ANATOMY & PHYSIOLOGY

2027 detailed review

Respiratory | Digestive | Immune

For two eighth-grade Division B students. Core means regional subject matter. State & National includes every additional topic marked that way in AP_RULE_2027.pdf (B3-B4).

Use this book with Review Hub.html: searchable concepts, flashcards, source-page image gallery and question index. The source gallery retains original diagrams and labels; the notes explain mechanisms and correct known source errors.

How to study

For each topic: read the mechanism; redraw a structure or graph; answer the retrieval question without notes; explain the common trap; then practice matched questions. For diseases, trace cause -> cell/tissue change -> organ dysfunction -> symptoms, then compare treatment and prevention principles.

Scope and evidence

The supplied Division B rule controls scope. The requested Division C webpage is a resource directory, not a substitute rule. The YR3 deck says it was prepared from draft rules. The three locally saved Scioly wiki PDFs are included as supplemental sources WD, WI and WR. Their original diagrams are preserved in the image gallery; source corrections are listed near the end of these notes. Histology is included as anatomy/practical application and requested enrichment; the rule does not supply a finite list of microscopic slides.

Source key

S27 = AP_Respiratory, Digestive, Immune Systems_Slides2027.pdf

S23 = AP_Respiratory, Digestive, Immune Systems_Slides2023.pdf

YR3 = 1-27 A&P-YR3_PDF.pdf

RES = 3-27A&P Textbook_Web Resources.pdf

DIG = 6-Digestive-System-and-How-It-Works.pdf

RESP = 7-Introduction-to-the-Respiratory-System.pdf

IMM = 8-Immune-system.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

Rule-to-note coverage checklist

Respiratory

3a.i | R-AIR | Air pathway and respiratory functions | Core

3a.i | R-TISSUE | Lungs, pleura and respiratory histology | Core

3a.ii | R-VENT | Pressure, volume, airflow and ventilation calculations | Core

3a.iii | R-PAT | Patterns of breathing | Core

3a.ii,iv | R-SPIRO | Spirograms, lung volumes and obstructive/restrictive patterns | Core

3a.v | R-GAS | Gas exchange, diffusion and ventilation-perfusion matching | Core

3a.v | R-HB | Oxygen transport, dissociation curves and carbon dioxide | Core

3a.vi | R-EX | Exercise and high altitude | Core

3a.ix | R-CTRL | Breathing control and acid-base chemistry | State & National

Disease recognition: 3a.vii-viii. Treatment/prevention for ALL listed diseases: 3a.x (State & National).

Digestive

3b.i,ii | D-ANAT | Food pathway, organs and functions | Core

3b.iii | D-WALL | Four gut-wall layers and enteric nerves | Core

3b.iv | D-HIST | Compare esophagus, stomach, small intestine and colon | Core

3b.v | D-MECH | Mechanical digestion, propulsion and chemical digestion | Core

3b.vi | D-CARB | Carbohydrate digestion and absorption | Core

3b.vi | D-PROT | Protein digestion, acid and gastric protection | Core

3b.vi | D-FAT | Fat digestion, micelles and lymphatic absorption | Core

3b.x; 3b.ii,vi foundations | D-LIVER | Liver, pancreas, Kupffer cells and digestive regulation | State & National

3b.vii | D-EX | Exercise, obesity and digestive function | Core

Disease recognition: 3b.viii-ix. Treatment/prevention for ALL listed diseases: 3b.xi (State & National).

Immune

3c.i | I-ORG | Immune organs and histology | Core

3c.ii | I-INN | Innate barriers, receptors and immune cells | Core

3c.ii | I-COMP | Complement and inflammation | Core

3c.iii | I-ADAPT | Adaptive immunity, antigen presentation and tolerance | Core

3c.iii,iv | I-AB | Antibodies and sources of immunity | Core

3c.iv | I-RESP | Response sequence, memory and allergy | Core

3c.v | I-HYP | Four hypersensitivity mechanisms | Core

3c.vi | I-GRAFT | Transplants and prevention of rejection | State & National

3c.vii | I-THER | Cancer immunotherapy: antibodies and CAR-T | State & National

Disease recognition: 3c.v,viii. Treatment/prevention for ALL listed diseases: 3c.ix (State & National).

RESPIRATORY

R-AIR | Air pathway and respiratory functions

Core | Rule 3a.i | S27 pp.5; S23 pp.5-9; YR3 pp.8-12; RESP pp.1-2; supplementary reading WR pp.1-4

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.

R-TISSUE | Lungs, pleura and respiratory histology

Core | Rule 3a.i | S27 pp.5,9; S23 pp.6-7; YR3 pp.9-10; supplementary reading WR pp.3-4

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.

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

Core | Rule 3a.ii | S27 p.6; S23 pp.10,13; YR3 pp.13,16; RESP p.2; supplementary reading WR pp.4-5

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 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.

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.

R-PAT | Patterns of breathing

Core | Rule 3a.iii | S27 p.7; S23 p.11; YR3 p.14; supplementary reading WR pp.5-6

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 CO_2 production, lowering arterial CO_2; hypoventilation means inadequate alveolar ventilation for CO_2 production, raising CO_2. 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.

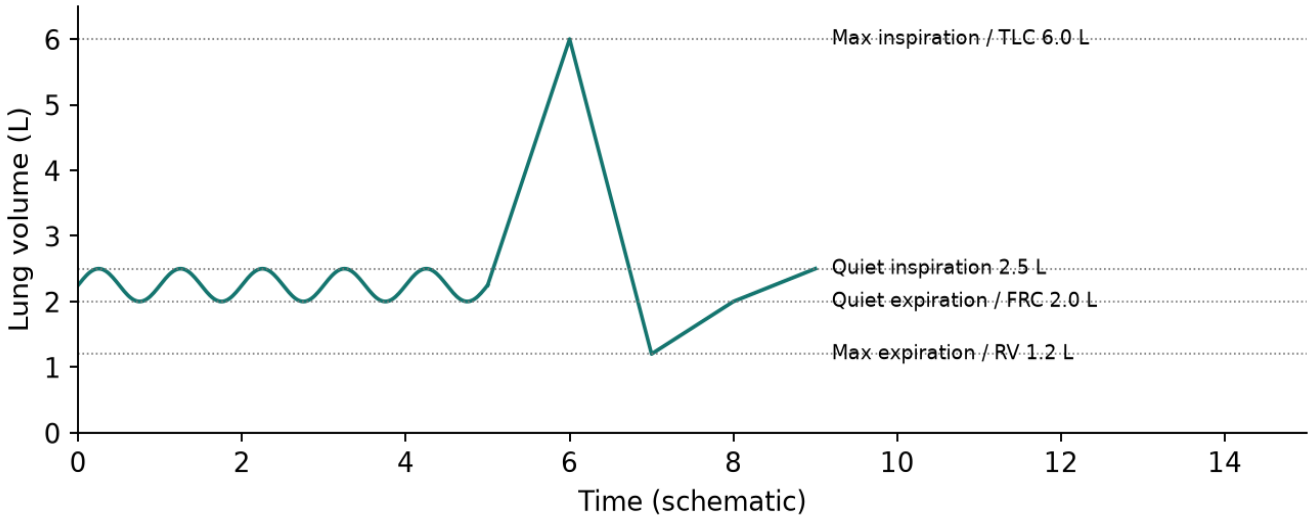
R-SPIRO | Spirograms, lung volumes and obstructive/restrictive patterns

Core | Rule 3a.ii,iv | S27 pp.6,8; S23 pp.12-14; YR3 pp.15-17; supplementary reading WR p.5

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.



Teaching spirogram: TV=0.5, IRV=3.5, ERV=0.8, RV=1.2 L. VC=4.8 and TLC=6.0 L. RV is supplied by the teaching model, not directly measured by simple spirometry.

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.

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

Core | Rule 3a.v | S27 p.9; S23 pp.16-17; YR3 pp.19-20; supplementary reading WR pp.7-8

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 principle 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.

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 rest.

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.

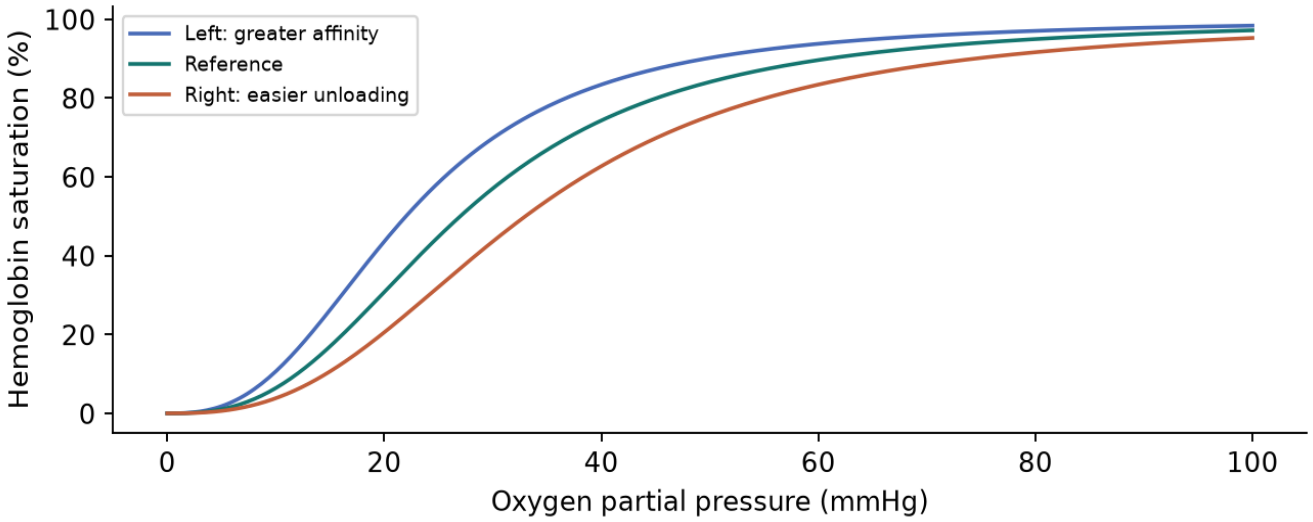
R-HB | Oxygen transport, dissociation curves and carbon dioxide

Core | Rule 3a.v | S27 p.10; S23 pp.16-18; YR3 pp.19-21; supplementary reading WR pp.7-8

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.

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



Illustrative Hill curves. Compare saturation at the SAME PO2. More CO2/H+, heat or 2,3-BPG favors unloading/right shift. Saturation is not total 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.

R-EX | Exercise and high altitude

Core | Rule 3a.vi | S27 p.11; YR3 p.22; supplementary reading WR pp.6-7

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

State & National | Rule 3a.ix | S27 p.14; S23 pp.15,18; YR3 pp.18,21; supplementary reading WR pp.6,12

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.

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.

Respiratory disease review

Recognition level is shown for each disease. Treatment and prevention depth for every disease is State & National. These are competition-study principles, not personal medical instructions.

R-DIS-01 | COPD

Core recognition | Rule 3a.vii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

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

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

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

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

R-DIS-02 | Asthma

Core recognition | Rule 3a.vii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

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

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

Treatment: Inhaled corticosteroid-containing treatment controls inflammation; bronchodilators relieve constriction.

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

R-DIS-03 | Emphysema

Core recognition | Rule 3a.vii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

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

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

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

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

R-DIS-04 | Pneumonia

Core recognition | Rule 3a.vii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

Recognition: Cough, fever, breathlessness; alveolar consolidation.

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

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

Prevention: Appropriate vaccines, hand hygiene and reducing aspiration risk where applicable.

R-DIS-05 | Sleep apnea

Core recognition | Rule 3a.vii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

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

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

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

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

R-DIS-06 | Cystic fibrosis

Core recognition | Rule 3a.vii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

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

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

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

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

R-DIS-07 | Tuberculosis

State & National recognition | Rule 3a.viii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

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

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

Treatment: Multiple appropriate antibiotics together, selected for susceptibility and clinical context.

Prevention: Airborne infection control, detection/treatment of infection and vaccination where indicated.

R-DIS-08 | Pulmonary edema

State & National recognition | Rule 3a.viii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

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

Distinguish: Fibrosis is scar tissue; edema is fluid.

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

Prevention: Manage contributing conditions; prevention depends on cause.

R-DIS-09 | Pleurisy

State & National recognition | Rule 3a.viii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

Recognition: Sharp pain worsened by inspiration or cough.

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

Treatment: Treat the underlying cause and control inflammation/pain as appropriate.

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

R-DIS-10 | Small-cell lung cancer

State & National recognition | Rule 3a.viii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

Recognition: Lung mass with possible systemic/paraneoplastic effects.

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

Treatment: Chemotherapy, radiotherapy and/or immunotherapy according to stage and clinical context.

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

R-DIS-11 | Non-small-cell lung cancer

State & National recognition | Rule 3a.viii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

Recognition: 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.

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

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

R-DIS-12 | Pulmonary fibrosis

State & National recognition | Rule 3a.viii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

Recognition: Restrictive physiology, low TLC and exertional breathlessness.

Distinguish: Opposite compliance tendency from emphysema.

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

Prevention: Reduce harmful exposures where relevant; many forms cannot be reliably prevented.

R-DIS-13 | Silicosis

State & National recognition | Rule 3a.viii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

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

Distinguish: Identify silica rather than coal dust or asbestos.

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

Prevention: Engineering controls, dust suppression and properly selected respiratory protection.

R-DIS-14 | Coal workers' pneumoconiosis

State & National recognition | Rule 3a.viii; treatment 3a.x | S27 pp.12-15; YR3 p.23

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

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

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

Treatment: Reduce further exposure, supportive respiratory care and treat complications.

Prevention: Dust monitoring, ventilation and exposure controls.

R-DIS-15 | Asbestosis

State & National recognition | Rule 3a.viii; treatment 3a.x | S27 pp.12-15; YR3 p.23

Mechanism: Inhaled asbestos fibers cause interstitial fibrosis.

Recognition: 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.

Treatment: Supportive care and prevention of further exposure; cancer evaluation when indicated.

Prevention: Safe asbestos management by qualified personnel and workplace controls.

DIGESTIVE

D-ANAT | Food pathway, organs and functions

Core | Rule 3b.i,ii | S27 pp.17-18; YR3 pp.25-37; DIG pp.1-3; supplementary reading WD pp.1-6

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.

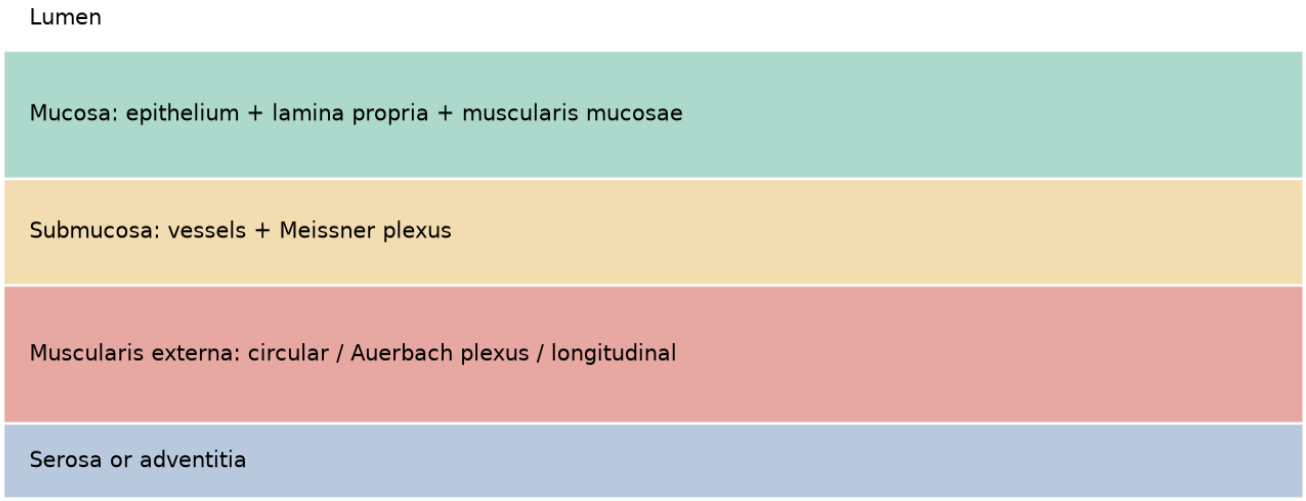
D-WALL | Four gut-wall layers and enteric nerves

Core | Rule 3b.iii | S27 p.19; S23 digestive anatomy; YR3 digestive section; supplementary reading WD pp.2-5

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.



Schematic layers from lumen outward; the muscularis mucosae is part of mucosa. The myenteric plexus sits between muscle layers of muscularis externa, not in the submucosa.

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

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

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

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

Core | Rule 3b.iv | S27 p.20; YR3 pp.31-37; histology resources listed in RES p.1; supplementary reading WD pp.4-6

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 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.

D-MECH | Mechanical digestion, propulsion and chemical digestion

Core | Rule 3b.v | S27 p.21-22; YR3 pp.26,38-39; DIG pp.1-2; supplementary reading WD pp.2-7

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 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.

D-CARB | Carbohydrate digestion and absorption

Core | Rule 3b.vi | S27 p.22; YR3 p.39; DIG p.2; supplementary reading WD pp.6-8

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.

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.

D-PROT | Protein digestion, acid and gastric protection

Core | Rule 3b.vi | S27 pp.20,22; YR3 pp.33,39; DIG p.2; supplementary reading WD pp.6-8

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.

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.

D-FAT | Fat digestion, micelles and lymphatic absorption

Core | Rule 3b.vi | S27 p.22; YR3 pp.35-39; DIG p.2; supplementary reading WD pp.6-8

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.

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.

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

State & National | Rule 3b.x; 3b.ii,vi foundations | S27 p.26; YR3 pp.35-36; DIG pp.2-3; supplementary reading WD pp.6-8

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.

D-EX | Exercise, obesity and digestive function

Core | Rule 3b.vii | S27 p.23; YR3 pp.41-44; supplementary reading WD pp.8-9

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.

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.

Digestive disease review

Recognition level is shown for each disease. Treatment and prevention depth for every disease is State & National. These are competition-study principles, not personal medical instructions.

D-DIS-16 | Stomach and duodenal ulcers / peptic ulcer disease

Core recognition | Rule 3b.viii; treatment 3b.xi | S27 pp.24-27; YR3 p.40

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

Recognition: 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.

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

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

D-DIS-17 | Digestive-system cancers

Core recognition | Rule 3b.viii; treatment 3b.xi | S27 pp.24-27; YR3 p.40

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

Recognition: 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.

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

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

D-DIS-18 | Diarrhea

Core recognition | Rule 3b.viii; treatment 3b.xi | S27 pp.24-27; YR3 p.40

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

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

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

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

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

D-DIS-19 | Lactose intolerance

Core recognition | Rule 3b.viii; treatment 3b.xi | S27 pp.24-27; YR3 p.40

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

Recognition: Bloating, gas and diarrhea after lactose exposure.

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

Treatment: Adjust lactose intake or use lactase while maintaining nutritional adequacy.

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

D-DIS-20 | Hepatitis

Core recognition | Rule 3b.viii; treatment 3b.xi | S27 pp.24-27; YR3 p.40

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

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

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

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

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.

D-DIS-21 | Appendicitis

Core recognition | Rule 3b.viii; treatment 3b.xi | S27 pp.24-27; YR3 p.40

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

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

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

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

Prevention: No reliable universal prevention; early recognition reduces complications.

D-DIS-22 | Diverticular disease

State & National recognition | Rule 3b.ix; treatment 3b.xi | S27 pp.24-27; YR3 p.40

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

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

Distinguish: Diverticulosis does not necessarily mean active infection.

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

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

D-DIS-23 | GERD

State & National recognition | Rule 3b.ix; treatment 3b.xi | S27 pp.24-27; YR3 p.40

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

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

Distinguish: Esophageal reflux is not a gastric ulcer.

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

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

D-DIS-24 | Ulcerative colitis

State & National recognition | Rule 3b.ix; treatment 3b.xi | S27 pp.24-27; YR3 p.40

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

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

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

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

Prevention: No established universal primary prevention; monitoring and treatment reduce complications.

D-DIS-25 | Crohn's disease

State & National recognition | Rule 3b.ix; treatment 3b.xi | S27 pp.24-27; YR3 p.40

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

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

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

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

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

D-DIS-26 | Celiac disease

State & National recognition | Rule 3b.ix; treatment 3b.xi | S27 pp.24-27; YR3 p.40

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

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

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

Treatment: Strict gluten-free diet with nutritional assessment and follow-up.

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

IMMUNE

I-ORG | Immune organs and histology

Core | Rule 3c.i | S27 p.29; YR3 pp.46-50; IMM pp.9-11; supplementary reading WI p.7

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.

I-INN | Innate barriers, receptors and immune cells

Core | Rule 3c.ii | S27 p.30; YR3 pp.51-55; IMM pp.3-8,11; supplementary reading WI pp.1-4

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 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.

I-COMP | Complement and inflammation

Core | Rule 3c.ii | S27 p.30; YR3 pp.54-56; IMM pp.3,11; supplementary reading WI pp.2-4

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.

I-ADAPT | Adaptive immunity, antigen presentation and tolerance

Core | Rule 3c.iii | S27 p.31; YR3 pp.57-65; IMM p.4; supplementary reading WI pp.4-6

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 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.

I-AB | Antibodies and sources of immunity

Core | Rule 3c.iii,iv | S27 pp.31-32; YR3 pp.60-68; supplementary reading WI pp.5-6

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.

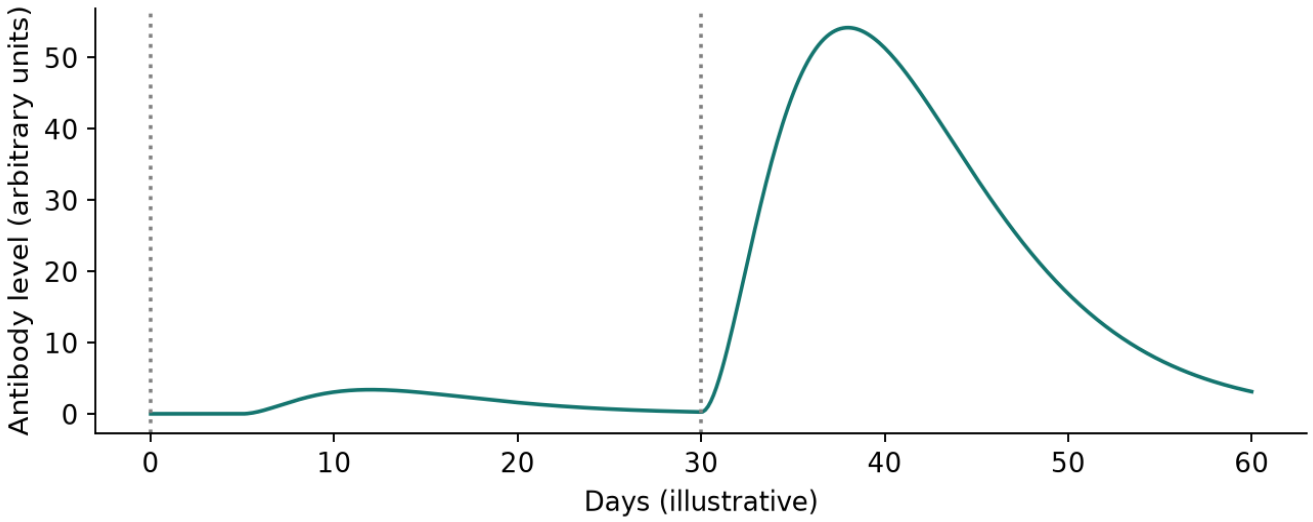
I-RESP | Response sequence, memory and allergy

Core | Rule 3c.iv | S27 p.32; YR3 pp.56-68,71; supplementary reading WI pp.4-6

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.



First exposure at day 0; repeat exposure to the same antigen at day 30. Memory produces a shorter lag and higher response. Exact times and heights are not universal.

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.

I-HYP | Four hypersensitivity mechanisms

Core | Rule 3c.v | S27 p.35; YR3 p.71; supplementary reading WI pp.7-8

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.

I-GRAFT | Transplants and prevention of rejection

State & National | Rule 3c.vi | S27 p.36; supplementary reading WI p.16

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.

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.

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

State & National | Rule 3c.vii | S27 p.37; YR3 p.70

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.

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.

Immune disease review

Recognition level is shown for each disease. Treatment and prevention depth for every disease is State & National. These are competition-study principles, not personal medical instructions.

I-DIS-27 | HIV/AIDS

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

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

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

Treatment: Combination antiretroviral therapy and prevention/treatment of associated infections.

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

I-DIS-28 | Common variable immunodeficiency (CVID)

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

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

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

Treatment: Immunoglobulin replacement when indicated and infection management.

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

I-DIS-29 | Severe combined immunodeficiency (SCID)

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

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

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

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

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

I-DIS-30 | Multiple sclerosis

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

Recognition: Neurologic deficits varying with lesion location and time.

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

Treatment: Disease-modifying immune therapies, relapse treatment and rehabilitation.

Prevention: No certain universal prevention; therapies aim to reduce relapses/progression.

I-DIS-31 | Rheumatoid arthritis

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

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

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

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

Prevention: No guaranteed prevention; early disease control limits damage.

I-DIS-32 | Hashimoto thyroiditis

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

Mechanism: Autoimmune thyroid injury commonly reduces thyroid hormone production.

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

Distinguish: Contrast Graves receptor stimulation and excess hormone.

Treatment: Thyroid hormone replacement when hypothyroidism is present.

Prevention: No established universal prevention; monitoring identifies hormone deficiency.

I-DIS-33 | Graves' disease

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

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

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

Treatment: Antithyroid medicines, radioiodine or surgery depending on context; symptom control.

Prevention: No established universal prevention; monitoring/treatment reduce complications.

I-DIS-34 | Anaphylaxis

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

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

Distinguish: More severe and systemic than isolated hives.

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

Prevention: Identify and avoid relevant triggers and use a clinician-provided emergency plan.

I-DIS-35 | Urticaria

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

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

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

Treatment: Antihistamines and cause-directed care; evaluate systemic symptoms separately.

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

I-DIS-36 | Myasthenia gravis

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

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

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

Treatment: Acetylcholinesterase inhibitors, immune-directed therapies and selected thymectomy.

Prevention: No universal primary prevention; treatment reduces weakness/complications.

I-DIS-37 | Arthus reaction

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

Recognition: Localized inflammatory reaction after antigen exposure.

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

Treatment: Avoid further relevant exposure and manage inflammation according to severity.

Prevention: Avoid identified causative exposures when appropriate.

I-DIS-38 | Serum sickness

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

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

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

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

Prevention: Document/avoid causative agents when alternatives are appropriate.

I-DIS-39 | Contact dermatitis

Core recognition | Rule 3c.v; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

Recognition: Itchy eczematous rash at exposure sites.

Distinguish: Type IV allergy differs from nonimmune irritant dermatitis.

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

Prevention: Exposure identification and protective measures.

I-DIS-40 | Systemic lupus erythematosus

State & National recognition | Rule 3c.viii; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

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

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

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

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

I-DIS-41 | Dermatomyositis

State & National recognition | Rule 3c.viii; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

Mechanism: Immune-mediated inflammatory muscle and skin disease.

Recognition: Proximal muscle weakness plus characteristic rash.

Distinguish: Skin plus muscle disease, unlike isolated psoriasis.

Treatment: Immune-directed treatment, rehabilitation and monitoring for associated conditions.

Prevention: No established universal prevention.

I-DIS-42 | Scleroderma

State & National recognition | Rule 3c.viii; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

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

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

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

Prevention: No universal prevention; early organ monitoring is important.

I-DIS-43 | Psoriasis

State & National recognition | Rule 3c.viii; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

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

Recognition: Raised scaly plaques; nail changes can occur.

Distinguish: Skin disease does not automatically mean joint involvement.

Treatment: Topical treatments, phototherapy and selected systemic/biologic drugs.

Prevention: No guaranteed prevention; trigger management and ongoing treatment reduce activity.

I-DIS-44 | Psoriatic arthritis

State & National recognition | Rule 3c.viii; treatment 3c.ix | S27 pp.33-39; YR3 pp.71-73

Mechanism: Inflammatory joint/enthesis disease associated with psoriasis.

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

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

Treatment: Disease-modifying/biologic therapies and rehabilitation tailored to disease pattern.

Prevention: No universal prevention; early treatment helps limit joint damage.

Histology practical: identify, justify, predict

Start at low magnification: find lumen, capsule, layers and repeated units. Then identify epithelium/cell shapes, glands, cartilage, muscle or lymphoid regions. Give two supporting features and one function. Never diagnose from stain color alone. The gallery contains supplied source figures; linked virtual slides provide real microscopy rather than synthetic images.

Trachea

Look for: Pseudostratified ciliated epithelium, goblet cells and cartilage. Function: Filter/move mucus while supporting an open airway. Contrast: Bronchiole lacks cartilage.

Concept R-TISSUE | https://histologyguide.com/slidebox/17-respiratory-system.html

Bronchiole

Look for: Smooth muscle around a small epithelial airway; no cartilage. Function: Control airflow resistance. Contrast: A vessel has endothelium and may contain red cells.

Concept R-TISSUE | https://histologyguide.com/slidebox/17-respiratory-system.html

Alveolar lung

Look for: Numerous thin-walled air spaces and fine septa. Function: Large area and short diffusion distance. Contrast: Emphysema removes septa; fibrosis thickens them.

Concept R-TISSUE | https://histologyguide.com/slidebox/17-respiratory-system.html

Esophagus

Look for: Stratified squamous lining; no villi. Function: Protect against abrasion. Contrast: Stomach has simple columnar surface and pits.

Concept D-HIST | https://histologyguide.com/slidebox/14-gastrointestinal-tract.html

Stomach

Look for: Gastric pits leading to glands; no villi. Function: Mucus protection and acid/enzyme secretion. Contrast: Do not identify gastric pits as villi.

Concept D-HIST | https://histologyguide.com/slidebox/14-gastrointestinal-tract.html

Duodenum

Look for: Villi plus submucosal Brunner glands. Function: Absorption and alkaline mucus protection. Contrast: Jejunum lacks Brunner glands.

Concept D-HIST | https://histologyguide.com/slidebox/14-gastrointestinal-tract.html

Jejunum

Look for: Prominent folds/villi without characteristic Brunner glands or aggregated Peyer patches. Function: Large absorptive surface. Contrast: Small fields can omit regional landmarks.

Concept D-HIST | https://histologyguide.com/slidebox/14-gastrointestinal-tract.html

Ileum

Look for: Villi and often aggregated Peyer patches. Function: Absorption and gut immune surveillance. Contrast: Colon lacks villi.

Concept D-HIST | https://histologyguide.com/slidebox/14-gastrointestinal-tract.html

Colon

Look for: Straight crypts, abundant goblet cells, no villi. Function: Mucus lubrication and remaining water/electrolyte recovery. Contrast: Not small intestine with absorptive villi.

Concept D-HIST | https://histologyguide.com/slidebox/14-gastrointestinal-tract.html

Liver

Look for: Hepatocyte plates, sinusoids, central vein and portal regions. Function: Metabolism, bile secretion and Kupffer-cell surveillance. Contrast: Blood and bile follow different pathways.

Concept D-LIVER | https://histologyguide.com/

Pancreas

Look for: Dark exocrine acini with paler endocrine islets. Function: Enzyme secretion versus hormone secretion. Contrast: Islets have no ducts for digestive secretions.

Concept D-LIVER | https://histologyguide.com/

Lymph node

Look for: Follicles in cortex, deeper paracortex/medulla and sinuses. Function: Filter lymph and organize adaptive responses. Contrast: Spleen has red/white pulp rather than this cortex-medulla arrangement.

Concept I-ORG | https://histologyguide.com/slidebox/10-lymphoid-system.html

Spleen

Look for: White pulp among abundant red pulp. Function: Blood surveillance and aged red-cell removal. Contrast: Not a lymph filter like a lymph node.

Concept I-ORG | https://histologyguide.com/slidebox/10-lymphoid-system.html

Thymus

Look for: Lobules with dark cortex, lighter medulla; Hassall corpuscles. Function: T-cell maturation and selection. Contrast: Normal thymus lacks lymph-node-type follicles.

Concept I-ORG | https://histologyguide.com/slidebox/10-lymphoid-system.html

Red bone marrow

Look for: Mixed developing blood cells, fat spaces and large megakaryocytes. Function: Hematopoiesis, including lymphocyte origins. Contrast: Yellow marrow is more fatty.

Concept I-ORG | https://histologyguide.com/

Neutrophil versus lymphocyte

Look for: Neutrophil has multilobed nucleus; small lymphocyte has dense round nucleus and scant cytoplasm. Function: Rapid phagocytosis versus lymphocyte functions. Contrast: B/T subsets cannot reliably be identified by ordinary smear appearance alone.

Concept I-INN | https://histologyguide.com/

Source corrections and exam cautions

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.

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.

Practice index and mock-test use

The 156 studio items have reviewed concept assignments and preserve their original IDs/options. Source-exam index entries are suggested mappings from extracted text; diagrams, shared stems, OCR and subparts must be checked against the linked original. The index is not a certified transcription or a replacement answer key. Use the supplied source key for source-exam scoring and record disputed or outdated answers for coach review.

Verified web resources

https://www.soinc.org/anatomy-and-physiology-c

https://www.nhlbi.nih.gov/health/lungs

https://www.nhlbi.nih.gov/health/asthma/treatment-action-plan

https://www.niddk.nih.gov/health-information/digestive-diseases

https://www.niams.nih.gov/health-topics

https://histologyguide.com/slidebox/17-respiratory-system.html

https://histologyguide.com/slidebox/14-gastrointestinal-tract.html

https://histologyguide.com/slidebox/10-lymphoid-system.html