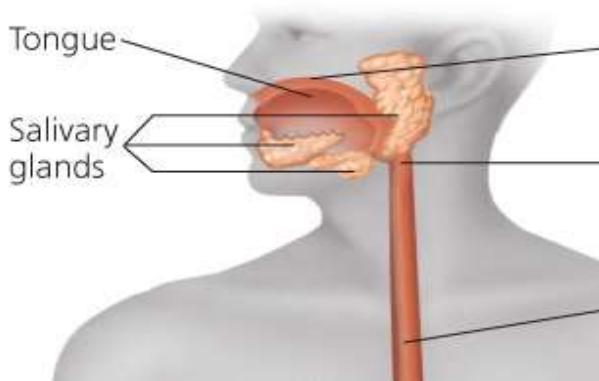


Marieb, Elaine N.; Hoehn, Katja, Human Anatomy & Physiology, 8th Edition
Electronically reproduced by permission of Pearson Education, Inc, Upp

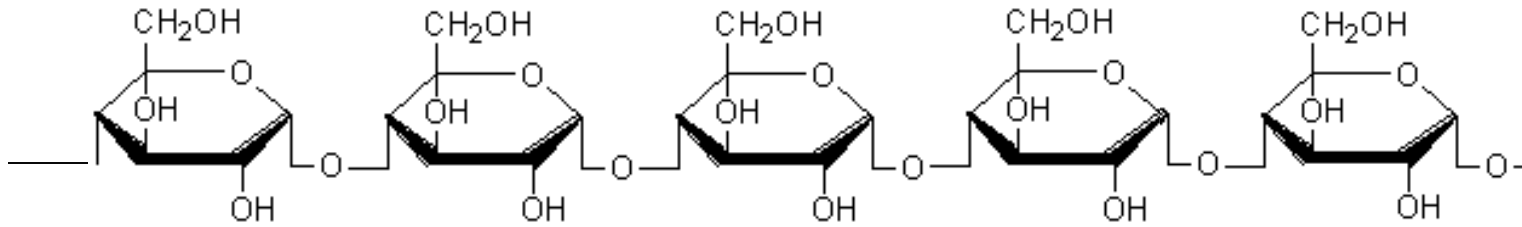
Digestion

Complex organic molecules \longrightarrow simpler molecules
digestion

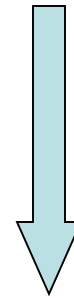
Digestive enzymes



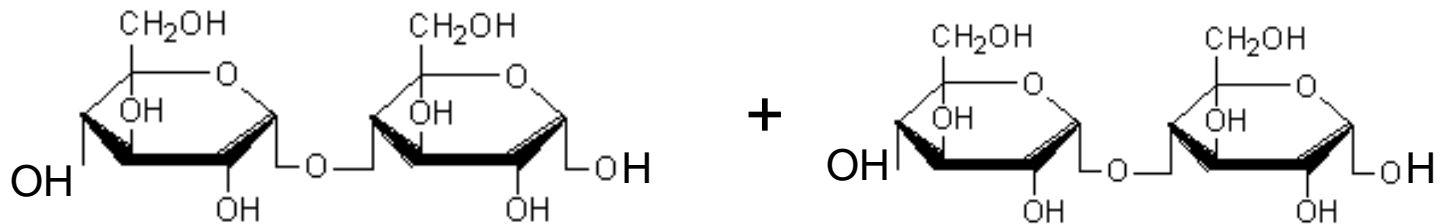
1. Oral cavity: teeth, salivary glands (in humans 3 pairs of major glands + ~600 smaller ones)



Starch = a polysaccharide

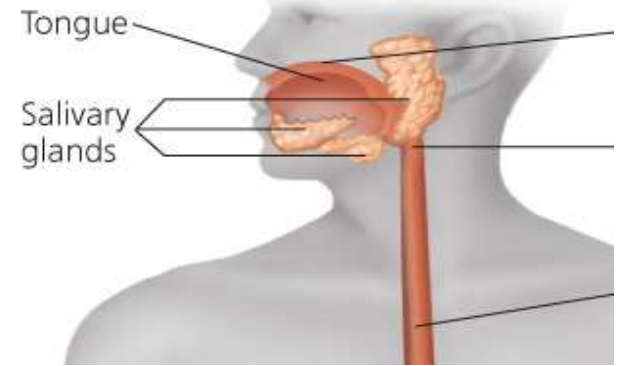


amylase



Maltose = 2 glucose units (disaccharide)

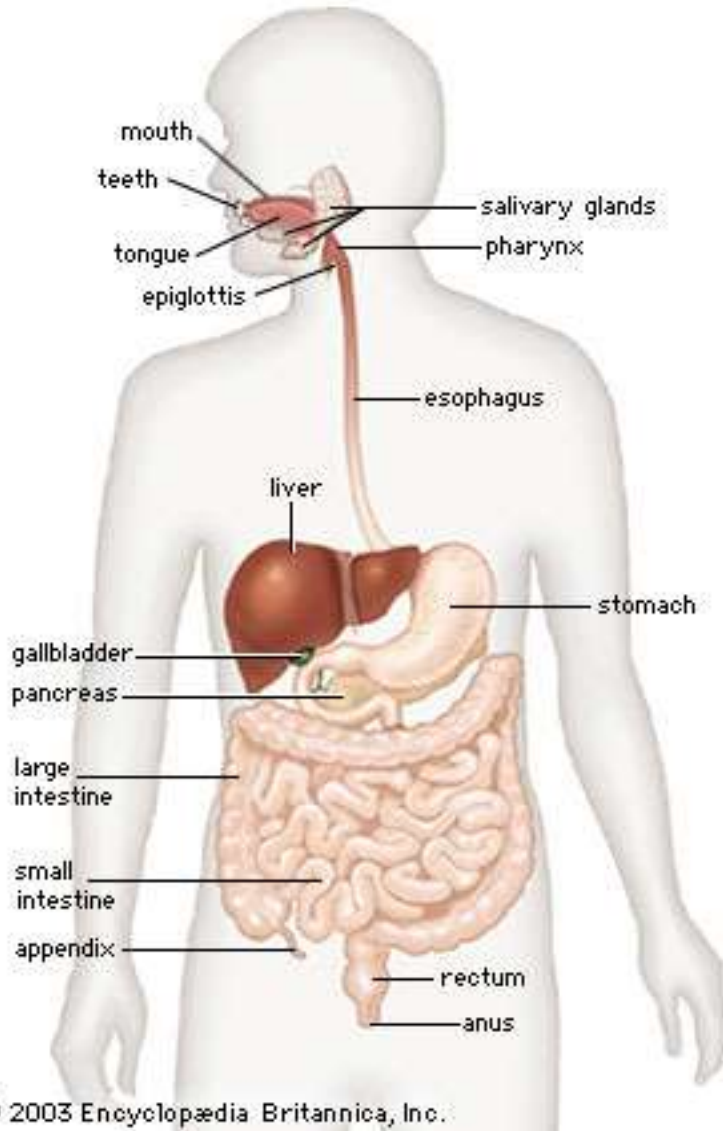
salivary amylase



2. The stomach

When food enters our stomach, gastric juice is secreted

Homework: please read about crops, the gizzard and the stomach (pg.946-949)



Gastric Juice

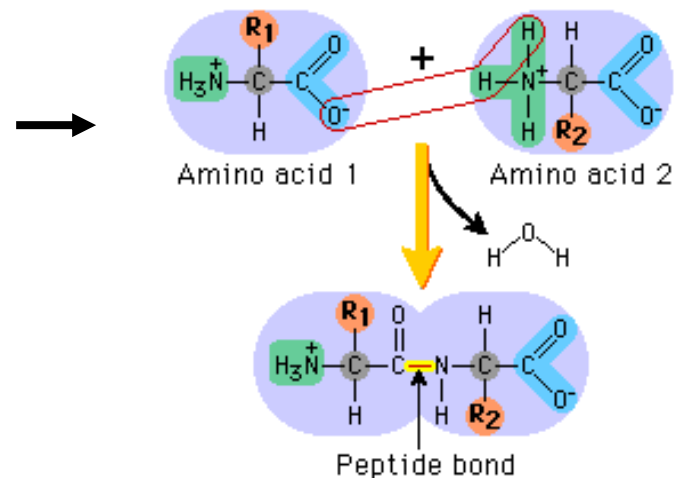
1. HCl
2. pepsin

Pepsin is a protease (member of an enzyme family that is important for the hydrolysis of proteins)



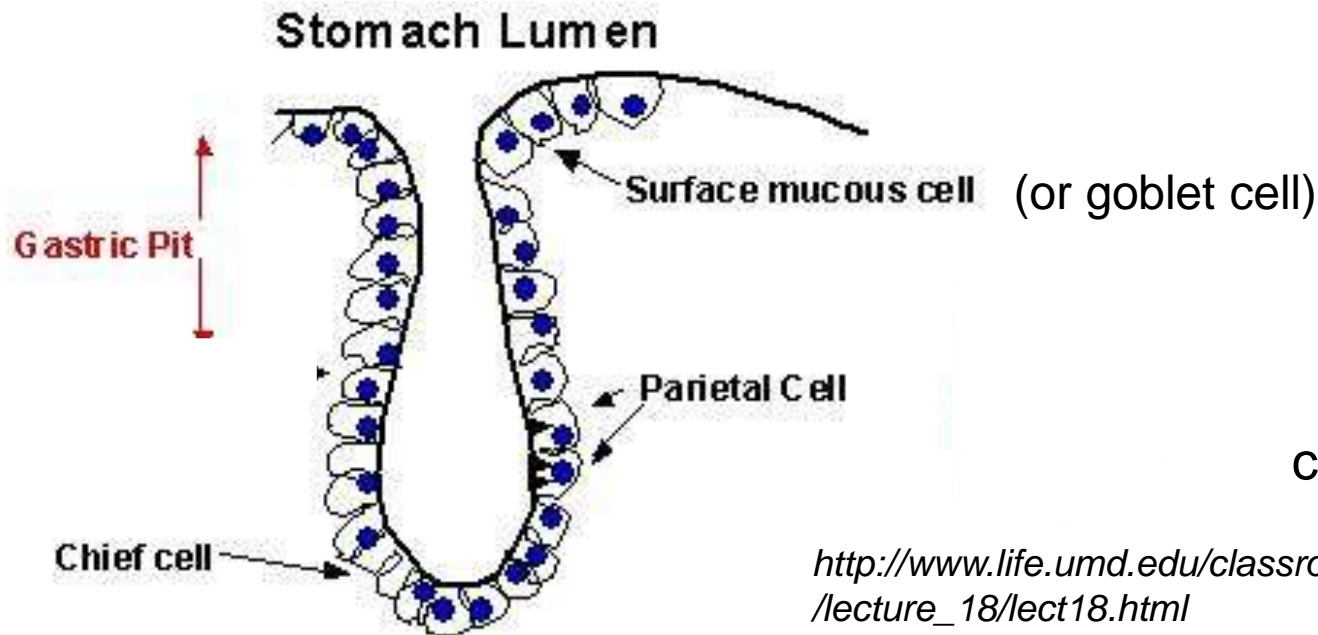
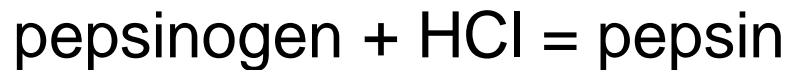
pepsin

Note: This is a review from Biology 102



So, why don't the cells in the stomach lining get hydrolyzed by pepsin and damaged by HCl?

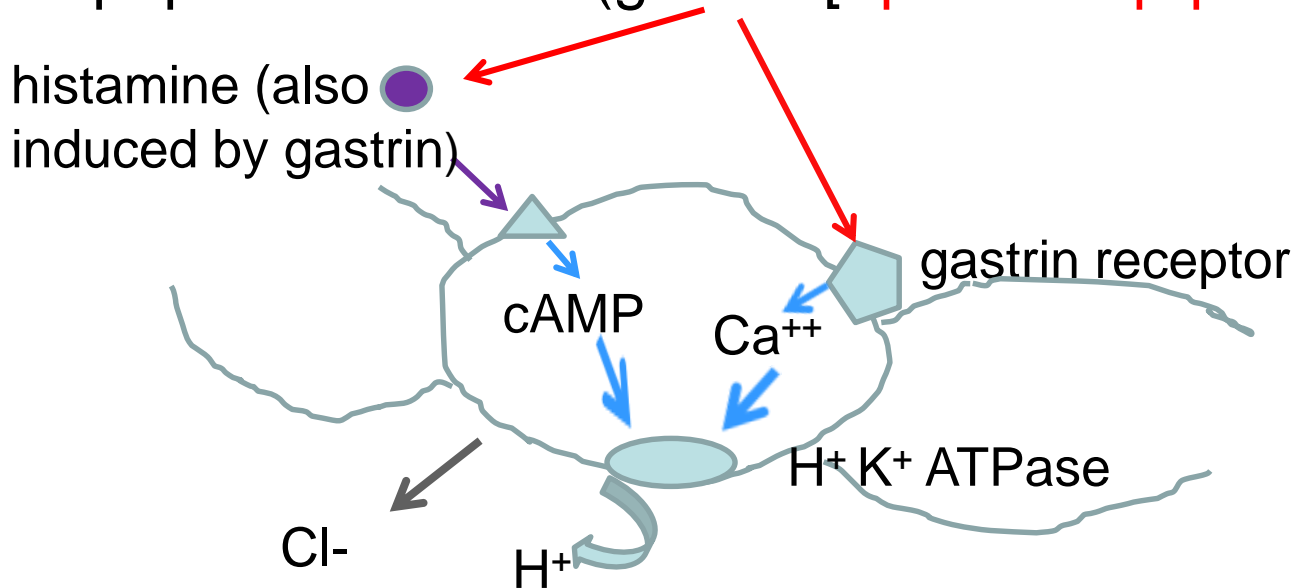
1. Gastric glands in the chief cells lining the stomach produce pepsin in an inactive form, pepsinogen (inactive enzymes are called zymogens and the pepsin zymogen = pepsinogen).



continued.....

Why cells in the stomach lining don't get hydrolyzed by pepsin and damaged by HCl continued....

2. Goblet cells that line the stomach produce a viscous mucous layer with a pH ~6
3. The parietal cells that produce the HCl don't accumulate the acid. It forms in the stomach after secretion of H^+ and Cl^- ions. (Note: the parietal cells are signaled to produce HCl by a peptide hormone (gastrin [$trp-met-asp-phe-NH_2$]))



H^+ & Cl^- ultimately are derived from the circulatory system

HCl acid (forms in the stomach lumen)

4. Only a few years ago, it was shown that cells lining the stomach have particularly resistant membranes

Are ulcers caused by “excess stomach acid”
caused by stress as is often heard?



Medical news: ~11 years ago the Nobel prize in physiology or medicine was awarded to Warren and Marshall, two Australians who discovered that ulcers were associated with the bacterium, *Helicobacter pylori** and could be treated with antibiotics (see text pg 948-9).

Peptic ulcers
may lead to
bleeding,
perforation,
or other
emergencies



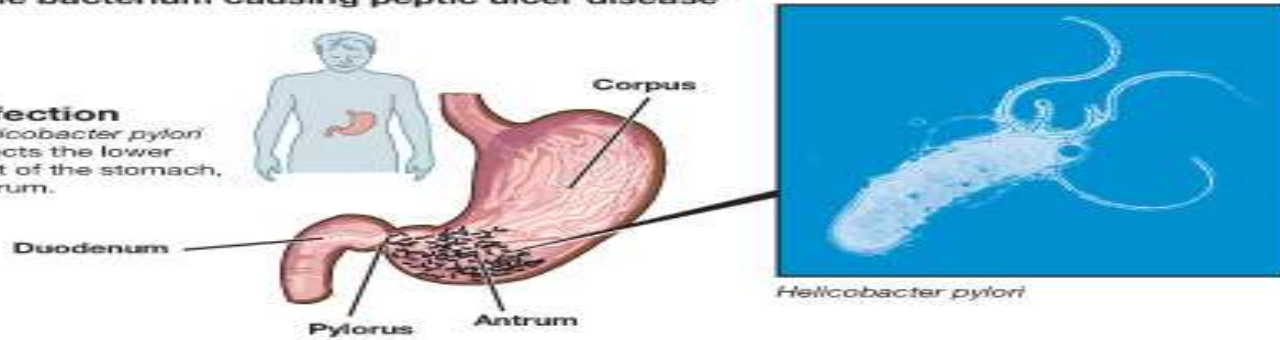
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* *New research:* gastrin stimulates the growth of these bacteria!

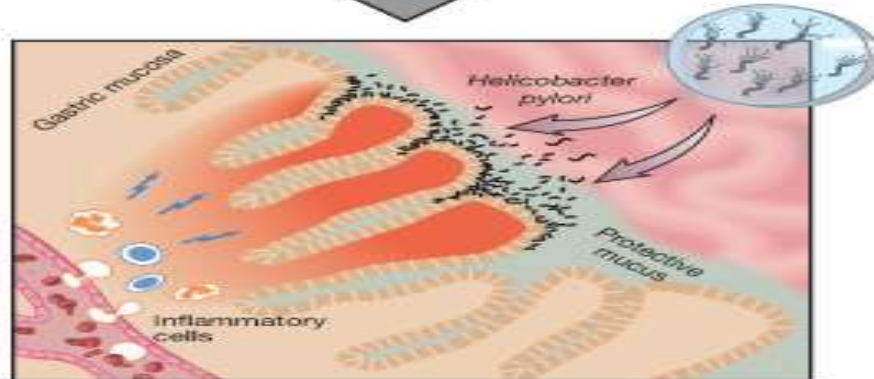
Helicobacter pylori

— the bacterium causing peptic ulcer disease

Infection
Helicobacter pylori infects the lower part of the stomach, antrum.



Inflammation
Helicobacter pylori causes inflammation of the gastric mucosa (gastritis). This is often asymptomatic.



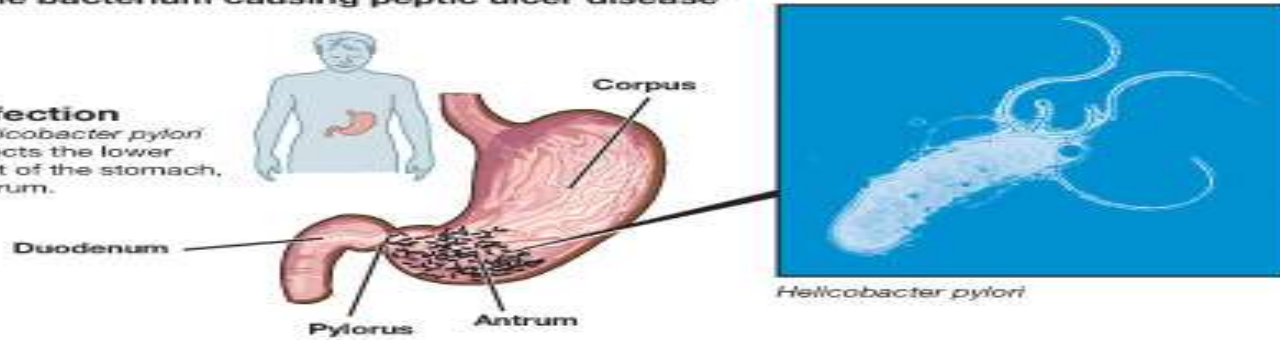
Ulcer
 Gastric inflammation may lead to duodenal or gastric ulcer. Severe complications include bleeding ulcer and perforated ulcer.



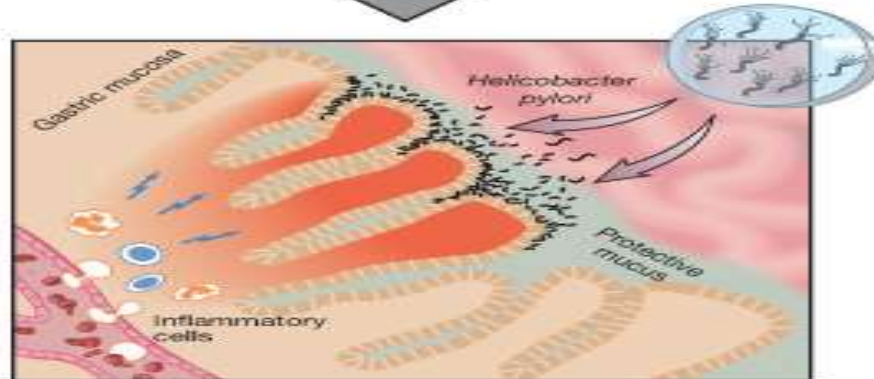
Helicobacter pylori

— the bacterium causing peptic ulcer disease

Infection
Helicobacter pylori infects the lower part of the stomach, antrum.



Inflammation
Helicobacter pylori causes inflammation of the gastric mucosa (gastritis). This is often asymptomatic.

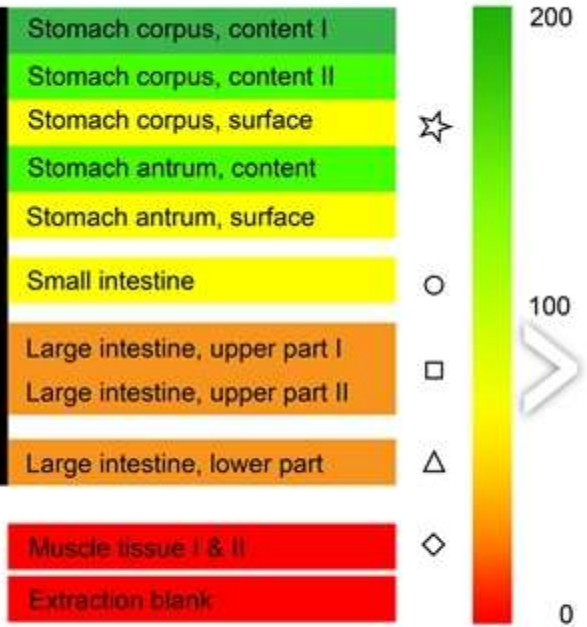
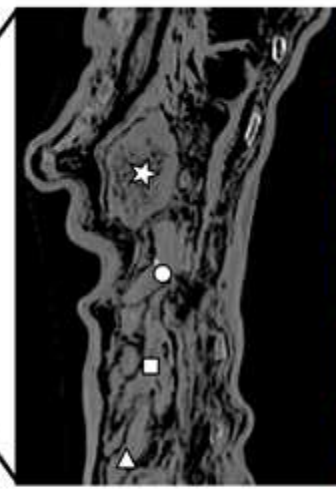


Ulcer
 Gastric inflammation may lead to duodenal or gastric ulcer. Severe complications include bleeding ulcer and perforated ulcer.



Iceman Melted to Reveal Stomach Bug

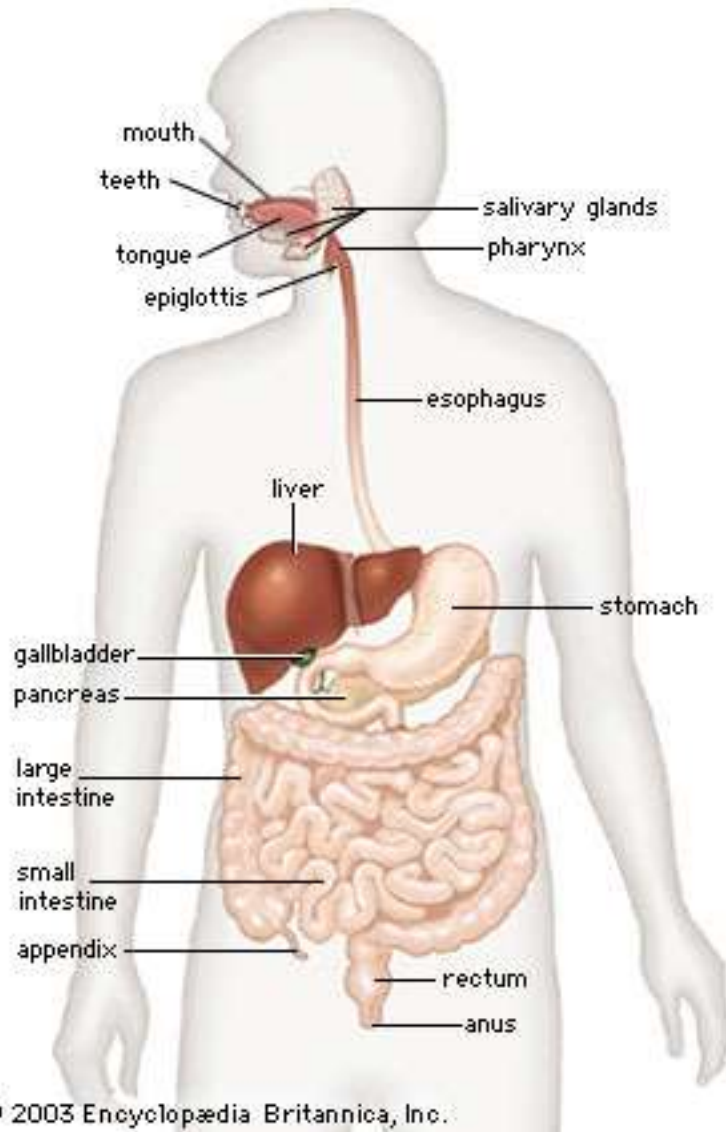
JAN 7, 2016 02:00 PM ET // BY ROSSELLA LORENZI



New DNA analysis has revealed that Oetzi, the 5,300-year-old mummy discovered in a melting glacier in the Italian Alps 25 years ago, harbored a pathogen in his stomach when he was murdered.

The bug, *Helicobacter pylori*, is common and gives people gastritis and stomach ulcers.

In order to make the discovery, scientists completely defrosted the mummy and took samples of its stomach.



3. Small intestine

The contents of the stomach (= acid chyme) then pass on to the small intestine where there is a large number of digestive enzymes produced by

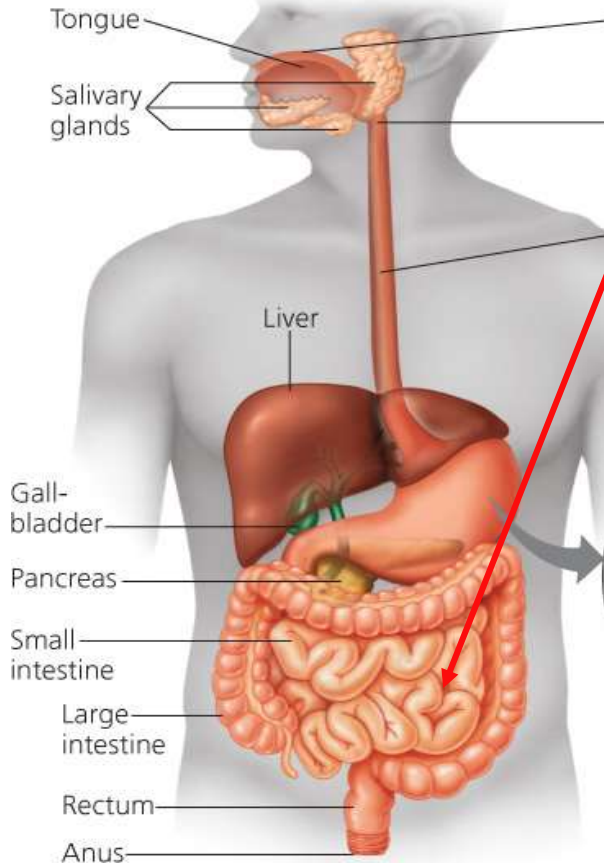
- (a) the intestinal glands
- (b) pancreas
- (c) liver

3. Small intestine

(a) the intestinal glands

(b) pancreas

(c) liver



Marieb, Elaine N.; Hoehn, Katja, Human Anatomy & Physiology, 8th Edition, 2012, Pearson Education, Inc. Electronically reproduced by permission of Pearson Education, Inc.

Summary:

Proteases



endoproteases

hydrolysis of peptide bonds
within a polypeptide

e.g. pepsin

trypsin

enterokinase

chymotrypsin

elastase

exoproteases

hydrolysis of terminal
peptide bonds

e.g. aminopeptidase

dipeptidase

carboxypeptidase

Digestion overview

(a) Intestinal glands: (i) maltase (ii) proteases = enterokinase, aminopeptidase, dipeptidase

(b) Pancreas: (i) proteases = carboxylpeptidase, trypsin, chymotrypsin, elastase

(ii) pancreatic amylase (starch \longrightarrow maltose)

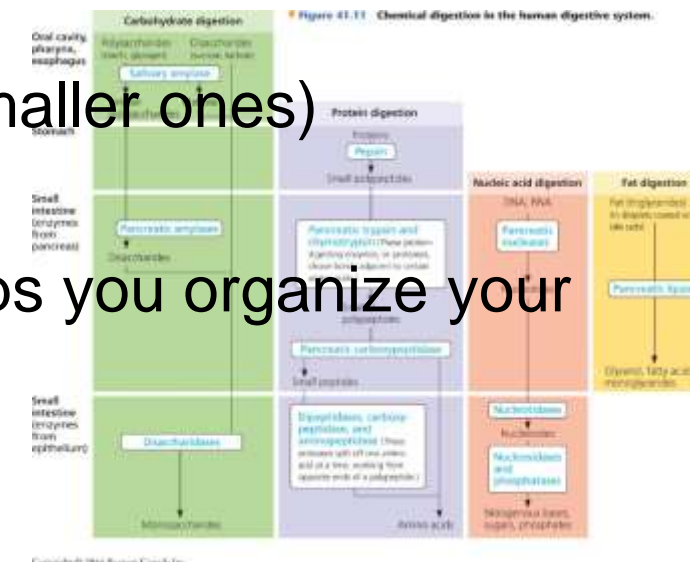
(iii) lipase (fats \longrightarrow fatty acids + monoglycerides)

(iv) nuclease (nucleic acids \longrightarrow nucleotides)

(v) bicarbonate ions (HCO_3^-)

(c) Liver: bile (large fat droplets \longrightarrow smaller ones)

* You may find that text Fig. 41.11 helps you organize your understanding



* We cannot do it all “ourselves”- please note that there is a very important role played by the bacteria that live in our digestive system (= our microbiome). We are only now discovering more of these contributions

What happens when we perturb our microbiome?

Abundance of bacteria (determined by DNA sequencing):

Control



25 mg/L Ag nanoparticles



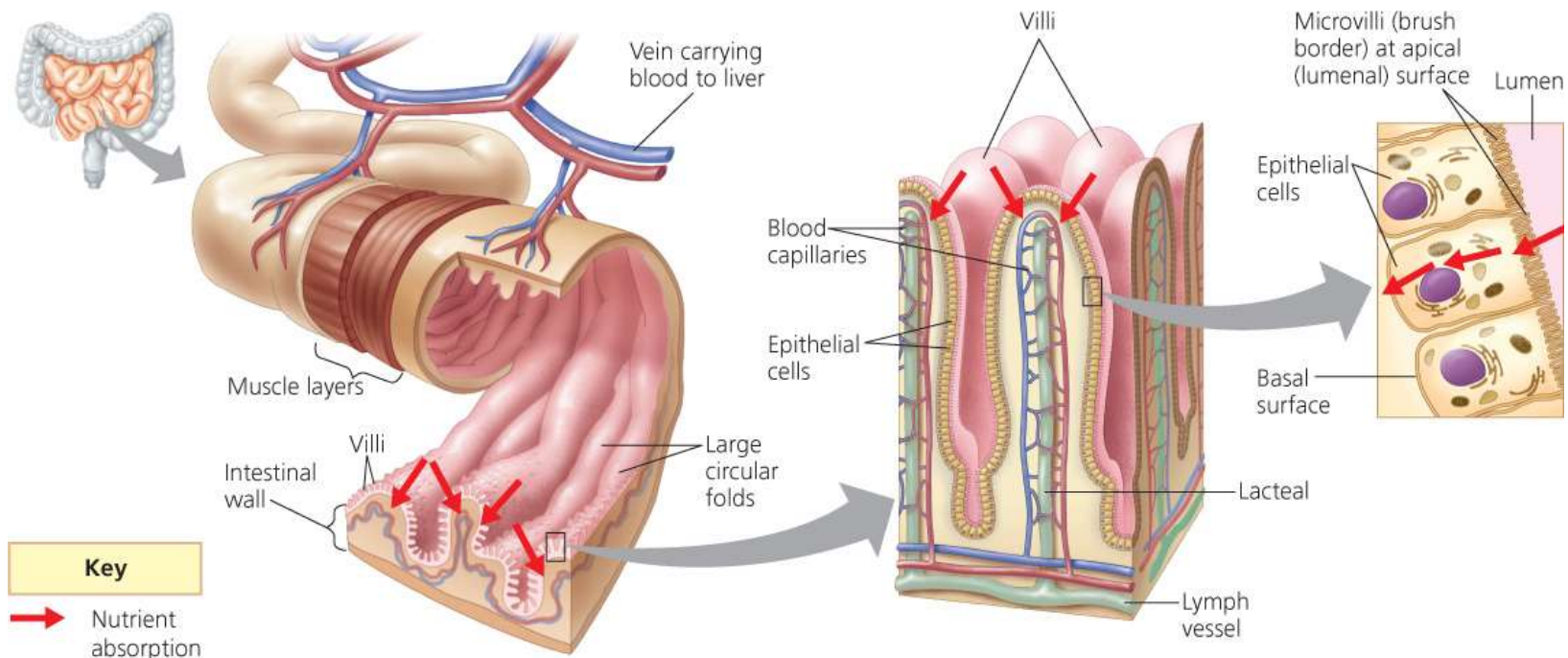
- Bacteroides ovatus
- Escherichia coli
- |
- Parabacteroides distasonis
- Roseburia faecis

- ↓ *Bacteriodes*: polysaccharide metabolism and nitrogen cycling
- ↑ *E. coli*: emerging ‘of interest’ in bowel disease
- ↓ *Parabacteriodes*: decreases intestinal inflammation
- ↓ *Roseburia faecis*: produces butyrate; used by intestinal epithelial cells

Research from my lab: 2014-16 **no, you don't need to read about it!**

Absorption

Absorption of the digested products, vitamins, minerals and water occurs in the small intestine. This is assisted by the huge surface area of the small intestine contributed by the villi (contains lacteals, and capillaries) and microvilli.



▲ **Figure 41.12** Nutrient absorption in the small intestine.

Homework: please read about absorption pg. 950-952

Digestion in insects

- Similar to digestion in vertebrates, but no pepsin
Why?
- A few interesting adaptations:
 - salivary glands (discussed)
 - active proteases eliminated with feces

