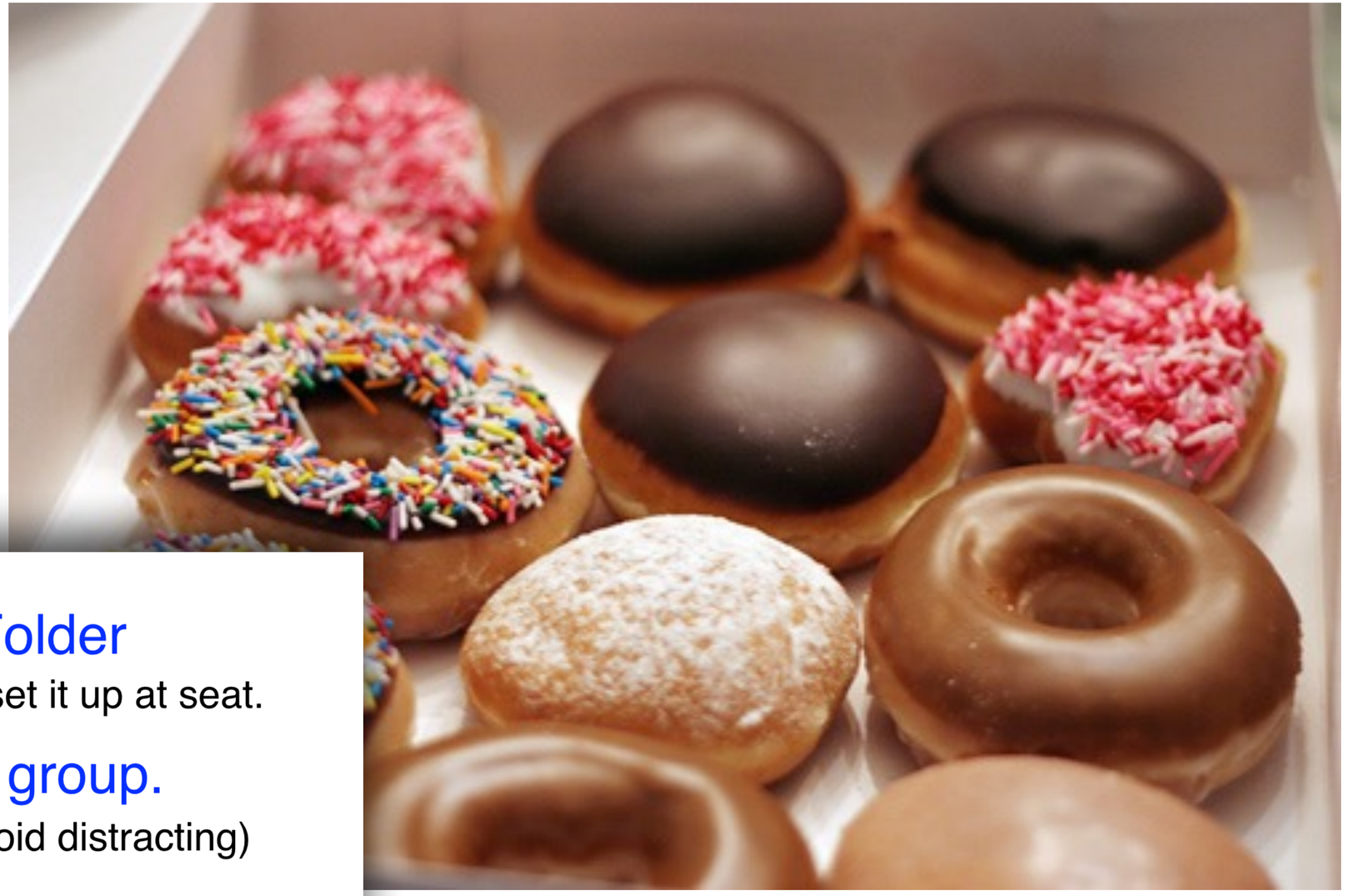


1. **Pick up** Name Folder

- Pick up name folder and set it up at seat.

2. **Sit** with your lab group.

- laptops almost closed (avoid distracting)

3. **Clicker** Attendance

- Launch your Top Hat, and get ready to click.

Budgeting homework time (70 min): Chapter 23, section 23.1 is 3891 words in length with 10 figures and three of which are data tables/figures that will require thinking and notetaking for the Trifecta. On the other hand, these Figures and Tables are not important, Figure 23.4, the second half of Table 23.2, the parietal cell video, and Figure 23.5. When done properly, when you pause to review important figures/tables, try Questions, and take notes, this assignment should take you more like 70 minutes.

1. _____ **For Thursday's lecture**, read **Chapter 23: Cells in Tissues**, first review the introductory page, and then carefully read section 23.1: "How do you break down and absorb nutrients from the food you eat?" (3891 words). As you read it be sure to take handwritten notes.

2. _____ **Try to answer these Integrating Questions : #1** about da Vinci, **#3** about Prout's research and **#7** on Muallem's work and be prepared to share your answers in class.

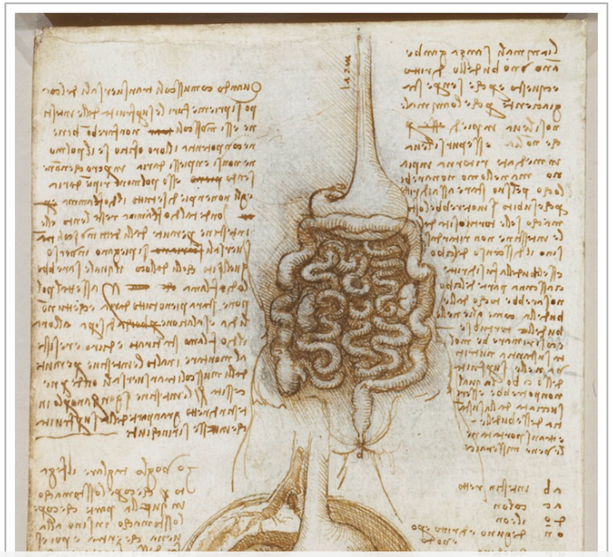
3. _____ (Trifecta): **Prepare to explain (aloud) Tables 23.1, 23.2(first half) and Figure 23.7 in class** (Purpose, Methods, Findings).

23.1 How do you break down and absorb nutrients from the food you eat?

- Context: Populations of cells perform different tasks within multicellular organisms.
- Major themes: Cell structure relates to function, cells maintain internal environments that differ from their external environments, and cells communicate with other cells.
- Bottom line: Multicellular organisms must have mechanisms to absorb nutrients, and digestive systems have populations of cells that perform specific functions.

Biology Learning Objectives

- Explain how cells of the digestive system break down and absorb nutrients.
- Describe the gross anatomy of the mammalian digestive system.
- Explain the major sites of absorption for proteins, carbohydrates, and water in the mammalian digestive system.



In most multicellular organisms, cells make up **tissues,** which make up organs, which make up organ systems. There may be various populations of cells, or tissues, within one organ. For instance, you learned how populations of muscle cells respond to exercise. In the course of that investigation, you learned that muscles are composed of more than just muscle cells. *{Connections: Muscle cells are examined in Section 9.2.}* There are populations of connective tissue cells, neurons, and other cells. Some of the cells within a tissue are actually part of other systems. Neurons are part of the nervous system and yet are found in all other systems. The cells, tissues, organs, and systems are all highly interconnected, and you will examine the impact of one population of cells upon an entire organism.

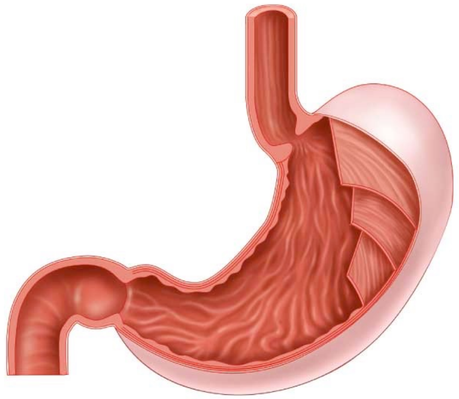
Even though you are investigating cells at the tissue and organismal levels in this chapter, you can think about your digestive system as an organ system that illustrates all of the Big Ideas of biology. Specialized populations of *cells* exist along the internal lining of the **alimentary canal** that are critical in secreting digestive enzymes and absorbing nutrients. Molecular *information* is detected by those cells, which triggers release of enzymes and performs other functions as needed after a meal is ingested. The processes of **ingestion, digestion,** and **assimilation** are critical for maintaining *homeostasis*. The components of the digestive system have evolved in different animals for the particular diet of each animal. The entire system is an *emergent property* that arises from

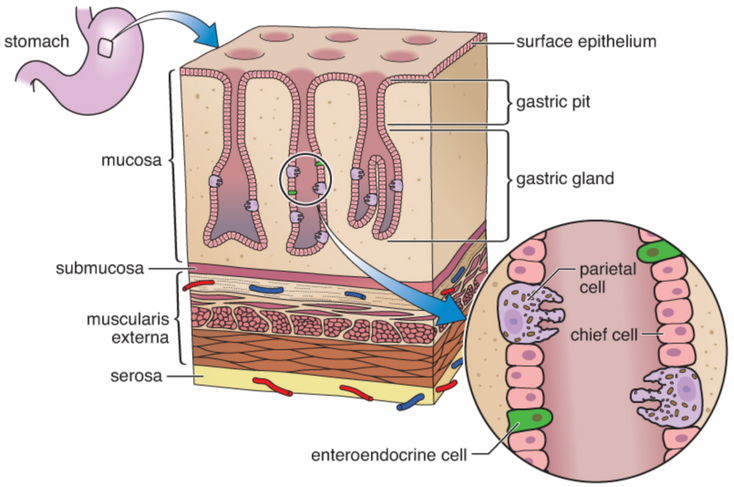
When does digestion begin?

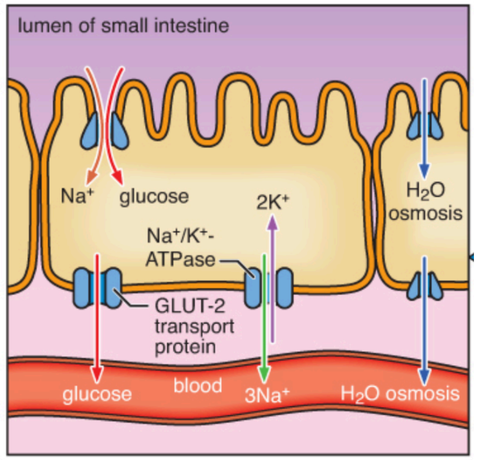
Digestion: Stomach

When you are eating, describe the process of digestion in the Stomach.

What **hormones** -> stimulate which **cells** -> to secrete what **enzymes/other** (or perform what **mechanical** actions) -> to digest what **food** molecules?







Prout
(Stomach)

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exact amount of base to neutralize acid (second fraction) (g)	1.56	0.76	0.40
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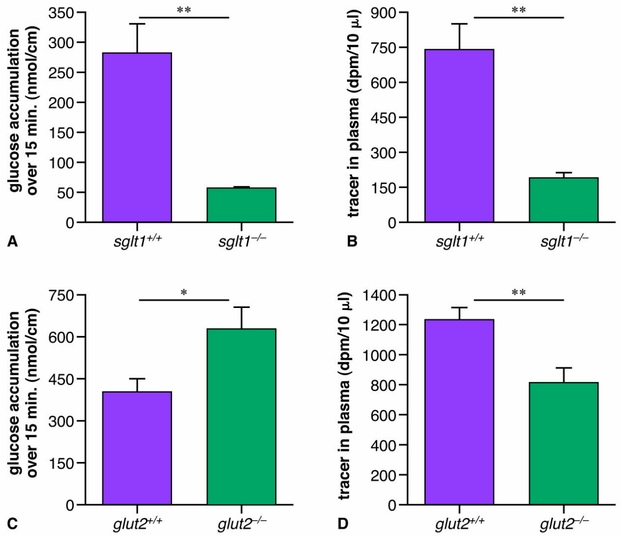
total amount of chloride (g)	3.27	3.93	4.83
other acids (fourth fraction)	0	0	0

Muallem
(Parietal cell)

treatment	ΔpH
no Na^+	−0.58
Na^+ added after exposed to no Na^+	0.56
Na^+ and Na^+/H^+ exchange inhibitor added after exposed to no Na^+	0
histamine	0.130 ± 0.038

Roder
(Absorption)



William Prout, 1823,

Purpose: Determine if stomach secreted an acid, if so which?

Methods: Animal experiments + dissection
(rabbits, horses, cows, dogs)

1. - feed animal, kill immediately, remove 'contents' of stomach
2. quantify amount acid
   - exposed 'contents' to distilled water + combine mixtures
   - next divide mixture into four portions (equal) #1, 2, 3 4

fractions

#1 → dry, burn, redissolve in dH_2O
use silver nitrate to react with Cl^- to determine $[Cl^-]$

#2 → neutralize [to pH 7.0] soln w/ KOH tritration

#3 → add large [KOH] → neutralize all to KCl + H_2O
use silver nitrate to determine $[Cl^-]$

#4 → look for evidence of ~~other~~ acids

→ data Table 23.1 Findings → it's HCl

*The Origin of the Hydrochloric Acid in the Gastric Tubules.**

By Mabel Purefoy FitzGerald.

(Communicated by Prof. A. B. Macallum, F.R.S. Received June 4, 1910.)

(From the Biochemical Laboratory of the University of Toronto.)

[Plates 7—9.]

In 1823 William Prout† brought forward the view that the acid normally existing in the stomach was free hydrochloric acid, or to quote his own words, "free or, at least, unsaturated muriatic acid." This opinion was based on the analyses made by him of the gastric contents of the rabbit and of other animals, and of the fluid ejected from the human stomach in severe cases of dyspepsia. He said further: "With respect to the nature of this acid, very various opinions have been entertained. Some of the older chemists seem to have considered it as an acid *sui generis*; by others it was supposed to be the phosphoric, the acetic, the lactic, etc. No less various have been the opinions respecting its origin and use, some supposing that it is derived from the stomach itself, and is essential to the digestive process, others that it is derived from the food, or is a result of fermentation, etc.; in short, there seems to be no physiological subject so imperfectly understood, or concerning which there has been such a variety of opinions."

These words written in retrospection by the first exponent of the free hydrochloric acid theory, when read in the twentieth century, have the significance also of a prognostication, for during the past eighty-seven years interminable discussion has ensued between the advocates of the mineral and organic acid theories respectively, and in spite of the efforts of the physiologist, biologist, and bio-chemist in their several fields, uncertainty still exists on many and similar points. This is true in particular of the structure or structures of the gastric mucosa directly concerned with the formation and secretion of the hydrochloric acid, as well as of the existence even of hydrochloric acid in a demonstrable form within the gland tubules.

Article

The origin of the hydrochloric acid in the gastric tubules

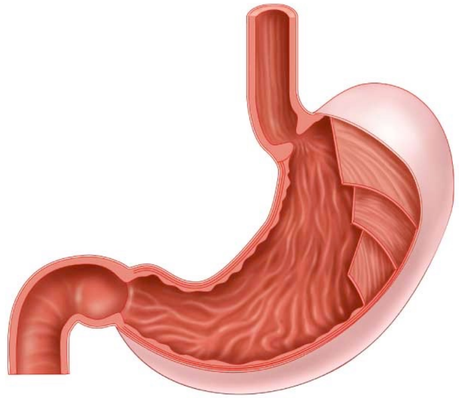
Mabel Purefoy Fitzgerald

Published: **26 November 1910** | https://doi.org/10.1098/rspb.1910.0067

Abstract

In 1823 William Prout brought forward the view that the acid normally existing in the stomach was free hydrochloric acid, or to quote his own words, "free or, at least, unsaturated muriatic acid." This opinion was based on the analyses made by him of the gastric contents of the rabbit and of other animals, and of the fluid ejected from the human stomach in severe cases of dyspepsia. He said further: "With respect to the nature of this acid, very various opinions have been entertained. Some of the older chemists seem to have considered it as an acid *sui generis*; by others it was supposed to be the phosphoric, the acetic, the lactic, etc. No less various have been the opinions respecting its origin and use, some supposing that it is derived from the stomach itself, and is essential to the digestive process, other that it is derived from the food, or is a result of fermentation, etc. ; in short, there seems to be no physiological subject so imperfectly understood, or concerning which there has been such a variety of opinions." These words written in retrospection by the first exponent of the free hydrochloric acid theory, when read in the twentieth century, have the significance also of a prognostication, for during the past eighty-seven years interminable discussion has ensued between the advocates of the mineral and organic acid theories respectively, and in spite of the efforts of the physiologist, biologist, and bio-chemist in their several fields, uncertainly still exists on many and similar points. This is true in particular of the structure or structures of the gastric mucosa directly concerned with the formation and secretion of the hydrochloric acid, as well as of the existence even of hydrochloric acid in a demonstrable form within the glad tubules.

Prout
(Stomach)



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Trifecta!

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Explain these results

Table 23.1

From Prout, 1824.

Results from chloride analysis of stomach contents of three rabbits

fraction of solution	rabbit 1	rabbit 2	rabbit 3
chloride salts (first fraction) (g)	0.12	0.95	1.71

Prout used silver nitrate to react with chloride salts to determine amount of chloride ion.

Table 23.1

From Prout, 1824.

Results from chloride analysis of stomach contents of three rabbits

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Prout used a known amount of potassium hydroxide to exactly neutralize the solution, to determine free acid present.

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From Prout, 1824.

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Prout added a large quantity of KOH, which neutralized all HCl to $KCl + H_2O$.

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Total chloride is sum of 1st 3 fractions.

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total amount of chloride (g)	3.27	3.93	4.83

Total chloride is sum of 1st 3 fractions.

Numbers vary from rabbit to rabbit, but total amount of chloride is fairly consistent.

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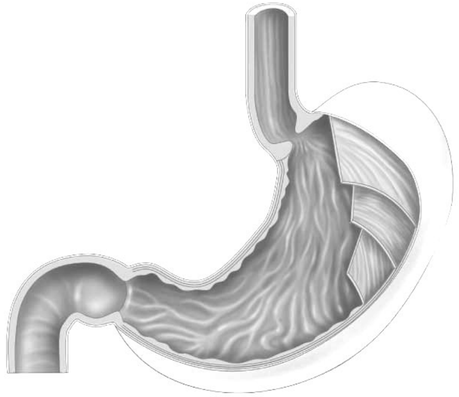

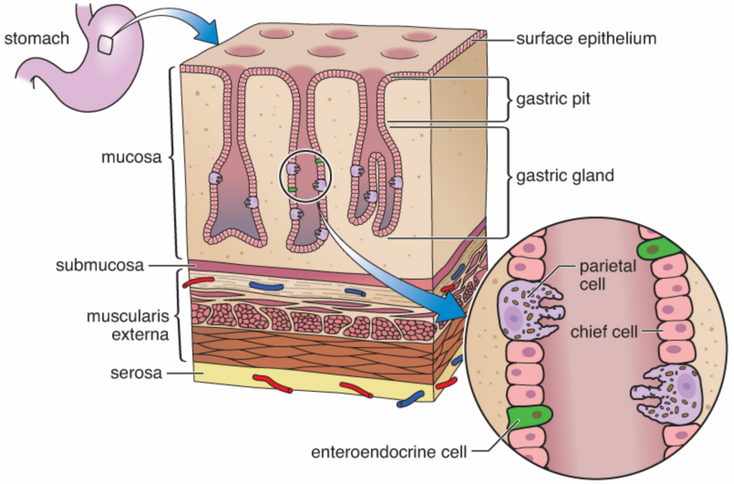

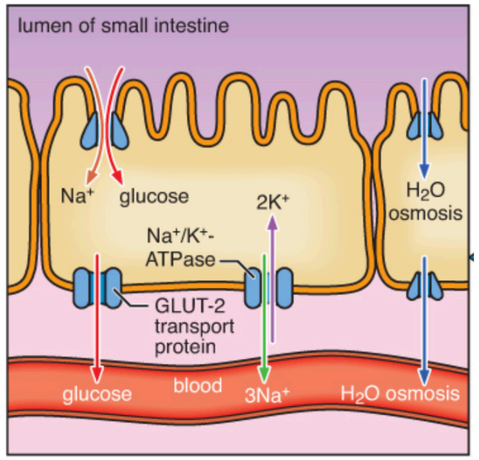

Prout
(Stomach)

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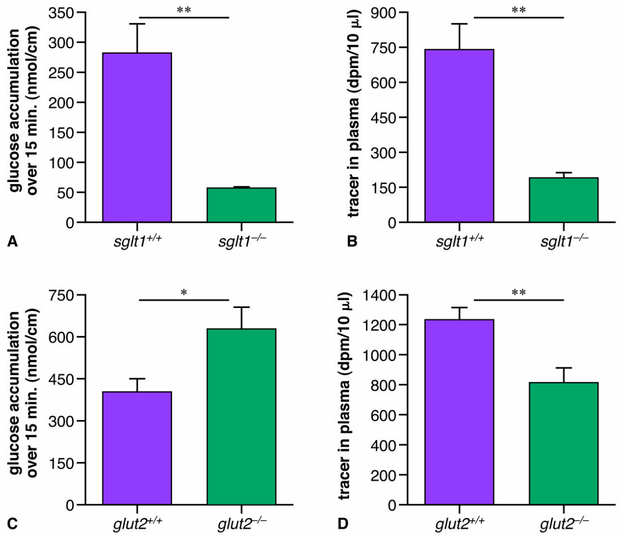
Muallem
(Parietal cell)

treatment	ΔpH
no Na^+	−0.58
Na^+ added after exposed to no Na^+	0.56
Na^+ and Na^+/H^+ exchange inhibitor added after exposed to no Na^+	0
histamine	0.130 ± 0.038

Roder
(Absorption)



Shumel Muallem etal 1988 — UCLA then later Texas

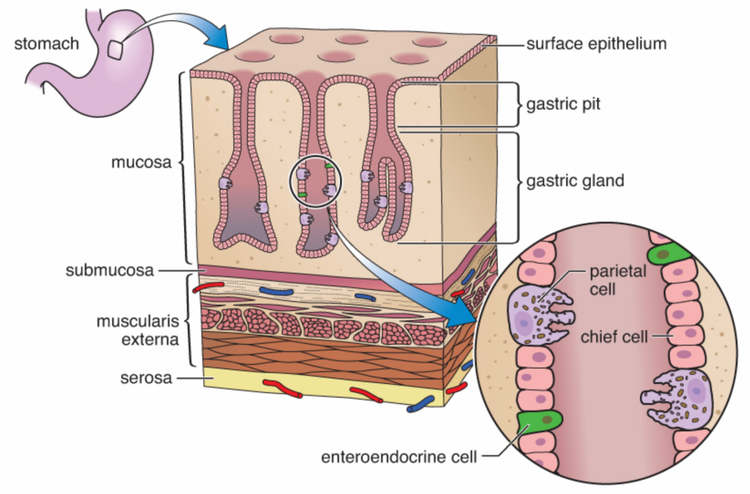
Purpose: Determine how stimulation of parietal cells affected pH inside the parietal cell.

(If H+ leaves cell its internal pH becomes more alkaline)

Methods: Cellular experiments - seperate parietal cells
- isolate rabbit upper stomach, use centrifuge to seperate parietal cells from other epithel.
- measure cellular internal pH with flourescent dye that is membrane permeable. emission at two λ's ratio = pH
- incubate cells w/ dye 20 min, then wash away any dye left outside cells.

1. Expose cells to solution missing sodium Na^+ ions
   → then pH_i → ↓ acidifies
   Return Na^+ to the solution pH_i → ↑ alkaline
2. Add chemical (ameloride?) that inhibits Na/H exchan
   slowed pH_i → ↑ when return Na^+ to solution in # 1
3. Add histamine → a stimulant of parietal cells
   Add histamine receptor blocker + Add forksolin ↑ cAMP

Muallem
(Parietal cell)



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histamine	0.130 ± 0.038

Muallem and colleagues performed a study to determine how stimulation of the parietal cells affected parietal cell internal pH. If pH of individual cells goes up, then you can conclude that hydrogen ions are being excreted from the cell and pH of the surrounding environment goes down. Although as you will see, there is a way to measure changes in pH inside and outside of the cells. *{Connections: Chloroplast pH is measured in a similar manner in Section 11.1.}* The scientists used a centrifugation technique to isolate parietal cells from the epithelium of the upper part of a rabbit's stomach. Keep in mind the structure of the parietal cell, especially the deep infolding on the lumen end of the cell, which increases the surface area for secretion.

The measurement of internal cell [redacted] as achieved through use of a dye that can permeate cells. This dye can be us[redacted]. The scientists measured the emission intensity of the dye at two differen[redacted]tensities is pH-dependent. Knowledge of the intensity ratio in solutions of known pH allows researchers to estimate intracellular pH. The scientists incubated cells with dye for 20 minutes and then washed the cells so that any dye not taken up by cells was washed away. Keep in mind that relatively small changes in pH, which is measured on a logarithmic scale, in single cell suspensions could actually lead to large decreases in pH in the stomach lumen.

Muallem and colleagues exposed cells to a medium that contained no sodium ions (Na^+) and found that intracellular pH went down (Table 23.2). When they added Na^+, the pH rapidly went up. Addition of a

treatment	ΔpH
no Na^+	−0.58
Na^+ added after exposed to no Na^+	0.56
Na^+ and Na^+/H^+ exchange inhibitor added after exposed to no Na^+	0
histamine	0.130 ± 0.038
no Na^+, then histamine	0.04

Trifecta

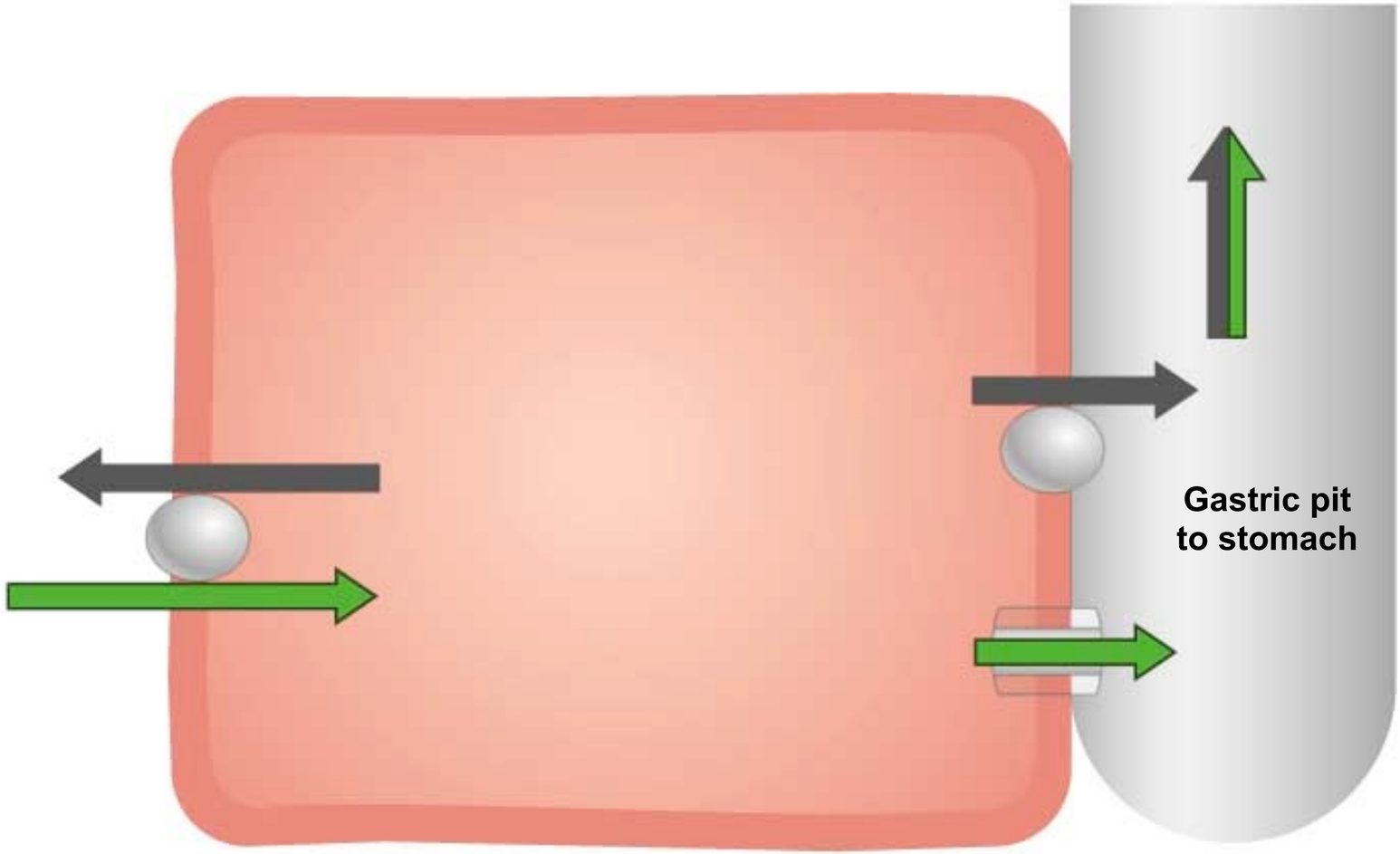
treatment	ΔpH
no Na^+	−0.58
Na^+ added after exposed to no Na^+	0.56
Na^+ and Na^+/H^+ exchange inhibitor added after exposed to no Na^+	0
histamine	0.130 ± 0.038

Explain these results

Table 23.2

Data from Muallem et al., 1988, text and Figures 2 to 8.

DRAW a model parietal cell



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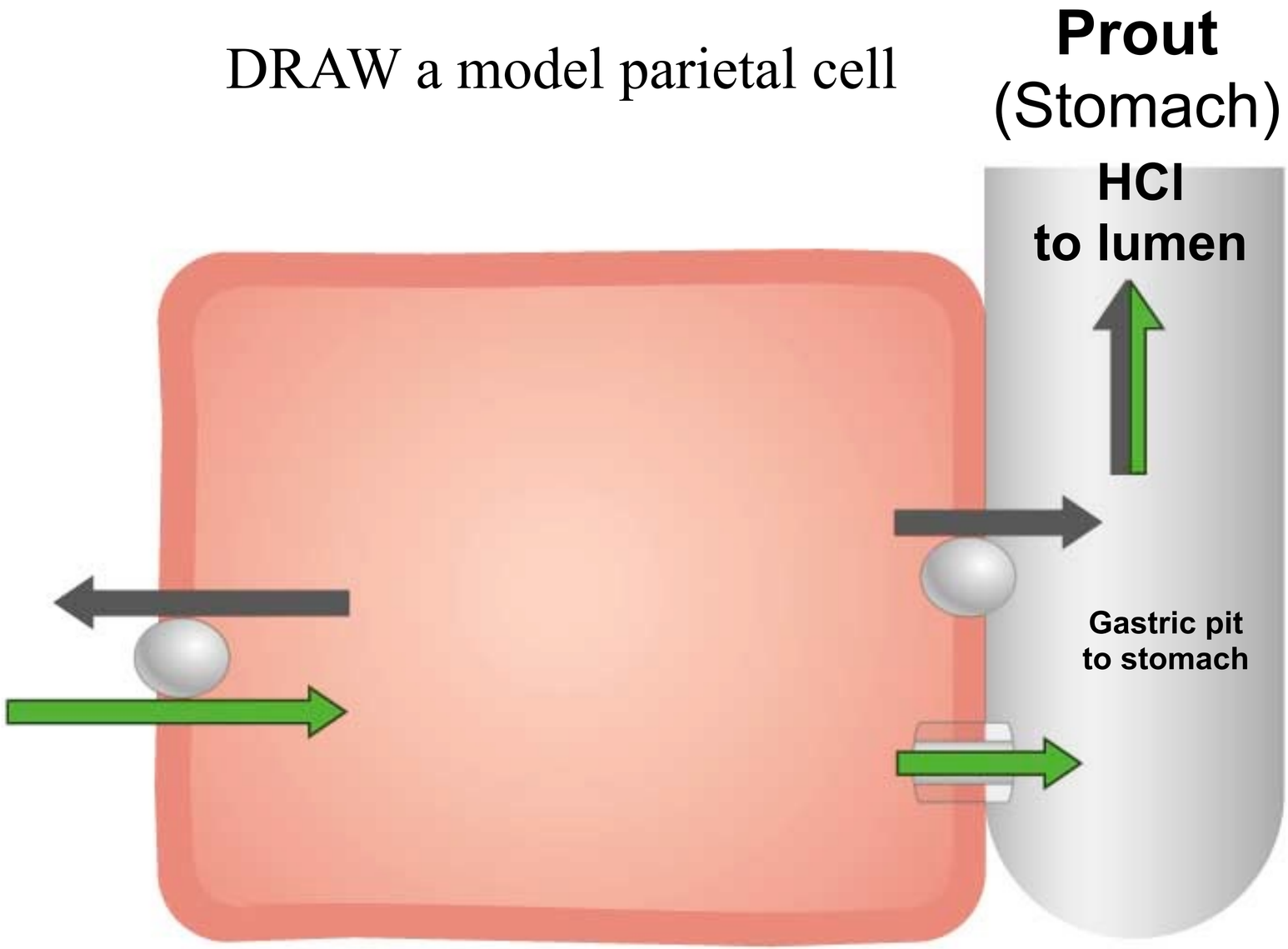
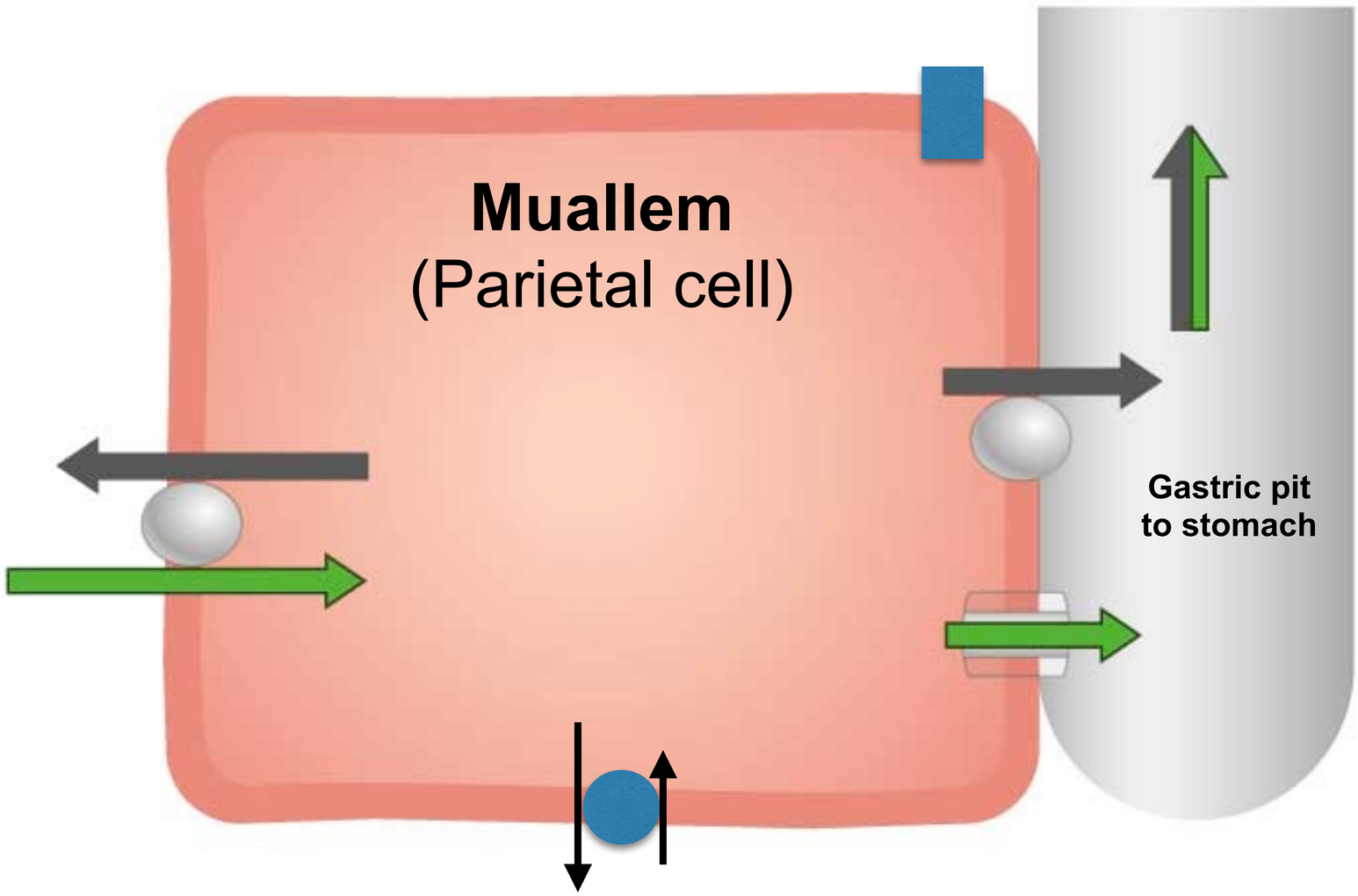
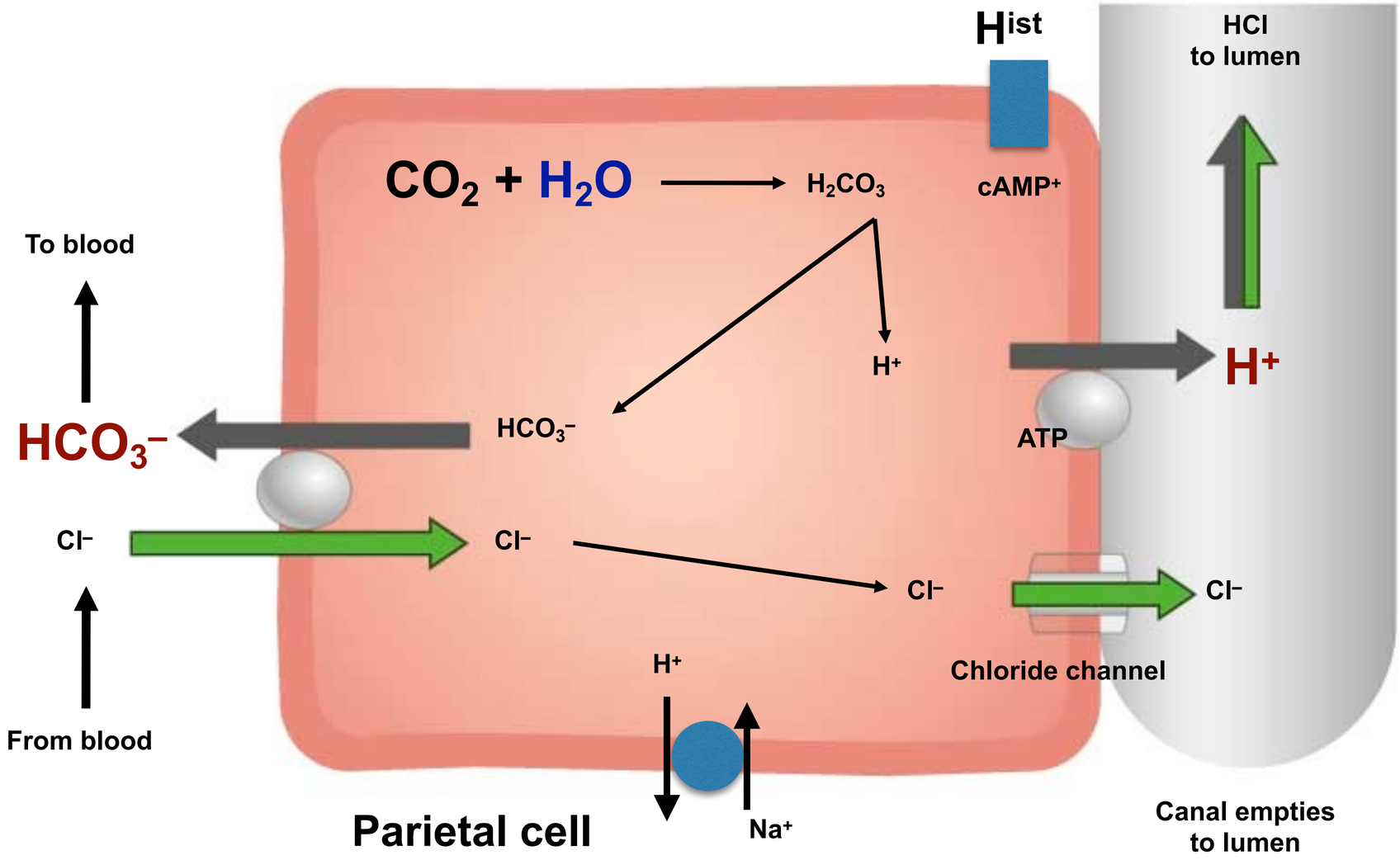


DRAW a model parietal cell



Secretion of HCl by parietal cells



THE JOURNAL OF BIOLOGICAL CHEMISTRY

Vol. 263, No. 29, Issue of October 15, pp. 14703–14711, 1988
Printed in U.S.A.

Activation of the Na^+/H^+ and Cl^-/HCO_3^- Exchange by Stimulation of Acid Secretion in the Parietal Cell*

(Received for publication, November 9, 1987)

Shmuel Muallem‡§, Douglas Blissard¶§, Edward J. Cragoe, Jr.‖, and George Sachs¶§

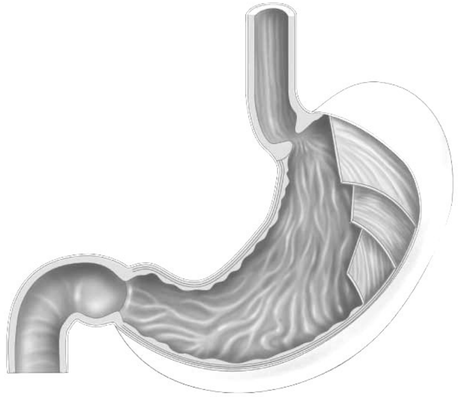
From the ‡Laboratory of Membrane Biology, Research Institute Cedars Sinai Medical Center, Los Angeles, California 90048, the ¶Center for Ulcer Research and Education, Veterans Administration, West Los Angeles, California 90073, the ‖Merck Institute, West Point, Pennsylvania 19487, and the §UCLA Department of Medicine and Physiology, Los Angeles, California 90024

Upon stimulation, the gastric parietal cell secretes a large quantity of isotonic HCl across its apical membrane which must be accompanied by the generation of base in the cytosol. The ability of this cell type to regulate cytosolic pH (pH_i) was examined as a function of stimulation of acid secretion by histamine or forskolin. The pH_i was estimated from the change of fluorescence of the trapped dye, 2′,7′-bis(carboxyethyl)-5(6)-carboxyfluorescein-bis-carboxyethylcarboxy fluorescein in a purified cell suspension of rabbit parietal cells. Stimulation of the cell suspension raised pH_i by an average of 0.13 ± 0.038 pH units. The H^+,K^+-ATPase inhibitor, SCH28080 (2-methyl-8-[phenylmethoxy]-imidazo-(1,2)-pyridine-3-acetonitrile) had only a small effect on the increase of pH_i due to cell stimulation. The increase of pH_i, therefore, was largely independent of H^+,K^+-ATPase activity. In Na^+-free medium, where Na^+/H^+ exchange would be absent, the

across the apical membrane of the parietal cell.

The regulation of cytosolic pH by cells usually requires the export of acid such as CO_2 or lactate that is generated metabolically or the extrusion of protons that enter the cell down their electrochemical gradient. An obligatory export of base is a less frequent situation.

The cell that is responsible for acid secretion in the stomach is the parietal cell. The transport enzyme that elaborates HCl is the HK-ATPase. In unstimulated cells, this enzyme is located in smooth-surfaced vesicular structures (tubulovesicles) in the cytoplasm. Upon stimulation, the HK-ATPase membranes move to the surface of the secretory canaliculus, forming microvilli. This canaliculus structure can be regarded as an inward extension of the apical membrane of the parietal cell. The secretion of protons is accompanied by an accumu-



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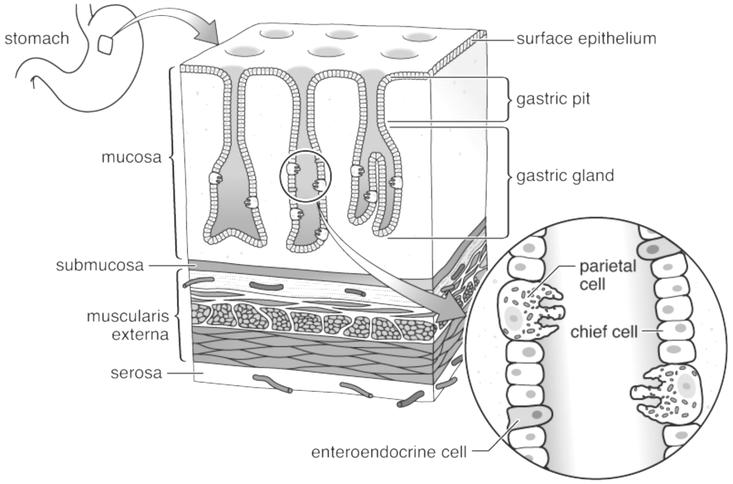

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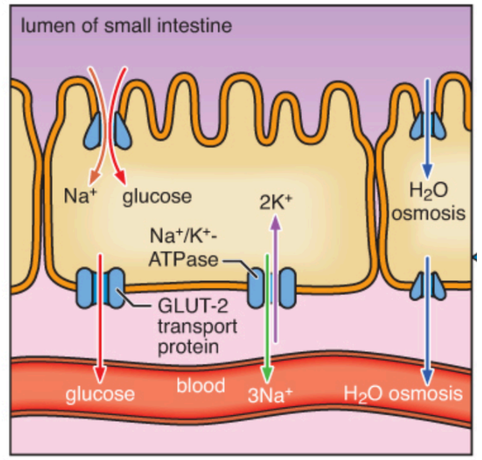
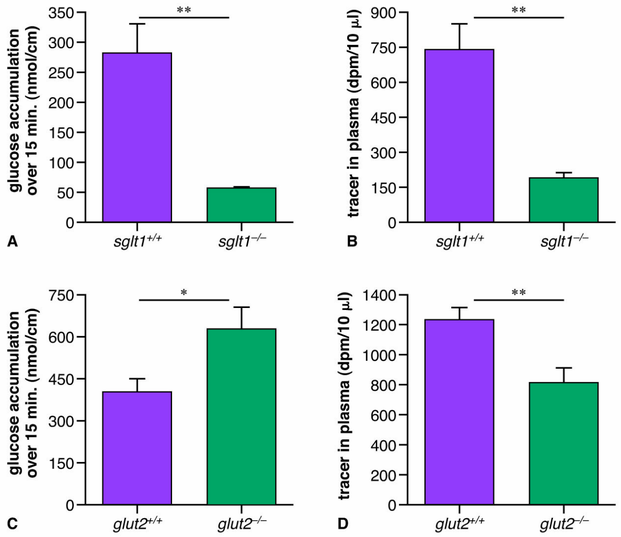
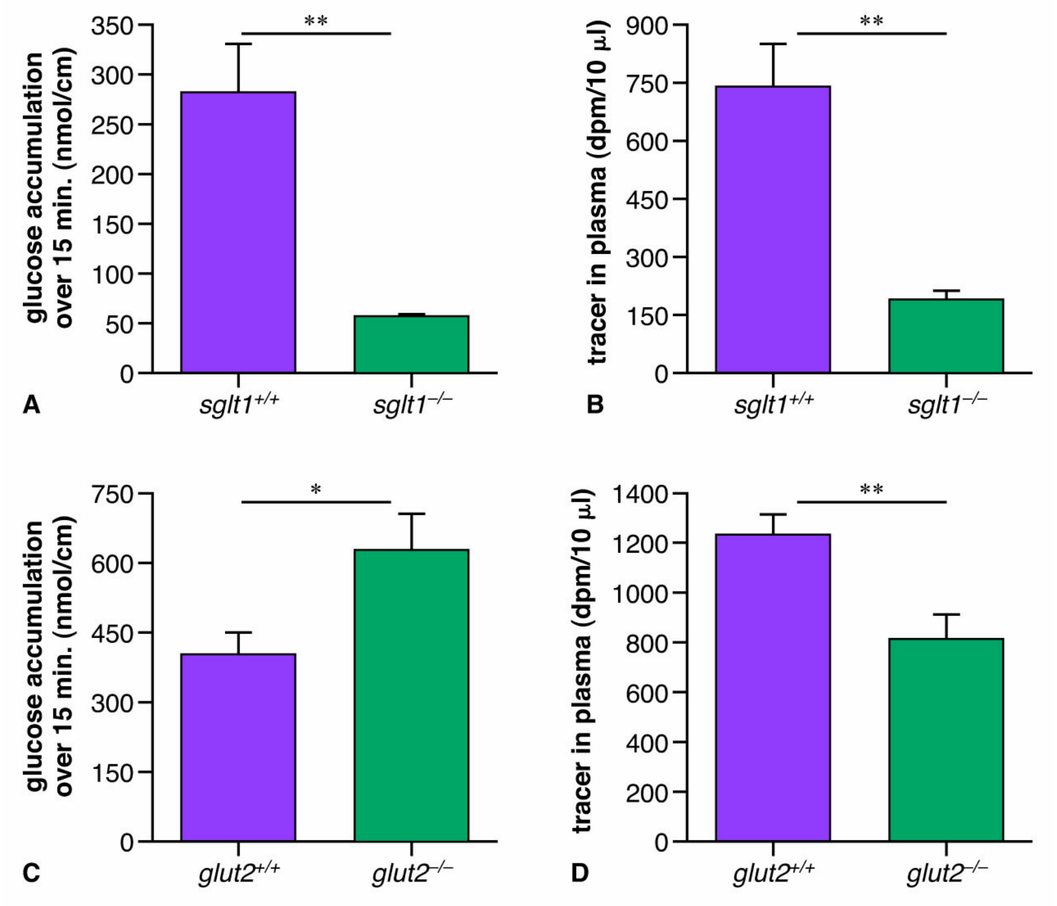
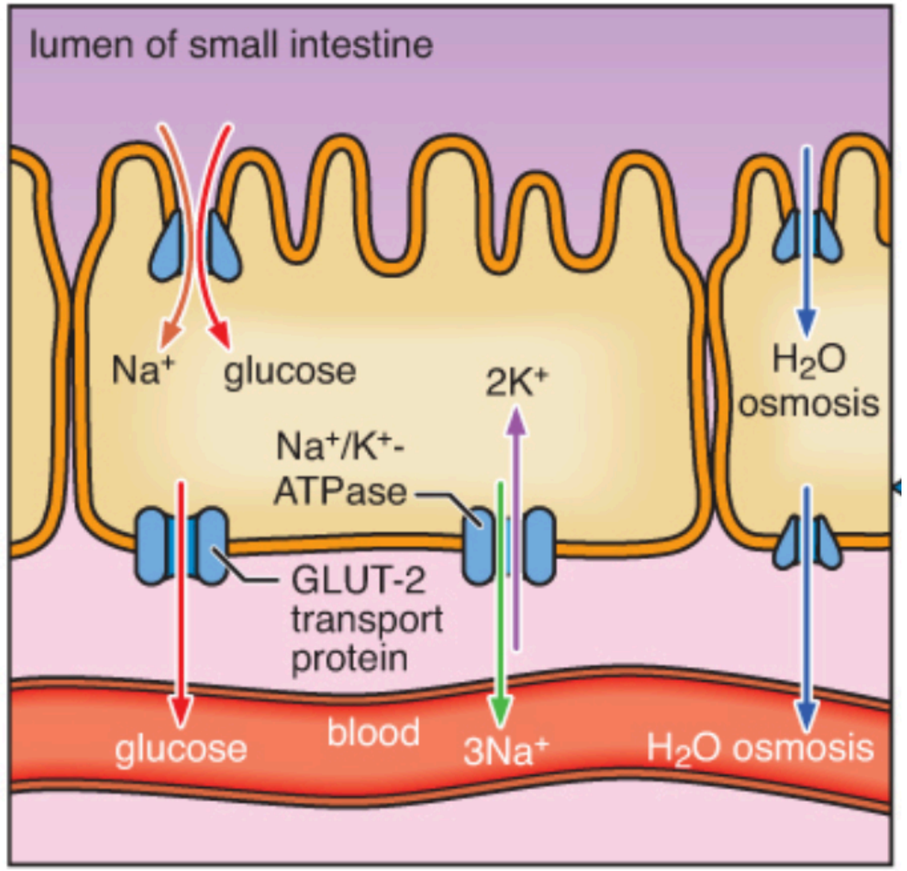


Roder
(Absorption)



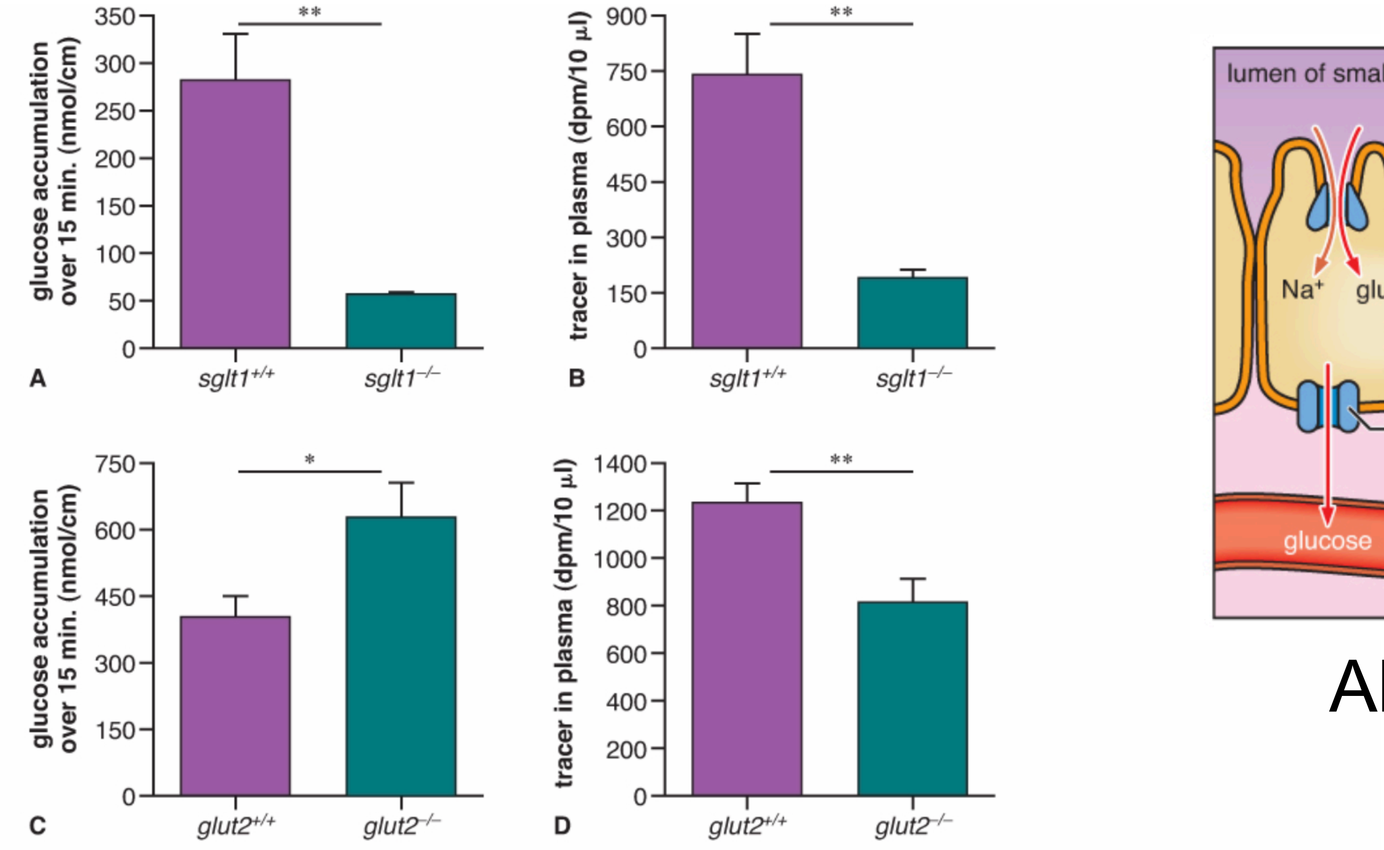
Roder
(Absorption)

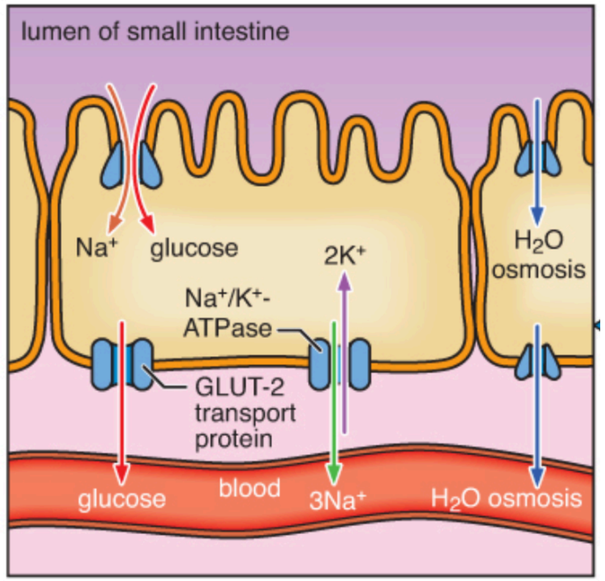




Roder
(Absorption)

Trifecta





Absorption

Figure 23.7 Effects of SGLT1 and GLUT2 on glucose accumulation and blood plasma glucose, as measured by amount of radioactive tracer. Purple bars are wild-type, and teal bars are mutant mice. **A,** Mean accumulation of glucose in intestinal tissue samples for *sglt1* wild-type and mutant mice. **B,** Mean amount of glucose in blood plasma for *sglt1* wild-type and mutant mice. **C,** Mean accumulation of glucose in intestinal tissue samples for *glut2* wild-type and mutant mice. **D,** Mean amount of glucose in blood plasma for *glut2* wild-type and mutant mice. Error bars represent ± 1 standard error (SE). Statistical analyses were performed using a t-test. *, *p*-value < 0.05; **, *p*-

Effects of SGLT1 and GLUT2 on glucose accumulation and blood plasma glucose

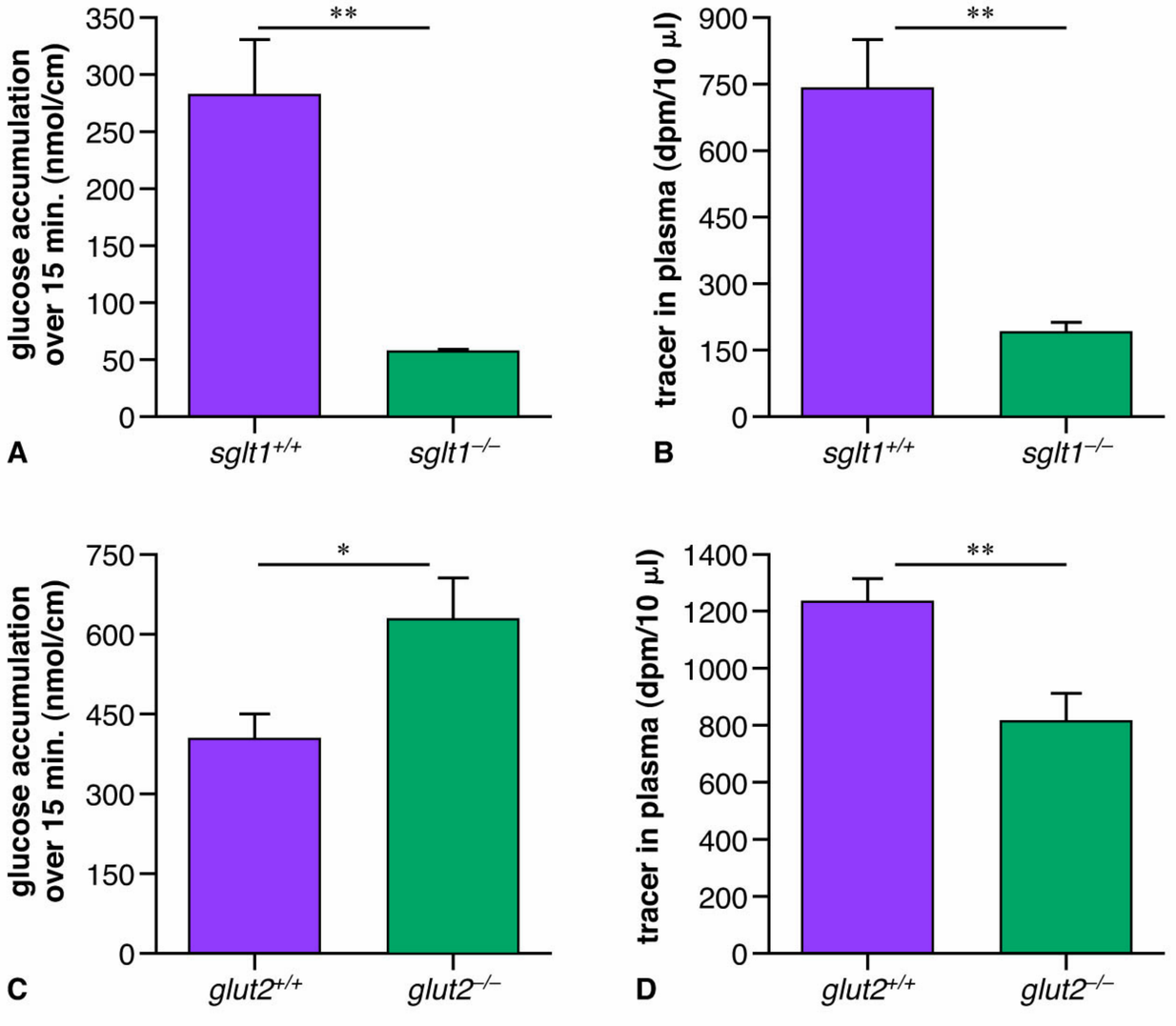


Figure 23.7

Effects of SGLT1 and GLUT2 on glucose accumulation and blood plasma glucose

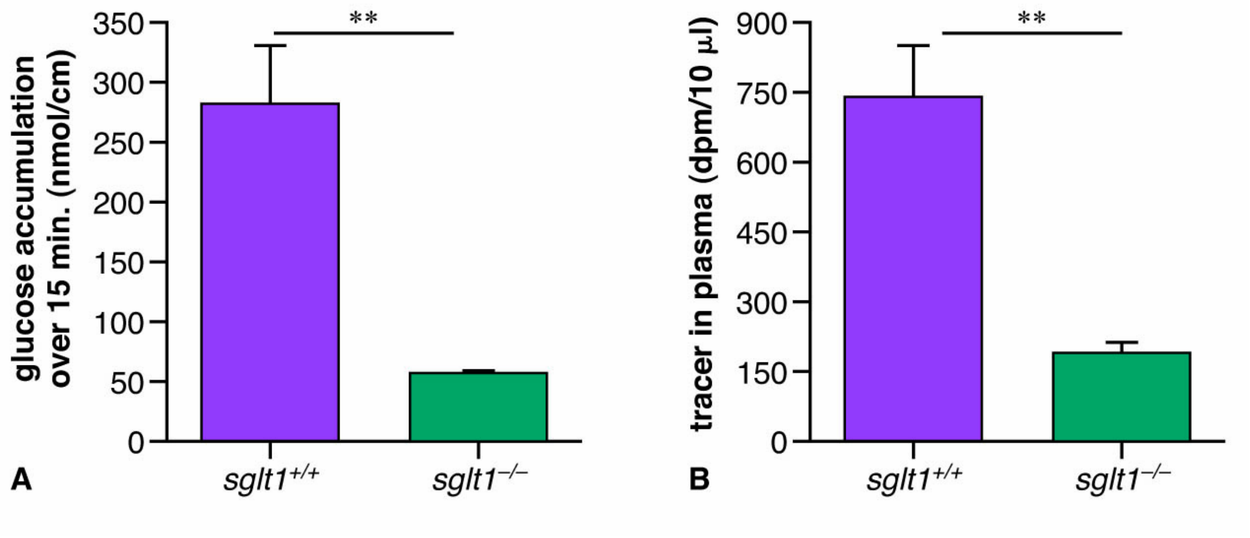
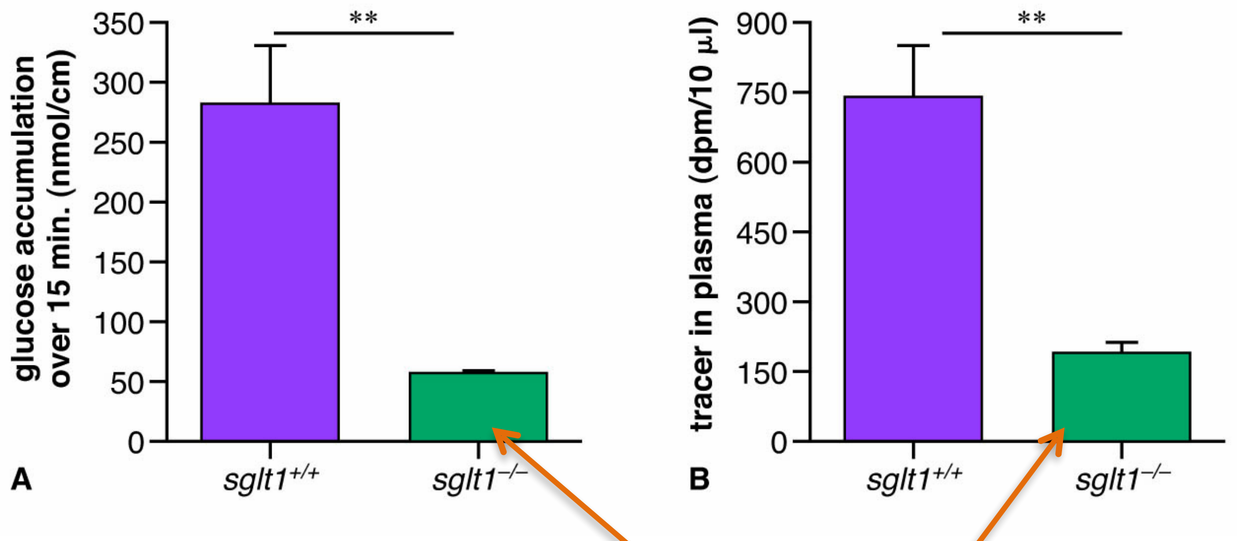


Figure 23.7

Effects of SGLT1 and GLUT2 on glucose accumulation and blood plasma glucose



Mutant *sglt1*$^{-/-}$ mice take up very little glucose into epithelial cells and show very little glucose in the blood relative to wild-type mice.

Figure 23.7

Effects of SGLT1 and GLUT2 on glucose accumulation and blood plasma glucose

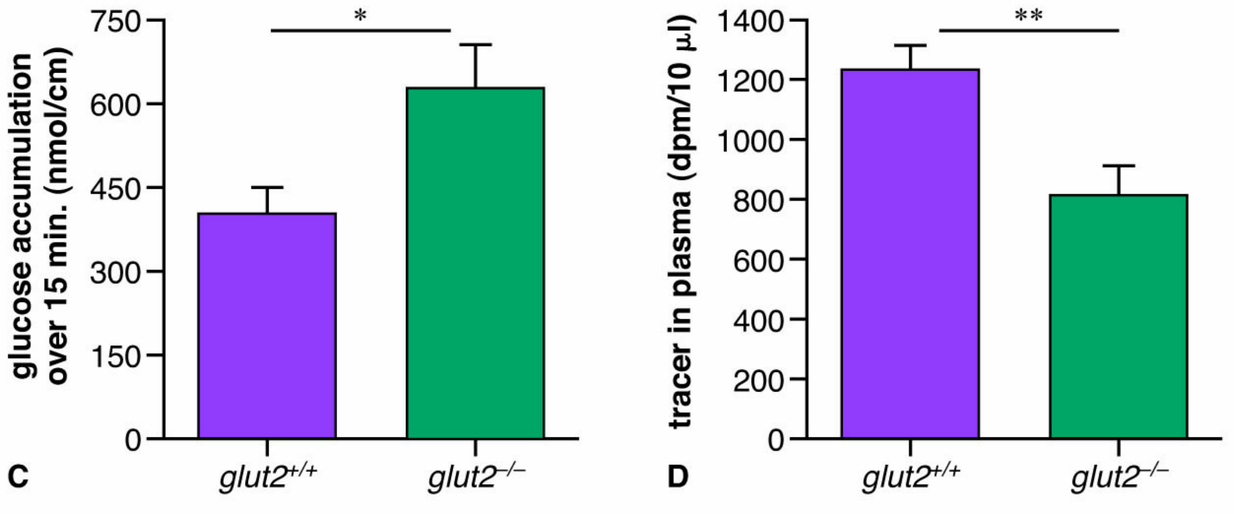


Figure 23.7

Effects of SGLT1 and GLUT2 on glucose accumulation and blood plasma glucose

Glucose accumulation is higher in mutant *glut2*$^{-/-}$ mice than wild-type mice. Glucose concentrations are lower in mutant than in wild-type mice blood.
Glucose can enter the epithelial cell but cannot get out.

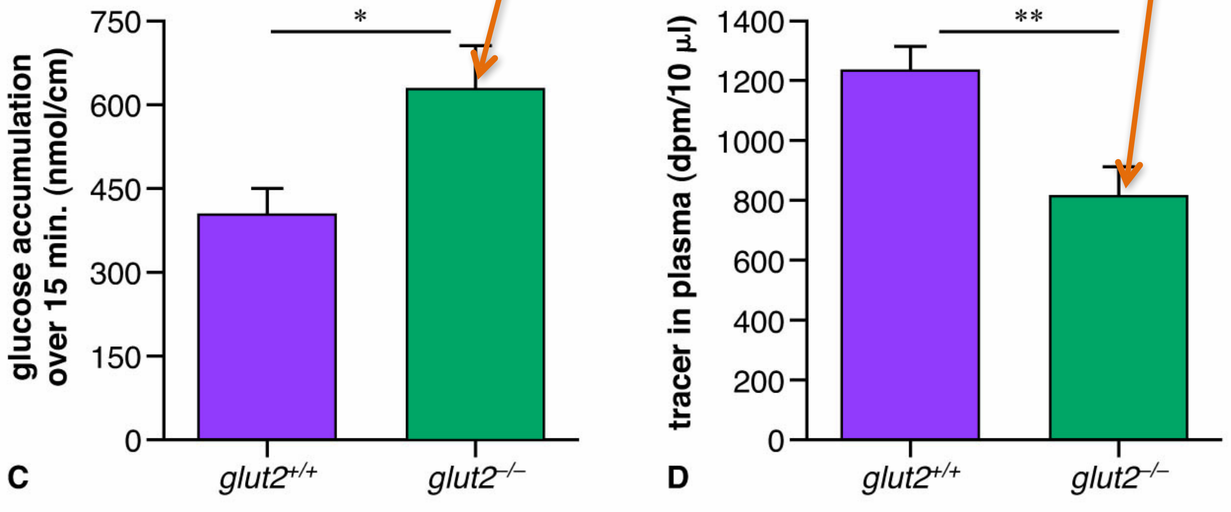


Figure 23.7

Effects of SGLT1 and GLUT2 on glucose accumulation and blood plasma glucose

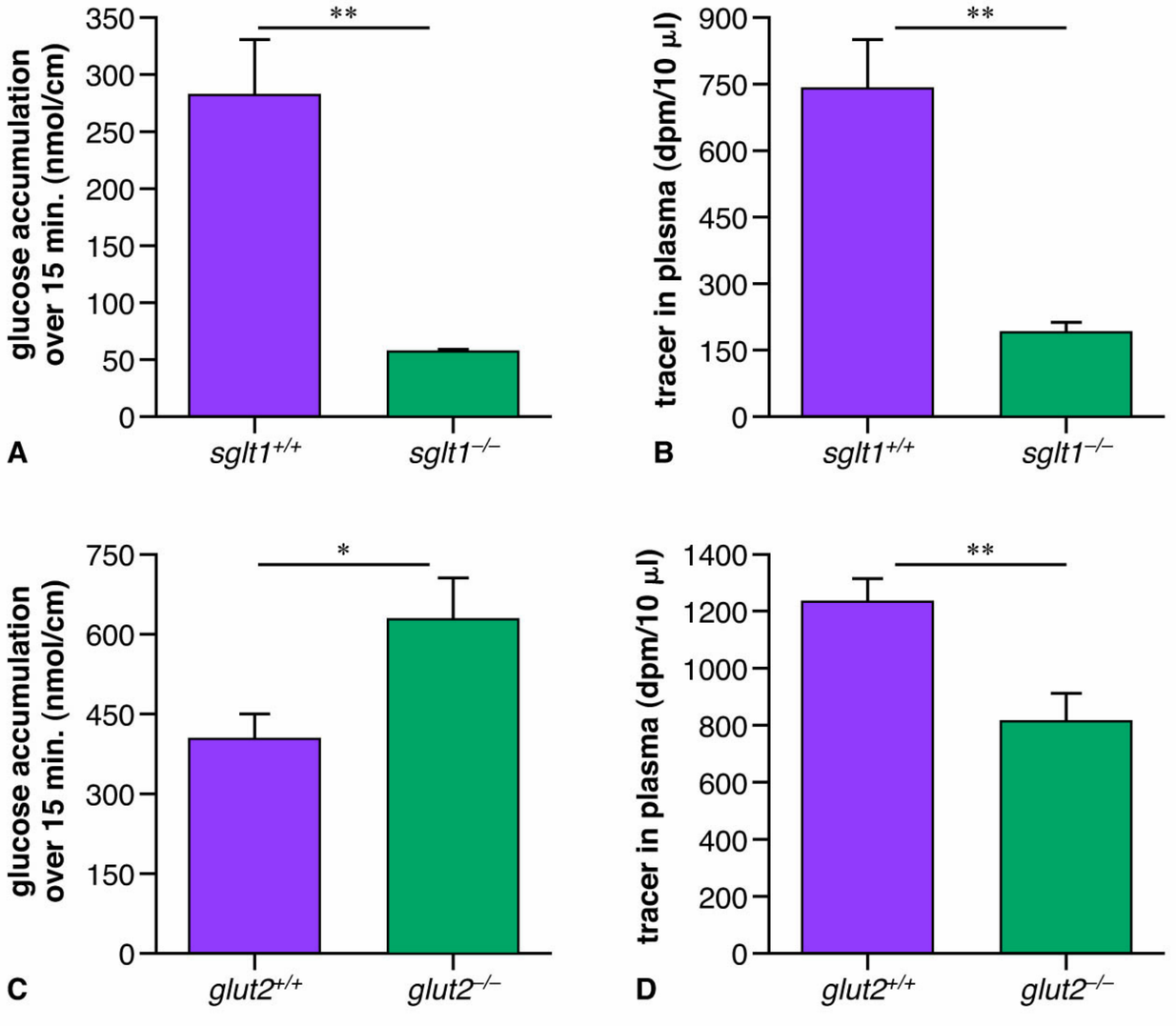


Figure 23.7

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PLOS ONE

The Role of SGLT1 and GLUT2 in Intestinal Glucose Transport and Sensing

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Abstract

Intestinal glucose absorption is mediated by SGLT1 whereas GLUT2 is considered to provide basolateral exit. Recently, it was proposed that GLUT2 can be recruited into the apical membrane after a high luminal glucose bolus allowing bulk absorption of glucose by facilitated diffusion. Moreover, SGLT1 and GLUT2 are suggested to play an important role in intestinal glucose sensing and incretin secretion. In mice that lack either SGLT1 or GLUT2 we re-assessed the role of these transporters in intestinal glucose uptake after radiotracer glucose gavage and performed Western blot analysis for transporter abundance in apical membrane fractions in a comparative approach. Moreover, we examined the contribution of these transporters to glucose-induced changes in plasma GIP, GLP-1 and insulin levels. In mice lacking SGLT1, tissue retention of tracer glucose was drastically reduced throughout the entire small intestine whereas GLUT2-deficient animals exhibited higher tracer contents in tissue samples than wild type animals. Deletion of SGLT1 resulted also in reduced blood glucose elevations and abolished GIP and GLP-1 secretion in response to glucose. In mice lacking GLUT2, glucose-induced insulin but not incretin secretion was impaired. Western blot analysis revealed unchanged protein levels of SGLT1 after glucose gavage. GLUT2 detected in apical membrane fractions mainly resulted from contamination with basolateral membranes but did not change in density after glucose administration. SGLT1 is unequivocally the prime intestinal glucose transporter even at high luminal glucose concentrations. Moreover, SGLT1 mediates glucose-induced incretin secretion. Our studies do not provide evidence for GLUT2 playing any role in either apical glucose influx or incretin secretion.

Citation: Röder PV, Geillinger KE, Zietek TS, Thorens B, Koepsell H, et al. (2014) The Role of SGLT1 and GLUT2 in Intestinal Glucose Transport and Sensing. PLoS ONE 9(2): e89977. doi:10.1371/journal.pone.0089977

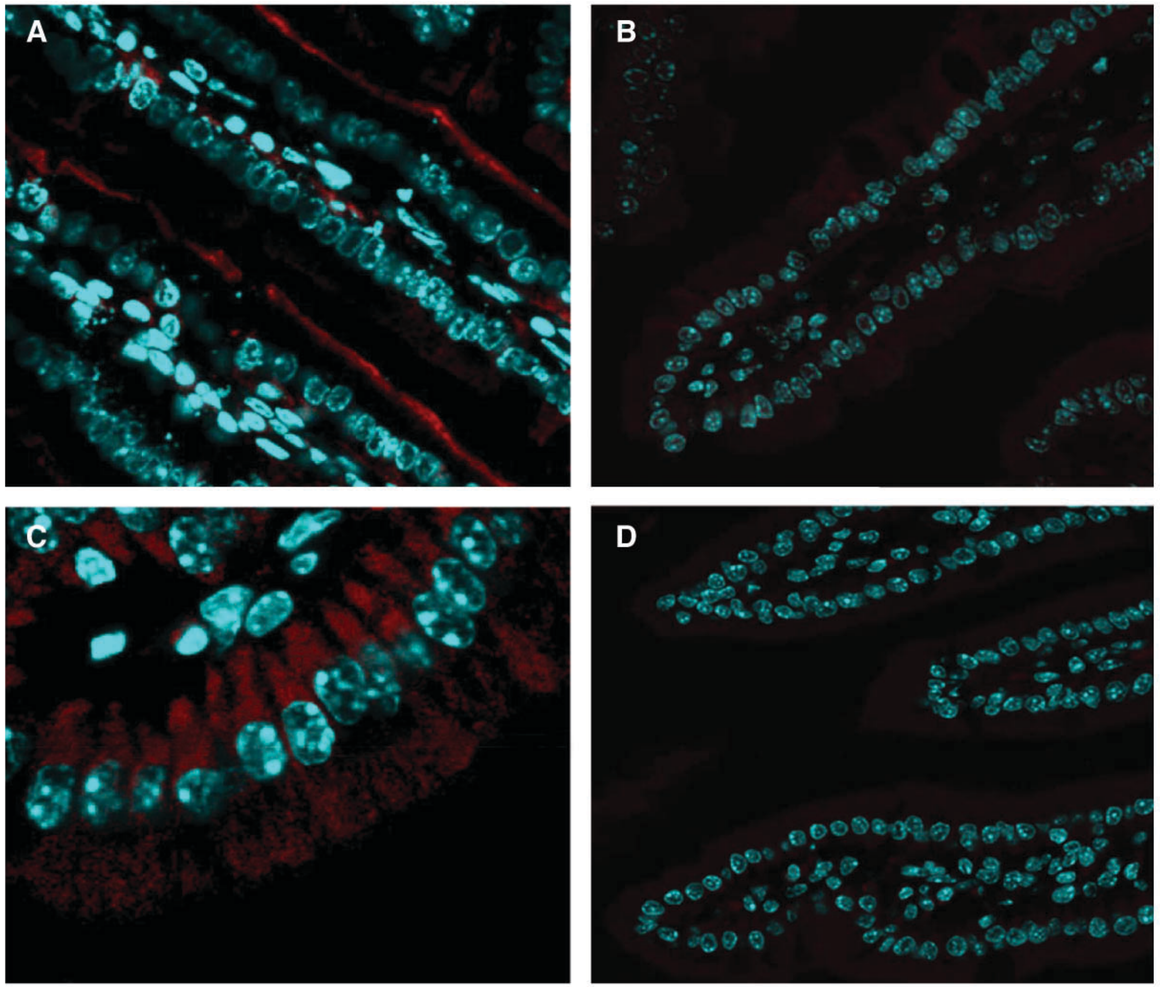
Editor: Marià Alemany, University of Barcelona, Spain

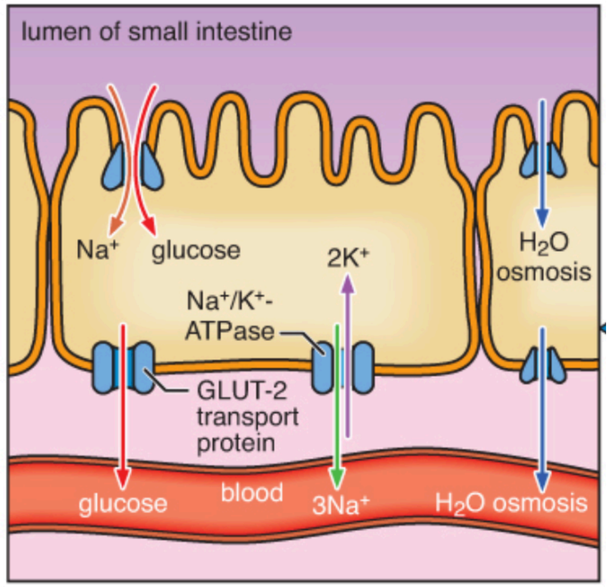
Received November 29, 2013; **Accepted** January 24, 2014; **Published** February 26, 2014

Funding: The work was supported by the Deutsche Forschungsgemeinschaft (GZ DA 190/10-1, Nutra2Sense). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing Interests: The authors have declared that no competing interests exist.

Immunostained jejunum epithelial cells showing localization of glucose transport proteins (red) and cell nuclei (blue)

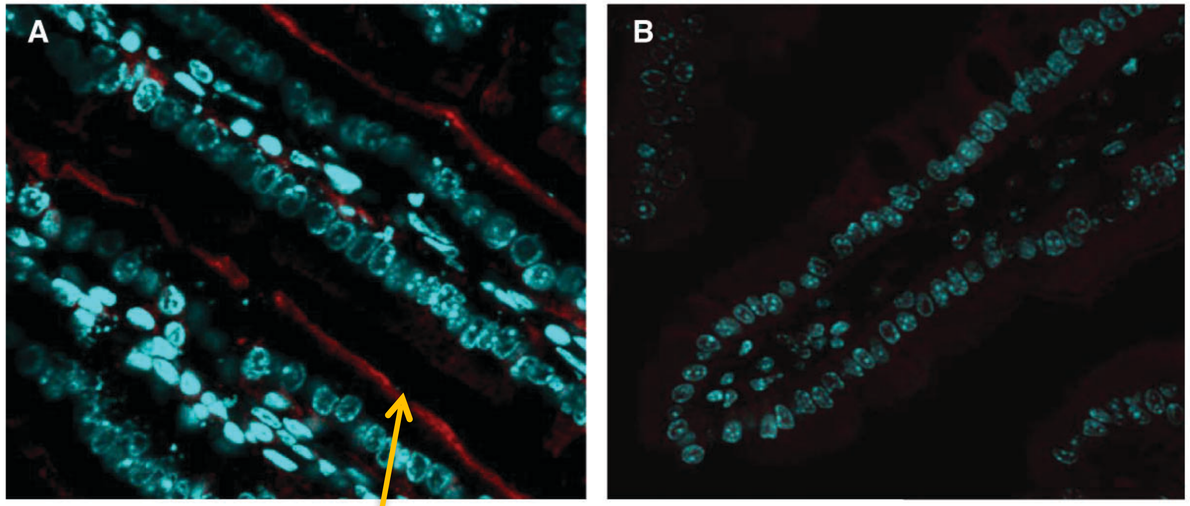




Absorption

Figure 23.8

Immunostained jejunum epithelial cells showing localization of glucose transport proteins (red) and cell nuclei (blue)



Localization of SGLT1 from *sglt1*$^{+/+}$ wild-type but not *sglt1*$^{-/-}$ mice

Figure 23.8

Immunostained jejunum epithelial cells showing localization of glucose transport proteins (red) and cell nuclei (blue)

Localization of GLUT2 from wild-type *glut2*$^{+/+}$ but not *glut2*$^{-/-}$ mice

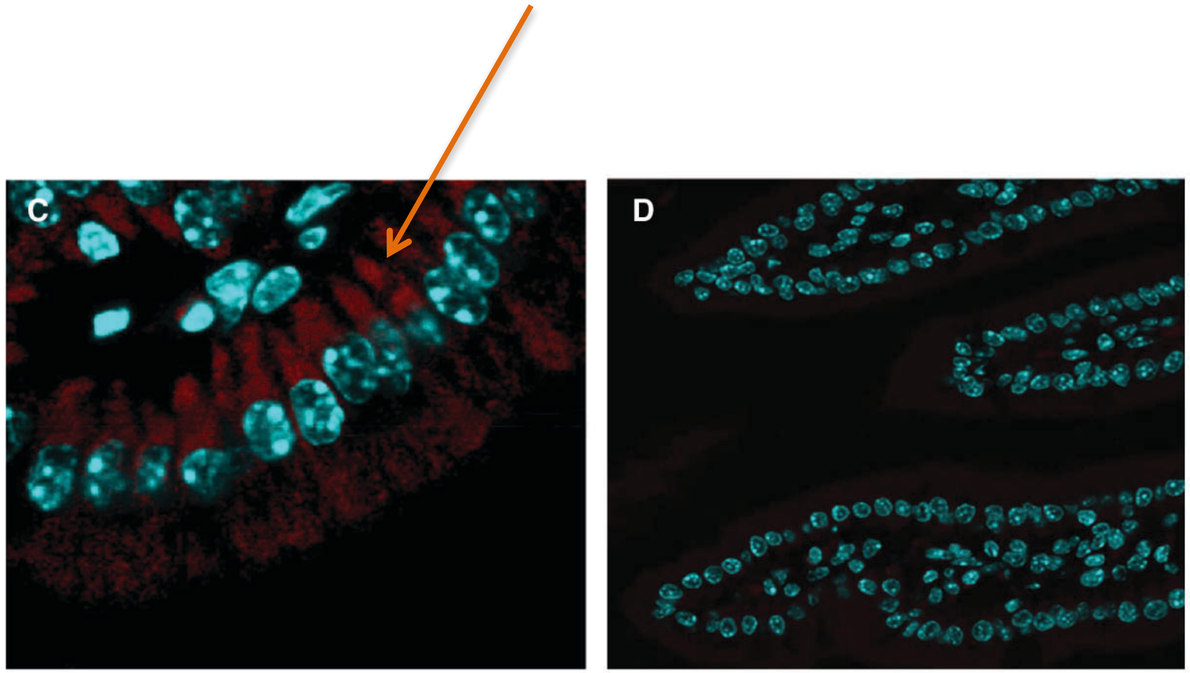
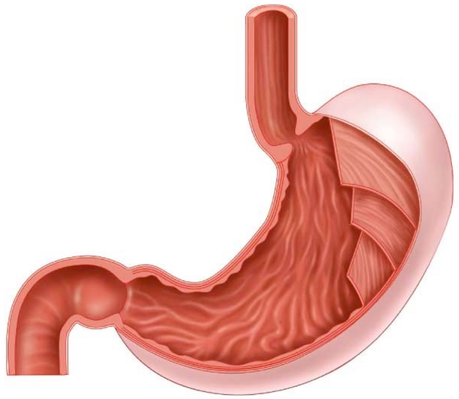
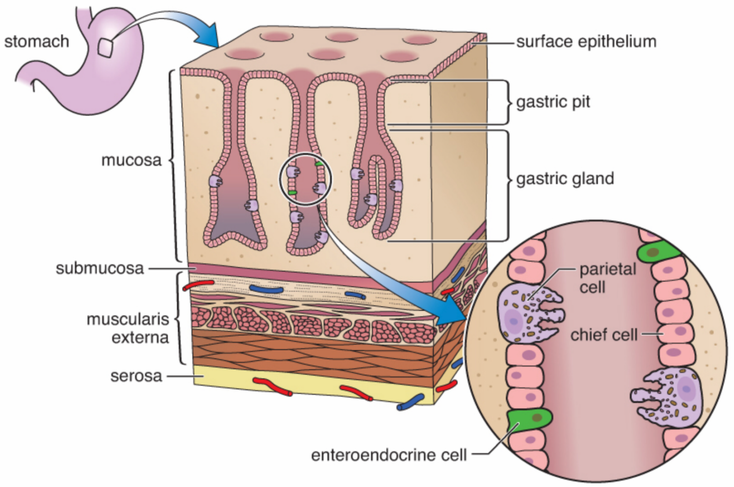
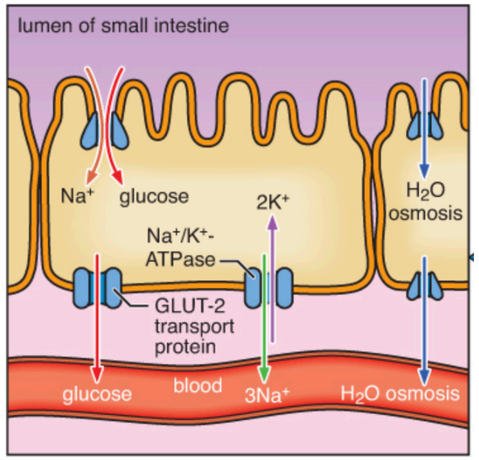


Figure 23.8







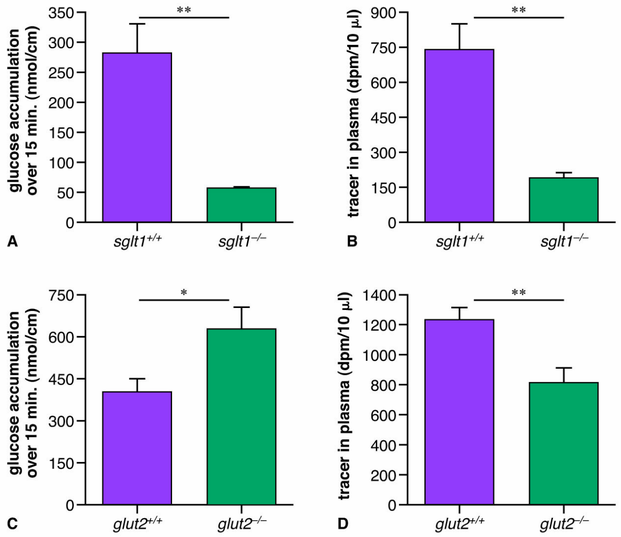
Prout
(Stomach)

fraction of solution	rabbit 1	rabbit 2	rabbit 3
chloride salts (first fraction) (g)	0.12	0.95	1.71
exact amount of base to neutralize acid (second fraction) (g)	1.56	0.76	0.40
chloride salts after neutralization (third fraction) (g)	1.59	2.22	2.72
total amount of chloride (g)	3.27	3.93	4.83
other acids (fourth fraction)	0	0	0

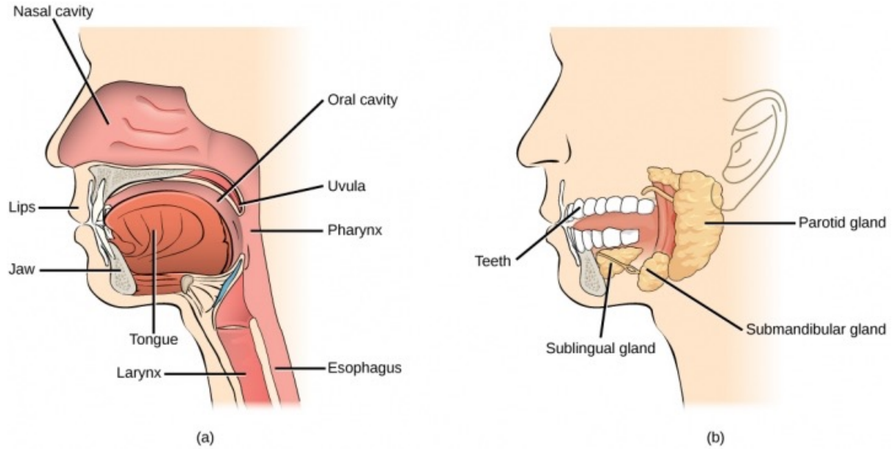
Muallem
(Parietal cell)

treatment	ΔpH
no Na^+	−0.58
Na^+ added after exposed to no Na^+	0.56
Na^+ and Na^+/H^+ exchange inhibitor added after exposed to no Na^+	0
histamine	0.130 ± 0.038

Roder
(Absorption)



VF



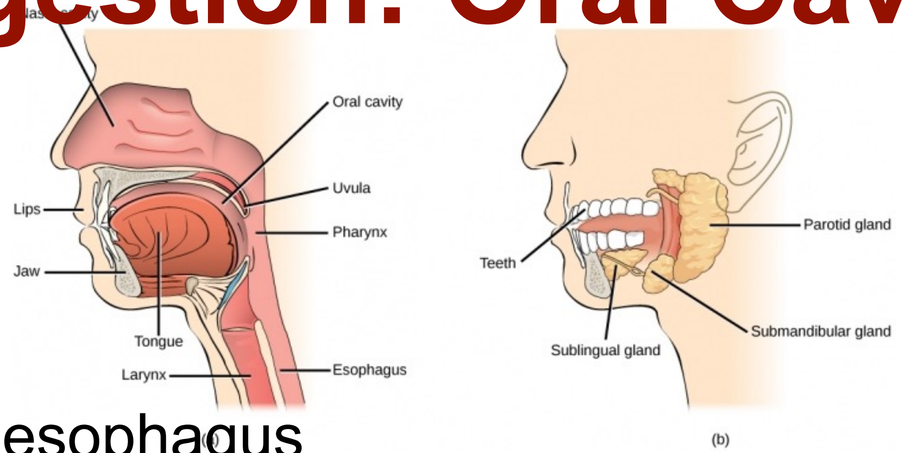
Digestion: Oral Cavity

When you are eating a Krispy Kreme donut, describe the process of digestion in the Oral Cavity (mouth).

What **hormones** -> stimulate which **cells** -> to secrete what **enzymes** (or perform what **mechanical** actions) -> to digest what **food** molecules?

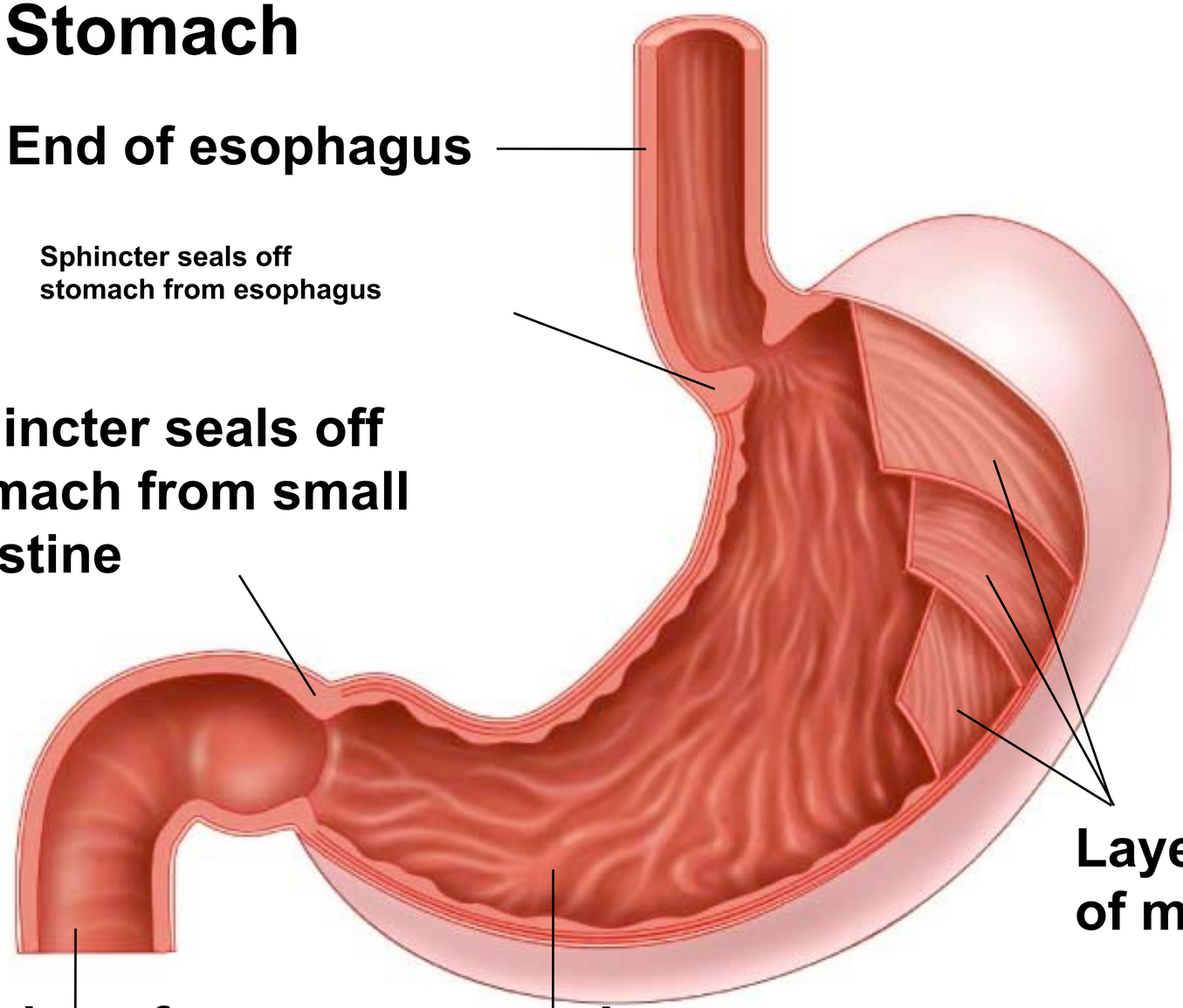
VF

Digestion: Oral Cavity



- The mouth and esophagus
  - Salivary amylase begins the process of carbohydrate digestion (starch i.e. amylose).
  - Lingual lipase initiates fat (i.e. lipid) digestion.
  - Hormones: (kinda) neurotransmitter via nerves from brain
  - other? Buffers, mucous, mechanical, antibacterial compounds
  - A combination of smooth and skeletal muscle moves food down the esophagus by peristalsis.

Stomach

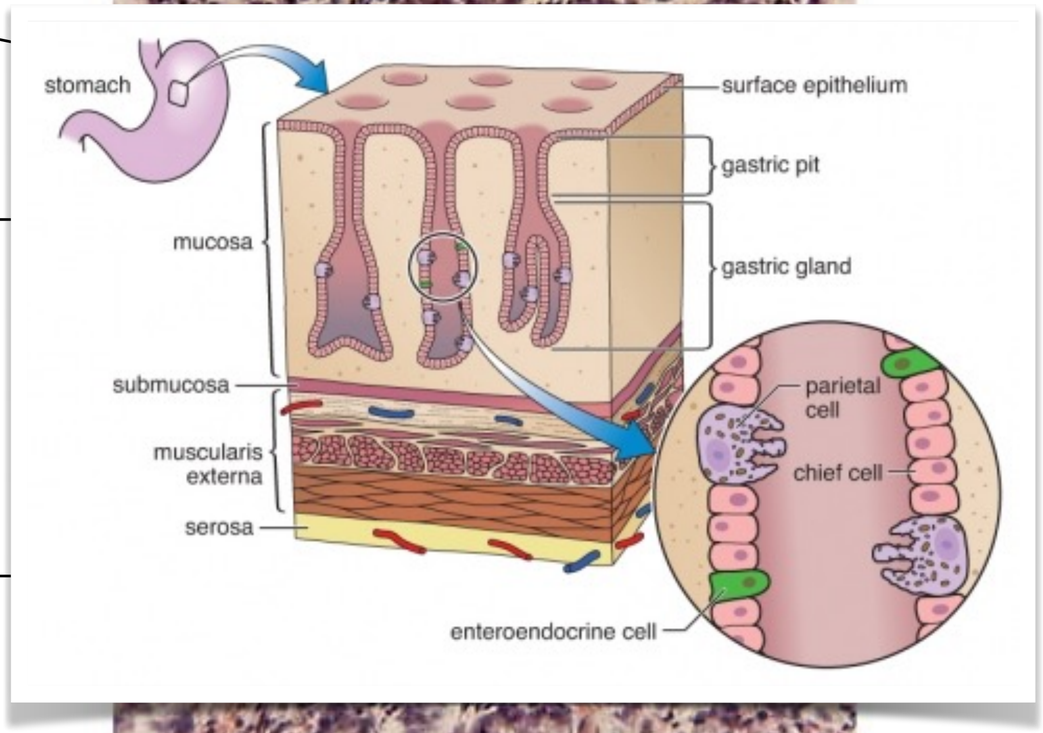

End of esophagus
Sphincter seals off stomach from esophagus
Sphincter seals off stomach from small intestine
Layers of muscle
Beginning of small intestine
Lumen (interior)

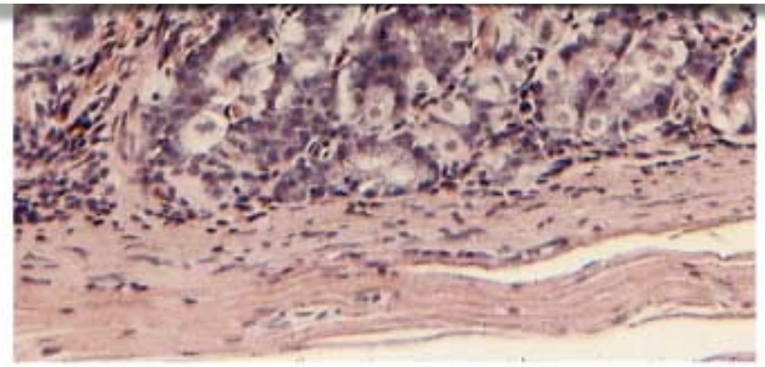
Stomach lining

Canal empties to lumen

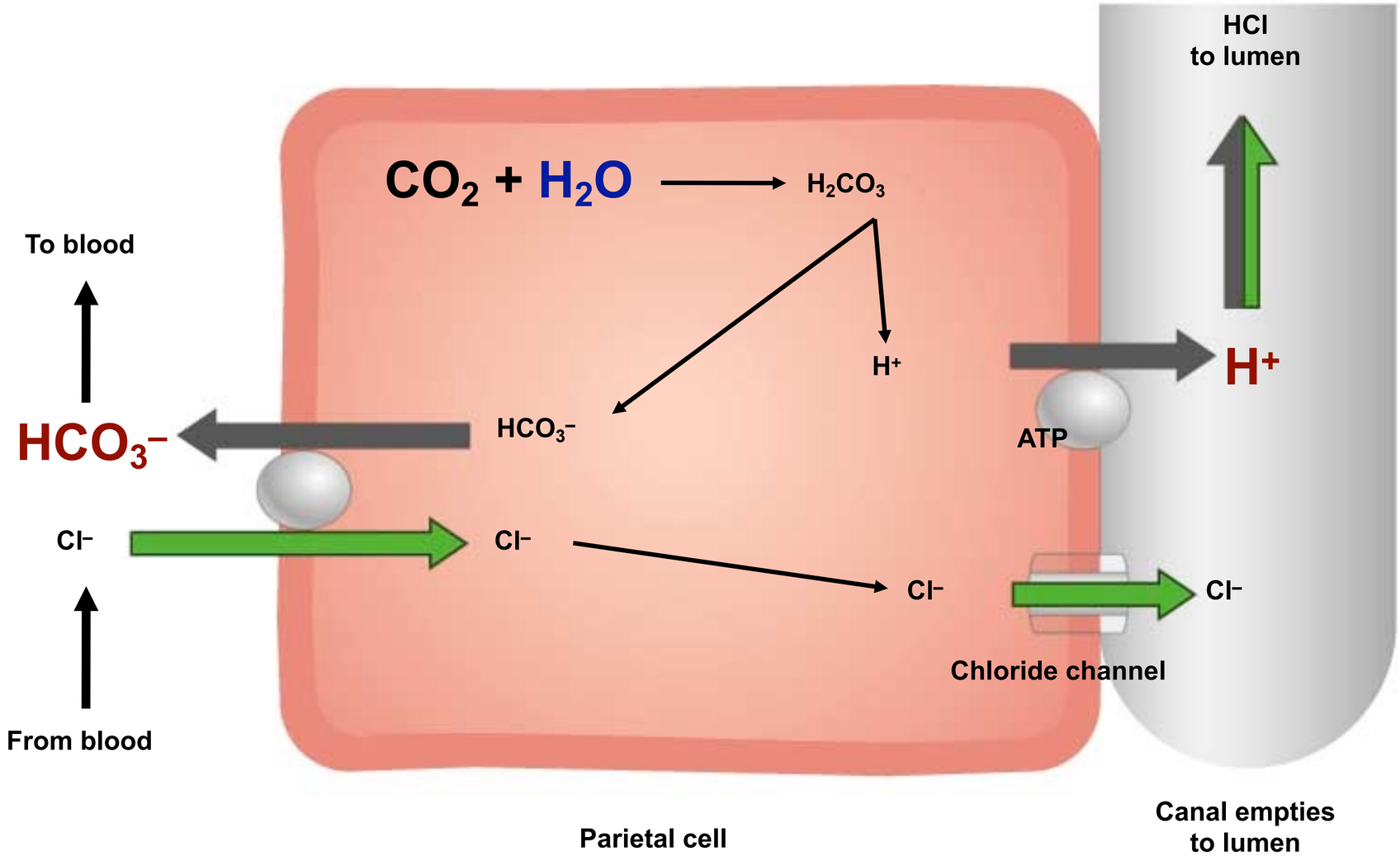
Parietal cells (secrete HCl)

Chief cells (secrete pepsinogen)





Secretion of HCl by parietal cells

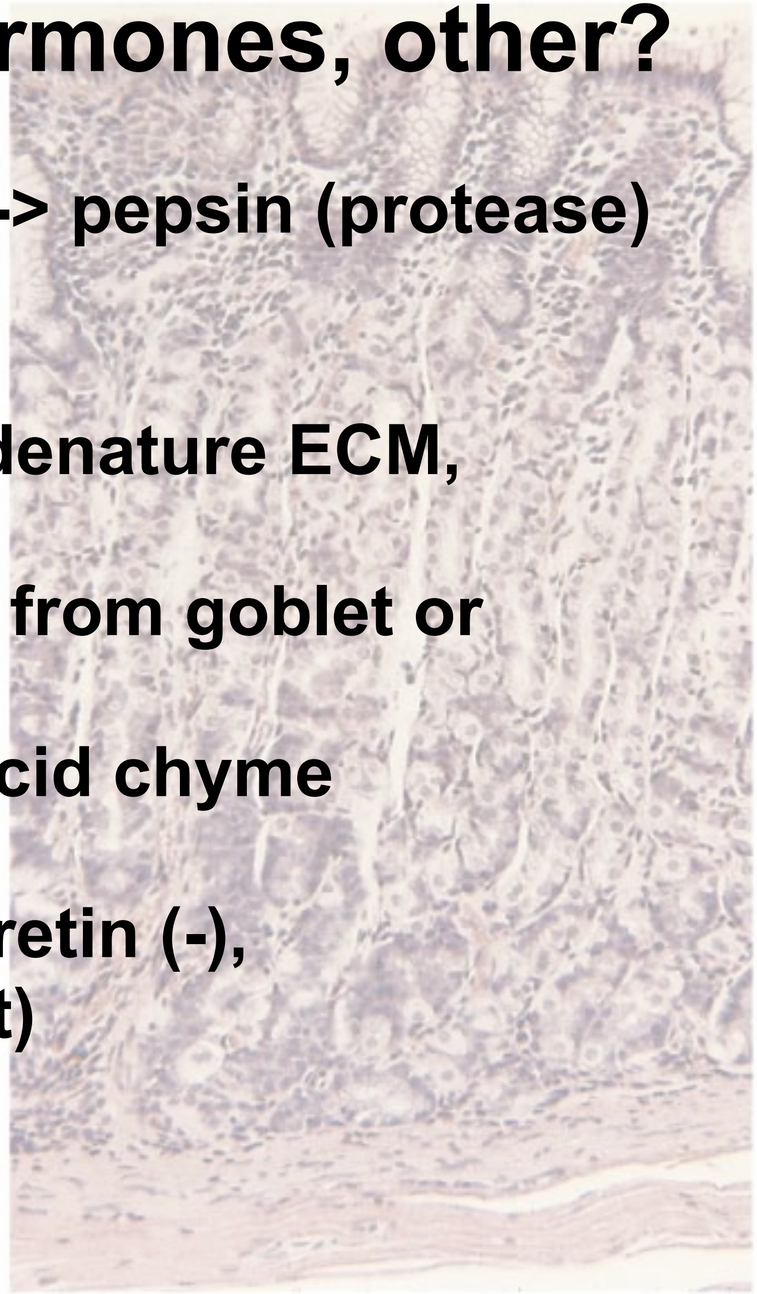


Stomach -Enzymes, Hormones, other?

- **Enzymes**: pepsinogen (inactive)-> pepsin (protease) from chief cells

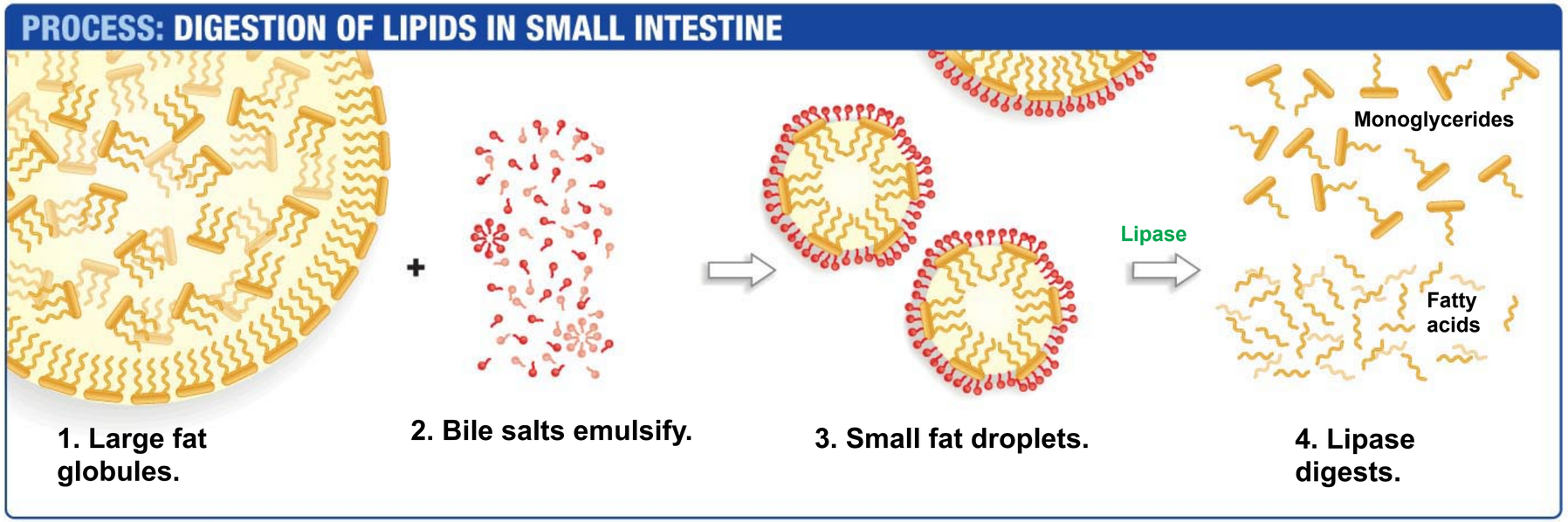
- ***Other***: HCl from parietal cells-> denature ECM, bacteria, activator of pepsin
- ***Other***: Mucins/mucus protective from goblet or mucus cells
- ***Other***: mechanical churning -> acid chyme

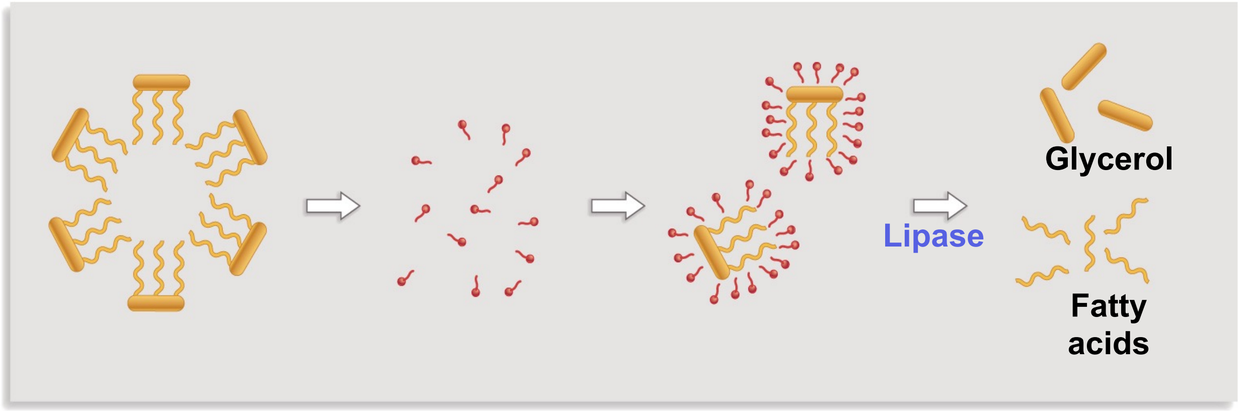
- **Hormones**: Gastrin (+), CCK/secretin (-), enterogastrone (-- pyloric sphinct)



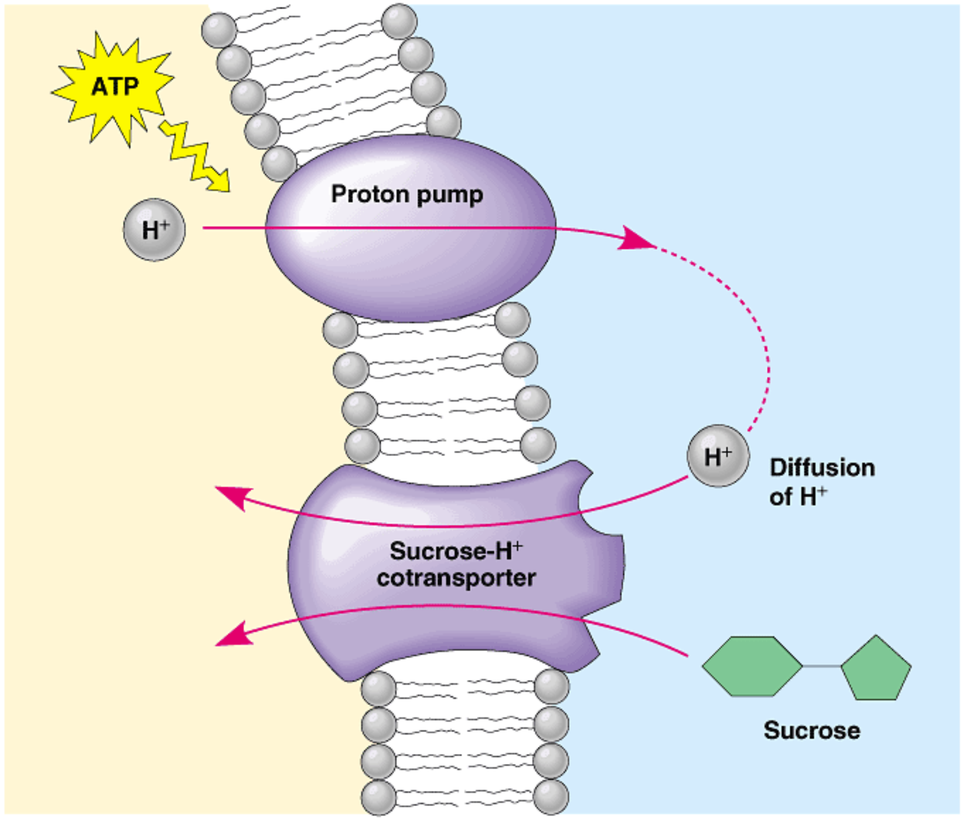
Digestion

- The small intestine
  - The hormone *cholecystokinin (CCK)* released from the duodenum into the bloodstream soon stimulates the secretion of enzymes from the pancreas and bile salts from the gall bladder, into the small intestine.
  - Bile salts emulsify fats (lipids) so they can be digested by the enzyme lipase secreted by the pancreas.
  - The epithelium of the small intestine is highly folded, and have fingerlike projections called microvilli which provide a large surface area for absorption of food into the body and bloodstream.

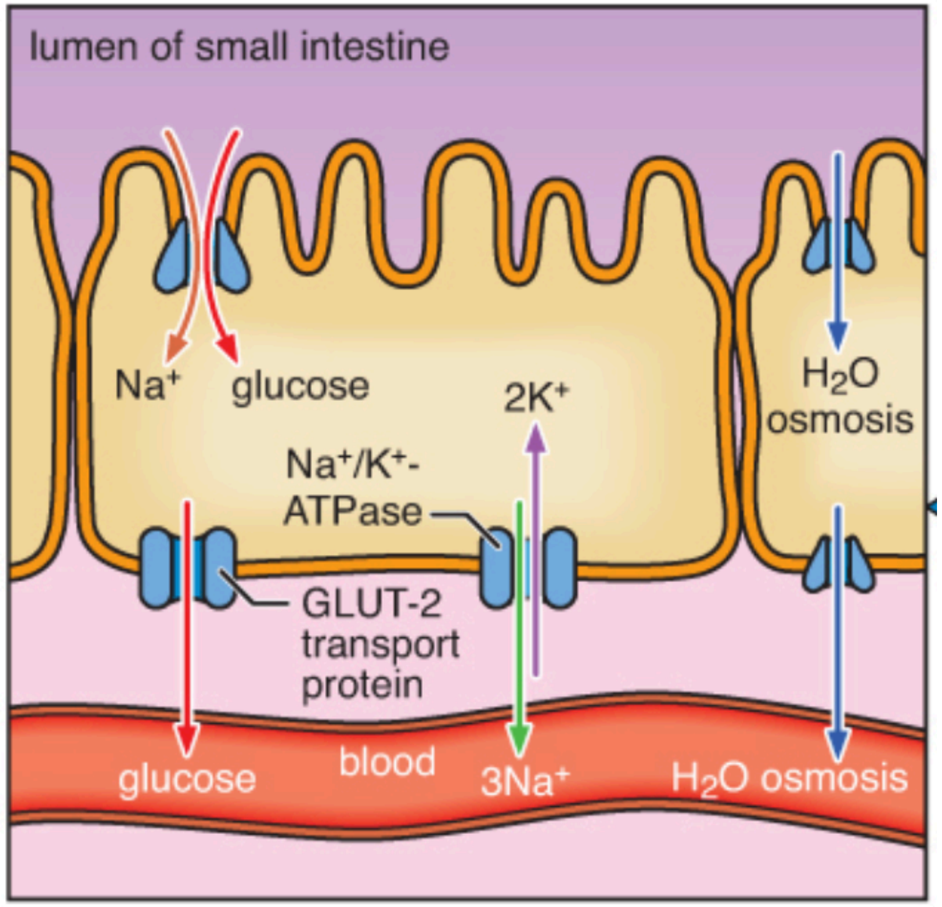




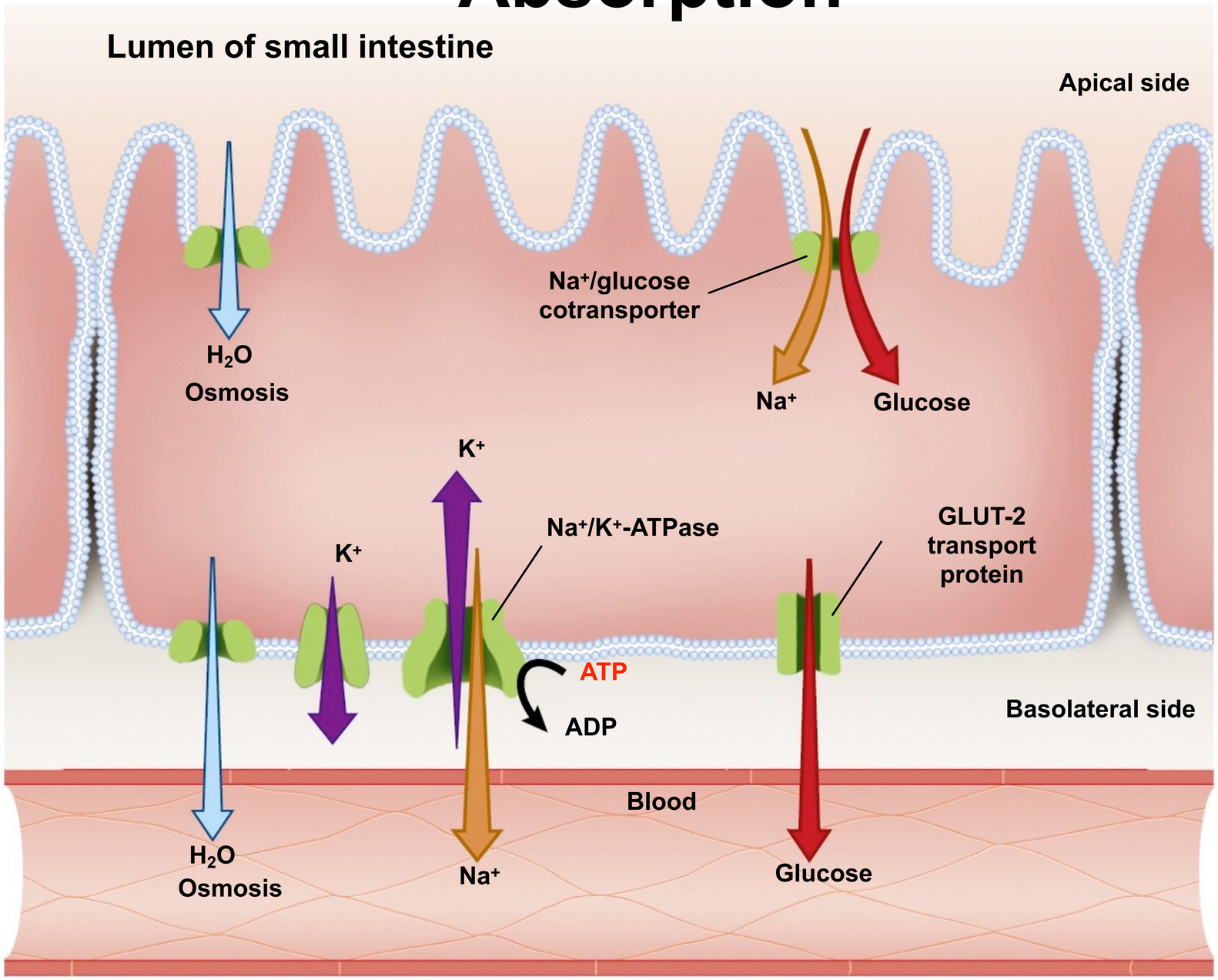
Absorption



Absorption


lumen of small intestine
Na+
glucose
2K+
H2O
osmosis
Na+/K+-
ATPase
GLUT-2
transport
protein
glucose
blood
3Na+
H2O osmosis

Absorption


Lumen of small intestine
Apical side
H_2O
Osmosis
Na^+/glucose
cotransporter
Na^+
Glucose
K^+
K^+
Na^+/K^+-ATPase
GLUT-2
transport
protein
ATP
ADP
Basolateral side
Blood
H_2O
Osmosis
Na^+
Glucose

Absorption +

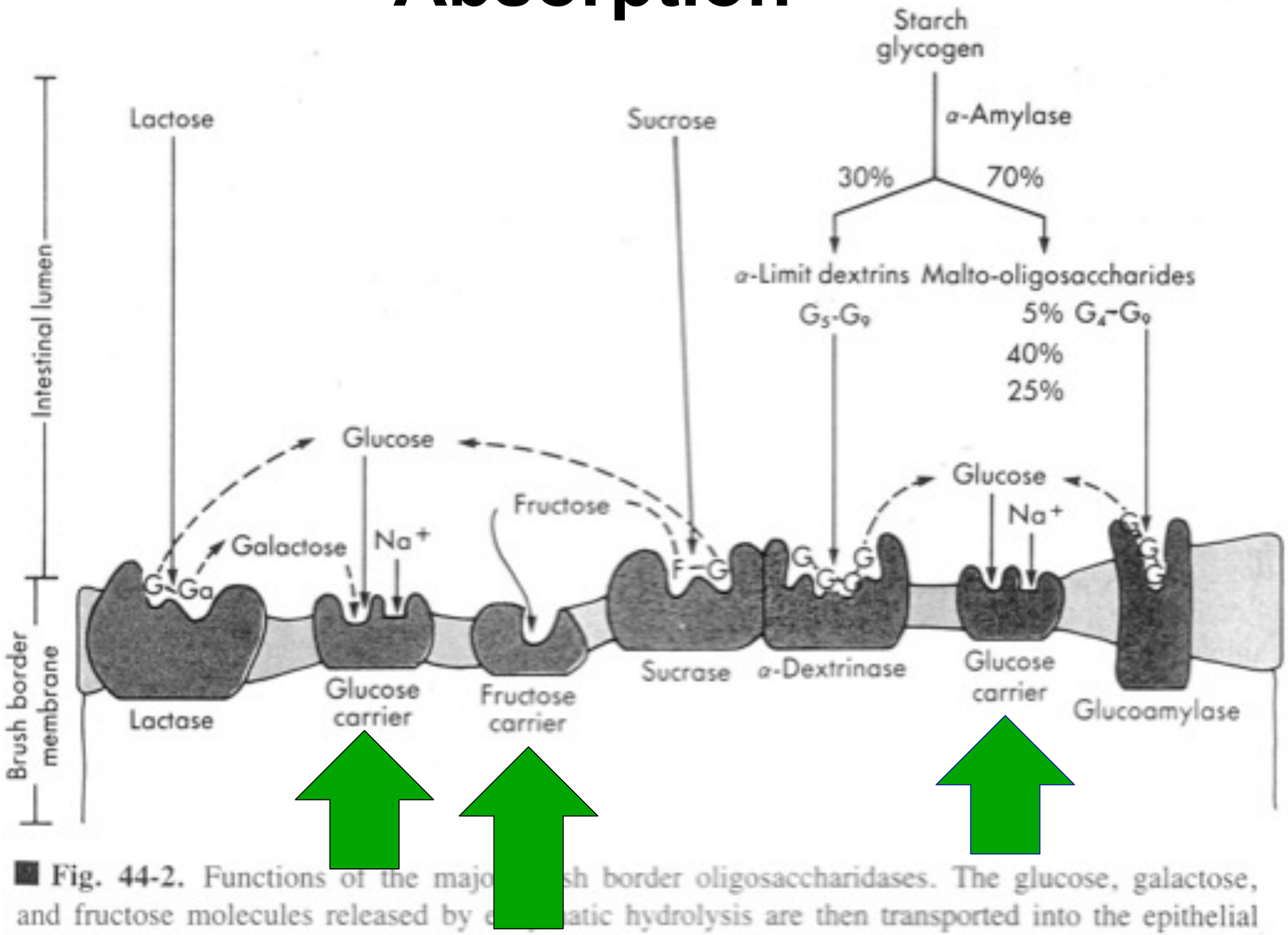


Fig. 44-2. Functions of the majo[illegible]sh border oligosaccharidases. The glucose, galactose, and fructose molecules released by e[illegible]atic hydrolysis are then transported into the epithelial cell by specific transport carrier proteins. *G*, Glucose; *Ga*, galactose; *F*, fructose. (From Gray, G.M.: N. Engl. J. Med. **292:**1225, 1975. Reprinted by permission of *The New England Journal of Medicine*.)