Anatomy & Physiology 2027

Illustrated review notes

Respiratory | Digestive | Immune

Core concepts, named diseases, labeled diagrams, mechanism explanations and LaTeX equations for Division B.

Level key

[Core] = required baseline at Regional, State and National. [S] = State; [N] = National. The supplied AP_RULE_2027 groups advanced requirements as State & National Level Only, so advanced entries carry both [S] [N]. There is no separate National-only list in this supplied rule. [E] = optional illustration/enrichment, not an additional named rule requirement.

How this revision differs

Figures are selected from all five sources and placed alongside the corresponding review notes. Title pages, contents, section dividers and decorative images are omitted. Historical food pyramids are excluded. Original source crops preserve existing labels; textbook diagrams provide clearer alternatives for dense or blurred figures. Some original bitmaps remain blurry at large magnification; the notes never claim enlargement restores missing detail.

Study each picture

Name the structures or axes. Trace the arrows. Explain why the mechanism works. Cover labels and redraw. Predict the effect of a disease. Read the caption and correction before memorizing an older diagram.

Sources

S23 = AP_Respiratory, Digestive, Immune Systems_Slides2023.pdf

S27 = AP_Respiratory, Digestive, Immune Systems_Slides2027.pdf

WD = Anatomy_Digestive System - Wiki - Scioly.org.pdf

WI = Anatomy_Immune System - Wiki - Scioly.org.pdf

WR = Anatomy_Respiratory System - Wiki - Scioly.org.pdf

Figure Selection Audit.csv records a decision for all 156 source pages. Original source PDFs remain unchanged. Textbook diagrams are stored locally for offline use.

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RESPIRATORY

R-AIR | Air pathway and respiratory functions

[Core] | Rule 3a.i

Trace a breath: nose or mouth -> pharynx -> larynx -> trachea -> main bronchi -> lobar bronchi -> segmental bronchi -> bronchioles -> terminal bronchioles -> respiratory bronchioles -> alveolar ducts -> alveoli. The conducting zone ends at terminal bronchioles; it transports, warms, moistens and cleans air. The respiratory zone begins where alveoli appear along respiratory bronchioles. Most exchange occurs in alveoli.

The nasal cavity has a large, moist, vascular surface. Mucus catches particles; cilia move mucus toward the pharynx. The pharynx is a shared passage, whereas the larynx protects the lower airway and contains vocal folds. The epiglottis and coordinated closure during swallowing help keep food out of the airway. Tracheal cartilage supports an open lumen. Bronchi contain cartilage; bronchioles lack cartilage and change resistance through smooth muscle.

Ventilation means bulk air movement. External respiration is exchange between alveoli and pulmonary blood; internal respiration is exchange between systemic blood and tissues. Cellular respiration is the intracellular process that uses nutrients to make ATP. Other respiratory functions include voice production, smell, airway protection and regulation of blood pH through CO_2 removal.

Recall: Where does the conducting zone end, and why does that boundary matter?

Terminal bronchioles; air in conducting passages does not directly exchange gas with blood.

Common trap: Do not equate breathing with cellular respiration or call every bronchiole a gas-exchange surface.

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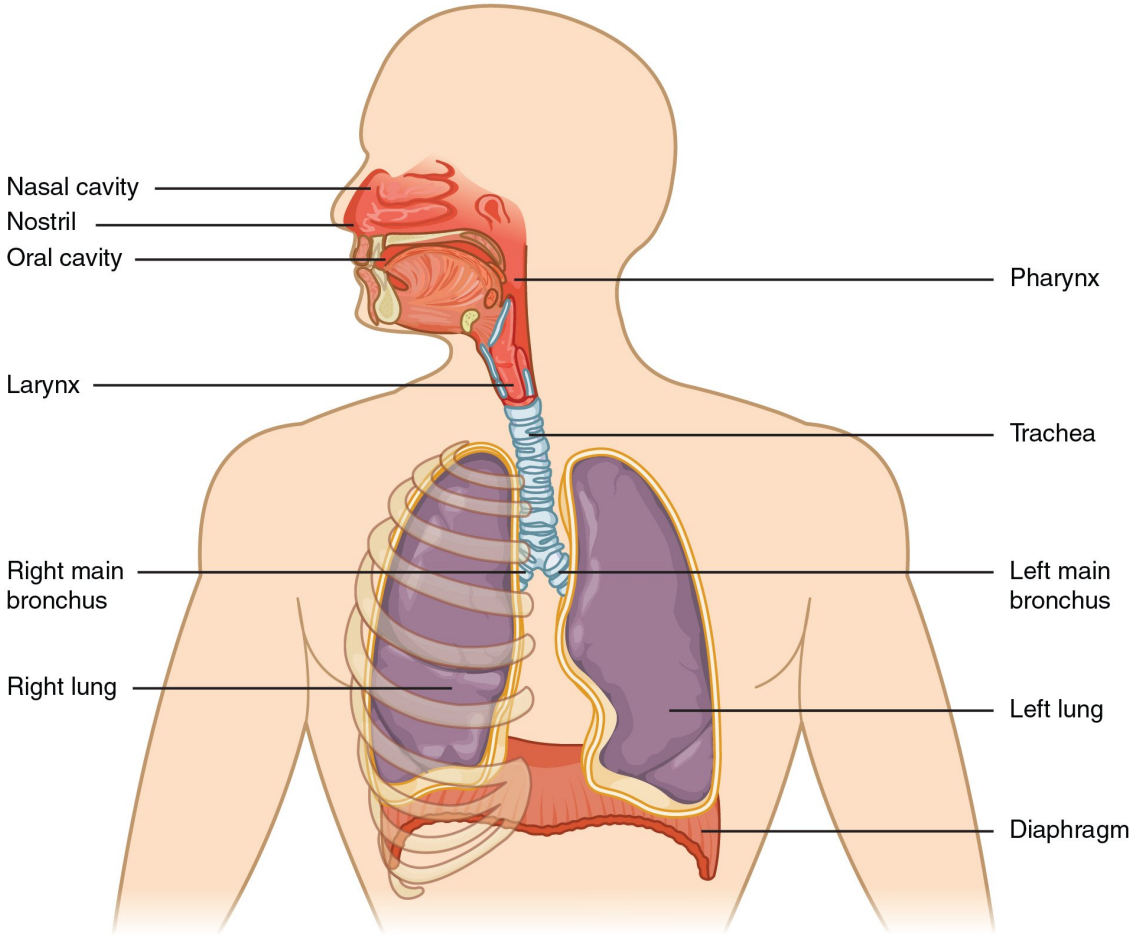
[Core] Major Respiratory Structures



Trace air from nasal cavity to trachea and bronchi; identify the diaphragm below the lungs. This larger textbook view supplements S23 p6.

OpenStax A&P 2e, Fig. 22.2 | https://openstax.org/books/anatomy-and-physiology-2e/pages/22-1-organs-and-structures-of-the-respiratory-system | Publisher original; 1933 x 1625 pixels

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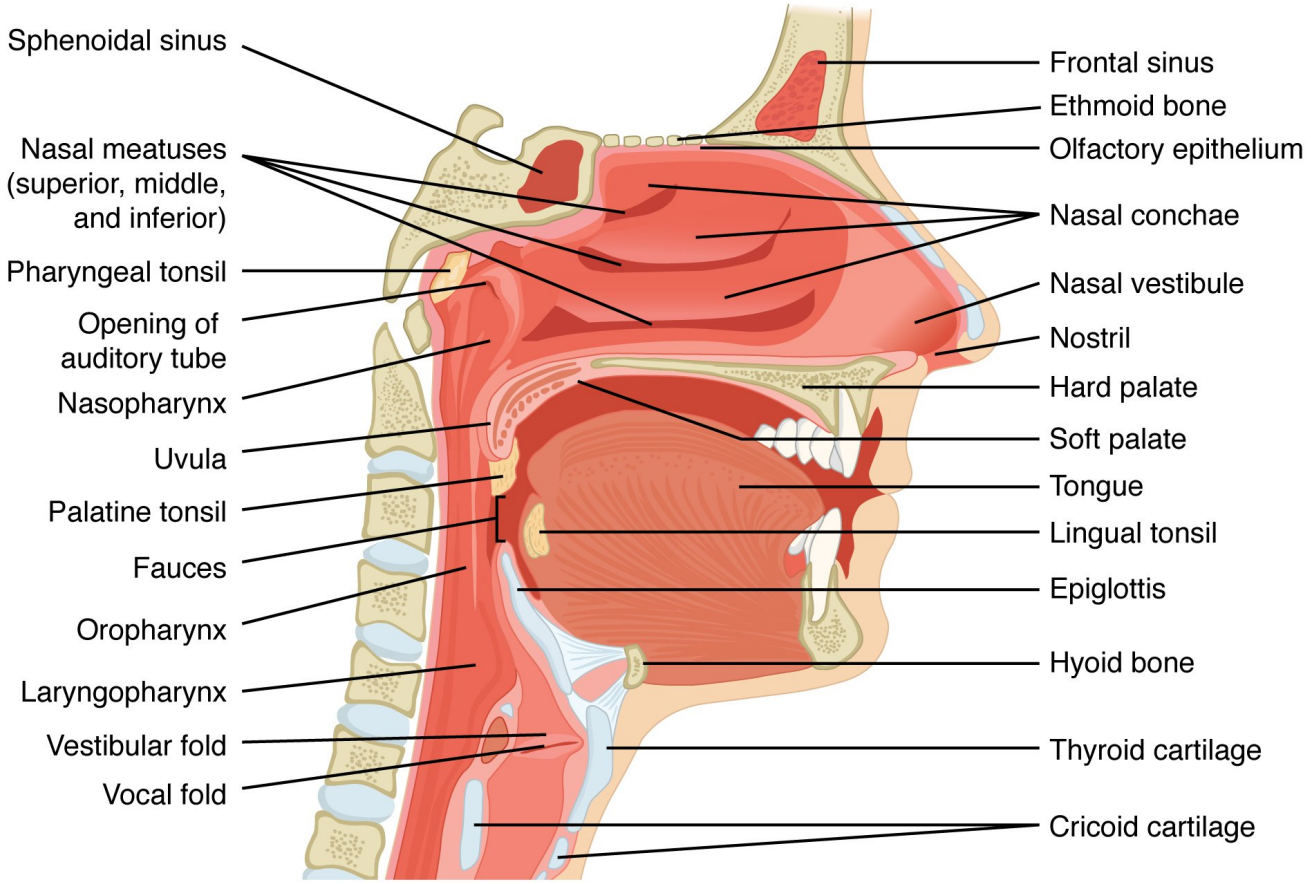
[Core] Upper Airway



Find nasal cavity, hard/soft palate and pharyngeal passage. Relate the separation of routes to swallowing and airway protection.

OpenStax A&P 2e, Fig. 22.4 | https://openstax.org/books/anatomy-and-physiology-2e/pages/22-1-organs-and-structures-of-the-respiratory-system | Publisher original; 1950 x 1351 pixels

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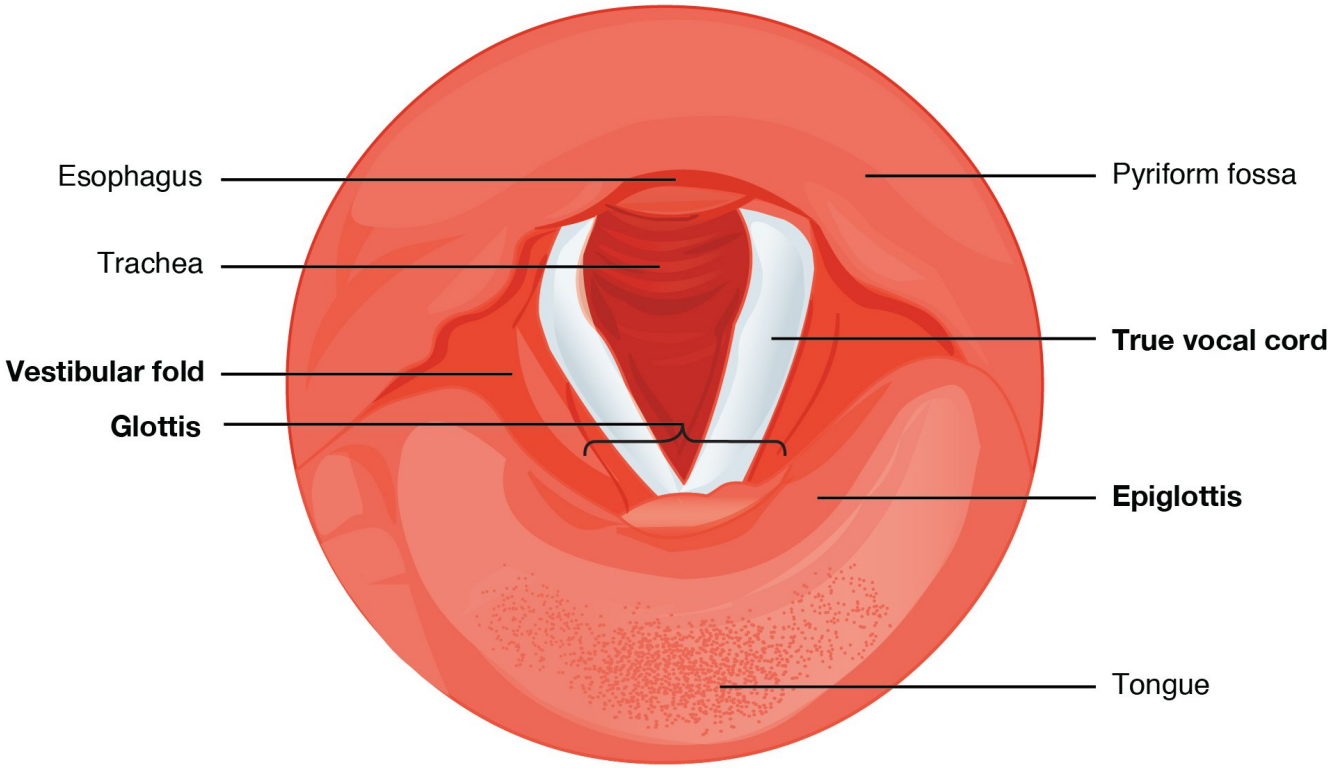
[Core] Vocal Cords



Compare the opening between vocal folds with the folds themselves. Explain how moving air and vibration produce sound.

OpenStax A&P 2e, Fig. 22.8 | https://openstax.org/books/anatomy-and-physiology-2e/pages/22-1-organs-and-structures-of-the-respiratory-system | Publisher original; 1938 x 1133 pixels

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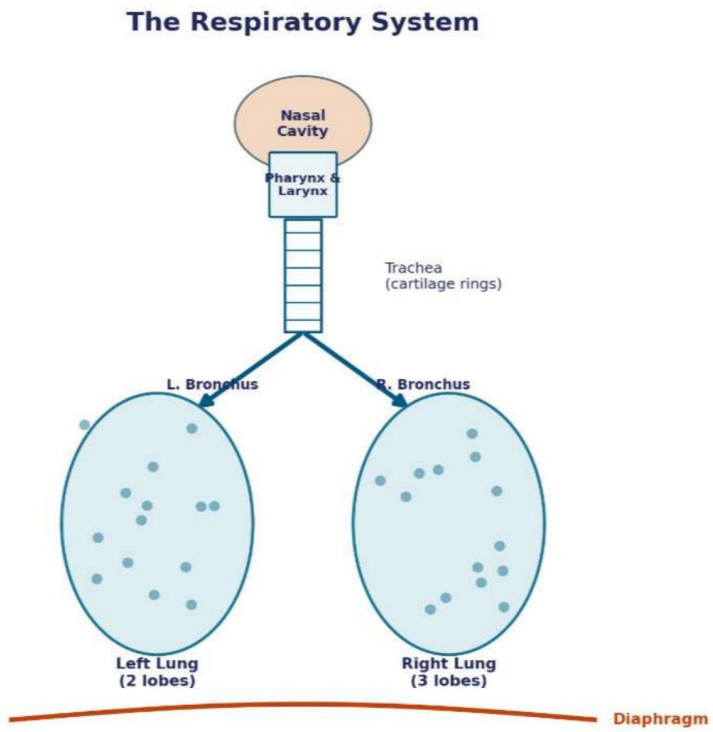
[Core] Airway and lung overview



Conducting zone (nose → trachea → bronchi) vs. respiratory zone (alveoli)

Follow the airway toward the alveoli. The two lungs are not identical: right has three lobes, left has two. The diaphragm forms the muscular floor of the thorax.

S27 p.5 | Original labels retained; figure region enlarged.

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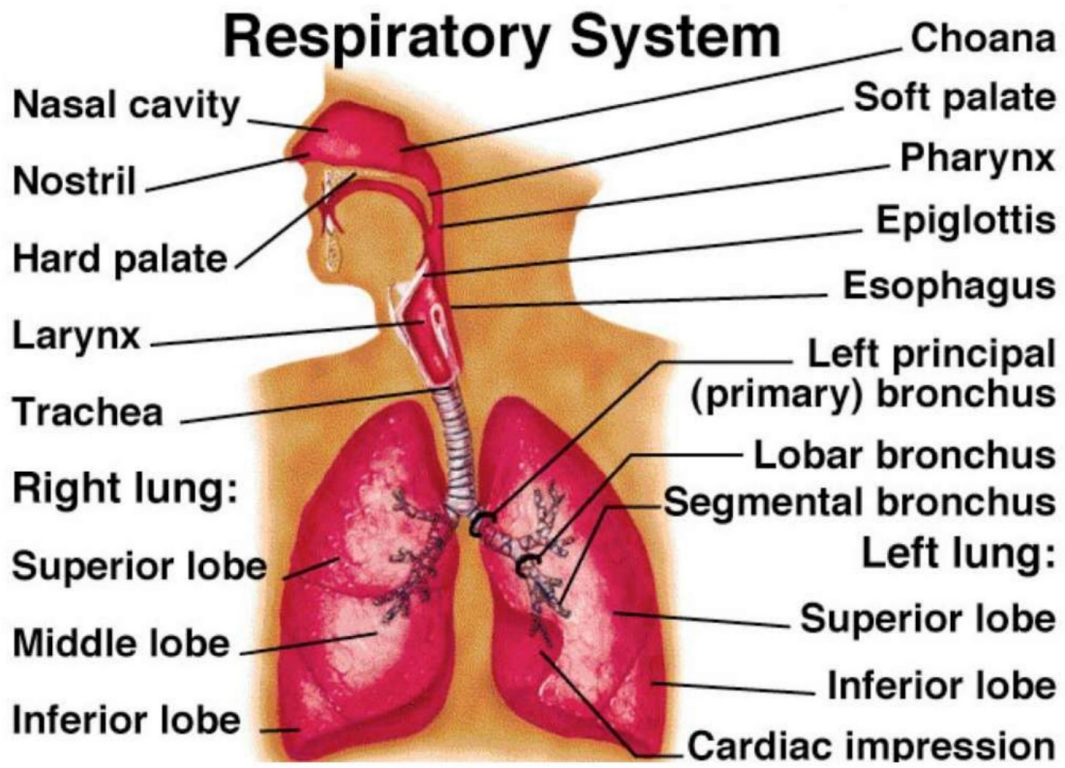
[Core] Principal respiratory organs



Your requested example: trace nasal cavity, pharynx, larynx, trachea and bronchi. Distinguish the posterior esophagus from the anterior airway. Identify the lung lobes and compare right and left lungs.

S23 p.6 | Original labels retained; figure region enlarged.

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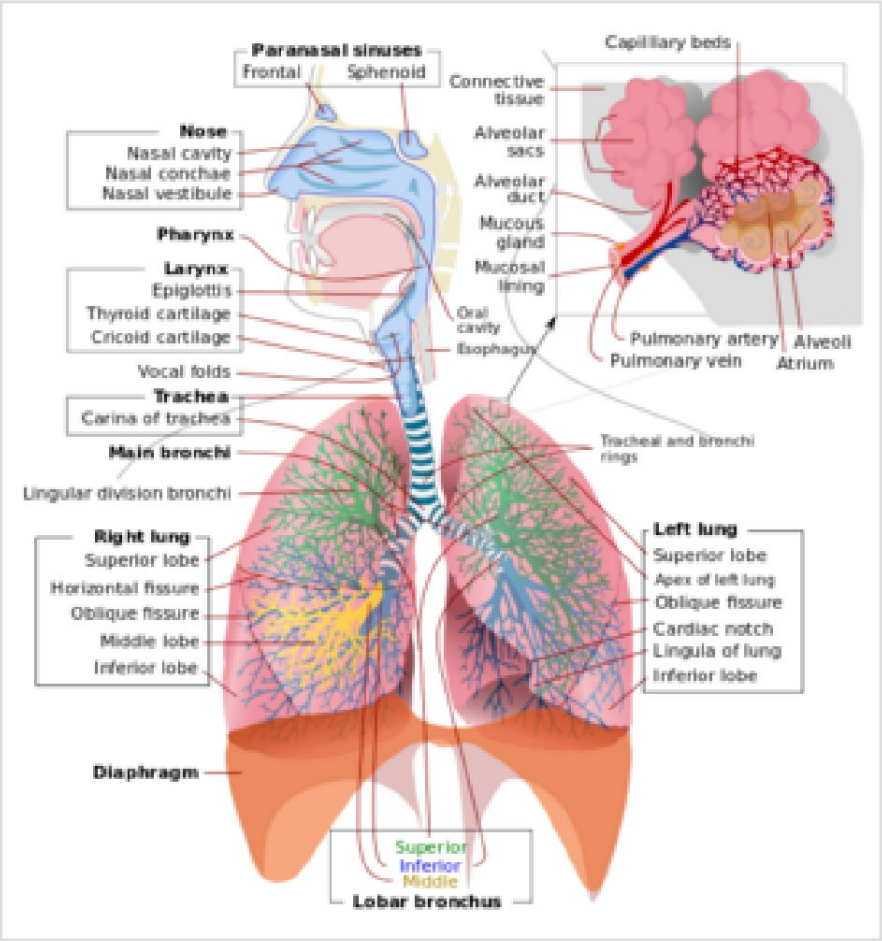
[Core] Whole respiratory pathway



Locate nasal cavity, pharynx, larynx, trachea and lungs, then relate bronchi to the lungs and diaphragm to ventilation.

WR p.1 | Original labels retained; figure region enlarged.

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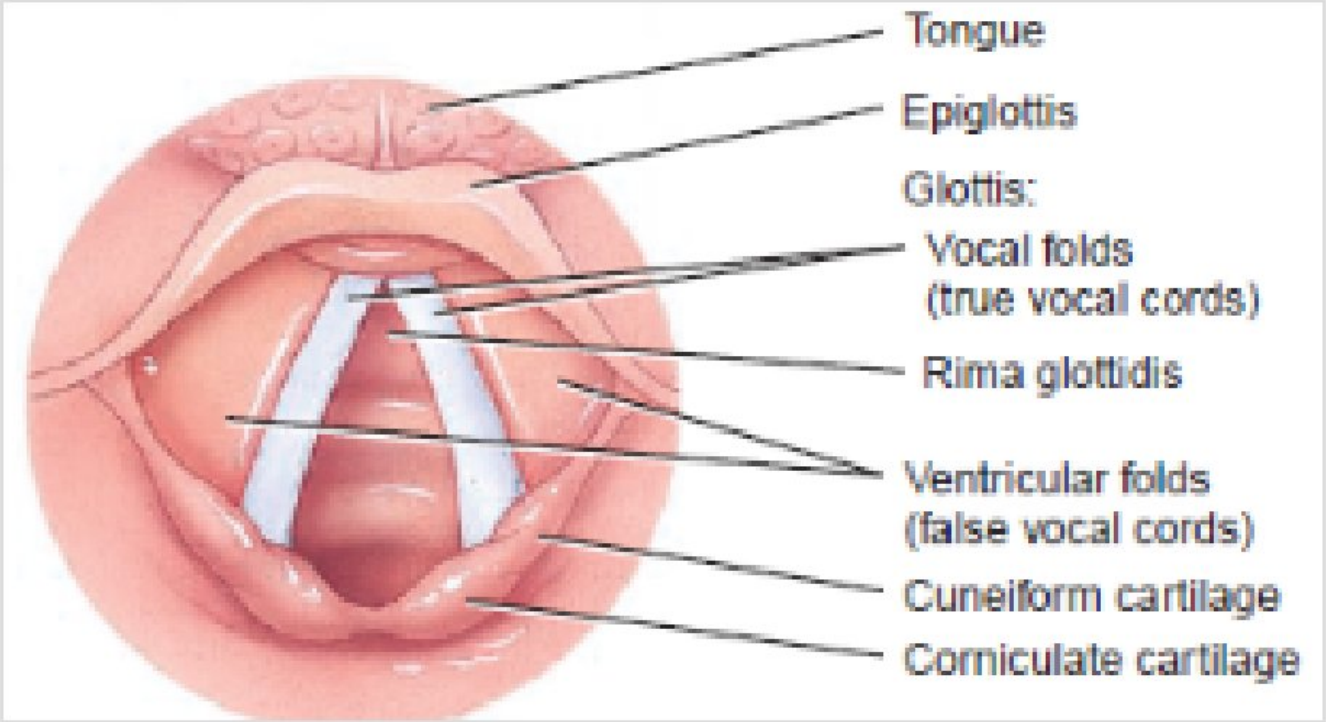
[Core] Vocal folds viewed from above



Locate true vocal folds and glottic opening. Air passing vibrating folds produces sound; they do not remain completely closed during voiced airflow.

WR p.2 | Original labels retained; figure region enlarged.

R-TISSUE | Lungs, pleura and respiratory histology

[Core] | Rule 3a.i

The right lung has three lobes and the left has two, with a cardiac notch. The right main bronchus is generally wider, shorter and more vertical, helping explain why aspirated objects often enter it. The diaphragm separates thoracic and abdominal cavities. Visceral pleura covers the lung; parietal pleura lines the thoracic wall. A thin fluid layer reduces friction and couples lung movement to chest-wall movement.

Recognize trachea by pseudostratified ciliated columnar epithelium, goblet cells and hyaline cartilage. Bronchi show cartilage plates and smooth muscle; bronchioles show smooth muscle but no cartilage or submucosal glands. Smaller bronchioles have simpler cuboidal epithelium. Alveoli look like many thin-walled air spaces rather than a single large airway lumen.

Type I alveolar cells are thin squamous cells covering most gas-exchange surface. Type II cells secrete surfactant and can replace damaged epithelial cells. Surfactant lowers surface tension, increases ease of inflation and limits collapse. Alveolar macrophages ingest particles and microbes. The blood-air barrier includes alveolar epithelium, thin interstitium/shared basal laminae and capillary endothelium; it is not literally one cell in total thickness.

Recall: A lung section shows an airway without cartilage, surrounded by smooth muscle. What is it?

A bronchiole. Look for neighboring alveoli and exclude a blood vessel by its epithelial lining and contents.

Common trap: A diagram is not a histological micrograph. On a micrograph, identify two visible features before naming an organ.

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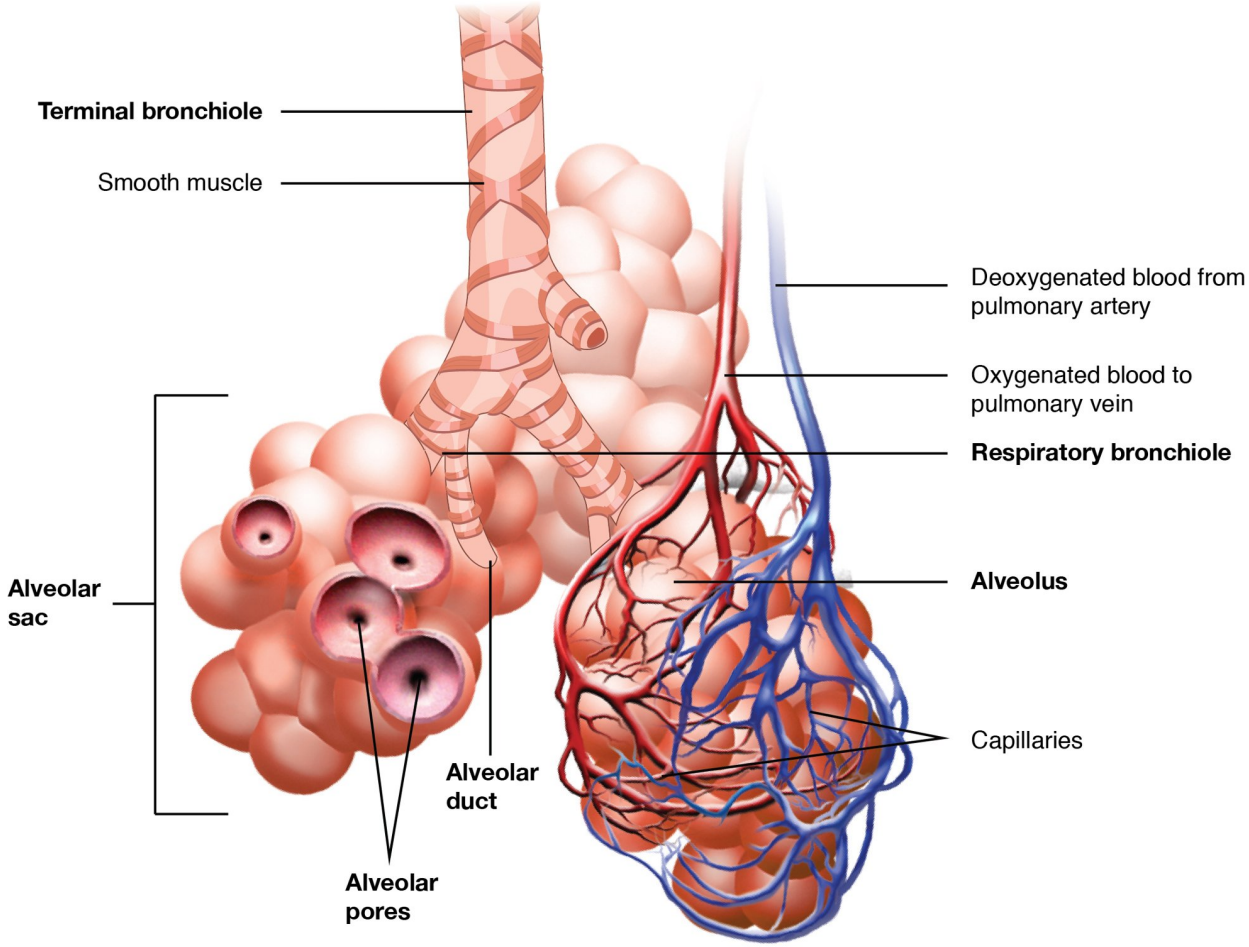
[Core] Respiratory Zone



Follow respiratory bronchiole to alveolar duct and sacs. Identify the capillary network surrounding gas-exchange surfaces.

OpenStax A&P 2e, Fig. 22.10 | https://openstax.org/books/anatomy-and-physiology-2e/pages/22-1-organs-and-structures-of-the-respiratory-system | Publisher original; 2188 x 1671 pixels

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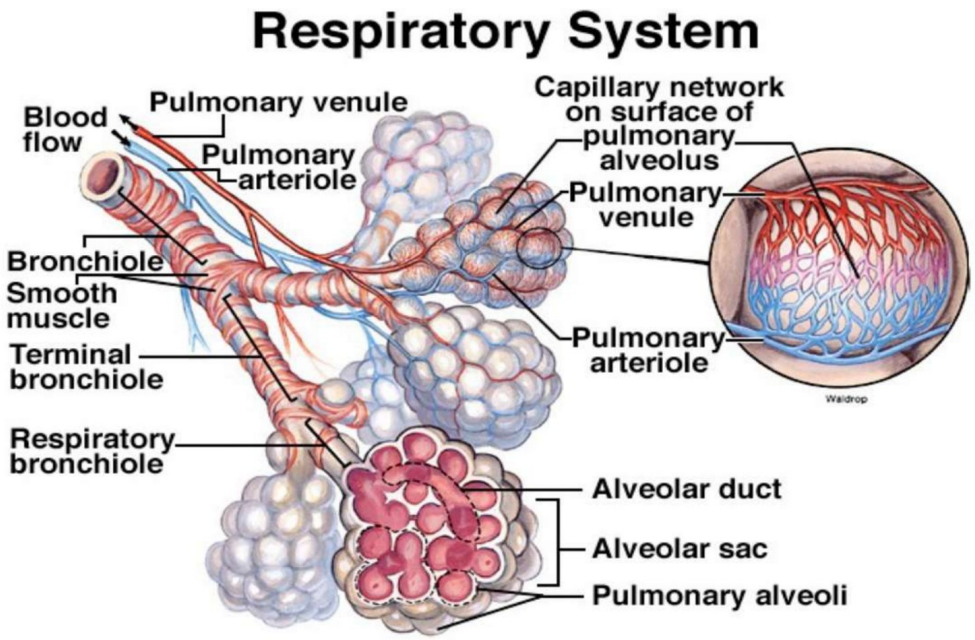
[Core] Bronchioles, alveoli and pulmonary vessels



Follow progressively smaller airways to alveolar ducts and sacs. Pulmonary arteries deliver relatively deoxygenated blood; pulmonary veins return oxygenated blood. Vessel names refer to direction relative to the heart.

S23 p.7 | Original labels retained; figure region enlarged.

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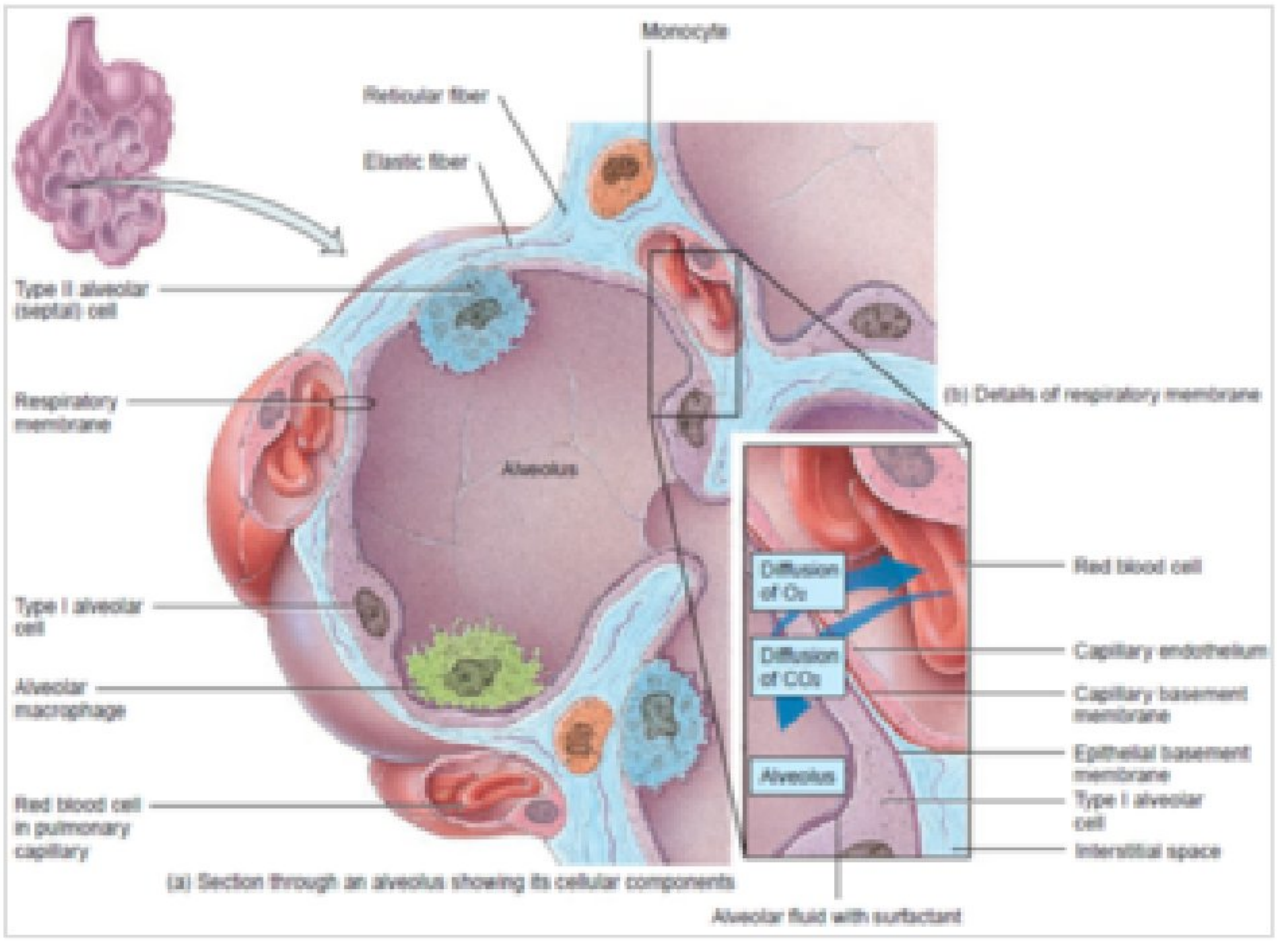
[Core] Alveolar structure and cell types



Identify air spaces, septa and adjacent capillaries. Thin type I cells permit diffusion; type II cells secrete surfactant; macrophages remove inhaled material.

WR p.2 | Original labels retained; figure region enlarged.

R-VENT | Pressure, volume, airflow and ventilation calculations

[Core] | Rule 3a.ii

At a constant temperature, Boyle's law relates pressure inversely to gas volume. During quiet inspiration the diaphragm contracts and descends, and external intercostals elevate the ribs. Thoracic volume increases, pleural pressure becomes more negative, the lungs expand, alveolar pressure briefly falls below atmospheric pressure and air flows inward. At the end of inspiration airflow stops when pressures equalize; the lung is still expanded.

Quiet expiration usually requires no active expiratory muscle contraction: inspiratory muscles relax and elastic recoil decreases volume, briefly raising alveolar pressure above atmospheric pressure. Forced expiration recruits abdominal muscles and internal intercostals. Compliance is volume change divided by pressure change: a compliant lung expands easily. Fibrosis lowers compliance; emphysema can raise compliance while reducing recoil. Narrower airways increase resistance; mucus and bronchoconstriction reduce airflow.

Minute ventilation = tidal volume x respiratory rate. Alveolar ventilation = (tidal volume - dead-space volume) x rate. Convert units first. With TV 500 mL, dead space 150 mL and rate 12/min, minute ventilation is 6000

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mL/min but alveolar ventilation is 4200 mL/min. Two people can have the same minute ventilation but different effective ventilation because each breath includes dead-space air. These are teaching values, not personal medical targets.

[Core] Boyle law

$$P_1V_1 = P_2V_2$$

At constant temperature and gas amount, pressure varies inversely with volume. This explains the pressure changes that help drive breathing.

[Core] Minute and alveolar ventilation

$$\dot{V}_E = V_T f \qquad \dot{V}_A = (V_T - V_D)f$$

VT: tidal volume; VD: dead-space volume per breath; f: breaths per minute. Example: (500 - 150) mL x 12/min = 4.2 L/min alveolar ventilation.

[Core] Flow and resistance

$$\dot{V} = \frac{\Delta P}{R} \qquad R \propto \frac{1}{r^4}$$

A smaller airway radius strongly raises resistance in the ideal laminar-tube model. The fourth-power relationship is a useful approximation, not an exact model of every airway.

Recall: Compare 600 mL x 10/min with 300 mL x 20/min when dead space is 150 mL.

Both minute ventilations are 6 L/min. Alveolar ventilation is 4.5 versus 3.0 L/min; deeper breaths waste a smaller fraction in dead space.

Common trap: Air moves because alveolar and atmospheric pressures differ, not because the lungs actively suck on their own.

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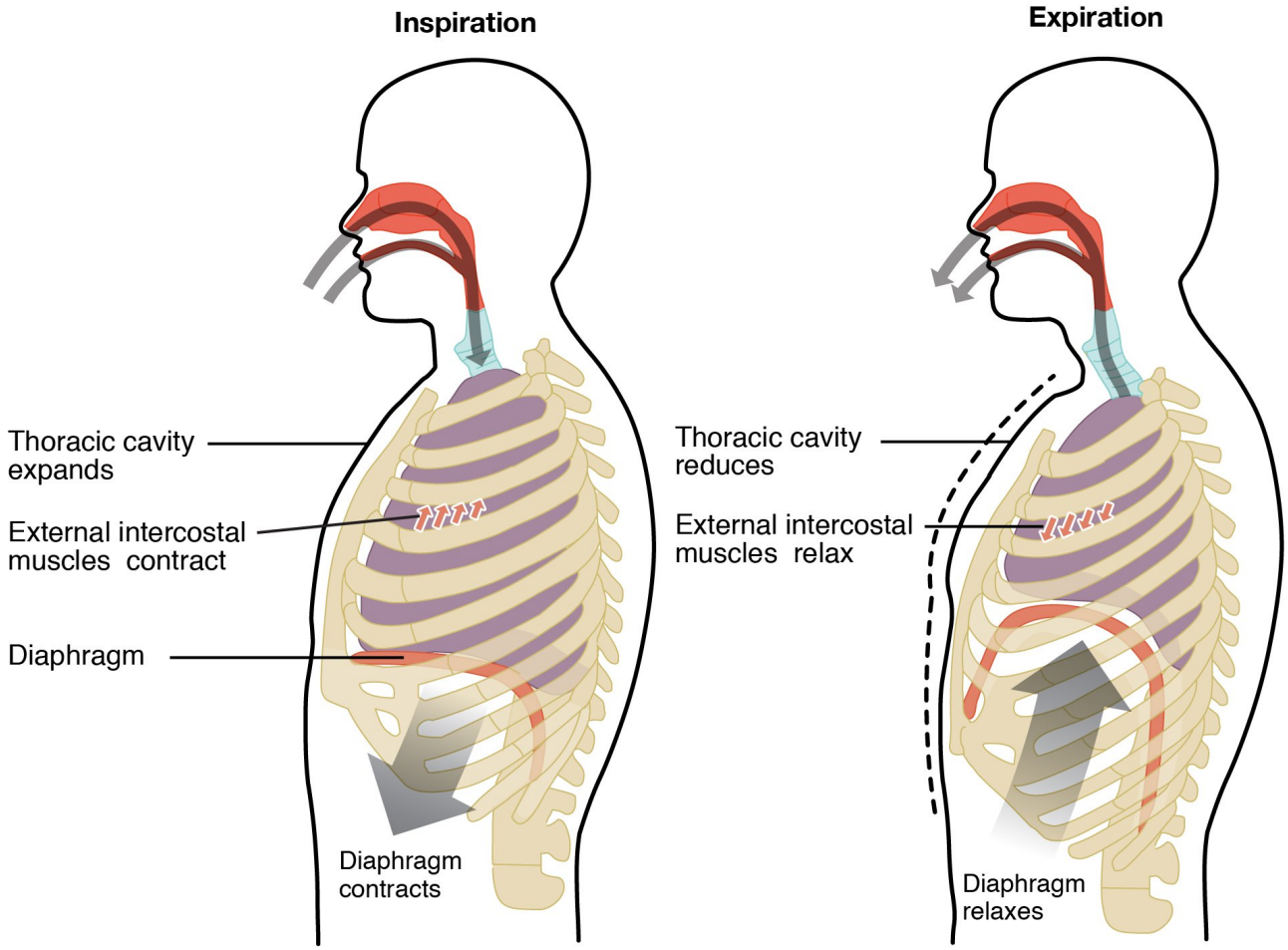
[Core] Normal Inspiration and Expiration



Compare muscle action in the two panels and predict the direction of air movement from the resulting pressure difference.

OpenStax A&P 2e, Fig. 22.17 | https://openstax.org/books/anatomy-and-physiology-2e/pages/22-3-the-process-of-breathing | Publisher original; 1925 x 1422 pixels

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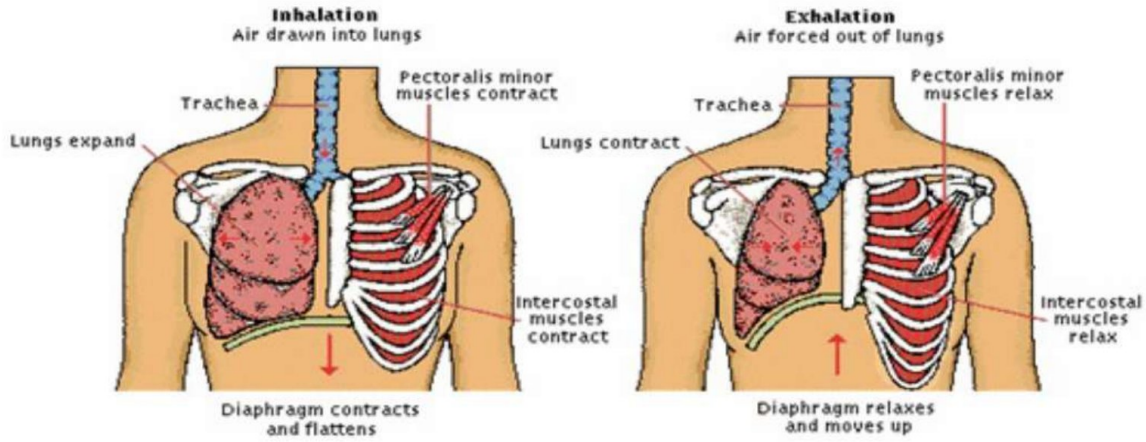
[Core] Inspiration and expiration



During inspiration the diaphragm contracts and descends as the chest expands. During quiet expiration it relaxes and elastic recoil reduces lung volume. Air follows a pressure gradient.

S23 p.10 | Original labels retained; figure region enlarged.

R-PAT | Patterns of breathing

[Core] | Rule 3a.iii

Eupnea is quiet normal breathing. Tachypnea means faster rate; bradypnea means slower rate. Hyperpnea is increased depth, often with rate, appropriate to increased demand such as exercise. Hyperventilation means alveolar ventilation exceeds CO2 production, lowering arterial CO2; hypoventilation means inadequate alveolar ventilation for CO2 production, raising CO2. Rate alone cannot establish either condition.

Apnea is cessation of airflow; dyspnea is the sensation of breathing difficulty; orthopnea is breathlessness while lying flat. Respiratory arrest means breathing has stopped; it does not mean that recovery is impossible. Coughs, sneezes, sighs, speech and hiccups are recognizable non-tidal air movements.

For pattern recognition, Kussmaul breathing is deep increased ventilation associated with compensation for severe metabolic acidosis. Cheyne-Stokes has waxing and waning depth interrupted by apnea. Ataxic/Biot-type patterns are irregular with pauses. Describe a trace in terms of depth, rate, regularity and pauses before naming it; these patterns are clues rather than unique diagnoses.

Recall: Can rapid breathing occur without hyperventilation?

Yes. Rapid shallow breaths may provide little alveolar ventilation, or ventilation may appropriately match increased metabolism.

Common trap: Hyperpnea during exercise is not automatically pathological hyperventilation.

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[Core] Nonrespiratory air movements

Table 17.5 Nonrespiratory Air Movements

Air Movement	Mechanism	Comments
Coughing	Deep inspiration followed by a closure of of the glottis. The forceful expiration that results abruptly opens the glottis, sending a blast of air through the upper respiratory tract.	Reflexive or voluntary. Stimulus may be foreign material irritating the larynx or trachea.
Sneezing	Similar to a cough, except that the forceful expired air is directed primarily through the nasal cavity. The eyelids close reflexively during a sneeze.	Reflexive response to irritating stimulus of the nasal mucosa. Sneezing clears the upper respiratory passages.
Sighing	Deep, prolonged inspiration followed by a rapid, forceful expiration.	Reflexive or voluntary, usually in response to boredom or sadness.
Yawning	Deep inspiration through a widely opened mouth. The inspired air is usually held for a short period before sudden expiration.	Usually reflexive in response to drowsiness, fatigue, or boredom, but exact stimulus-receptor cause is unknown.
Laughing	Deep inspiration followed by a rapid convulsive expiration. Air movements are accompanied by expressive facial distortions.	Reflexive; may be voluntary to express emotions.
Crying	Similar to laughing, but the glottis remains open during entire expiration and different facial muscles are involved.	Somewhat reflexive but under voluntary control.
Hiccuping	Spasmodic contraction of the diaphragm while the glottis is closed, producing a sharp inspiratory sound.	Reflexive; serves no known function.

Compare cough, sneeze, hiccup and speech-related movements. These reuse respiratory muscles or airflow but have different triggers and protective or vocal functions.

S23 p.8 | Original labels retained; figure region enlarged.

R-SPIRO | Spirograms, lung volumes and obstructive/restrictive patterns

[Core] | Rule 3a.ii,iv

TV is the change during a quiet breath. IRV is the additional inhalable volume above an ordinary inspiration. ERV is the additional exhalable volume below an ordinary expiration. RV remains after maximal expiration. VC = IRV + TV + ERV; IC = IRV + TV; FRC = ERV + RV; TLC = VC + RV. FRC is the resting end-expiratory volume. A volume between two levels is a difference, not the value of either level alone.

On a volume-time trace, locate resting end-expiration, resting end-inspiration, maximal inspiration and maximal expiration. If these levels are 2.0, 2.5, 6.0 and 1.2 L, TV = 0.5 L, IRV = 3.5 L, ERV = 0.8 L, RV = 1.2 L, VC = 4.8 L, IC = 4.0 L and FRC = 2.0 L. Ordinary spirometry measures moved air and cannot directly measure RV, FRC or TLC; a teaching graph can supply those values from another method.

FVC is the forced vital capacity; FEV1 is the volume exhaled in its first second. FEV1/FVC is a fraction or percentage. Obstructive disease disproportionately lowers FEV1, commonly lowering the ratio and causing air trapping. Restriction lowers TLC; FVC may be low with a preserved or raised ratio. Low FVC alone does not prove restriction. Interpret age-appropriate reference ranges rather than treating a fixed adult threshold as universal for eighth graders.

[Core] Inspiratory and functional residual capacity

$$IC = V_T + IRV \qquad FRC = ERV + RV$$

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IC starts at the end of quiet expiration. FRC is the air remaining after quiet expiration.

[Core] Vital and total lung capacity

$$VC = IRV + V_T + ERV \qquad TLC = VC + RV$$

RV remains after maximal expiration. A simple spirogram cannot directly measure RV, FRC or TLC.

[Core] Forced expiratory ratio

$$FEV_1/FVC$$

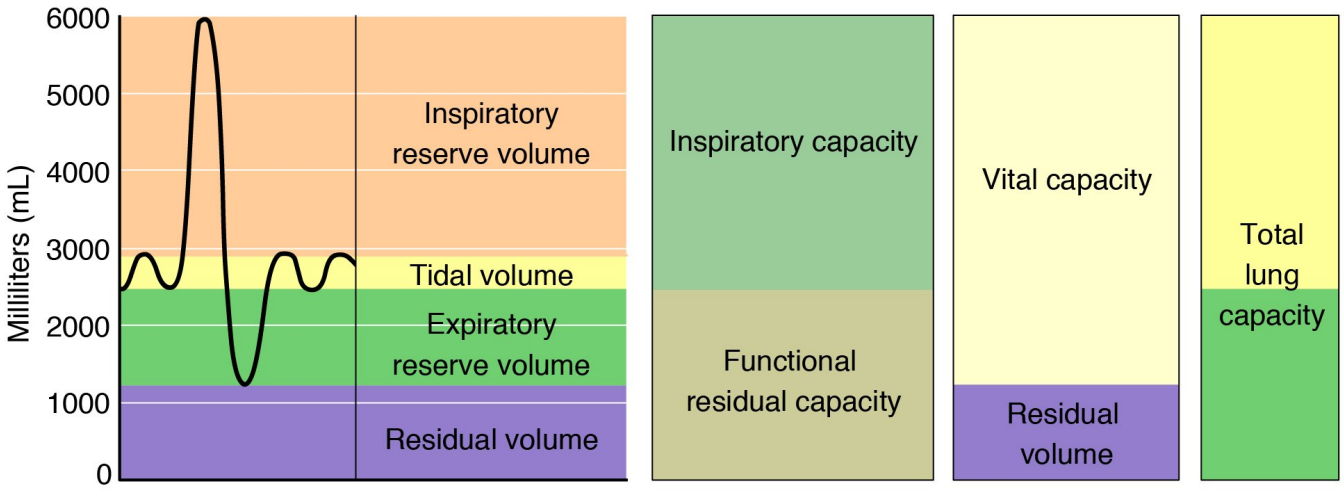
FEV1: volume exhaled in the first second of a forced maneuver; FVC: total forced vital capacity. The ratio commonly falls with obstruction; do not apply one adult cutoff to all children.

Recall: FEV1 = 1.8 L and FVC = 3.6 L. Calculate and interpret.

1.8/3.6 = 0.50 = 50%; a reduced ratio suggests obstruction, interpreted against the appropriate reference range.

Common trap: Vital capacity excludes residual volume; total lung capacity includes it.

[Core] Respiratory Volumes and Capacities



Read the volume axis, identify component volumes and add them to form capacities. RV-containing capacities cannot be measured directly by simple spirometry.

OpenStax A&P 2e, Fig. 22.18 | https://openstax.org/books/anatomy-and-physiology-2e/pages/22-3-the-process-of-breathing | Publisher original; 1940 x 781 pixels

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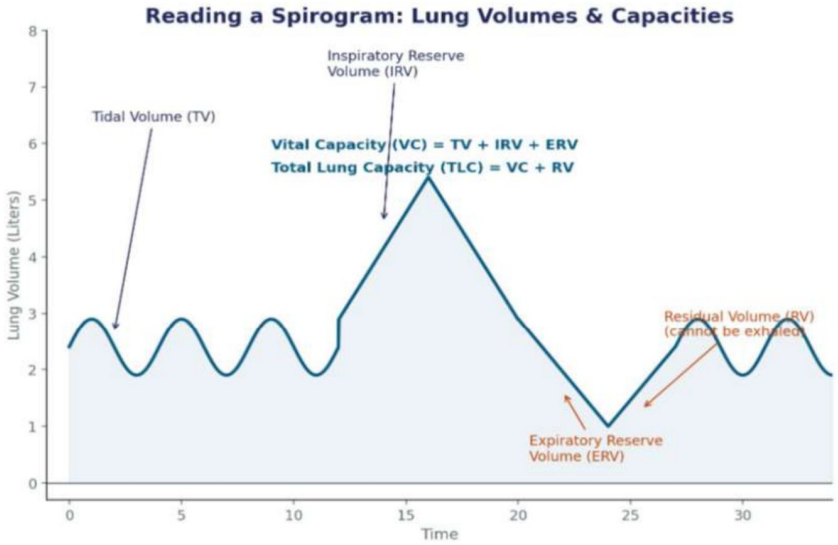
[Core] Schematic spirogram



Lung volumes/capacities plotted on a spirometer trace

Identify quiet tidal breaths, maximal inspiration and maximal expiration. Read changes vertically on the volume axis. Source schematic is qualitative: use the formula panel and textbook graph for standard volume relationships. Source caution: its RV arrow and capacity annotations are oversimplified; use the textbook graph and equations for quantitative practice.

S27 p.8 | Original labels retained; figure region enlarged.

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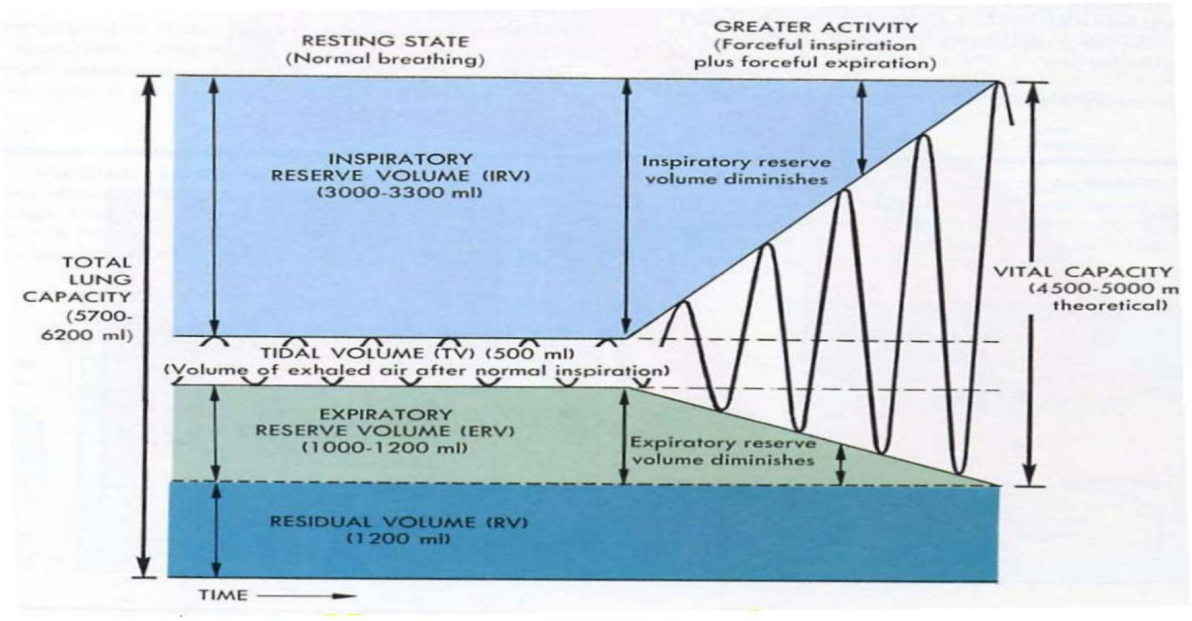
[Core] Respiratory volumes and capacities



Locate TV, IRV, ERV and RV, then assemble IC, FRC, VC and TLC. The small tidal oscillations sit above RV plus ERV. Values are illustrative, not universal normal values.

S23 p.12 | Original labels retained; figure region enlarged.

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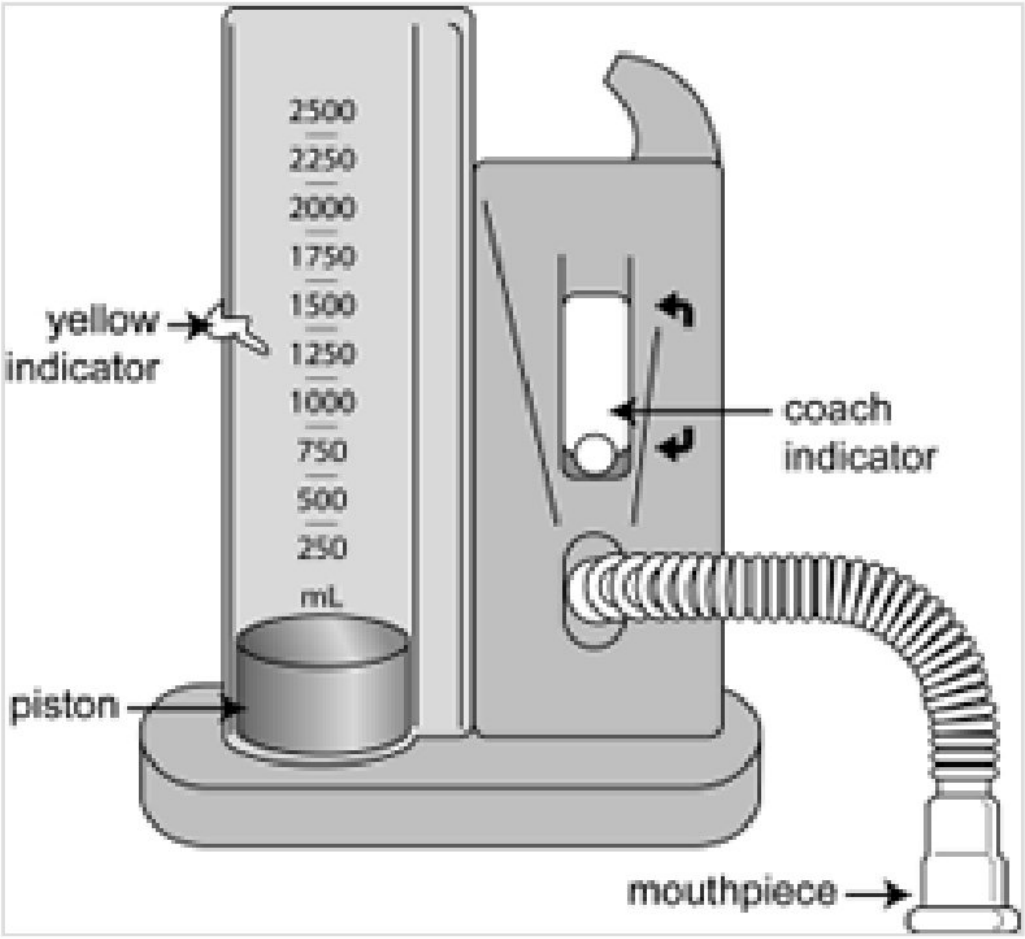
[Core] Incentive spirometer



This pictured device is an incentive spirometer: the patient inhales to raise a piston and uses an indicator as a goal. It is a breathing-exercise device, not the diagnostic apparatus that records a forced expiratory spirogram. Do not use this drawing to infer an FEV1 measurement.

WR p.4 | Original labels retained; figure region enlarged.

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[Core] Spirogram with capacities

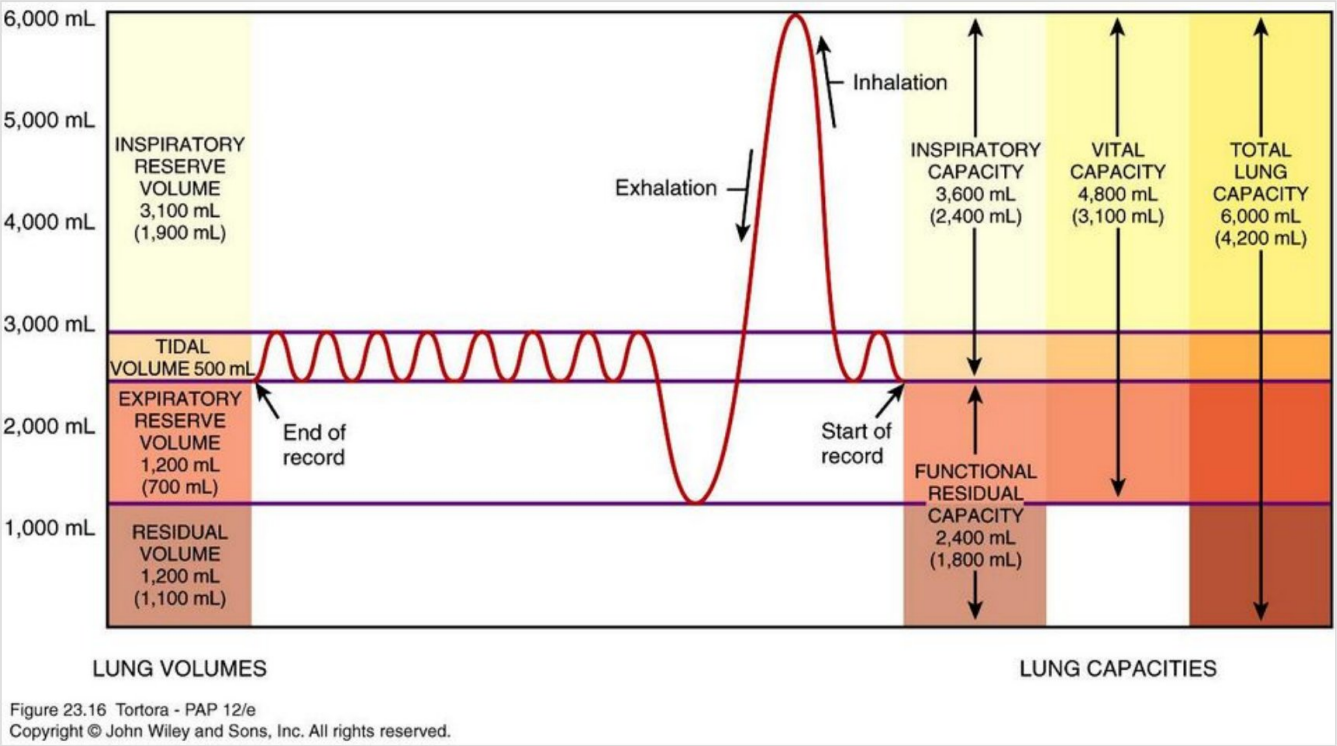

Figure 23.16 Tortora - PAP 12/e
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Read tidal breaths, maximal inspiration and maximal expiration, then add component volumes. The displayed numbers are teaching examples rather than fixed values for every person.

WR p.5 | Original labels retained; figure region enlarged.

R-GAS | Gas exchange, diffusion and ventilation-perfusion matching

[Core] | Rule 3a.v

Each gas diffuses down its own partial-pressure gradient. Oxygen moves from alveolar air into arriving pulmonary blood; carbon dioxide moves from that blood into alveoli. In systemic tissues the directions reverse. Pulmonary arteries bring relatively oxygen-poor blood to the lungs; pulmonary veins return relatively oxygen-rich blood to the heart.

Fick's diffusion law predicts more diffusion with greater area, greater pressure difference and higher diffusivity, and less diffusion with greater barrier thickness. Emphysema removes surface area; edema adds fluid and diffusion distance; fibrosis thickens and stiffens the exchange barrier. Thin alveolar walls and a dense capillary network support exchange.

Ventilation is air supply and perfusion is blood supply. Good exchange requires both. A blocked airway can leave perfusion without adequate ventilation, producing a low V/Q region. A blocked pulmonary vessel leaves ventilation with little perfusion, increasing physiological dead space. This V/Q language is useful explanatory depth; memorize the causal chain rather than unrelated diagnostic formulas.

[S] [N] Blood-chemistry extension - chloride shift: in systemic capillaries, carbonic anhydrase in red cells accelerates CO2 + H2O <-> H2CO3 <-> H+ + HCO3-. Bicarbonate exits in exchange for chloride, preserving electrical neutrality; the exchange reverses in lungs. This is not an exchange because the ions occupy opposite sides of a pH scale. Venous blood still contains oxygen; hemoglobin is not completely emptied at

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rest.

[Core] Diffusion relationship

$$\text{Rate} \propto \frac{A\,\Delta P}{T}$$

A: surface area; delta P: partial-pressure gradient; T: barrier thickness. More area or gradient favors diffusion; greater thickness impairs it.

[Core] Partial pressure

$$P_{\text{gas}} = F_{\text{gas}} P_{\text{total}}$$

For a dry gas mixture, partial pressure equals fraction times total pressure. At altitude, oxygen fraction stays near 21% while atmospheric pressure falls.

Recall: Why can an alveolus full of fluid cause low blood oxygen even when blood still flows past it?

Ventilation is impaired and diffusion distance increases, so the oxygen supply to perfused blood is reduced.

Common trap: Partial pressure, oxygen saturation and total oxygen content are different quantities.

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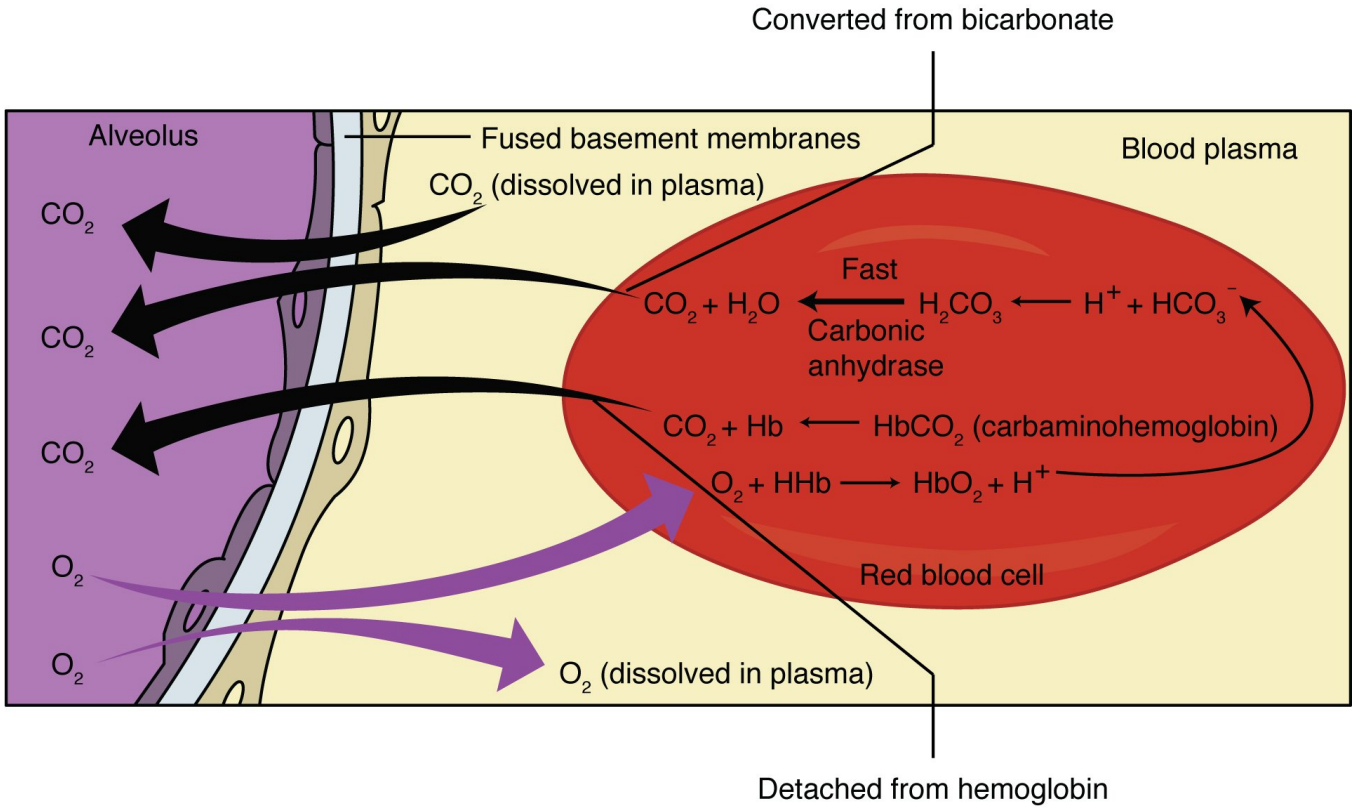
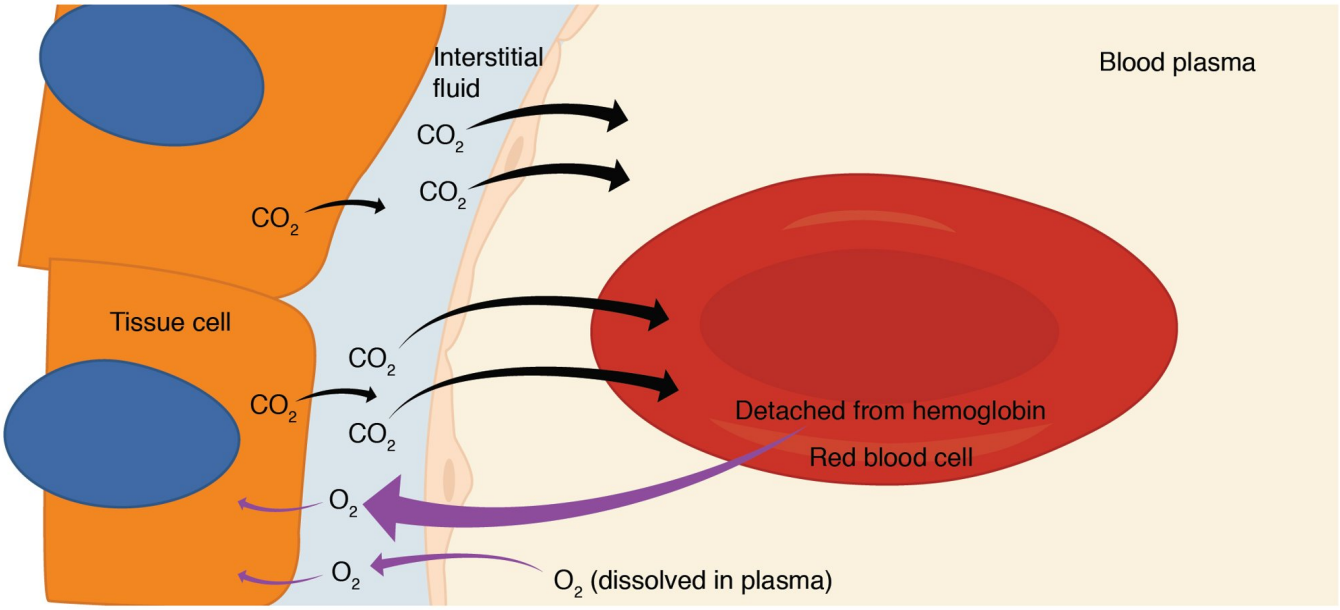
[Core] External Respiration



Trace oxygen into pulmonary blood and CO2 into the alveolus. Compare partial pressures rather than total gas amounts.

OpenStax A&P 2e, Fig. 22.22 | https://openstax.org/books/anatomy-and-physiology-2e/pages/22-4-gas-exchange | Publisher original; 1921 x 1152 pixels

[Core] Internal Respiration



Reverse the exchange direction at tissues: oxygen leaves systemic blood and CO2 enters it.

OpenStax A&P 2e, Fig. 22.23 | https://openstax.org/books/anatomy-and-physiology-2e/pages/22-4-gas-exchange | Publisher original; 2165 x 980 pixels

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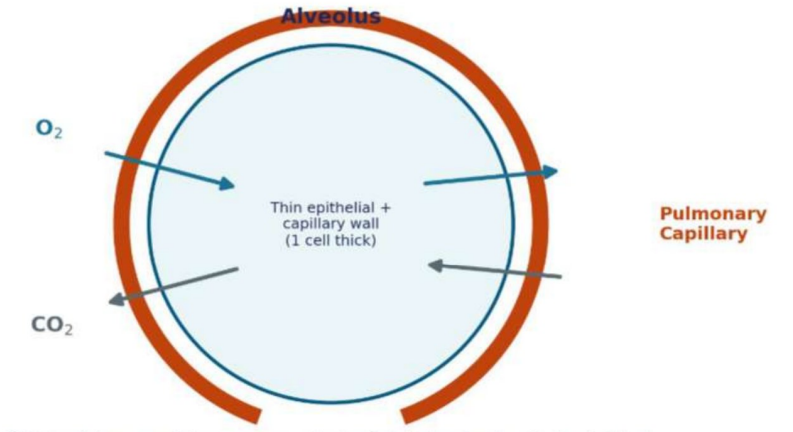
[Core] Alveolus and pulmonary capillary



O_2 and CO_2 diffuse down partial-pressure gradients across the alveolar-capillary membrane

Oxygen diffuses from alveolar air into blood; carbon dioxide moves from blood to air. The barrier includes alveolar epithelium, thin intervening structures and capillary endothelium, not one cell total.

S27 p.9 | Original labels retained; figure region enlarged.

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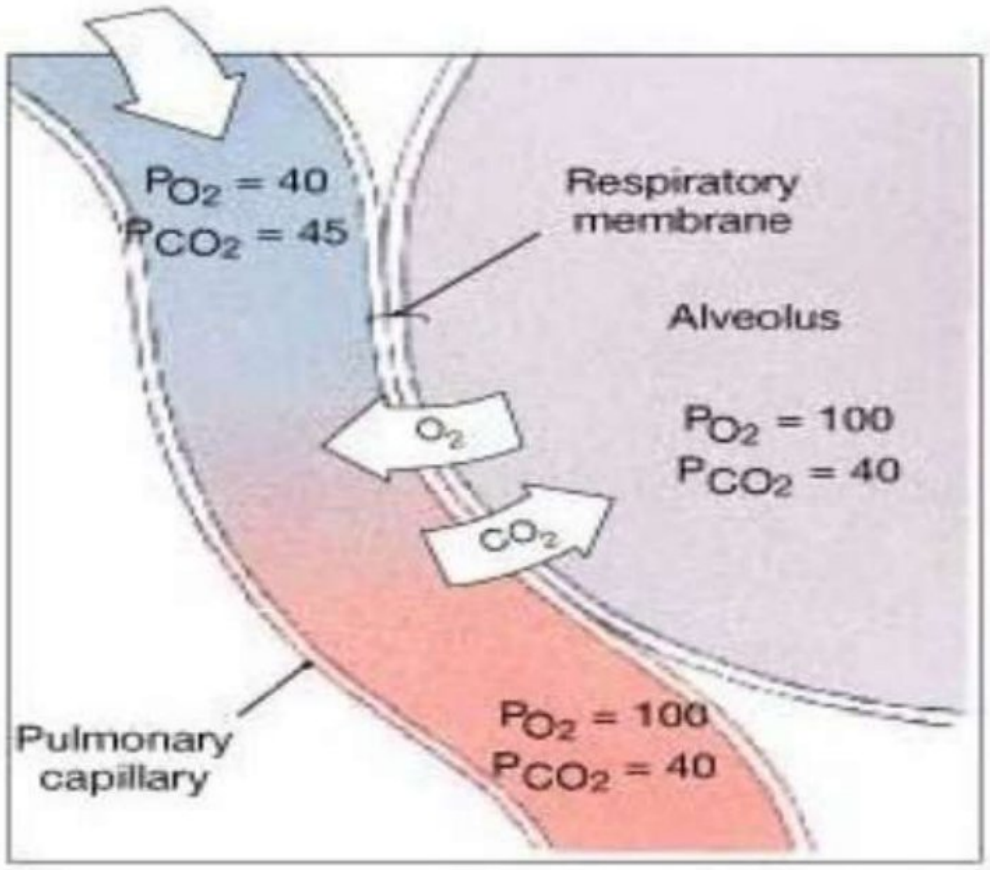
[Core] External gas exchange



In pulmonary capillaries, oxygen enters blood and carbon dioxide enters alveolar air down partial-pressure gradients. This crop omits the slide text that incorrectly describes most CO2 as carbonic acid.

S23 p.16 | Original labels retained; figure region enlarged.

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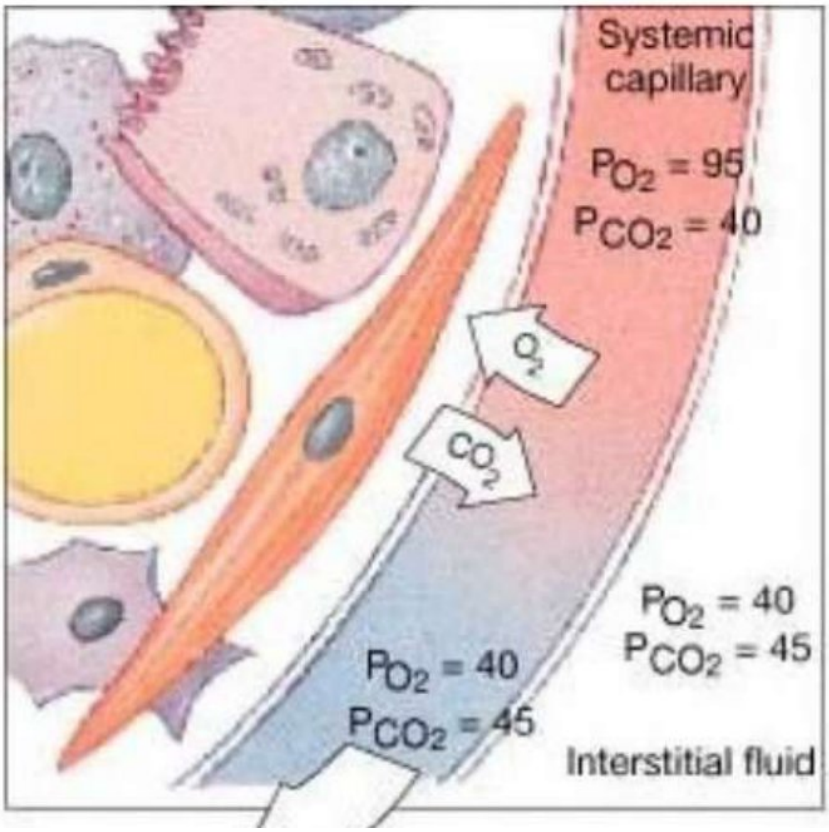
[Core] Systemic gas exchange



In tissue capillaries, oxygen leaves blood and metabolically produced carbon dioxide enters blood. [S] [N] Blood-chemistry extension: deoxygenated hemoglobin more readily carries CO2 and buffers H+, contributing to the Haldane effect.

S23 p.17 | Original labels retained; figure region enlarged.

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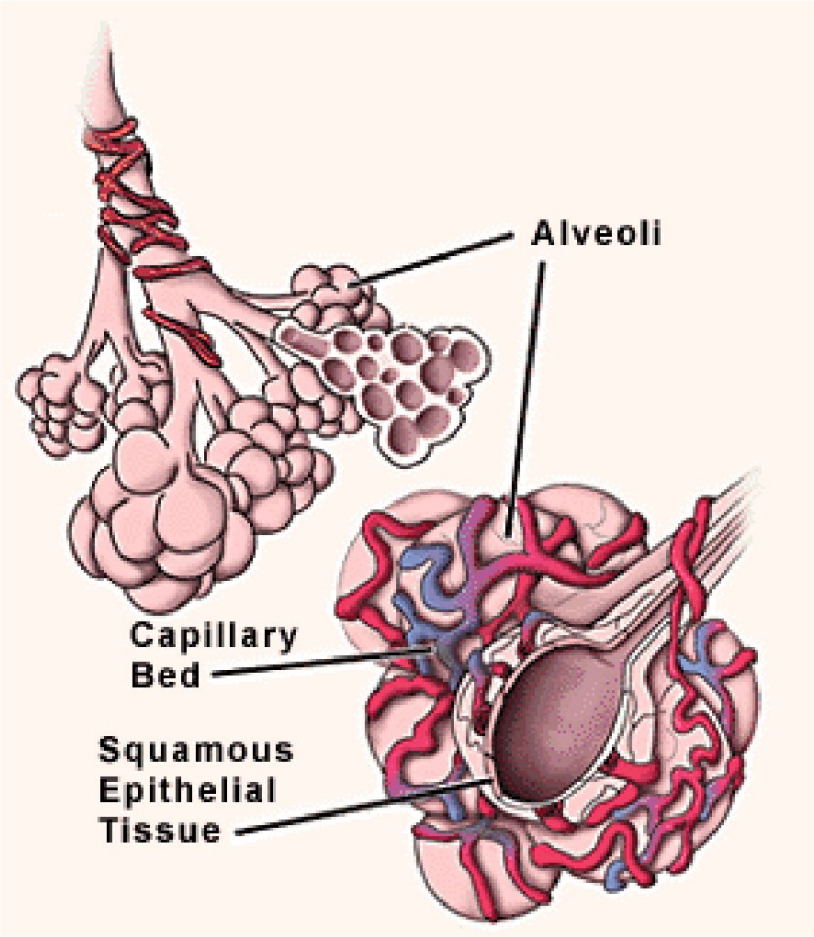
[Core] Alveolar-capillary gas-exchange surface



Match squamous epithelium and capillary walls to a short diffusion path. Oxygen and CO2 move in opposite directions because their partial-pressure gradients differ.

WR p.7 | Original labels retained; figure region enlarged.

R-HB | Oxygen transport, dissociation curves and carbon dioxide

[Core] | Rule 3a.v

Most oxygen is bound reversibly to hemoglobin; only a small amount is dissolved. Partial pressure reflects dissolved oxygen, saturation is the fraction of binding sites occupied, and oxygen content also depends on how much hemoglobin is present. Anemia can lower oxygen content despite a normal saturation.

The oxygen-hemoglobin curve is S-shaped because binding is cooperative. The plateau favors loading in the lungs; the steeper section permits substantial unloading as tissue PO2 falls. At the same PO2, a right shift means lower saturation and easier release. Increased CO2, increased temperature, decreased pH and increased 2,3-BPG favor a right shift. Opposite changes favor a left shift. The Bohr effect specifically links CO2/H+ to reduced oxygen affinity. Compare curves at the same horizontal coordinate.

[S] [N] CO2 transport and blood chemistry: CO2 travels mainly as bicarbonate, with smaller fractions bound to proteins such as hemoglobin or dissolved. Carbonic anhydrase in red cells speeds CO2 + H2O <-> H2CO3 <-> H+ + HCO3-. Bicarbonate exchange with chloride helps transport it in plasma. The Haldane effect means deoxygenated hemoglobin carries/buffers CO2 and H+ more readily; oxygenation in the lungs promotes CO2

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unloading. Approximate fractions vary; do not memorize the older slide's claim that 90% travels as carbonic acid.

[Core] Hemoglobin saturation

$$S_{O_2} = \frac{\text{occupied } O_2 \text{ binding sites}}{\text{total } O_2 \text{ binding sites}} \times 100\%$$

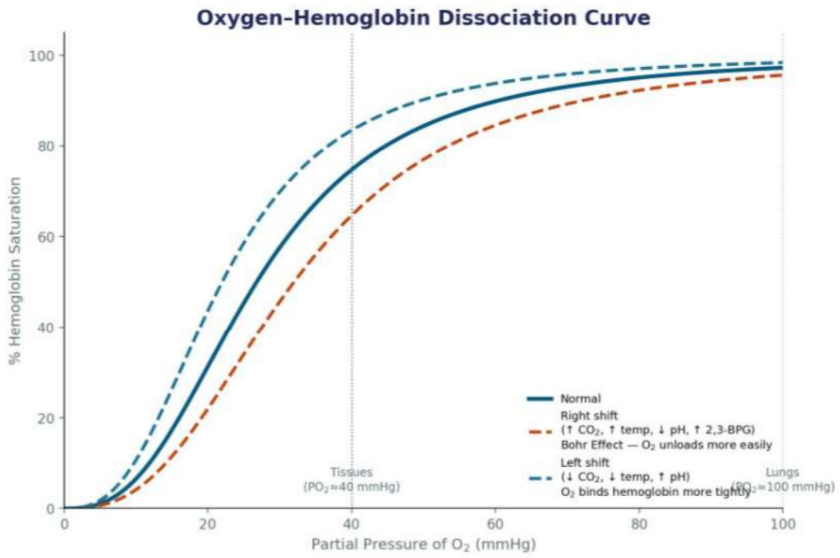
Read saturation on the vertical axis of the dissociation curve. Saturation is not identical to total blood oxygen content.

Recall: At one tissue PO2, curve B is below curve A. What does that mean?

B has lower hemoglobin saturation at the same PO2, consistent with greater oxygen unloading from a right-shifted curve.

Common trap: Bohr = effects on oxygen binding; Haldane = effects of oxygenation on CO2/H+ carriage.

[Core] Oxygen-hemoglobin dissociation curves



Sigmoidal O_2-hemoglobin dissociation curve, with Bohr-effect shifts

Compare saturation at the same oxygen partial pressure. A right shift means lower affinity and easier oxygen unloading; the plateau protects loading at high partial pressures.

S27 p.10 | Original labels retained; figure region enlarged.

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R-EX | Exercise and high altitude

[Core] | Rule 3a.vi

Exercise increases oxygen consumption and CO2 production. Ventilation increases in depth and rate; cardiac output and tissue oxygen extraction also rise. Heat, CO2 and H+ in active muscle facilitate oxygen unloading. Normal exercise does not require a large sustained rise in arterial CO2: anticipatory neural drive and feedback increase ventilation to meet demand.

At altitude the oxygen fraction remains near 21%, but total barometric pressure falls, so inspired oxygen partial pressure is lower. The immediate response includes increased ventilation, which lowers CO2 and can raise pH. Over days the kidneys help compensate for alkalosis; over longer periods erythropoietin supports increased red-cell production. Increased 2,3-BPG can aid unloading.

Distinguish a rapid response (breathing faster/deeper) from acclimatization (renal compensation, more red cells). Altitude illness and high-altitude edema are relevant connections; fitness alone does not prevent them. Training improves oxygen delivery/use and exercise tolerance, but healthy lung size or vital capacity does not necessarily increase greatly in every athlete.

Recall: Why is there less usable oxygen at altitude despite a similar percentage of oxygen?

Lower atmospheric pressure reduces oxygen partial pressure and the gradient driving oxygen into blood.

Common trap: Do not say the oxygen percentage falls substantially with ordinary altitude exposure.

R-CTRL | Breathing control and acid-base chemistry

[S] [N] | Rule 3a.ix

Brainstem networks in the medulla generate respiratory rhythm; pontine networks help shape transitions and pattern. Motor output reaches the diaphragm via phrenic nerves and intercostal muscles via spinal nerves. Voluntary cortical control, emotion, pain and exercise can modify breathing, but chemical feedback limits prolonged voluntary breath holding.

Central chemoreceptors respond chiefly to changes in cerebrospinal-fluid H+ caused by CO2 crossing the blood-brain barrier. Peripheral chemoreceptors in carotid and aortic bodies detect arterial PO2, CO2 and pH; low oxygen becomes a strong stimulus when arterial PO2 falls markedly. They sense partial pressure, not simply total oxygen content.

Hypoventilation retains CO2, shifts the carbonic-acid equilibrium toward H+ and produces a primary respiratory acidosis. Hyperventilation lowers CO2 and tends toward respiratory alkalosis. Metabolic acidosis can stimulate compensatory increased ventilation. Kidneys change bicarbonate handling more slowly than lungs alter CO2. In a problem, first determine which variable changed, then predict CO2, pH and the compensating organ; do not call compensation a cure of the underlying cause.

[S] [N] Carbon dioxide buffer system

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

Carbonic anhydrase speeds the first equilibrium in red cells. More retained CO2 tends to increase H+; ventilation helps regulate pH.

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[S] [N] pH definition

$$pH = -\log_{10}[H^+]$$

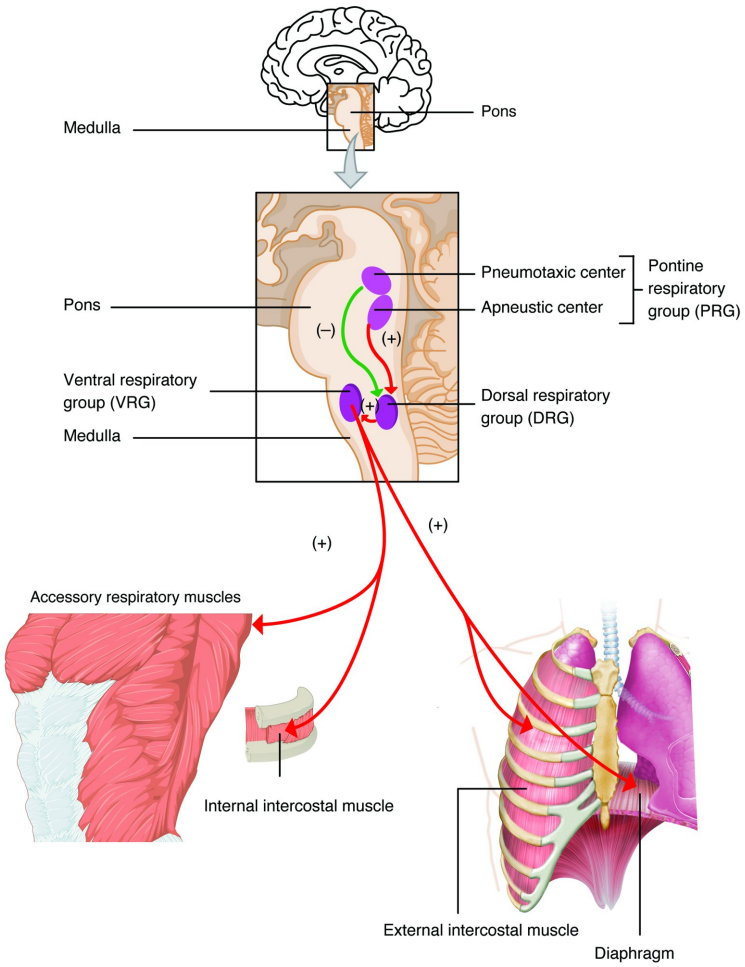
Higher hydrogen-ion concentration means lower pH. This is a logarithmic scale.

Recall: A person hypoventilates. Predict CO2 and pH before renal compensation.

CO2 rises and pH falls: respiratory acidosis.

Common trap: The brain primarily senses CO2 through CSF acidity; peripheral receptors are the major low-arterial-oxygen sensors.

[S] [N] Respiratory Centers of the Brain



Identify medulla and pons in the brainstem. Connect rhythm generation to modulation and motor output to breathing muscles.

OpenStax A&P 2e, Fig. 22.20 | https://openstax.org/books/anatomy-and-physiology-2e/pages/22-3-the-process-of-breathing | Publisher original; 1883 x 2447 pixels

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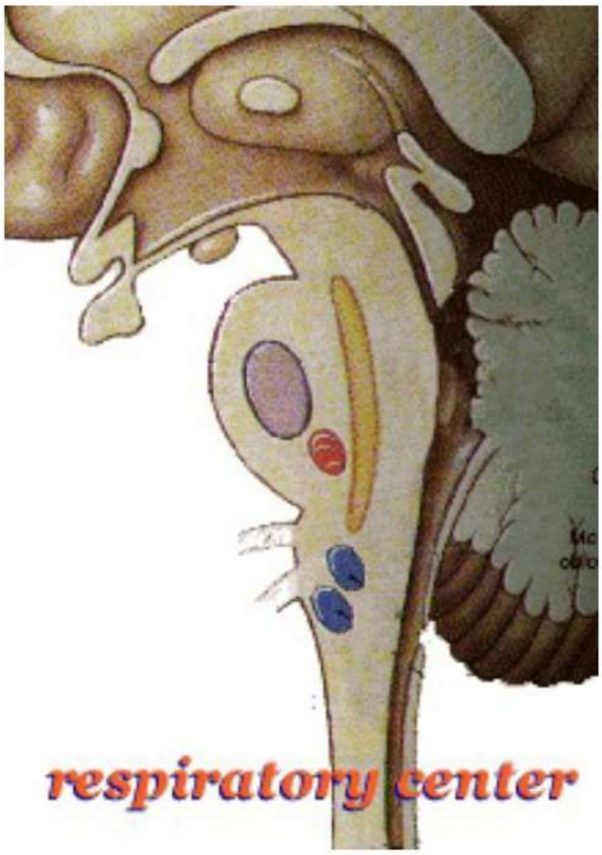
[S] [N] Brainstem respiratory centers



Locate medulla and pons. Brainstem networks generate and modify rhythm, while sensory feedback adjusts ventilation. The diaphragm is the effector muscle rather than the rhythm generator.

S23 p.15 | Original labels retained; figure region enlarged.

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Respiratory diseases

COPD

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Persistent airflow limitation from airway disease and/or emphysema; tobacco smoke and other inhaled exposures are major risks.

Recognition clues: Chronic cough, sputum, exertional breathlessness; reduced FEV1/FVC.

Distinguish: Chronic bronchitis emphasizes mucus/airway inflammation; emphysema emphasizes loss of alveolar walls and recoil.

[S] [N] Treatment: Bronchodilators, pulmonary rehabilitation; selected patients receive inhaled anti-inflammatory treatment or oxygen.

[S] [N] Prevention: Avoid tobacco and harmful inhaled exposures; indicated vaccination and exposure control reduce additional harm.

Asthma

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Variable airway inflammation, smooth-muscle constriction and mucus reduce airway caliber.

Recognition clues: Episodic wheeze, cough, chest tightness; variable airflow obstruction.

Distinguish: Usually more variable/reversible than COPD; both can obstruct airflow.

[S] [N] Treatment: Inhaled corticosteroid-containing treatment controls inflammation; bronchodilators relieve constriction.

[S] [N] Prevention: Reduce relevant triggers and smoke exposure; control plans reduce attacks, but there is no universal guaranteed prevention.

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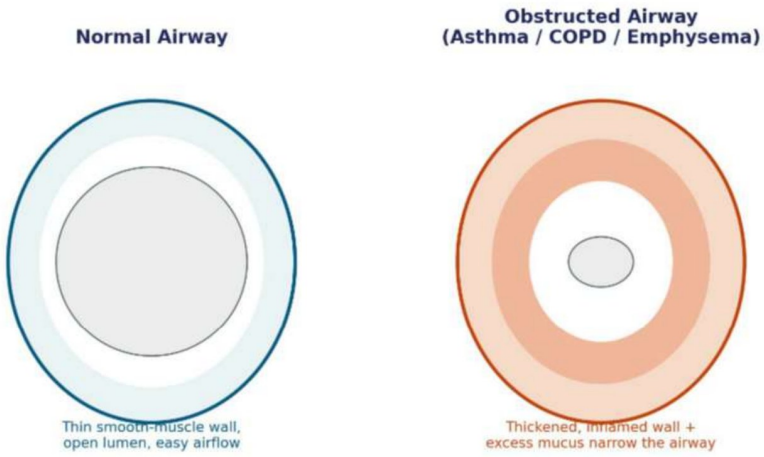
[Core] Normal and obstructed airways



Normal vs. inflamed/obstructed airway (asthma, COPD)

Compare lumen size and wall thickness. Smooth-muscle contraction, inflammation and mucus increase airflow resistance. Emphysema also involves alveolar destruction; this airway drawing does not show that mechanism.

S27 p.12 | Original labels retained; figure region enlarged.

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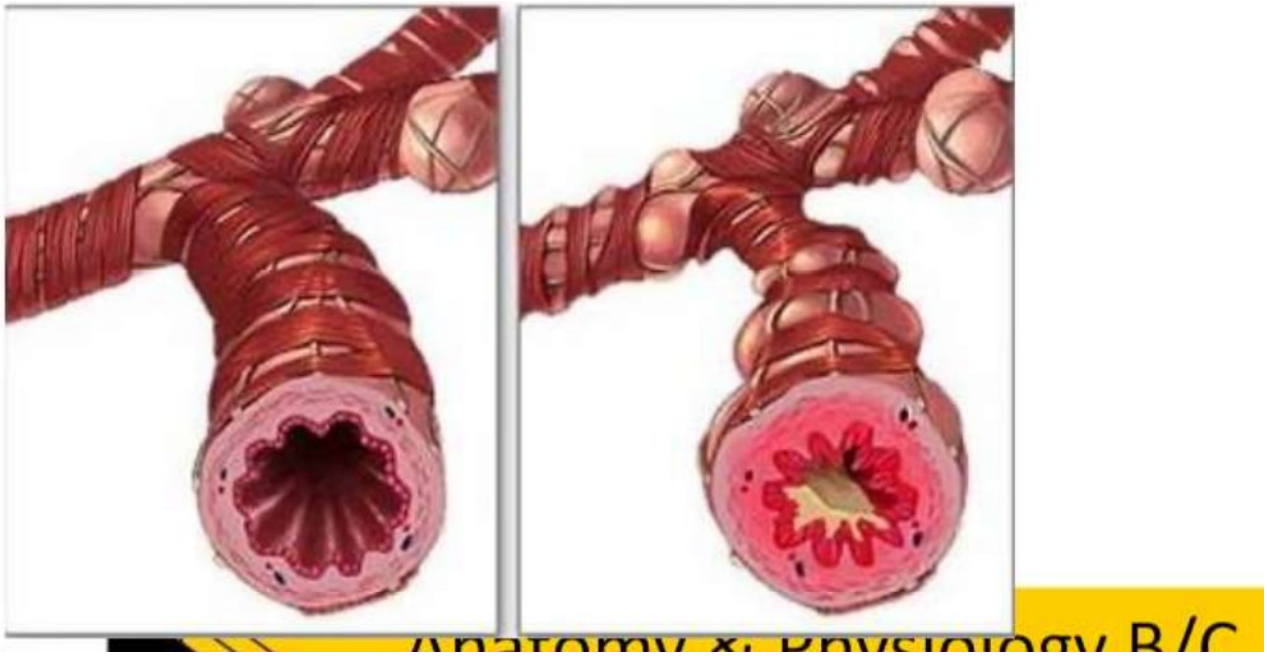
[Core] Normal versus asthmatic bronchiole



Look for airway smooth-muscle contraction, mucosal swelling and excess mucus. Each narrows the lumen and increases resistance, especially affecting expiration.

S23 p.70 | Original labels retained; figure region enlarged.

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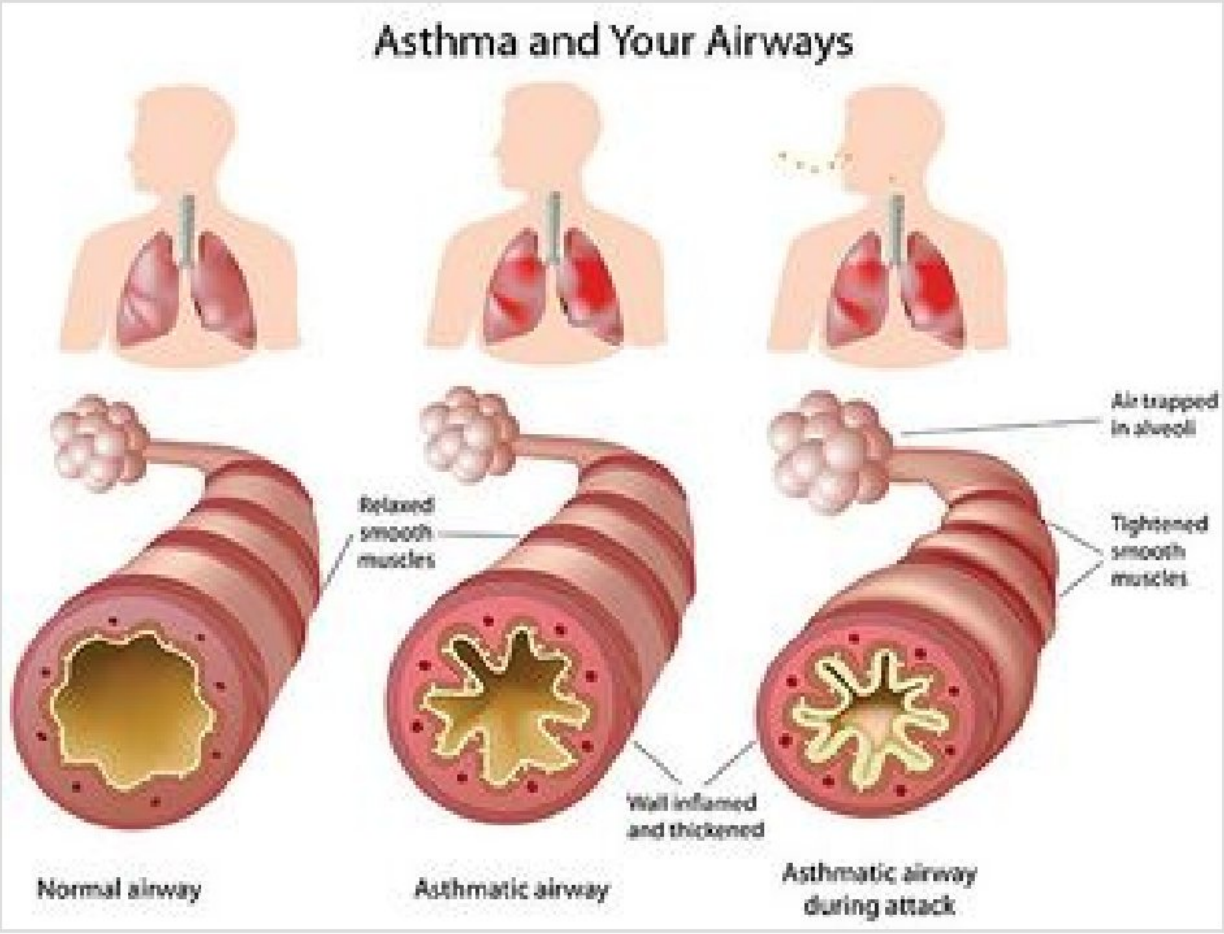
[Core] Asthma airway comparison



Compare healthy and narrowed airways. Connect inflammation, smooth muscle and mucus to airflow resistance rather than treating asthma as alveolar destruction.

WI p.7 | Original labels retained; figure region enlarged.

Emphysema

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Alveolar-wall destruction reduces surface area and elastic recoil, promoting air trapping.

Recognition clues: Hyperinflation, breathlessness; enlarged air spaces on tissue sections.

Distinguish: Unlike fibrosis, compliance can increase while recoil decreases.

[S] [N] Treatment: COPD care, rehabilitation; selected severe cases may receive lung-volume reduction or transplantation.

[S] [N] Prevention: Smoking avoidance/cessation and control of occupational exposures; inherited alpha-1 antitrypsin deficiency is a separate risk.

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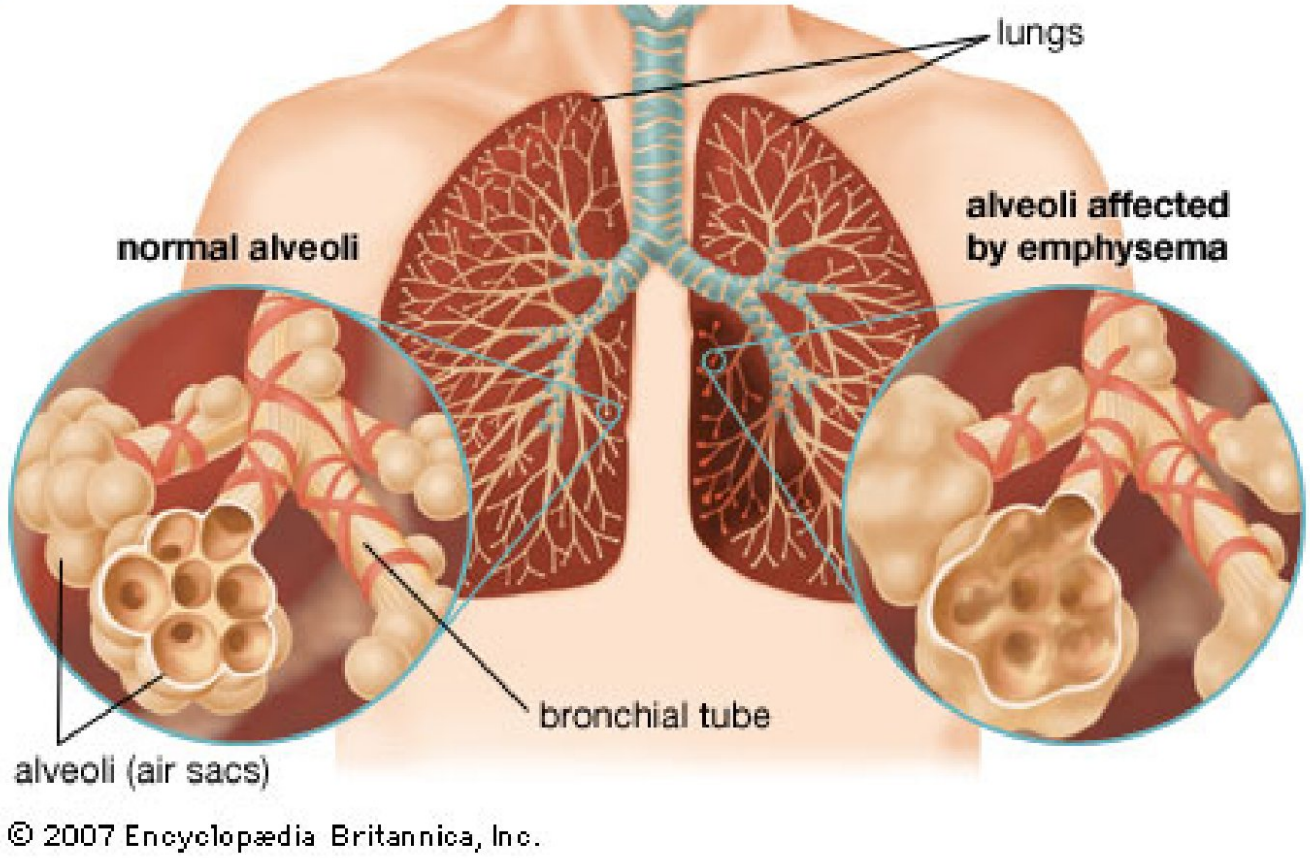
[Core] Normal and emphysematous alveoli



Compare many small air spaces with enlarged spaces and destroyed septa. Less surface area impairs exchange; loss of elastic recoil promotes air trapping.

WR p.7 | Original labels retained; figure region enlarged.

Pneumonia

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Infection produces inflammation and material within air spaces, reducing effective gas exchange.

Recognition clues: Cough, fever, breathlessness; alveolar consolidation.

Distinguish: Edema is fluid accumulation that can be noninfectious; pneumonia is infectious inflammation.

[S] [N] Treatment: Cause-directed antimicrobial therapy when indicated plus supportive care; antibiotics do not treat all viral causes.

[S] [N] Prevention: Appropriate vaccines, hand hygiene and reducing aspiration risk where applicable.

Sleep apnea

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Obstructive apnea involves upper-airway collapse; central apnea involves reduced respiratory drive.

Recognition clues: Repeated sleep-related breathing pauses, disrupted sleep and daytime sleepiness.

Distinguish: A sleep-associated pattern, not the same as persistent lower-airway obstruction.

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[S] [N] Treatment: Positive airway pressure for many obstructive cases; selected oral devices or surgery; central causes need tailored care.

[S] [N] Prevention: Address modifiable risks and avoid aggravating sedatives when clinically advised; not all cases are preventable.

Cystic fibrosis

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Inherited CFTR dysfunction disrupts salt/water movement, producing dehydrated airway secretions and often pancreatic-duct obstruction.

Recognition clues: Recurrent respiratory infections, thick mucus, poor fat absorption, elevated sweat chloride.

Distinguish: Connect respiratory obstruction with digestive enzyme deficiency; not an acquired infection.

[S] [N] Treatment: Airway clearance, infection treatment, pancreatic enzymes when needed and mutation-appropriate CFTR modulators.

[S] [N] Prevention: Inherited disease has no routine lifestyle prevention; screening and early care reduce complications.

Tuberculosis

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Mycobacterium tuberculosis infection can produce granulomatous inflammation; latent and active infection differ.

Recognition clues: Persistent cough and systemic symptoms in active disease; granulomas may be seen histologically.

Distinguish: Latent infection is not equivalent to symptomatic contagious pulmonary disease.

[S] [N] Treatment: Multiple appropriate antibiotics together, selected for susceptibility and clinical context.

[S] [N] Prevention: Airborne infection control, detection/treatment of infection and vaccination where indicated.

Pulmonary edema

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Fluid enters interstitium/alveoli, impairing oxygen transfer; cardiogenic and noncardiogenic causes exist.

Recognition clues: Breathlessness, crackles and impaired oxygenation; fluid in exchange spaces.

Distinguish: Fibrosis is scar tissue; edema is fluid.

[S] [N] Treatment: Treat the cause; oxygen/ventilatory support when needed; diuretics for appropriate fluid-overload cases.

[S] [N] Prevention: Manage contributing conditions; prevention depends on cause.

Pleurisy

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Inflammation of pleural surfaces causes painful friction with breathing.

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Recognition clues: Sharp pain worsened by inspiration or cough.

Distinguish: Pleural pain differs from loss of alveolar surface area.

[S] [N] Treatment: Treat the underlying cause and control inflammation/pain as appropriate.

[S] [N] Prevention: Prevent or manage contributing infections/conditions; no single preventive strategy fits every cause.

Small-cell lung cancer

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: An aggressive neuroendocrine lung cancer, strongly associated with smoking and often early spread.

Recognition clues: Lung mass with possible systemic/paraneoplastic effects.

Distinguish: Often treated systemically; do not assume surgery is the main treatment for all stages.

[S] [N] Treatment: Chemotherapy, radiotherapy and/or immunotherapy according to stage and clinical context.

[S] [N] Prevention: Avoid tobacco and carcinogenic exposures; screening eligibility is age/risk specific.

Non-small-cell lung cancer

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: A group including adenocarcinoma and squamous-cell carcinoma; risk factors and molecular features vary.

Recognition clues: Mass, cough or other variable symptoms; stage and tumor testing guide care.

Distinguish: A category, not a single uniform cancer; differs from small-cell biology.

[S] [N] Treatment: Surgery for selected localized tumors; radiation, chemotherapy, targeted therapy or immunotherapy as indicated.

[S] [N] Prevention: Tobacco avoidance, radon/exposure control and appropriate risk-based screening.

Pulmonary fibrosis

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Scarring thickens/stiffens lung tissue, reducing compliance and diffusion.

Recognition clues: Restrictive physiology, low TLC and exertional breathlessness.

Distinguish: Opposite compliance tendency from emphysema.

[S] [N] Treatment: Treat the cause; antifibrotic drugs for selected fibrotic diseases, oxygen/rehabilitation and sometimes transplant.

[S] [N] Prevention: Reduce harmful exposures where relevant; many forms cannot be reliably prevented.

Silicosis

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Inhaled crystalline silica causes inflammatory/fibrotic lung injury.

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Recognition clues: History of silica-dust exposure; increased susceptibility to tuberculosis.

Distinguish: Identify silica rather than coal dust or asbestos.

[S] [N] Treatment: Exposure cessation, supportive care and management of complications; established fibrosis is not simply reversed.

[S] [N] Prevention: Engineering controls, dust suppression and properly selected respiratory protection.

Coal workers' pneumoconiosis

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Coal-mine dust exposure can produce chronic lung disease and fibrosis.

Recognition clues: Occupational history is central; severe progressive massive fibrosis may occur.

Distinguish: Different exposure from silicosis, although mixed dust exposure occurs.

[S] [N] Treatment: Reduce further exposure, supportive respiratory care and treat complications.

[S] [N] Prevention: Dust monitoring, ventilation and exposure controls.

Asbestosis

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Inhaled asbestos fibers cause interstitial fibrosis.

Recognition clues: Occupational exposure; asbestos/ferruginous bodies can be a tissue clue.

Distinguish: Asbestos exposure also raises mesothelioma and lung-cancer risk; these are not synonyms for asbestosis.

[S] [N] Treatment: Supportive care and prevention of further exposure; cancer evaluation when indicated.

[S] [N] Prevention: Safe asbestos management by qualified personnel and workplace controls.

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DIGESTIVE

D-ANAT | Food pathway, organs and functions

[Core] | Rule 3b.i,ii

Food travels mouth -> pharynx -> esophagus -> stomach -> duodenum -> jejunum -> ileum -> cecum -> ascending, transverse, descending and sigmoid colon -> rectum -> anus. The appendix arises from the cecum. Ingestion brings food in; propulsion moves it; digestion breaks it down; absorption transfers nutrients across epithelium; defecation removes undigested residue. Digestion is mostly extracellular in the tract lumen.

Teeth perform mechanical breakdown: incisors cut, canines tear, premolars and molars crush/grind. Tongue positions food and forms a bolus. Parotid glands lie near the ears; submandibular and sublingual glands lie below the jaw and tongue. Saliva lubricates and begins starch digestion. Swallowing coordinates airway closure and esophageal propulsion.

The stomach stores and churns food into chyme; identify cardia, fundus, body and pylorus. The pyloric sphincter regulates delivery to the duodenum. The small intestine performs most nutrient absorption and much digestion; the large intestine recovers remaining water/electrolytes, supports microbial fermentation and stores feces. Liver, gallbladder and pancreas are accessory organs: secretions enter the tract but food does not pass through them. Most water absorption occurs in the small intestine, even though water recovery is a major colon function.

Connect the GI and immune systems: Peyer patches are aggregated lymphoid nodules, especially prominent in the ileum; they sample threats near the gut lining. They are not separate lymph nodes. The uvula is an extension of the soft palate, not a substitute name for a tonsil.

Recall: Which structures receive food: pancreas, duodenum, gallbladder or jejunum?

Duodenum and jejunum. The pancreas and gallbladder deliver secretions through ducts.

Common trap: The colon is not the site of most total water absorption.

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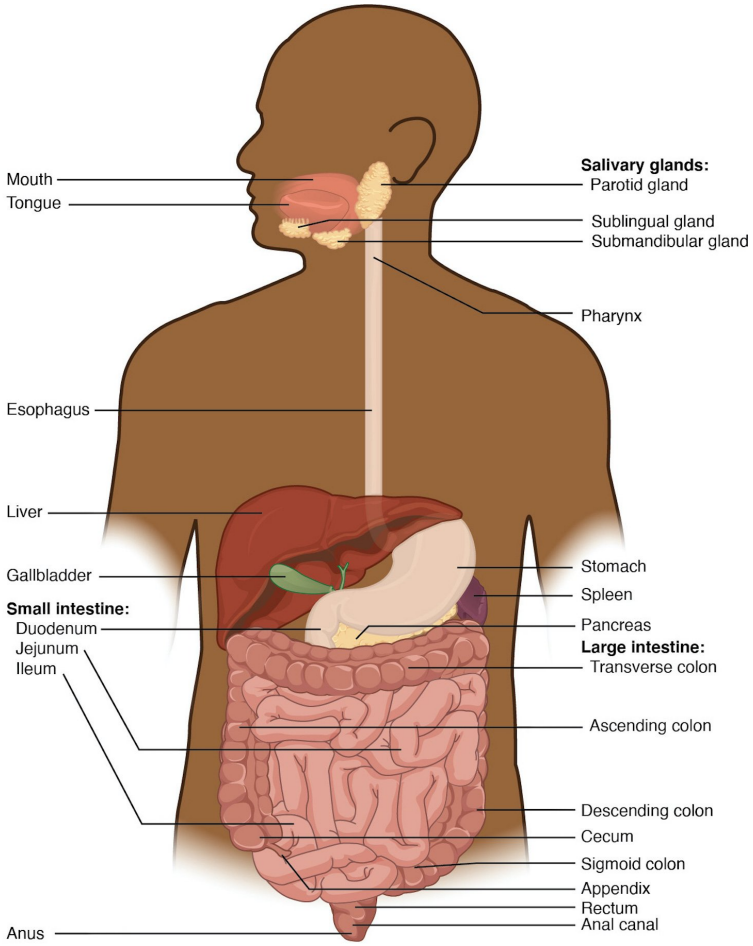
[Core] Components of the Digestive System



Use this large labeled overview to orient the canal and accessory organs before learning their individual structures.

OpenStax A&P 2e, Fig. 23.2 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-1-overview-of-the-digestive-system | Publisher original; 1500 x 1864 pixels

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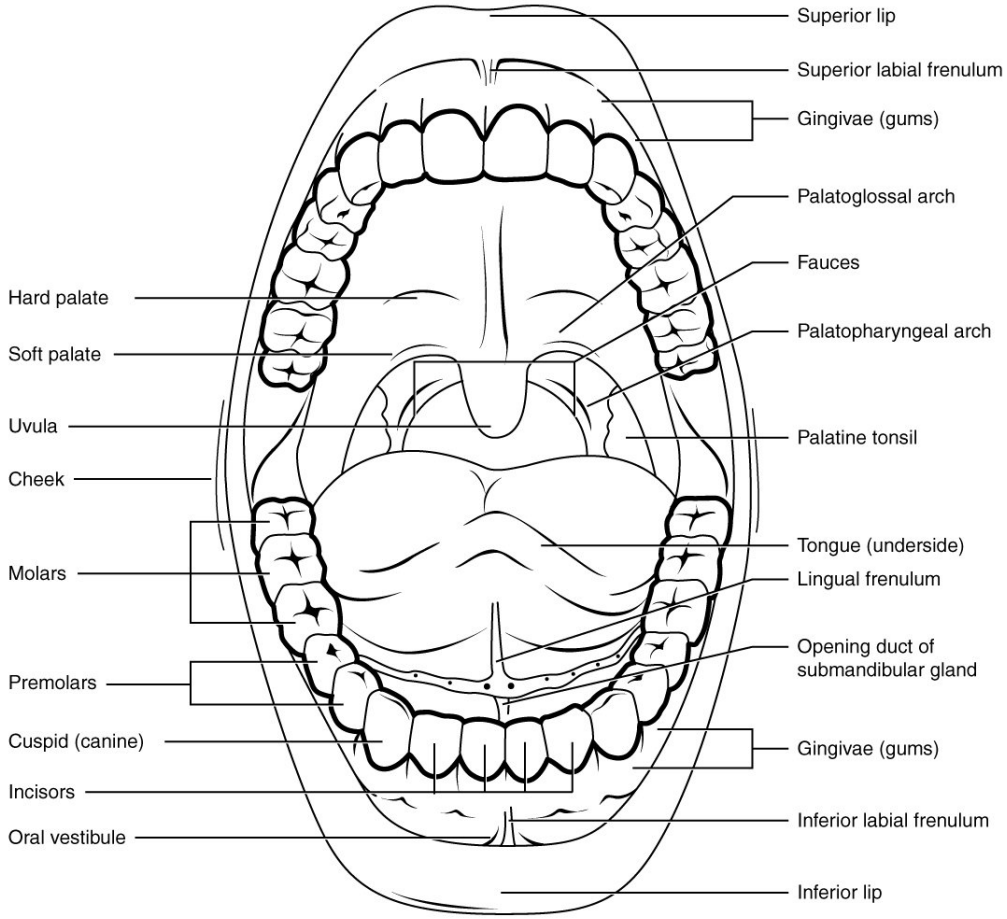
[Core] Mouth



Identify tongue, palate and oral opening. Connect position to chewing, bolus formation and swallowing.

OpenStax A&P 2e, Fig. 23.7 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-3-the-mouth-pharynx-and-esophagus | Publisher original; 1075 x 1028 pixels

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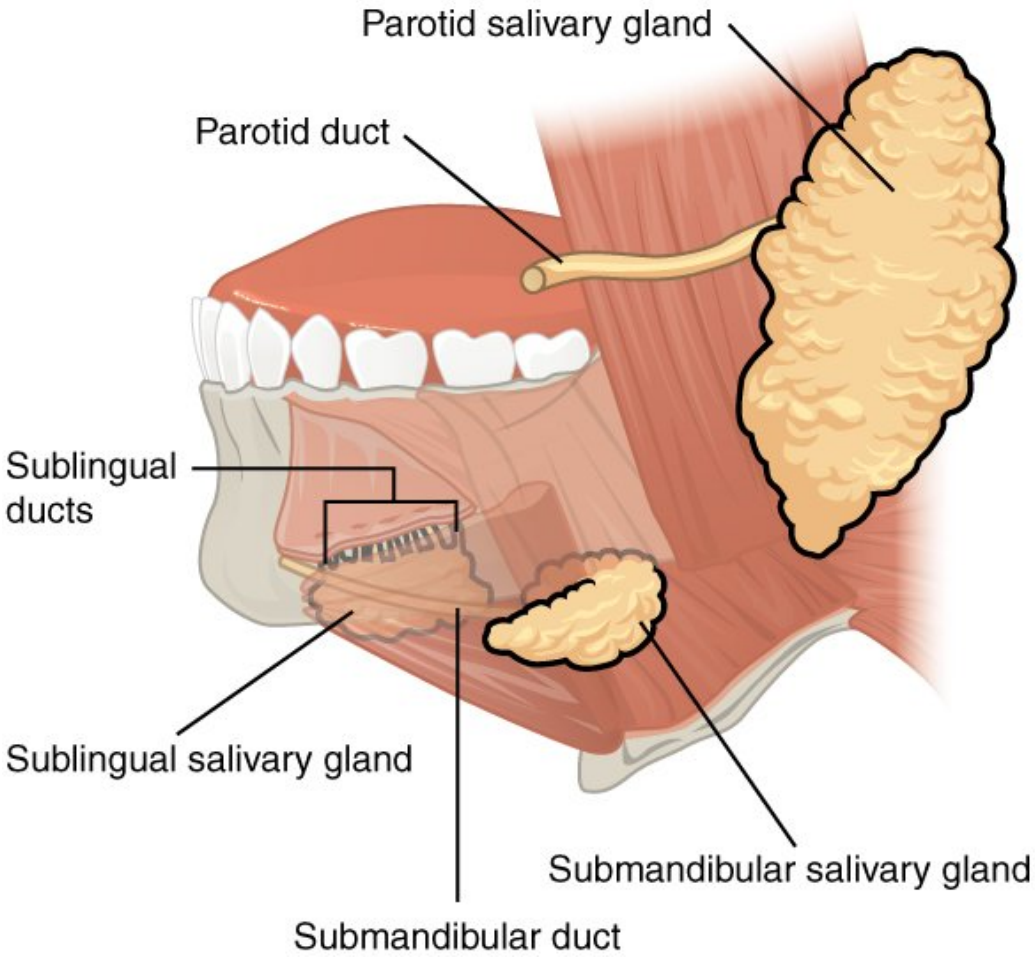
[Core] Salivary glands



Identify the three paired major salivary glands and their ducts. Ducts carry secretion to the oral cavity.

OpenStax A&P 2e, Fig. 23.9 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-3-the-mouth-pharynx-and-esophagus | Publisher original; 586 x 542 pixels

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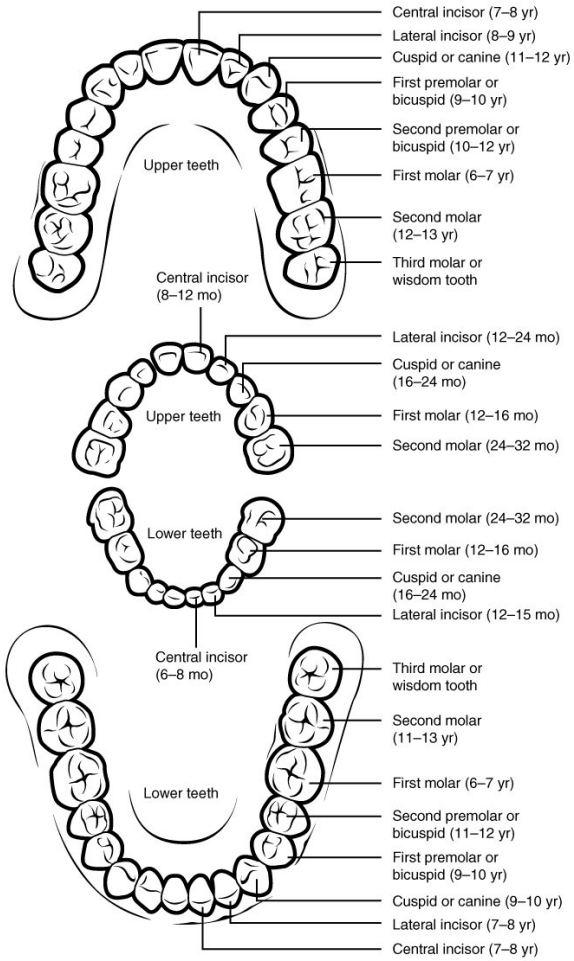
[Core] Permanent and Deciduous Teeth



Compare adult and deciduous tooth patterns. The image is an anatomy aid; detailed dental numbering is enrichment.

OpenStax A&P 2e, Fig. 23.10 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-3-the-mouth-pharynx-and-esophagus | Publisher original; 660 x 1110 pixels

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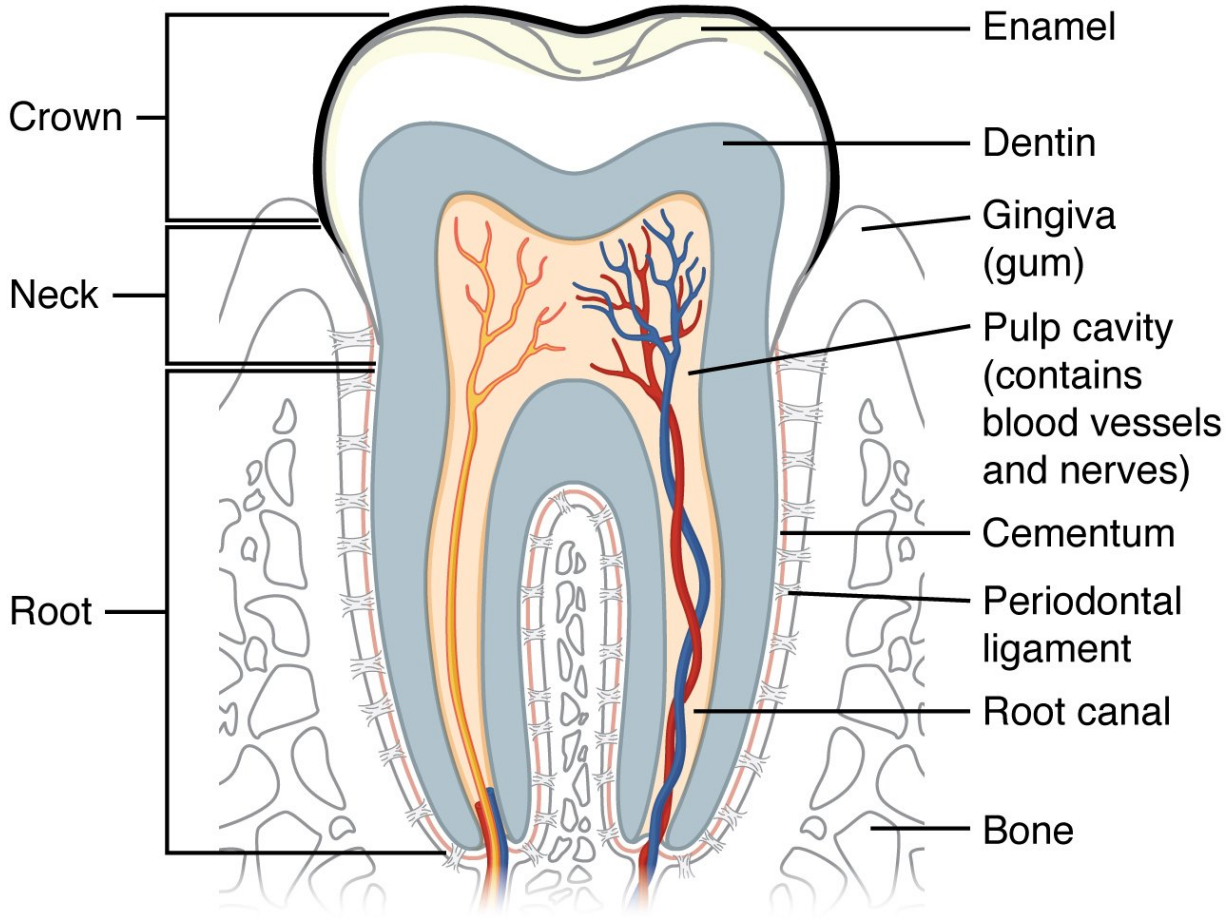
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] The Structure of the Tooth



Locate enamel, dentin, pulp, root and supporting tissue. Contrast hard protective layers with living vascular pulp.

OpenStax A&P 2e, Fig. 23.11 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-3-the-mouth-pharynx-and-esophagus | Publisher original; 1281 x 1010 pixels

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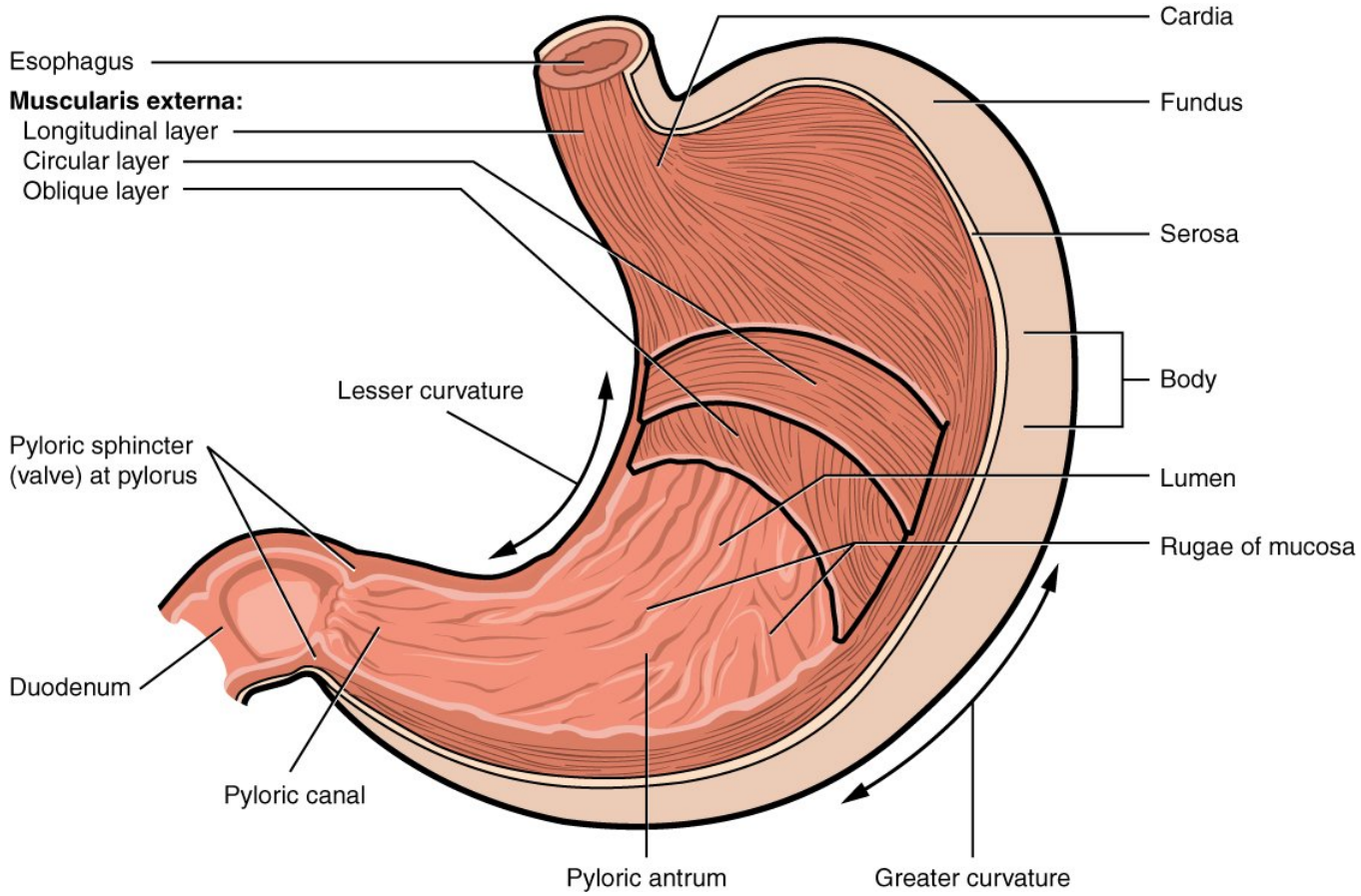
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Stomach



Identify stomach inlet, regions, muscle layers and pyloric outlet. Rugae are folds, not villi.

OpenStax A&P 2e, Fig. 23.15 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-4-the-stomach | Publisher original; 1122 x 741 pixels

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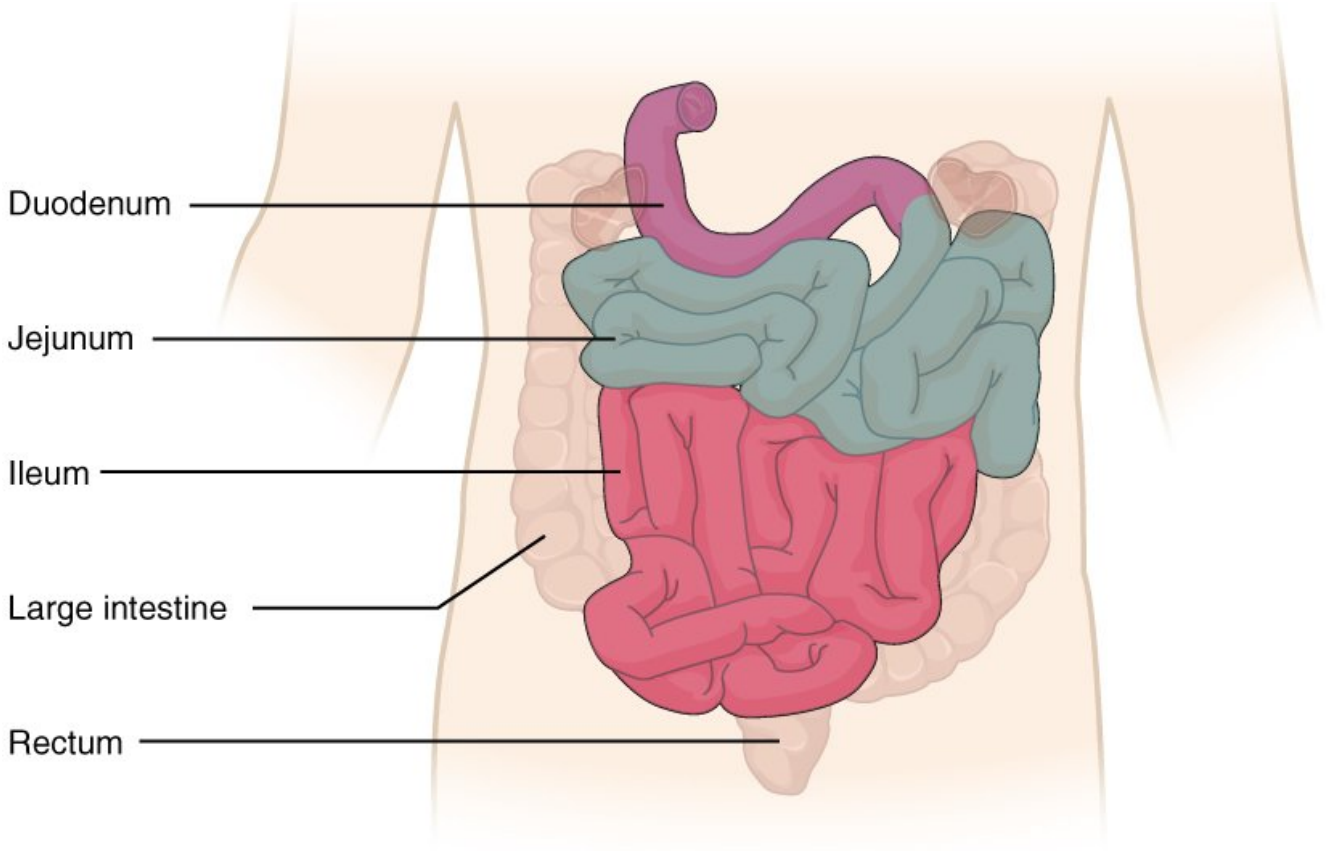
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Small Intestine



Trace duodenum, jejunum and ileum from stomach to cecum.

OpenStax A&P 2e, Fig. 23.18 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-5-the-small-and-large-intestines | Publisher original; 839 x 552 pixels

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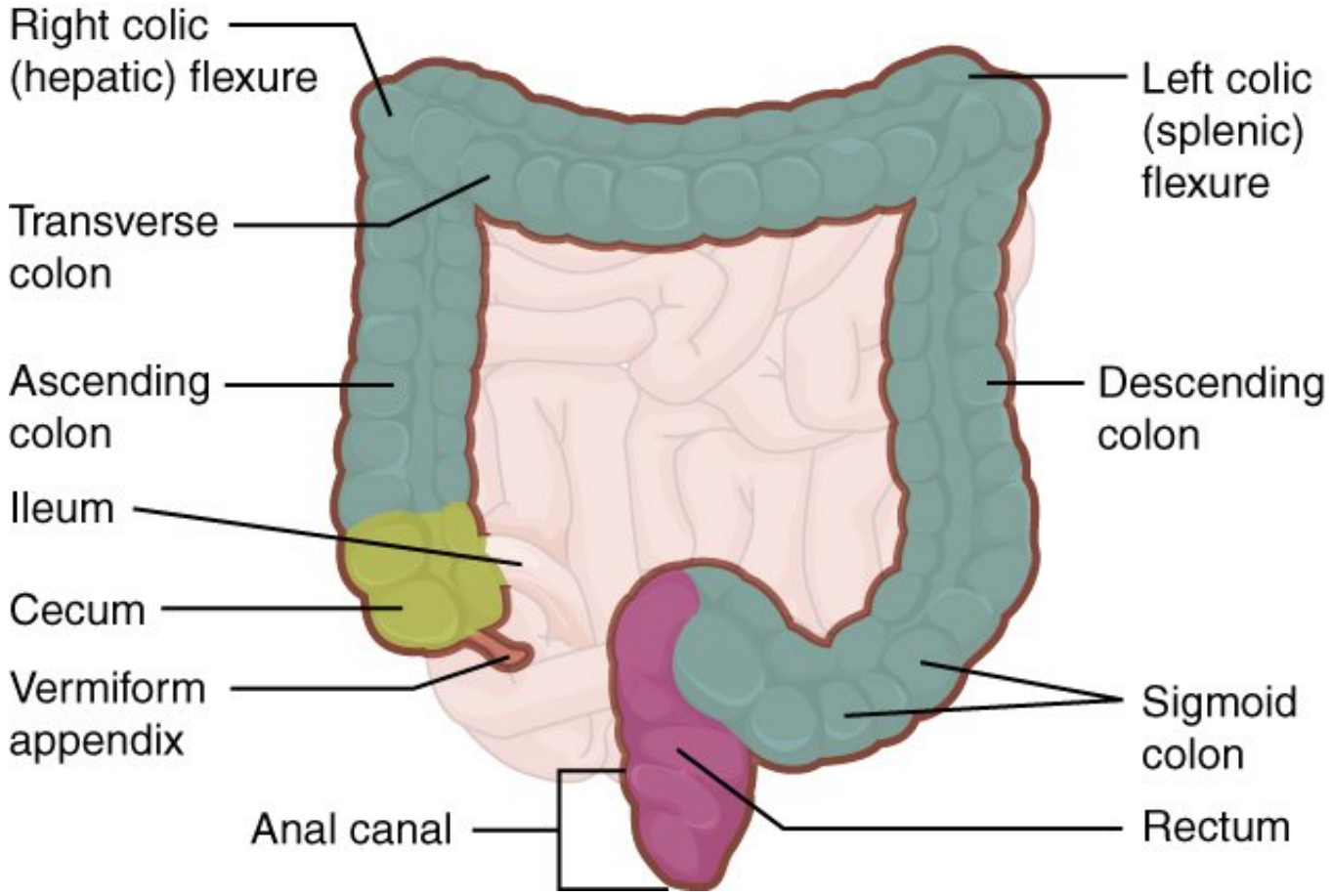
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Large Intestine



Follow the colon around the small intestine and identify rectum, cecum and appendix.

OpenStax A&P 2e, Fig. 23.21 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-5-the-small-and-large-intestines | Publisher original; 633 x 427 pixels

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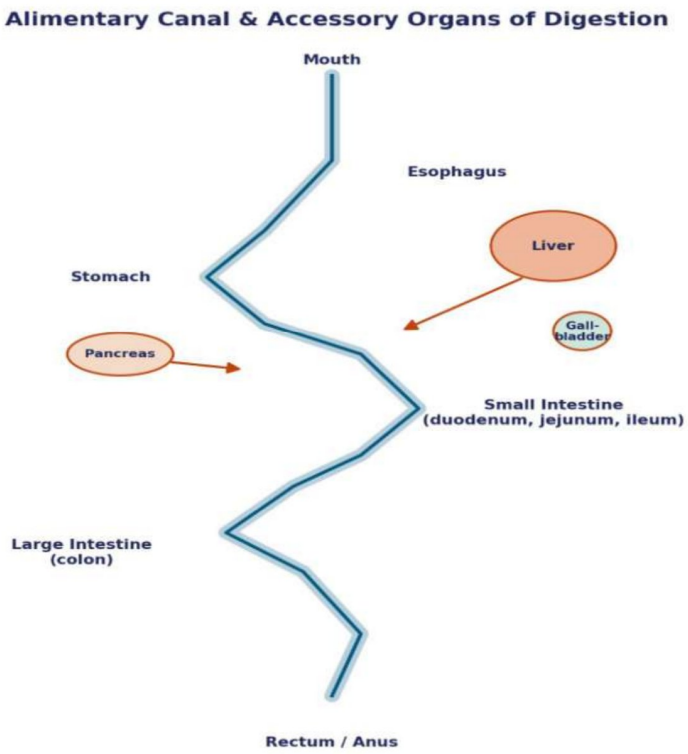
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Alimentary canal and accessory organs



The ~9-meter alimentary canal, plus accessory organs

Trace food along the continuous canal. Liver, gallbladder and pancreas deliver secretions through ducts; food does not pass through these accessory organs.

S27 p.17 | Original labels retained; figure region enlarged.

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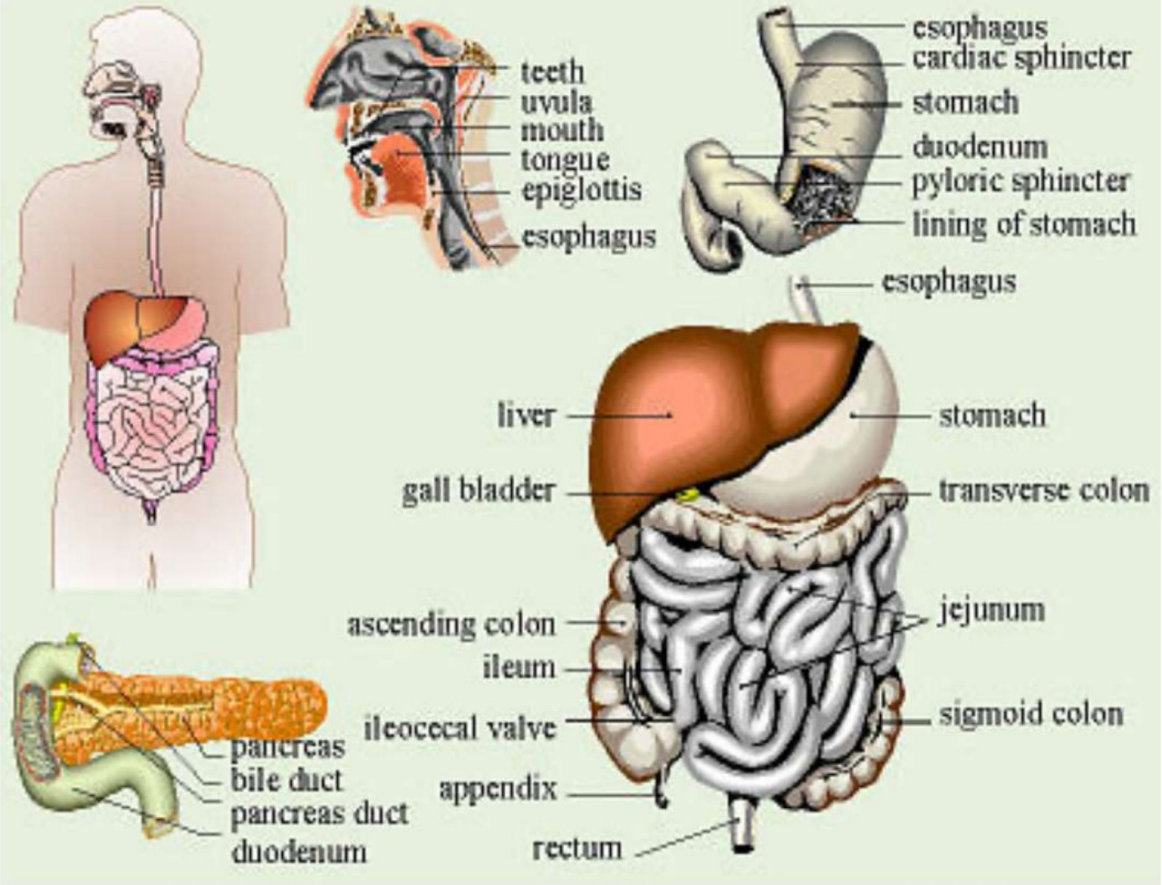
[Core] Digestive organs and accessory glands



Follow the canal from mouth to anus, then identify salivary glands, liver, gallbladder and pancreas. Separate the route of food from the routes of secretions.

S23 p.22 | Original labels retained; figure region enlarged.

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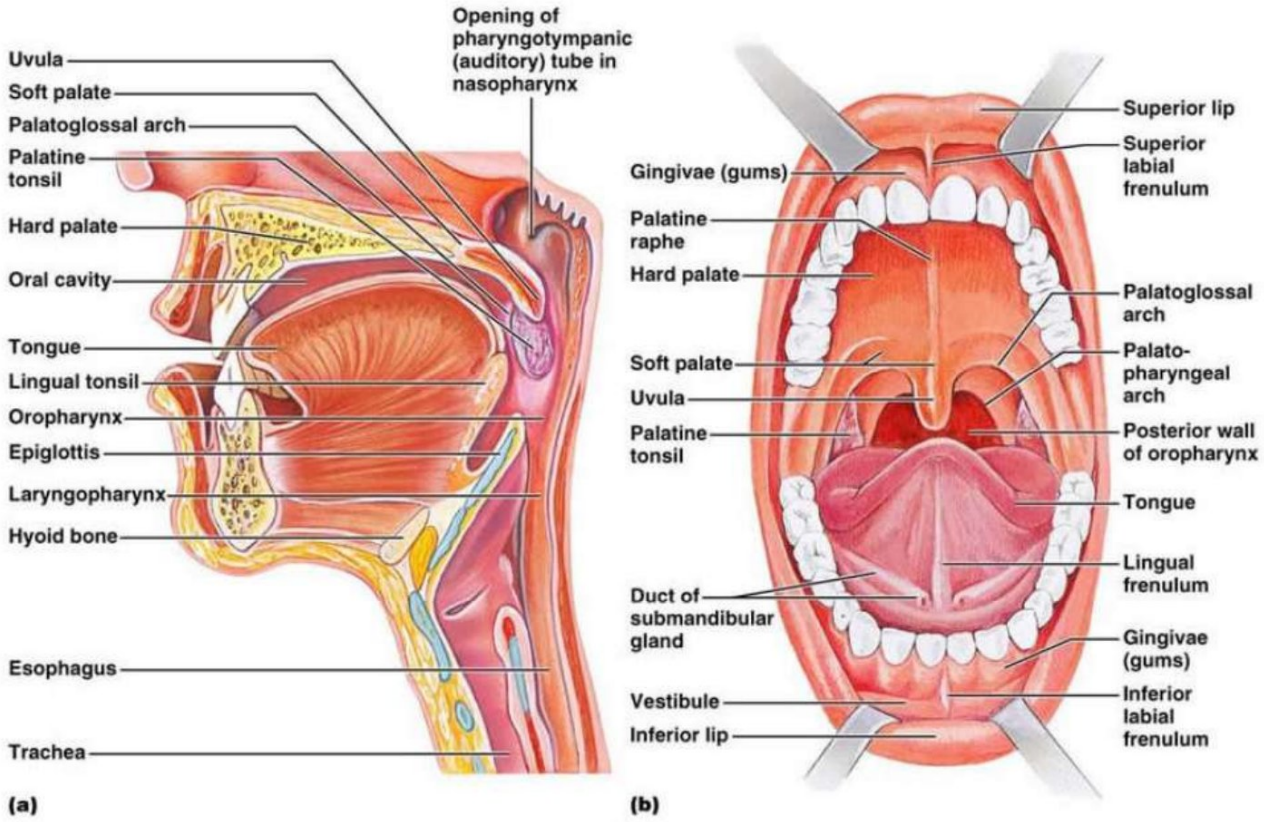
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Oral cavity and salivary structures



Find tongue, hard and soft palates and salivary structures. Teeth and tongue mechanically process food; saliva lubricates and begins starch digestion.

S23 p.26 | Original labels retained; figure region enlarged.

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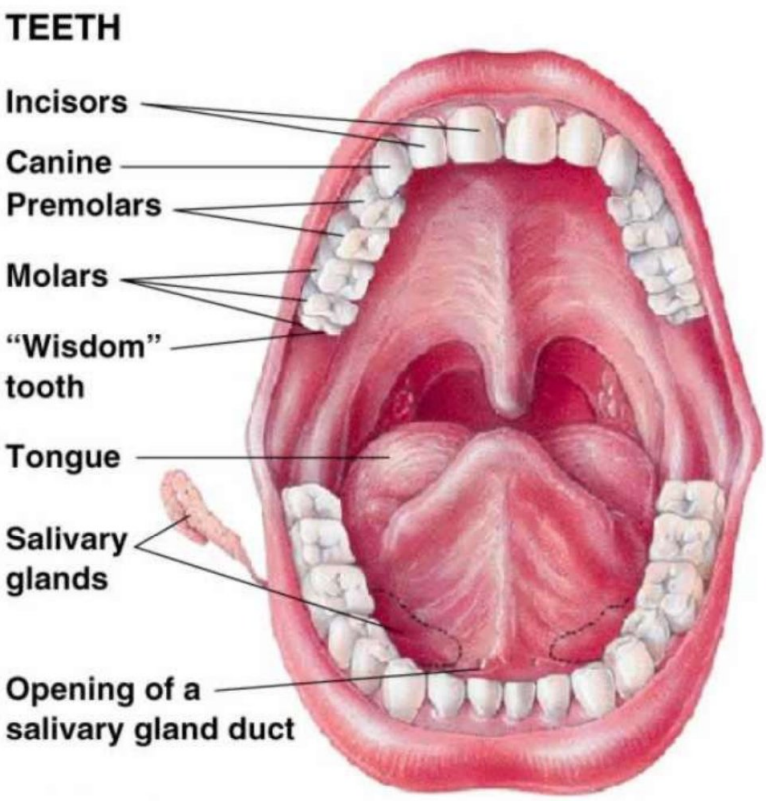
[Core] Teeth and palate



Identify incisors, canines, premolars and molars and relate shape to cutting, tearing and grinding. Teeth perform mechanical digestion; they do not hydrolyze nutrients.

S23 p.27 | Original labels retained; figure region enlarged.

Access for free at https://openstax.org/books/anatomy-and-physiology-2e/pages/1-introduction
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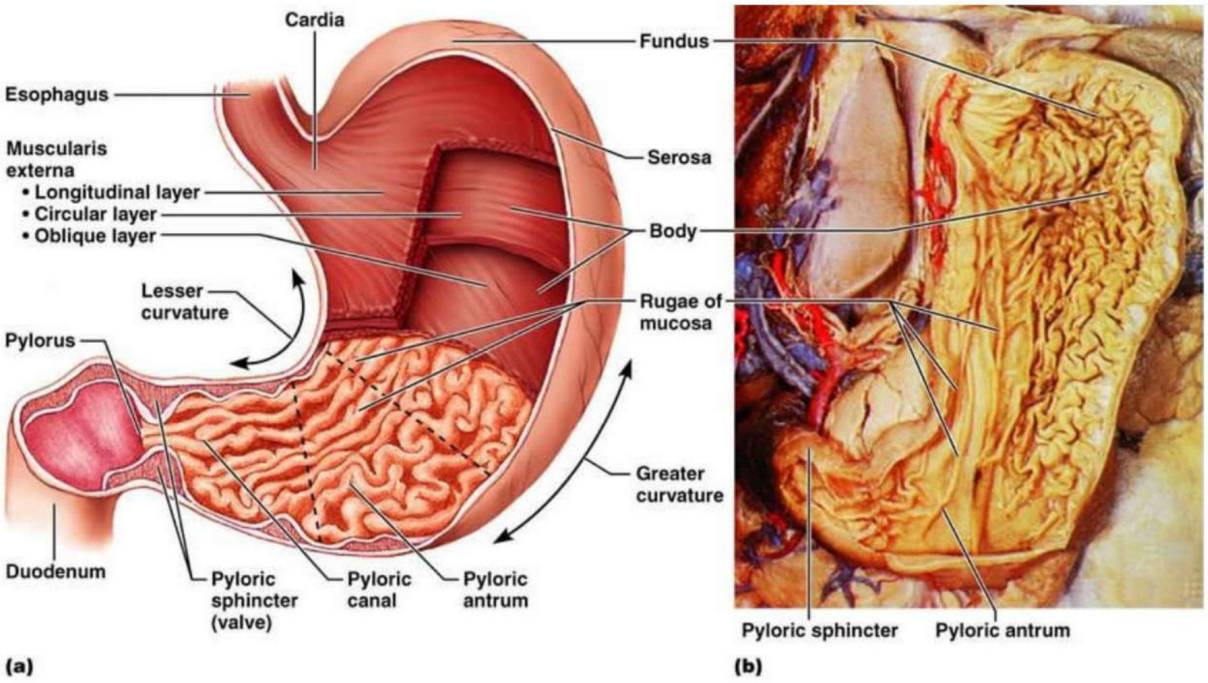
[Core] Stomach regions and internal folds



Identify cardia, fundus, body, pylorus and rugae. Rugae allow expansion; they are not small-intestinal villi. The pyloric sphincter regulates passage to the duodenum.

S23 p.29 | Original labels retained; figure region enlarged.

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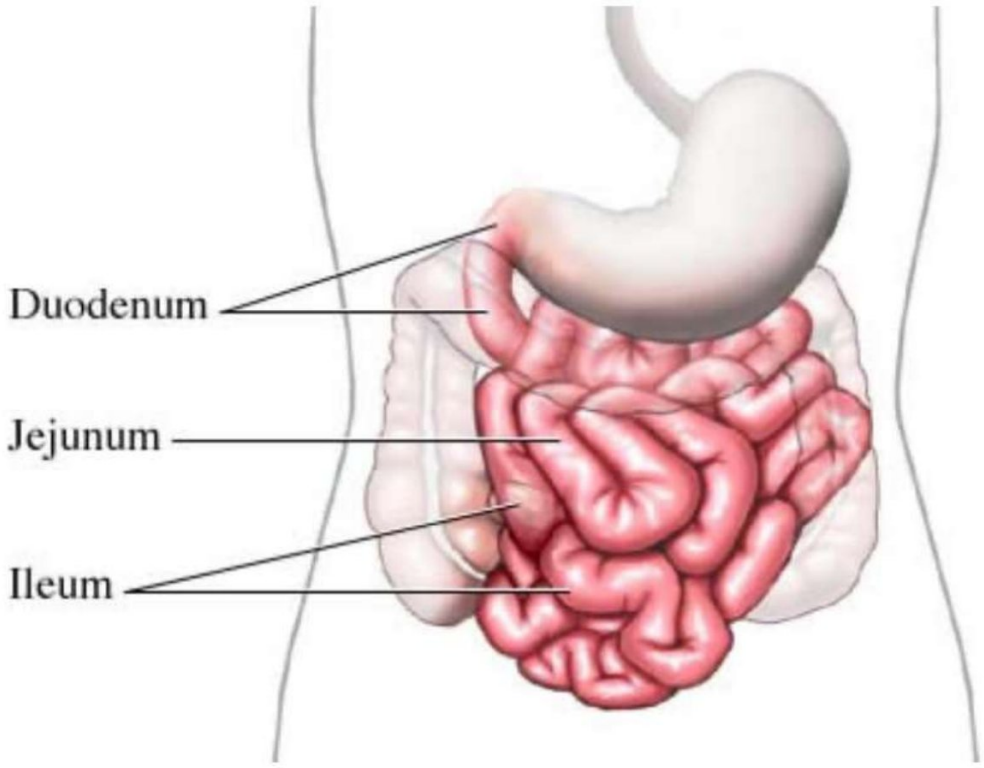
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Duodenum, jejunum and ileum



Trace the three small-intestinal regions in order. Digestion and absorption occur throughout, with regional specializations; do not equate all absorption exclusively with one region.

S23 p.31 | Original labels retained; figure region enlarged.

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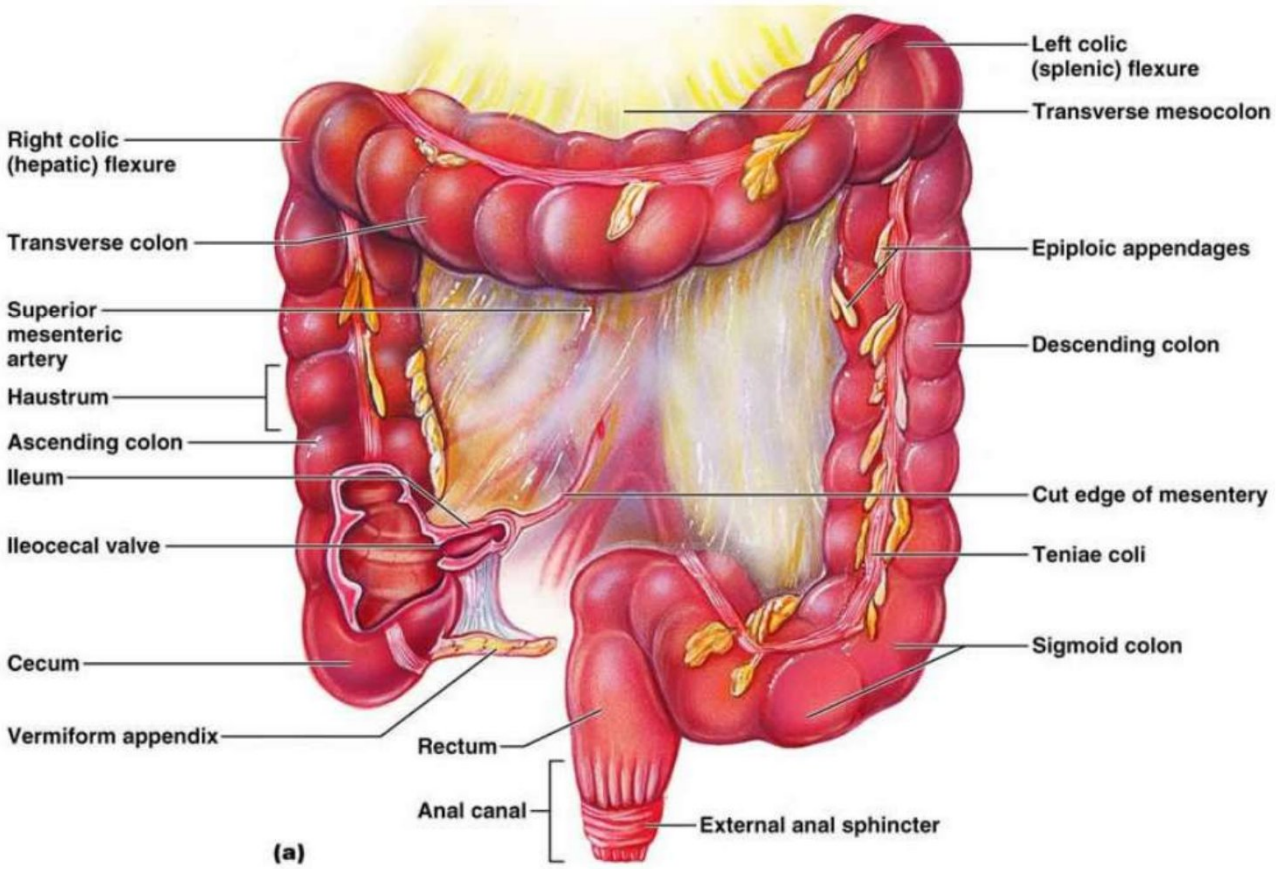
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Large intestine



Identify cecum, appendix, ascending/transverse/descending/sigmoid colon and rectum. Distinguish haustra from the fingerlike villi of the small intestine.

S23 p.34 | Original labels retained; figure region enlarged.

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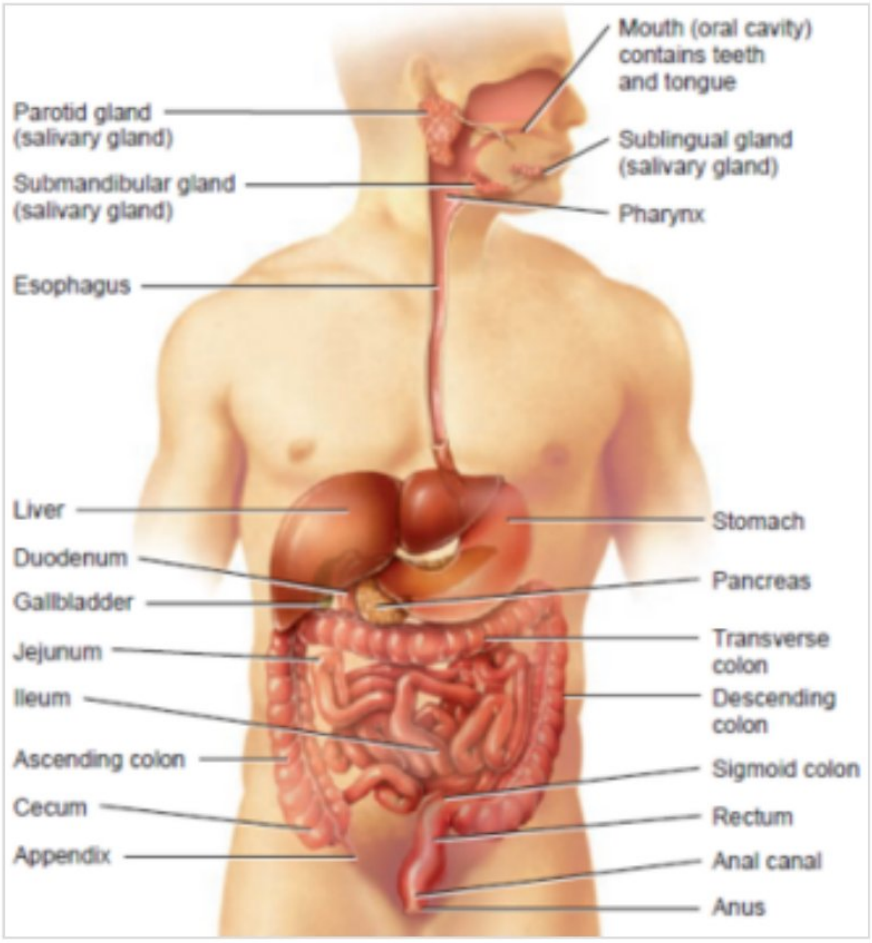
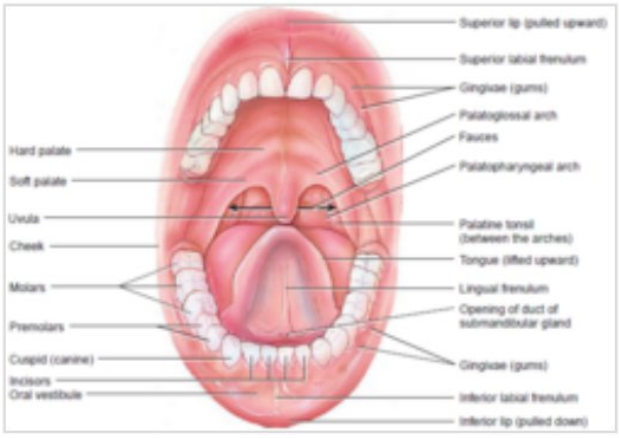
[Core] Digestive tract orientation

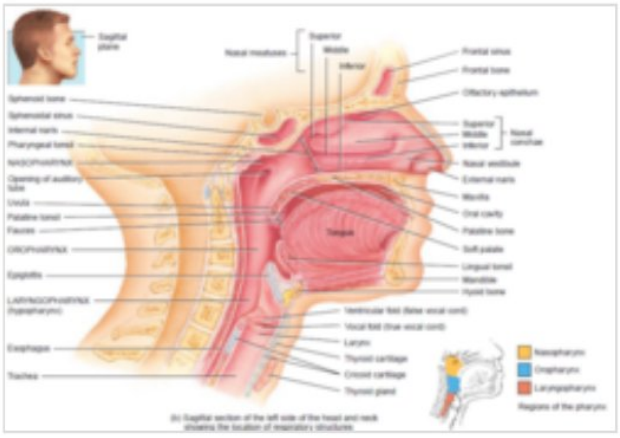


Trace oral cavity, esophagus, stomach, small intestine and colon. Identify the accessory organs without placing them in the food pathway.

WD p.1 | Original labels retained; figure region enlarged.

[Core] Oral cavity: front and side views





Compare frontal and sagittal orientations. Find tongue, palate and the transition to the pharynx; do not confuse the oral passage with the nasal cavity.

WD p.2 | Original labels retained; figure region enlarged.

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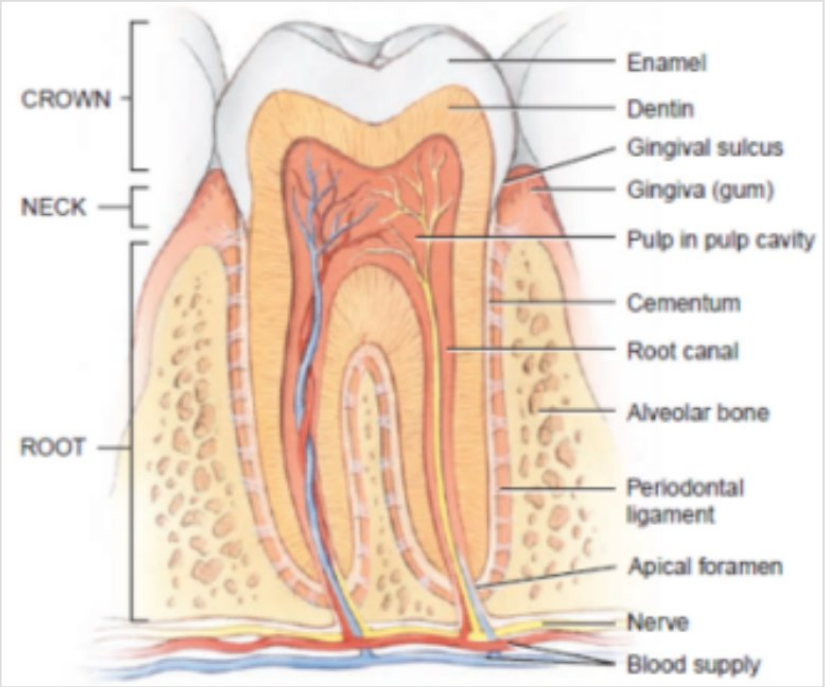
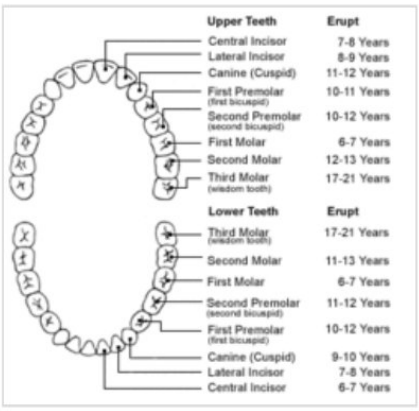
[Core] Tooth cross-section and dental arch





A labeled diagram of all teeth

Identify enamel, dentin and pulp, then compare crown, neck and root. The dental arch illustrates relative tooth positions.

WD p.3 | Original labels retained; figure region enlarged.

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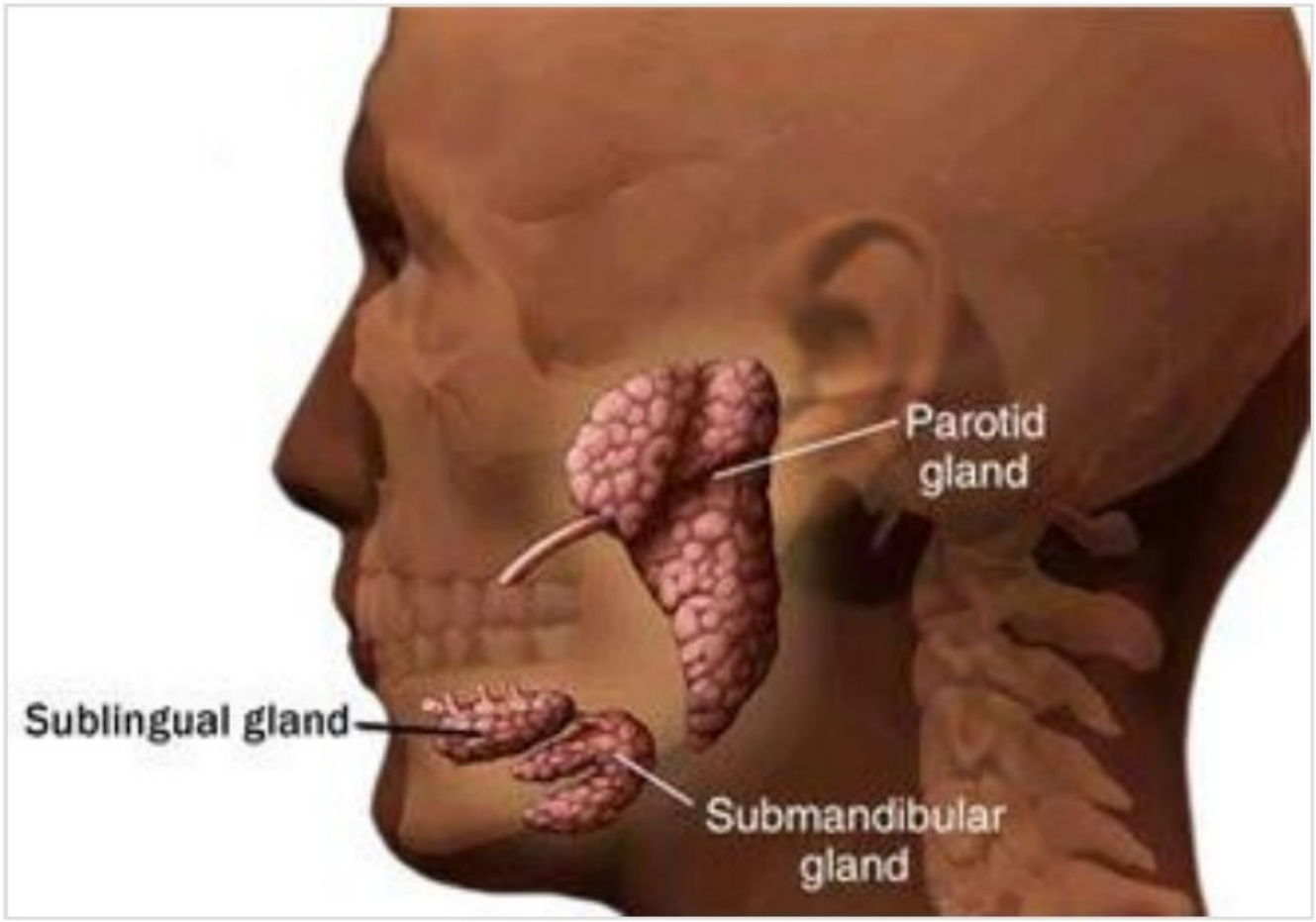
[Core] Major salivary glands



Locate parotid, submandibular and sublingual glands. Salivary ducts deliver lubricating fluid and enzymes to the mouth.

WD p.3 | Original labels retained; figure region enlarged.

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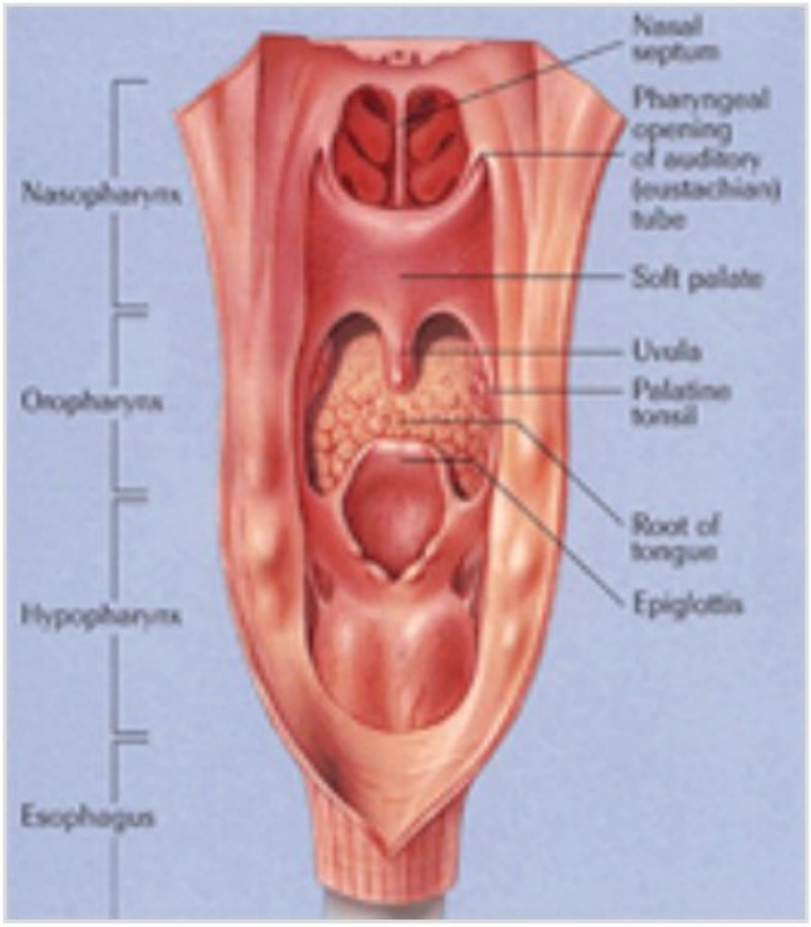
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Pharyngeal passage



Locate the shared throat passage and relate it to swallowing and airway protection. The pharynx is not the same structure as the larynx.

WD p.4 | Original labels retained; figure region enlarged.

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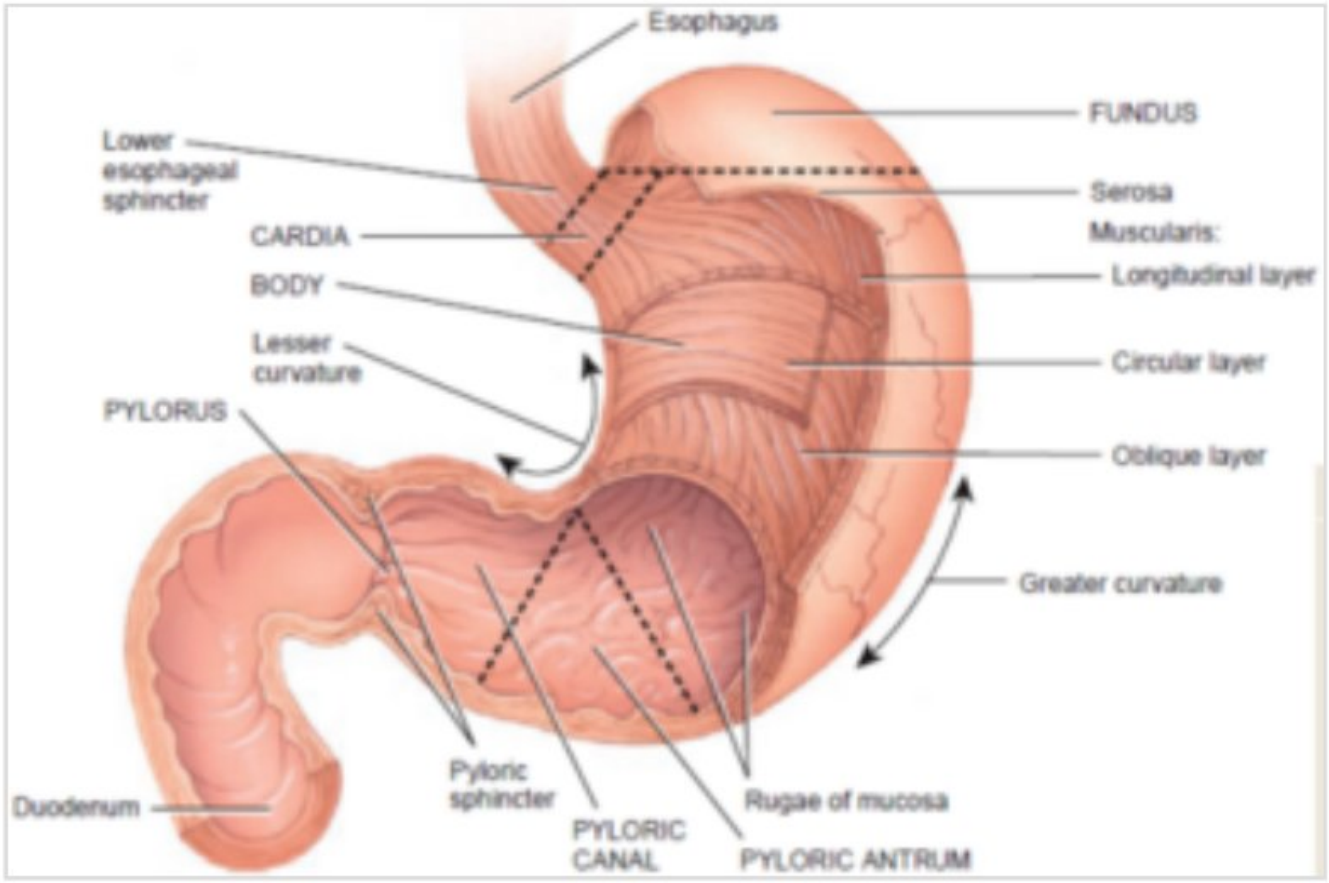
[Core] Stomach regions



Identify inlet, fundus, body and pyloric outlet. Distinguish the stomach lumen from the folds and muscle layers.

WD p.4 | Original labels retained; figure region enlarged.

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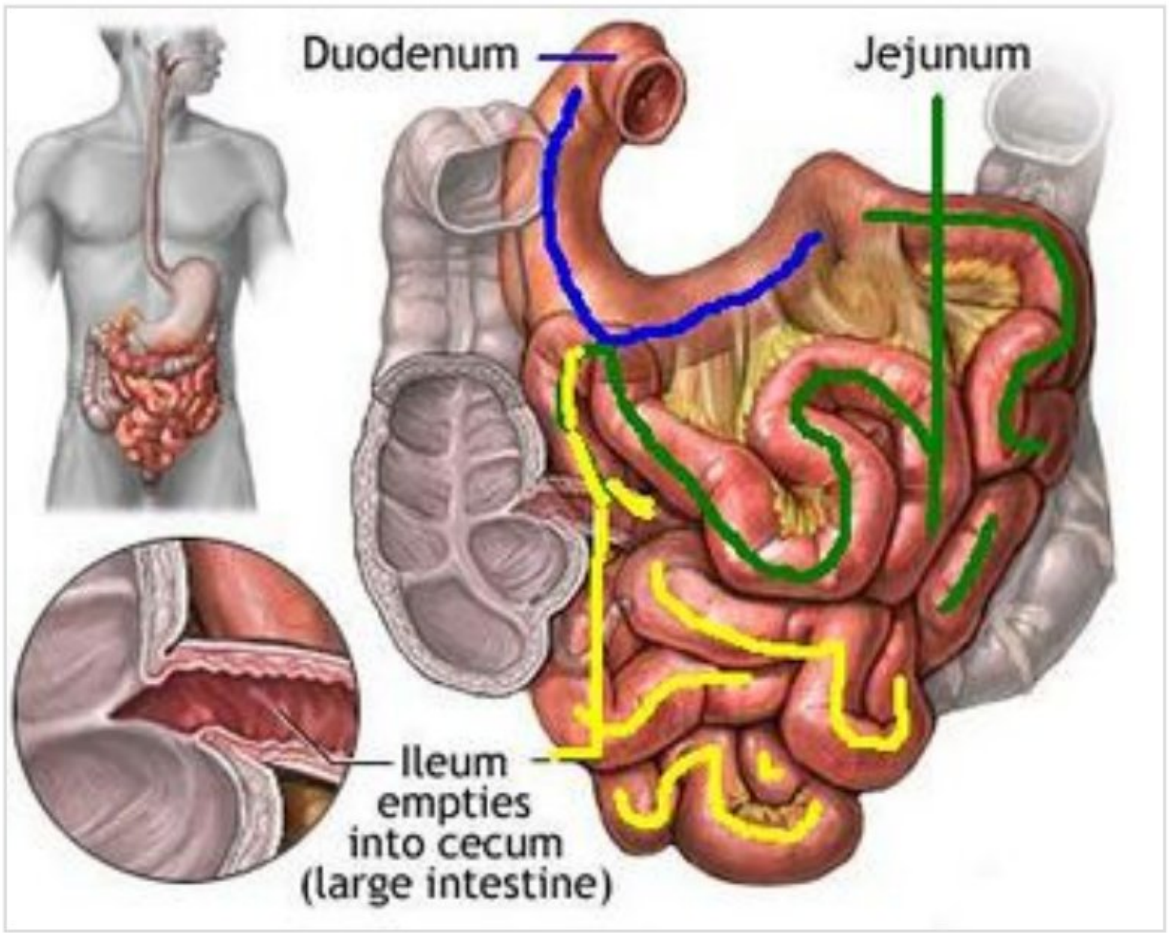
[Core] Small-intestinal regions



Follow duodenum, jejunum and ileum. The colon frames many small-intestinal loops in the abdominal view.

WD p.4 | Original labels retained; figure region enlarged.

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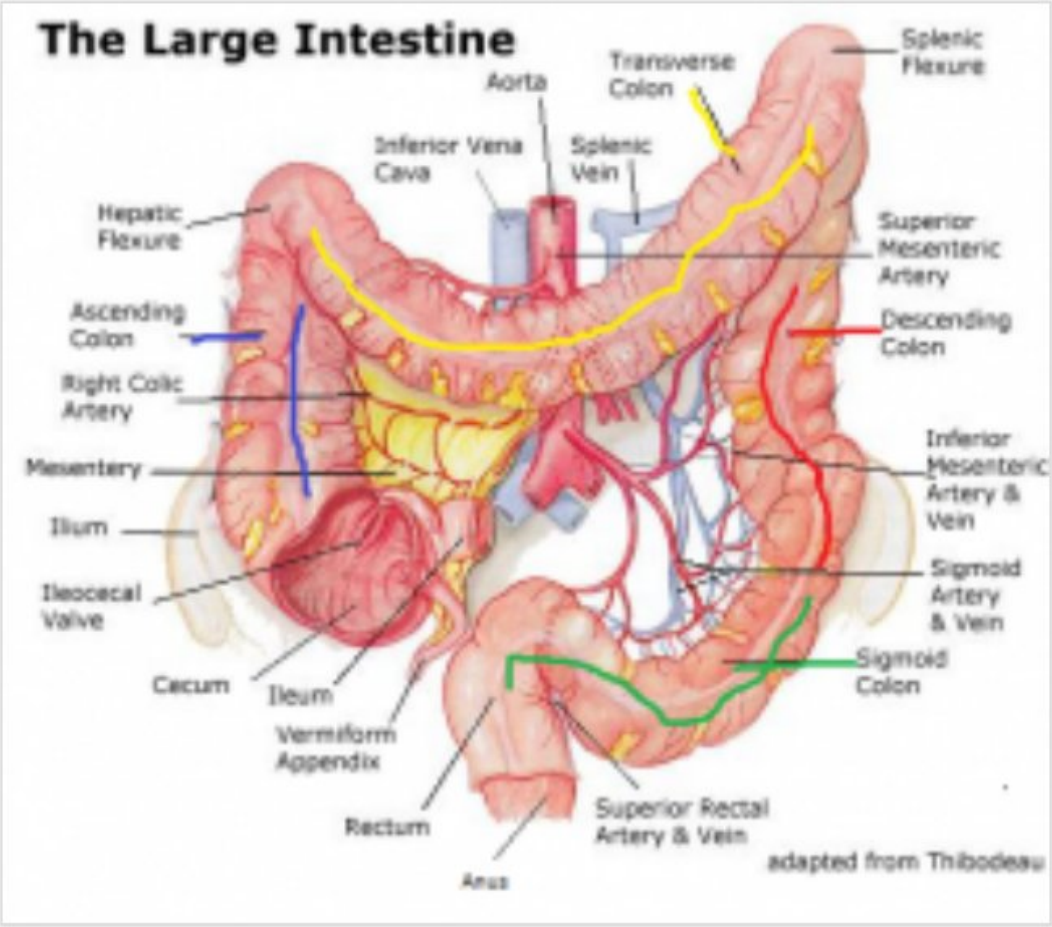
[Core] Colon and rectum



Trace cecum to ascending, transverse, descending and sigmoid colon, then rectum and anal canal.

WD p.6 | Original labels retained; figure region enlarged.

D-WALL | Four gut-wall layers and enteric nerves

[Core] | Rule 3b.iii

From lumen outward: mucosa, submucosa, muscularis externa, serosa/adventitia. Mucosa itself contains epithelium, lamina propria (connective tissue with vessels and immune cells) and muscularis mucosae (thin smooth muscle). It is the interface for secretion, protection and absorption.

Submucosa contains larger blood/lymph vessels and the submucosal or Meissner plexus, which helps regulate secretion and local blood flow. Muscularis externa usually has inner circular and outer longitudinal muscle; the myenteric or Auerbach plexus lies between them and coordinates motility. The stomach adds an oblique muscle layer. Esophageal muscle transitions from skeletal toward smooth down its length.

Serosa consists of connective tissue covered by mesothelium where an organ is covered by visceral peritoneum. Adventitia is connective tissue that anchors certain regions to neighboring structures and lacks that mesothelial covering. On a section, first locate lumen and epithelium, then the thin muscularis mucosae, vessel-rich submucosa and thick muscularis externa. Do not identify all pink tissue as the same muscle layer.

Recall: Where is the myenteric plexus relative to the circular and longitudinal muscle?

Between them in muscularis externa; it chiefly coordinates gut movement.

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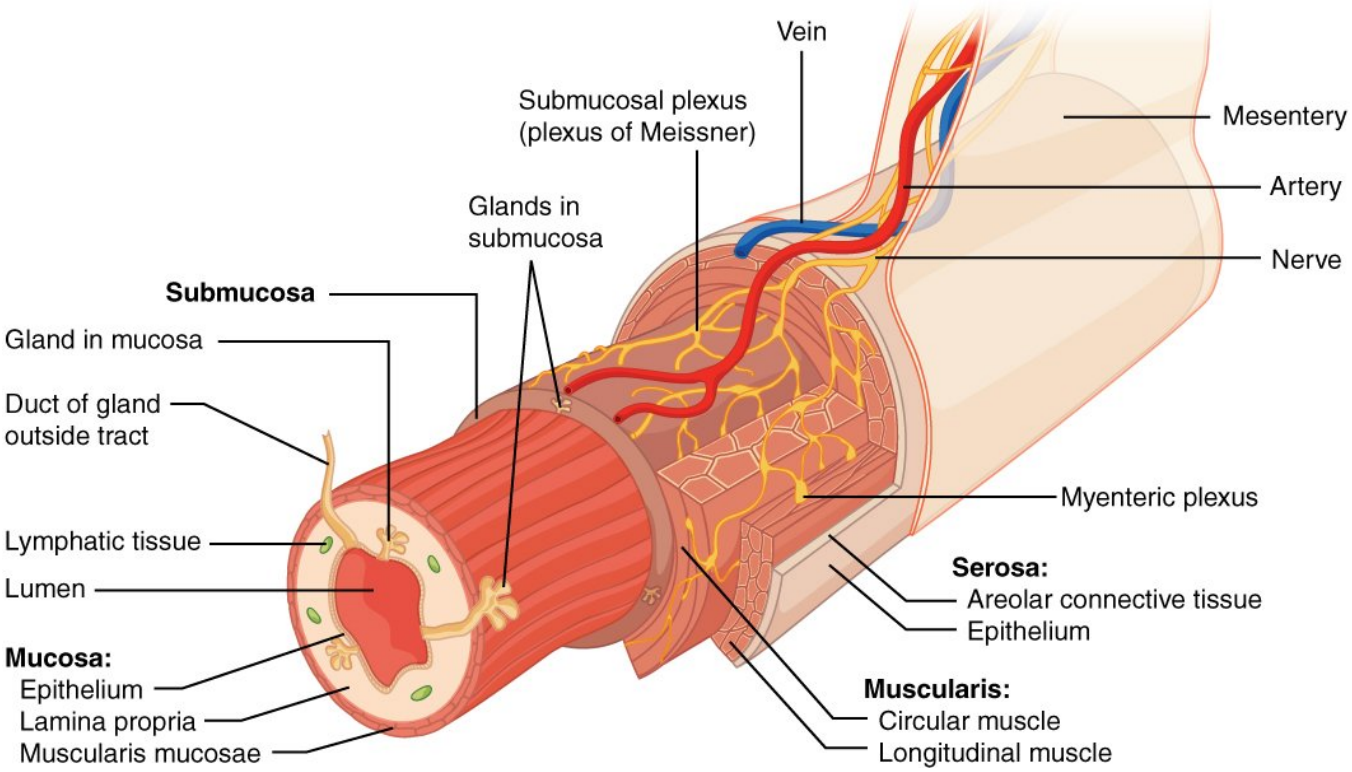
Common trap: Muscularis mucosae belongs to mucosa and is not muscularis externa.

[Core] Layers of the Alimentary Canal



Read lumen outward. Locate mucosa, submucosa, muscularis externa and outer covering; distinguish their nervous plexuses.

OpenStax A&P 2e, Fig. 23.3 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-1-overview-of-the-digestive-system | Publisher original; 1059 x 604 pixels

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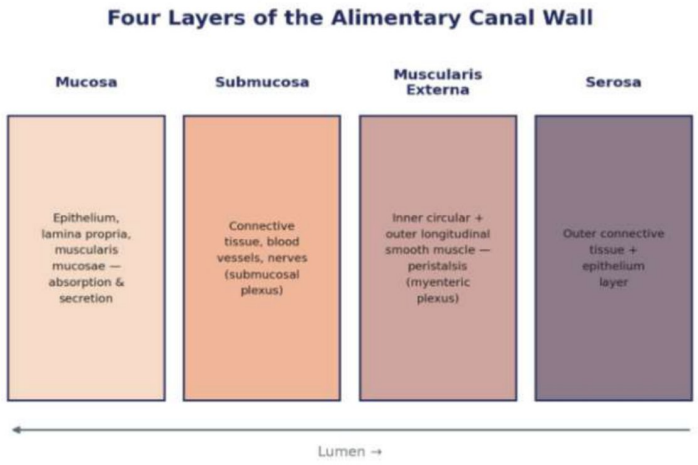
[Core] Four gut-wall layers



Mucosa → submucosa → muscularis externa → serosa, lumen to exterior

Read from lumen outward: mucosa, submucosa, muscularis externa, serosa/adventitia. Distinguish the thin muscularis mucosae from the much thicker muscularis externa.

S27 p.19 | Original labels retained; figure region enlarged.

D-HIST | Compare esophagus, stomach, small intestine and colon

[Core] | Rule 3b.iv

Esophagus: nonkeratinized stratified squamous epithelium resists abrasion. Multiple cell layers and flattened superficial nuclei are useful clues. It has no intestinal villi. Stomach: simple columnar surface mucous cells lead into gastric pits and glands; normal stomach has no villi. Parietal cells secrete acid and intrinsic factor; chief cells release pepsinogen. G cells produce gastrin, especially in the antrum; enterochromaffin-like cells release histamine and D cells somatostatin.

Small intestine: simple columnar absorptive cells and goblet cells cover villi, with crypts between them. Villi are tissue projections containing blood capillaries and a lacteal; microvilli are microscopic apical projections on individual enterocytes. Circular folds contain mucosa and submucosa. Duodenum has submucosal Brunner glands; jejunum typically has prominent folds/villi without those glands or large aggregated Peyer patches; ileum often has Peyer patches and shorter villi. Absence of one feature in a small field is not conclusive.

Colon: straight crypts with many goblet cells and no villi. Mucus protects and lubricates increasingly solid contents. On low power, compare overall architecture before individual cells. Histology questions may ask

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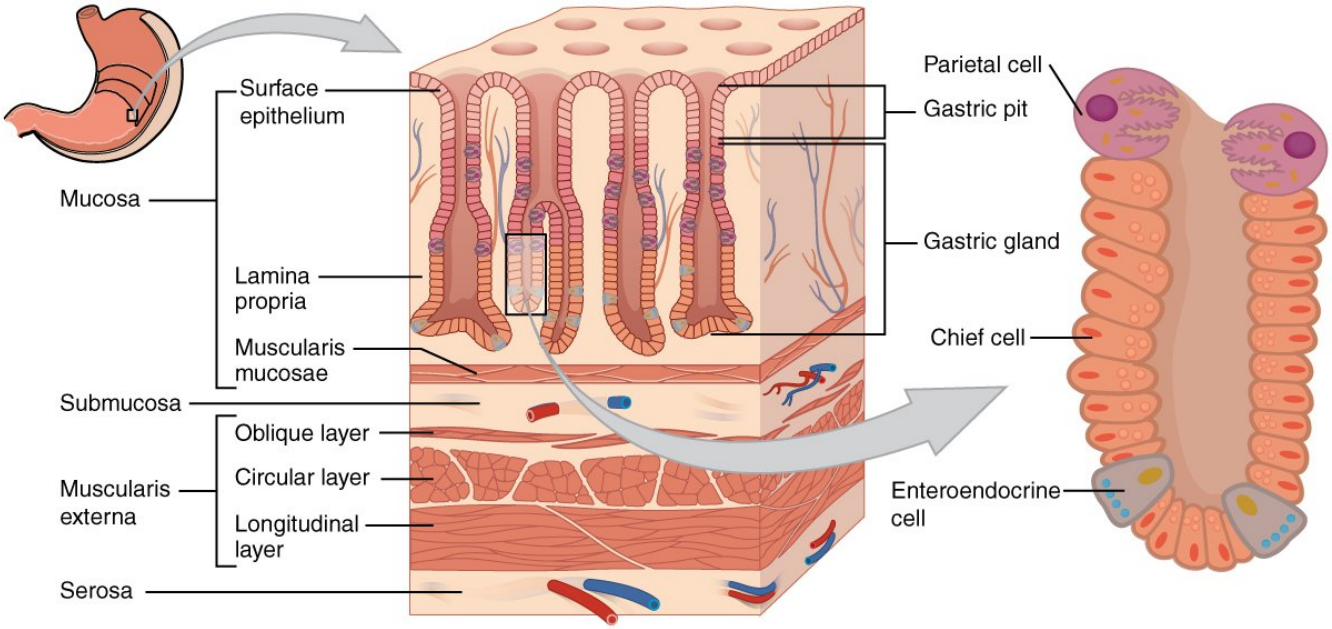
what function follows from structure: many thin projections imply absorption; multiple layers imply protection; secretory glands imply secretion. H&E commonly stains nuclei blue-purple and cytoplasm/extracellular proteins pink; staining color alone cannot identify a tissue.

Recall: A specimen has straight crypts, many goblet cells and no villi. What is the best identification?

Large intestine/colon. Explain the mucus-producing and water/electrolyte-recovery functions.

Common trap: A villus is multicellular; a microvillus is part of one cell and usually not separately resolved on a routine low-power slide.

[Core] Histology of the Stomach



Locate gastric pits, glands and secretory cell types. Relate acid, enzyme precursor and protective mucus to the labeled cells.

OpenStax A&P 2e, Fig. 23.16 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-4-the-stomach | Publisher original; 1214 x 568 pixels

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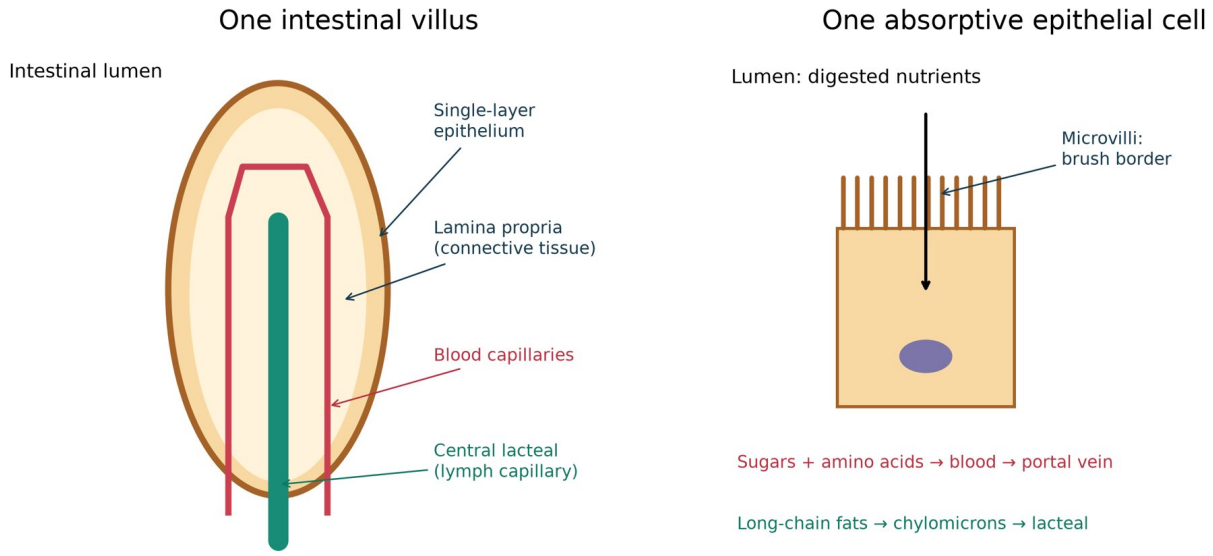
[Core] Villus, microvilli and nutrient transport



Villi are tissue projections; microvilli are projections of individual cells. Schematic, not to scale.

The villus contains connective tissue, blood capillaries and a blind-ended lymph capillary called a lacteal. Its epithelial cells bear microvilli. Monosaccharides and amino acids enter portal blood. Most absorbed long-chain lipids are packaged into chylomicrons and enter lacteals, then lymph before reaching blood. Short-chain fatty acids can enter portal blood directly. This is a teaching diagram, not a histology micrograph.

Original vector teaching schematic. p. | Original labels retained; figure region enlarged.

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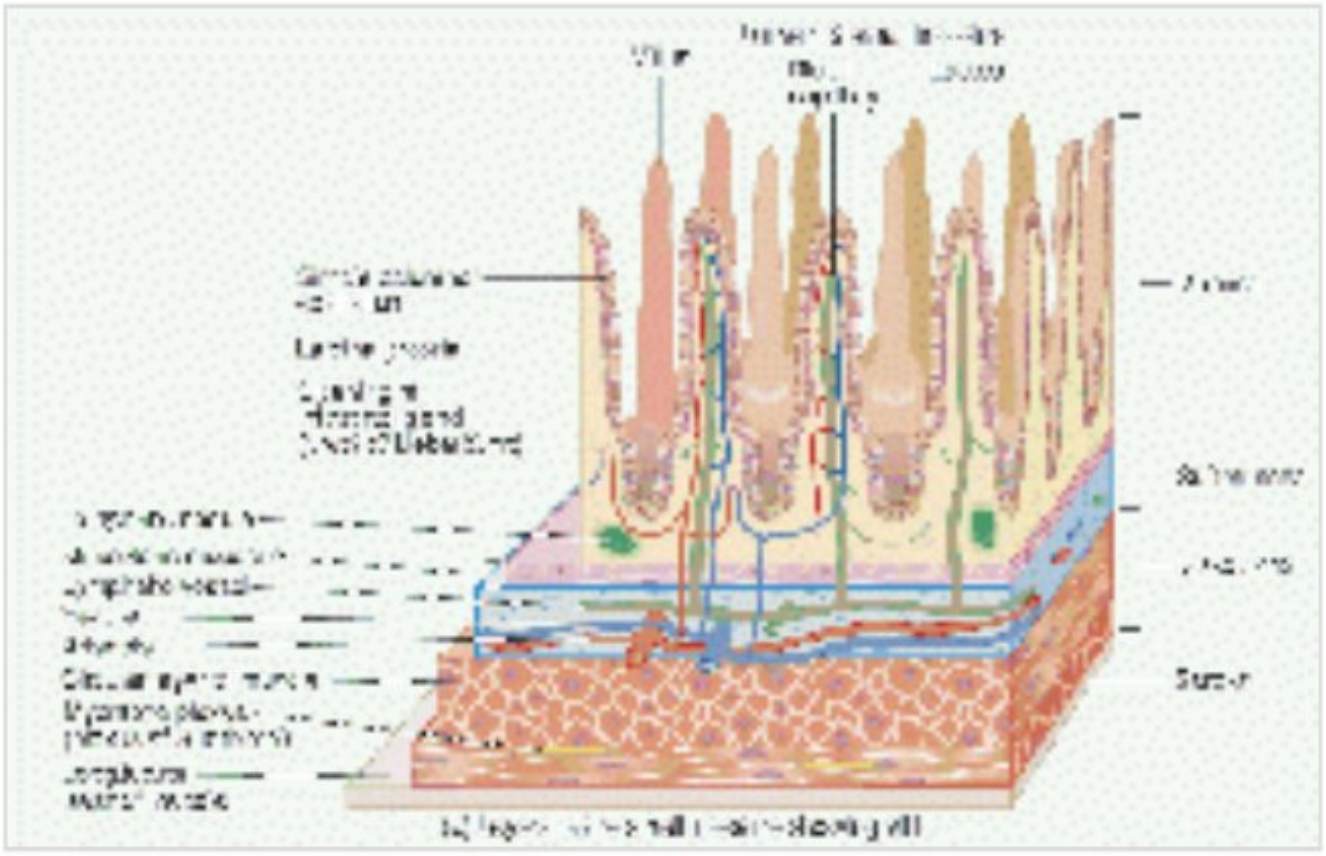
[Core] Small-intestinal wall and villi



Connect folds, villi and microvilli to progressively larger absorptive area. Locate mucosa, submucosa and muscular layers; villi project into the lumen.

WD p.4 | Original labels retained; figure region enlarged.

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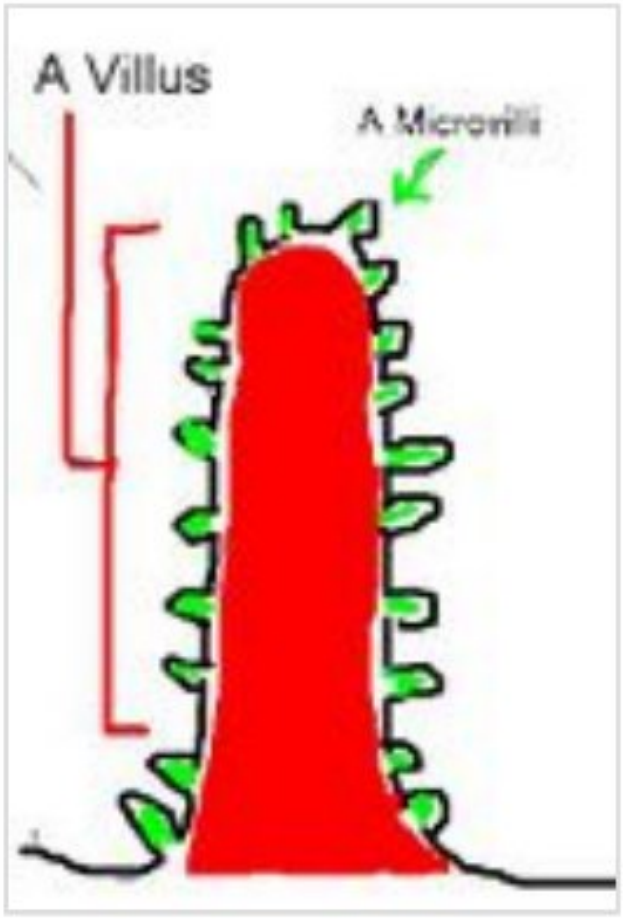
[Core] Villus and absorptive routes



Distinguish one villus from its much smaller microvilli (brush border). The drawing compares surface scales, not the full internal vessels. In a complete villus, sugars/amino acids enter blood capillaries and chylomicrons enter a central lacteal.

WD p.5 | Original labels retained; figure region enlarged.

D-MECH | Mechanical digestion, propulsion and chemical digestion

[Core] | Rule 3b.v

Mechanical processing changes size and mixing without hydrolyzing nutrient molecules: chewing, stomach churning and intestinal segmentation are examples. Peristalsis primarily propels contents with coordinated contraction behind and relaxation ahead of a bolus. Segmentation mainly mixes chyme and increases contact with the mucosa.

Chemical digestion hydrolyzes bonds in large food molecules using enzymes. Acid denatures proteins and helps activate pepsin, but acid is not itself a digestive enzyme. Bile emulsifies large fat globules into smaller droplets and helps form micelles; it is physical/chemical assistance without enzymatic triglyceride hydrolysis. Pancreatic lipase performs that hydrolysis.

Increasing surface area exposes more substrate to enzymes. Enzyme activity depends on pH, temperature and substrate availability. In an experimental table, identify the independent variable, controlled factors and measured product. A bile-plus-lipase sample may produce more fatty acid than lipase alone because more fat

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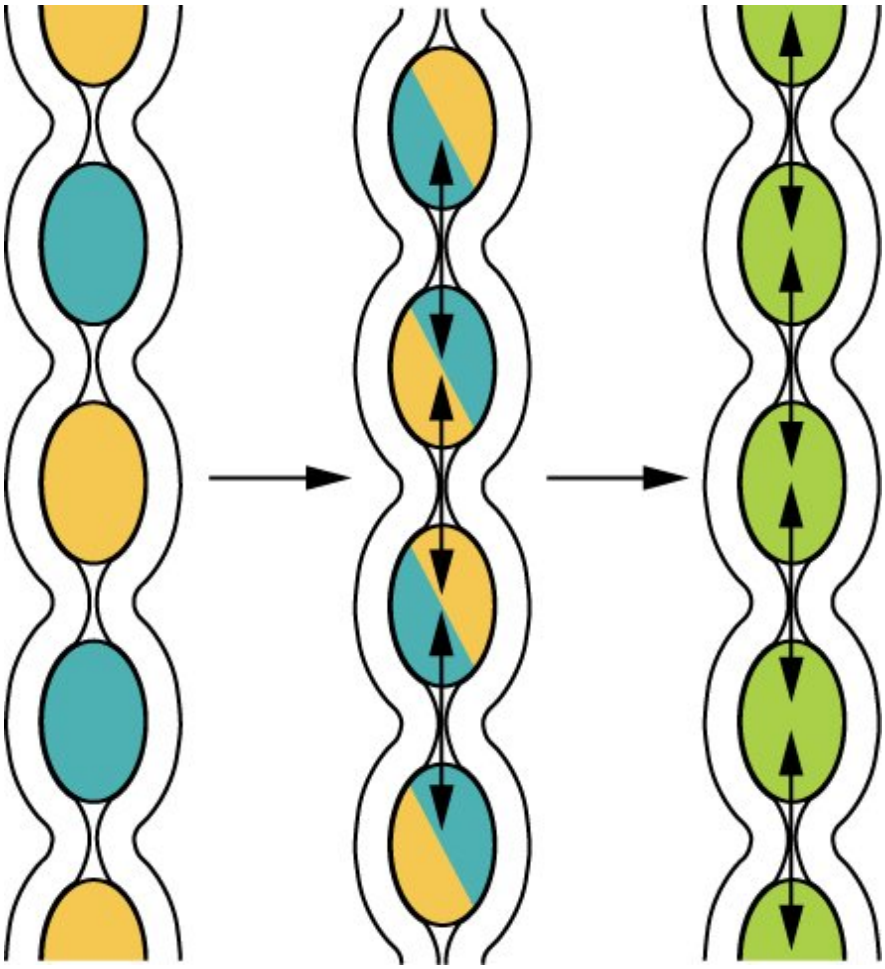
surface is accessible, not because bile is a second lipase.

Recall: Why does bile increase the effectiveness of lipase without being an enzyme?

It disperses fat into small droplets, increasing accessible surface, and supports transport of digestion products in micelles.

Common trap: Peristalsis is movement; not every movement directly breaks food into smaller pieces.

[Core] Segmentation



Alternating local contractions mix chyme. Compare segmentation with a propagating peristaltic wave.

OpenStax A&P 2e, Fig. 23.20 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-5-the-small-and-large-intestines | Publisher original; 442 x 484 pixels

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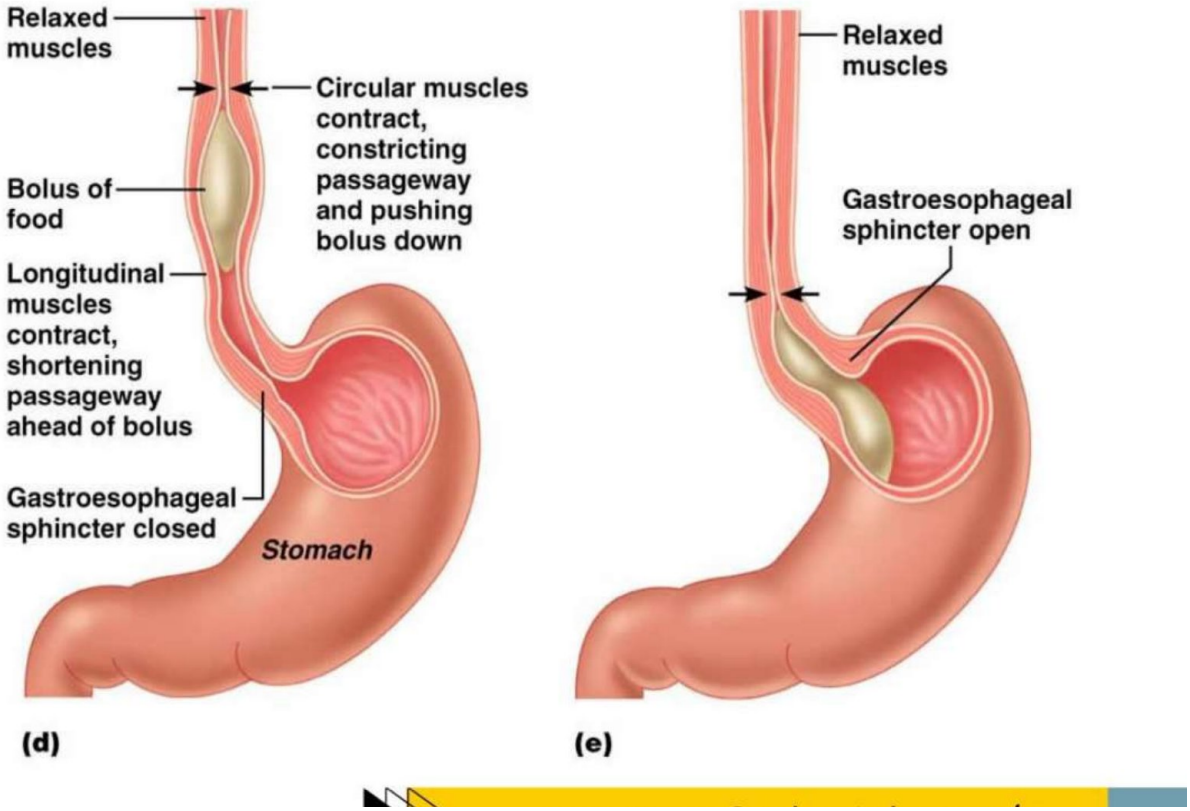
[Core] Peristalsis and the lower esophageal sphincter



A coordinated contraction behind the bolus and relaxation ahead move food toward the stomach. The lower esophageal sphincter helps prevent reflux.

S23 p.28 | Original labels retained; figure region enlarged.

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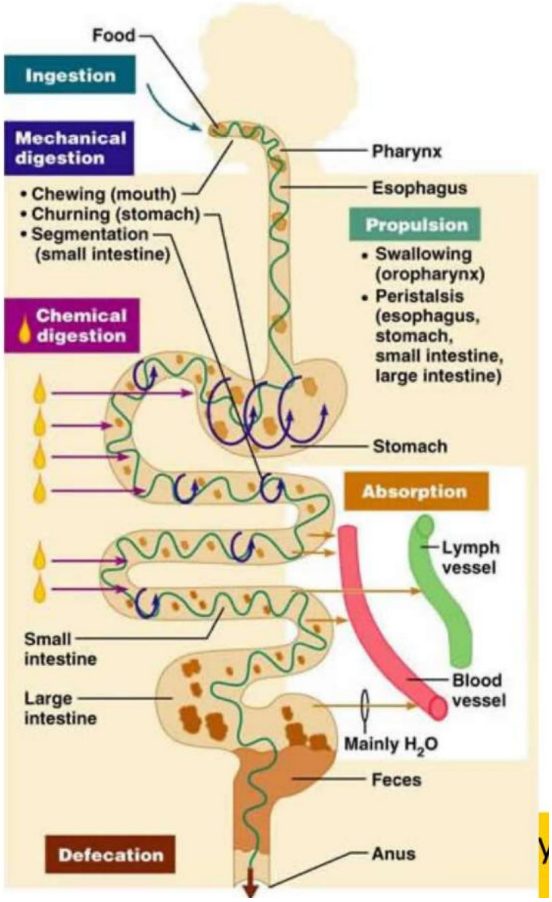
[Core] Digestive process map



Trace ingestion, propulsion, digestion, absorption and defecation. Absorption moves digested material across epithelium; defecation removes unabsorbed residue.

S23 p.35 | Original labels retained; figure region enlarged.

D-CARB | Carbohydrate digestion and absorption

[Core] | Rule 3b.vi

Salivary amylase begins hydrolysis of starch; it loses activity as stomach contents become acidic. Pancreatic amylase continues digestion in the small intestine, producing smaller carbohydrates rather than completing all digestion to glucose. Brush-border enzymes finish the process: maltase acts on maltose, sucrase on sucrose, and lactase on lactose. Absorbable principal products are glucose, galactose and fructose.

Glucose and galactose enter enterocytes by sodium-linked cotransport; fructose uses facilitated diffusion. Sugars exit toward intestinal capillaries and travel through the hepatic portal circulation to the liver. The sodium gradient ultimately depends on the sodium-potassium pump. This transport detail explains why glucose and sodium together support intestinal water absorption.

Humans lack enzymes that digest cellulose; fiber can add bulk, retain water and be fermented by colonic microbes to varying degrees. Lactase deficiency leaves lactose in the lumen, drawing water osmotically; bacteria ferment it and produce gas. This differs from an immune reaction against milk proteins.

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[Core] Lactose hydrolysis

$$\text{lactose} + H_2O \xrightarrow{\text{lactase}} \text{glucose} + \text{galactose}$$

Lactase is a brush-border enzyme. Low activity can cause osmotic water retention and fermentation of undigested lactose.

Carbohydrates

Enzymes/process: Salivary/pancreatic amylase; brush-border enzymes. Products: Monosaccharides. Route: Enterocyte -> blood capillaries -> hepatic portal vein.

Proteins

Enzymes/process: Pepsin; pancreatic proteases; peptidases. Products: Amino acids; small peptides enter cells then are further digested. Route: Enterocyte -> portal blood.

Long-chain fats

Enzymes/process: Bile assists; pancreatic lipase hydrolyzes. Products: Fatty acids + monoglycerides. Route: Micelles -> enterocyte -> reassembled lipids -> chylomicrons -> lacteal.

Recall: What products result from lactase acting on lactose?

Glucose and galactose; lack of lactase causes osmotic/fermentative symptoms rather than milk-protein allergy.

Common trap: Amylase does not digest protein, fat or cellulose.

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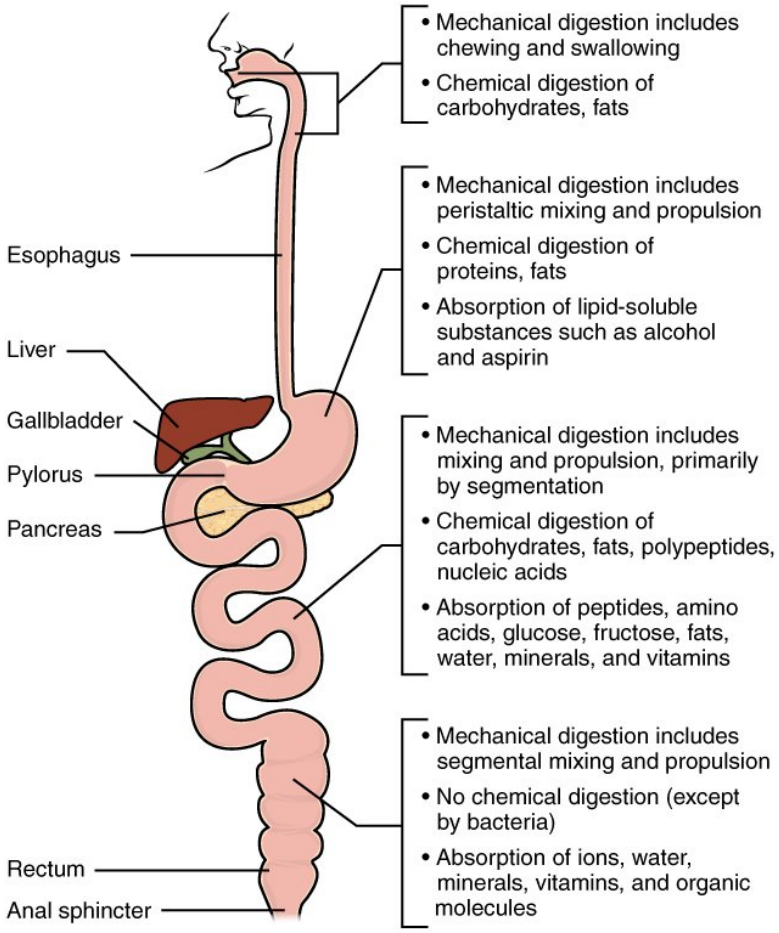
[Core] Digestion and Absorption



Follow the sites of nutrient breakdown and uptake. Use the adjacent nutrient table for the core protein, fat and carbohydrate pathways.

OpenStax A&P 2e, Fig. 23.28 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-7-chemical-digestion-and-absorption-a-closer-look | Publisher original; 636 x 790 pixels

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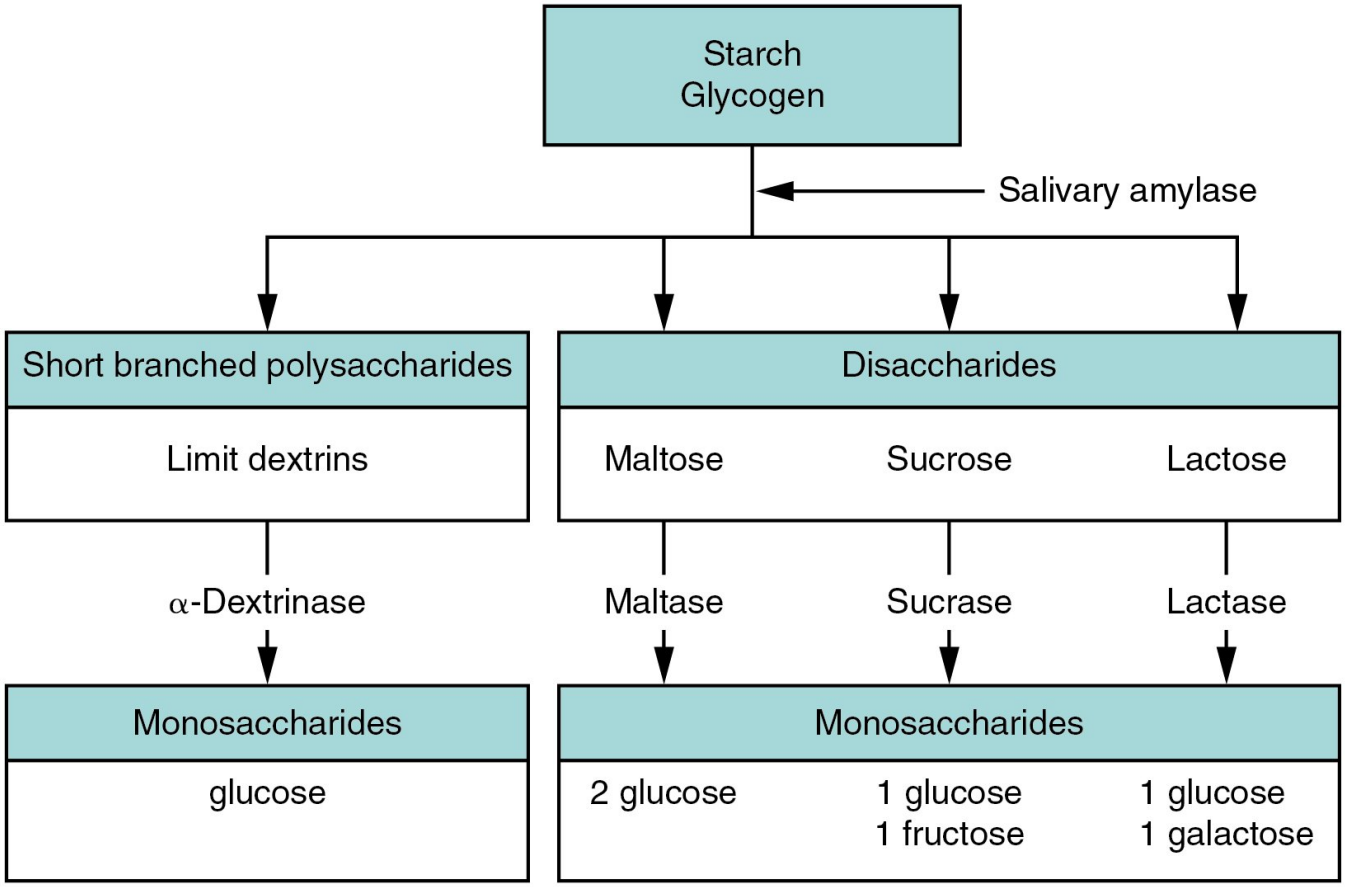
[Core] Carbohydrate Digestion Flow Chart



Trace starch to smaller carbohydrates and then absorbable monosaccharides. Lactase acts on lactose, not starch.

OpenStax A&P 2e, Fig. 23.29 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-7-chemical-digestion-and-absorption-a-closer-look | Publisher original; 1635 x 1080 pixels

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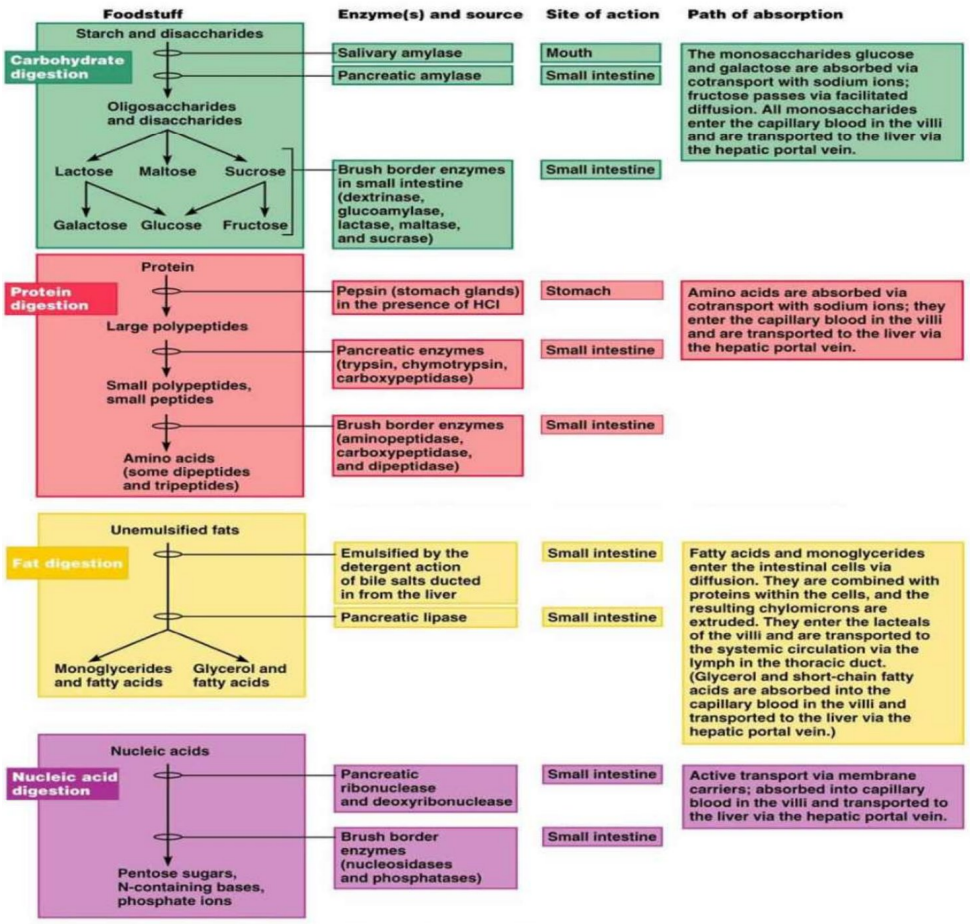
[Core] Chemical digestion overview



Read each nutrient pathway from large molecule to absorbable products. The readable nutrient tables beside this figure restate the essential carbohydrate, protein and fat pathways; nucleic-acid digestion is enrichment.

S23 p.36 | Original labels retained; figure region enlarged.

D-PROT | Protein digestion, acid and gastric protection

[Core] | Rule 3b.vi

Parietal cells release HCl, which denatures food proteins and helps convert chief-cell pepsinogen to pepsin. Pepsin begins protein hydrolysis in an acidic environment, producing peptides. Mucus, bicarbonate at the epithelial surface, tight junctions, blood flow and epithelial renewal protect the stomach from its own contents.

Pancreatic proteases are secreted largely as inactive zymogens to limit self-digestion. Duodenal brush-border enteropeptidase activates trypsinogen to trypsin; trypsin activates additional protease precursors, including more trypsinogen. Trypsin, chymotrypsin and carboxypeptidases act on different peptide bonds. Brush-border and intracellular peptidases complete digestion.

Amino acids and small peptides cross enterocytes by specific transport systems; absorbed peptides are mostly broken into amino acids before entering portal blood. Intrinsic factor is also made by parietal cells, but its role is vitamin B12 absorption in the terminal ileum, not peptide hydrolysis. An ulcer represents failure of mucosal defenses relative to damaging factors, often H. pylori or NSAID exposure.

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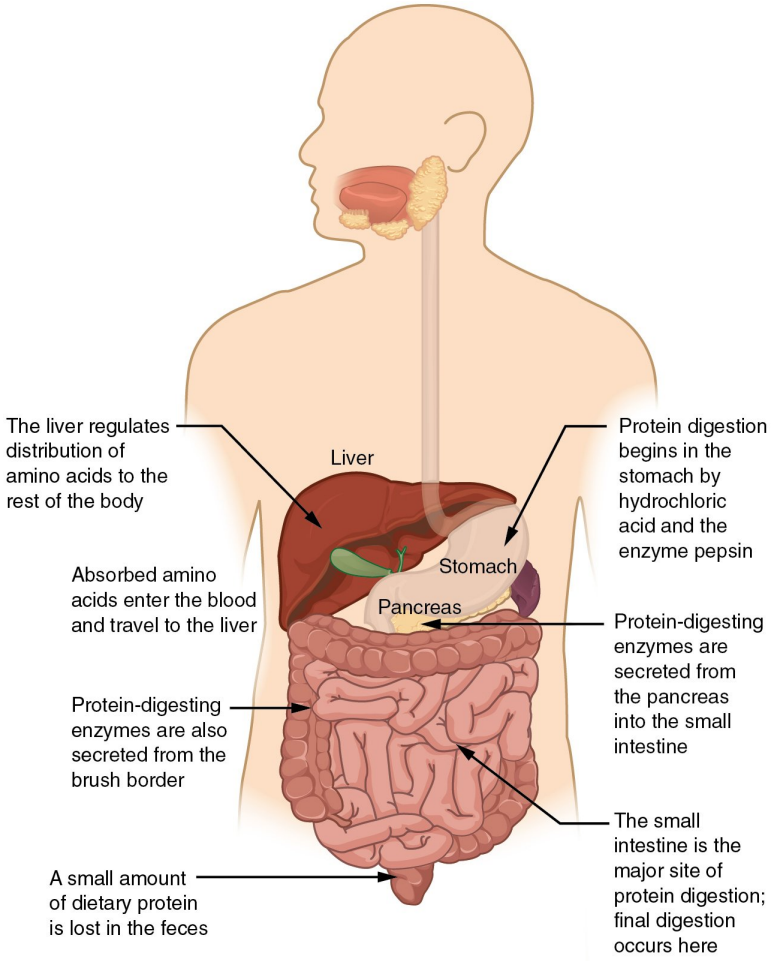
Recall: Why does the pancreas release trypsinogen rather than active trypsin?

Inactive secretion helps prevent pancreatic self-digestion; activation normally begins in the intestinal lumen.

Common trap: Pepsinogen comes from chief cells, while HCl and intrinsic factor come from parietal cells.

[Core] Digestion of Protein



Track stomach and pancreatic enzymes through protein digestion; match the organ to the site of enzyme action.

OpenStax A&P 2e, Fig. 23.30 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-7-chemical-digestion-and-absorption-a-closer-look | Publisher original; 1281 x 1602 pixels

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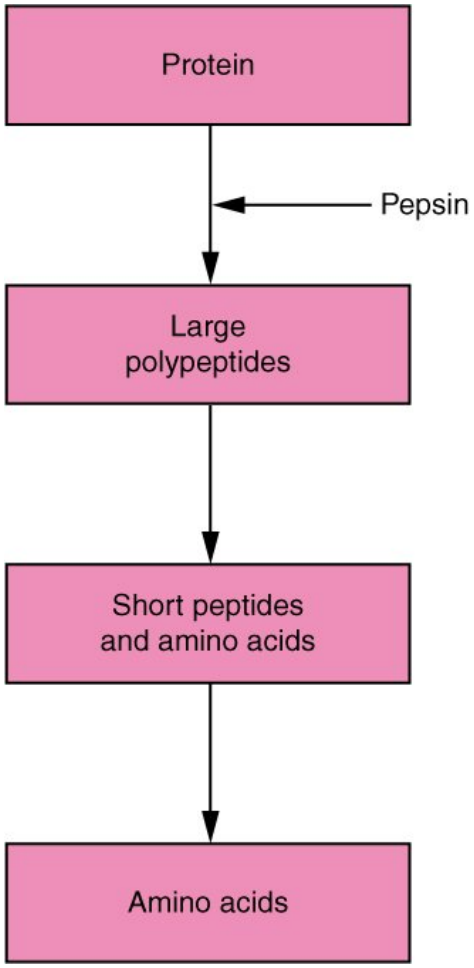
[Core] Digestion of Protein Flow Chart



Follow the progression from proteins to peptides to amino acids. Small peptides can enter enterocytes before further intracellular digestion.

OpenStax A&P 2e, Fig. 23.31 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-7-chemical-digestion-and-absorption-a-closer-look | Publisher original; 328 x 677 pixels

D-FAT | Fat digestion, micelles and lymphatic absorption

[Core] | Rule 3b.vi

Bile salts made by the liver emulsify dietary fat. Pancreatic lipase, aided by colipase, hydrolyzes triglycerides mainly into free fatty acids and monoglycerides. Bile-salt micelles carry poorly water-soluble products and fat-soluble vitamins toward the brush border. Micelles are not the same as chylomicrons.

Long-chain lipid products enter enterocytes, are reassembled into triglycerides and packaged with proteins into chylomicrons. Chylomicrons enter villus lacteals, travel through lymph and then enter venous blood. Many short- and medium-chain fatty acids can reach portal blood directly. Therefore, do not claim every absorbed fat follows exactly the same route.

Vitamins A, D, E and K are fat-soluble. Poor bile delivery, pancreatic enzyme deficiency or extensive small-intestinal disease can impair fat absorption, causing greasy stools and vitamin deficiencies. Compare glucose (capillaries -> portal vein -> liver) with most long-chain dietary fat (lacteal -> lymph -> blood). Water-soluble nutrients generally follow the capillary route.

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[Core] Pancreatic triglyceride digestion

$$\text{triglyceride} + 2H_2O \rightarrow 2\text{–monoglyceride} + 2\,\text{fatty acids}$$

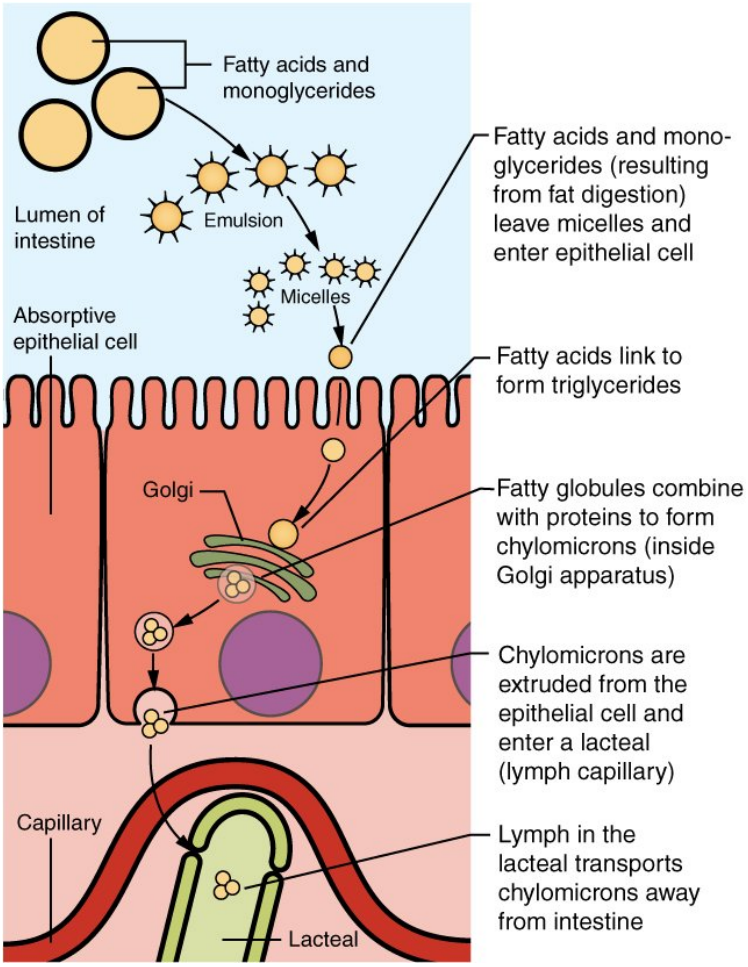
Pancreatic lipase commonly yields a 2-monoglyceride and two free fatty acids. Bile aids emulsification and micelles; it is not the hydrolyzing enzyme.

Recall: Distinguish a micelle from a chylomicron.

A micelle assists lipid delivery in the intestinal lumen; a chylomicron is assembled inside an enterocyte and transports absorbed lipid through lymph.

Common trap: Bile is made in liver, stored/concentrated in gallbladder, and is not a digestive enzyme.

[Core] Lipid Absorption



Trace bile-assisted delivery, enterocyte uptake, repackaging and chylomicron entry into a lacteal. Micelles and chylomicrons are different structures.

OpenStax A&P 2e, Fig. 23.33 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-7-chemical-digestion-and-absorption-a-closer-look | Publisher original; 640 x 827 pixels

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D-LIVER | Liver, pancreas, Kupffer cells and digestive regulation

[S] [N] | Rule 3b.x; 3b.ii,vi foundations

Core foundation: identify these organs and their basic secretions. [S] [N]: detailed liver/pancreatic function, including Kupffer cells. Figure badges distinguish these depths.

The liver produces bile, processes absorbed nutrients, stores glycogen and vitamins, synthesizes proteins such as albumin and many clotting factors, and metabolizes many chemicals. The hepatic portal vein brings nutrient-rich blood from digestive organs; the hepatic artery supplies oxygenated arterial blood. Blood moves through sinusoids toward central veins; bile moves through canaliculi toward bile ducts in the opposite general direction within a lobule.

Kupffer cells are resident hepatic macrophages associated with sinusoids. They phagocytose bacteria and debris reaching the liver, linking digestive anatomy to innate immunity. Hepatocytes perform most metabolic and bile-producing functions. In histology, look for plates of hepatocytes, sinusoids, central veins and portal areas; do not confuse a macrophage with a bile duct cell.

Pancreatic acinar cells produce digestive enzymes; duct cells supply bicarbonate-rich fluid to neutralize acidic chyme. Islets release hormones into blood (insulin/glucagon), a separate endocrine function. CCK, released in response to fat/protein in the small intestine, promotes enzyme secretion and gallbladder contraction. Secretin responds to acid and promotes bicarbonate secretion. Gastrin promotes gastric activity; enteric nerves and parasympathetic input coordinate motility/secretion. Study these as cause-and-effect loops, not isolated hormone names.

For a liver image, locate a portal region containing branches of the hepatic artery, portal vein and bile duct. Blood flows from portal regions through sinusoids toward central veins. Bile flows through canaliculi toward bile ducts in the opposite overall direction. Distinguish hepatocytes that make bile, gallbladder that stores/concentrates it, and pancreas that makes pancreatic juice.

Recall: What does a Kupffer cell do that a hepatocyte does not primarily do?

It acts as a resident phagocytic immune cell that removes microbes and debris from sinusoidal blood.

Common trap: The hepatic portal vein is not the same as the hepatic vein that drains the liver.

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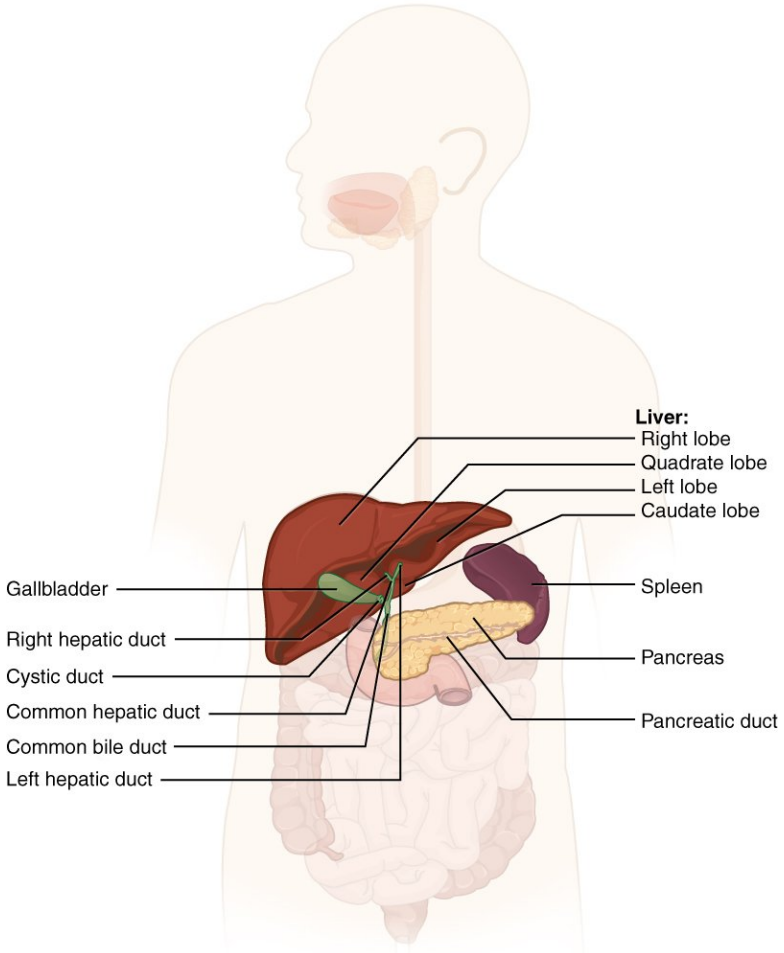
[Core] Accessory Organs



Relate liver, gallbladder and pancreas to the duodenum. Food remains in the canal while secretions arrive through ducts.

OpenStax A&P 2e, Fig. 23.24 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-6-accessory-organs-in-digestion-the-liver-pancreas-and-gallbladder | Publisher original; 805 x 1002 pixels

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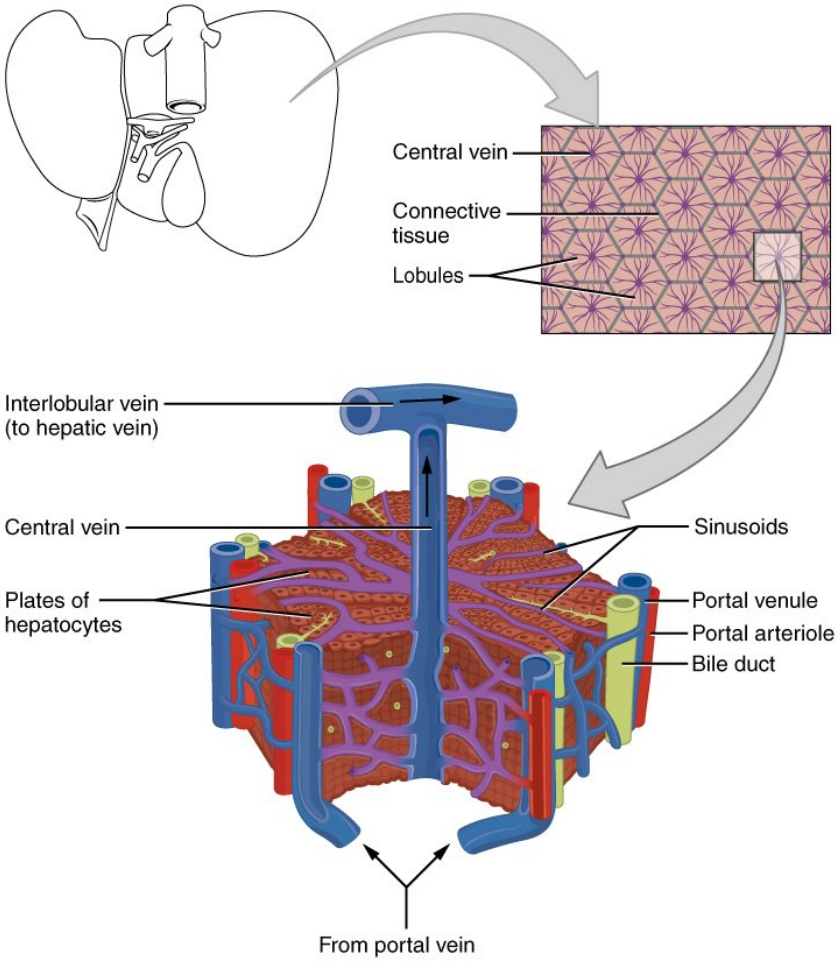
[S] [N] Microscopic Anatomy of the Liver



Trace blood from portal regions through sinusoids toward the central vein. Trace bile in the opposite overall direction toward ducts.

OpenStax A&P 2e, Fig. 23.25 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-6-accessory-organs-in-digestion-the-liver-pancreas-and-gallbladder | Publisher original; 721 x 824 pixels

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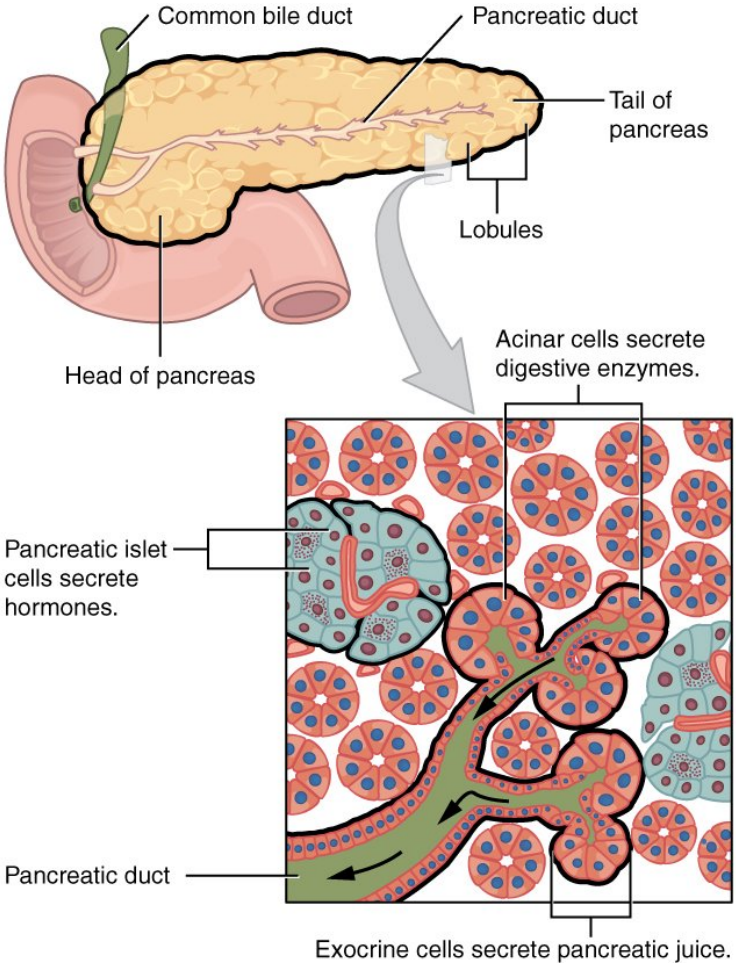
[S] [N] Exocrine and Endocrine Pancreas



Separate exocrine acini/ducts from endocrine islets. Digestive enzymes and bicarbonate reach the intestine; hormones reach blood.

OpenStax A&P 2e, Fig. 23.26 | https://openstax.org/books/anatomy-and-physiology-2e/pages/23-6-accessory-organs-in-digestion-the-liver-pancreas-and-gallbladder | Publisher original; 637 x 827 pixels

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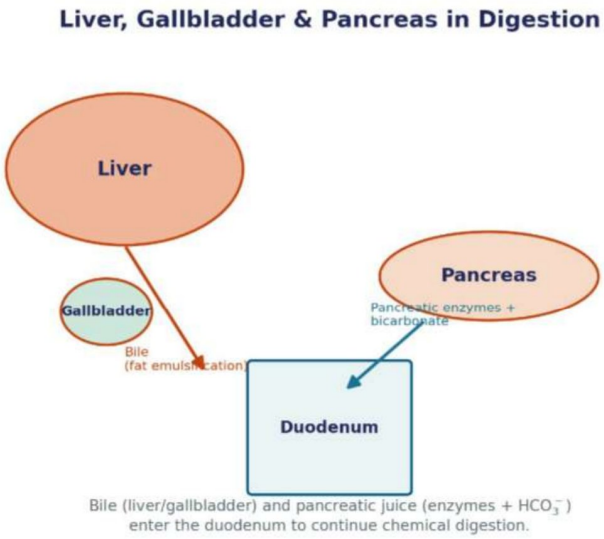
[S] [N] Bile and pancreatic secretion routes



Bile (liver/gallbladder) and pancreatic secretions both enter the duodenum

Liver makes bile; gallbladder stores/concentrates it; pancreatic ducts supply digestive enzymes and bicarbonate. Both secretions reach the duodenum. Basic organ roles are Core; detailed function and Kupffer cells are S/N.

S27 p.26 | Original labels retained; figure region enlarged.

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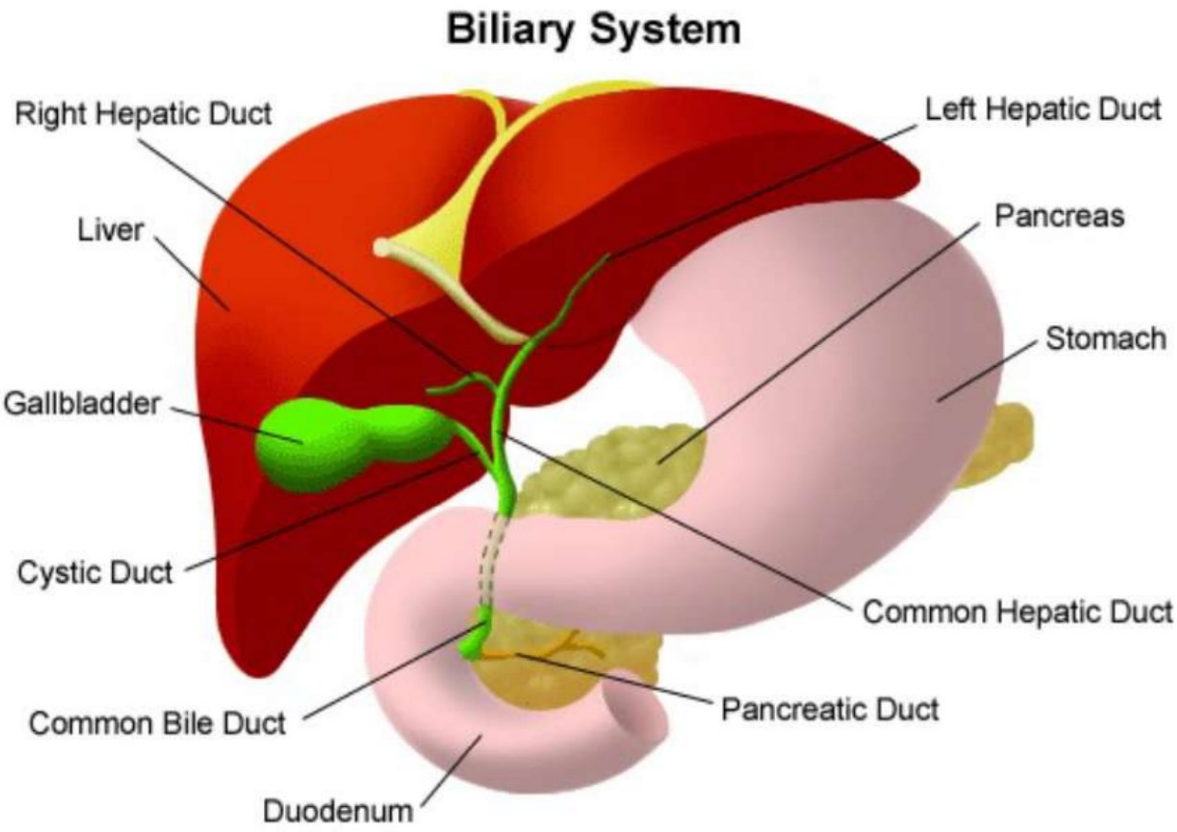
[Core] Biliary tree



Trace bile from liver through hepatic ducts and the common bile duct toward the duodenum. The cystic duct connects the gallbladder. Bile emulsifies fat; it is not a digestive enzyme.

S23 p.32 | Original labels retained; figure region enlarged.

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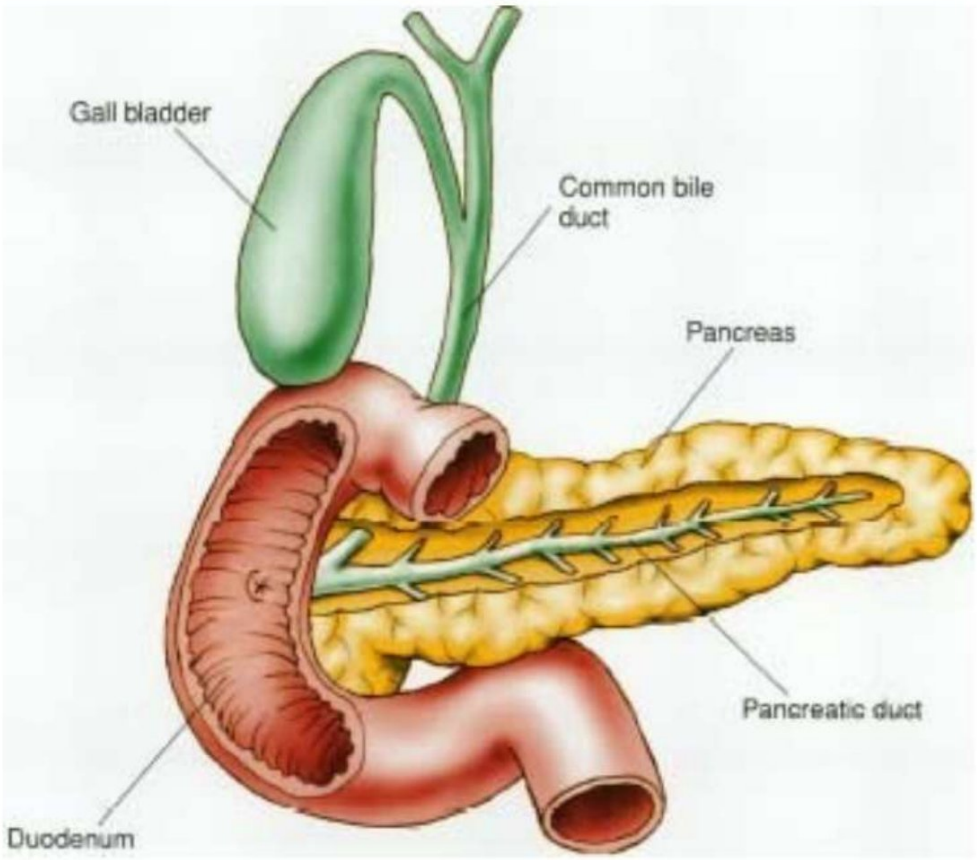
[S] [N] Pancreatic duct and duodenum



Locate the pancreas within the duodenal curve and trace the pancreatic duct. Exocrine acini supply enzymes, duct cells add bicarbonate, and endocrine islets release hormones into blood.

S23 p.33 | Original labels retained; figure region enlarged.

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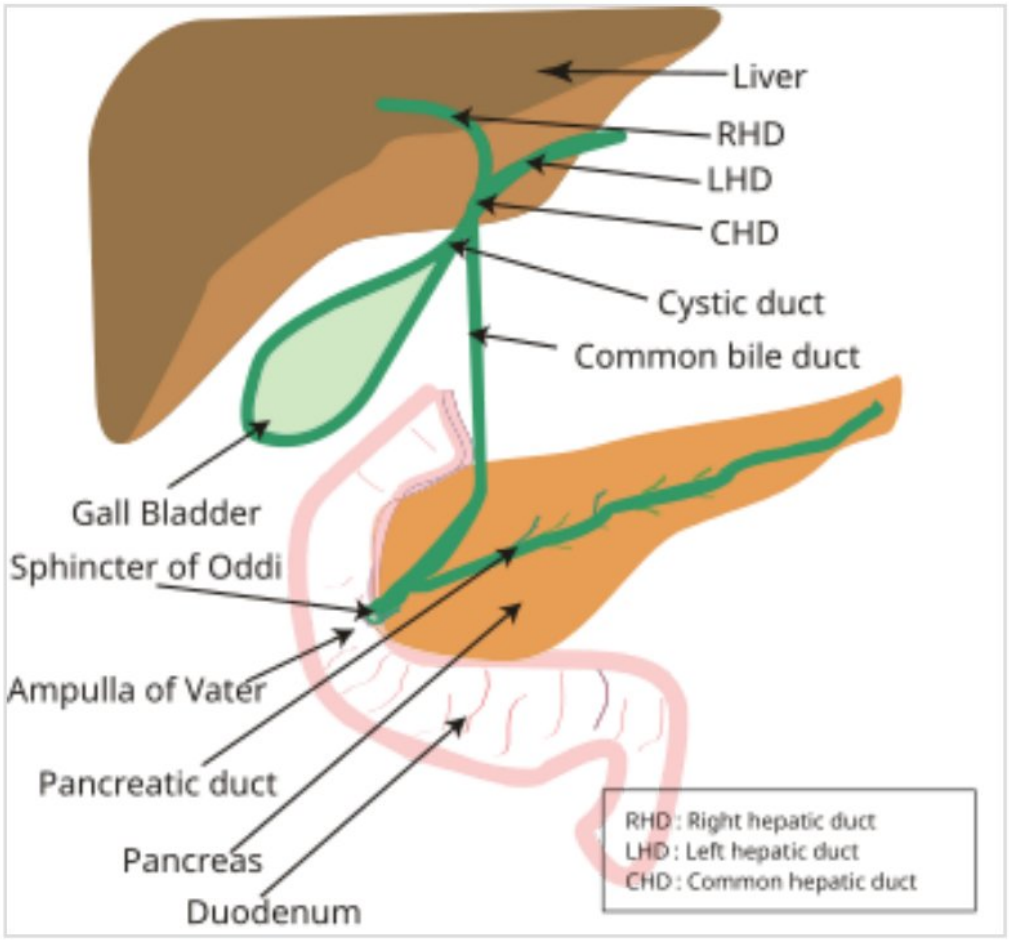
[Core] Hepatobiliary and pancreatic ducts



Locate hepatic, cystic, common bile and pancreatic ducts. Follow their connections to the duodenum; gallbladder secretion is bile, not pancreatic juice.

WD p.2 | Original labels retained; figure region enlarged.

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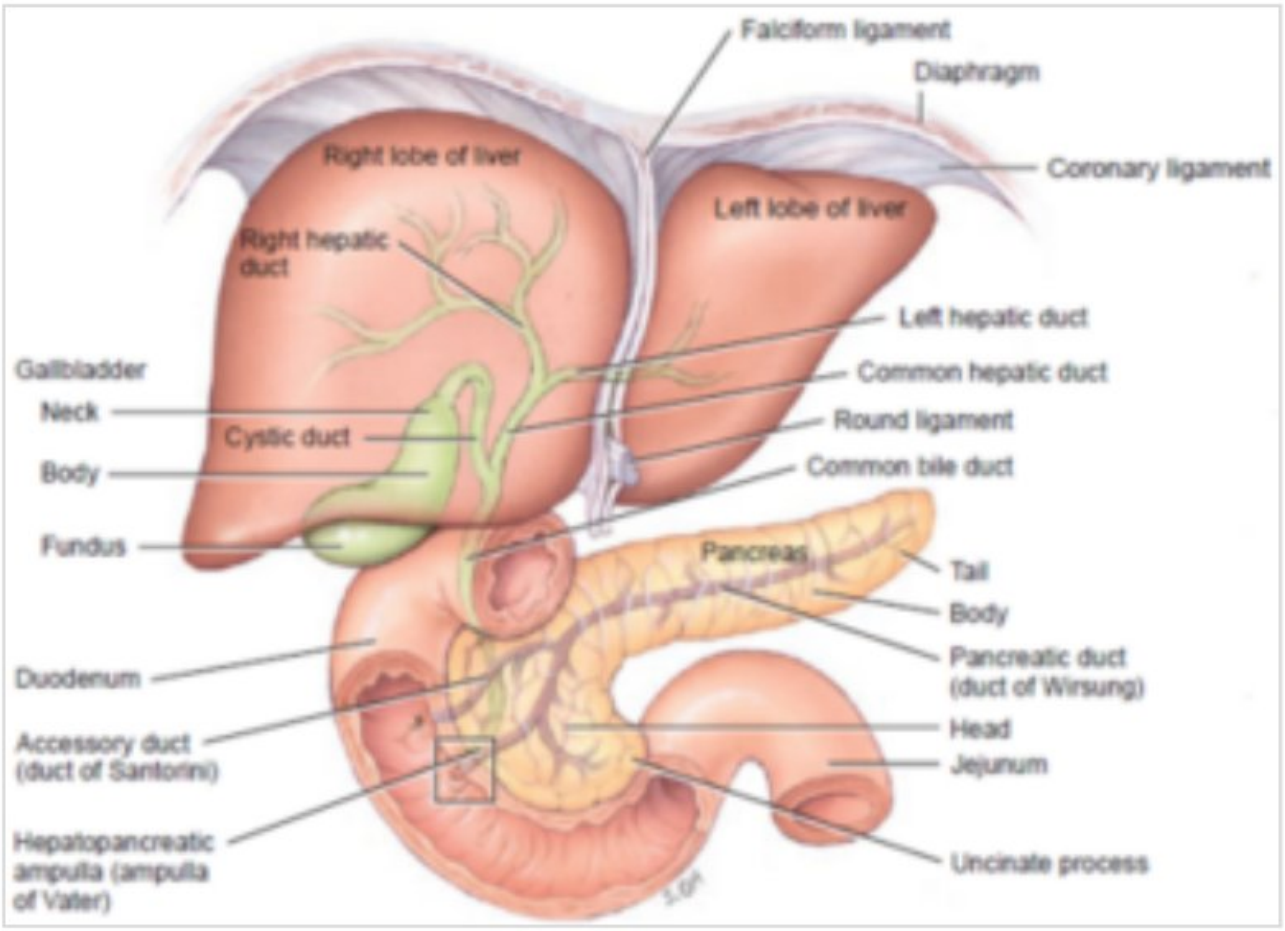
[Core] Accessory digestive organs



Identify liver, gallbladder and pancreas relative to the duodenum. Match each organ to its secretions.

WD p.5 | Original labels retained; figure region enlarged.

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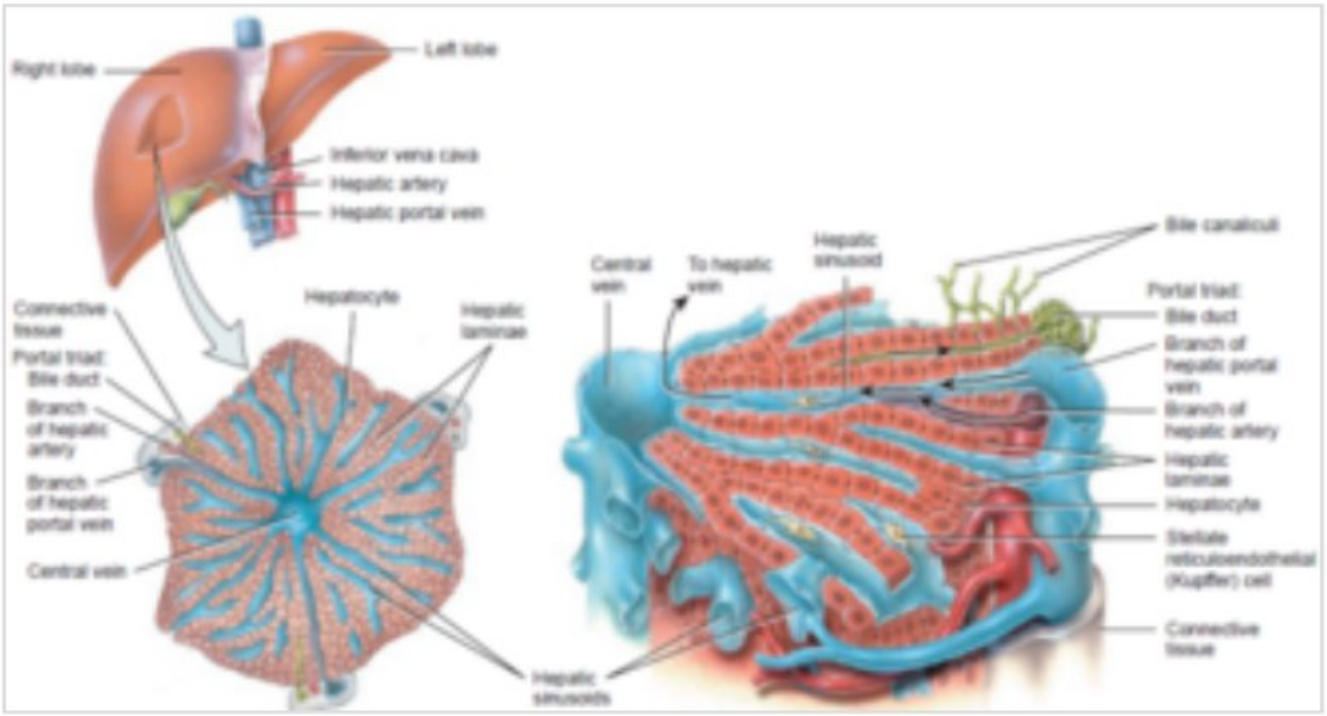
[S] [N] Liver lobule and portal region



Locate portal region, sinusoids and central vein. Blood flows toward the central vein; bile flows toward bile ducts. Kupffer cells line sinusoids and phagocytose material from portal blood.

WD p.5 | Original labels retained; figure region enlarged.

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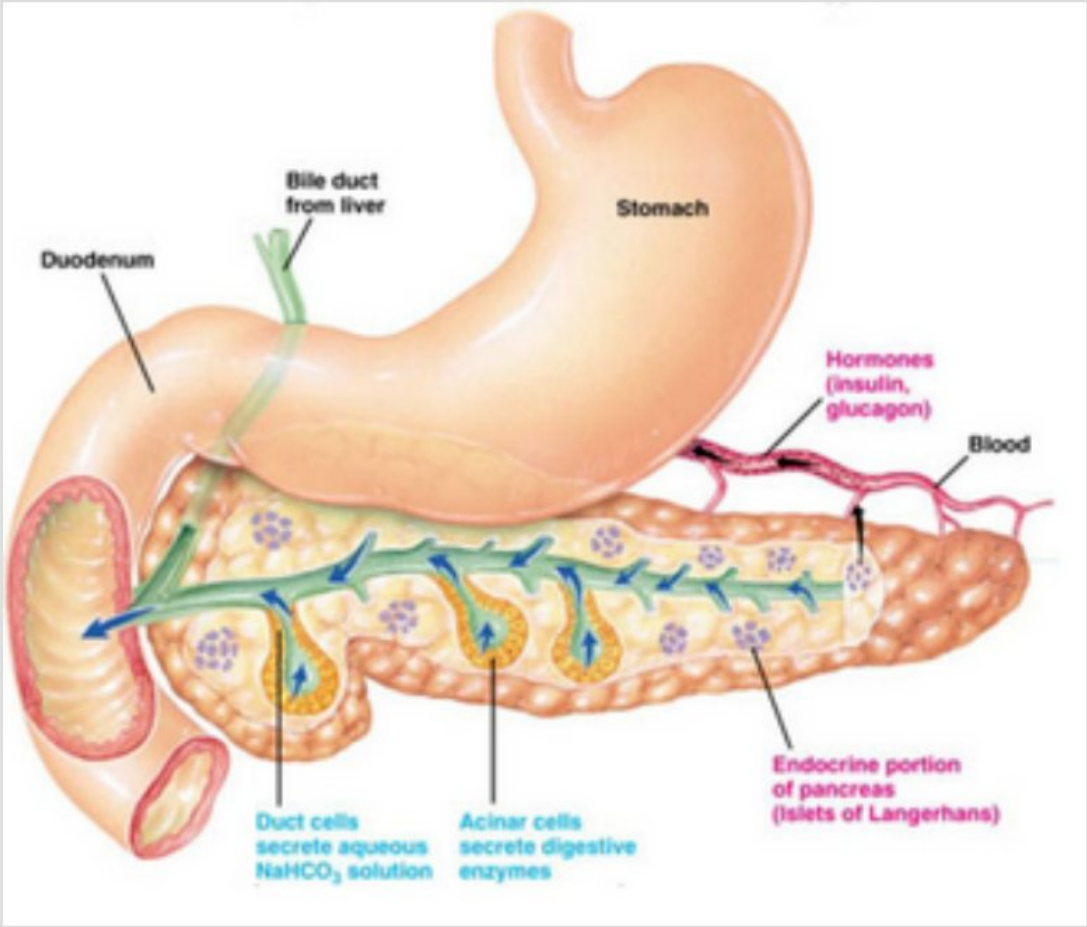
[S] [N] Pancreas, stomach and duodenum



Trace pancreatic juice to the duodenum. Bicarbonate neutralizes acid; enzyme activities digest macronutrients. Islets belong to endocrine function.

WD p.6 | Original labels retained; figure region enlarged.

D-EX | Exercise, obesity and digestive function

[Core] | Rule 3b.vii

Regular moderate activity can support intestinal motility and metabolic health. During very intense exercise, blood flow is redistributed toward working muscle and away from the gut; dehydration, altered motility and reduced intestinal perfusion can contribute to cramps or diarrhea. The effect depends on intensity, hydration, meal timing and the individual.

Obesity is associated with increased risk of reflux, gallstones, fatty liver disease and several digestive cancers. Increased abdominal pressure can favor reflux; altered metabolism can promote fat accumulation in the liver. Explain the mechanism and distinguish an association from certainty: a risk factor does not mean a person necessarily has a disease.

Dietary fiber, appropriate hydration and balanced nutrition support digestive function, but older food-pyramid slides and a 2000-calorie example are background material, not a personalized diet prescription. For an experiment, compare groups fairly and consider confounders such as diet and activity before claiming that weight alone caused the observed outcome.

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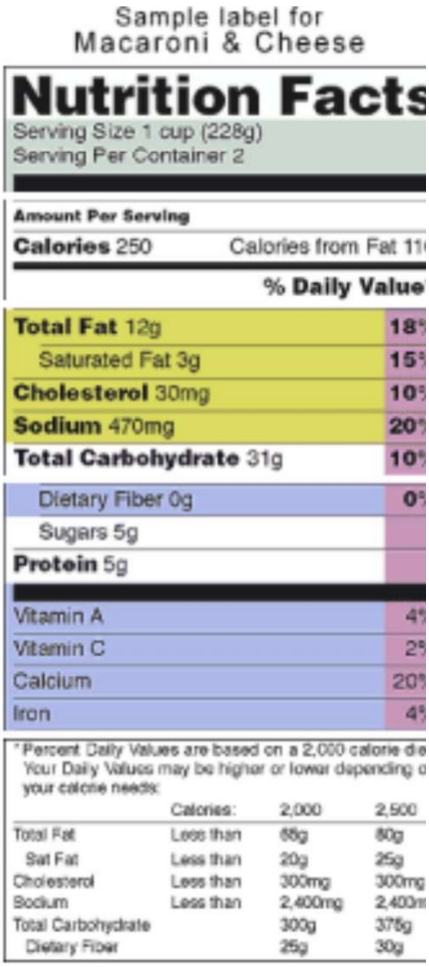
Recall: Why can strenuous exercise cause GI symptoms while moderate activity helps motility?

Intensity changes physiology: heavy exertion can reduce gut perfusion and alter fluid balance/motility, whereas regular moderate activity can support normal function.

Common trap: Do not convert a population-level risk association into a diagnosis.

[E] enrichment Legacy nutrition label


Sample label for
Macaroni & Cheese

Nutrition Facts

Serving Size 1 cup (228g)
Serving Per Container 2

Amount Per Serving

Calories 250 Calories from Fat 11

% Daily Value

Total Fat 12g	18%
Saturated Fat 3g	15%
Cholesterol 30mg	10%
Sodium 470mg	20%
Total Carbohydrate 31g	10%
Dietary Fiber 0g	0%
Sugars 5g	
Protein 5g	
Vitamin A	4%
Vitamin C	2%
Calcium	20%
Iron	4%

* Percent Daily Values are based on a 2,000 calorie die
Your Daily Values may be higher or lower depending o
your calorie needs:

	Calories:	2,000	2,500
Total Fat	Less than	65g	80g
Sat Fat	Less than	20g	25g
Cholesterol	Less than	300mg	300mg
Sodium	Less than	2,400mg	2,400m
Total Carbohydrate		300g	375g
Dietary Fiber		25g	30g

Practice distinguishing serving size from servings per container and calories per serving from calories in a whole package. This is a historical label, not current labeling guidance.

S23 p.41 | Original labels retained; figure region enlarged.

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Digestive diseases

Stomach and duodenal ulcers / peptic ulcer disease

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Mucosal injury often follows H. pylori infection or NSAID-related loss of protective mechanisms, with acid contributing.

Recognition clues: Upper abdominal pain; bleeding or perforation can occur.

Distinguish: Gastric = stomach; duodenal = first small-intestinal segment. The rule repeats PUD in the advanced list; study it once with advanced treatment depth.

[S] [N] Treatment: Acid suppression and H. pylori eradication when present; manage NSAID exposure; complications may require procedures.

[S] [N] Prevention: Reduce avoidable ulcer-promoting exposures and treat identified infection; not simply avoidance of spicy food.

[Core] Gastric ulcer image



Locate the focal mucosal defect. Ulceration breaches protective mucosa; common mechanisms involve H. pylori or NSAID-associated loss of protection. This image is not sufficient for diagnosis.

WD p.8 | Original labels retained; figure region enlarged.

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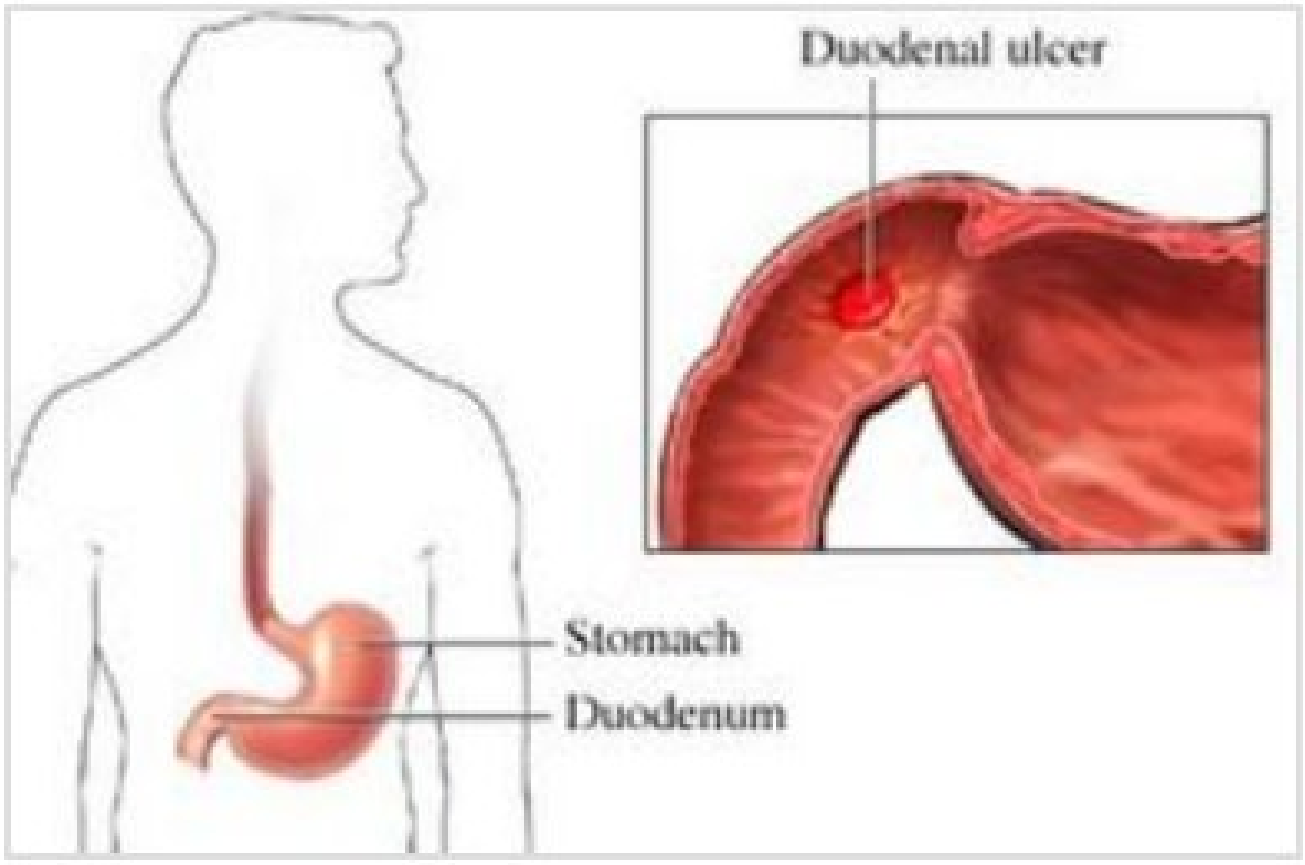
[Core] Duodenal ulcer location



Distinguish duodenum from stomach: both can develop peptic ulcers but are different anatomic sites.

WD p.8 | Original labels retained; figure region enlarged.

Digestive-system cancers

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Uncontrolled cell growth can affect esophagus, stomach, colon/rectum, liver, pancreas and other digestive structures.

Recognition clues: Effects depend on site: obstruction, bleeding, altered digestion or weight loss can occur.

Distinguish: Know location and consequence; no one symptom or treatment identifies all digestive cancers.

[S] [N] Treatment: Site/stage-directed surgery, systemic drugs and/or radiation; selected tumors use targeted or immune therapies.

[S] [N] Prevention: Tobacco/alcohol risk reduction, relevant hepatitis prevention and age/risk-appropriate screening; prevention is not guaranteed.

Diarrhea

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Excess luminal water results from secretion, poor absorption, osmotic solutes, inflammation or altered transit.

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Recognition clues: Loose stools with dehydration/electrolyte-loss risk.

Distinguish: Lactose malabsorption can cause osmotic diarrhea; inflammatory disease can damage absorption.

[S] [N] Treatment: Fluid/electrolyte replacement and cause-specific treatment; antibiotics are not universally appropriate.

[S] [N] Prevention: Safe food/water and hygiene for infectious causes; address specific underlying disease.

Lactose intolerance

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Low brush-border lactase leaves lactose unabsorbed; osmotic water retention and microbial fermentation cause symptoms.

Recognition clues: Bloating, gas and diarrhea after lactose exposure.

Distinguish: An enzyme deficiency, unlike milk-protein allergy.

[S] [N] Treatment: Adjust lactose intake or use lactase while maintaining nutritional adequacy.

[S] [N] Prevention: Often not preventable; symptom prevention depends on tolerated intake and enzyme support.

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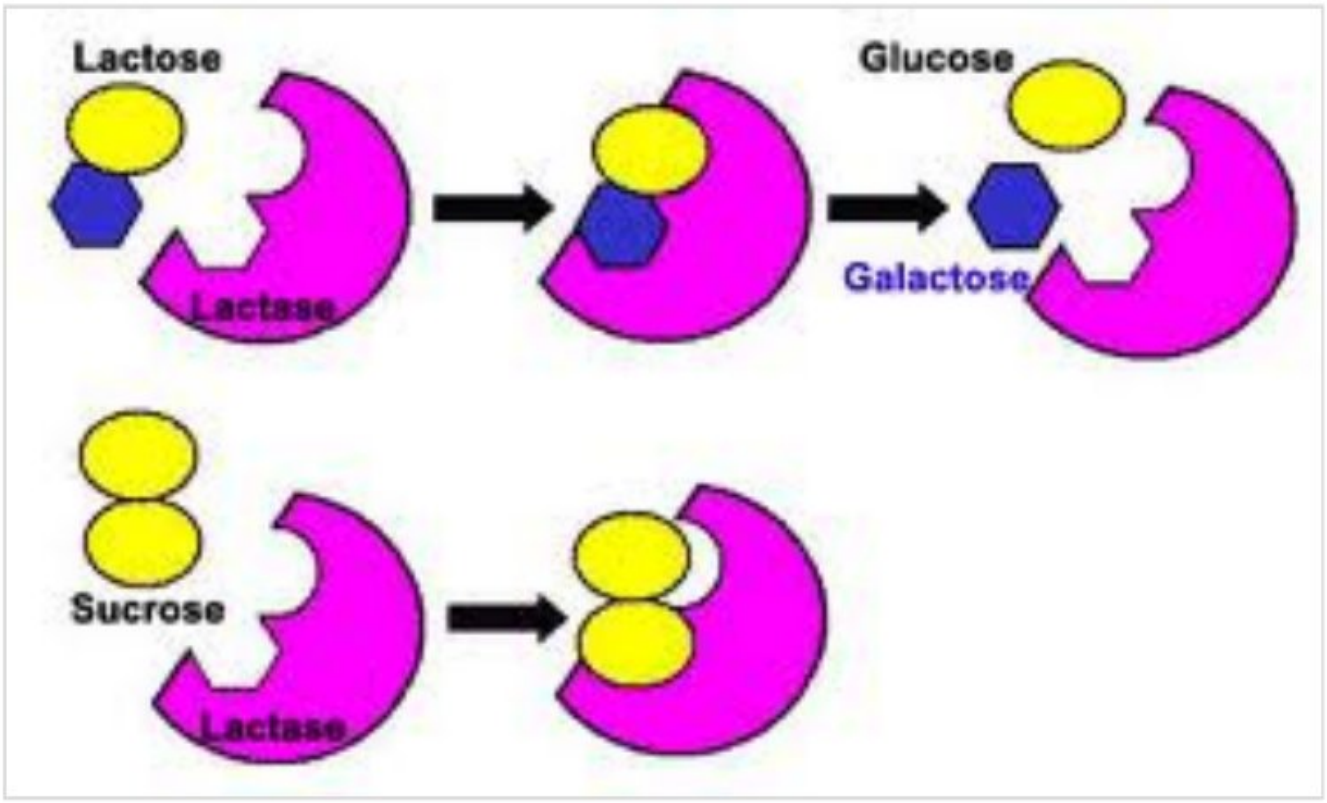
[Core] Lactase hydrolysis mechanism



Follow enzyme-substrate binding and splitting of lactose into glucose plus galactose. Low lactase leaves lactose to retain water and undergo bacterial fermentation. The lower panel contrasts sucrose: lactase does not hydrolyze sucrose. That illustrates enzyme specificity.

WD p.11 | Original labels retained; figure region enlarged.

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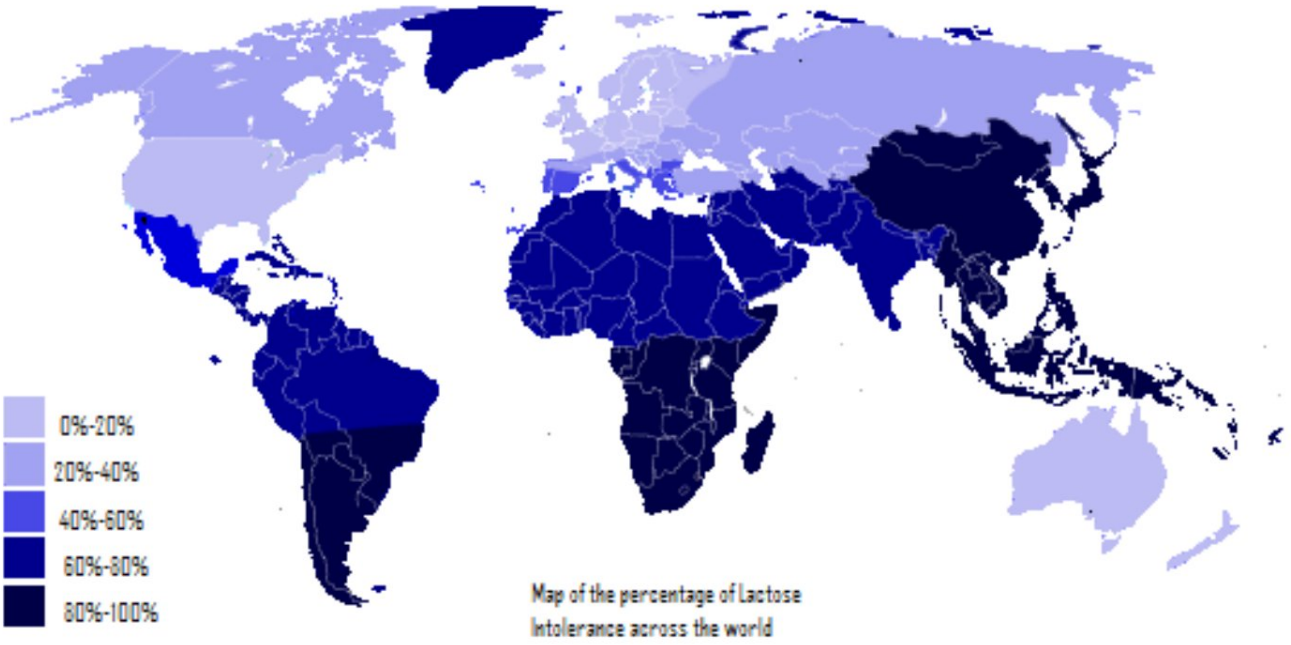
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[E] enrichment Historical lactose-malabsorption distribution



Use only to recognize population variation. Do not infer an individual's symptoms from ancestry or memorize these unsourced historical percentages.

WD p.11 | Original labels retained; figure region enlarged.

Hepatitis

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Liver inflammation can result from viruses, toxins, alcohol, autoimmunity or other injury.

Recognition clues: Variable symptoms; jaundice can reflect impaired bilirubin handling.

Distinguish: Hepatitis is inflammation, not automatically cirrhosis or cancer.

[S] [N] Treatment: Treat the cause: selected antivirals, removal of harmful exposure or immune-directed therapy as appropriate.

[S] [N] Prevention: Vaccines for hepatitis A and B; safe food/water and avoidance of blood-borne exposure according to the virus. No routine hepatitis C vaccine.

Appendicitis

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Inflammation of the appendix, sometimes following luminal obstruction, can progress to perforation.

Recognition clues: Typical evolving abdominal pain; location and signs vary.

Distinguish: A localized acute problem distinct from chronic inflammatory bowel disease.

[S] [N] Treatment: Prompt clinical evaluation; appendectomy is common, with antibiotics used according to the clinical situation.

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[S] [N] Prevention: No reliable universal prevention; early recognition reduces complications.

Diverticular disease

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Pouches form in the colonic wall (diverticulosis); inflammation of pouches is diverticulitis.

Recognition clues: Many pouches are asymptomatic; inflammation can cause localized pain and fever.

Distinguish: Diverticulosis does not necessarily mean active infection.

[S] [N] Treatment: Management depends on symptoms/severity; dietary measures, selected antibiotics, drainage or surgery in appropriate cases.

[S] [N] Prevention: Healthy bowel habits and fiber as appropriate; avoid presenting one diet as suitable during every acute episode.

GERD

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Reflux of stomach contents injures or irritates esophagus, often with impaired antireflux barrier function.

Recognition clues: Heartburn and regurgitation; chronic injury may alter epithelium.

Distinguish: Esophageal reflux is not a gastric ulcer.

[S] [N] Treatment: Behavioral measures and acid-suppressing medicines; selected cases receive procedures/surgery.

[S] [N] Prevention: Address individual triggers, meal timing and modifiable risks; not all reflux can be prevented.

Ulcerative colitis

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Chronic immune-mediated inflammation involves colon/rectum, typically continuous and mainly mucosal.

Recognition clues: Bloody diarrhea; long-term inflammation can increase colorectal cancer risk.

Distinguish: Compare Crohn: continuous colon disease versus patchy disease potentially anywhere in GI tract.

[S] [N] Treatment: Anti-inflammatory/immune-modifying drugs and biologics; colectomy for selected severe disease.

[S] [N] Prevention: No established universal primary prevention; monitoring and treatment reduce complications.

Crohn's disease

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Immune-mediated, often patchy transmural inflammation can affect any GI region.

Recognition clues: Skip lesions, strictures and fistulas can occur; malabsorption depends on location.

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Distinguish: Transmural disease helps explain fistulas; not limited to colonic mucosa.

[S] [N] Treatment: Immune-modifying/biologic therapy, nutritional support and surgery for complications; surgery does not guarantee cure.

[S] [N] Prevention: No guaranteed prevention; avoiding smoking and maintaining care can reduce adverse outcomes.

Celiac disease

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Gluten-triggered immune injury damages small-intestinal villi in susceptible people.

Recognition clues: Malabsorption, deficiencies and variable GI or extraintestinal symptoms.

Distinguish: Autoimmune enteropathy, not lactase deficiency or simply wheat allergy.

[S] [N] Treatment: Strict gluten-free diet with nutritional assessment and follow-up.

[S] [N] Prevention: No established universal prevention; avoiding gluten after diagnosis prevents ongoing trigger exposure.

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IMMUNE

I-ORG | Immune organs and histology

[Core] | Rule 3c.i

Red bone marrow produces blood cells and supports B-cell maturation. T-cell precursors originate in marrow but mature in the thymus. These are primary lymphoid organs. Lymph nodes, spleen and mucosa-associated lymphoid tissue are secondary sites where immune cells encounter antigen and organize responses.

Lymph nodes filter lymph from tissues. Their cortex contains B-cell follicles, the paracortex is rich in T cells, and medullary cords/sinuses lie deeper. Spleen filters blood: white pulp supports immune responses, while red pulp removes aged/damaged red blood cells. Thymus contains darker cortex and lighter medulla in lobules; Hassall corpuscles are a useful medullary clue. Do not identify a thymus by B-cell follicles; normal thymus does not organize them like a lymph node.

Skin is a physical barrier with keratinized stratified squamous epidermis, antimicrobial secretions, immune cells and underlying connective tissue. MALT includes gut-associated tissue such as Peyer patches and tonsils. Marrow sections contain mixed developing blood cells, adipocytes and large megakaryocytes. Yellow marrow is largely fatty; the older handout's wording should not replace red marrow as the principal hematopoietic tissue.

Recall: Which organ filters blood and which filters lymph?

Spleen filters blood; lymph nodes filter lymph.

Common trap: B cells mature in bone marrow; T cells mature in thymus, although both originate from marrow precursors.

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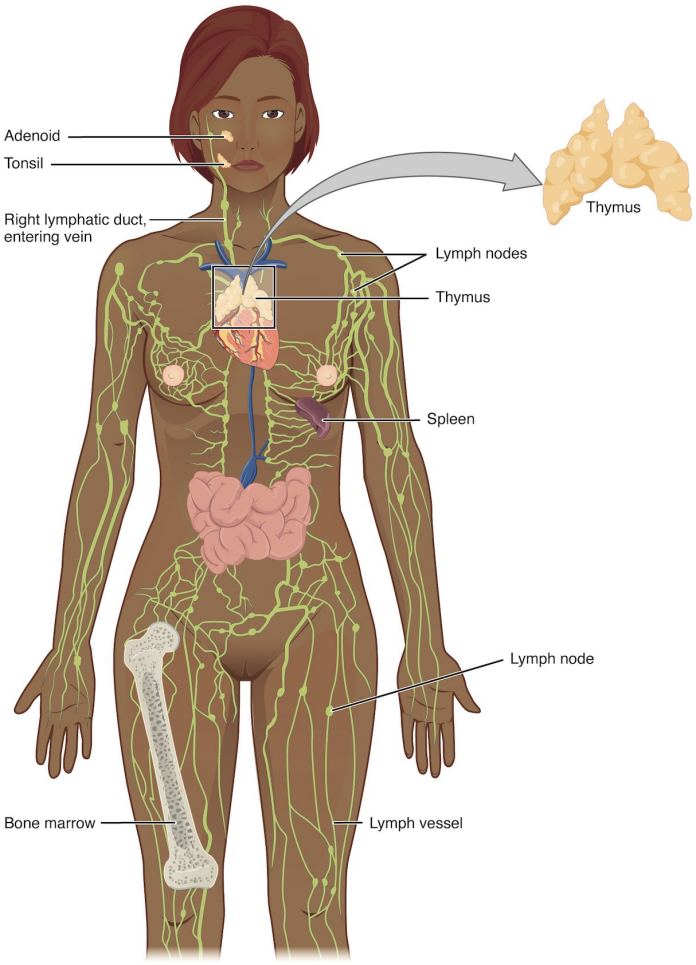
[Core] Anatomy of the Lymphatic System

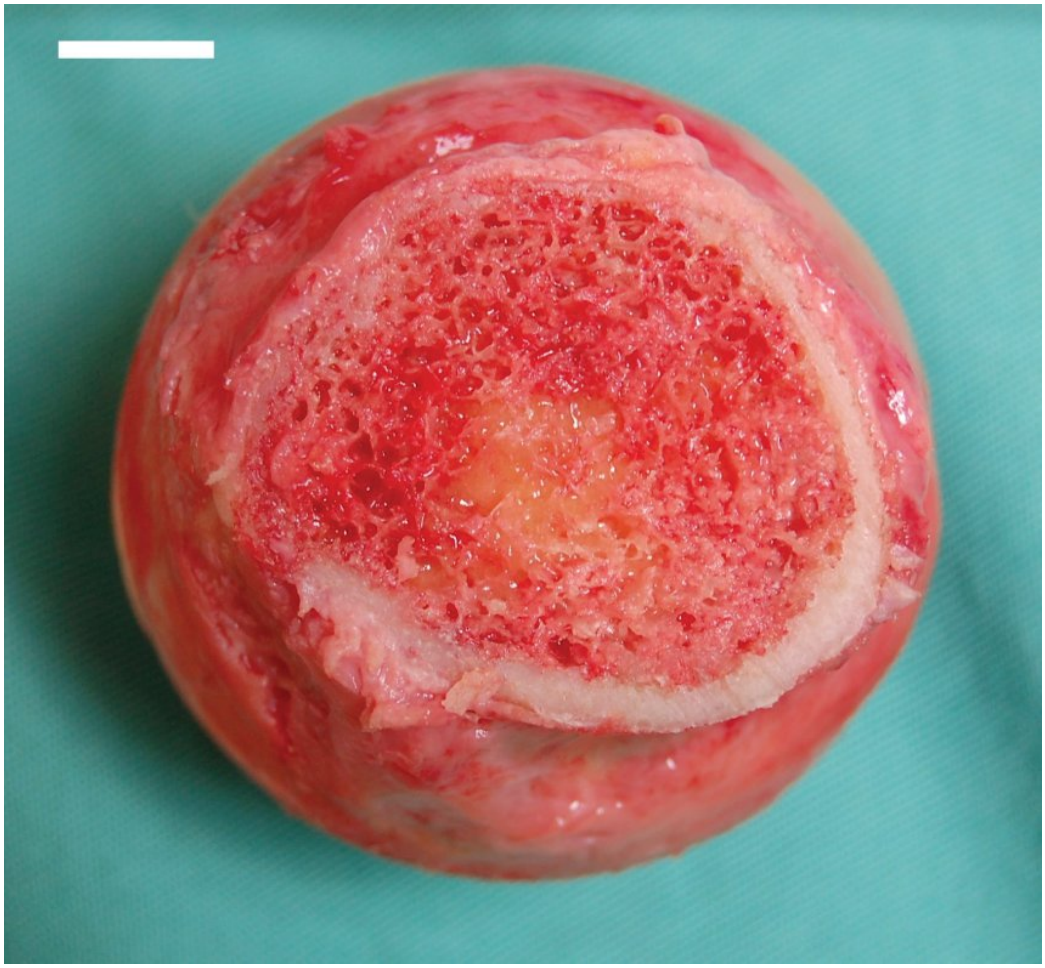


Locate marrow, thymus, spleen and lymph nodes. Pair each with development, maturation or surveillance functions.

OpenStax A&P 2e, Fig. 21.2 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-1-anatomy-of-the-lymphatic-and-immune-systems | Publisher original; 1200 x 1678 pixels

Access for free at https://openstax.org/books/anatomy-and-physiology-2e/pages/1-introduction
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Bone Marrow



Connect marrow spaces to blood-cell production. Red marrow is hematopoietic; yellow marrow is predominantly fatty.

OpenStax A&P 2e, Fig. 21.6 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-1-anatomy-of-the-lymphatic-and-immune-systems | Publisher original; 854 x 791 pixels

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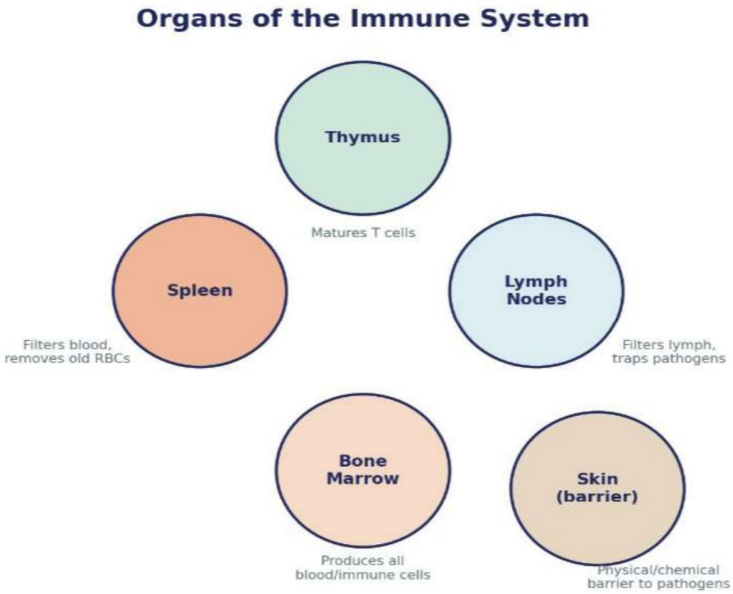
[Core] Immune organ functions



Bone marrow, thymus, spleen, lymph nodes, and skin

Connect marrow to blood-cell production, thymus to T-cell maturation, lymph nodes to lymph filtration, spleen to blood surveillance and skin to barrier defense.

S27 p.29 | Original labels retained; figure region enlarged.

Access for free at https://openstax.org/books/anatomy-and-physiology-2e/pages/1-introduction
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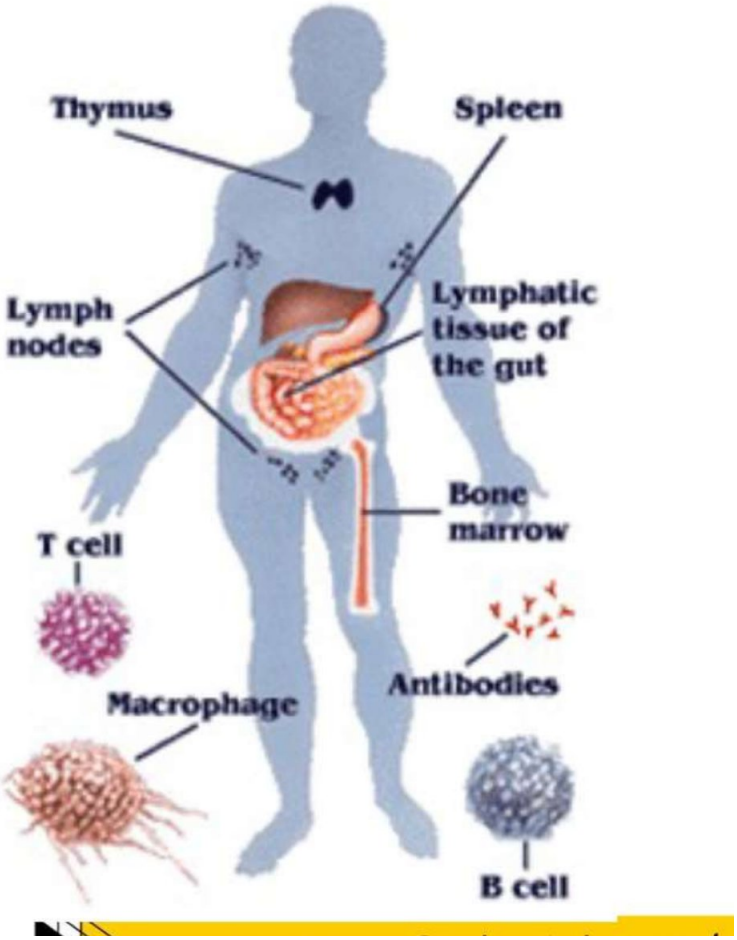
[Core] Immune cells and organs overview



Identify thymus, spleen, marrow and lymph nodes, then connect them to immune-cell development or surveillance. A duplicate appears on S23 page 46.

S23 p.44 | Original labels retained; figure region enlarged.

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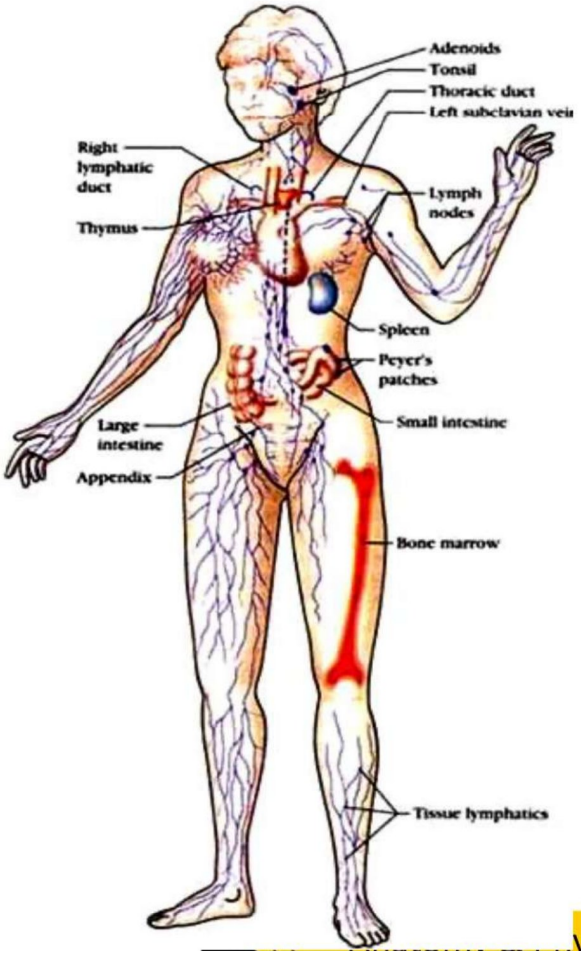
[Core] Lymphatic vessel network



Trace peripheral lymph vessels toward major ducts and venous return. Nodes filter lymph along the route. The spleen instead monitors blood.

S23 p.48 | Original labels retained; figure region enlarged.

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[Core] Lymphatic organ locations

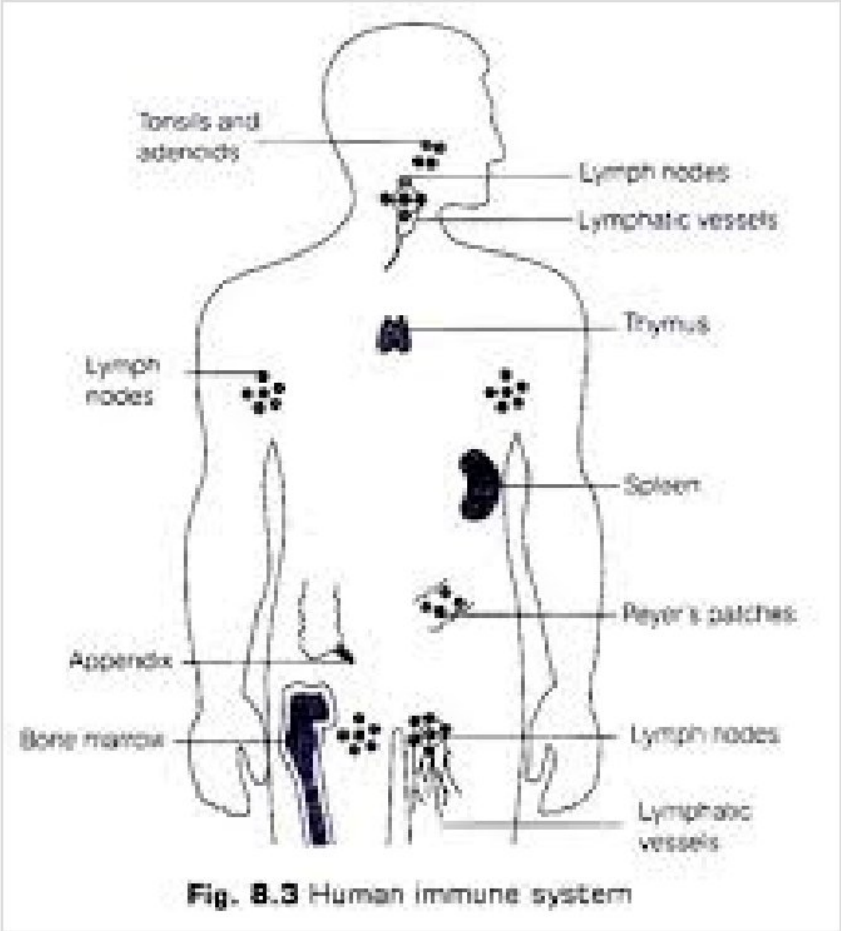

Fig. 8.3 Human immune system

Locate marrow, thymus, spleen and lymph nodes. Distinguish primary lymphoid organs where cells develop from secondary sites where responses are organized.

WI p.7 | Original labels retained; figure region enlarged.

I-INN | Innate barriers, receptors and immune cells

[Core] | Rule 3c.ii

Innate defenses act rapidly using inherited pattern-recognition mechanisms. Barriers include intact skin, mucus, cilia, tears/saliva, antimicrobial molecules, stomach acid and normal microbial communities. They reduce entry but are not impenetrable. Toll-like receptors recognize conserved microbial patterns (PAMPs) and participate in inflammatory signaling; innate sensing can also respond to damage signals.

Neutrophils are abundant rapid phagocytes, recognizable by multilobed nuclei. Monocytes circulate and can differentiate into macrophages; macrophages phagocytose, release signals and present antigen. Dendritic cells are important activators of naive T cells. Natural killer cells detect stress signals or reduced self-MHC I and can kill abnormal cells without prior antigen-specific sensitization. NK cells are lymphocytes but are innate, not adaptive T cells.

Eosinophils participate in parasite responses and allergy, often with bilobed nuclei and conspicuous granules. Basophils circulate, whereas mast cells reside in tissues and release mediators such as histamine. B and T lymphocytes can look similar on ordinary blood smears; appearance alone usually cannot distinguish their subsets. Innate immunity is not blind to microbial differences: it recognizes patterns rather than the highly

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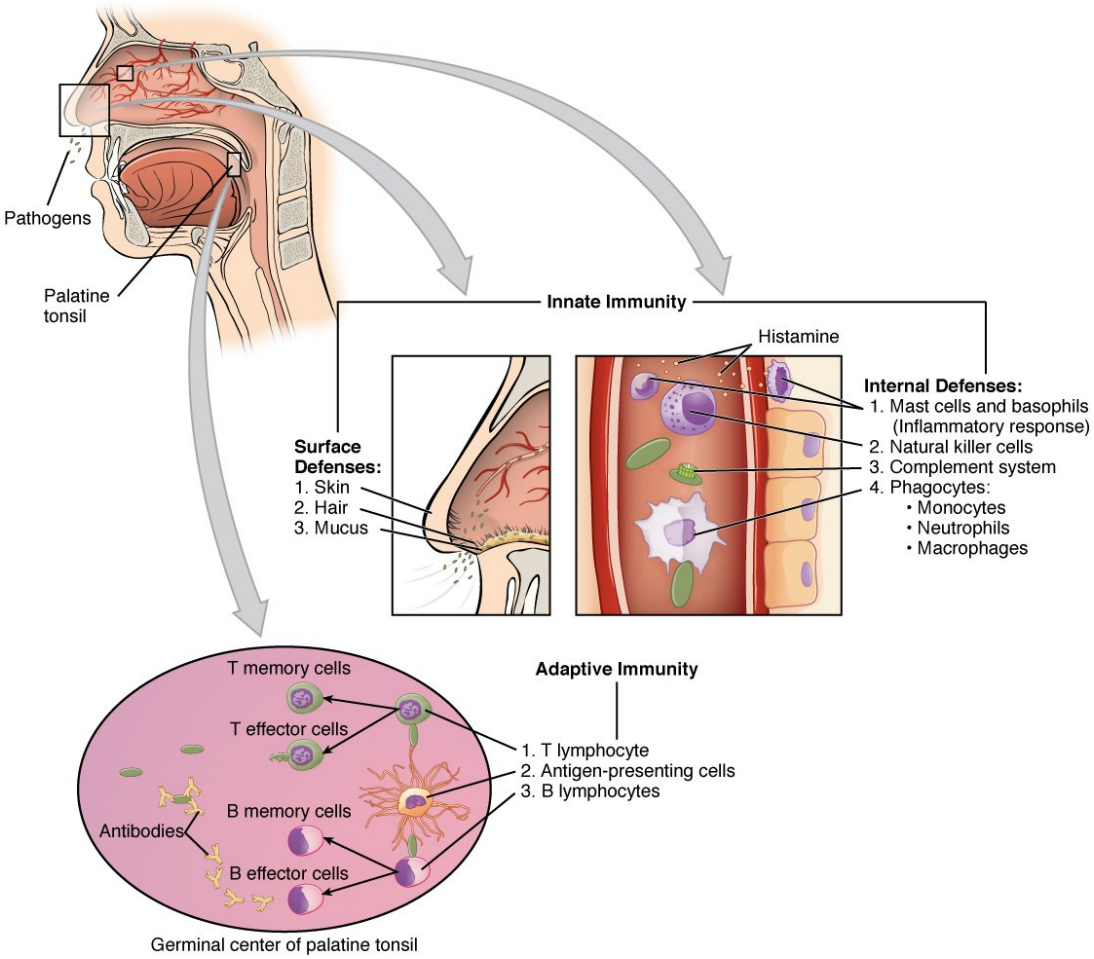
diverse rearranged antigen receptors of adaptive immunity.

Recall: Why is a natural killer cell classified as innate even though it is a lymphocyte?

It uses innate activating/inhibitory recognition rather than a rearranged antigen-specific T-cell receptor and prior clonal sensitization.

Common trap: Do not label every lymphocyte adaptive or every white cell a phagocyte.

[Core] Cooperation between Innate and Adaptive Immune Responses



Follow communication between innate recognition and antigen-specific lymphocytes. Neither branch works completely alone.

OpenStax A&P 2e, Fig. 21.12 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-2-barrier-defenses-and-the-innate-immune-response | Publisher original; 1119 x 986 pixels

Access for free at https://openstax.org/books/anatomy-and-physiology-2e/pages/1-introduction
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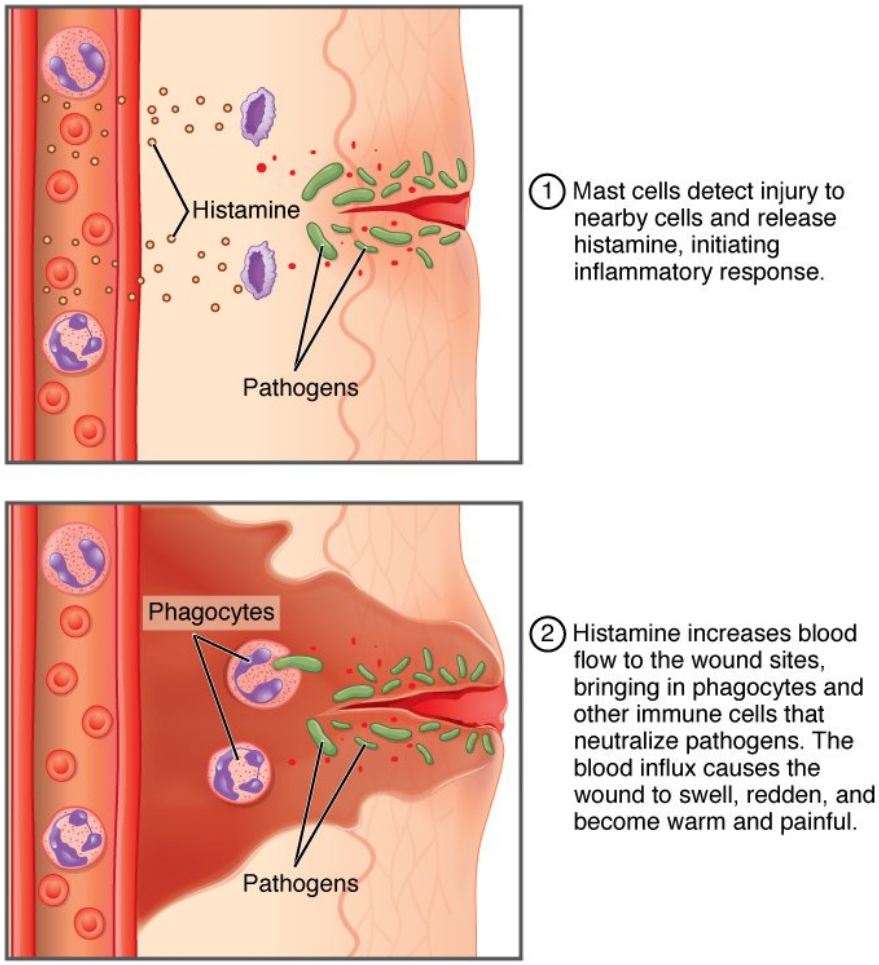
[Core] The inflammatory response



Trace tissue injury to mediators, vascular changes and recruited phagocytes. Vasodilation and increased permeability explain different visible signs.

OpenStax A&P 2e, Fig. 21.14 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-2-barrier-defenses-and-the-innate-immune-response | Publisher original; 675 x 743 pixels

Access for free at https://openstax.org/books/anatomy-and-physiology-2e/pages/1-introduction
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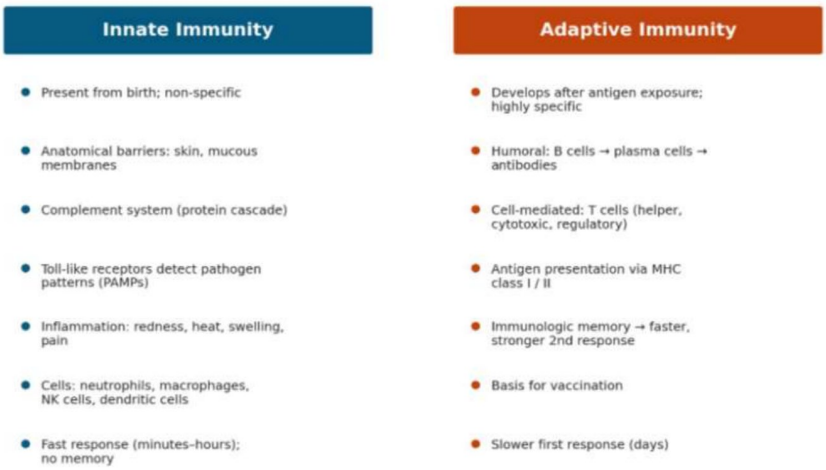
[Core] Innate versus adaptive defense



Innate vs. adaptive immunity, side by side

Innate responses rapidly recognize shared patterns. Adaptive responses select antigen-specific lymphocyte clones and form memory. The two branches cooperate rather than acting as isolated systems.

S27 p.30 | Original labels retained; figure region enlarged.

Access for free at https://openstax.org/books/anatomy-and-physiology-2e/pages/1-introduction
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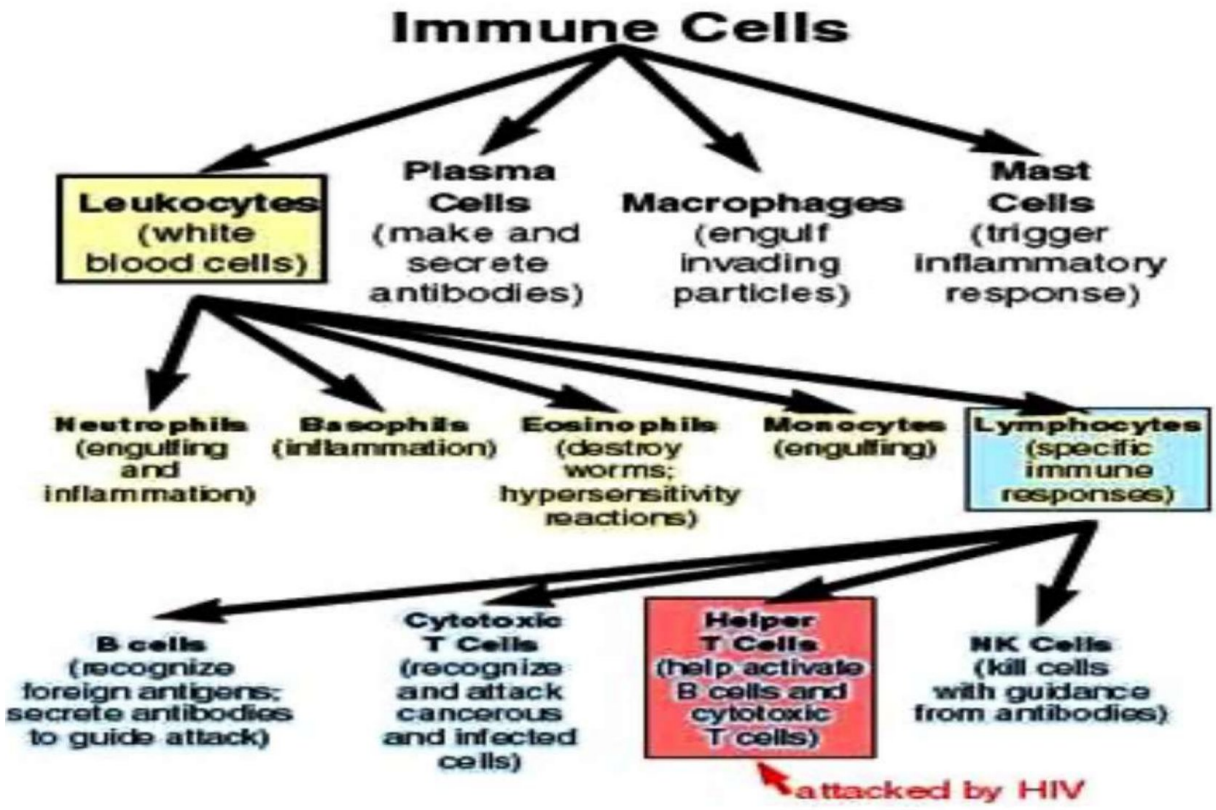
[Core] Immune-cell relationships



Separate lymphocytes from granulocytes and monocyte-derived cells. Plasma cells are differentiated B cells. Do not use this simplified chart as a complete modern hematopoietic lineage tree.

S23 p.47 | Original labels retained; figure region enlarged.

Access for free at https://openstax.org/books/anatomy-and-physiology-2e/pages/1-introduction
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[Core] Visible inflammation



Redness and swelling can reflect vascular dilation and increased permeability. A photograph alone does not identify the cause or prove an infection.

S23 p.52 | Original labels retained; figure region enlarged.

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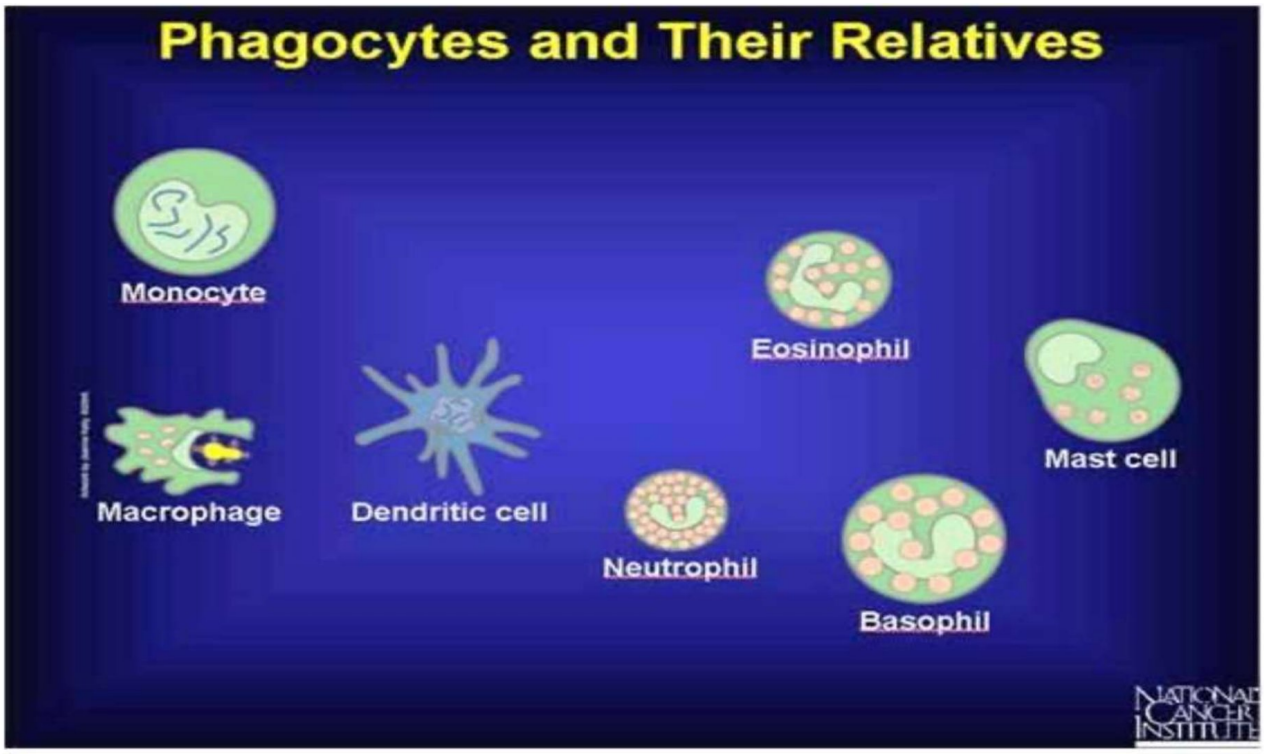
[Core] Innate immune-cell appearance



Compare a multilobed neutrophil, a large macrophage and a dendritic cell. Despite the slide title, not every cell shown is a professional phagocyte; mast cells and basophils chiefly release mediators.

S23 p.53 | Original labels retained; figure region enlarged.

Access for free at https://openstax.org/books/anatomy-and-physiology-2e/pages/1-introduction
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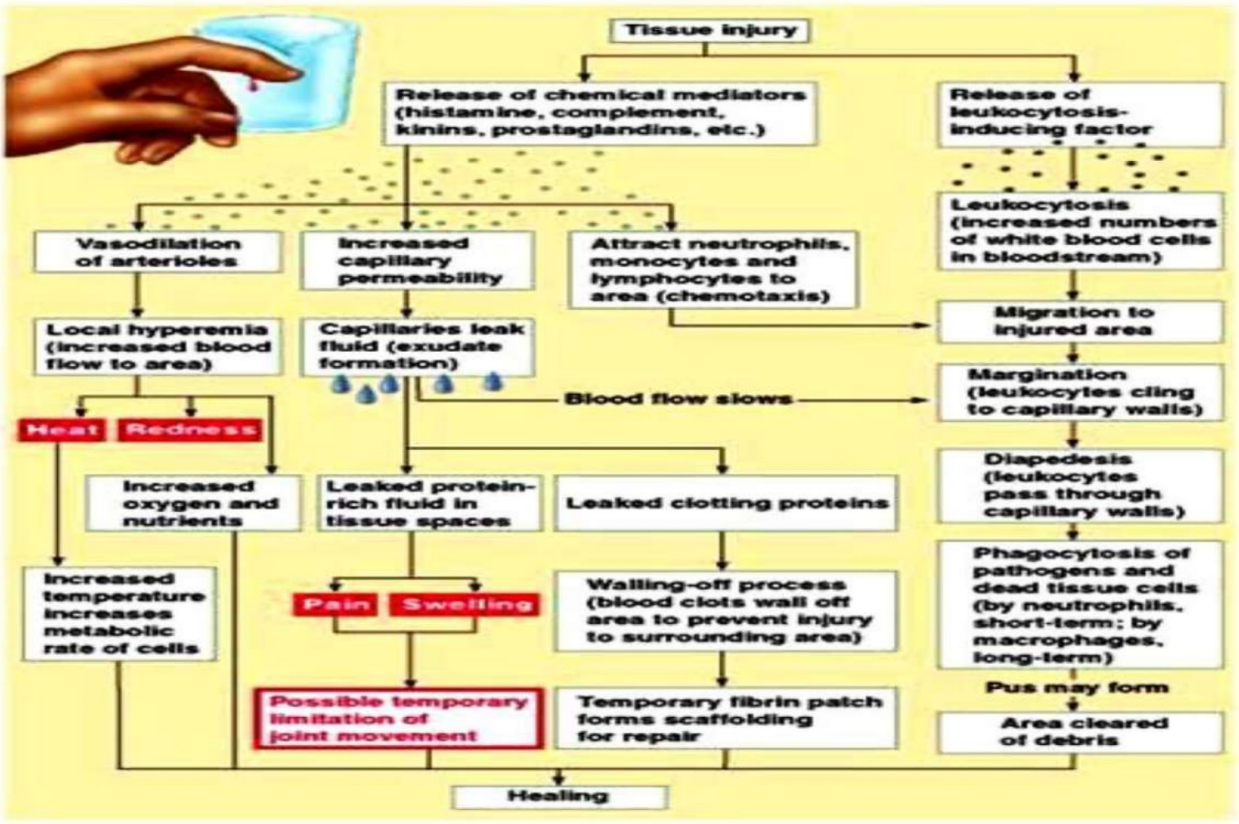
[Core] Inflammation cascade

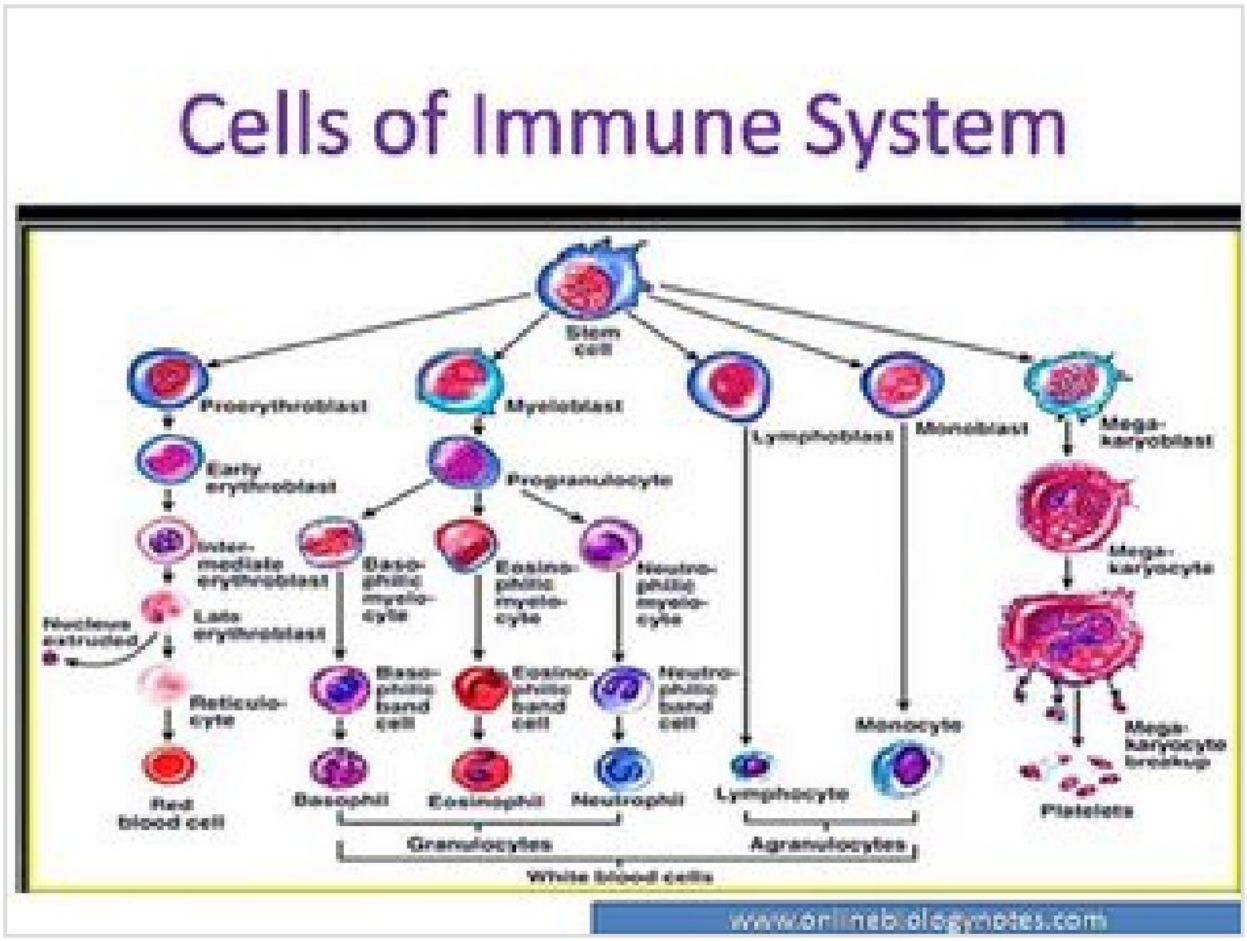


Trace injury recognition, mediator release, altered blood flow/permeability and recruitment of leukocytes. Use the clear textbook inflammation figure for small labels that remain blurred here.

S23 p.54 | Original labels retained; figure region enlarged.

Access for free at https://openstax.org/books/anatomy-and-physiology-2e/pages/1-introduction
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Immune-cell lineage overview



Separate myeloid and lymphoid branches. Relate neutrophils/monocytes to phagocytosis and lymphocytes to adaptive or NK-cell functions; not all lymphocytes are adaptive.

WI p.2 | Original labels retained; figure region enlarged.

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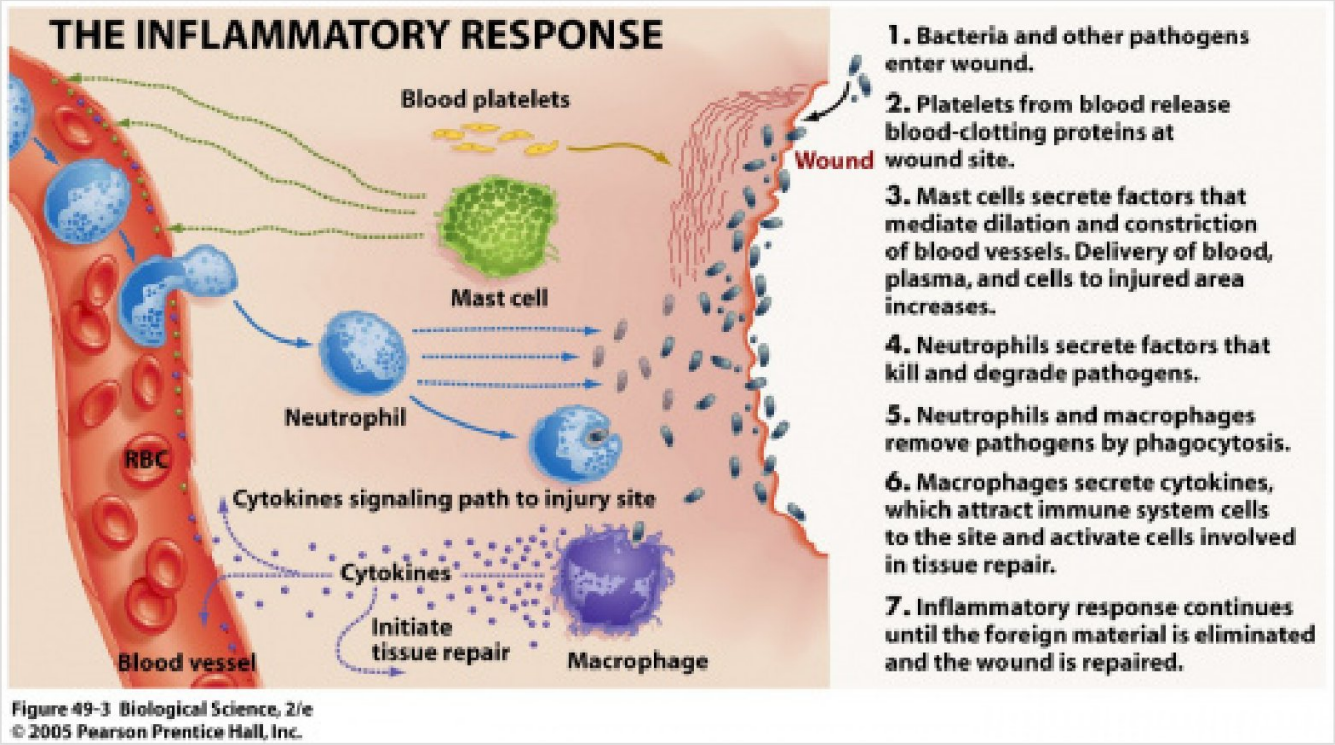
[Core] Local inflammatory response



Follow microbes at the injury site, mediator release, vascular changes and neutrophil recruitment. Dilation and permeability are separate processes.

WI p.4 | Original labels retained; figure region enlarged.

I-COMP | Complement and inflammation

[Core] | Rule 3c.ii

Complement is a protein cascade. Classical activation can be initiated by antibody-antigen complexes; lectin and alternative pathways can activate without antibody. The pathways converge on C3 activation. C3b coats targets for phagocytosis (opsonization); C3a and C5a promote inflammation, and C5a is a strong chemotactic signal. The terminal C5b-C9 membrane attack complex forms pores in susceptible targets.

Inflammation recruits defenses to damaged or infected tissue. Vasodilation increases blood flow, causing redness and warmth. Increased vascular permeability allows fluid/proteins into tissue, causing swelling; inflammatory mediators and tissue pressure contribute to pain. Leukocytes adhere to vessel walls, leave blood by diapedesis and follow chemical gradients by chemotaxis.

Phagocytosis involves recognition/attachment, engulfment, phagosome formation, fusion with lysosomes and killing/digestion. Opsonins improve recognition. Inflammation can occur without infection, and uncontrolled inflammation can damage host tissue. Complement helps innate defense while also collaborating with antibodies; it is not an antibody class.

Recall: Name one complement function each for C3b and C5b-C9.

C3b opsonizes; C5b-C9 forms the membrane attack complex.

Common trap: Redness is primarily increased blood flow; swelling is mainly fluid leaving permeable vessels.

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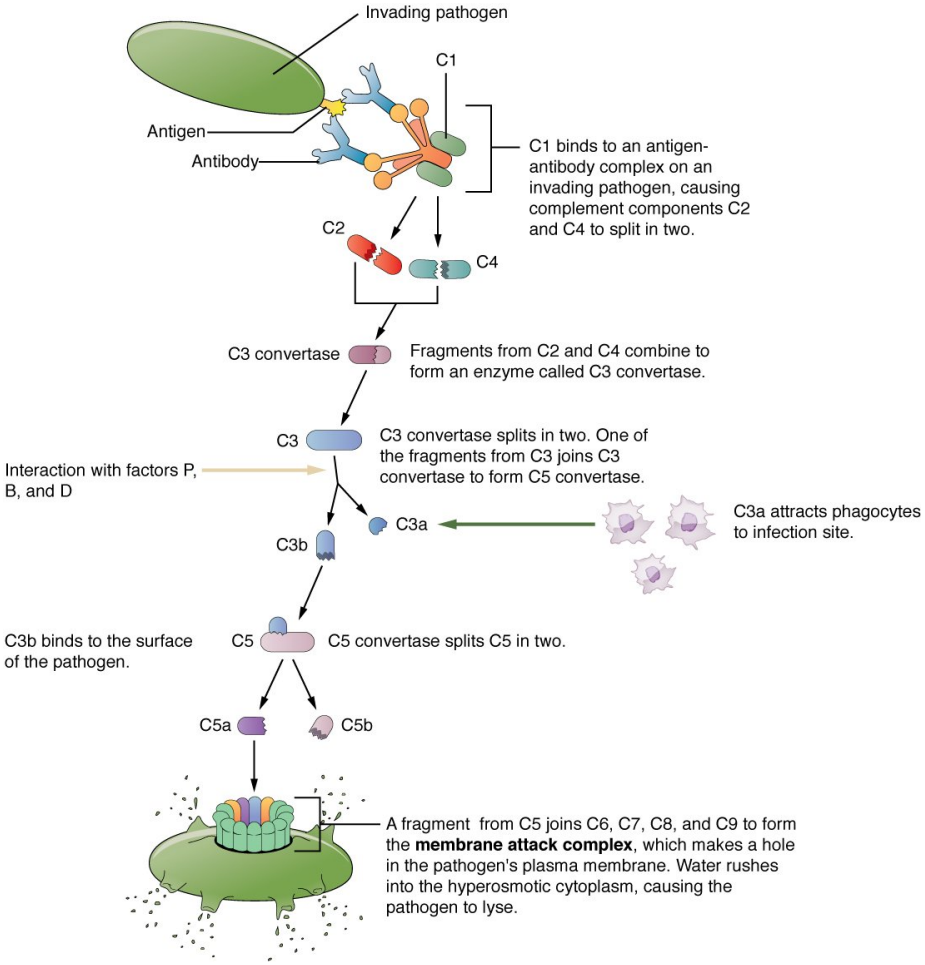
[Core] Complement Cascade and Function



Identify opsonization, inflammatory signaling and membrane attack as different complement outcomes. This is a classical-pathway overview, not every step of all three pathways.

OpenStax A&P 2e, Fig. 21.13 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-2-barrier-defenses-and-the-innate-immune-response | Publisher original; 1102 x 1154 pixels

I-ADAPT | Adaptive immunity, antigen presentation and tolerance

[Core] | Rule 3c.iii

Adaptive immunity uses antigen-specific B and T cells, clonal expansion and memory. An antigen is a structure recognized by an immune receptor; an epitope is the specific recognized part. Antigens can be foreign or self, and not all are proteins. Loss of self-tolerance can cause autoimmunity.

B-cell receptors can bind native antigen. Activated B cells can become plasma cells that secrete antibody and memory B cells. Helper T cells provide signals supporting many B-cell responses, macrophage activation and other immune functions. Cytotoxic CD8 T cells kill infected or abnormal host cells; regulatory T cells help restrain responses. Killing a host cell is different from secreting antibody into fluid.

T cells recognize peptide presented on MHC. MHC I on most nucleated cells presents to CD8 T cells; MHC II on professional antigen-presenting cells presents to CD4 helper T cells. Dendritic cells, macrophages and B cells are major professional APCs. Activation of naive T cells requires appropriate recognition and additional

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costimulatory signals; recognition alone is not always enough. Positive/negative selection and peripheral regulation reduce harmful self-reactivity.

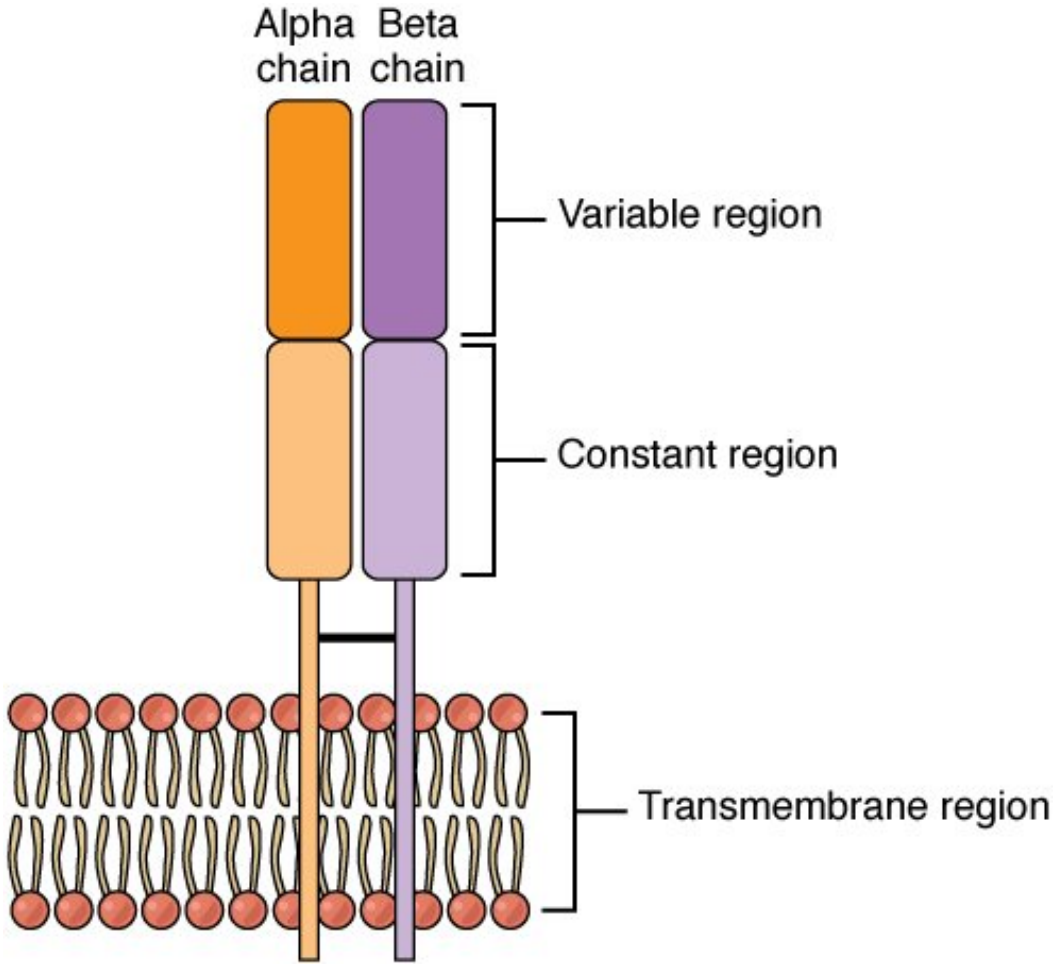
Clonal selection selects lymphocytes with pre-existing receptors suited to an antigen; exposure does not teach a cell to invent the matching receptor. For most protein antigens, effective B-cell responses also involve helper-T-cell signals. MHC I presents peptides to CD8 T cells; MHC II presents peptides to CD4 T cells. Mature red blood cells lack the usual MHC I expression of nucleated cells.

Recall: Pair MHC I and MHC II with the responding T-cell populations.

MHC I -> CD8 cytotoxic T cells; MHC II -> CD4 helper T cells.

Common trap: Antibodies bind antigen; T-cell receptors typically recognize peptide-MHC complexes.

[Core] Alpha-beta T Cell Receptor



Identify the T-cell receptor that recognizes peptide-MHC, rather than free native antigen alone.

OpenStax A&P 2e, Fig. 21.15 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-3-the-adaptive-immune-response-t-lymphocytes-and-their-functional-types | Publisher original; 494 x 452 pixels

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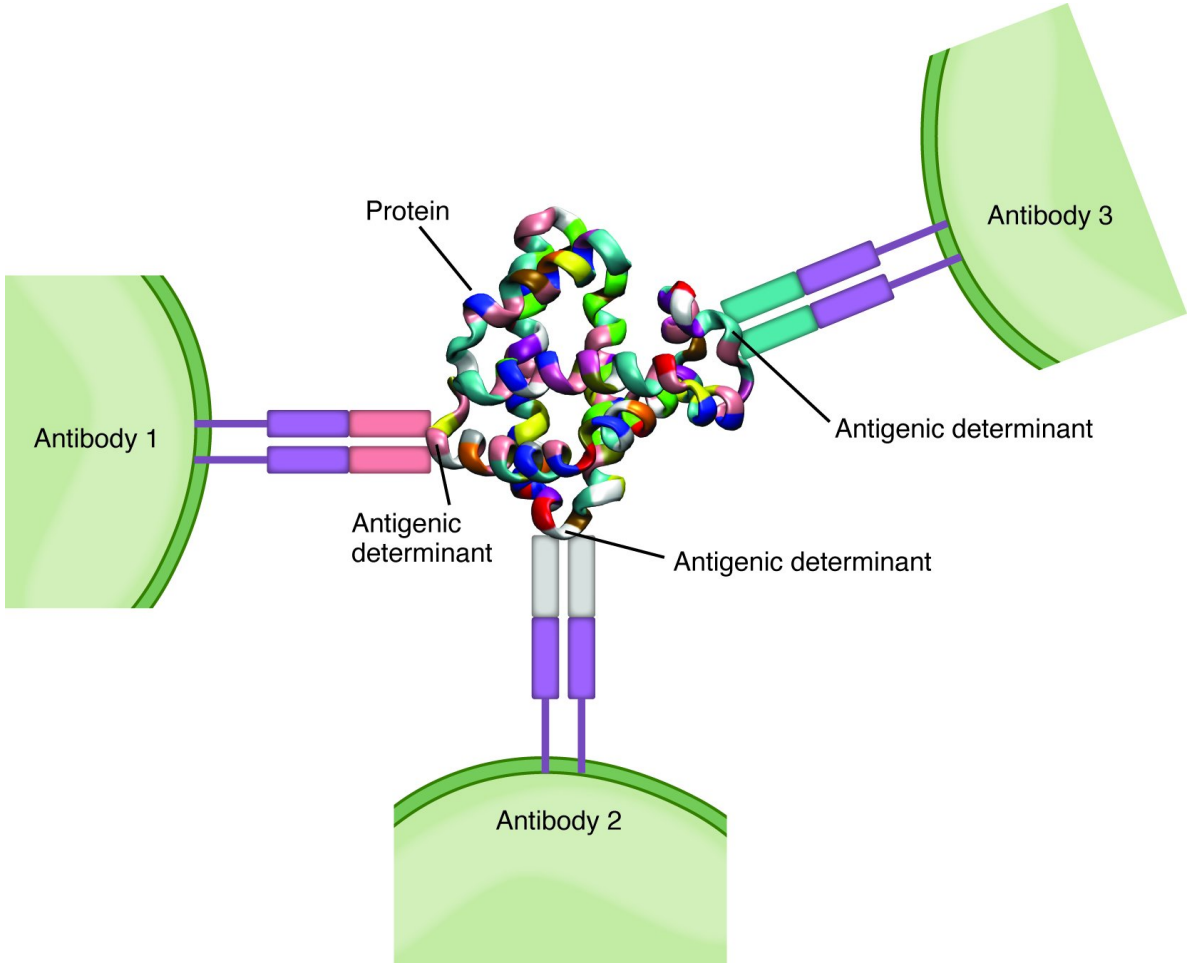
[Core] Antigenic Determinants



Distinguish one antigen from its multiple epitopes. Different clones may recognize different sites on the same antigen.

OpenStax A&P 2e, Fig. 21.16 |
https://openstax.org/books/anatomy-and-physiology-2e/pages/21-3-the-adaptive-immune-response-t-lymphocytes-and-their-functional-types |
Publisher original; 1658 x 1347 pixels

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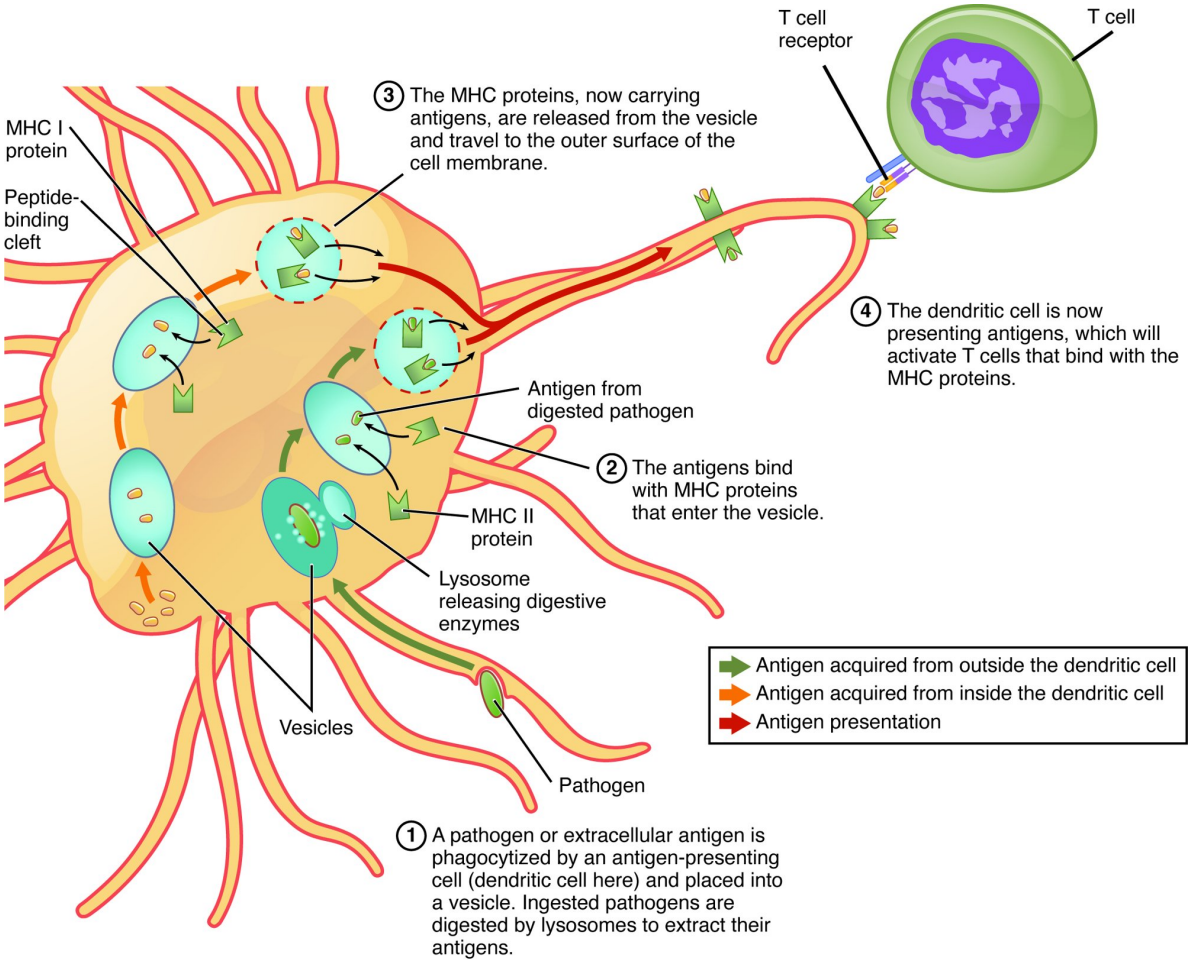
[Core] Antigen Processing and Presentation



Compare intracellular versus engulfed antigen processing. Match MHC I to CD8 and MHC II to CD4.

OpenStax A&P 2e, Fig. 21.17 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-3-the-adaptive-immune-response-t-lymphocytes-and-their-functional-types | Publisher original; 2193 x 1780 pixels

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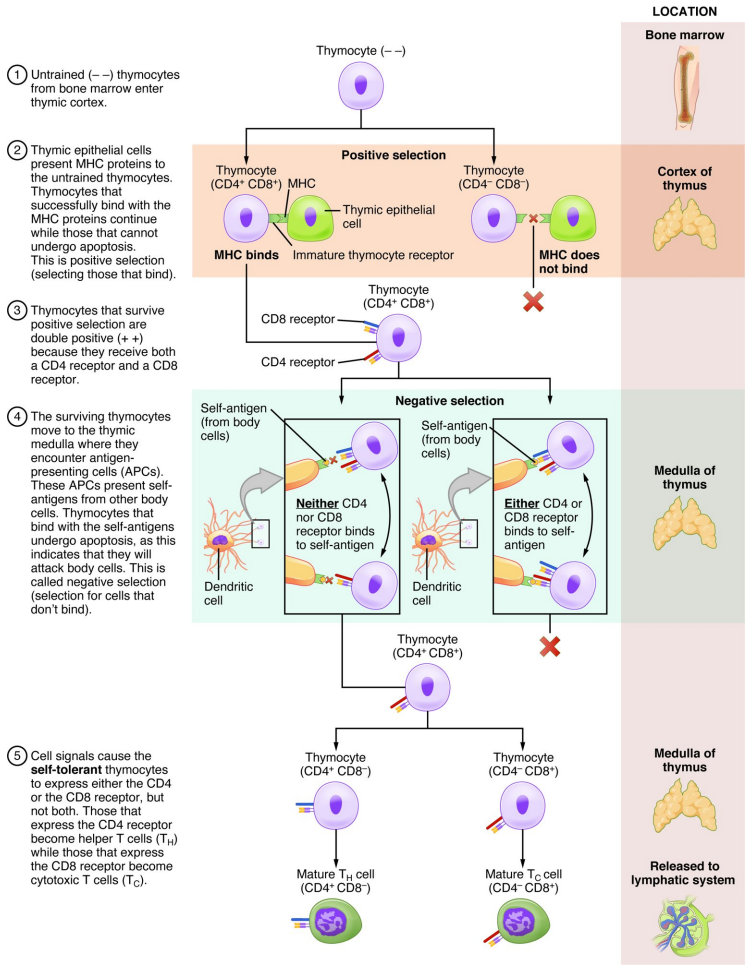
[Core] Differentiation of T Cells within the Thymus



Trace T-cell development and selection. Selection promotes useful recognition while reducing dangerous self-reactivity.

OpenStax A&P 2e, Fig. 21.18 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-3-the-adaptive-immune-response-t-lymphocytes-and-their-functional-types | Publisher original; 2230 x 2903 pixels

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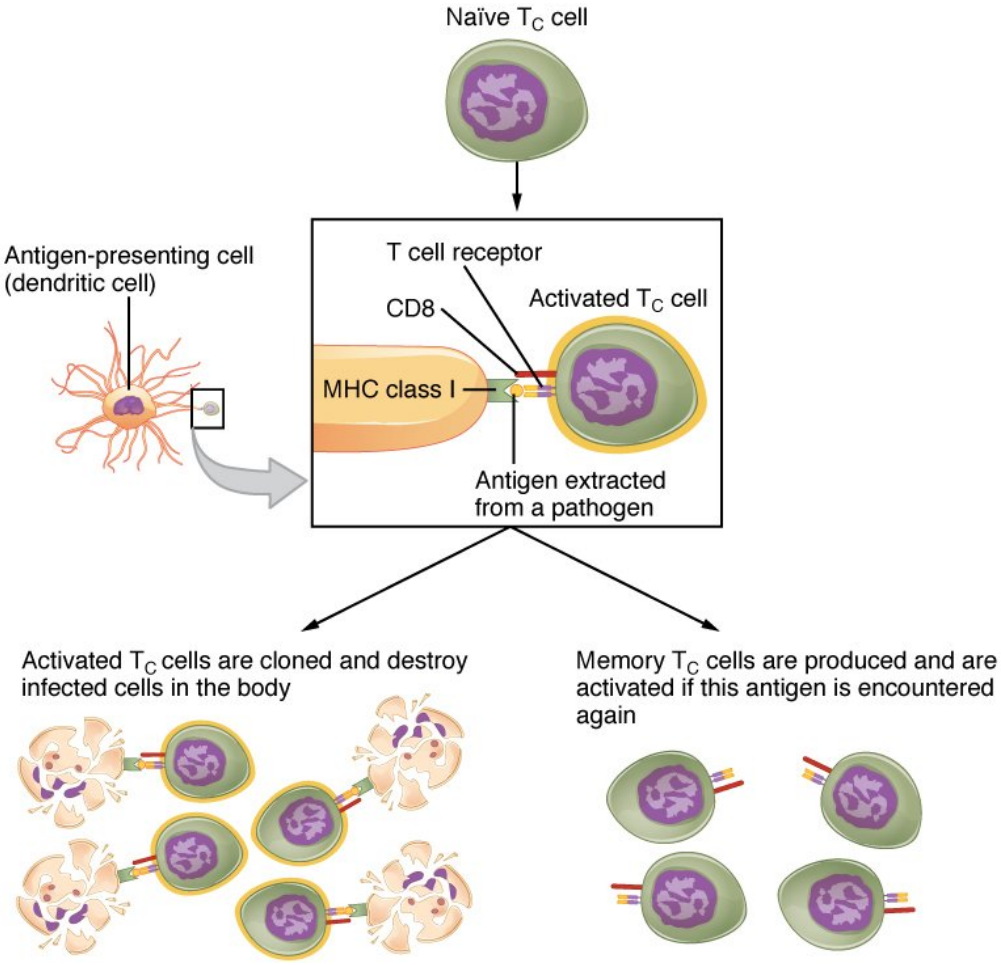
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Clonal Selection and Expansion of T Lymphocytes



Follow selection of a pre-existing specific clone, then proliferation and differentiation. The antigen does not invent the receptor.

OpenStax A&P 2e, Fig. 21.19 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-3-the-adaptive-immune-response-t-lymphocytes-and-their-functional-types | Publisher original; 764 x 733 pixels

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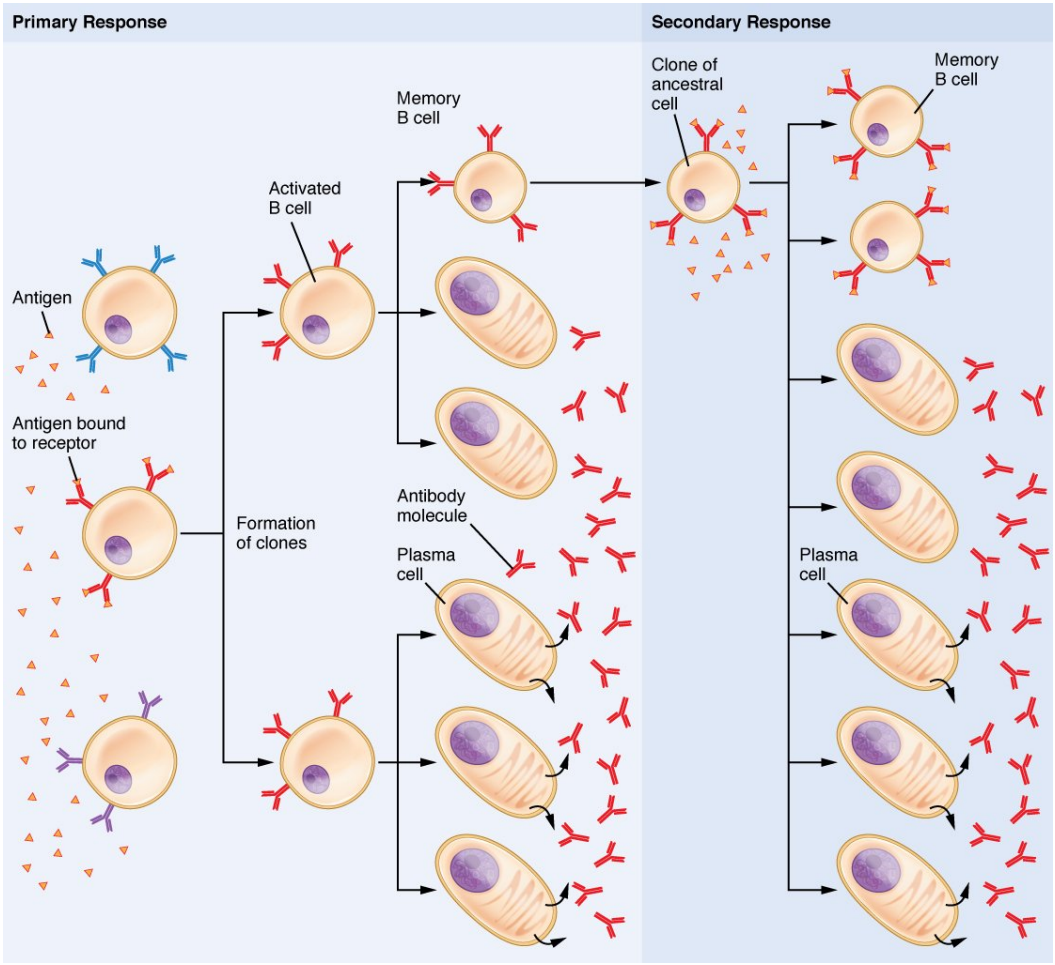
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Clonal Selection of B Cells



Trace antigen-selected B cells to antibody-secreting plasma cells and memory cells.

OpenStax A&P 2e, Fig. 21.23 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-4-the-adaptive-immune-response-b-lymphocytes-and-antibodies | Publisher original; 1117 x 1023 pixels

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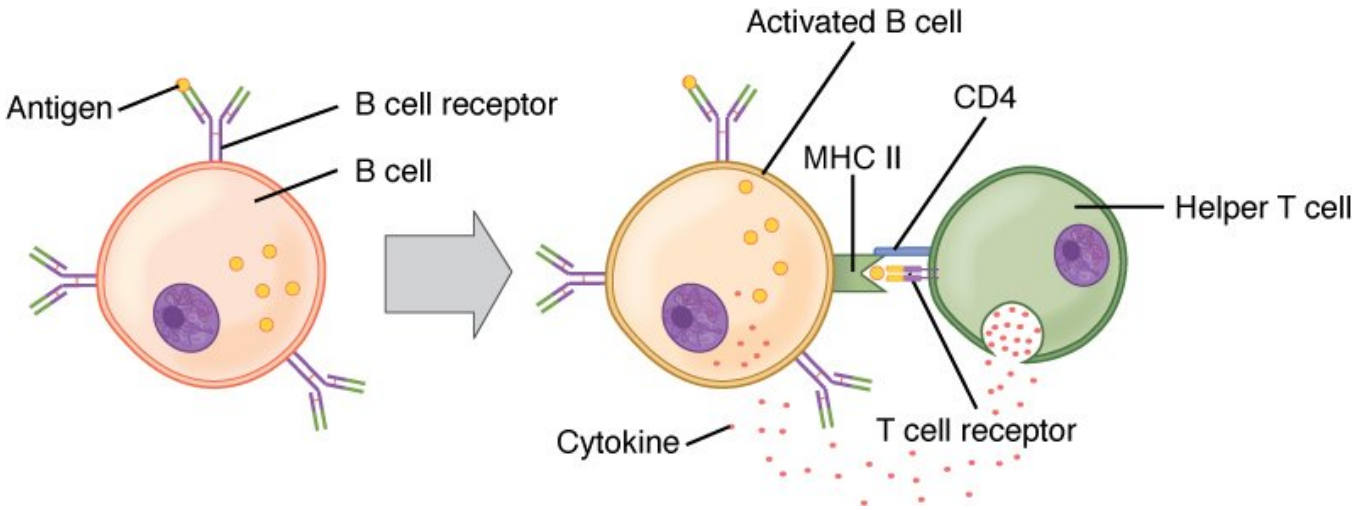
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] T and B Cell Binding



Follow B-cell antigen uptake and presentation to a helper T cell. Distinguish B-cell native-antigen recognition from T-cell peptide-MHC recognition.

OpenStax A&P 2e, Fig. 21.25 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-4-the-adaptive-immune-response-b-lymphocytes-and-antibodies | Publisher original; 796 x 294 pixels

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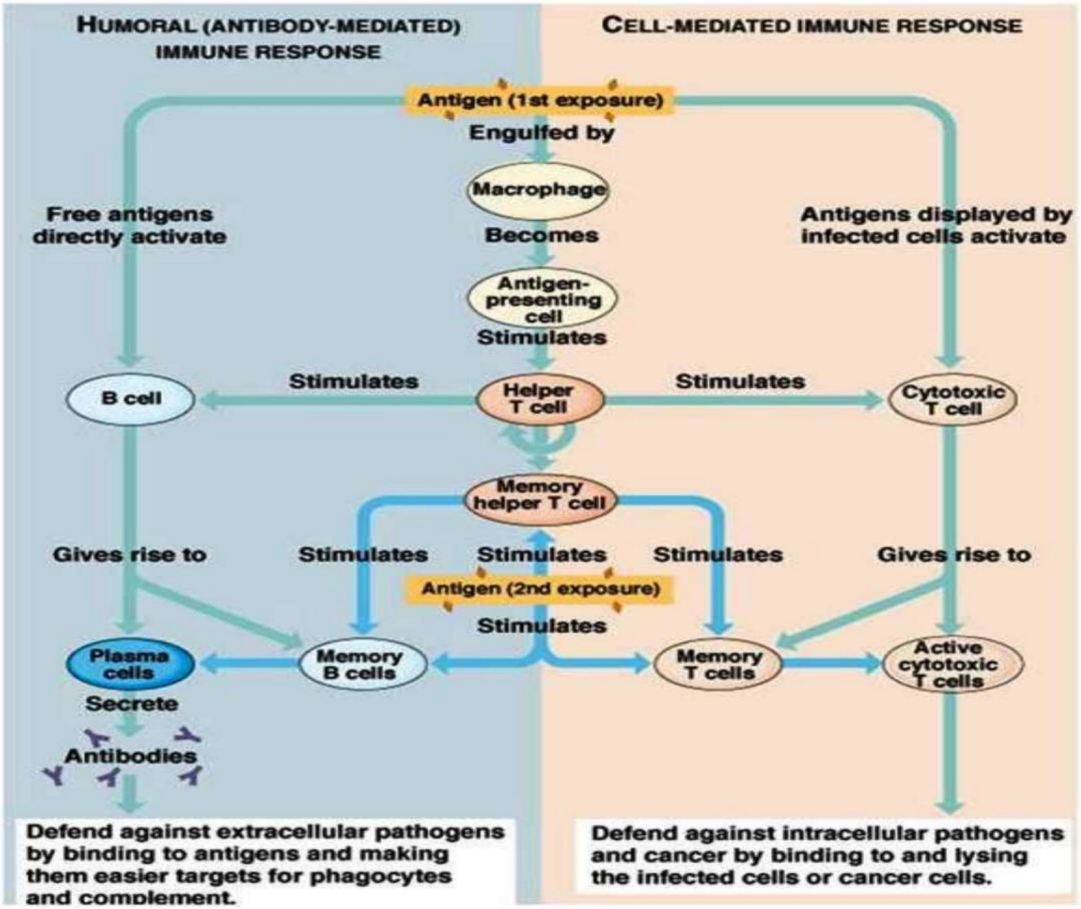
[Core] Humoral and cell-mediated response pathways



Compare B-cell/plasma-cell antibody production with cytotoxic T-cell killing. Helper T-cell signals coordinate both branches. Antigen selection expands pre-existing specific clones.

S23 p.57 | Original labels retained; figure region enlarged.

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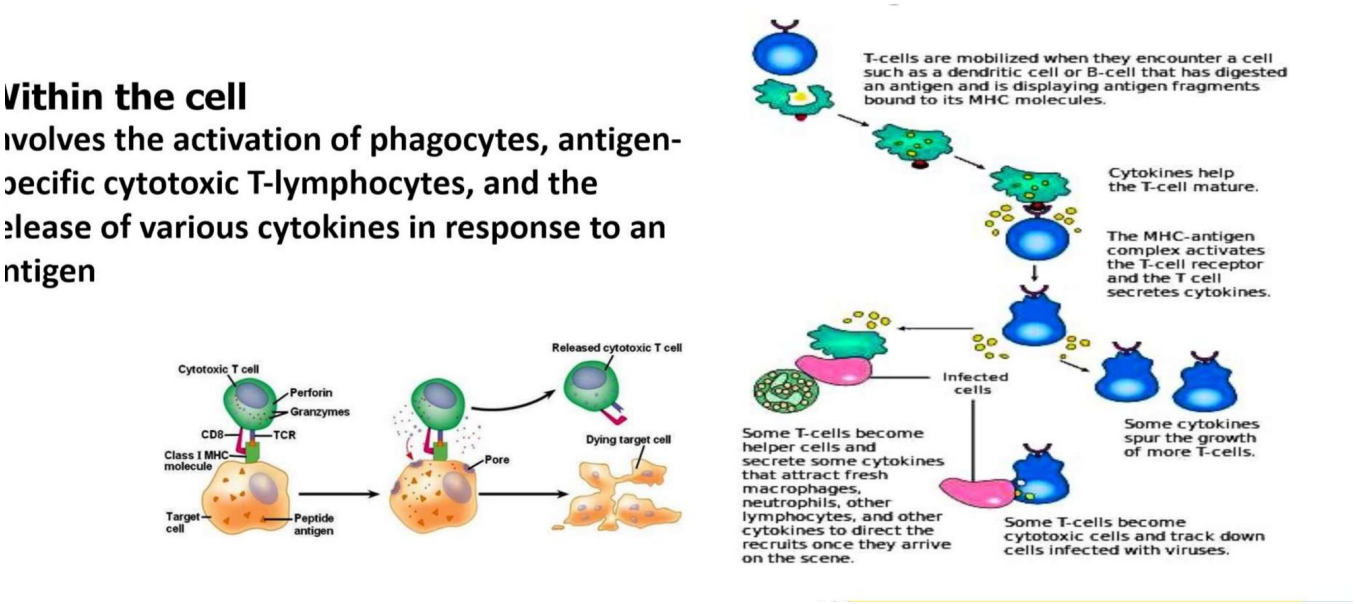
[Core] Cytotoxic T-cell response



Trace antigen recognition, activation, clonal expansion and target-cell killing. CD8 T cells recognize peptide-MHC I; CD4 helper cells generally recognize peptide-MHC II.

S23 p.62 | Original labels retained; figure region enlarged.

I-AB | Antibodies and sources of immunity

[Core] | Rule 3c.iii,iv

Antibodies contain two heavy and two light chains. Variable regions bind epitopes; constant regions help determine effector functions. Antibodies can neutralize toxins/viruses, agglutinate targets, opsonize and activate complement. They do not ingest pathogens themselves.

IgM is often the first major secreted class in a primary response; secreted IgM is typically pentameric, whereas membrane IgM is monomeric. IgG is prominent in blood and secondary responses and can cross the placenta. Secretory IgA is usually dimeric and protects mucosal surfaces, tears, saliva and breast milk; serum IgA is mainly monomeric. IgE binds mast cells/basophils and is important in immediate allergy and parasite defense. IgD mainly functions as a receptor on naive B cells.

Active immunity results from the recipient's own response, after infection or vaccination, and can form memory. Passive immunity transfers ready-made antibodies, such as maternal IgG, breast-milk IgA or therapeutic immunoglobulin; protection is rapid but fades without producing the same recipient memory. Natural/artificial and active/passive describe different axes. A vaccine is artificial active immunity; an antibody injection is artificial passive immunity.

Recall: Which antibody protects mucosal surfaces, and which crosses the placenta?

Secretory IgA protects mucosal surfaces; IgG crosses the placenta.

Common trap: Not every IgA molecule is a dimer, and passive antibodies are not a vaccine.

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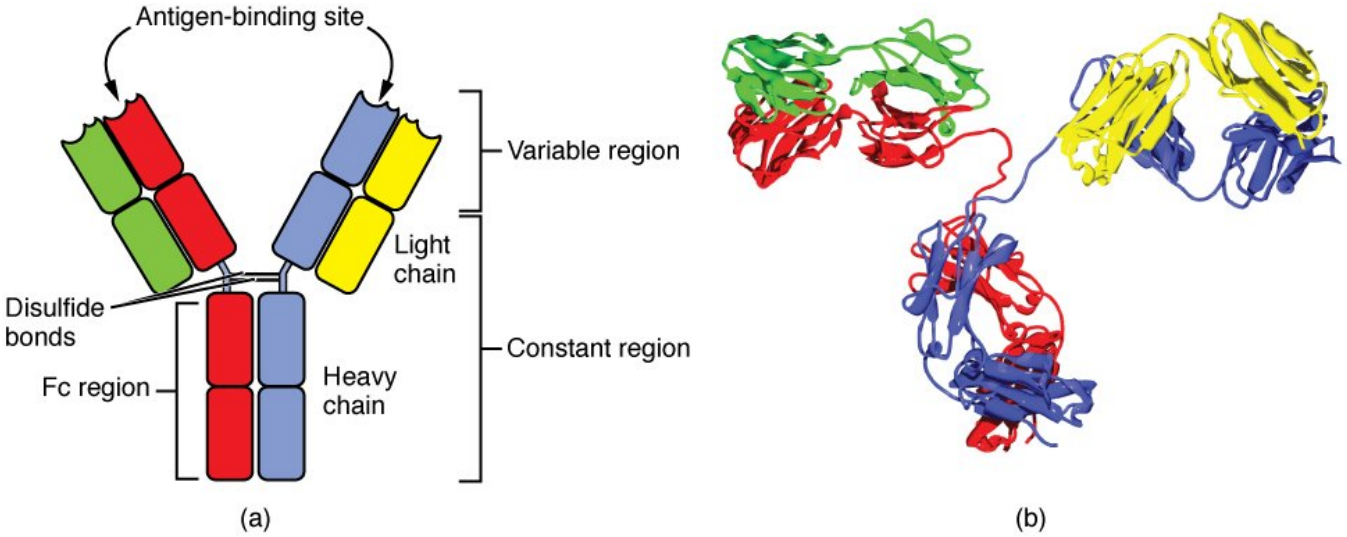
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Antibody and IgG2 Structures



Identify heavy/light chains, variable antigen-binding regions and the Fc region. The source credits panel (b) to a modification of work by Tim Vickers.

OpenStax A&P 2e, Fig. 21.21 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-4-the-adaptive-immune-response-b-lymphocytes-and-antibodies | Publisher original; 970 x 386 pixels

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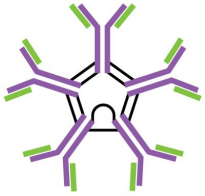
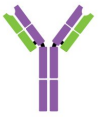
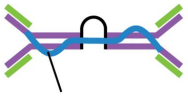
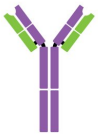
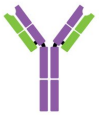
[Core] Five Classes of Antibodies

The Five Immunoglobulin (Ig) Classes					
	IgM pentamer	IgG monomer	Secretory IgA dimer	IgE monomer	IgD monomer
					
Heavy chains	μ	γ	α	ε	δ
Number of antigen binding sites	10	2	4	2	2
Molecular weight (Daltons)	900,000	150,000	385,000	200,000	180,000
Percentage of total antibody in serum	6%	80%	13%	0.002%	1%
Crosses placenta	no	yes	no	no	no
Fixes complement	yes	yes	no	no	no
Fc binds to		phagocytes		mast cells and basophils	
Function	Main antibody of primary responses, best at fixing complement; the monomer form of IgM serves as the B cell receptor	Main blood antibody of secondary responses, neutralizes toxins, opsonization	Secreted into mucus, tears, saliva, colostrum	Antibody of allergy and antiparasitic activity	B cell receptor

Compare antibody classes. Secreted IgM is generally pentameric, secretory IgA commonly dimeric, and the other pictured classes monomeric.

OpenStax A&P 2e, Fig. 21.22 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-4-the-adaptive-immune-response-b-lymphocytes-and-antibodies | Publisher original; 2242 x 2100 pixels

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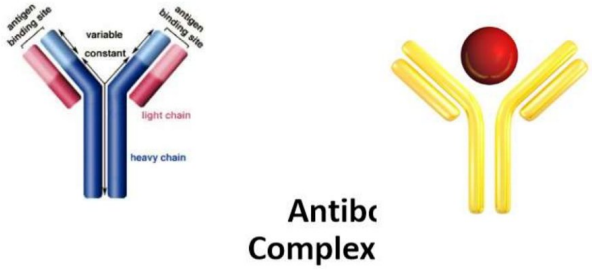
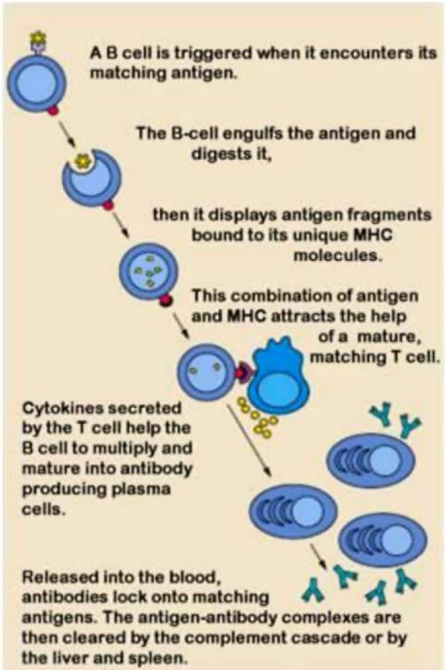
[Core] Antibody binding and B-cell activation

- **B cells**
- **Plasma Cells -produce antibodies**



- **Helper T Cells**
- **Memory Cells**



Locate antibody antigen-binding tips and follow B-cell activation to plasma and memory cells. Antibody binding and helper signals are distinct events.

S23 p.58 | Original labels retained; figure region enlarged.

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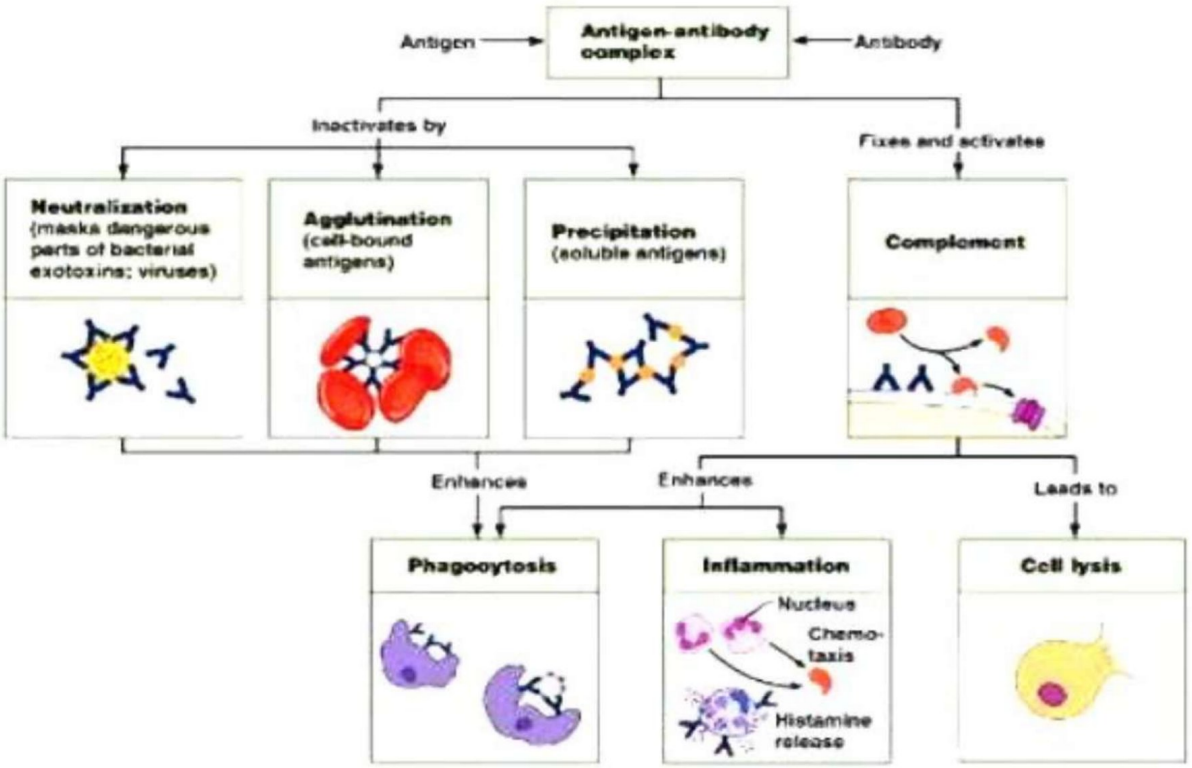
[Core] Antibody effector mechanisms



Follow neutralization, clumping and complement recruitment. Opsonization promotes uptake by phagocytes; antibodies do not directly eat a pathogen.

S23 p.59 | Original labels retained; figure region enlarged.

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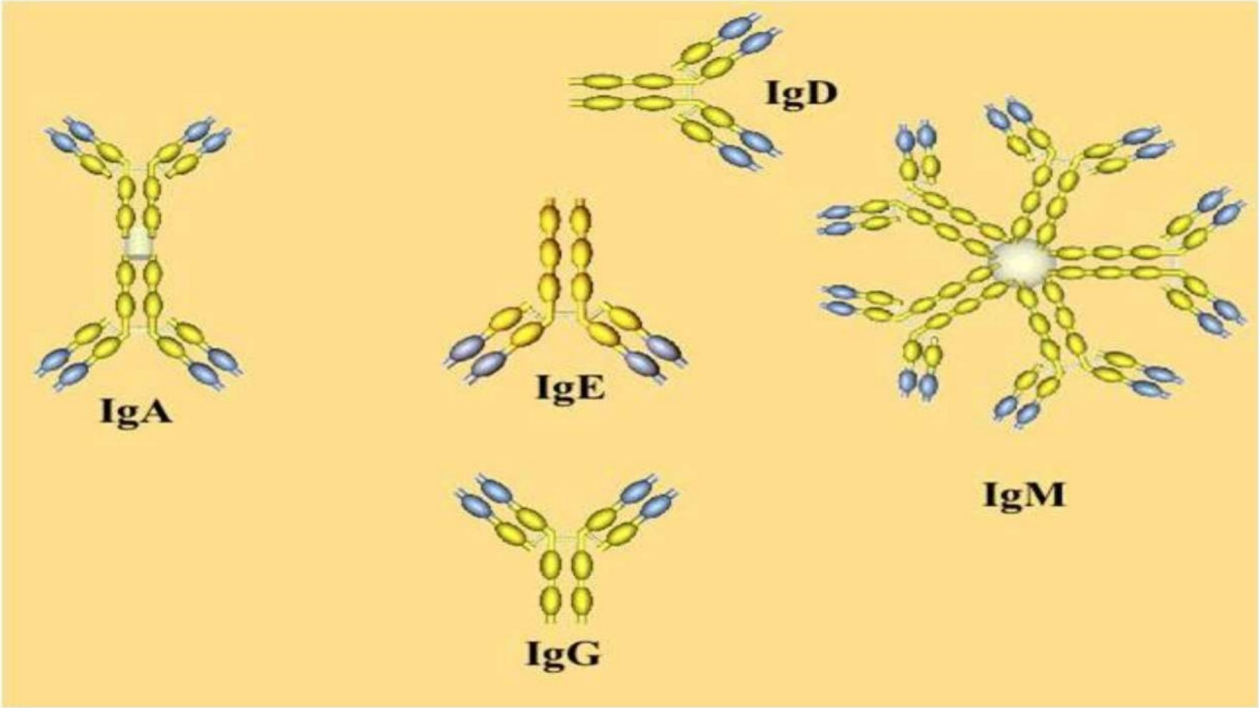
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Five immunoglobulin classes



Compare monomeric IgG, IgD and IgE, commonly dimeric secretory IgA, and pentameric secreted IgM. The membrane form of IgM is monomeric.

S23 p.60 | Original labels retained; figure region enlarged.

[Core] Antibody class-function table

IgA	**Antibodies are dimmers – contain two Y shaped structures. Found in mucosal areas, such as the gut, respiratory tract and urogenital tract. Also found in saliva, tears, and breast milk. They attack microbes and prevents colonization by pathogens before they reach the blood stream so it is most important antibody in local immunity**
IgD	**Functions mainly as an antigen receptor on B cells that have not been exposed to antigens. It has been shown to activate basophils and mast cells to produce antimicrobial factors.**
IgG	**In its four forms, provides the majority of antibody-based immunity against invading pathogens. It makes up about 75 % of all human antibodies and is the body's major defense against bacteria. The only antibody capable of crossing the placenta to give passive immunity to fetus. It is the most versatile of antibodies because it carries out functions of the other antibodies as well.**
IgE	**Binds to allergens and triggers histamine release from mast cells and basophils, and is involved in allergy. Also protects against parasitic worms.**
IgM	**Expressed on the surface of B cells and in a secreted form with very high avidity. Eliminates pathogens in the early stages of B cell mediated (humoral) immunity before there is sufficient IgG.**

Pair IgA with mucosal secretions, IgE with immediate allergy/parasites, IgG with serum and placental transfer, IgM with early responses, and IgD with naive B-cell receptors.

S23 p.61 | Original labels retained; figure region enlarged.

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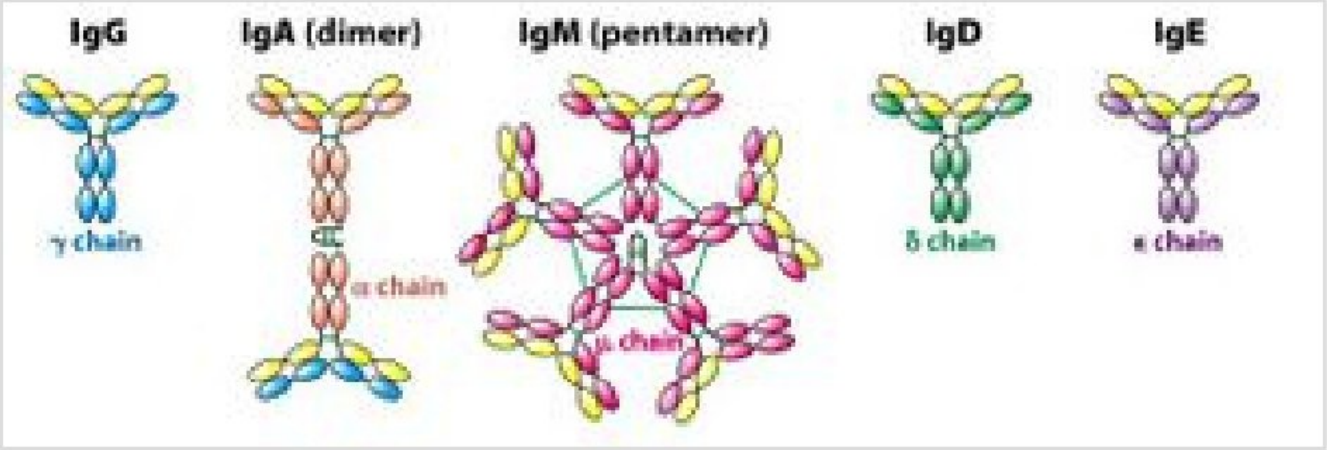
[Core] Immunoglobulin structures



Compare antibody shapes and number of linked units. Shape helps identify class but function depends on the Fc region and tissue context.

WI p.5 | Original labels retained; figure region enlarged.

I-RESP | Response sequence, memory and allergy

[Core] | Rule 3c.iv

A useful sequence is barrier breach -> innate recognition/inflammation -> antigen delivery/presentation -> activation and expansion of specific lymphocytes -> effector response -> contraction and memory. These events overlap; innate defenses continue while adaptive immunity develops. Clonal selection means antigen favors activation of cells whose receptors already fit, rather than instructing all cells to create a matching receptor.

On an antibody-versus-time graph, a primary response has a longer lag and often a smaller peak. A secondary response to the same antigen is typically quicker, stronger and longer lasting, reflecting memory and affinity maturation/class switching. A different antigen still requires its own primary response. Read axes, exposure times and whether antibody concentration is plotted on a linear or logarithmic scale.

In a typical IgE-mediated allergy, initial sensitization leads to IgE binding high-affinity receptors on mast cells. On re-exposure, allergen cross-links cell-bound IgE and triggers degranulation. Histamine and other mediators can cause itching, swelling, mucus secretion and bronchoconstriction. Not every allergic or hypersensitivity disorder is IgE-mediated; contact dermatitis is a T-cell example.

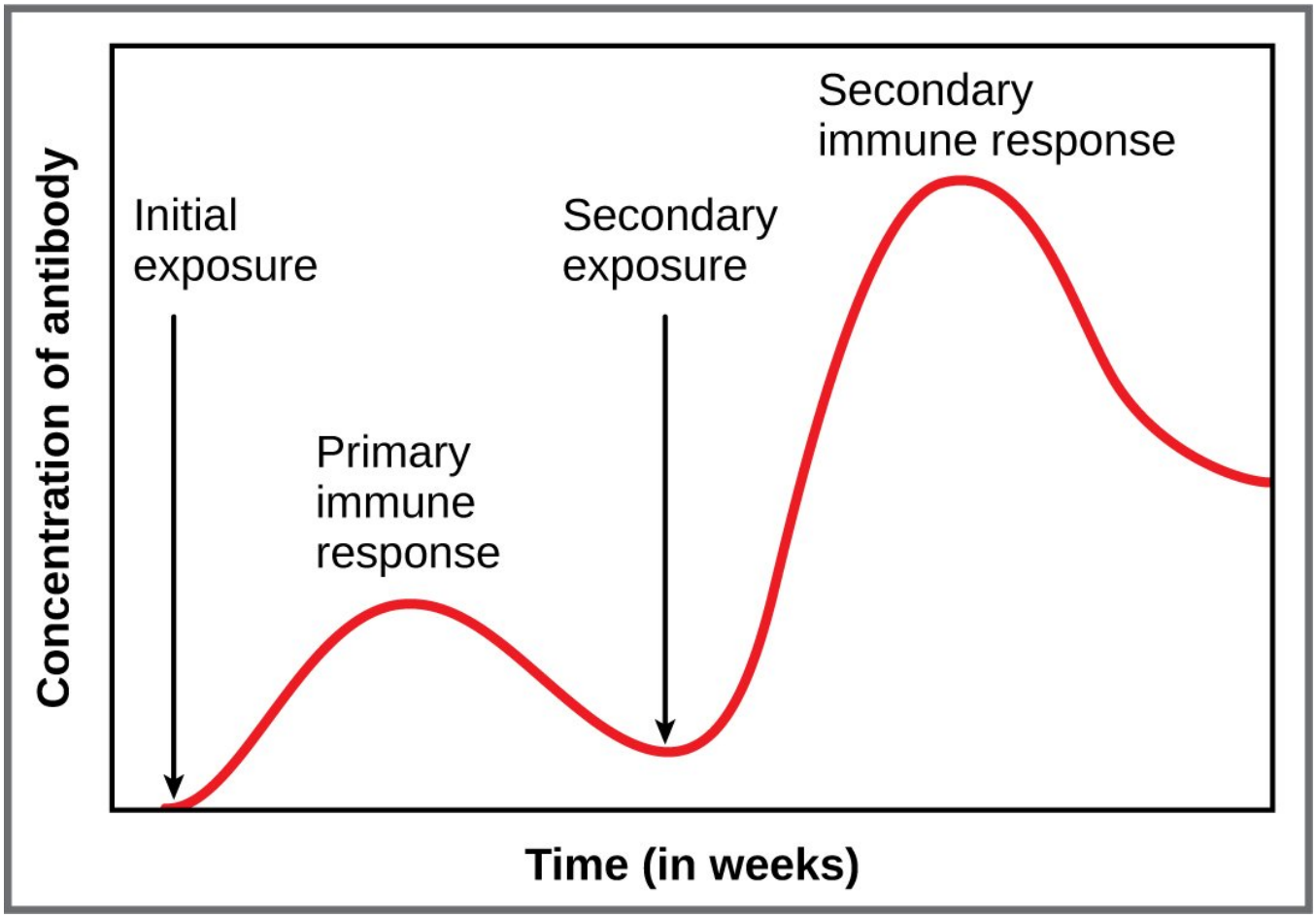
Recall: What is different when a memory B-cell response encounters the same antigen again?

A shorter lag, more rapid expansion and usually a stronger, higher-affinity antibody response.

Common trap: A second exposure to a different antigen is not automatically a secondary response to that antigen.

Access for free at https://openstax.org/books/anatomy-and-physiology-2e/pages/1-introduction
OpenStax-derived sections and original notes: CC BY-NC-SA 4.0. Supplied source figures retain original credits.

[Core] Primary and Secondary Antibody Responses



Compare the repeat response to antigen A with a first response to antigen B. Memory is antigen-specific; a new antigen still produces a primary response.

OpenStax A&P 2e, Fig. 21.24 | https://openstax.org/books/anatomy-and-physiology-2e/pages/21-4-the-adaptive-immune-response-b-lymphocytes-and-antibodies | Publisher original; 1088 x 756 pixels

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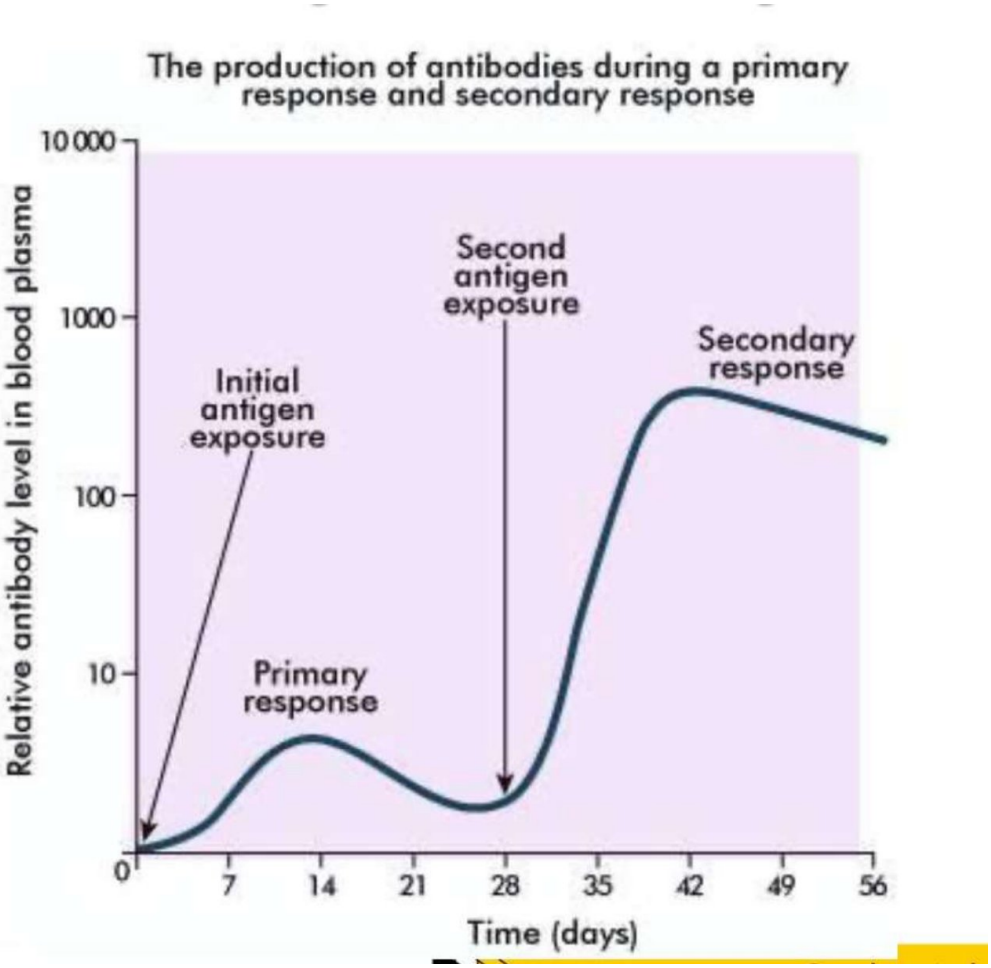
[Core] Primary and secondary antibody response



Identify the exposure times, lag, peak height and persistence. Secondary responses to the same antigen are usually faster and stronger because memory cells are present.

S23 p.64 | Original labels retained; figure region enlarged.

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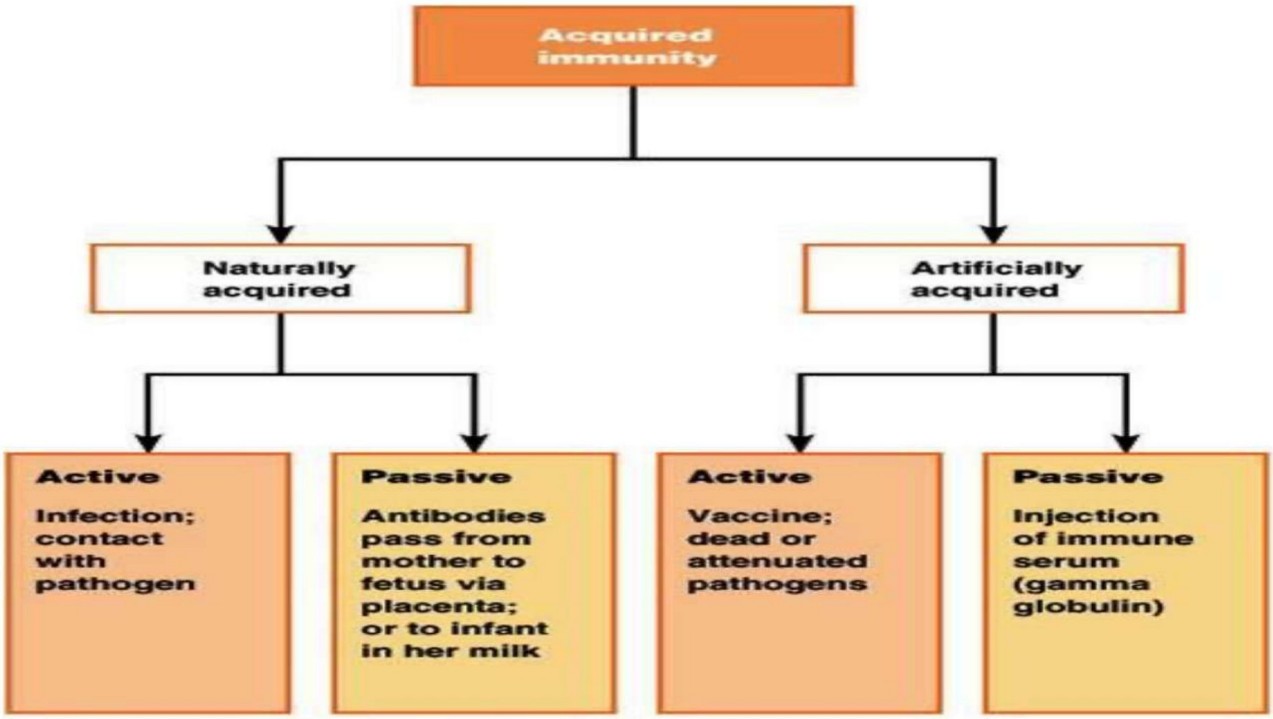
[Core] Natural/artificial and active/passive immunity



Active immunity uses the recipient's response and can form memory. Passive immunity transfers antibodies and gives immediate temporary protection; it does not create recipient memory by itself.

S23 p.65 | Original labels retained; figure region enlarged.

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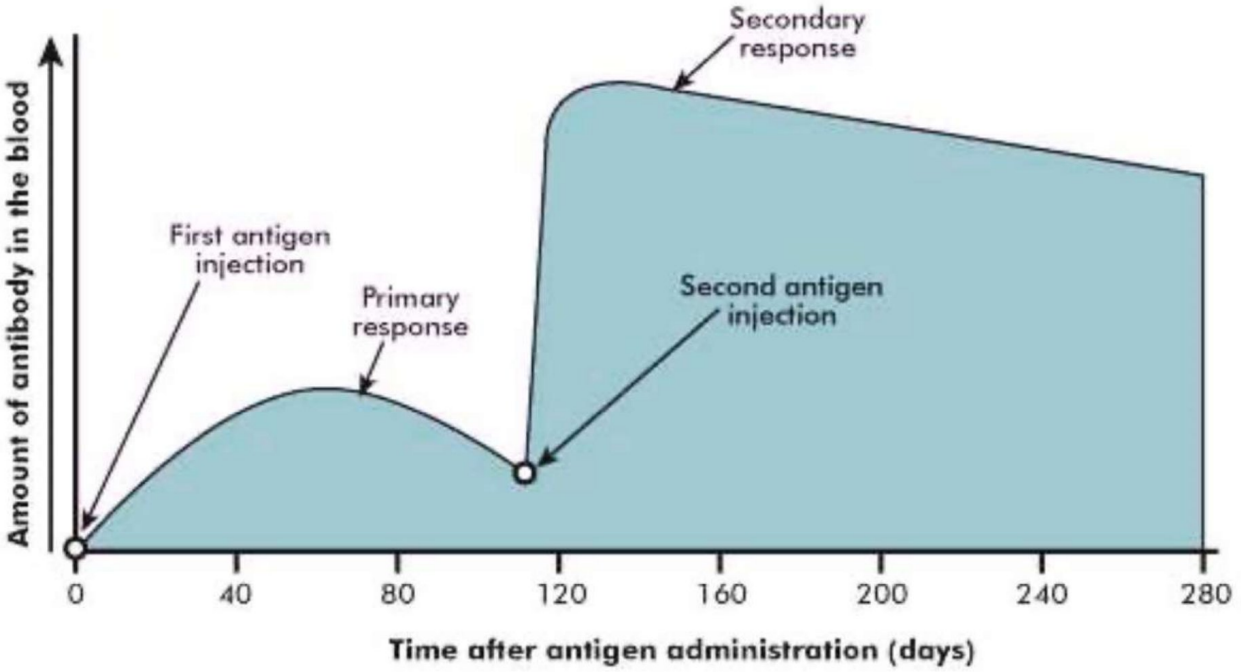
[Core] Immunization and antibody response



Compare the first and repeat exposure. Interpret axes before judging magnitude: this is a schematic, not a universal vaccination schedule or measured clinical curve.

S23 p.66 | Original labels retained; figure region enlarged.

I-HYP | Four hypersensitivity mechanisms

[Core] | Rule 3c.v

Type I: immediate IgE-mediated mast-cell activation; examples include anaphylaxis and many forms of urticaria. Local wheals are not the same as systemic anaphylaxis with potentially dangerous airway/circulatory effects. Type II: IgG/IgM against cell-surface or matrix targets, causing destruction, inflammation or altered receptor function. Myasthenia gravis impairs neuromuscular acetylcholine-receptor function; Graves disease stimulates the TSH receptor.

Type III: soluble antigen-antibody complexes form and deposit, activating complement and inflammation. Serum sickness is a systemic example; an Arthus reaction is a localized immune-complex reaction in a sensitized person. Type IV: sensitized T cells cause delayed inflammation or cytotoxic injury; contact dermatitis is a classic example. No antibody is required for the type IV mechanism.

Use mechanism first, then timing and example. A rapid reaction alone does not establish type I. A receptor antibody is type II even without cell lysis. Types are useful models; complex autoimmune diseases can involve more than one mechanism. Learn the rule's named examples individually rather than assigning every autoimmune disorder to one oversimplified category.

Recall: Compare serum sickness with contact dermatitis.

Serum sickness: type III immune complexes/complement. Contact dermatitis: type IV T-cell-mediated delayed inflammation.

Common trap: Type II does not always destroy cells; it can stimulate or block receptors.

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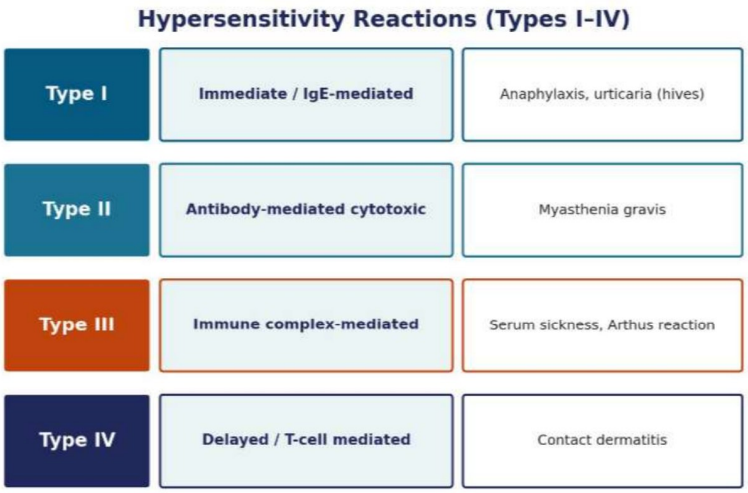
[Core] Hypersensitivity types I-IV



The four hypersensitivity types, mechanism and a classic example

Match mechanism to example: I IgE/mast cells; II antibodies against cell or receptor targets; III immune complexes; IV T cells. Not every allergic reaction is type I.

S27 p.35 | Original labels retained; figure region enlarged.

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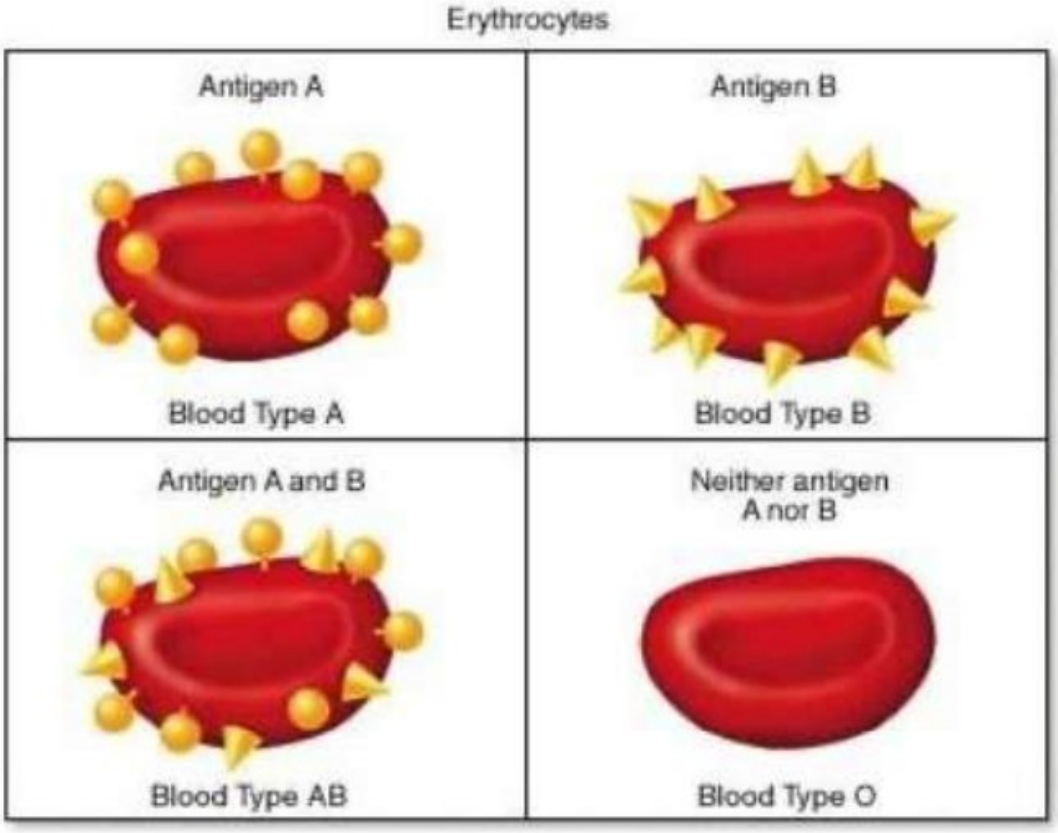
[E] enrichment ABO antigen patterns



A and B designate red-cell surface antigens. Type O lacks A/B antigens, not all antigens. Antibody-mediated incompatible transfusion illustrates a type II mechanism.

S23 p.73 | Original labels retained; figure region enlarged.

I-GRAFT | Transplants and prevention of rejection

[S] [N] | Rule 3c.vi

An autograft moves tissue within the same person; an allograft transfers tissue between genetically different people of the same species. An autograft avoids the usual alloimmune mismatch, but can still fail from infection, poor blood supply or other non-rejection causes.

Allograft rejection reflects recognition of donor antigens, particularly HLA/MHC, by recipient immune mechanisms. Hyperacute rejection involves pre-existing antibodies and vascular injury; acute rejection can involve T cells and/or antibodies; chronic rejection develops progressive vascular and fibrotic injury. Timelines are approximate, and mechanisms matter more than memorizing exact day cutoffs.

ABO compatibility where relevant, HLA matching and crossmatching help reduce risk. Immunosuppressive combinations can include calcineurin inhibitors, antiproliferative agents and corticosteroids. Suppression protects the graft but raises infection and some cancer risks; matching reduces rather than eliminates rejection. Most solid-organ allografts require ongoing suppression, but not every tissue transplant follows an identical regimen.

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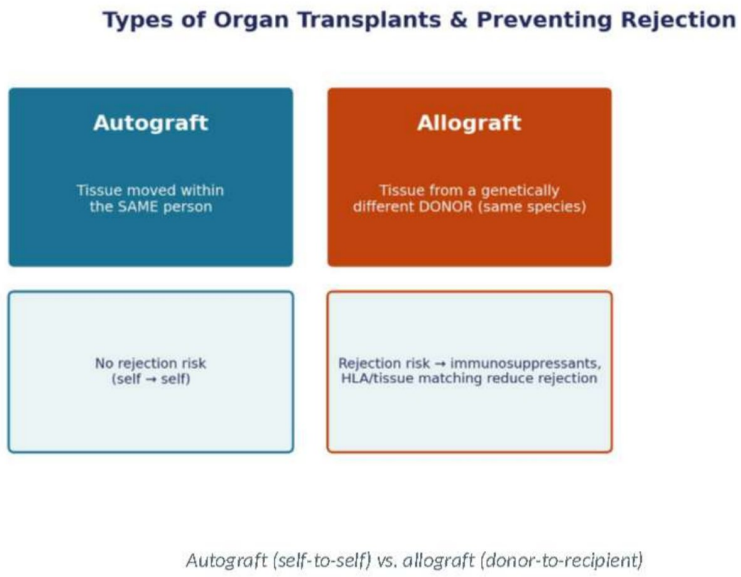
Recall: Why is matching alone insufficient to prevent every allograft rejection?

Matching reduces antigen differences but cannot eliminate all immune recognition; immunosuppression and monitoring are often still needed.

Common trap: Autograft means same individual, not merely the same species.

[S] [N] Autograft and allograft



Autograft (self-to-self) vs. allograft (donor-to-recipient)

Autograft uses the same person and avoids alloimmune rejection; allograft comes from another person of the same species. The source phrase no rejection risk refers to alloimmune rejection, not freedom from infection or surgical failure.

S27 p.36 | Original labels retained; figure region enlarged.

I-THER | Cancer immunotherapy: antibodies and CAR-T

[S] [N] | Rule 3c.vii

Monoclonal antibodies bind selected targets. Some mark tumor cells for immune destruction, block growth signals or carry a toxic payload. Checkpoint-blocking antibodies remove inhibitory signals on immune cells, increasing antitumor T-cell activity; they can also produce immune-related inflammation in normal organs.

CAR-T therapy modifies T cells to express a chimeric antigen receptor that recognizes a chosen surface antigen, then expands and administers those cells. In the common autologous approach the cells come from the patient. The receptor combines target recognition with intracellular activation. This is a living cellular therapy, unlike giving a vial of antibody protein.

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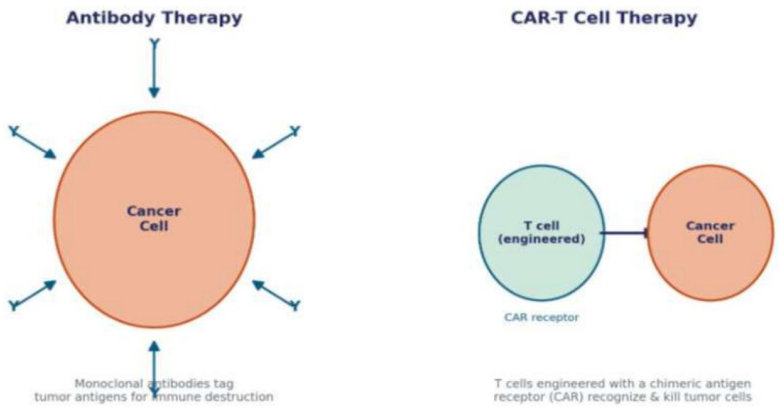
Target selection matters: normal cells sharing the antigen can be affected, and tumors can escape by losing the target. Cytokine release syndrome and neurologic toxicity are important potential complications. These are broad treatment principles for the event, not instructions for choosing therapy; efficacy depends strongly on cancer type, target and clinical setting.

Recall: What is engineered in CAR-T, and how does it differ from an antibody drug?

T cells are engineered with a receptor and infused as cells; an antibody drug delivers antibody protein targeting a molecule.

Common trap: Checkpoint inhibitors release immune inhibition; they are not simply general immune suppression.

[S] [N] Antibody therapy and CAR-T cells



Antibody therapy and CAR-T cell therapy, simplified

An antibody binds a target antigen; engineered CAR-T cells recognize selected surface antigens and kill target cells. Treatment selectivity does not eliminate adverse effects.

S27 p.37 | Original labels retained; figure region enlarged.

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Immune diseases

HIV/AIDS

[Core] recognition; [S] [N] treatment/prevention

Mechanism: HIV infection progressively damages CD4 T-cell-mediated coordination; AIDS is an advanced clinical/immunologic stage.

Recognition clues: Opportunistic infections become more likely with severe immune loss.

Distinguish: Acquired immunodeficiency, not a genetic B-cell disorder.

[S] [N] Treatment: Combination antiretroviral therapy and prevention/treatment of associated infections.

[S] [N] Prevention: Prevent exposure through established blood/sexual/perinatal prevention measures; appropriate prophylaxis and sustained treatment reduce transmission.

Common variable immunodeficiency (CVID)

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Impaired effective antibody production leads to low immunoglobulins and poor specific responses in a heterogeneous disorder.

Recognition clues: Recurrent infections, often respiratory; some people develop autoimmunity.

Distinguish: Predominantly antibody dysfunction versus severe combined T/B dysfunction in SCID.

[S] [N] Treatment: Immunoglobulin replacement when indicated and infection management.

[S] [N] Prevention: No universal prevention of the underlying disorder; reduce infection risk through individualized care.

Severe combined immunodeficiency (SCID)

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Genetic defects cause profound T-cell dysfunction with impaired adaptive function, including B-cell responses; patterns vary.

Recognition clues: Severe early infections and failure to thrive can occur.

Distinguish: A family of disorders, not one mutation or identical cell count pattern.

[S] [N] Treatment: Early protective care and hematopoietic stem-cell transplantation; selected forms use gene/enzyme therapy.

[S] [N] Prevention: Newborn screening and early treatment reduce complications; inherited disease is not prevented by ordinary lifestyle changes.

Multiple sclerosis

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Immune injury to central nervous system myelin and related structures disrupts conduction.

Recognition clues: Neurologic deficits varying with lesion location and time.

Distinguish: Targets CNS myelin, not the neuromuscular acetylcholine receptor.

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[S] [N] Treatment: Disease-modifying immune therapies, relapse treatment and rehabilitation.

[S] [N] Prevention: No certain universal prevention; therapies aim to reduce relapses/progression.

Rheumatoid arthritis

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Chronic immune-mediated synovial inflammation damages joints and can affect other organs.

Recognition clues: Inflammatory joint swelling/stiffness; often symmetric small-joint involvement.

Distinguish: Not simply mechanical wear-and-tear osteoarthritis.

[S] [N] Treatment: Disease-modifying antirheumatic drugs, including selected biologics; symptom control and rehabilitation.

[S] [N] Prevention: No guaranteed prevention; early disease control limits damage.

Hashimoto thyroiditis

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Autoimmune thyroid injury commonly reduces thyroid hormone production.

Recognition clues: Hypothyroid physiology, such as fatigue and cold intolerance, may develop.

Distinguish: Contrast Graves receptor stimulation and excess hormone.

[S] [N] Treatment: Thyroid hormone replacement when hypothyroidism is present.

[S] [N] Prevention: No established universal prevention; monitoring identifies hormone deficiency.

Graves' disease

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Antibodies stimulate TSH receptors, increasing thyroid hormone production.

Recognition clues: Hyperthyroid physiology such as heat intolerance and rapid pulse; eye involvement can occur.

Distinguish: Functional type II antibody effect rather than simple destruction.

[S] [N] Treatment: Antithyroid medicines, radioiodine or surgery depending on context; symptom control.

[S] [N] Prevention: No established universal prevention; monitoring/treatment reduce complications.

Anaphylaxis

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Systemic immediate hypersensitivity can produce airway narrowing and vascular leakage/hypotension.

Recognition clues: Rapid multisystem reaction; skin symptoms may be absent.

Distinguish: More severe and systemic than isolated hives.

[S] [N] Treatment: Epinephrine is first-line emergency treatment; supportive emergency care follows. Antihistamines do not replace it.

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[S] [N] Prevention: Identify and avoid relevant triggers and use a clinician-provided emergency plan.

Urticaria

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Mast-cell mediators cause transient itchy wheals; causes include allergic and nonallergic mechanisms.

Recognition clues: Raised itchy skin lesions that often move/disappear.

Distinguish: Not every hive is IgE allergy and not every hive episode is anaphylaxis.

[S] [N] Treatment: Antihistamines and cause-directed care; evaluate systemic symptoms separately.

[S] [N] Prevention: Avoid established triggers where possible; many cases have no single preventable cause.

Myasthenia gravis

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Autoantibodies impair neuromuscular signaling, commonly through acetylcholine receptors.

Recognition clues: Fatigable skeletal-muscle weakness; ocular/bulbar symptoms may occur.

Distinguish: Type II functional antibody disease; differs from CNS demyelination in MS.

[S] [N] Treatment: Acetylcholinesterase inhibitors, immune-directed therapies and selected thymectomy.

[S] [N] Prevention: No universal primary prevention; treatment reduces weakness/complications.

Arthus reaction

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Local immune-complex deposition activates complement and inflammation in a sensitized person.

Recognition clues: Localized inflammatory reaction after antigen exposure.

Distinguish: Type III; serum sickness is the systemic comparison.

[S] [N] Treatment: Avoid further relevant exposure and manage inflammation according to severity.

[S] [N] Prevention: Avoid identified causative exposures when appropriate.

Serum sickness

[Core] recognition; [S] [N] treatment/prevention

Mechanism: Systemic immune-complex disease following exposure to particular foreign proteins or medicines.

Recognition clues: Fever, rash and joint symptoms may follow a delay.

Distinguish: Type III immune complexes, unlike immediate IgE anaphylaxis.

[S] [N] Treatment: Remove the trigger and provide severity-appropriate anti-inflammatory/supportive care.

[S] [N] Prevention: Document/avoid causative agents when alternatives are appropriate.

Contact dermatitis

[Core] recognition; [S] [N] treatment/prevention

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Mechanism: Allergic contact dermatitis is delayed T-cell-mediated inflammation after skin sensitization.

Recognition clues: Itchy eczematous rash at exposure sites.

Distinguish: Type IV allergy differs from nonimmune irritant dermatitis.

[S] [N] Treatment: Avoid the allergen, protect skin and use appropriate topical anti-inflammatory treatment.

[S] [N] Prevention: Exposure identification and protective measures.

Systemic lupus erythematosus

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Systemic autoimmunity can cause immune-complex injury in multiple tissues.

Recognition clues: Skin, joints, kidneys and other organs may be involved.

Distinguish: Type III mechanisms are important but do not explain every lupus manifestation.

[S] [N] Treatment: Hydroxychloroquine and organ/severity-directed immune-modifying drugs, including selected biologics.

[S] [N] Prevention: No guaranteed prevention; trigger management, sun protection and monitoring reduce some flares/complications.

Dermatomyositis

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Immune-mediated inflammatory muscle and skin disease.

Recognition clues: Proximal muscle weakness plus characteristic rash.

Distinguish: Skin plus muscle disease, unlike isolated psoriasis.

[S] [N] Treatment: Immune-directed treatment, rehabilitation and monitoring for associated conditions.

[S] [N] Prevention: No established universal prevention.

Scleroderma

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Autoimmunity, vascular dysfunction and excess connective-tissue deposition cause skin and sometimes organ fibrosis.

Recognition clues: Skin thickening; systemic forms may affect vessels, lungs and GI tract.

Distinguish: Fibrosis is a central feature, not just a superficial inflammatory rash.

[S] [N] Treatment: Organ-specific therapy and selected immune/fibrosis-directed treatments; management is individualized.

[S] [N] Prevention: No universal prevention; early organ monitoring is important.

Psoriasis

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Immune-mediated inflammation accelerates epidermal turnover and produces plaques.

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Recognition clues: Raised scaly plaques; nail changes can occur.

Distinguish: Skin disease does not automatically mean joint involvement.

[S] [N] Treatment: Topical treatments, phototherapy and selected systemic/biologic drugs.

[S] [N] Prevention: No guaranteed prevention; trigger management and ongoing treatment reduce activity.

Psoriatic arthritis

[S] [N] recognition; [S] [N] treatment/prevention

Mechanism: Inflammatory joint/enthesis disease associated with psoriasis.

Recognition clues: Joint pain/swelling, enthesitis or swollen digits can occur.

Distinguish: Adds musculoskeletal disease to the psoriasis spectrum; not every person with psoriasis develops it.

[S] [N] Treatment: Disease-modifying/biologic therapies and rehabilitation tailored to disease pattern.

[S] [N] Prevention: No universal prevention; early treatment helps limit joint damage.

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Source corrections

Scope

AP_RULE_2027 B3-B4 explicitly says State & National Level Only. The compact slide decks do not override it.

Gas carriage

CO2 is transported predominantly as bicarbonate, not predominantly as carbonic acid. Percentages are approximations.

Barrier

The blood-air barrier contains alveolar epithelium and capillary endothelium with thin intervening structures, not one cell total.

Bile

Bile emulsifies fat and supports micelles; lipase hydrolyzes triglycerides. Bile is not an enzyme.

Water

Most total water uptake occurs in the small intestine; the colon recovers remaining water.

Marrow

Red marrow is the main blood-cell-production site; yellow marrow is predominantly fatty.

Spirometry

Simple spirometry cannot directly measure RV, FRC or TLC. VC is not maximum total lung volume.

Type II

Antibodies may destroy cells OR change receptor function; Graves and myasthenia illustrate functional effects.

Innate recognition

Innate immunity recognizes patterns; saying it treats every microbe identically is too strong.

Allergy

IgE explains type I allergy, not all hypersensitivity; type IV contact allergy is T-cell-mediated.

Clinical details

Old tournament keys can contain outdated drug indications. Learn broad rule-level mechanisms/treatment categories; do not use these notes for personal treatment.

Saved wiki scope

The three saved Scioly PDF snapshots are supplemental reading. Their older Nationals headings and extra disease lists do not override AP_RULE_2027. Ataxia-telangiectasia, APS, Burkitt lymphoma and DiGeorge syndrome are enrichment rather than named 2027 Division B diseases.

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Digestive wiki: organ identity

WD p.2 incorrectly associates pancreatic juice with the gallbladder: the pancreas produces pancreatic juice. WD p.5 calls Peyer patches lymph nodes; they are aggregated lymphoid nodules.

Digestive wiki: appendix

WD p.12 places McBurney point on the left. The classic landmark is on the right, one-third of the distance from the right anterior superior iliac spine to the umbilicus. Tenderness alone is not a diagnostic test.

Respiratory wiki: compliance

WR pp.8-9 generalizes decreased compliance to COPD. Emphysema typically increases compliance while decreasing elastic recoil; fibrosis decreases compliance.

Respiratory wiki: transport

WR pp.7-8 contains misleading wording about tissue hemoglobin, complete oxygen dissociation and chloride exchange. Oxygen unloads from red-cell hemoglobin; venous hemoglobin remains partly saturated. Chloride/bicarbonate exchange preserves electrical neutrality.

Immune wiki: selection and organs

WI pp.4-7 oversimplifies lymphocyte activation and tonsil function. Receptors precede clonal selection; tonsils provide mucosal immune surveillance. The thymus is the main site of T-cell maturation.

Older disease statistics and treatments

Do not memorize saved-wiki survival figures, screening ages or drug lists as universal current facts. Use the rule-aligned disease summaries; the original wiki images retain their original, sometimes outdated wording.

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